SURGICAL COMPLICATIONS

Editor

Assoc. Prof. Dr. Serkan Arslan

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PREFACE

Dear readers; We are proud to present to you, our valuable readers, our book, "Surgical Complications", which consists of 15 chapters in different categories, current and covering many health departments in the field of medicine and health in health sciences.

This book has been written by academics who are experts in their field. In each section, you will be able to see up-to-date information on its own subject. In each chapter, the authors presented their theoretical knowledge on their subjects by blending a detailed literature knowledge and their own experiences. I believe that this information will be both a guide for scientists and surgeons who have taken a new step, and a foresight in terms of practical knowledge they can acquire on these issues.

We would like to thank the coordinators, referees, authors and publishing house for their valuable contributions.

Assoc. Prof. Dr. Serkan ARSLAN

Editor

CONTENTS

CHAPTER I

COMPLICATIONS OF LAPAROSCOPIC NEPHRECTOMY

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After it was first described by Clayman et al in 1991, the indications of laparoscopic kidney surgery have increased rapidly and it has become a standard procedure in many centers today (1). Although laparoscopic surgery was defined primarily for benign diseases of the kidney, a wide range of indications including radical nephrectomy and partial nephrectomy has emerged over time, with the development of hemostatic agents, the rapid development of surgical devices and imaging technologies in this area, and the adequate level of surgical technique. With the spread of laparoscopy and its clinical routine in many urological fields other than the kidney, an increase in complications secondary to surgery has been observed in parallel with this development, and many studies have been conducted in this field (2,3). In many studies, it has been reported that complications are concentrated in the early learning process of surgeons (4). Ultimately, the durability of a surgical procedure is based on its safety and decreased postoperative morbidity, defined by reported complications. The National Cancer Institute Common Toxicity Criteria (NCI-CTC) has been described as a tool to assess and compare complications(5). The NCI-CTC is described the complications and then divided it into grades according to specific criteria from one to five. According to this system, the five grades represent mild, moderate, severe, life-threatening, and death. In this section, in addition to the general complications of laparoscopic surgery, specific complications of laparoscopic nephrectomy and renal surgery will be discussed.

1. Common Complications

1.1. Rhabdomyolysis

Rhabdomyolysis in laparoscopic nephrectomy is usually associated with patient position. Long periods of time spent in the full flank position cause a certain amount of muscle tissue damage, and for this reason, the modified lateral decubitus position has been used recently (6). Rhabdomyolysis can lead to serious consequences, especially in patients with solitary kidneys, leading to permanent renal failure. Rhabdomyolysis should be suspected in the presence of skin changes and abnormal pain in the hip and thigh in the postoperative period. Urine myoglobin test is confirmatory, but early treatment with clinical findings is appropriate. Abundant hydration and alkalization of the urine can prevent kidney damage. Men with large muscle mass, obesity and prolonged operation time are important risk factors.

1.2. Nerve Injuries

Nerve damage in laparoscopic renal surgeries is reported in the literature at a rate of about 1-3% (7). In the lateral decubitus position, the brachial plexus may be damaged due to the pressure spreading to the axilla. In addition, if adequate support is not provided to the hands and feet in the lateral decubitus position, postoperative low hand or foot may be seen. All these complications can be prevented by providing soft and adequate support to the axilla, hands or feet while preparing the patient's position. Emergency neurology consultation should be made in all motor neuropathies that develop postoperatively. Sensory neuropathies are usually due to damage to nerves extending along the quadratus lumborum or psoas muscle during port incision or dissection (n.ilioinguinalis, n.iliohypogasticus, n.genitofemoralis) and are usually reversible. Oral gabapentin and amitriptyline may be helpful in accelerating recovery.

1.3. Gas Leakage

Gas leakage is a condition that is seen secondary to the size of the incision made for the hand port, especially in hand-assisted laparoscopy(8). However, this situation can also be seen in other laparoscopic procedures because the port incision is made large or the trocar cannot be fixed well. The size of the incision made is the most important factor. The incision should be wide enough to allow the trocar to enter and allow the surgeon's hand movements during the operation, maintain the stability of the working instruments at the entrance and exit, and contain skin, subcutaneous and disaster layers to prevent gas leakage (9).

1.4. Wound Complications

Although it can be seen in other laparoscopic procedures, wound complications are more common, especially in hand-assisted laparoscopy due to the size of the hand port (10). Periumbilical midline incision should be preferred in patients with normal body structure. However, in obese patients, paramedian incision provides more advantages than midline incision in reaching the ipsilateral kidney. Although complications such as hernia, wound dehiscence and wound infection are seen in all procedures, they are more common in hand-assisted laparoscopy (11). The incidence of incisional hernia in hand assisted laparoscopy ranges from 3% to 5% (12). In addition, wound complications of hand-assisted laparoscopy, especially wound infection, are more common in obese patients. Careful preoperative antibiotic therapy and irrigation of subcutaneous tissue to prevent infection may reduce the incidence of these complications.

1.5. Injuries Developing Secondary to Needle and Trocar

Complications associated with the use of trocars include bleeding, visceralinjury, andhernias. These complications are rare (lesst han1%) but may require conversion to the open procedure(13). Trocar insertion by Veress technique utilizes a needle to assess penetration into the peritoneum and allow for insufflation. Inherent to its blind insertion are potential perforation of abdominal wall vessels and associated bleeding or visceral injury damage. Transillumination and appreciation for epigastric vessel anatomy are critical to trocar placement. Bleeding-associated trocar insertion requires a thorough inspection of the peritoneum to rule out an intraabdominal vascular injury and if no intra-abdominal bleeding is encountered, a second trocar should be inserted to asist with the inspection of the initial trocar site for abdominal wall vessel bleeding. Abdominal wall vessel bleeding may require superficial or peritoneal electrocautery or suture repair. Careful visualization of the peritoneum after trocar insertion is imperative as serious intra-abdominal injury due to trocar placement may go unnoticed. Mases documented an injury to the aorta with the Veress needle that was detected nearly 60 min into surgery because of unexplained hypertension and a sudden decrease in end-tidal carbon dioxide (14).

Visceral injury should be suspected upon aspiration of blood or gastrointestinal contents upon placement of the Veress needle or if elevated insufflation pressures are required to maintain pneumoperitoneum. Should this occur, the Veress needle should be repositioned, trocar placement ensured,

and careful examination followed by laparoscopic or open repair of visceral perforation (14). Hasson trocar insertion may be preferred in patients who are at higher risk for intra-abdominal adhesions (from previous surgery or suppurative abdominal disease), although they are cumbersome and can be associated with air-leak. Open trocar insertion is also associated with higher incidence of incisional hernias. In general, all facial defects greater than 10 mm must be closed with sutures.

Advances in trocar design and insertion techniques are ongoing and may improve on already low complication rates associated with Veress needle insertion. Optical access trocar placement as described by Thomas, involves placement of a trocar through a clear blunt-tipped obturator with a recessed knife blade that can be deployed for controlled sharp dissection. Four complications were reported in 88 patients: two epigastric vessel injuries, one injury to the bowel, and one injury to the mesentery. Three of these injures were identified and repaired immediately with a re-operation required to repair an abdominal wall hematoma secondary to epigastric artery injury (15).

2. Complications Associated With Visual Area

2.1. Bowel injuries

Intestinal damage and thermal injuries at the entrance are seen at a rate of approximately 1.5% (16). It is recommended to use cold shears for column mobilization to prevent thermal induced column damage. The duodenum on the right side should be removed from the renal hilt, because the duodenum can be injured very easily and its complications can cause serious consequences. In minor serosal injuries, a few support sutures can be placed and the patient can be followed closely. In full-thickness injuries, general surgery consultation should be requested (17). The damaged area is closed twice with laparoscopic or open surgery, depending on the surgeon's experience. Avoiding the use of cautery in areas close to the colon and being careful especially when changing instruments are important factors in minimizing the risk of colon damage.

2.2. Spleen injuries

Less than 1.3% of spleen injuries were reported in left laparoscopic renal surgeries (16). Minor thermal injuries and damages can be controlled with hemostatic agents or compression (18). In larger injuries, supportive treatment with blood transfusion, embolizationb, or splenectomy may be necessary. Therefore, the patient should be followed closely (19).

2.3. Pleural injury

In left renal surgeries, pleural damage may occur during the release of the upper pole of the kidney from the spleen and the release of the liver from the upper pole on the right (20). In minor injuries, underwater drainage with a 14-gauge angiocatheter can be performed from the chest wall and the defect can be closed with absorbable sutures. For larger injuries, a chest tube is placed and thoracic surgery can perform an open repair if necessary.

2.4. Pancreatic injury

Pancreatic injury usually occurs during left nephrectomy when the tail of the pancreas is released to view the renal hilum. In pancreatic damage, the patient has postoperative abdominal pain, nausea, vomiting, fever and deterioration in laboratory values (21). While minor injuries can usually be improved by stopping oral feeding and conservative monitoring, general surgery consultation is required in more major injuries.

2.5. Hepatobiliary injury

Minor lacerations and thermal damage to the liver do not require primary repair. In large capsule lacerations and parenchymal damage, intervention with argoncoagulation or hemostatic agents may be necessary (22). In gallbladder damage, cholecystectomy may be necessary and general surgery consultation should be requested.

2.6. Vascular injuries

On the left, the gonadal vein drains into the renal vein. Especially in women, this vein can be wide and its injury during dissection can cause serious bleeding. This vessel can be controlled and cut with a stapler or vessel clip if necessary. The iliac vessels are also at risk during mobilization of the distal ureter or descending colon. Other vessels at risk are the inferior mesenteric artery, superior mesenteric artery, and inferior mesenteric vein. Since intestinal ischemia may develop in the superior mesenteric artery damage, cardiovascular surgery consultation should be requested. Another risk on the left side is aortic damage and requires open revision because laparoscopic repair of the aorta is difficult.

On the right side, the gonadal vein drains directly into the vena cava and its injury creates a greater problem. Therefore, the vein should be located distally and dissection should be done laterally. Since its wall is thinner and more fragile than the artery, sharp dissection or gentle blunt dissection should be performed

around the vena cava. In inferior vena cava injuries, pressure should be applied to the vena cava and repair should be done by quickly switching to open surgery (23).

3. Affects Of Laparoscopic Renal Surgery Type On Complication

3.1. Simple nephrectomy

Simple nephrectomy generally describes nephrectomy performed because the kidney becomes non-functional as a result of benign diseases. Many etiological factors such as xanthogranulomatous pyelonephritis or other infectious causes, reflux disease, stone disease, failed pyeloplasty, polycystic kidney disease and atrophic kidney can be evaluated in this context. It has been reported that the most common conversion to open surgery among laparoscopic kidney surgeries is during simple nephrectomy with a rate of 3.7% (16). The most common reason for this is the deterioration of the normal anatomical structure of the tissues and the renal unit due to inflammation and excessive adhesion to the surrounding tissues. Colon damage risk is 1% and bowel cleansing should be done before the operation (24). In cases where the tissues are adhered, it is necessary to see the vessels directed towards the colon and careful dissection to avoid colon and mesenteric vessel damage. The treatment approach in colon injuries depends on the extent of the damage. Small mesenteric openings should be closed because of the risk of internal hernia.

Another important problem in simple nephrectomy is the difficulty in releasing the renal hilum. Perihilar inflammation and tissue reaction secondary to hydronephrosis due to xanthogranulomatous pyelonephritis or renal stone disease makes dissection of this region very difficult. The renal artery and vein are controlled by placing a separate clip. However, in cases where adhesion to the reaction is high, the renal artery and vein can be ligated en-bloc with the help of a stapler(25). The surrounding of the renal hilum should be thinned sufficiently while the stapler is placed. If the reacted adherent tissues do not allow this, hand assisted laparoscopy or open surgery should be performed. If the periphery of the hilum is not sufficiently freed, the stapler may fail, resulting in serious vascular injury and bleeding(26). If the laparoscopic procedure cannot be continued in cases such as chronic infection, previous stone surgery or nephrostomy insertion, ureter surgeries, open surgery should be performed(27). In case of post-operative infection and secondary abscess, the abscess should be drained, if necessary, a drain should be placed in the lodge and appropriate antibiotic therapy should be administered.

3.2. Laparoscopic donor nephrectomy

LDN is a safe alternative to open nephrectomy for kidney transplantation. While complication rates have previously been reported to occur at about 6.4–16.5%,14,34 a contemporary review of 300 patients at the University of California Los Angeles found complications to occur in 4% of cases: 0.6% were intraoperative (major) and 3.3% were postoperative. It has been reported that only 1% of these complications are major. (28) Hand-assisted LDN has been described previously and is a common approach to donor nephrectomy. In addition to improvements in operative time and length of stay over open donor nephrectomy, the hand-assisted LDN cohort was found to have lower mean creatinine values at 7 days and one month postoperatively as well as lower urological complications and higher graft survival at one year postoperatively (29).

3.3. Radical nephrectomy

Port site metastases and local recurrences have been reported after laparoscopic radical nephrectomy. Port site metastases may develop secondary to tumor morcellation or may be related to the contact of the tumor to the port site during removal (30,31). It has been reported that correct morcellation minimizes the risk of metastasis (32). Although en-bloc removal of the tumor requires enlarging the incision area, it has the advantages that the macroscopic features of the tumor can be seen fully and its cellular properties can be better examined.

Renal cell carcinoma exhibits less scar tissue grafting characteristics than transitional cell carcinoma. Extreme care should be taken during morcellation or complete extraction to prevent the mass from scattering around. If the specimen is to be removed as a whole, placing the specimen in the specimen bag beforehand will prevent this risk. If morcellation is made, this should be done in a double layer bag and the site and port hole where the morcellation is made should be washed with sterile saline.

3.4. Laparoscopic partial nephrectomy

Laparoscopic partial nephrectomy (LPN) has emerged as an acceptable alternative procedure to open partial nephrectomy (OPN) (33). Early experience with LPN has been summarized by Rassweiler and was associated with a 33% complication rate (34).The most frequent complications reported were vascular/bleeding, followed by urinary leak. Complication rates for LPN is comparable to OPN. In a review of 200 patients undergoing OPN *vs* LPN, only

five intraoperative complications were noted in the 100 patients in the LPN cohort compared to zero in the OPN group (35). Although none of these patients required conversion to the open procedure, one patient with a solitary kidney did require re-operation and nephrectomy for on-going hemorrhage. Postoperative complications associated with the LPN group and the OPN group were 7/100 and 2/100, respectively. Three patients with urine leak and four patients with hemorrhage were observed in the LPN group. One ureteral obstruction was observed in the OPN group, but there were none in the LPN group. Reasons provided for increased likelihood of vascular complications in the LPN cohort were mainly technical and were described earlier in this study. In a retrospective review of the initial 200 patients undergoing LPN at the Cleveland Clinic, intraoperative, postoperative, and delayed complications occurred in 5.5%, 15%, and 12.5% of patients, respectively (36). Hemorrhage and urinary leak were the most frequently encountered urological complications in this study. Hemorrhagic complications were noted to improve with the use of Floseal (Hemostatic Matrix; Baxter International; Deerfield, Illinois, USA). Urine leaks were noted to occur in 4.5% of cases, the majority of which were managed with a double-J ureteral stent. These complication rates were noted to be comparable with or improved compared with previously reported OPN complication rates in this study. Non-urological complications counted as pulmonary, cardiovascular, gastro-intestinal, and sepsis were discovered in 5%, 4.5%, 2%, and 1% of cases (36). With comparable complication rates to OPN, LPN could offer the benefit of decreased LOS, pain medication requirement, and time to convalescence (35).

4. Conclusion

Today, the laparoscopic procedure has been accepted as the standard in nephrectomy performed for many benign and malignant diseases. The first step in establishing adequate technical skills and understanding the expected anatomical features and steps of the whole procedure should avoid potential complications. In oncological cases, adhering to oncological principles is another additional requirement in laparoscopy. Surgeons who adopt the laparoscopic approach should initially select the appropriate case and be careful in this regard. Because the surgeon's experience level is directly related to the complication rate.

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CHAPTER II

CURRENT PRACTICE FOR DIAGNOSIS AND TREATMENT FOR ACUTE UROLOGICAL CONDITIONS

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1. Flank Pain And Renal Colic

There are basically two types of pain in patients with flank pain:

a) Local pain: It is a directly related pain in the immediate vicinity of the causing organ.
b) Referred pain: Pain that is distant but associated with the causative organ. It is explained by the fact that these structures have common nerve innervation. (For example, pain in the ipsilateral testis in a ureteral stone)

There are many diseases that can cause flank pain. The main ones are abdominal aortic aneurysm, pneumonia, myocardial infarction, ovarian pathologies, acute appendicitis, testicular torsion, inflammatory bowel disease. (Crohn's, ulcerative colitis), diverticulitis, ectopic pregnancy, peptic ulcer.

If the flank pain is of kidney or ureter origin, it is called renal colic. In renal colic, the pain usually starts suddenly and at rest. Like all colic pains, it progresses in the form of crescendo and decrescendo. The patient is in constant motion in an effort to reduce pain, unlike in peritoneal irritation. Typical kidney pain is felt as a dull and constant pain. It is localized just below the costovertebral angle and the 12th rib with the sacrospinal muscle. If this pain is of ureteral origin, it usually spreads through the subcostal region towards the umbilicus and can be felt up to the testis (1) . *(Figure* 1)

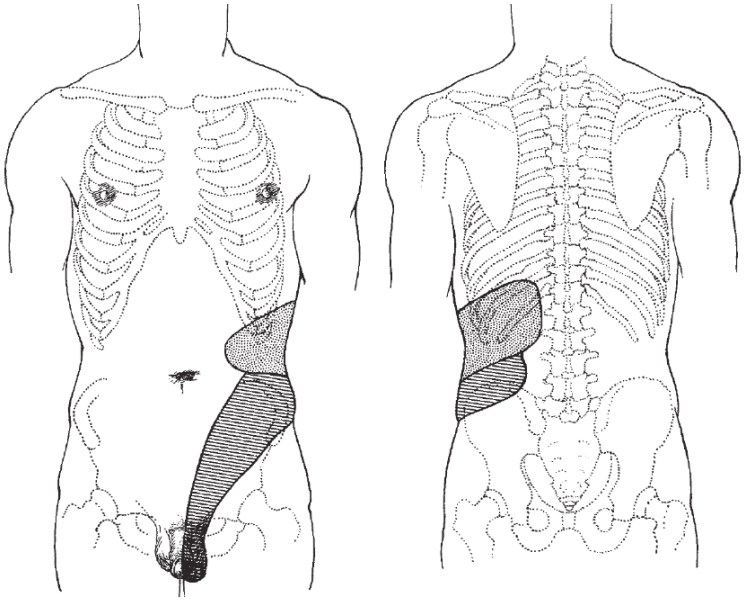


Figure 1 Pain regions in renal colic (renal pain with dots, ureteral pain with lines are schematized.)

Renal colic is seen in 50% of patients with flank pain, and is often associated with kidney or ureteral stones. Pain develops due to sudden distension of the renal capsule. Besides stones, acute pyelonephritis (sudden renal edema), renal and ureteral tumors, and acute ureteral obstruction (increased intrarenal pressure) also cause this typical pain.

Renal colic is often accompanied by nausea, vomiting, and hematuria (usually microscopic). The examinations to be performed in patients presenting with these symptoms are summarized in Table-1 ([2]) .

Table-1 Investigations for Renal Colic

Physical Exam
Ultrasound (KUB) or CT-KUB
Urine Analysis and Microscopy
UE's and FBC

In physical examination, renal palpation and costvertebral angle tenderness are important. For kidney palpation, the left hand is placed behind the patient

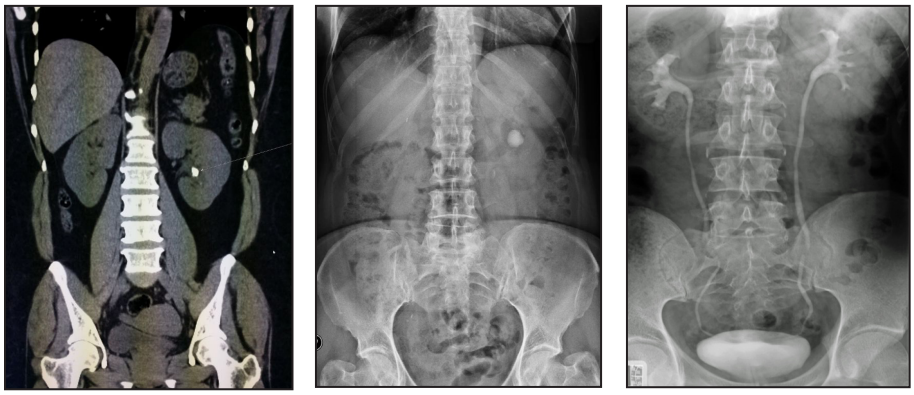
between the rib cage and the iliac crest, and the right hand is placed under the right costal margin. While the hands are pressed tightly, the patient is asked to take a deep breath. In this way, the lower pole of the kidney is tried to be felt. It is checked whether the patient has costovertebral angle tenderness by placing one hand on the palpated area and gently tapping this area with the other hand 1 .

Urinary system ultrasonography is a very fast and inexpensive imaging method with a very high sensitivity. Kidney, ureter (difficult to see the middle section), bladder and prostate can be visualized by ultrasonography. Renal colic is a condition that can be seen frequently in pregnant women. Especially after the 20th week, the growing uterus tends to put pressure on the right ureter. This condition is considered physiological. If there is 1 or 2 hydronephrosis and the patient does not have pain that affects his general condition, only follow-up is recommended. However, if the patient's pain is severe; or if grade 3 or 4 hydronephrosis develops, there is a risk of triggering preterm labor. In this case, patients must be consulted by a urologist (3).

Especially infectious pathologies can be diagnosed with complete urinalysis and microscopy. The presence of 5 or more leukocytes in each area in the urinalysis suggests urinary infection, while the presence of 5 or more erythrocytes (hematuria) suggests more stones or tumoral pathologies. In addition, some crystals that can be seen can give an idea about urinary system stone disease. The presence of leukocytosis and increase in CRP in the complete blood count should suggest infectious pathologies, the presence of anemia or polycythemia and increase in erythrocyte sedimentation should suggest tumoral pathologies.

In emergency cases, it is sufficient to perform urea and creatinine tests in venous blood to see kidney functions. In the presence of a normofunctional kidney, it is expected that kidney function tests will not be impaired, regardless of the pathology in the other kidney. If the kidney function is high in a patient presenting with unilateral colic, it is important to clarify the underlying pathologies (for example, diabetic nephropathy).

After these examinations, the underlying pathology will be clarified in most of the patients. However, if a clear diagnosis cannot be made, advanced imaging methods can be applied considering the patient's symptoms. Non-contrast CT, Direct Urinary System X-ray or Intravenous Pyelography (IVP) giving information about the anatomy of the collecting system can be used. (Respectively figures 3a,b,c)



Follow-up and Treatment

The final treatment of the pathology causing renal colic will be done by the urology specialist. However, before reaching this stage, especially the analgesic treatments of the patients are important. After distinguishing between emergent and elective pathologies, analgesic and expulsive treatment should be initiated for non-emergency conditions.

Renal colic with an indication for emergency urology consultation:

1. Pain unresponsive to narcotic analgesics
2. Pyelonephritis findings
3. Renal dysfunction
4. hematuria leading to anemia
5. Patients with solitary kidney
6. Pregnant women with 3rd or 4th degree right hydronephrosis or left hydronephrosis

Renal Colic Treatment Algorithm:

a) Non-Steroid Anti-Inflammatories

The drugs recommended in the first place in the treatment of renal colic are non-steroidal anti-inflammatories. The three drugs recommended by the EAU (European Association of Urology) guidelines are diclofenac, indomethacin or ibuprofen. Although it is stated that diclofenac may worsen renal functions; This is known to be reversible in people with normal renal function.

b) Opioids

Opioids are not recommended as first-line treatment for renal colic. Other opioids other than pethidine (meperidine), hydromorphine, pentazocine and tramadol are recommended as second-line therapy in cases where NSAIDs are insufficient.

c) Other Analgesics

Metamizole and paracetamol should be considered especially in patients with critical renal function.

d) Alpha-blockers and calcium channel blockers

It is known that alpha blockers and calcium channel blockers, which are used for expulsion of stones smaller than 10 mm in the ureter, reduce the need for analgesics by reducing ureter peristalsis, as well as contributing to the reduction of the stone.

e) Spasmolytics

Spasmolytics aim to relieve pain by preventing hyperperistalsis in the ureteral smooth muscle. Hyoscine-butylbromide can be used with ketorolac (4) .

2. Suprapubic Pain

Many reasons can cause pain in the suprapubic region. Especially patients with bladder outlet obstruction due to prostate enlargement and lower urinary tract symptoms may suddenly become unable to urinate for various reasons (acute decompensation). Causes include abstinence from sexual intercourse, infection, alcohol intake, prostate infarction, some drugs (anticholinergics, sympathomimetics). Uterine and ovarian pathologies may also cause suprapubic pain in women. In addition, it should not be forgotten that suprapubic pain may occur in the early stages of appendicitis.

Each patient should undergo a physical examination. Especially in bladder and uterine pathologies, tenderness is expected when pressing on the midline above the pubic bone. Considering that the patient may have urinary retention or a uterine mass, the examination should be performed as sensitively as possible. In acute urinary retention, there is usually 500-800 ml of urine in the bladder. On physical examination, the suprapubic globe can be felt vesically. Palpation is painful. The patient's history is expected to be inability to urinate or dripping. Bladder outlet obstruction, impaired bladder contraction (detrusor dysfunction),

interruption of sensory or motor innervation, inflammatory, infective, and neoplastic conditions of the bladder may be present.

The treatment of acute urinary retention is emergency decompression. If there is no additional pathology for this, urethral catheterization should be tried first. If successful, it is not recommended to empty very high volume urine retention quickly and at once. Sudden pressure change may cause bleeding in the superficial veins of the bladder. For this reason, it is correct to empty the bladder by giving intervals of a few minutes. If urethral catheterization is not successful, it is recommended to seek urology consultation without forcing the urethra too much. It should not be forgotten that iatrogenic urethral injuries may be caused at this stage. While suprapubic aspiration may be a temporary solution in patients who cannot be catheterized urethrically, insertion of a percutaneous cystostomy will be a medium-term solution until the final treatment. In Figures 4a and 4b these attempts are schematized (5) .

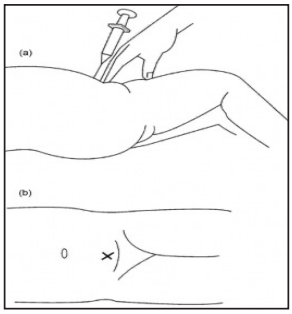


Fıgure 4a- Suprapubic Needle Insertion - The location and angle

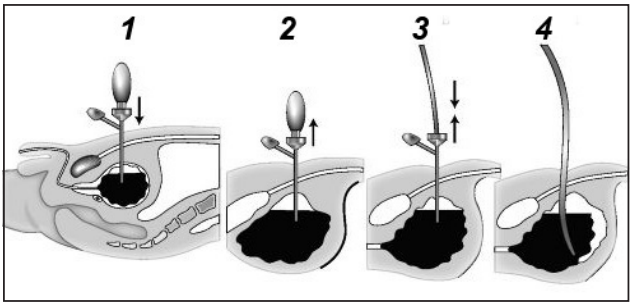


Figure 4b – Suprapubic catheter insertion method

In chronic retention, it can be seen that the bladder forms a swelling that can extend to the umbilical region, especially in weak patients. This condition is painless. Patients have overflow incontinence, and renal function impairment can be detected with bilateral hydroureteronephrosis. Urinary infection is common. Such patients, although painless, require rapid intervention in terms of renal functions. Catheterization is the first thing to be done. It is necessary to empty the bladder intermittently and slowly.

3. Scrotal Pain

The main diseases included in the differential diagnosis of scrotal pain are:

- Spermatic Cord Torsion
- Appendix Testis and Epididymal Torsion
- Epididymitis, Epididymoorchitis
- Inguinal Hernia
- Hydrocele, Epididymal Cyst
- Trauma
- Testicular Tumor
- Varicocele

In patients presenting with scrotal pain, the examinations that should be performed in the first stage after the physical examination are as follows:

- Scrotal Doppler Ultrasonography
- Complete Blood Count and CRP
- Testicular Tumor Markers

If torsion of the testis or its appendages is suspected at this point, it is necessary to take the patient to exploratory surgery without waiting for other examination results. Otherwise, a process leading to irreversible damage and even loss of testis may develop (6).

3/a. Testicular Torsion

It is a condition caused by the rotation of the spermatic cord pedicle of the testis on its axis.

and it is dangerous due to ischemia. Although it is usually seen in prepubertal and early adolescence boys, it can be encountered at any age. Symptoms of torsion syndromes are acute onset scrotal pain, swelling, nausea and vomiting.

An important finding on physical examination is the disappearance of the cremaster reflex. This reflex consists of stroking the back and inner part of the thigh. The normal response is to pull the testis up on both sides with contraction of the cremaster muscle. Other findings are erythematous and edematous scrotum, abdominal pain in the ipsilateral lower quadrant, and testicular pain that does not decrease or even increases with elevation.

When diagnosed, the testis is usually lost if not surgically corrected within 6 hours. The contralateral partner of a torsioned testis also exhibits histological

abnormalities. And in case this is a general torsion tendency, torsion prophylaxis is performed by fixing the contralateral testis in people who have undergone testicular torsion surgery (7) .

Testicular torsion is examined in two main groups. These are intravaginal and extravaginal torsions. One of the scrotal torsion syndromes is the torsion of the appendix testis, which is an embryological remnant on the testis, due to its rotation in a way that impairs blood circulation. These three torsion syndromes are schematized in Figures 5A,B,C. Extravaginal torsion is usually seen in the intrauterine or neonatal period. Intravaginal torsion is seen in advancing ages.

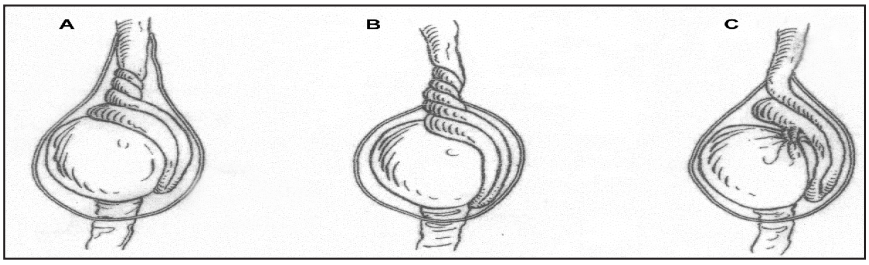


Figure 5a - intravaginal 5b-ekstravaginal 5c-appendixs testis

Diagnosis is made by physical examination. If there is doubt, it is confirmed by emergency scrotal Doppler ultrasonography. Urgent surgical exploration and necessary intervention are performed in the treatment.

3/b. Epididymo-orchitis

It should be considered primarily in patients presenting with scrotal pain, fever, increased temperature, massive swelling, and redness. Bacterial infections of testis and epididymis. Systemic infection should be evaluated in people with a pre-diagnosis of epididymo-orchitis, and predisposing factors such as urinary tract infection or sexually transmitted diseases should be investigated [2] . Epididymo-orchitis is usually caused by a bacterial infection. Infection can result from surgery, spread of infections elsewhere in the bladder or urinary tract. Sometimes, especially in young men, it can be caused by a sexually transmitted disease. Rare causes include infection by certain viruses or fungi. Sometimes no focus of infection may be found. In conditions such as high urethral pressure, urine may leak out of the urethra (this may be due to lifting something very heavy) and travel into the epididymis.

With the preservation of the cremasteric reflex from testicular torsion in the physical examination; It can be distinguished from testicular tumor by

the presence of temperature increase and tenderness. Infection is confirmed by complete blood count and CRP. Scrotal Doppler ultrasound supports the diagnosis of infection with high sensitivity. If there is an abscess focus, it should be drained first and then the treatment should be completed with antibiotic therapy. Quinolones and 3rd generation cephalosporins are preferred in the treatment of epididymo-orchitis (8) .

4. Priapism

It is an erection that lasts longer than 4 hours and is not accompanied by sexual desire. It is a pathology that requires urgent intervention. Two types are available.

- *ischemic (veno-occlusive, low flow)* (more common)
  - Hematological diseases, malignant infiltration of the corpora cavernosa or drugs.
  - Sore
- *Non-ischemic* (arterial, high flow)
  - Arteriovenous fistula due to perineal trauma
  - Painless

In patients presenting with priapism, if there is an obvious reason (eg, drug use or penile doppler ultrasound performed on the same day), the erection is terminated by medical (alpha-adrenergics) or interventional intervention (corpora-cavernosal shunt). If the cause is unknown, the tests to be performed for the differential diagnosis are as follows:

- Hemoglobin electrophoresis; sickle cell test
- Penile Doppler USG (in terms of ischemic – non-ischemic distinction)
- Blood gas from both cavernous bodies,
  - **low-flow** (dark blood; pH <7.25 (acidosis); pO2 <30mmHg (hypoxia); pCO2 >60mmHg (hypercapnia)
  - **high-current** (light red, arterial-like blood at room temperature; pH = 7.4; pO2 >90mmHg; pCO2 <40mmHg)

The earlier the priapism treatment is done, the more successful the results. However, it should not be forgotten that every patient has a risk of developing impotence (9) .

5. Phimosis

Phimosis is a condition in which the foreskin of the penis cannot be pulled back. A balloon-like swelling may appear under the foreskin while urinating. In teens and adults, it may cause pain during erection, but otherwise not painful. Affected individuals have a higher risk of inflammation of the glans, known as balanitis, than in the normal population.

In young children, it is normal not to be able to fully retract the foreskin. In more than 90% of cases, this disability will regress spontaneously by age seven and in 99% of cases by age 16. Sometimes phimosis can be caused by an underlying condition such as balanitis or scarring due to balanitis xerotica obliterans. This can typically be diagnosed by seeing the scarring of the opening of the foreskin.

Table 3 – Phimosis Grades

1: the foreskin can be fully retracted.
2: Partially visible glans, preputial adhesions usually begin in the middle part of the glans.
3: partial retraction can be done, only the external urethral meatus can be seen.
4: slight retraction, but some distance can be seen between the tip and the glans, that is, neither the meatus nor the glans can be exposed.
5: Absolutely no foreskin retraction can be done.

Low-grade phimosis (1,2) will typically resolve spontaneously by age three without treatment. A topical steroids can be used to loosen skin that is tight in palliative treatments. If this method is not effective when combined with stretching exercises, other treatments such as circumcision may be recommended. A potential complication of phimosis is paraphimosis, a condition that requires immediate attention. Permanent and definitive treatment is circumcision, especially in advanced degrees of phimosis (4,5, 10)

6. Paraphimosis

Pulling the overly narrow preputium into the glanular sulcus behind the glans penis can compress the shaft and cause oedema. Penile perfusion is impaired in the distal part of the pinched ring and this creates the risk of necrosis.

Paraphimosis is almost always caused by the parents or child pushing back the foreskin voluntarily.

Immediate intervention is required to prevent necrosis. In the treatment, the paraphimotic band, which is narrow from the dorsal region, can be temporarily incised and thus the permanent treatment can be postponed, or the patient is circumcised in the same session and permanent treatment is provided (11).

7. Hematuria:

The presence of blood in the urine microscopy (microscopic hematuria) or the patient's statement that he has seen blood in his urine (macroscopic hematuria) is a danger signal for the urinary system. Stone disease, kidney, prostate or bladder cancer, infections or nephritic syndromes are the most important diseases to be considered.

In patients presenting with hematuria, accompanying symptoms should be questioned first. Hematuria with dysuria usually suggests infectious pathologies, hematuria with renal colic suggests stone disease or polycystic kidney disease, and hematuria with painless clots suggests bladder cancer. All patients should be questioned about the drugs they use (especially anticoagulant or antiaggregant drugs) and additional diseases (for example, hemophilia, glomerulonephritis, polycystic kidney disease). The causes of hematuria are summarized in Table 4.

Table 4- Causes of Hematuria

Sore	**Painless**
• Kidney stone • Urinary tract infection • pyelonephritis • polycystic kidney disease • Renal vein thrombosis • Trauma (blunt trauma to the urinary tract or insertion of a traumatic Foley catheter)	• Kidney or bladder cancer • Exercise-induced hematuria • Glomerulonephritis • Vascular anomalies in the urinary system • Coagulation or aggregation disorders

Red urine color does not always mean the presence of hematuria. To confirm this, erythrocytes should be evaluated in urine microscopy. Some substances can mimic hematuria by either changing the color of the urine or causing a red color on the urine stick. In addition, urinalysis performed only with strips can be misleading in this regard. Causes of a false positive urine stick include hemoglobin (in the absence of red blood cells), semen, myoglobin, porphyrins, betanin (after eating beets), and medications (rifampicin, phenazopyridine, and sulfonamides). Substances that mimic hematuria by causing the color of red or

brown urine include drugs (such as sulfonamides, quinine, rifampin, phenytoin), betanin, and menstrual fluid.

After confirming the authenticity of hematuria, detailing and learning whether it is partial (initial, terminal) or total (present throughout the entire voiding) is often helpful in estimating the etiology. For example, hematuria seen at the beginning and decreasing towards the end of voiding suggests an anterior urethral lesion (eg, urethritis, stricture, meatal stenosis in young men); Terminal hematuria usually originates from the posterior urethra, bladder neck, or trigone. Common causes include posterior urethritis and bladder neck polyps or tumors. In the presence of total hematuria, it is more appropriate to expect to see a pathology at or above the bladder level (eg stone, tumor, tuberculosis, nephritis) (12) .

Hematuria is a symptom. Treatment should be directed towards the underlying pathology. However, emergency intervention should be performed in cases such as massive bleeding, clot retention, severe hypotension or profound anemia. First of all, after the vital signs and general condition of the patient are stabilized, necessary examinations should be performed.

8. Fournier's Gangrene

It is a gangranous type necrotizing fasciitis of the penis, scrotum, and perineum. Necrosis begins when an infection that starts in this region causes occlusions, especially in small vessels. With the addition of anaerobic factors, the classical Fournier table is formed. Common causative agents are Streptococci, Staphylococci, Enterococci, Corynebacteria, E.coli, Klebsiella, Proteus, Bacterioides and Clostridia type bacteria. Typically, only erythema and edema can be seen in the scrotum and perineum in the early stage. Most patients are diabetic or immunocompromised. It is not usually a very painful disease. It persists as a foul-smelling infection in the perineal region and is a disease that leads to high morbidity and mortality, with extensive necrosis if left untreated. Like all other gangrene, primary treatment is broad-spectrum antibiotics and debridement. Hyperbaric oxygen therapy has also been shown to be beneficial (13) .

9. Dysuria

Painful urination is usually associated with acute inflammation of the bladder, prostate, or urethra. There is a burning sensation in urine only during micturition. It is usually a symptom secondary to urinary tract infection. However, prostate

diseases, urinary system stone disease, interstitial cystitis, bladder and urethral cancers should be considered in the differential diagnosis.

Table 7 – Causes of Dysuria

- Chemical irritants (eg soaps, tampons, toilet paper)
- Medications (eg anticholinergics, non-steroidal pain relievers)
- Capsaicin consumption (eg Jalapeno peppers)
- Benign prostatic hyperplasia (male)
- Endometriosis (female)
- Prostate cancer (male)
- Prostatitis (male)
- Vaginitis (women)
- Cystitis
- Kidney stones
- Malignant (eg bladder cancer, prostate cancer or urethral cancer)
- Benign prostatic hyperplasia (male),
- Prostatitis (male)
- Pyelonephritis
- Sexually transmitted diseases
- Urethral stricture
- Parasites in the urinary tract (eg Schistosomiasis)
- Bacterial urinary tract infection (UTI)

Dysuria is a symptom. Treatment should be directed towards the underlying pathology. However, especially in severe manifestations of urinary infections, conditions such as sepsis, which may impair the general condition, should not be forgotten, and urgent intervention should be given to these. First of all, after the vital signs and general condition of the patient are stabilized, necessary examinations should be performed.

10. Acute Pyelonephritis

Pyelonephritis is a urinary tract infection that has reached the pyelum (pelvis) of the kidney (Nephros in Ancient Greek). It is used synonymously with the term "urosepsis" if the infection is serious. The presence of nitrites and leukocytes in the urine of patients with typical symptoms is sufficient for the diagnosis of pyelonephritis and is a guide for treatment. Definitive diagnosis is made by urine culture; A blood culture may also be required if there is doubt as to where the infection originated.

If a kidney stone is suspected (for example, due to colic pain, or if there is an excessive amount of blood in the urine), X-ray examination of the kidney, ureter, and bladder, CT may be used to detect the presence of X-ray-proof stones. In case of recurrence of ascending (ascending) urinary tract infections, the possibility of an anatomical abnormality (vesicoureteral reflux (backflow of urine from the bladder into the ureter) or polycystic kidney disease) may need to be excluded. In this case, the examinations performed are ultrasound and voiding cystourethrography.

Patients with high fever and leukocytosis should be treated by hospitalization. Intravenous antibiotics are recommended for these patients. Although quinolone group antibiotics are used most frequently, the final treatment should be determined according to the urine or blood culture results. Although the mainstay of treatment is antibiotics, the root cause of the disease must be eliminated (14) .

11. Cystitis

Bladder inflammation, especially seen in women. Typical findings are dysuria, pollakiuria, urgency (urgency in urine), hematuria (hemorrhagic cystitis) and fever. Although bacterial infections are the most common cause, these symptoms can also be seen in some non-infectious conditions such as interstitial cystitis and radiation cystitis. Differential diagnosis includes urinary tract stone disease, lower urinary tract cancers, infections of the pelvic organs (eg, pelvic inflammatory disease, prostatitis) and genital infections (eg, urethritis, vaginitis).

Since cystitis is usually a noisy clinical picture, empirical antibiotic therapy should be started without waiting for culture results. Oral treatments (for example, fosfomycin, trimethoprim + sulfamethoxazole, nitrofurantoin or quinolones) are often preferred here. Final treatment should be based on culture antibiogram test results.

The condition of having cystitis more than twice a year is called recurrent cystitis. In this case, only the treatment of the existing infection is not sufficient. Additional underlying causes should be investigated. The main reasons that may cause recurrent infections are kidney stones, urinary tract strictures, malignancies, immune system deficiencies, vesico-ureteral reflux, hygiene problems, inadequate previous treatments. An important step in the treatment of recurrent cystitis is the treatment of the sexual partner. It is known that some of the agents of cystitis are sexually transmitted. Therefore, simultaneous partner treatment should be performed in all cases of recurrent cystitis.

12. Acute Prostatitis

Acute prostatitis is a serious bacterial infection of the prostate gland. This infection is a medical emergency. It should be differentiated from other prostatitis such as chronic bacterial prostatitis and chronic pelvic pain syndrome. Although the actual incidence is unknown, it is estimated that acute bacterial prostatitis accounts for approximately 10% of all prostatitis cases . Most acute bacterial prostatitis infections are community acquired, but some occur after transurethral manipulation procedures such as urethral catheterization and cystoscopy, or after transrectal prostate biopsy. Diagnosis is based mainly on symptoms and physical examination, but can be supported by urinalysis. It is very important to do a rectal touch, especially in people with suspected prostatitis. There is usually a sign of tenderness on the prostate on the touch. Another important point here is the suspicion of prostate abscess. Abscess should be considered if there is fluctuating fever, deterioration in general condition or unresponsiveness to antibiotic therapy; In this case, rectal tapping should be avoided and the patient should be directed to the urology physician and parenteral treatment should be started.

A urine culture should be obtained in all patients suspected of having acute bacterial prostatitis to identify the responsible bacteria and the pattern of antibiotic susceptibility. If possible, this culture can be taken after the prostate examination, so that more factors in the prostate can pass into the urethra. Additional laboratory studies may be performed based on risk factors and disease severity. Radiological examinations are generally useless. However, transrectal USG can be helpful in the differential diagnosis of prostate abscess.

Initial treatment should be parenteral until fever and symptoms are controlled. An ampicillin/cephalosporin+gentamicin combination may be an option. Then, outpatient treatment continues with oral antibiotics and supportive measures. Typical antibiotic regimens include ceftriaxone and doxycycline, ciprofloxacin and piperacillin/tazobactam (15) . Infection indicators (hemogram, CRP, sedimentation, urinalysis and culture, PSA) are checked with weekly controls. In this process, alpha-blocker drugs are added to the treatment, especially in those with urination difficulties. Treatment should be done in a sufficient time so that the infection does not become chronic. This is considered as 1 month for a quinolone group antibiotic.

Another important issue regarding prostate infections is PSA elevation. PSA is a test used in routine prostate cancer screening and may be elevated in some non-cancerous conditions. Prostate infections are one of them. It is one of

the medical conditions that increase PSA the most, and this increase cannot be said to be significant in the presence of infection. After the end of the prostatitis treatment, the PSA examination should be renewed considering that the half-life is 3 days. A still high PSA after all signs of infection have resolved, raises suspicion of cancer and prostate biopsy should be taken from these patients.

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CHAPTER III

RHINOPLASTY COMPLICATIONS

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Rhinoplasty is a very popular surgery in recent years which is applied with functional and aesthetic indications(1). As with any surgery, rhinoplasty operations also have complications. In the literature, it is reported that the incidence of complications after rhinoplasty ranges from %5 to %28(2-4). However as in every operation, if patients are well evaluated and informed preoperatively, if the surgeons choose the cases according to their surgical experince and if the surgical instruments are sufficient complication rates can be reduced

1. Early Complications

1.1. Bleeding

Bleeding is one of the most common complications after rhinoplasty(5, 6). It can be seen in the first 48 hours as well as between 10-14 days after surgery(7). It is reported that the incidence of bleeding-related complications after rhinoplasty is from 0.2% to 6.7%(8-11). Preoperatively, patients should be asked about the use of anticoagulant drugs and herbal medicines. Especially recently, the use of tranexamic acid whic is an antifibrinolytic agent, has become widespread in order to reduce surgery-related bleeding. There are studies showing that preoperative administration of tranexamic acid significantly reduces intraoperative bleeding and postoperative edema and ecchymosis among patients undergoing rhinoplasty(12, 13). Furthermore, the use of desmopressin is also recommended to reduce surgical bleeding during rhinoplasty(14, 15).

Bleeding often originates from nasal mucosa and incision sites. Bleeding can be controlled with absorbable or nonabsorbable packing materails when necessary(11). Although rare, in less than 1% of patients, resistant bleeding

may originate from the sphenopalatine artery(5, 11). In this case, if bleeding is persistant, hospitalization may be required.

1.2. Hematoma

Septal hematoma, must be drained promptly. Otherwise, it can cause cartilage necrosis and saddle nose deformity(11). Nasal soft tissue hematomas may lead to excessive fibrosis and distortion of tissues. So they can cause irregularities under skin if untreated(16). It should be kept in mind that hematomas of dorsal skin may occur during plaster or cast removal and care should be taken(7).

1.3. Infection

Although it is considered an unsterile surgery, infection rates after rhinoplasty is 0% to 4% in the literature(8-10). Soft tissue cellulitis can be treated with oral antibiotics but soft tissue abscess or septal abscess must be treated with incision and drainage along with intravenous antibiotics(11).

The guideline for rhinoplasty of American Academy of Otolaryngology Head and Neck Surgery do not recommend routinely postoperative antibiotics for greater than 24 hours but there are exceptions such as revision or complicated surgery, baseline colonization of methicillin-resistant Staphylococcus aureus (MRSA), extensive cartilage grafting, immuncompromised patients, comorbid conditions requiring antibiotics and nasal packing(17).

Nasal packing used for hemostasis can rarely cause toxic shock syndrome(18-20). Nausea, fever, tachycardia and hypotension are the major symptoms of toxic shock syndrome. Systemic antibiotics and close follow up are needed for the treatment. Cavernous sinus thrombosis, meningitis and brain abscess are rare but serious complications(21).

It has been reported that the incidence of MRSA colonization in the US population is 1.5%(22). However it is considered that this rate is higher in health care workers and patients who have been recently hospitalized(23, 24). It has been shown that the risk of clinical infection is 4-fold higher in patients with MRSA colonization(25). Therefore preoperative 5-day course topical treatment of mupirocin to the nasal vestibules and perioperative antibiotic treatment is recommended for patients at high risk for MRSA colonization undergoing rhinoplasty(26).

1.4. Edema and Ecchymosis

Although degree of edema and ecchymosis varies among patients, they are expected results after rhinoplasty. Edema in the nasal tip and dorsum resolve

after 6-12 months so it is necessary to wait for this period to see the final results(7). Periorbital ecchymosis due to lateral osteotomy may persist for 2-4 weeks after rhinoplasty. The risk of ecchymosis can be reduced with the use micro-osteotomes(27).

1.5. Skin and Soft Tissue Complications

Minor skin reactions and contact dermatitis related to tape and splint can be seen after rhinoplasty. Topical cortisone is used when necessary for the treatment. So patients should be asked about any known allergies to taping materials(28).

Skin necrosis is a rare complication with the incidence of 1.7%(9). Excessive pressure from the splint, inappropriate plane of dissection, excessive thinning of the subcutaneous layer, vascular compromise of the nasal tip skin, inadequate treatment of infection and patient smoking are the possible risk factors for development of skin necrosis(7, 11).

The correct dissection plane is very important to preserve the blood supply of soft tissues. The five tissue layers over the bony and cartilaginous framework are perichondrial/periosteal layer, deep areolar layer, fibromuscular layer, superficial areolar layer and skin, respectively. Because the blood supply is in the deep fat layer, dissection should be performed below this layer to maintain the blood supply to the tissues(29, 30).

Smoking is an important factor that impair wound healing(31). It should be considered that there may be problems with wound healing in smokers and patients who have smoking history should be informed about possible complications(11).

1.6. Complications Related to Costal Cartilage

Complications related to costal cartilage may originate from the donor site or recipient site. Donor site related complication rates are reported as 3.2% whereas recipient site related complication are 11.4%. The most common complication related to donor site is scar development with the rate of 2.9%. this is followed by pleural tears with the rate of 0.6% and pneumothorax 0.1%. Pneumothorax is the most serious complication. Removal of only a central segment of costal cartilage has been recommended to avoid pneumothorax and pleural tears(32, 33).

The most common recipient site complications are warping with the rate of 5.2%, infection with the rate of 2.5%, graft resorption with the rate of 0.9%, displacement with the rate of 0.6% and graft fracture with the rate of 0.2%(34).

Warping may result in changes in nasal structure that will eventually result in the need for revision surgery. It is reported that increase in calcification of

the costal cartilage with increasing age decrease in warp rates(35). Oblique split carving technique was first described to avoid warping by Tastan et al. in 2013(36).

2. Late Complications

2.1. Scarring

Scars can be seen after rhinoplasty, especially in patients who have a predisposition to the hypertrophic scarring. The incidance rate of scarring after rhinoplasty has been reported as 0% to 7%(10, 37, 38). Intralesional steroid and 5-fluorouracil injections can be used for keloid treatment(11).

2.2. Septal Perforation

The incidence rate of septal perforation ranges from 0% to 2.6% in the literature(39-41). Septal perforation can occur due to improperly drained septal hematomas, infections, necrosis or symmetrical mucosal tears(7). Small perforations can be closed by wound contraction but larger perforations are difficult to close. Septal perforation causes complaint such as crusting, whistling, bleeding and nasal airway obstruction. Airway obstruction occurs because laminar airflow is distrupted. Perforations located in the anterior septum and larger in size cause more frequent sypmtoms. Nasal saline irrigations and septal button are conservative treatment options(5).

2.3. Need for Revision Surgery

Revision rates after rhinoplasty ranges from 0% to 10% in the literature(10, 42). Most revisions are requested for cosmetic reasons rather than functional ones. Independent risk factors for revision surgery are female sex, younger age, autoimmune diseases, anxiety and surgery for cosmetic or congenital deformities(10).

3. Technical Complications

3.1. Saddle Nose Deformity

Saddle nose deformity occurs as a result of creating a low dorsum in the upper 2/3 of the nose. Projection and rotation of the tip are two important criteria in determining the height of nasal dorsum. Except for excessive resection of middle nasal roof, trying to correct the faltness of nasal root with dorsal resection instead of augmentation, due to deterioration of the key area, may cause this

deformity. Iatrogenic, neoplastic, infectious or granulomatous etiologies causing septal perforation may result in saddle nose deformity(43, 44). Middle vault support is very important when performing septoplasty in order to avoid iatrogenic collapse. Grafting materials commonly costal cartilage are used to provide dorsal caudal support and correct this deformity(45).

3.2. Pollybeak Deformity

Pollybeak deformity is seen 40% to 60% in revision rhinoplasties(46-48). Pollybeak deformity can occur for various reasons. Morever, pollybeak deformity has been reported as the most frequent cause of revision rhinoplasty by Kamer et al.(48). Insufficient lowering of the cartilaginous dorsum is the most common cause of pollybeak deformity(11). After rhinoplasty if the tip ptosis develops over time, pollybeak deformity may occur. In order to prevent tip ptosis sufficient tip support restoration is very crucial. This deformity may also develop because of overresection of the bony dorsum. In addition to these unfrequently, excessive scar formation after traumatic dissection of the soft tissue may result in pollybeak deformity. Also improper splinting can cause excessive fibrosis and scar tissue in the dead space of the supratip area. In this case triamcinolone injection can be effective(7, 47, 49). Injections can be continued every 2 months until the scar tissue is reduced. Possible side effects of steroid injections are dermal atrophy, telengiectasias, depressions, color changes and contour irregularity(5).

3.3. Rocker Deformity

Rocker deformity is a complication of lateral osteotomy. This deformity may occur when the lateral osteotomy lines are too high in the medial canthus area extending in thicker frontal bone. In case of rocker deformity is seen, crushed cartilages and fascia can be utilized in order to camouflage bony steps(50).

3.4. Open Roof Deformity

Following dorsal hump reduction, if there is a gap between septum and nasal bones nasal bridge appears flat and trapezoid in shape. This deformity is called open roof deformity(7, 11). In order to correct this deformity, the space between septum and nasal bones should be closed by lateral osteotomies. Furthermore, incomplete osteotomies resulting in lack of full mobilization of bones should be avoided to prevent open roof deformity(47).

3.5. Inverted V Deformity

Separation of upper lateral cartilages from the nasal bones causes shadow effect on the dorsum and this deformity is called inverted V deformity(11). After aggressive dorsal hump resection, if the support of the upper lateral cartilages is reduced and insufficient upper lateral cartilages may collapse causing inverted V deformity(51). Patients with short nasal bones and longer upper lateral cartilages predispose to this deformity so in such a situation, dislocating of upper lateral cartilages from the undersurface of nasal bones should be avoided. In order to prevent development of inverted V deformity, upper lateral cartilages can be fixed to the septum with suspension sutures and spreader grafts can be used(7, 11).

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CHAPTER IV

MANAGEMENT OF POSTOPERATIVE RESPIRATORY COMPLICATIONS IN THE INTENSIVE CARE UNIT

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1. Introduction

Among the reasons for hospitalization to the intensive care unit in the postoperative period in surgical cases, respiratory problems are in the first place decisively. The type of surgery and the comorbidities exist in patient play an important role in the emergence of these problems. Postoperative respiratory failure is one of the important reasons for the increase in length of hospital stay, costs, morbidity and mortality.(1-5) The incidence of postoperative pulmonary complications (PPCs) has been reported to vary between 2-40% in the literature.(6-8) However, the occurrence of these complications can be reduced by measures to be taken both preoperatively and peroperatively. First of all, the evaluation of risky patient groups preoperatively and the identification of patients with a high risk of postoperative complications should be the first step to reduce these complications. Although different scoring systems(3,9-11) are used for this purpose, the use of the ARISCAT score, which provides a simple and rapid evaluation of seven parameters, can help identify risky cases and can be easily used during preoperative evaluation.(3) The ARISCAT score uses simple variables taken from the patient's history and physical examination that can predict the risk of pulmonary complications and does not include any intervention that will increase the health cost for the patient.(12)

Table 1: Ariscat Score (12)

Predictor	Risk score
Age: ≤5**0** years old	0
Age: 51-8**0** years old	3
Age: > 8**0 y**ears old	16
Preoperative **S**pO2: ≥96%	0
Preoperative S**pO2: 91-95%**	**8**
Preoperative SpO2: ≤ 90%	**24**
Respiratory tract infection in the last 1 month: No	**0**
Respiratory tract infection in the last 1 month: Yes	**17**
Preoperative Hemoglobin<10 gm/dl: No	**0**
Preoperative Hemoglobin<10 gm/dl: Yes	**11**
Peripheral incision	**0**
Upper abdominal incision	**15**
Intrathoracic incision	**24**
Surgery time <2 hours	**0**
Surgery time = 2-3 hours	**16**
Surgery >3 hours	**23**
Emergency procedure: No	**0**
Emergency procedure: Yes	**8**
Low risk: <26, Medium risk: 26-44, High risk: ≥45	

Recognition of modifiable and non-modifiable anesthesia and surgical factors peroperatively, especially in patients with high ARISCAT scores, may help identify patients at risk of developing PPCs. Appropriate perioperative strategies can then be applied to minimize the occurrence of pulmonary complications. Studies have shown that surgical and anesthetic risk factors such as general anesthesia, emergency surgery, upper abdominal incision, operation period more than 3 hours, having postoperative NG tube, intraoperative blood transfusion, intraoperative pulmonary complications and high ARISCAT score are significantly associated with PPC formation.(12)

Studies have shown that high age is an independent risk factor for PPCs. (4,13-15) Depending on age; The emergence of ventilation perfusion mismatch due to decreased lung elasticity, decreased alveolar surface area and increased dead distance may predispose to increased work of breathing and PPC development.(8)

Anemia and low oxygenation of arterial blood can cause tissue hypoxia and increase the risk of infection by affecting the immune system. (16,17) The fact that patients encounter respiratory problems intraoperatively may pose a risk for these patients after surgery. Studies have shown that the incidence of postoperative complications is higher in patients with intraoperative respiratory complications.(18,19)) The duration and type of the surgery can be effective in the emergence of postoperative respiratory complications. For this reason, it is important to discuss the surgical and anesthesia application methods in risky patient groups by the surgeon and anesthetist in order to prevent complications. In high-risk patients, alternative anesthetics (regional or neuraxial anesthesia instead of general anesthesia) and modified surgical techniques (minimally invasive surgical procedures instead of open surgery and limitation of operation duration) should be considered.(20-22)The entity of a nasogastric (NG) tube has been reported as a risk factor for PPCs. (23) NG tube may cause ineffective coughing, retention of secretions, and atelectasis. (24) It may cause micro-aspirations and pneumonia by causing leakage in the lower esophageal sphincter. Identification of risk factors is helpful in predicting postoperative outcomes of patients. The patient and surgeon should be informed about the increased risk of PPC associated with the presence of non-modifiable risk factors such as high age. Modifiable factors such as respiratory tract infection should be treated preoperatively and routine NG use should not be recommended. (12)

Although operations are performed considering all the factors mentioned in detail above, sometimes respiratory complications may be encountered and patients may need to be followed up in the intensive care unit postoperatively.

As a postoperative pulmonary complication; respiratory tract infection, respiratory failure, bronchospasm, atelectasis, pleural effusion, pneumothorax or aspiration pneumonia are the most common clinical manifestations.

Table 2: Description of postoperative pulmonary complications(12)

Complication	Description
Respiratory Failure	PaO2 <60 mmHg, PaO2/FIO2 <300 or
	SPO2 <90% by pulse oximetry in postoperative room air
Respiratory infection / Pneumonia	Treatment with antibiotics for respiratory tract infection, and at least one of the following criteria: New or altered sputum, New or changed lung opacities on clinically indicated chest radiograph, Fever>38.3 °C, Leukocyte count <4,000, >12,000/mm3
Pleural effusion	Chest radiograph showing atrophy of the costophrenic angle, loss of sharp silhouette of the ipsilateral hemidiaphragm (in the upright position), evidence of displacement of adjacent anatomical structures, or a hazy opacity in a hemithorax with preserved vascular shadows (in the supine position)
Atelectasis	Lung opacification with displacement of the mediastinum, hilum, or hemidiaphragm toward the affected area and increased aeration in the adjacent non-atelectatic lung
Pneumothorax	Air in the pleural space without the vascular bed-parenchyma surrounding the visceral pleura on chest radiograph
Bronchospasm/ Exacerbation of Obstructive Pulmonary Diseases (Asthma, COPD…)	New-onset expiratory wheezing treated with bronchodilators/Occurring expiratory wheezing based on preexisting disease
Aspiration pneumonia	Respiratory failure after aspiration of gastric contents

2. Postoperative Pulmonary Complications

2.1 Respiratory Failure

Postoperative respiratory failure is among the most common postoperative complications, and its incidence is estimated to vary between 2% and 5.6%. It has been shown that post-extubation respiratory failure is one of the most important factors associated with poor patient outcomes, leading to longer hospital stays, higher financial costs (25), and an increase in 30-day mortality up to 18 times (26,27). Predicting which patients are at high risk for postextubation respiratory

failure is clinically important as it contributes to triage for the follow-up of these patients in the postoperative period, and will also provide more intensive and careful follow-up of these patients.(26)

Respiratory failure can be examined in 4 groups as Type 1 (Hypoxemic), Type 2 (Hypercapnic), Type 3 (Perioperative) and Type 4 (Shock). Respiratory failure developed in the postoperative period may present with findings such as dyspnea, tachypnea (respiratory rate >20/min), bradypnea (respiratory rate <8/min), cyanosis, altered consciousness, tachycardia, use of auxiliary respiratory muscles, and paradoxical breathing pattern.(28) In addition to clinical findings, the presence of PaO2<60 mmHg, PaCO2>45 mmHg, PaO2/FiO2<300 and PH<7.35 should be considered as diagnostic criteria for respiratory failure. There are many reasons for the emergence of these clinical pictures postoperatively.

2.2 Atelectasis

One of the most common causes of postoperative respiratory failure is atelectasis. Depending on the applied surgical areas, atelectasis may develop in the lungs at certain rates. While this rate is highest in thorax surgery, it is respectively followed by upper and lower abdominal surgery. Developing atelectasis causes volume loss and may last for several days.(29) Although the etiology of atelectasis in the perioperative setting has not been fully explained, three important 'physiological' mechanisms have been found to cause or contribute to its development. These include compression, alveolar gas resorption, and surfactant disturbance.(30) Atelectasis can develop within minutes after the induction of general anesthesia and can be seen in 85-90% of healthy adults, it can affect up to 15% of the whole lungs and can develop more especially in the basal regions.(31) Developing atelectasis causes a true pulmonary shunt in approximately 5-10% of cardiac output.(32) Anesthesia given under high oxygen concentrations, especially without the addition of positive end-expiratory pressure (PEEP), muscle relaxants and mechanical ventilation cause permanent atelectasis and postoperative pulmonary complications.(33-35) PEEP increases expiratory lung volume and prevents airway closure by having a predominant effect on dependent lung regions.(36) Intraoperative administration of individualized optimal PEEP not only reduces driving pressure, but also improves respiratory compliance and oxygenation, and reduces the incidence and severity of postoperative atelectasis. (37) Therefore, the application of lung protective ventilation (38,39), which has been shown to improve outcomes in patients undergoing general anesthesia, may prevent the occurrence of these

complications. Despite taking the necessary precautions, sometimes atelectasis developing postoperatively can cause respiratory failure. Atelectasis should be one of the first diagnoses that should come to mind in patients admitted to the intensive care unit with respiratory failure. In order to confirm this preliminary diagnosis, first of all, physical examination findings (decreased breath sounds on auscultation) and imaging to confirm the diagnosis radiologically would be appropriate. Knowing those risk factors; type and duration of general anesthesia, type and duration of surgery, underlying lung disease, neuromuscular disease, chronic systemic disease, weakness, obesity, age, airway obstruction, pleural effusion, prolonged bed rest (especially with limited position changes), and Knowing that shallow breathing (pain and splint result) as causes of atelectasis will help in planning the treatment. (40)

Postoperative pain control is critical in the prevention of PPCs. Pain can cause lung volumes to decrease and restrict lung expansion, impairing the ability to breathe deeply and cough effectively. Painful patients have difficulty cooperating with mobilization and lung expansion maneuvers. (42) Prophylactic respiratory care should be provided routinely after major surgery to minimize the adverse effects of surgical trauma and anesthesia on the pulmonary system. A group of therapeutic modalities known as airway clearance therapy (ACTs) have been developed to promote lung expansion and secretion mobilization. They are applied to improve lung volumes, strengthen respiratory muscles, activate retained pulmonary secretions and resolve atelectasis. ACT techniques/modalities include exercise spirometry (IS), deep breathing exercises, chest physiotherapy techniques, airway oscillation therapies including continuous positive pressure breathing, positive expiratory pressure, and nasal high frequency oscillation techniques. (40.43.44) Oxygen can be given by nasal or mask according to the SPO2 of the monitored patient or the PaO2 and PaCO2 levels of the taken blood gas.mRespiratory physiotherapy should be done together with oxygen therapy. If there is no response to this treatment, high-flow nasal oxygen therapy can be administered to patients with higher oxygen requirements. In patients who do not respond to oxygen therapy and respiratory physiotherapy, respiratory support can be provided with positive pressure ventilation (appropriate PEEP and tidal volume).

2.3 Pneumothorax

One of the important causes of acute respiratory failure developing in the early postoperative period is due to the development of pneumothorax, and this clinical

picture can sometimes reach life-threatening levels. Pneumothorax is defined as the presence of air in the pleural space. Air in the pleural space occurs when one of three events occurs: (1) communication between the alveolar spaces and the pleura, (2) direct or indirect communication between the atmosphere and the pleural space, or (3) the presence of gas-producing organisms in the pleural space.(45,46) Pneumothorax is divided into 3 groups as spontaneous (primary and secondary), catamenial and traumatic (iatrogenic and noniatrogenic).(47) Here, we will focus mainly on iatrogenic pneumothorax due to the content of this chapter**.**

Iatrogenic pneumothorax most commonly occurs after transthoracic needle biopsy (24%), subclavian vein catheterization (22%), thoracentesis (20%), transbronchial lung biopsy (10%), pleural biopsy (8%), and positive pressure ventilation (7%). comes out.(46) It is necessary to be more careful in terms of iatrogenic pneumothorax in patients undergoing such invasive procedures. In addition, paying attention to mechanical ventilation (MV) parameters and applying protective lung ventilation will protect the patient from the risk of pneumothorax due to MV. Pneumothorax presents with acute and sudden chest pain along with shortness of breath in 95% of patients. This pain may be more severe on inhalation and may be localized to the area where the pneumothorax developed. The severity of symptoms such as dyspnea is proportional to the size of the pneumothorax, although 5% of patients may be asymptomatic.(48)

In patients who develop respiratory failure in the postoperative period, if the respiratory sounds cannot be obtained bilaterally in auscultation, it should be immediately considered and checked by direct chest X-ray. Small and asymptomatic iatrogenic pneumothorax often do not require any treatment and resolve spontaneously. For larger or symptomatic pneumothorax, simple manual aspiration or chest tube insertion is usually effective. (49)

2.3.1 Tension Pneumothorax

In tension pneumothorax, air flows into the pleural space during inhalation, but is trapped in the pleural space during exhalation and therefore cannot exit, resulting in a gradual increase in intrapleural pressure. Tension pneumothorax can develop from spontaneous pneumothorax or traumatic pneumothorax.

During a tension pneumothorax, the affected lung collapses completely ipsilateral to the pneumothorax and the contralateral lung and heart are pressurized. The result is severe shortness of breath leading to death, cyanosis, and hypotension. Therefore, tension pneumothorax should be treated promptly with needle decompression.(48)

2.3.2 Pneumothorax Treatment

Oxygen therapy:

Gas in the pleural space is absorbed by diffusion and can be facilitated by changing the composition of the gas within the pleural space. Oxygen is absorbed 62 times faster than nitrogen, and carbon dioxide (CO2) is absorbed 23 times faster than oxygen. When the patient breathes 100% oxygen, nitrogen is lost from the pleural space, leaving only oxygen that is more rapidly absorbed from the pleural space into the veins. It is recommended to use high flow oxygen (10 L/min) in symptomatic patients.(48)

Observation:

It is recommended that only clinically stable patients diagnosed with small volume pneumothorax be observed. According to the American College of Chest Physicians (ACCP) guidelines, clinically stable patients should be observed for 3-6 hours and if a repeated chest radiograph excludes progression of the pneumothorax, continue monitoring and treatment.(48)

Simple aspiration:

Aspiration of a pneumothorax is performed using a small catheter. The catheter is inserted into the pleural space and removed immediately after draining the air from the pleural space or if it is observed, it may remain inserted in the patient. (50)

Chest tube placement:

A chest tube should be placed in patients with a large pneumothorax or clinically unstable patients. The ACCP recommends placing a thick chest tube (24 to 28F) in a patient who is experiencing a large-scale air leak, such as a bronchopleural fistula, or is being mechanically ventilated. In the stable patient, the ACCP recommends chest tube thickness of 14 to 22F or less. If the air leak persists for 2-14 days, surgical intervention is usually recommended to eliminate the leak. (51,52)

2.4 Postoperative Pneumonia

Postoperative pneumonia can be defined as hospital-acquired pneumonia (pneumonia that develops 48-72 hours after hospitalization) or ventilator-

associated pneumonia (VAP, pneumonia that develops 48-72 hours after endotracheal intubation) after surgery. Today, postoperative pneumonia is the third most common complication in all surgical procedures and is associated with increased patient morbidity and mortality.(53)The pathogenesis of postoperative pneumonia is multifactorial and typically begins with colonization of the aero-digestive tract, aspiration of contaminated tract secretions, and decreased host defense (critical illness, comorbidities, or drugs).(54) Most cases of postoperative pneumonia are caused by gram-negative, aerobic bacteria, including Pseudomonas, Klebsiella and Enterobacter species, among others. Regarding Gram-positive bacteria, methicillin-resistant Staphyloccocus aureus is the most common cause. Even more troubling is the increasing resistance to antimicrobial drugs, making treatment increasingly difficult.(55) The incidence of postoperative pneumonia (POP) is estimated to be between 1.3% and 18.6%. (56) The overall mortality rate of POP has been reported to range from 9% to 46%, depending on the population studied.(10,57,58) Studies have shown that the risk of postoperative lung infection may be higher in patients with a high ASA score, COPD, functional status, 70 years of age ≥ diabetes mellitus, anemia, hypoalbuminemia, general anesthesia, and operative period ≥120 minutes.(59,60) It should be considered that the incidence of postoperative pneumonia may be high in the patient group with these factors before and after the operation, and necessary precautions should be taken.Taking preventive measures for patients at high risk of postoperative pneumonia can improve the prognosis and reduce hospitalization costs.Measures to be taken include maintaining an upright posture while eating, encouraging coughing and deep breathing exercises, raising the head of the bed to at least 30 degrees, good pain control, and oral hygiene with chlorhexidine twice a day.(53)In addition to these measures, appropriate antibiotic therapy should be started according to the culture results of the patients.

2.5 Aspiration Pneumonia

The infectious pulmonary process that occurs after the abnormal entry of fluids into the lower airways is called aspiration pneumonia. Aspirated fluid may also be oropharyngeal secretions, particulate matter, or gastric contents.(61)

Aspiration is one of the clinical situations encountered during intubation of patients, especially in cases taken under emergency conditions. In addition, predictive clinical factors for the development of aspiration pneumonia in older adults receiving nursing care were determined to be ASA classification ≥ 3,

male gender, inability to sputum, impaired swallowing function in the last 3 months, dehydration, and dementia. (62.63) It should be known that patients with these clinical features are at risk for aspiration in the postoperative period and necessary precautions should be taken. Studies have shown that aspiration pneumonia is seen around 1% in patients undergoing surgery and has a high mortality rate. (63.64) Mortality from aspiration pneumonia is highly dependent on the volume and content of the aspirate and can reach 70%.(65,66) Aspiration of large volumes of gastric contents may lead to the development of chemical pneumonia and then severe respiratory failure with the development of acute respiratory distress syndrome (ARDS). The etiology of aspiration pneumonia depends on the content of the aspirate. Aspiration of the normal oropharyngeal flora may initiate the infectious process and cause aspiration pneumonia. If the bacterial load of the aspirate is low, normal host defense will clear secretions and prevent infection.(61) A prospective study of 95 patients showed that gram-negative bacilli contributed 49%, followed by anaerobes (16%). The main anaerobes isolated were Fusobacterium, Bacteroides and Peptostreptococcus. Gram-negative organisms, particularly Pseudomonas aeruginosa, should be considered in hospital-acquired aspiration pneumonia.(67,68)

Antibiotic treatment should be started immediately in patients with suspected aspiration pneumonia, and imaging studies should not delay treatment. Commonly used imaging studies are chest X-ray and computed tomography of the chest to assist in localization of the aspiration site. The right lower lobe is most commonly involved in chest X-ray. Bilateral lower lobe involvement may exist in patients aspirate vertically. Patients lying in the left lateral decubitus position tend to have left-sided infiltrates. Arterial blood gas can be used to assess pH status and oxygenation. Bronchoscopy is indicated when food particles are aspirated. The technique also allows it to be taken for culture.

In the treatment, the position of the patient should be adjusted first, followed by aspiration of the oropharyngeal contents by placing the nasogastric tube. In non-intubated patients, humidified oxygen is administered and the head of the bed should be elevated 45 degrees. Close monitoring of the patient's oxygen saturation is important and if hypoxia gets worse, intubation should be considered and mechanical ventilation should be started. In hospital-acquired aspiration pneumonia, antibiotics are needed, including resistant gram-negative bacteria and S. aureus. Therefore, the use of vancomycin and the piperacillin-tazobactam combination is most common. After culture results are obtained, the antibiotic regimen should be narrowed to organism specific.(69-71)

2.6 Postoperative Pulmonary Edema

Postoperative pulmonary edema is a well-known postoperative complication that occurs as a result of many etiological factors that can be easily detected with careful follow-up in the postoperative period. Postoperative pulmonary edema is most commonly seen as cardiogenic pulmonary edema in patients with severe cardiovascular disease, followed by non-cardiogenic pulmonary edema or negative pressure pulmonary edema (NPPE), which may be due to fluid overload in the postoperative period.(72) Various etiological factors have been identified that may lead to postoperative pulmonary edema, and there is ample evidence to suggest that many of these patients have pre-existing heart disease. Excessive administration of intravenous fluids in the perioperative period may precipitate cardiac dysfunction, which can be fatal.(73)

Etiology:

Cardiogenic pulmonary edema; It is the most important and most common cause of postoperative pulmonary edema in patients with pre-existing heart disease. Myocardial infarction causes fluid leakage into the interstitium as a result of left ventricular dysfunction and increased hydrostatic pressure in the pulmonary circulation in these patients. Any excessive stress during the surgical and anesthetic procedure, particularly intubation-related stress and extubation response, can be considered as potential causes.(74)

Non-cardiogenic pulmonary edema may develop in patients without any heart disease and with any underlying pathology.

Neurogenic pulmonary edema; Clinical manifestations of pulmonary edema can be attributed to disruption of the autonomic nervous system, resulting in an exaggerated sympathetic response. Thus, it increases the pressure in the pulmonary capillaries and causes the extravasation of fluid to the pulmonary tissues.(75)

Fluid overload; Pulmonary edema can be extremely fatal if not diagnosed early and treated appropriately. Often, it may be the first clinical manifestation of excessive fluid administration in some cases such as sepsis, excessive intraoperative bleeding, resuscitation during trauma, and so on.(76,77)

Negative pressure pulmonary edema; Negative pressure pulmonary edema (NPPE) is a pathophysiological syndrome that occurs with the acute development of negative intrathoracic pressure during spontaneous respiratory efforts against an obstructed upper airway. The most common cause of NPPE is laryngospasm that develops after extubation.(72,78)

Treatment; Management of postoperative pulmonary edema is usually directed towards the treatment of the underlying cause. While the majority of patients show good results with conservative and symptomatic treatment,few require intubation and initiation of mechanical ventilation with application of positive end-expiratory pressure. While cardiogenic pulmonary edema responds to treatment for the cardiac event causing pulmonary edema, it usually responds to fluid restriction and diuretic therapy due to fluid overload. (72) Noninvasive ventilation support has replaced invasive intubation and ventilation strategies in the treatment of respiratory failure. The role of noninvasive ventilation in the treatment of pulmonary edema may be important as it greatly reduces the increased work of breathing, thereby preventing muscle fatigue. Besides this benefit, noninvasive ventilation can reduce ventricular afterload and cause minimal disturbances of hemodynamic parameters. (72,79,80) Resolution of pulmonary edema usually occurs within 3-12 hours after the initiation of appropriate therapy with complete resolution of chest radiograph findings. However, in a few cases total resolution may take up to 12-48 hours. Postoperative pulmonary edema has a good prognosis if patients are diagnosed early and appropriate treatment is started. However, delay in diagnosis may cause significant complications. (81,82)

3. Conclusion

Postoperative pulmonary complications are closely associated with morbidity, mortality, long hospital stays and high costs. Knowing the factors that are effective in the emergence of these preoperative complications and taking the necessary precautions can prevent the occurrence of these complications. Despite the precautions taken, sometimes these complications can be encountered. One of the important points is to recognize the complications early and to make the necessary interventions in time. It should not be forgotten that when time is lost, more severe clinical consequences will be encountered.

Keep in mivd

- Postoperative pulmonary complications (PPCs) are directly associated with increased morbidity, mortality and health costs. The incidence of PPCs increases severely in proportion to aginγ.
- PPCs is a multifactorial syndrome in which atelectasis plays an important role. Evidence suggests that the development of atelectasis is an almost universal precursor and accompanying feature of PPCs.(40)

- Prevention or aggressive early management of atelectasis is vital to minimize progression to serious/life-threatening PPCs and unsustainable economic expenditure.(40)
- Expanding awareness and understanding of the role of atelectasis as a cause of PPCs is a key step towards minimizing prevalence, improving clinical outcomes and reducing the financial burden of this potentially preventable complication.(40)
- Postoperative respiratory failure is associated with high morbidity and mortaliτy
- Among the independent predictors for postoperative reintubation; ASA score of 3 or more, emergency surgery, high-risk surgical service, history of congestive heart failure, and chronic lung disease.(26)
- Although we have some criteria, it is difficult to predict which patients will need postoperative reintubatiον.
- Postoperative pulmonary edema has a good prognosis if patients are diagnosed early and appropriate treatment is started. However, delay in diagnosis can cause significant complicationσ.
- It is the most important and most common cause of postoperative pulmonary edema in patients with pre-existing heart disease. Excessive administration of intravenous fluids in the perioperative period may precipitate cardiac dysfunction, which can be fataλ.
- Postoperative pneumonia is among the most common complications in all surgical procedures and is associated with increased patient morbidity and mortality.

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CHAPTER V

MEDIASTINITIS AFTER OPEN HEART SURGERY

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1. Introduction

Postoperative mediastinitis is a rare, but dreaded complication of open heart surgery. It is associated with high rates of morbidity, mortality, prolonged hospitalization and health care costs. The incidence of postoperative mediastinitis ranges between 1-5%. According to the Centre for Disease Control and Prevention (CDC), diagnosis of postoperative mediastinitis requires an organism isolated culture of mediastinal fluid or tissue and fever (>38^{o}C), chest pain or sternal instability in combination with purulent discharge from the mediastinum or an organism isolated from the blood or mediastinal drainage (1). The diagnosis of postoperative mediastinitis is relatively easier in patients who developed bacteremia accompanied with sternal wound drainage and/or infection within 14 days following open heart surgery. Management of postoperative mediastinitis consists of antimicrobial therapy and surgical treatment. This chapter includes pathogenesis, incidence, risk factors, clinical features, diagnosis, management and prevention of postoperative mediastinitis occurring following open heart surgery.

2. Pathogenesis

Patients undergoing open heart surgery are at high risk of mediastinitis due to impaired immune responses and a number of potential ports of entry for bacterial pathogens. Pathogenesis of postoperative mediastinitis is complex and multifactorial. The most common factor is intraoperative contamination of the wound. Such contamination is likely to occur in all patients due to large sternotomy wounds that are open for a long time during cardiac surgery. The type and degree of contamination are influenced by patient factors such as

sufficiency of blood supply, immunologic status, and nutritional status. Some other pre- and intraoperative factors may play a role in the development of postoperative mediastinitis. These factors include impairment of blood supply to the sternum during operation, tissue trauma from electrocautery and early postoperative wound disruption.

The mediastinum is covered by a thin layer of fibrin and the mediastinal structures are relatively soft and mobile in the early stages of wound infection. Chronic mediastinitis develops within a few weeks and is characterized by the formation of sinus tracts that extend to the posterior and middle mediastinum. The mediastinal structures are then covered by a thick fibrous cortex, which prevents them from moving into the anterior mediastinum.

2.1. Microbiological Etiology

Monomicrobial infection is more common in patients with postoperative mediastinitis. Two reviews including 350 patients with postoperative möediastinitis have reported that a single pathogen was isolated from the blood or mediastinum in the majority of the patients (83%) (2, 3).

In fact, any organism can cause mediastinitis. In a prospective study by Trouillet et al. including 316 patients with mediastinitis, which had occurred less than 30 days following heart surgery, the most commonly isolated pathogens included methicillin-susceptib*le Staphylococcus aur*eus (MSSA) by 45%, gram-negative bacilli by 17%, methicillin-resista*nt Staphylococcus aur*eus (MRSA) by 16%, coagulase-negative staphylococci (CONS) by 13% and streptococci by 5% (3). On the other hand, CONS have been reported to be common pathogens isolated in postoperative mediastinitis following cardiac operations (4). CONS are the most commonly isolated when stainless steel wires are used to close the sternum (5). Furthermore, CONS are common in the skin flora, facilitating infection of the surgical wound (6). In postoperative mediastinitis, MSSA is more commonly isolated in the case of preoperative nasal MSSA colonization, while MRSA is more commonly found through nosocomial transmission between patients (7). Although rare, fungi have also been reported as causative agents in a small portion of patients with postoperative mediastinitis (8).

3. Incidence

The incidence of postoperative mediastinitis has been reported between 1-5% in various studies (9), while rates >2% usually indicate poor quality of care

in cardiac surgeries (10). However, the incidence may be higher in certain subsets of patients undergoing cardiac surgery. Higher incidence rates have been reported in patients undergoing cardiac transplant surgery, CABG with thoracic aortic surgery and CABG with valvular surgery. Lower incidence rates have been associated with isolated CABG, valvular repairs and thoracic repairs (11).

Oliveira et al. reported the incidence of mediastinitis as 4.2% in 1322 patients who underwent coronary artery bypass graft (CABG) operation (12). Diez et al. reported the incidence as 2.65% in 1700 patients after cardiac surgery (10). Although the incidence of postoperative mediastinitis is relatively low, mortality associated with postoperative mediastinitis is extremely high as reported between 14% and 47% (13***).***

3.1. Risk Factors

Common risk factors of developing postoperative mediastinitis are shown in Figure 1.

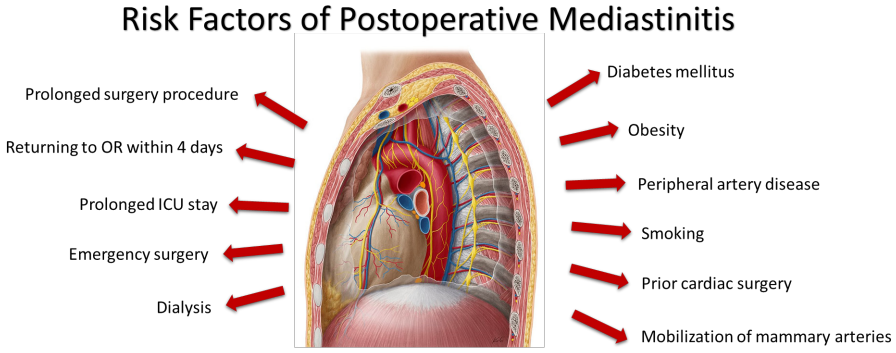

Figure 1. Risk factors of developing postoperative mediastinitis after cardiac surgery

There is no consensus on which risk factors are more crucial and whether the risk factors in Figure 1. are independent predictors of an increased risk of mediastinitis following cardiac surgery. However, it is obvious that all or many of the risk factors above are especially important in selected patients, especially if two or more factors are present (14).

The possible mechanism through which obesity increases the risk of developing mediastinitis has not been fully understood, but it is thought that

poor distribution of perioperative antibiotics in adipose tissue and inadequate skin preparation due to deep skin folds may facilitate contamination (4). In diabetes mellitus, raised blood glucose levels disrupt wound healing. Continuous use of intravenous insulin has been shown to decrease the incidence of deep sternal wound infection in diabetic patients (15). Prolonged procedure duration increases the risk of intraoperative contamination, and thus the risk of developing mediastinitis (16).

4. Clinical Features

Mediastinitis manifestations can vary from subacute to critical. Postoperative mediastinitis is typically accompanied by tachycardia, fever, and local signs of wound infection in the surgical site. These symptoms occur within the first 30 days after the procedure in two thirds of patients. The most common presentation is an operative wound dehiscence with local secretions and pain, inflammation, purulent distal from the mediastinal area and sternal instability (12). Other local findings may include edema and crepitus of the chest wall, and Hamman's sign, which is a crunching sound auscultated over the precordium during systole. Although signs of sternal wound infection can appear before or after the recognition of postoperative mediastinitis, systemic symptoms and fever are evident first in most patients.

Bacteremia is a common manifestation in postoperative mediastinitis. It may precede or follow the recognition of mediastinitis. Since bacteremia can be the first sign of postoperative mediastinitis, the possibility of mediastinitis should be considered in all bacteremic patients who had undergone cardiac surgery (9).

4.1. History and Physical Examination

Medical history including important risk factors such as smoking, obesity, diabetes mellitus, renal failure and immunocompromised state should be recorded carefully. In addition, obtaining a surgical history is also critical, because longer operative times, excessive cautery or repeat thoracic surgeries increase the risk of developing postoperative mediastinitis. The physical examination in patients with symptoms suggestive of mediastinitis will usually reveal sternal instability, pain, wound discharge and tenderness. In the case of a slow postoperative recovery, mediastinitis should be considered in differential diagnosis. Majority of patients present within 30 days of cardiac surgery, although some patients can present with mediastinitis up to a year after the surgery (17***).***

4.2. Evaluation

In patients with suspected mediastinitis, a prompt primary assessment should be performed including airway, circulation and breathing. Once a patient is stabilized, imaging examination will be the next step of evaluation. Postoperative mediastinitis may be challenging to assess with imaging due to physiological postoperative changes that are frequently recognized in radiographic studies**.**

4.2.1. Laboratory and radiological features

Leukocytosis is present in almost all patients with postoperative mediastinum, but it is not a specific finding. Mediastinal widening on chest X-ray is a rare finding in postoperative mediastinitis. Other imaging abnormalities that are rarely observed include air fluid levels in the mediastinum or subcutaneous tissue and mediastinal air on lateral chest X-rays. However, mediastinal air on lateral X-rays is not specific in patients evaluated immediately after cardiac surgery.

The two hallmark findings of postoperative mediastinitis, i.e. localized mediastinal fluid and pneumomediastinum (gas bubbles in the mediastinum) are better demonstrated with computed tomography (CT). Although gas bubbles and mediastinal fluid collections may be normal findings following cardiac surgery, they disappear usually within 21 days (18). Therefore, presence of gas bubbles beyond 21 days may be useful indicators if they are accompanied with the other factors. These bubbles are present in more than half of patients with postoperative mediastinitis.

5. Diagnosis

Postoperative mediastinitis is usually diagnosed easily when patients develop infection within several weeks after heart surgery. These patients generally present with characteristic findings of leukocytosis, fever, sternal instability and sternal wound discharge with or without bubbles. The diagnosis is established when these findings are accompanied by bacteremia or systemic symptoms such as chills, fever or signs of sepsis. Wound dehiscence, purulent discharge, erythematous painful incisions accompanied with sternal instability are typical findings. In addition, positive blood culture, leukocytosis and elevated C-reactive protein are often observed. In the absence of local signs and symptoms, leukocytosis and fever may be the only presenting features in a small portion

of patients. Wound discharge is the most common presentation, occurring in 70-90% of cases (4). Patients should be clinically evaluated on a daily basis in the immediate postoperative period and a high index of suspicion should be maintained in order to ensure an early diagnosis. The diagnosis can be made definitely by taking such patients to surgery immediately and by the finding of pus in the mediastinum.

The diagnosis may be more challenging in patients presenting with leukocytosis and fever, but no sternal drainage or infection. The majority of such patients develop sternal wound infection within a few days. It is difficult to make distinction between sternal wound infection due to postoperative mediastinitis and superficial sternal wound infection.

Subxiphoid aspiration and/or CT may be useful in patients with systemic symptoms of bacteremia when there are no findings of sternal wound suppuration and those with subacute symptoms. Later imagings are more useful diagnostically. In a study by Exarhos et al., CT scans had a sensitivity of 100%, but specificity of 33% when performed before postoperat[iv]e 14th day, but they had both specificity and sensitivity of 100% when performed later (18). In addition, CT scans are used for localization of the infected tissue (4).

Sternal puncture has also been used for the diagnosis of postoperative mediastinitis (19). A 21 Gauge needle was entered within approximate margins of sternotomy for aspiration. This approach resulted in the finding of the same organism in all patients. However, further studies are needed to determine safety, effectiveness, sensitivity and specificity of this method.

In a study by Fowler et al., utilization of blood culture for the diagnosis of postoperative mediastinitis was investigated in 5,500 patients undergoing open heart surgery with median sternotomy (20). Among these patients, 46 of 60 (77%) who had bacteremia due to S. Aureus within 90 days of heart surgery developed postoperative mediastinitis due to the same organism. In the same study, patients with negative blood cultures were less likely to develop mediastinitis (20).

In patients presenting with early-onset postoperative mediastinitis, cultures of epicardial pacing wires had a sensitivity of 75% and specificity of 83% in the diagnosis of mediastinitis (21). In such cultures sensitivity, specificity and positive predictive value were even higher when the isolated organis*m was S.* Aur***eu***s.

5.1. Differential Diagnosis

Differential diagnosis of postoperative mediastinitis is shown in Figure 2 (22, 23).

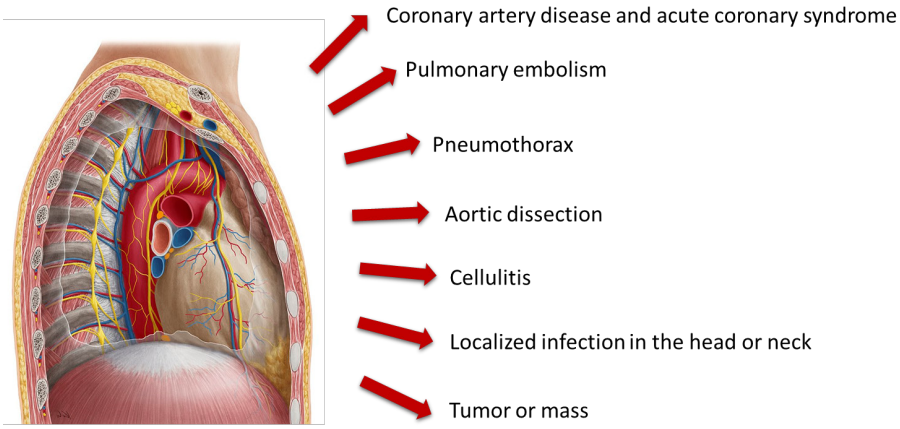

Figure 2. Differential diagnosis of postoperative mediastinitis

6. Treatment / Management

6.1. Antimicrobial Treatment

Systemic antimicrobial treatment should be established once the diagnosis of postoperative mediastinitis is established or suspected and blood cultures are obtained. The initial empiric treatment should have a broad coverage against gram-negative bacilli and gram-positive cocci. A typical regimen consists of i.v. vancomycin and a third generation cephalosporin, an aminoglycoside or a quinolone. This regimen should be adjusted once the results of blood cultures or sternal wound drainage become available. Antimicrobial therapy is generally administered over 2 to 3 weeks. However, 4 to 6 weeks of antimicrobial therapy is required in case of sternal debridement performed without resection.

6.2. Surgical Treatment

Surgical debridement is the primary surgical treatment for postoperative mediastinitis followed by a primary closure. Furthermore, an open wound care may be performed followed by primary or delayed flap closure. In patients with open sternal wounds after debridement for postoperative mediastinitis, vacuum-assisted closure (VAC) or topical negative pressure wound therapy is used before subsequent delayed flap repair.

In a systematic review by Yu et al. including studies evaluating the results of negative pressure wound therapy compared to other methods, negative pressure wound therapy was associated with decreased length of stay in hospital, re-infection and the rate of mortality (24). In a retrospective study including 90 patients undergoing CABG, effectiveness of negative pressure wound therapy was compared with immediate closure with closed irrigation and debridement followed by delayed closure with open dressings. Negative pressure wound therapy has been reported to be associated with lower 90-day mortality and higher 1-year survival (25).

7. Conclusion

Postoperative mediastinitis is a rare, but significant complication of open heart surgery. In case of diagnosed or suspected postoperative mediastinitis, following prompt CT scans early broad-spectrum antibiotics should be initiated. Intensive postoperative care should be performed after surgical intervention. These patients should be closely monitored as worsening symptoms or delayed healing may require repeat surgery. Prevention is the most important aspect of improved outcomes in the management of postoperative mediastinitis. Evaluation of risk factors prior to open heart surgery is helpful in the prevention. Prophylactic antibiotics are also helpful. The most critical measures include sterile techniques and prevention of contamination during operation.

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CHAPTER VI

COMPLICATIONS OF CARDIOPULMONARY BYPASS

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1. CBP Circulation Line Components and Related Complications

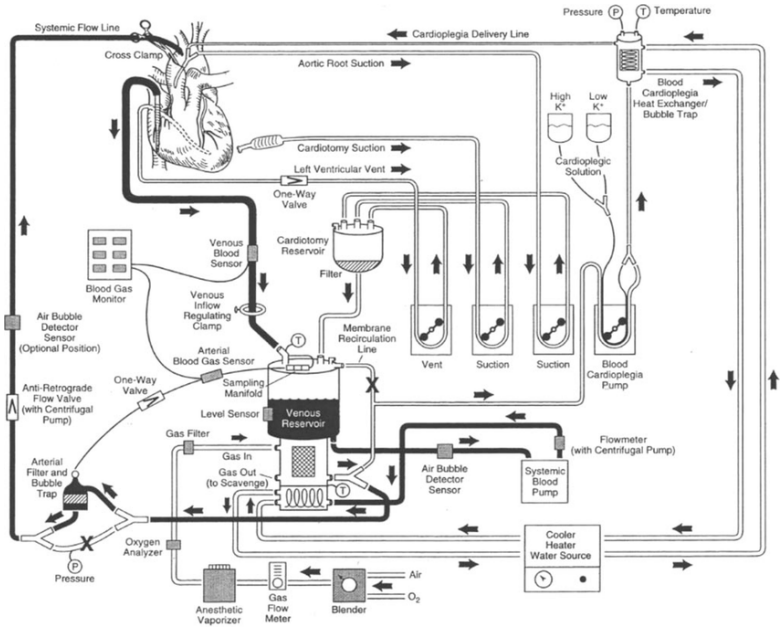

Figure 1

2. Venous Cannules

They can be made of different designs, materials and sizes. They are classified as one-stage (atrial) or two-stage (cavo-atrial).

2.1. Venous Cannulation

Atrial arrhythmias or hemodynamic instability may occur due to manipulation of the heart during venous cannulation. Especially, pulling the heart to the left while the ivc purse sutures are being placed may adversely affect the hemodynamics. This condition is usually temporary. Electrical cardioversion can be performed due to atrial arrhythmia, but this may pose a risk for clotting. A decrease in systemic temperature during sternotomy and cannulation will increase the risk of arrhythmia. In cases where bicaval cannulation will be performed, only svc cannulation and ivc cannulation after entering the pump may reduce the possibility of encountering some hemodynamic disorders. During cannulation, incision should be made as required size. After placing the cannulas, the location of the cannulas should be checked manually by hand and even TEE support can be taken if necessary. Venous cannulas can sometimes pass through the

innominate vein, hepatic vein, coronary sinus, or even through the septal defect to the left side of the heart. If the SVC is cannulated, the incision should be repaired in a way that does not narrow the SVC after the cannula is removed.

During cannulation, pulmonary artery catheters and central venous catheters previously inserted may become dislodged. Care must be taken about this. Rarely, caval sutures can fixate the intracardiac catheters and this may be a reason for reoperation.

Persistent left superior vena cava (PSSVK) should not be forgotten during venous cannulation. The frequency of PSSVK in the general population is rare. However, it is the most common anomaly among systemic venous system anomalies. PSSVK is seen in 0.2 - 0.5% of the general population, but it has been reported to be detected in 3-10% of those with congenital heart disease. It usually opens into the coronary sinus. PSSVK does not pose a significant problem when two stage cannula is used. In patients who will undergo bicaval cannulation, blood coming from the coronary sinus may deteriorate the clean surgical field. Aspiration with retrograde cannula may solve this problem. If there is a shunt during placement of venous cannulas, systemic air embolism may occur. In bicaval cannulation, venous cannulas may prevent physiological venous return. Depending on this, hemodynamic disorders may occur. In this case, cardiopulmonary bypass should be performed immediately.

3. Arterıal Cannul*es*

3.1. Arterial cannulation

The aorta is the most common cannulation site in cardiac surgery. There are also other areas such as the femoral artery or the axillary arter*y.*

3.2. Aortic Cannulation

Calcific plaque displacement at the cannulation site and embolism are important causes of neurological complications in cardiac surgery. Although embolization of small particles does not produce clinical symptoms, it may cause postoperative neurocognitive chang**es.(**1)

Some techniques can be used to prevent systemic embolization. It is possible to detect the presence of calcific plaque by manual palpation of the ascending aorta, aortic arch, and proximal aorta by the surgeon. It is extremely important that the planned arterial and cardioplegia cannulation sites, proximal anastomosis areas, and the area to be cross-clamped in the aorta are free from

any significant vascular disease. Unfortunately, the degree of atherosclerotic disease in the aorta can sometimes be overlooked with manual palpati**on.(**2)

The more widespread use of TEE in cardiac surgeries will help us to evaluate atherosclerosis in the ascending aorta more efficiently. Although the middle parts of the aortic arch cannot be fully evaluated with TEE, the ascending and descending aorta can be evaluated with this techni**que(2).** Recently, significant atherosclerotic disease in the ascending aorta can be examined more sensitively with epiaortic ultrasound before cannulation**(2).** With this technique, the ultrasound probe in a sterile sheath is taken to the operating field, the pericardium is filled with serum sale, the probe is directly contacted with the aorta, and the cross-sectional area and longitudinal imaging of the aorta is performed. If significant atherosclerotic disease is encountered, some alterations can be made in the surgical plan such as:

1. Axillary or femoral artery cannulation
2. Selection of atherosclerosis-free zones in the aorta
3. Off-pump approach in patients with only CABG planned
4. Incorporation of ascending aortic replacement into the original surgical plan.

During intraluminal insertion of the cannula, it is bled back, allowing pulsatile blood flow to flow into the arterial line. This back-bleeding process is beneficial in preventing embolization of both small particles and small amounts of air bubbles at the cannula tip.

Cannula may not be placed properly when inserting. Inadequate aortotomy may be due to wall fibrosis or calcific plaque.

The intramural inserted cannula causes obstruction of arterial blood flow. This situation should be recognized immediately. Vigorous pushing of the cannula may result in rupture of the cannulation site or posterior aortic wall. Misdirection of the aortic cannula tip may result in cannulation facing the aortic valve, retrograde cannulation, or cannulation facing the innominate artery. Intramural hematomas that may occur at the cannulation site should be prevented by rapid opening of the adventitia.**(3)**

Bleeding after decannulation should be stopped with additional stitches. Recurrent hemorrhage and pseudoaneurysm are late complications of aortic cannulation.

3.3. Femoral Artery Cannulation

The femoral artery is cannulated percutaneously or by exploration usually. Direct injury to the femoral artery, bleeding, dissection, pseudoaneurysm formation, lymphocele, peripheral nerve injury, retrograde dissection of the aorta, air or calcific plaque embolism to the distal extremity are the complications that may develop during cannulation. Distal extremity ischemia can be prevented by using a graft with end-to-side anastomosis technique, cannulating the graft, or placing a lower number of cannula into the femoral artery.

Retrograde cannulation with the femoral artery can cause retrograde embolism of existing plaques in the iliac artery or descending aorta. If calcification is detected in the descending aorta with TEE, axillary artery cannulation may be preferred or the patient can be cannulated according to CT angio images.

3.4. Axillary Artery Cannulation

The right axillary artery should be preferred over the left axillary artery. The atherosclerotic process progresses more slowly in the axillary artery than in the aorta and femoral artery. Cannulation can be done with an end tos ide graft.

4. Oxygenator

The two basic oxygenators are the membrane (more common) and air bubble oxygenators (4). The oxygenator is responsible for both oxygenation and ventilation (CO2 removal) functions. In membrane oxygenators, the contact between the blood and the membrane occurs only at the beginning of the CBP. The protein layer formed on the membrane for a while after the onset of CBP prevents blood-membrane contact. Membrane oxygenators do less hemolysis than air bubble oxygenators. At the same time, less complement granulocyte and platelet activation and cerebral microembolism rates are reported. The use of heparin-coated oxygenators has recently become widespread. In this way, there will be a further decrease in the systemic inflammatory response**.**

5. The Pump

Roller pumps consist of sets of tubes placed in curved troughs. Cylinders or rollers rotating around the tubes provide arterial flow by compressing the silicone tube and the blood in the grooves. In any roller pump, the blood flow rate is directly proportional to the pump speed, that is, the number of revolutions per minute. The degree of clogging of the cylindrical rollers to the tube set is

important here. Excessive pressure on the tube set will cause hemolysis, while insufficient pressure will decrease the effective flow rate. The most ideal amount of pressure is provided even though the rollers do not physically occlude the tube set end to end. In the complications specific to roller pumps, attention should be paid to improper calibration (59) and fractures in the silicone tubes. Massive air leakage into the arterial system is one of the most sensitive points of roller pumps, especially when the venous reservoir level is not carefully monitored and emptied undesirably.

Fragmentation and rupture (spalliation) of microparticles in silicone tubes are one of the most undesirable effects of these pumps. This complication can be limited by arterial filters. If there is an undesirable blockage (unnoticed clamp) anywhere on the line in roller pumps, there will be tearing / disintegration in the silicone tube or its connections.

The driver or impeller design (vortex effect) in centrifugal pumps ensures completely nonocclusive operation. The flow rate in this type of pump depends on the rotational speed of the pump as well as the afterload of the patient and on the perfusion line.

If the pump does not work, blood may flow back from the patient to the pump, and if the arterial line is not clamped, very severe hypovolemia may develop (63). By the same mechanism, massive air escaping to the patient for any reason can be withdrawn from the arterial cannulation site. While the pump is running, an obstruction on the arterial line will result in the device stopping, so it does not cause a rupture in the tube set as in the roller pump. Massive air embolism does not develop in centrifugal pumps as in other pumps. In large quantities of air embolism, the centrifugal pump is self-stopping (de-prime). Smaller amounts of air bubbles can easily be pumped through the arterial line to the pati**ent.**

6. Heat Exchanger

A heat exchanger is added to the perfusion line to cool or warm the blood. Malfunction of heat exchanger may cause the patient's temperature to be uncontrollable duri**ng CPB.**

7. Cardıotomy Aspırator

Blood evacuated from the surgical site reaches the perfusion line through a cardiotomy or venous reservoir through a microfilter and foam reducing cavity. These lines work connected to the roller pump. These lines can cause hemolysis,

air, fat, particle microembolism, coagulation activation, fibrinolysis, cellular aggregation, or platelet dysfunction.

8. Auto Transfusion Device (Cell Saver)

One of the methods used to reduce the use of homologous blood and blood products in open heart surgery is to return the accumulated blood to the patient with the 'cell saver' device. By using the cell saver device instead of the cardiotomy aspirator, the blood evacuated from the surgical field can be cleaned again and given to the patient. Thanks to this technique, the cleaned cells are washed with serum sale, centrifuged and separated from the plasma. The blood cells can then be autotransfused into the patient by intravenous or pump delivery. Contrary to cardiotomy aspiration, the blood returning to the patient with the cell saver can be cleaned from air-fat-tissue particles as a result of filtration and then reinfused. The disadvantage of the cell saver technique is the destruction of clotting factors, platelets, and other plasma proteins during the centrifugation process. From a practical point of view, cell saver can be used in surgeries such as CABG, where much bleeding is not expected in the surgical field. Cell saver aspiration alone may not be practical in surgeries where larger amounts of hemorrhage are expected, such as redo surgeries and aortic surgery. The cell saver technique or autotransfusion is very helpful in delivering large volumes of blood remaining in the venous reservoir after termination of CPB.

9. Decompression of The Left Heart (Aspiration With Vent)

In cardiac surgery, the left heart can be decompressed with aspiration lines or vents. (5)

During cardioplegic arrest, the bronchial, thebessian vein, and coronary sinus blood flow draining to the right heart will pass to the left side of the heart unless ventilated. In aortic regurgitation, the left ventricle fills.

The left heart can be ventilated in various ways.

Possible complications :

There may be rupture of the aortic vent, cannulation site, early or late aortic dissection.

Risk of bleeding at the apex of the heart, left ventricular injury and pseudoaneurysm formation in the late period are complications that can be seen.

Air may be leaked into the left heart during insertion and removal of vent catheters. Excessive aspiration of the left vent can cause air to escape from purse sutures above the heart or from open coronary artery anastomoses. Inadvertently giving positive pressure air into the vent line is dangerous and care must be taken.

De-airing of intracardiac air with left ventricular ventilation can be done carefully with the help of TEE.

10. Mechanical Complications That May Develop During *CBP*

10.1. Energy Outage

Battery equipment is requir***ed.***

10.2. Massive Air Embolism

Massive air embolism affects the systemic circulation rather than the pulmonary circulation. Its incidence is around two per thou**sand(**7). An average of 50 percent of those who suffer from these complications die or suffer permanent neurological damage.

Air embolism may occur at different stages during surgery.

Before aortic clamping, air may enter from the aortic or cardioplegia cannulation sites. This can be avoided with the back bleed technique.

When the left heart opens, air can enter. This air must be carefully discharged with the help of a vent cannula and TEE.

In a CABG case, air may enter from the coronary artery openings when the vent withdraws excessively.

Careless emptying of the venous reservoir may result in the pressure of air from the arterial line into the systemic circulation. This situation can be prevented by systems that give an alarm when the venous reservoir is at a low le**vel.**(8)

If air is detected in the arterial line, CBP should be stopped immediately and air should be evacuated from the line, then should be re-entered to the CBP.

Retrograde cerebral perfusion and deep hypothermia may limit permanent neurological damage if excess air has escaped into the arterial circulati**on.** (9) Pharmacological therapy may also be beneficial. Corticosteroids, anticonvulsants, barbiturates, and diuretics can prevent neurological damage or symptoms of that damage. It has been reported that postoperative hyperbaric O2 therapy may be beneficial in such case**s.**

11. Monitoring of CBP and Related Complic*ati*on*s*

11.1. Arterial Catheters

The most common cannulated artery is the radial artery. Femoral, carotid, and brachial arteries can also be used for cannulation. Complications include infection, embolization ischemia, and hematoma formation. Cellulite may be seen at the cannulation site. Less common complications include skin necrosis, arterio venous fistula formation, median nerve neuropathy.

Sterile femoral artery cannulation is performed in patients who will have radial artery grafts. Infection at the cannulation site, bacteremia, distal embolization, a-v fistula formation, and femoral nerve neuropathy are among the potential complications.

11.2. Central Venous Catheters

IV entry of drugs, liquid blood products

CVP monitoring

It can be used for pulmonary artery cannulation with Swan ganz catheter.

The most common early complications of central venous catheters are:

Undesirable injury to the adjacent artery

Incorrect placement of the catheter

Pneumothorax(10)

Pulsatile blood coming from the catheter should suggest an arterial puncture. In such a case, the catheter should be withdrawn immediately. The incidence of carotid artery puncture in jugular procedures is 4%. If the jugular or femoral route is used, effective compression should be applied instead of cannulation to prevent hematoma formation. The benefit of manual compression in subclavian artery punctures is controversial. Rarely, arterial cannulation may not be noticed until after catheter insertion. Arterial catheterization can be determined by the inability of IV fluid to flow easily through the catheter, the appearance of an arterial pressure curve, or the observation of the catheter in the arterial trace on the thorax. During the operation, unexplained blood loss or hemodynamic instability after CVC should suggest a vascular injury due to CVC and the ipsilateral pleural space should be examined.

Catheters placed by the jugular or subclavian route should be placed in the area between the SVC and the right atrial junction. In the evaluation

of an unexpected hematoma or pleural effusion, the location of the tip of the catheter is observed with a chest X-ray taken immediately after surgery. The catheter may be in the wrong localizations. It can be counted that the catheter turns in the opposite direction and goes to the contralateral neck region, distal subclavian vein, vena cava inferior, contralateral subclavian vein, and loops through the right atrium. An incorrectly positioned catheter may not be harmful to the patient, however, catheter withdrawal is recommended in all cases. A guidewire that is involuntarily lost in the central circulation should be retrieved immediately.

Pneumothorax is observed in 1-4% compared to the CVC route. Pneumothorax should be suspected when there is hemodynamic disorder or low S02, and urgent telecardiography should be performed and air should be decompressed by inserting a tube into the pleura.

Venous thrombosis can be observed among the late complications of SVC. Unilateral upper extremity or neck swelling and discomfort are in the foreground. Diagnosis can be made with USG. The catheter should be withdrawn and systemic anticoagulation should be given. Redness and afflux at the catheter insertion site, fever, leukocytosis, bacteremia suggest catheter-related infection. It is necessary to withdraw the catheter. If the need for a catheter continues, it may be necessary to insert a catheter from another location.

11.3. Transesophageal Echocardiography

Intraoperative TEE is a method that is widely used today, especially in valve and aortic operations, and provides cardiac imaging in the repair of congenital defects. Perop TEE is a method that helps to evaluate the most appropriate surgical approach (valve replacement, repair), provides information about heart anatomy and functions, and helps to predict the early outcome of the surgery. In large series, intraoperative TEE morbidity rates are reported as 2% and mortality rates as 0%.**(11)**

Transient odynophagia is the most common complication of intraoperative therapy**(12).** While placing the TEE probe, care should be taken to ensure that there is no dental injury, no displacement of the endotracheal tube, and no upper GI bleeding due to mechanical irritation of the intraoperative TEE.

The most serious complication is GIS perforation. Dysphagia in preoperative patients is a risk factor for perforation.

12. Anticoagulation and Complications in C*BP*

12.1. Anticoagulation for CPB

The most commonly used is heparin. Other anticoagulants may also be used. Unfractionated heparin is a mixture of mast cell polysaccharides. Its anticoagulant effect is achieved by potentiating its (AT3) activity, by binding directly to cofact 2 and inhibiting thrombin. Although anaphylaxis, pulmonary edema and DIC are rare, they can be seen.

The most common complication of heparin is bleeding. Despite the risk of excessive bleeding during CPB, the use of heparin is essential. The use of heparin in excessive doses during CPB may cause fibrinolysis and platelet activation. Inadequate anticoagulation during CPB causes overuse of coagulation factors.

Heparin loading dose in CPB is 200-400 IU/kg, and the maintenance dose is calculated as 50-100 IU/kg during CPB. In addition, 10000-20000 heparin is added to the pump before entering the CP***B.***

12.2. Heparin Resistance

It is the situation where adequate anticoagulation cannot be achieved despite taking standard dose heparin before entering CBP. Congenital AT3 deficiency, thrombocytosis, pregnancy, sepsis, hypercoagulation and coagulapathies may cause heparin resistance. AT3 deficiency is the most common etiologic cause and can be treated with FFP or recombinant AT3 administratio***n.***

12.3. Heparin Induced Thrombocytopenia

Heparin-induced tombocytopenia (HIT) can be seen in 10% of patients treated with heparin and in 1-5% of surgical patie**nts(1**3). This high incidence in surgical patients is partly attributed to the indication for extensive use of heparin and partly to the high prevalence of heparin-related antibodies found in patients referred for cardiac surgery**.(13)**

The main treatment is heparin discontinuation and reduction of thrombin production with another anticoagulant agent. A subset of HIT cases is thrombosis due to HIT, and care should be taken in terms of thrombosis-related end-organ damage.

12.4. Heparin Alternatives

Alternatives for anticoagulation should be used in a patient with heparin or protamine allergy or known to have HIT. These are warfarin, low molecular

weight dextran, low molecular weight heparin, antirombin agents (hirudin, argotroban) and prostocyclines.

13. Anticoagulant Monitorization for CPB

For CPB, heparin starts with a certain loading dose, then heparin levels are periodically monitored with activated clotting time (ACT).

Goal;

1. To ensure adequate anticoagulation before starting CPB
2. Evaluation of required additional heparin dose during CPB
3. It is the demonstration that heparin levels are sufficiently neutralized after C**PB**

14. Neutralization of Anticoagulation

When CPB is to be terminated, the anticoagulant effect of heparin is neutralized with protamine sulfate. Overdose of protamine may cause platelet dysfunction, postoperative bleeding tendency, and unnecessary blood transfusion requirement.

Various methods can be used to calculate the adequate dose of protamine to be administered. The simplest method is to administer a fixed dose of protamine iv to the amount of heparin given. The dose calculation here can be based on the initial heparin dose or the total heparin dose. Various approaches have been described in this application; There are applications such as protamine neutralization at doses ranging from 1 to 5 mg to 1 mg of heparin. A second method for calculating the appropriate protamine dose is the system based on the measurement of activated clotting time (ACT) measured before and after CPB and reflecting heparin dose-response curves. With the curve obtained, the appropriate protamine dose is calculated by terminating CPB. It has been reported that lower doses of protamine are administered with this method compared to the fixed dose administration. However, the heparin dose-response curve method also has potential disadvantages. In fact, the fact that heparin dose-response curves are not linear may result in erroneous dose adjustments at very low or high anticoagulation levels. Protamine titration measurement is the most advantageous system compared to the other two methods. In this way, 30-40% less protamine can be applied compared to the heparin dose-response curve calculation method. Very serious undesirable side effects may occur

during protamine administration. Predisposing risk factors for these adverse reactions include the patient's pulmonary hypertension, previous exposure to protamine or protamine-containing insulin preparations, previous vasectomy, and fish allergy. Protamine reactions can generally be described in three main groups: type I transient hypotension, type II anaphylaxis, and type II pulmonary vasoconstriction. Type I protamine reaction is mediated by histamine released from mast cells and basophils. This effect becomes more pronounced with rapid injection of the drug. Therefore, it is recommended to administer protamine for 5-10 minutes or longer.

Although histamine receptor blockade before protamine initiation reduces this effect, it does not completely eliminate the possibility of a type I reaction. Histamine release causes a decrease in systemic arterial and central venous pressure. There will also be a decrease in cardiac output, often due to a decrease in preload.

Classically, in the type II protamine reaction, protamine interacts with the IgE on the mast cell surface and causes degranulation. Its symptoms include rash, bronchospasm, edema, stridor, hypotension, and cardiovascular collapse. Exposure to protamine or protamine-containing insulin preparations (NPH, or protamine-zinc insulin), past vasectomy, and fish allergy increase the risk of type 2 reactions. In Type 3 protamine reaction, patients develop acute pulmonary hypertension, decrease in left atrial pressure, right ventricular failure and systemic hypotension. It is not known exactly whether the protamine dose is effective in the formation of this picture . It may be necessary to re-enter CPB. It is not possible to predict whether the same reaction will occur with the re-administration of protamine. Although the mechanism of the type III reaction is not understood, protamine-heparin complexes cause the release of vasoactive substances (free oxygen radicals, thromboxane A2, etc.) via complement. estimated .

When protamine is given, there may be residual heparin in the circulation if the dose is insufficient or if heparin is released from protein-bound heparin from heparin-protamin complexes or from elsewhere. This may cause undesirable bleeding after **CPB.**

15. CPB and Bleeding

Cardiac surgery patients especially tend to have postoperative mediastinal bleeding. Although the incidence of severe bleeding after CPB varies according to definition, the frequency of 24-hour postoperative drainage greater than 2

liters has been reported to range from 5-7% . The incidence of reexploration due to excessive postoperative drainage in patients undergoing adult cardiac surgery has been reported as 3-5% . Reexploration following a cardiac surgical approach is associated with an ultimately increased morbidity and mortality. In a large series study, it was reported that reexploration doubled the operative mortality and caused significant renal failure, ARDS, prolonged mechanical ventilation, sepsis, and atrial arrhythmias. In addition, blood and blood product transfusions due to excessive bleeding; causes potential adverse events such as the transmission of blood-borne diseases (hepatitis, HIV), wound infection, transfusion react**ions. (14)**

16. Physiopatological Consequences of Cardiopulmonary Bypass

Blood-surface contact

Effects and consequences of hypothermia

Metabolic effects

It will be limited by the effects of CPB on the pulmonary, renal and neurolo**gical systems.**

17. Blood-Foreign Surface Contact Results

Physiologically, blood and plasma are in contact only with vessels covered with endothelium. In cardiac surgery procedures using cardiopulmonary bypass technology, different components of the blood and plasma perfusion line come into contact with foreign surfaces such as open wounds. As a result of this contact, some specific systemic reactions begin against CPB.

Following contact with a nonendothelial surface, the plasma proteins are instantly adsorbed and form a monolayer of different proteins. Among the plasma proteins that are adsorbed, the most important is fibrinogen . The type and relative mix of proteins vary with the nonendothelial surface. The aim here is to examine the results of stimulation of the contact activation system, intrinsic extrinsic coagulation pathways, fibrinolytic and complement systems during CPB; In addition, platelets, endothelial cells, neutrophil monocytes and lymphocytes are **also evalu**at**ed.**

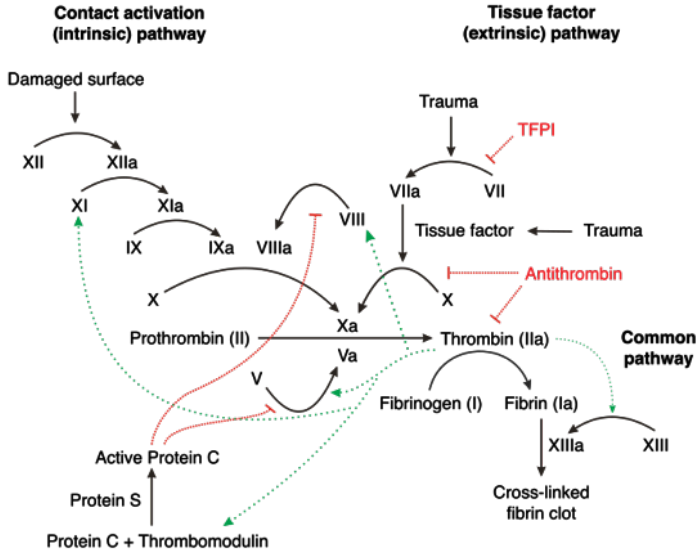

Figure 1.2

18. Contact Activation System

Adsorption of factor XII (Hageman factor) upon contact with a nonendothelial surface initiates a cascade in the contact activation system pathway (figure 1.2). The presence of pre-kallikrein and high molecular weight kininogen (HMWK) forms active protease factor XIIa and XIIf .Factor XIIa converts factor XI to Xla by the action of Kallikrein and HMWK. This is the beginning of the intrinsic coagulation pathway; eventually, thrombin will be formed. Kallikrein and factor XIIa are also a direct agonist of neutrophils.

18.1. Intrinsic Coagulation Pathway

Although it has been suggested that the extrinsic pathway may be more important in the systemic reaction to cardiopulmonary bypass, there is evidence that activation of the intrinsic coagulation pathway also plays an important role. The complex consisting of phospholipid (PL), FVIIa and FIXa binds to FXa and forms FX. This is the beginning of the entry into the coagulation pathway.

18.2. Extrinsic Coagulation Pathway

Tissue factor is a membrane-bound protein that does not come into contact with blood under physiological conditions. Activated monocytes and endothelial cells also have tissue factor.

During cardiopulmonary bypass, tissue factor activated F7a and PL both convert F9 to F11a and F10 to F10a, promoting entry into the extrinsic pathway. Factor Xa occurs in both intrinsic and extrinsic pathways. Basically, the intrinsic pathway is activated in the perfusion line, and the extrinsic pathway is activated in the surgical wound area. The common product of both coagulation pathways is thrombin, a circulating protease.

18.3. Fibrinolysis

One important effect of circulating thrombin is to activate endothelial cells, causing the release of tissue Plasminogen activator (tPA) and binding with fibrin. The combination of fibrin, tPA, and plasminogen cleaves plasminogen into plasmin: plasmin is then converted to fibrin.

18.4. Complement System

During cardiopulmonary bypass, both classical and alternative complement pathways are activated. Blood-surface contact in the perfusion line activates the classical pathway; C3 convertase occurs via C1, C2, and C4: C3, dividing into C3a and C3b. In the alternative route, factors B and D provide the formation of C3b.

It is emphasized that the second pathway may be more important during cardiopulmonary bypass . C3b divides C5 into C5a and C5b. C5b binds with C6-C7-C8-C9 to form the terminal complement complex (TCC). TCC interacts with the cell membrane, causing cell lysis and additionally increased thrombin formation . The released factors C3a, C4a and C5a are vasoactive substances; it is also a C5a major neutrophil agonist.

18.5. Platelets

Circulating platelets undergo many undesirable effects during CPB. The first noticable effect of this is probably the decrease in their number in the circulation due to dilution during priming of the perfusion line. Heparin prolongs bleeding time by preventing platelets from binding with von Willebrand factor. Heparin also increases platelet susceptibility to circulating agonists such as thrombin, C5b, plasmin, Cathepsin G, serotonin, and epinephrine. This interaction causes both reduction in platelet count and platelet dysfunction.

The adhesion and aggregation of circulating platelets with each other is another factor that causes a decrease in their number. Some cell surface glycoprotein receptors expressed by activated platelets promote aggregation.

Apart from the aggregation among themselves, platelets also form aggregates with monocytes and neutrophils.

After CPB, there is a 30-50% decrease in the number of platelets in the circulation. When CPB is terminated, platelet fragments are found at a high rate in addition to the platelets that remain intact. A decrease in all platelet functions and a prolongation of the calculated bleeding time are observed.

18.6. Endothelial cells

Endothelial cells are activated by thrombin, C5a, different cytokines (IL-1, tumor necrotizing factor-TNF) during CPB.

18.7. Neutrophils

Neutrophils are largely responsible for the systemic inflammatory response that occurs during cardiopulmonary bypass. These cells are strongly activated by kallikrein and C5a during CPB. In addition, F12a activates neutrophils in various substances such as heparin, leukotriene B4, IL-1, IL-8, and TNF. Activated neutrophils secrete damaging substances such as elastase, cathepsin G, lysozyme, myeloperoxidase, acid hydrolase, bacterial permeability agent, lectoferrin, collagenase, hydrogen peroxide, hydroxyl radicals, hypobromus and hypochlorous acids.

18.8. Monocytes

Monocytes are activated by monocyte chemotactic protein-1, C5a, immune complexes, endotoxin and IL-1. Activated monocytes are tissue factor both at the perfusion line and at the open wound site ; other than that, it secretes different cytokines such as IL-1, IL-2, TNF-α, which peak a few hours after the onset of CPB. The number of circulating monocytes does not change during CPB, but increases a few hours after the onset of CPB.

18.9. Lymphocytes

B and T lymphocytes decrease in number and function a few days after CPB.

19. Physiopathological Consequences of Hypothermia

The aim in hypothermia is to protect different organ systems from ischemic damage that may develop during surgery. It is related to the oxygen requirement and supply of the organs related to body temperature.

The direct effect of hypothermia in the nervous tissue is the preservation of high-energy bonds and the reduction of the secretion of excitatory neurotransmitters. Decreased tissue oxygen use during hypothermia allows CPB to be administered with lower flow rates.

Hypothermia can cause heart block and atrioventricular arrhythmias in the heart. Hypothermia in the lungs will cause decreased ventilation and increased renal vascular resistance in the kidneys. It causes polyuria and glucosuria with decreased tubular reabsorption.

Hypothermic CPB often leads to hyperglycemia.

Gluconeogenesis and glucogenolysis increase, while insulin secretion decreases. Furthermore, insulin insensitivity to exogenous insulin during hypothermia can be troubling. Therefore, it may be late for the surgical team to closely monitor serum glucose levels and to intervene with exogenous insulin when necessary. There are many evidence that controlling even moderate hyperglycemia can significantly reduce postoperative wound infection (15).

Along with hypothermia, changes occur in the fluid-electrolyte balance. Hypothermia causes a decrease in free water clearance and serum potassium concentrations and an increase in serum osmolality.

Systemic and pulmonary vasoconstriction occur with hypothermia below 26°C . The resulting arterio-venous shunts can have a very detrimental effect on oxygen transport to the tissue. Increase in blood viscosity, red blood cell aggregation and roll formation may further impair tissue oxygenation. These undesirable effects of hypothermia can be limited with the help of appropriate anesthesia, hemodilution and vasodilators.

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CHAPTER VII

COMPLICATIONS IN CHEST WALL RESECTION SURGERY

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Chest wall resection is defined as full-thickness removal of bone and soft tissues. Common indications of chest wall resection includes tumors (primary, metastatic, or recurrent chest wall tumors), infection, trauma, and local invasive radiation necrosis. The most frequent complications are respiratory or wound complications. When bone structures are resected, reconstruction is necessary to preserve rigidity and prevent respiratory failure. Chest wall resection and reconstruction presents a big challenge for thoracic surgeons, both in neoplastic and non-neoplastic patients. Although much progress has been made since Clagett first reported his experience about chest wall resections for primary tumors, many technical aspects are still being debated and several different techniques have been described over the years. (1,2). Modern prosthetic materials offer excellent changes for replacing resected tissues even in case of very extended resections, but despite improved surgical techniques and prosthetic materials, several major complications, especially respiratory complications, are still reported in range of 14%–46% (3–5).

Complications can be examined in three main sections:

1. Respiratory complications related to the size of the defect after resection and the size of the flail formed.
2. Surgical complications after reconstruction including bleeding, seroma, loss of graft, graft infection, hematoma and necrosis of the flap, omentum necrosis, and empyema.
3. General complications related to the interventions include cardiac complications, acute renal failure, deep venous thrombosis, and urinary and anesthetic complications.

Preoperative predictive risk factors for complications after chest wall resections include:

1. Additional systemic diseases, diabetes mellitus, chronic obstructive pulmonary disease (COPD), hypertension, etc.
2. Smoking,
3. Low lung capacity,
4. Advanced age,
5. Additional surgical intervention, (combined chest wall and lung resections)
6. Previous chemotherapy and radiotherapy

Moreover, ulceration of the tumor, sternal resection, the use of omentum and the size of defect are factors that increase complications rates (5).

Respiratory complications are the most frequent complications, seen at a rate ranging from 20%–24% (6). Full-thickness chest wall resection leaves behind an area that paradoxically participates in respiration. Respiratory failure rates vary widely and range between 0% and 20% in literature (6–8). This wide variability in the respiratory failure rates may explained due to several factors, including specific risk factors, the type of tumor, associated pulmonary resections, and the extent **of resection.** Reconstruction provides structural stability and helps sustain the respiratory mechanism. Reconstruction is also required for patients with ≥3 resected ribs and defects that are ≥5 cm in diameter, even for with smaller defects those suspected thoracic cage instability. Posterior defects up to 10 cm can be closed without prosthesis because of the overlaying scapula covering the chest wall (9,10). Anterior defects need reconstruction since anterior and anterolateral movements are greater than posterior movements (11). Rigid reconstruction has widely been used due to its effectiveness in stabilizing the thorax, but it can also induce severe complications, including deep infections (12). All rigid reconstructions should be closed with soft tissue; thus, omentoplasties, skin grafts or pedicled myocutenous flaps are used in combination to close the prosthesis in complex chest wall defects. Combined chest wall and lung tumor resection has high morbidity when compared to tumors rising from the chest wall (9). Flail-related respiratory failure may occur after extensive chest wall resections.

Seroma

Uninfected fluid may accumulate around prostheses used in reconstruction after chest wall resection. Leusa et al. reported a seroma ratio of 5% in their series

(13). It is recommended to use free drains during the operation to prevent seroma formation. The catheter should not be withdrawn unless the drainage is below 25 cc. When seroma occurs, aspiration and pressure dressing is recommended, and in the case of failure, a catheter can be placed, but it should be kept in mind that placing a catheter may predispose the patient to infection.

Infection and prothesis complications

Wound infection and complications related to prosthesis is the second most common complication in chest wall resection and reconstruction and it is seen in range of 7%–20% (5). The use of prosthetic material on contaminated and infected wounds is not recommended**.** Wound infection has been reported at a rate between 4.6% and 16% in different series (3,10). Both prosthetic and wound complications often require removal of the prosthesis, but the decision to remove the prosthesis should be made after clinical observation and radiological examinations (5).

Fracture and displacement of prothesis

Fractures are commonly seen in reconstructions with methylmethacrylate. Sometimes a fracture of the prosthesis and methacrylate toxicity can be seen. Fractures can also be observed in titanium meshes, similar to methylmethacrylate meshes (14).

Flap hematoma and necrosis

Autologous tissue transfers are used in 19%–57% of patients (14). Pedicled muscle or musculocutaneous flaps include the latissimus dorsi muscle flap, pectoralis major muscle flap, rectus abdominis muscle flap, and variants [vertical rectus abdominis muscle (VRAM), transversal rectus abdominis (TRAM)], external oblique muscle, serratus anterior muscle flap, and omentum flap. Flap hematoma and necrosis occur in 5% of patients (5). Flap necrosis is seen especially in large flaps due to impaired blood supply. Ulceration of the tumor, radionecrotic areas, and omental flap reconstruction lead to wound healing problems. Lans et al. suggested that care should be taken not to fold the omental flap while preparing it (10). Abdominal hernia may occur after use of TRAM and VRAM.

General complications

Due to insufficient coughing and an inability to clear secretions, atelectasis and subsequent pneumonia may develop. Mansour et al. reported a pneumonia

ratio of 14% (15). Acute respiratory distress syndrome (ARDS), pleural fluid accumulation, empyema, bleeding, prolonged air-leakage, aspiration, acute renal failure, and pancreatitis may also be seen (15). The mortality rate has been reported as 3%–7% (5).

New arguments will continue with the development of surgical techniques and prosthetic material technologies such as 3D, biomaterial, and stem cell (16–18). Currently, minimally invasive treatment in thoracic surgery is being used extensively. When compared to open surgery, it has obvious advantages, such as less postoperative pain, fast recovery, and reduced postoperative complications. Improvements in technical proficiency has led video-assisted thoracic surgery (VATS) to take place in this field as well (19). VATS may be a preferable option for appropriately selected patients (20).

A multidisciplinary approach is required in chest wall resection and reconstruction. Appropriate patient and prosthetic material selection, accurate postoperative care, pain control, and early mobilization are important to prevent complications

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CHAPTER VIII

COMPLICATIONS IN REGIONAL LYMPH NODE DISSECTIONS

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1. Cervical dissections

Cervical neck dissection includes the removal of the lymph nodes that localize between the mandible and the sternum-clavicle in the vertical plane, and between the omohyoid venter superior and anterior border of the trapezius muscle. Cervical lymph nodes are enveloped by the investing fascia of the neck (1) and studied in five levels. Level I includes the submandibular and submental triangles divided by the venter anterior of the digastric muscle into Levels IA and IB. Levels II to IV are jugular lymph nodes, with Level II located at the most cranial position. A transverse line passing through the hyoid bone divides Levels II and III, whereas omohyoid muscle divides Levels III and IV. Level V is the posterior cervical triangle between the posterior border of the sternocleidomastoid (SCM) muscle and the anterior border of the trapezius muscle.

In cervical dissections, the position of the patient is critical. Back support should be placed under the shoulders for the cervical extension. Many incisions have been identified, including Martin, Schobinger, Apron, Lahey and Macfee (2). Location, shape, and lengths of the incisions have specific functional effects on exploring underlying lymph nodes and preventing complications. Even though elevating the skin flaps under the platysma muscle helps the perfusion, the tips of the triangular skin flaps are still at risk for skin necrosis. When at least two incisions are used, skin incisions should be positioned so that the tips of the triangular flaps are not close to the major vascular structures such as carotid arteries or internal jugular veins. Another important point about the incisions is the length of the incision. The incision should go back enough to expose

the posterior aspect of the SCM muscle to allow wide exposure even in the supraomohyoid selective dissections. The posterior part of the incisions usually crosses the external jugular vein and the great auricular nerve, which should be kept in mind.

Musculocutaneous flaps are elevated in the cranial and caudal directions to expose the lymphatic tissue enveloped by the investing fascia of the neck. Needle tip monopolar cautery is usually used to elevate the flaps. However, a suitable-sized blade should be used to elevate the cranial flap to prevent damage to the marginal mandibular nerve.

After the flaps have been elevated, the fatty tissue that includes lymph nodes is resected according to the oncologic needs (Figure 1). The complications and ways to prevent them can be grouped in the levels:

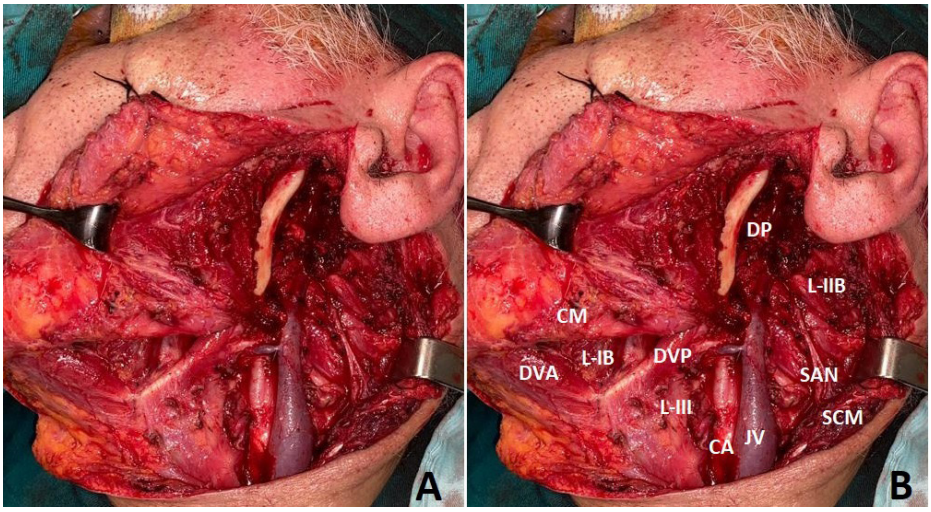

Figure 1. Photo of the patient after superficial and deep parotidectomy and cervical lymph node dissection. (Abbreviations: CA: Carotid Artery, CM: Corpus of the mandible, DP: Deep lobe of the parotid gland(removed), DVA: Digastricus venter anterior, DVP: Digastricus venter posterior, JV: Jugular Vein, L-IB: Level IB lymph nodes, L-IIB: Level IIB lymph nodes, L-III: Level III Lymph nodes, SAN: Spinal Accessory Nerve, SCM: Sternocleidomastoid Muscle)

Level I: Marginal mandibular branch of the facial nerve (this nerve will be named the marginal mandibular nerve in the remaining part of this chapter) passes in the Level IB, and it is usually within one finger length from the inferior border

of the mandible (3). However, it may go below this level (4) and it can even pass 3-4 cm below the mandible (5). Incising the investing fascia 4 centimeter below the lower edge of the mandible (2) or below the submandibular gland and elevating the cranial platysma flap deep to the investing fascia have the marginal mandibular nerve stayed in the flap. If the cranial platysma flap is elevated superficial to the investing fascia up to the mandibular rim, then the flap should be elevated precisely to protect the marginal mandibular nerve, which usually passes in very close proximity to the surface. Monopolar cautery can easily damage the nerve when used for this purpose. Another possible way to protect the marginal mandibular nerve is the Hayes-Martin Maneuver (6). The marginal mandibular nerve is always superficial to the facial artery and vein. Hayes Martin Maneuver includes exploring the posterior facial vein, ligating it, and elevating the flap deep to the facial vein to protect the nerve.

Another important structure that traverses Level I is the facial artery. The facial artery is a branch of the external carotid artery that arises deep to the stylohyoid and the posterior belly of the digastric muscle (7). It makes a loop posterior to the submandibular gland (8), gives off small branches to nourish the submandibular gland, and goes to the medial direction, where it gives off the submental artery. The submental artery is a sacrifiable vessel that usually passes deep to the anterior belly of the digastricus muscle (9, 10) and usually causes annoying bleeding during Level IA dissections. As it passes deep to the anterior belly of the digastricus muscle, it usually bleeds twice during Level I dissections.

The submandibular gland is an important structure localized in Level IB. It is the source for 70% of the non-stimulated saliva secretion (11) and is usually resected during Level I dissections; however, bilateral resections should be avoided to prevent dry-mouth (12). Before resection, it is retracted downwards to expose the underlying anatomic structures. The facial artery is a large calibrated vessel that localizes in the lateral part, and Wharton's duct is localized in the medial part. The Wharton duct is ligated to remove the gland. The lingual artery have a genu that touches the deep part of the gland and is preserved by dissecting off the gland.

Level II: Level II is divided into Levels IIA and IIB. Level IIB contains jugulodigastric nodes and is localized deep to the upper segment of the sternocleidomastoid (SCM) muscle, lateral to the internal jugular vein, and caudal to the mastoid bone. The lymphatic vessels from the facial region as well as mouth, tonsils and pharynx drain into jugulodigastric nodes (13-15), which

makes this region important. The spinal accessory nerve traverses obliquely from the caudal border of Level IIB (2) and should be protected. Jugulodigastric nodes can be peeled off from the underlying splenius capitis and levator scapulae muscles and can be passed deep to the spinal accessory nerve to have an en-block lymphatic specimen. This maneuver is called the Osvaldo Suarez Maneuver (16). Elevating the jugulodigastric nodes from Level IIB usually causes troublesome bleeding.

Level IIA is the inferomedial part of Level II above the transverse line passing from the level of the hyoid bone and caudal border of the spinal accessory nerve. The hypoglossal nerve passes through level IIA and passes deep to the posterior belly of digastricus to reach Level I (17). It is usually not encountered during Level II dissections; however, it should be protected to preserve tongue movements.

Level III: Out of ansa cervicalis, there are no specific anatomic structures to preserve in Level III except the carotid arteries and jugulary veins. The ansa cervicalis is a loop of upper and lower roots that emerge from C1, C2, and C3 cervical plexus roots (18). It is located on the anterior surface of the internal jugular vein and should be preserved to protect the strap muscles' function. Anterior jugular veins usually causes troublesome bleedings during the Level II and III dissections. These bleedings are significantly worse when the patient has a node-positive (N+) neck connected to enlarged anterior jugulary veins. They should be ligated to prevent postoperative hematoma, which can be life-threatening.

Level IV: Level IV is the deep and caudal region to the omohyoid muscle venter inferior. Important structures in this region include transverse cervical vessels and the thoracic duct on the left side. Transverse cervical vessels are the branches of the thyrocervical artery and vein (19, 20). The ductus thoracicus is the most distal part of the lymphatic system that comes from the cisterna chyli. After having a genu, it joins the left subclavian vein, and is a very important structure to be preserved in order to prevent chylous leaks and fistula (21). Accessory (right) lymphatic duct joins the right subclavian as well (2). After dissecting Level III nodes, the omohyoid muscle is retracted upwards with a strong retractor, and fatty tissue of Level IV is bluntly peeled off from the underlying pretracheal fascia with a peanut or with a finger that's wrapped with a gauze. Transverse cervical vessels usually form the dissection's lower boundary and should be protected. A feeding artery usually comes from the transverse cervical artery and feeds Level IV nodal tissues. This artery should be found

and ligated to prevent bleeding. One should suspect the damage to the ductus thoracicus if he/she sees a thin yellowish layer of liquid on the surface of the strap muscles that contain small droplets. It is always advised to explore and see the ductus to protect it: If it is seen, then it probably would be protected.

Level V: Level V is the region between the posterior border of the SCM muscle and the anterior border of the trapezius muscle (1). The clavicle forms the lower border of the triangle. Out of Level II, the spinal accessory nerve is also in this level. This usually runs obliquely from the posterior border of SCM muscle deep to the Erb's point (22). This nerve should be protected, again in Level V. Also, transverse cervical vessels are in Level V and should be protected. The most important structures of this level include roots and trunks of the brachial plexus. They come into view between the anterior and the middle scalene muscles. Roots form the trunks, and the trunks leave Level V by going deep to the clavicle. The subclavian artery also passes caudal to the inferior roots and the trunks of the brachial plexus. The brachial plexus is partly covered by the omohyoid venter inferior muscle. The phrenic nerve also exists in Level V and goes inferior on the anterior scalene muscle, and this nerve should be preserved to protect diaphragm function.

2. Axillary dissections

Skin incisions of the axillary dissections should be placed either on the anterior or posterior axillary line to prevent axillary contractures (23, 24) . Skin incisions should also be elongated on a natural skin crease for the same purpose.

The patient is positioned with the arm abducted 90 degrees on a sterile lateral table (24). The arm is draped and should be mobile, as shoulder flexion can be needed during the operation.

Skin flaps are elevated between the Camper's fascia and Scarpa's fascia. Camper's fascia is a thin layer of fascia and fat lobules (25). Fat lobules are small and larger on the superficial and deep to this fascia, respectively. As noted, skin flaps are elevated above the larger fat lobules. The axillary lymphatic tissue is the fatty tissue between the lateral border of the pectoralis major muscle, anterior border of the latissimus dorsi muscle and axillary vein. The deep border is the fascia of the serratus muscle. This tissue is Level I axillary lymph group, where level II is behind the pectoralis minor muscle, and Level III is medial to the pectoralis minor (24).

There are different techniques to remove axillary lymphatic tissue. In our clinical practice, borders of the lymphatic tissue are deepened without resecting

the lymphatic vessels that connect Level I to Level II. First, the anterior border is deepened using a monopolar cautery until the lateral border of the pectoralis major muscle is reached. Especially in women, there are lymphatic vessels which connect the breast to the axillary nodes. When being large caliber, these vessels should be ligated to decrease postoperative fluid accumulation and drainage levels. As they do not contain blood cells, and coagulation is impossible, resecting lymphatic vessels with monopolar cautery would not help close them. Depending on this, ligating the ones with larger calibers should be kept in mind. After retracting the pectoralis major muscle medially, the pectoralis minor muscle is exposed. The superficial surface of the pectoralis minor muscle is traced up to the level of its tendinous segment. The upper segment of the pectoralis minor muscle is located superficial to the axillary vein (26), and this relationship is used to find the axillary vein. After that, the posterior border of the specimen is deepened to reach the lateral border of the latissimus dorsi muscle. Again, large caliber lymphatic vessels that come from the back of the patient should be ligated. The lateral border incision is traced up to the level of the axillary vein. As opposed to the pectoralis minor muscle, the axillary vein passes superficial to the latissimus dorsi and tracing the latissimus dorsi muscle in the upward direction should be done cautiously. The anterior and posterior borders are connected on the level of the axillary vein, and this upper border of the incision exposes the axillary vein. After all of the borders have been identified, the lateral rim of the latissimus dorsi is retracted posterior to expose the undersurface of the muscle.

The hilus of the muscle is the point where the thoracodorsal neurovascular bundle enters the muscle fascia and is usually located approximately 4 centimeters from the distal scapular border (27). The hilus is explored, and the thoracodorsal bundle is found. Another way to find these vessels is to trace the serratus branch of the thoracodorsal artery once it is encountered. After the thoracodorsal bundle is exposed, the thoracodorsal vessels and thoracodorsal nerve are dissected up the apex of the axilla and freed from the surrounding fatty lymphatic tissue. Then, the specimen is peeled off from the underlying fascia of the serratus muscle. Another important anatomic structure to prevent is the long thoracic nerve (28). It emerges from the roots of the brachial plexus (29), and this early emergence makes it close to the thorax wall. It goes down on the lateral surface of the thoracic wall under the fascia. It is not covered by fatty lymphatic tissue. It should be protected while peeling lymphatic tissue off the serratus fascia. Damage to the nerve results in the winging of the scapula (28).

After all lymphatic tissue is peeled off the underlying fascia, it is still attached to the structures surrounding the axillary vein. Many lymphatic vessels usually come from the upper extremity around the axillary vein. These lymphatic vessels should be ligated as well. According to the oncologic needs, the sheath of the axillary vein can be dissected off.

After all these steps have been performed, the specimen is still attached to Level II nodes with the large caliber lymphatic vessels travelling under the pectoralis minor muscle. These vessels should be ligated before the removal of Level I nodes. There can be numerous lymphatic vessels in this connection, and they can be ligated all together with a number 0 or 2/0 silk suture. When Level II nodes need to be removed, the arm is abducted from the shoulder to mobilize the pectoralis minor muscle off the chest wall (24). After that, Level II nodes can be removed en-bloc with Level I nodes (Figure 2).

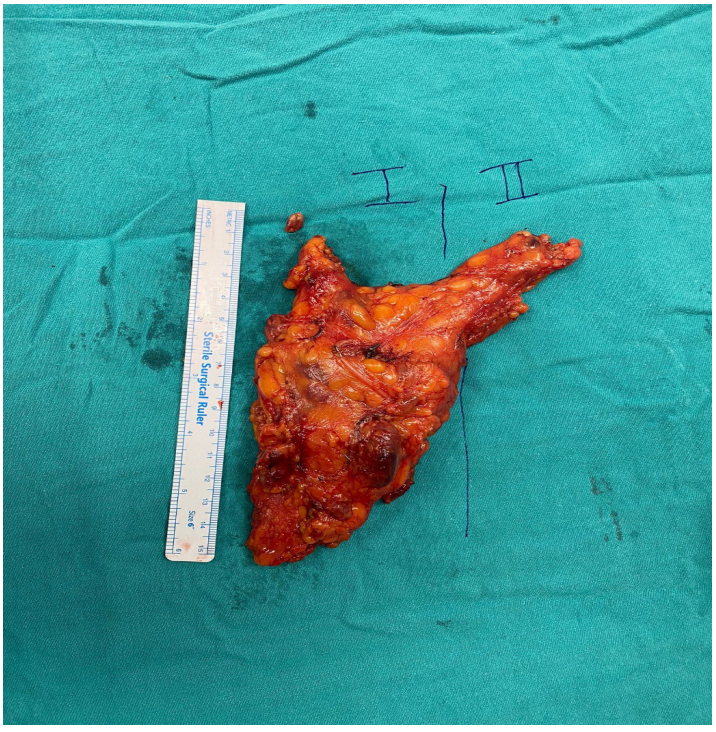

Figure 2. Axillary Levels I and II resected en-bloc.

3. Inguinal Dissections

Inguinal lymph nodes are localized in triangular fatty tissue in the Scarpa's triangle, namely between the medial border of the sartorius muscle and the

lateral border of the adductor longus muscle (30). The inguinal ligament is the upper border of the triangular base.

The patient is positioned in a frog leg position (31). The skin incision is positioned in a lazy S fashion. The direction of the lazy S incision is important in this operation. By the author, lazy S is positioned in a vertical direction, and the open mouth of the parabola looks lateral in the upper half to leave the medial skin flap shorter in the transvers direction. This area is susceptible to skin necrosis. Decreasing its transverse length helps to prevent skin necrosis. Including a skin island in the specimen may help to prevent skin necrosis. Skin flaps are elevated, similar to the technique that was outlined in the axillary dissection part. The great saphenous vein is usually encountered during the elevation of the medial flap at the surface or near the ground.

After elevation of the flaps, specimen's lateral border is deepened until the lateral border of the sartorius is reached. Then the specimen is peeled off from the underlying fascia latae and the deep muscle fascia. The femoral nerve is encountered first at the lateral edge of the bundle. It usually looks thin to the unfamiliar eyes. The sheath of the femoral vein is incised then. The fatty tissue around the femoral vein can be included in the specimen according to the oncologic needs. There is a strong septum between the femoral artery and vein. It is commonly transected in an inattentive way; however, this septum may contain thin arterial branches of the femoral artery that go medially, and these small arteries might be unnoticed depending on the pulling of the specimen.

In the upper half, fascia latae becomes thin, and this circular area is called fossa ovalis and it is covered by cribriform fascia (30). In the fossa ovalis, the saphenous vein joins the femoral vein on the medial side. It is ligated with great attention to include the saphenous vein in the specimen. In this region, deep femoral vessels, superficial circumflex iliac vessels, superficial epigastric vessels, and superficial external pudendal vessels are encountered and dissected.

This lymphatic tissue located in the femoral triangle is Level I nodes. Lymph from this nodes drain to subinguinal lymph nodes in the femoral triangle, and then to the lymph nodes that are located near the external iliac vessels in the preperitoneal region above the inguinal ligament (30). The lymphatic vessels connecting the subinguinal and scarpa's nodes to the external iliac chain nodes pass the undersurface of the inguinal ligament medial to the common femoral vein through the femoral canal (30). Level II nodes can be removed according to the oncologic needs by tracing these lymphatic connecting vessels through the femoral canal and ring.

Transposing the Sartorius muscle flap under the incision line before the skin closure supplies a strong support for the underlying vascular structures in case of incision dehiscence.

4. Popliteal Dissections

A skin incision of the popliteal dissection can be performed in many different styles. Figure 3A shows the preference of our institution. After the skin flaps have been elevated, popliteal fascia is exposed. Popliteal fascia is a strong fascia that is continuous with fascia latae. It has dense transverse fibers (Figure 3B).

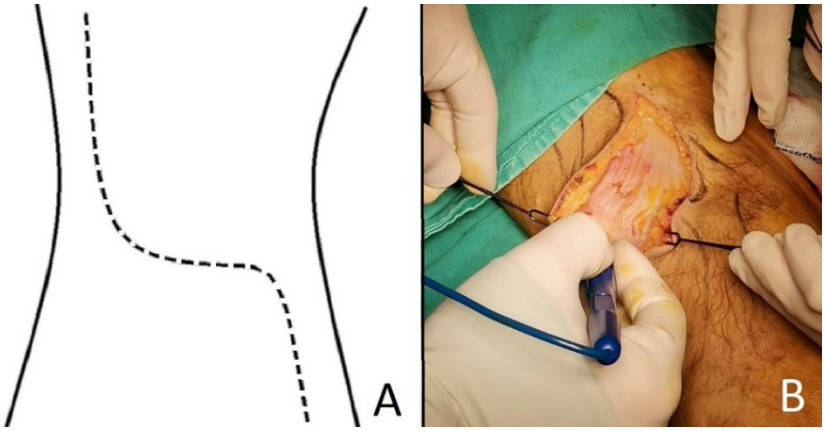

Figure 3. A: sample incision for popliteal dissection,
B: popliteal fascia with transverse fibers.

The popliteal fossa is a diamond-shaped region on the knee joint's posterior surface. Bounders of the popliteal lymphatic tissue are the medial belly of the gastrocnemius muscle on the inferomedial edge, the lateral belly of the gastrocnemius muscle and the plantaris muscle on the inferolateral edge, tendons and muscle bellies of the semimembranosus and semitendinosus on the superomedial edge, and muscle bellies of the heads of biceps femoris muscle on the superolateral edge (32).

Popliteal vessels and the tibial nerve traverse the fossa from the proximal to the distal direction, where the artery is in the medial, the vein is in the middle, and the tibial nerve is in the lateral. However, the tibialis nerve spirals around the popliteal vein before it enters the hiatus of the soleus muscle and passes to the medial side of the vein.

Out of these structures, the common peroneal nerve traverses the popliteal fossa on the lateral side. It comes from the paramedian line and goes inferolateral

(Figure 4A). In the upper half of the fossa, it is parallel to the biceps muscles and tendons, and in the lower half of the fossa, it is located superficial to both the plantaris muscle belly and the lateral head belly of the gastrocnemius (Figure 4B). The nerve is located deep in the popliteal fascia; however, it can be close to the undersurface of the fascia and should be preserved to protect from foot drop.

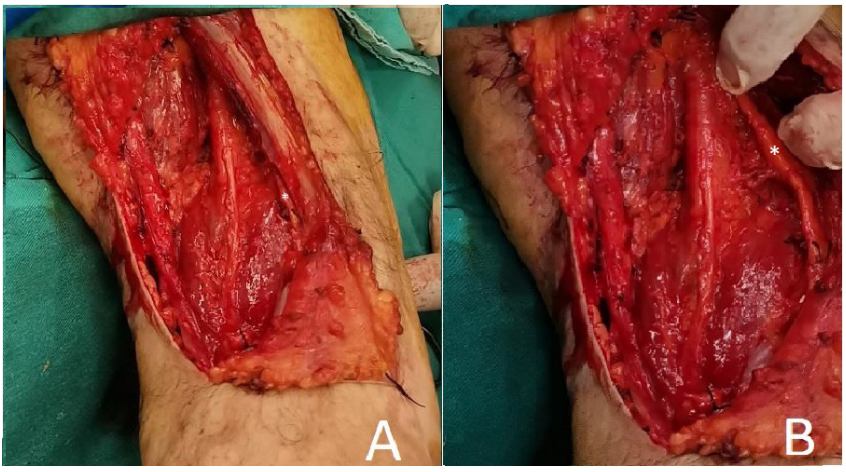

Figure 4. A: Tibial nerve (medial) and common peroneal nerve (lateral), B: Common peroneal nerve on the lateral side after short and long heads of the biceps femoris muscle is retracted laterally (*: indicates common peroneal nerve).

5. Superficial Parotidectomy

Superficial parotidectomy is performed in oncologic surgery when preauricular lymph nodes drain the tumor. The superficial lobe of the parotid gland contain preauricular lymph nodes and usually drains the region, which is located above a line that connects the angle of the mandible to the middle of the lower eyelid and the medialmost point of the upper eyelid (33). The upper border of the region is the bicoronal line.

Peripheral branches of the facial nerve pass between the deep and superficial lobes of the parotid gland. The branches may easily get harmed during the dissection of the gland. Depending on this, the surgery should be applied meticulously.

The most common two techniques for superficial parotidectomy include anterograde and retrograde approaches (34).

In the anterograde approach, the main trunk of the facial nerve is found around its exit from the cranial base, followed distally, and the superficial lobe is removed afterward. The posterior aspect of the parotid gland has strong connections to the cartilage of the external auditory canal and the upper quarter of the SCM muscle. These connections can be dissected sharply to mobilize the posterior edge of the gland. After that, the posterior belly of the digastric muscle comes into view. The posterior belly of the digastric muscle also inserts on the mastoid bone as SCM does. However, the digastric muscle is anterior and deep to the SCM muscle. The main trunk of the facial nerve comes to the surface anterior to the digastricus muscle. As mentioned above, after the gland is mobilized from the cartilage of the external auditory canal and SCM muscle, the posterior belly of the digastricus muscle is exposed and followed up. The tragal pointer comes into view in the deep portion of the external auditory canal. The anterior of the digastric muscle is bluntly and precisely dissected to find the main trunk of the nerve. The main trunk is positioned 1 centimeters deep and anteroinferior to the tragal pointer (35). Small bleedings can be accepted as an indicator of proximity to the nerve. These bleedings should not be cauterized recklessly to prevent damage to the main trunk. After the main trunk is identified, it is followed distally to remove the superficial lobe of the parotid gland. One very important trick here is identifying the bifurcation of the main trunk since it may have bifurcated proximal than usual. If this was not performed, the identified nerve would mistakenly be thought to be the main trunk, whereas it is either the cervicofacial or temporofacial branch. In this poor scenario, the other main branch can get damaged during the "so-called" safe removal of the superficial lobe.

Retrograde removal of the superficial lobe includes identifying the peripheral branches of the facial nerve, and removing the gland from the distal to the proximal direction. The parotid gland sits on the posterior aspect of the masseter muscle. Peripheral branches of the facial nerve leave the anterior edge of the gland and travel medially on the surface of the anterior aspect of the masseter muscle belly. These two important anatomic structures, the masseter muscle and the parotid gland, are covered together with a deep fascia called parotidomasseteric fascia, which is located deep to superficial musculoaponeurotic system (SMAS).

In the retrograde approach for superficial parotidectomy, subcutaneous fatty tissue and SMAS is transected vertically to expose the parotidomasseteric fascia. The skin flap is elevated medially under the SMAS layer or under the

parotidomasseteric fascia. If the flap was elevated under the parotidomasseteric fascia, the peripheral branches of the facial nerve would be seen uncovered after the edge of the parotid, where one should perform the dissection precisely. If the flap was elevated above the parotidomasseteric fascia, then the peripheral branches of the fascial nerve would be seen covered by the fascia where they are on the masseter muscle. Either way, after one of the peripheral branches is identified, the other branches are found in the same surgical plane, and the superficial gland is removed from the distal to the proximal direction (Figure 5**).**

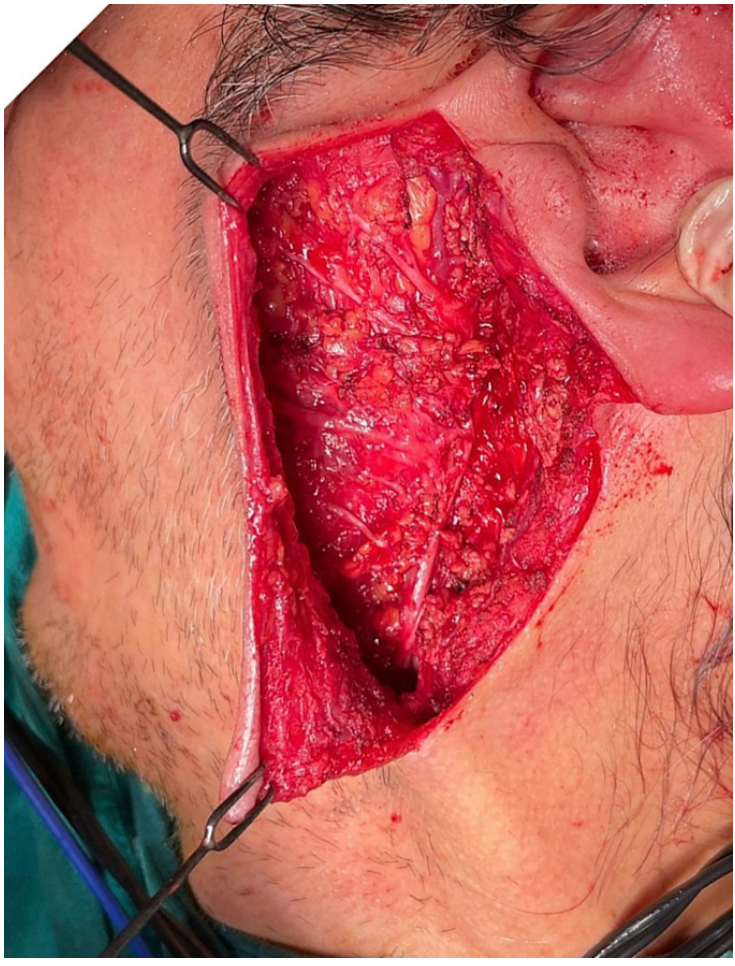

Figure 5. peripheral branches of the facial nerve after superficial parotidect**omy.**

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CHAPTER IX

BREAST REDUCTION COMPLICATIONS

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1. Introduction

Breast reduction is a common surgical procedure in today's plastic surgery practice around the world. Macromastia is defined as the fact that the breast is much larger, and drooping compared to the body size in adult age. Macromastia is mentioned when the weight of each breast is 1000-2000 g and above. Large breasts seen in adolescence are defined as virginal hypertrophy or gigantomasti. Breasts larger than normal sizes cause physiological and psychological discomfort in women. The reason for these ailments is that large breasts cause severe back, neck, and shoulder pain by disrupting the body posture, rather than cosmetic conditions. Also, larger breasts cause hygienic problems, dermatitis under the breast, and fungal infections. More rarely, complications such as numbness and pain in the hands and fingers can be seen due to the compression on the nerves in the areas innervated by the nervus ulnaris in brachial plexus and cervical arthritis. It is not possible to reduce hypertrophic breasts with any medication or hormone therapy, and the only treatment option is surgical treatment. Although there are many different techniques to perform breast reduction surgery, the inferior pedicle technique and free nipple technique are generally applied in gigantomasti. Other techniques for breast reduction are superior pedicle, lateral pedicle, medial pedicle and, superomedial pedicle reduction mammaplasty. All these techniques have advantages as well as some complications (1,2,3). Therefore, surgeons should be aware of this issue and diagnose before it causes seriosus problem for the patient.

2. History

There is no ideal and single method in breast reduction surgery as with every procedure. Therefore, a wide variety of surgical techniques have been described in the past, and they all have limitations. Paulus Aeginata described reduction mammoplasty in gynecomastia correction operations in the early 6th century. Diefenbach was probably the first to perform reduction mammoplasty by removing the lower two-thirds and posterior segments of both breasts in 1848. Many of the operations performed in the late 1800s and early 1900s were applied to correct sagging breasts. In these operations, many types of skin and gland excision methods have been used to attach the remaining breast higher on the chest wall, regardless of the position of the nipple area. Between 1909 and 1925, more attention was paid to the position of the nipple area. Morestin was the first to perform the idea of changing the nipple position in 1909, followed by Villandre in 1911, Lexer in 1912, and Thorek in 1921. After these improvements in breast reduction operations, further techniques are focused on providing blood circulation to the skin, protecting breast tissue and nipple area. In the technique published by Aubert in 1923, they did not excise the skin and glands attached to the subdermal plexus to prevent vascular complications. In 1930, Schwarzman drew attention to the preservation of the nipple areolar complex (NAC) by leaving the periareolar skin ring with its arterial and venous connections; increased the viability of the NAC and paved the way for new techniques with flaps in which the NAC localization was changed. Following techniques in breast reduction surgery focused on skin incisions and pedicle designs to create a more aesthetic appearance and preserve vascularity. In 1948, Bames emphasized the importance of marking the planned incision and resection sites before the operation. Aufricht explained the importance of skin support in breast form postoperatively (4-6). Robert Wise designed a skin resection pattern adapted from the bra design in 1956, and significant progress has been made in recent history. This pattern has come to be known as the inverted T. Significant advances in pedicle techniques were made by Pitanguy, who introduced the superior pedicle in the 1960s. McKissock defined vertical bipedicle in 1972, Robbins, Courtiss and Goldwyn introduced the inferior pedicle in 1977. These pedicles were adapted to the Wise skin resection model and taught to plastic surgery residents around the world. Superior pedicle

was popularized by Lassus, Marchac, de Olarte, and Lejour et al. The focus was on eliminating the horizontal scar length using the superior pedicle. This approach also improved breast shape by reducing the boxy appearance that sometimes occurs with inverted T. The vertical skin incision model is adapted to the inferior, medial, superomedial, and lateral pedicles. When vacuum lipectomy became popular in the 1980s, this volume reduction method was adapted to the breast to avoid direct parenchymal resection approaches. Thus, it caused the nipple to be slightly elevated and the skin tightened by significantly reducing blood flow and sensory innervation (7-10).

3. Neurovascular Anatomy of the Breast

Embryologically, the breast develops as the fourth intercostal space structure and there are arterial and vena comitants that arise between the fourth and fifth ribs, usually originating from the internal mammary vascular system. The superficial circulation comes mainly from the superficial branch of the lateral thoracic system and the thoracoacromial system and the internal mammary artery. As the breast develops during puberty, the superficial skin and subcutaneous tissue through which the vessels pass are pushed out. These vessels move towards the nipple and surround the breast. The superior pedicle is supplied by the perforator artery from the internal mammary system, usually coming from the second intercostal space. The medial pedicle is supplied by a perforator that comes from the medial aspect of the breast, usually from the third intercostal space. The lateral pedicle is supplied by the superficial branch of the lateral thoracic artery. The inferior and central pedicles are supplied by perforators from the fourth intercostal space. Perforators from the fifth intercostal space arise around the level of the inframammary fold and provide extra blood supply to the inferior pedicle. The venous system is often visible through the skin and drains mainly superomedially. The main nerve supply to the nipple and breast skin is the anterolateral branch of the fourth intercostal nerve, which gives a deep branch over the pectoralis fascia and a superficial branch to the subcutaneous tissue. The deep branch runs along the mammary meridian towards the nipple, which can be protected by medial and inferior pedicles (Fig. 1). However, this is not the only innervation of NAC. There are also anteromedial branches from the third to fifth intercostal nerves (10-13).

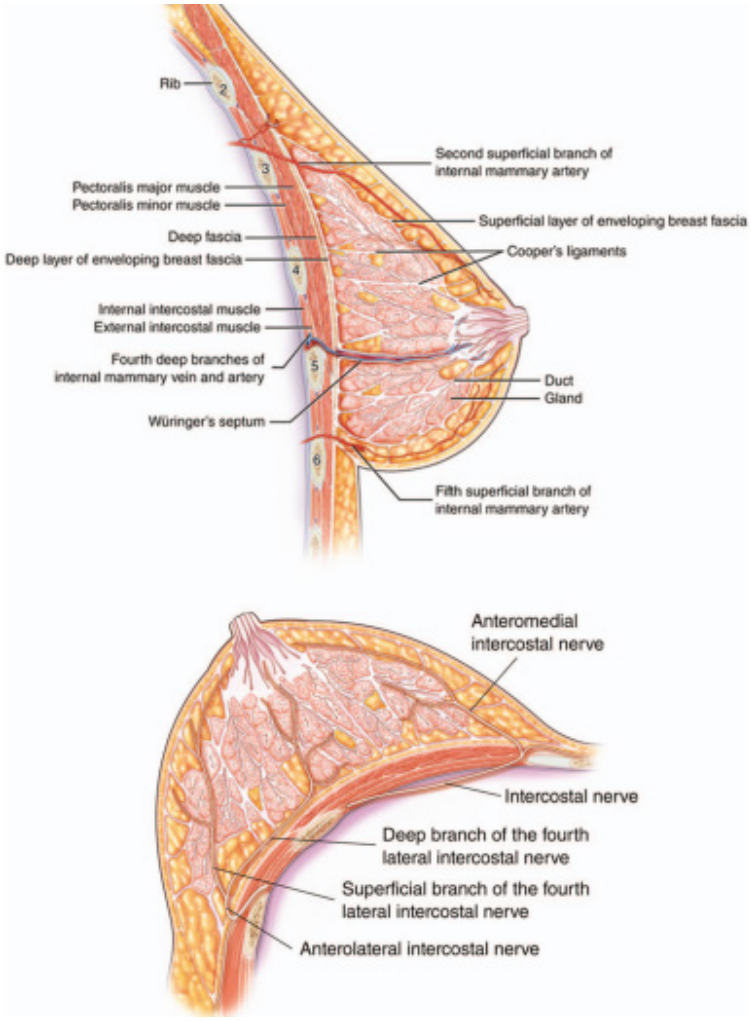

Figure 1: (Above) The blood supply to the breast consists of a deep artery and vein running from the fourth intercostal space to the parenchyma. The rest of the blood flow (mainly from the internal mammary system) comes from around the breast and then extends subcutaneously to the nipple. The vessels go deep into the dermis and most of them run superomedially. (Below) The innervation of the nipple is mainly from the anterior branch of the fourth lateral intercostal nerve. Except for the lateral pedicle, there is a superficial branch that will be completely damaged. The deep branch running over the pectoralis muscle can be protected in medial and inferior pedicles. There are many other nerves come from the second, third and fourth intercostal space that also provide the innervation of the nipple (10).

4. Breast Reduction Techniques

There are two main issues to be considered in breast reduction surgery. These are the skin removal pattern and the type of pedicl***e.***

4.1. Pedicle Techniques

Horizontal Bipedicle Technique; It is a method applied by excising the breast parenchyma under the pedicle on both sides. It provides the breast to be supplied by two dermal pedicles. This is sufficient for arterial and venous system, areola, and nipple nutrition (14,15).

Vertical Bipedicle Technique; In this technique defined by McKissock; The superior flap tissue is separate from the underlying breast parenchyma. The NAC is left with only the dermis. It is advantageous in vascular nutrition as the NAC is protected (15,16).

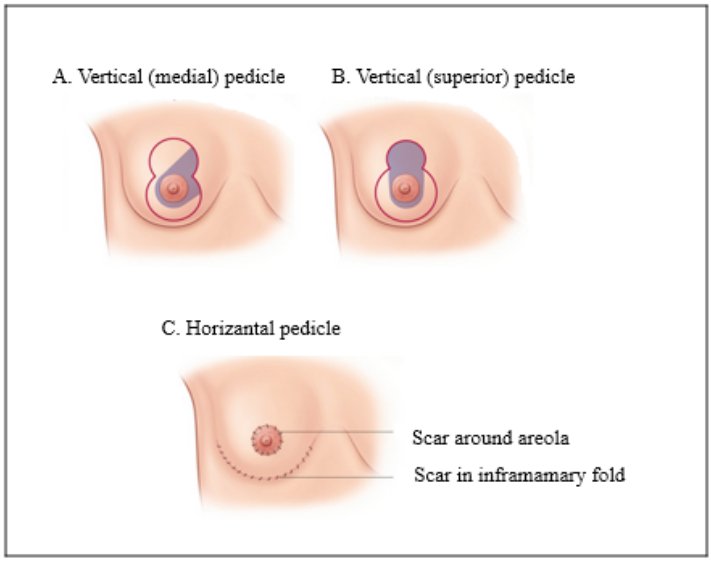

Figure 2: Pedicle designs. B, Vertical with medial pedicle. C, Vertical with superior pedicle. G, Horizontal pedicle (15).

Inferior pedicle technique is the technique in which the inferior dermal structure, NAC is preserved. In this technique, NAC is transposed over an inferior dermoglandular flap. It is a very advantageous technique as it protects the arterial and venous system of the NAC. At the same time, it is important in breastfeeding and maintaining nipple sensation (15,17).

Superior pedicle technique; The superior pedicle has a reliable artery comes from the internal mammary system at the level of the second or third intercostal space. Here, the superior dermoglandular structure is preserved. Therefore, although pedicle nutrition is good, it is difficult to shape and needs to be thinned. Since it is a dermal pedicle, the mammary glands are excised (15,18).

The central pedicle technique is like the inferior pedicle technique, but the dermis connecting the lower part is separated. It is supplied by perforators of the intercostal arteries. There is no need for a dermal bridge because there are sufficient arterial and venous networks (15,19).

The lateral pedicle technique can be considered as a technique in which half of the Strombeck method is preserved. The lateral pedicle is supplied via the superficial thoracic artery (a branch of the lateral thoracic artery extending just to the margin of the pectoralis muscle), (15,20).

The medial pedicle technique is one of the most frequently used techniques in general. The vascular supply of the medial pedicle comes from the medial branches of the internal mammary system (possibly from the third and fourth intercostal spaces). It is a pedicled technique in which the medial dermoglandular structure is preserved, the nutrition of the NAC is good and nipple sensation is preserved (15,21).

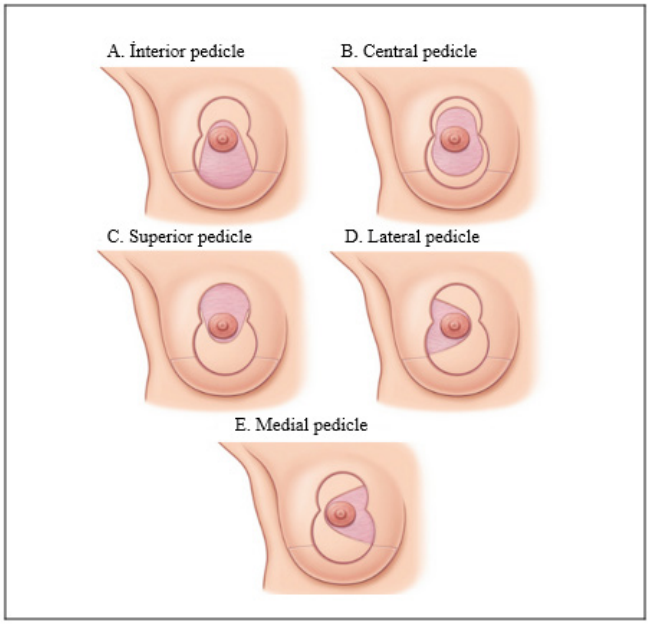

Figure 3: Pedicle designs for nipple-areola complex. A, Inferior. B, Central. C, Superior. D, Lateral. E, Medial. https://plasticsurgerykey.com/breast-reduction/

4.2. Types of Skin Excision in Breast Reduction

Inverted T resection (wise pattern) is used especially in large breasts and especially in cases where skin elasticity is weak (e.g., in patients with excessive weight loss), and in patients with excess skin. Although it is generally used in the inferior pedicle, it also gives good results in other pedicled techniques (15,21).

Vertical resection is the second most common skin resection pattern. The most common pedicles are superior and medial pedicles. After removing the excess skin and parenchyma in the vertical line, it is used to shape the breast by using the breast parenchyma in the pillars. The vertical technique is safe, easy to apply, and has limited complications. Except for the excision areas of the breast, the skin does not need to be separated from the subcutaneous tissue. These both reduces the possibility of circulatory problems and accelerates recovery.

Lateral resection; excision of excess skin and subcutaneous tissue is performed only from the pillar, areola and lateral part instead of leaving a reverse T scar. This process is applied in order not to leave a scar on the medial part.

Circumareolar resection; Excess skin tissue around the areola is excised. It is suitable for minor modifications and permanent stitching is required.

Vertical non-scarring technique; It is an inferior pedicle technique. But upper pole lacks fullness and becomes flat. Scar tissue also tends to pull the breast down.

Liposuction method; This method is not suitable for large breasts, but only for breasts with high fat content. It is a surgery that depends on the elasticity of the breast skin. It is a technique that preserves the sensation of the NAC (2,10,15,21,22).

5. Complications

Although breast reduction surgeries are important in terms of eliminating physiological problems (such as neck and back pain, intertrigo, coracoid compression syndrome, submammary dermatitis and fungal formation) rather than psychological and aesthetic concerns, some complications can be seen after the operations. In some cases, it can be difficult to prevent these complications. It is very important to diagnose complications to initiate appropriate treatment. The surgeon should know how to avoid complications and how to treat these complications in a timely manner.

5.1. Calcification

Yalın et al. reported that calcifications after breast reduction have mammographic and ultrasonographic findings. They stated that masses with coarse and thick spiculations, irregular borders, central radiolucency, amorphous and pleomorphic, dystrophic, coarse, and branching microcalcifications were seen. They also observed the presence of fat cyst calcifications like eggshells. While fat necrosis and fat cysts are associated with any surgical procedure in the breast, Brown and Miller have reported parenchymal redistribution, asymmetry, scarring, parenchymal or retro areolar linear bands and calcifications. Mendelson drew attention to the thickening of the skin around the incision sites in the periareolar area of the lower pole of the breast and in the inframammary fold. Also, the presence of needle-like calcifications has been reported. Mitnick et al. reported that calcifications were found in the breast skin, especially in the periareolar region, after breast reduction. **(referans)** Heywang-Kobrunner reported fat necrosis and calcifications of fat cysts with round, ring, or eggshell-like wall calcifications. **(referans)**

5.2. Early Wound Complications and Delayed Wound Healing

Wound healing, open wounds and skin loss are the most common complications in patients undergoing breast reduction surgery. Smoking, avoidance of steroid therapy, and control of diabetes or connective tissue diseases are vital in wound complications. In addition, technical considerations should be considered to avoid skin ischemia, which causes skin loss and delayed healing. Sufficient tissue should be removed from the pedicle so that the wounds are approximated without excessive tension. The incidence of these complications increases with the weight of the breast resection. Although skin loss is rare, it is among the complications that can be seen. Especially in surgeries where very large breasts are reduced, this risk increases. The harmful effects of smoking after breast reduction have been investigated and confirmed in the literature in both prospective and retrospective studies.

5.3. Hematoma and Seroma

The rate of major hematoma in breast reduction is approximately 1-2 percent. **(referans)** It is important to keep any pre-existing hypertension under control in preventing hematoma. Also, while providing hemostasis, it is necessary to provide normotensive conditions and to ensure optimal awakening from anesthesia without sudden increases in blood pressure. When a hematoma

occurs, incision and drainage should be made to protect the tissue necrosis caused by the hematoma and the risk of emerging infectious complications. An aspiration catheter or penrose must be placed for at least 24 hours. Seromas usually appear later and can be aspirated. Seromas are less likely to resolve spontaneously with an inverted T skin resection pattern compared to a vertical pattern because submammary scarring can block drainage.

5.4. Nipple Areolar Complex ischemia

NAC ischemia is one of the most feared and devastating complications of breast reduction. Although partial or complete nipple necrosis is a well-defined complication of breast reduction, it can be devastating for the patient and surgeon. It occurs due to arterial insufficiency after resection of the breast parenchyma that impairs NAC perfusion. It is not always possible to diagnose nipple ischemia, especially in black or dark-skinned patients. Pale or dark nipples in light-skinned patients suggest circulatory problems. In large-sized breast reduction surgery, the pedicle can be folded, and its compression can cause a decrease in circulation. Surgery may be performed if any problem is suspected. Sometimes just removing the stitches restores circulation.

5.5. Fat necrosis

There may be loss of vascular supply in fat-dense areas along the incision sites. This may result in postoperative drainage of necrotic fat. An untreated area of fat necrosis can cause cyst and calcification. Fat necrosis may also occur at the distal end of the pedicle due to lack of blood supply.

5.6. Infection

Some undesirable results may develop after any surgical intervention. Swelling that can be seen within a few days after the operation may be a sign of infection; In this case, the infected necrotic tissues should be debrided, and antibiotic treatment should be applied.

5.7. Long Term Complication

The most common long-term complications of breast reduction procedure are scar formation such as hypertrophic scar and keloid, loss of breast fullness, loss of shape, nipple misposition and asymmetry. Also, the common problem of almost all breast reduction techniques is that the lower pole of the parenchymal tissue comes down.

5.8. asymmetries

All people's bodies are asymmetrical. With liposuction, these asymmetries can be corrected; It can help treatment in reducing the volume of an operated breast. Also, skin envelope symmetry, epithelialization and re-approximation and optimization of the skin edge can be achieved to correct the breast shape, and the skin envelope can be tightened (10,23-27)

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CHAPTER X

CATARACT SURGERY COMPLICATIONS AND MANAGEMENT

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Cataract surgery is the most common surgical procedure performed by eye surgeons. The outcomes of cataract surgery have greatly improved with a reduced risk of complications since the transition from extracapsular extraction to phacoemulsification.

Although developments in cataract surgery technology prevent serious complications, each new surgical technique brings its complications. Complications of cataract surgery may occur in the early or late intraoperative or postoperative period (1, 2). The intraoperative and postoperative complications of cataract surgery and the available management options are discussed in this section. Our clinical practice, discussions with colleagues, data presented at international conferences, and literature from the PubMed database were made use of while preparing this article.

Preoperative Conditions

One of the most important steps to prevent possible complications in cataract surgery is to identify and inform the cases considered to be at higher risk, and to plan if any changes in the surgical technique or additional equipment are required for the cases considered to be risky. For example, a patient with pseudoexfoliation syndrome and associated phacodonesis may require the placement of a capsular tension ring during surgery. Such steps are important to avoid over-anxiety and

frustration for both surgeon and patient must any complications occur, and to ensure optimal management during surgery. Intraocular pressure values of all patients must also be measured and recorded, and IOP must be lowered first before surgery. The presence of systemic disease (e.g. Diabetes, Hypertension, Prostate, etc.) must be questioned. Again, the use of anticoagulant-like drugs must be questioned, and if necessary, these drugs must be discontinued before the surgery. Anamnesis and biomicroscopic examination are extremely important as well as questioning the age of the patient, how long the cataract has been present and evaluating its stiffness. In this way, the patient can be provided with the best possible surgical outcome.

Intraoperative Complications

Posterior Capsule Rupture

Posterior Capsule Rupture (PCR) is still the most common intraoperative complication with a prevalence of 0.5%-5.2% (1, 3). PCR is a serious complication potentially complicating surgery because of loss of all or part of the lens piece, vitreous prolapse or traction, retinal decollement, endophthalmitis, high intraocular pressure, intraocular inflammation or bleeding, corneal edema, cystoid macular edema, and difficulties in inserting the intraocular artificial lens because of loss of capsular support. PCR increases the risk of endophthalmitis by 6 times (4) and the probability of retinal decollement by approximately 15 to 18 times (1, 5). Jaycock et al. conducted a study and reported posterior capsule rupture as 1.92 percent (6). According to this study, the factors increasing the risk of PCR were reported as increasing age (2.37%), male gender (1.28%), glaucoma 1.3%, diabetic retinopathy 1.63%, presence of dense cataracts (2.99%), pseudoexfoliation/phacodonesis 2.92%, presence of vitreous opacities (2.46%), the narrow pupil (1.45%), the axial length greater than 26.0 mm (1.47%), the use of doxazosin as a systemic drug (1.51%), the inability to lie flat on the back (1.27%), and the inexperience of the operating surgeon (3.73%) (6).

Posterior Capsule Ruptures are usually caused by the contact of the surgical instrument with the posterior capsule and can occur at any stage of the procedure. Capsular block that stems from excessively intense hydrodissection can cause capsular rupture even before phacoemulsification. Other common causes of capsule rupture occur when the phacoemulsification probe directly contacts the posterior capsule and aspirates the posterior capsule during phacoemulsification of the lens, or the irrigation/aspiration of the probe or other instruments used

in surgery during cortex cleaning. Also, the anterior capsule may rupture when the surgical instruments touch in the capsulorhexis stage, and this rupture may extend posteriorly and cause posterior capsule rupture. Less common causes of Posterior Capsular Rupture include trauma during intraocular lens insertion and puncture through loose cannulas during hydration of wounds or intracameral injections at the end of surgery. There is almost no surgical stage in which posterior capsular rupture might not occur.

It is already known that some cataract types carry a higher risk for intraoperative PCR because of the presence of posterior capsule defects. Congenital posterior polar cataracts have a high incidence of a natural capsular defect in the region of lens opacity. After the patient is informed about the condition of the cataract, hydrodelineation must be performed in such cataracts by injecting fluid between the epinucleus and the nucleus, instead of hydrodissection, which is performed by giving fluid between the cortex and the nucleus to remove the lens from the capsule during surgery. In patients with a previous vitrectomy, cataracts may occur when the vitrectomy probe touches the lens during vitrectomy. The probability of posterior capsular defect is higher in such cases. Also, PCR is more likely to be observed during phacoemulsification because of loss of stabilization in the absence of vitreous in these cases.

The challenges faced by the surgeon following PCR vary depending on the stage at which it is faced. Rupture, which usually occurs in the early stages of the surgery, can be much more problematic than rupture occurring after the lens and cortex have been aspirated. In an early rupture, it may be necessary to enlarge the corneal main incision to remove the lens, which can bring new challenges. Although removing the lens from the incision by widening the main incision may prevent nuclear fragments from falling into the vitreous cavity in whole or in part in the early stage following PCR, which will require later posterior vitrectomy, it may not be possible to prevent the lens fragments from falling and posterior vitrectomy may be required in some cases. Despite the psychological trauma of a second surgery for both patient and surgeon, it was reported that 56 percent of patients had better than 20/40 vision in the postoperative period (6). If posterior vitrectomy is required, the timing of vitrectomy and lensectomy must generally be within three weeks of the surgery. This is the time needed for corneal edema and intraocular pressure to recover and the risk of intraocular infection to disappear significantly. Also, early intraoperative vitrectomy may further improve visual outcomes by reducing the risk of glaucoma, cystoid macular edema, and retinal decollement (7).

If adequate anterior vitrectomy is not performed after PCR, the existing vitreous in the anterior chamber may cause the intraocular lens to not be easily centralized, postoperative pupil deformity, presence of vitreous throughout the surgical wound increasing the risk of endophthalmitis, chronic uveitis, and risk of retinal rupture and decollement that may develop following the surgery and cystoid macular edema. It may be necessary to inject triamcinolone into the anterior chamber for visualization of the vitreous and easy aspiration by the vitrectomy (8).

The decision for Intraocular Lens (IOL) placement depends on the size of the posterior capsular defect. If a small portion of the posterior capsule has ruptured, the lens can still be carefully inserted into the capsule after the surgeon is satisfied that manipulation will not enlarge the rupture. It will be safer if this posterior capsulorhexis is done (9). In case of doubt and larger defects, the IOL must be placed in the sulcus (lens haptics placed between the anterior capsule and the iris) if the anterior capsule is intact and there is sulcus support. In such a condition, although there is no optimal IOL for sulcus placement, currently available three-piece foldable lenses are used (10).

In the postoperative period, the peripheral retina of the patient must be carefully checked for ruptures or ruptures that may have been caused by intraoperative vitreous traction. When managed properly, the visual outcome of patients who experience posterior capsule rupture is still very good, and more than 90 percent of patients have an improvement in vision when compared to preoperative levels (11).

Zonular Separation

In previous studies, the percentage of zonular separation was reported as 0.46 (6). To evaluate the stability of the lens and the presence of weak zonulas, it is necessary to take a careful anamnesis before the surgery and to perform a detailed lens examination by using biomicroscopy. Ruptured zonulas most commonly occur in the case of ocular trauma or pseudoexfoliation syndrome. In Marfan Syndrome, although the outcome is the same, the zonulas usually stretch rather than rupture. In such a condition, it is helpful to investigate the presence of vitreous in the anterior chamber and to inform the patient appropriately of the risks associated with the surgery.

Capsulorhexis may be more difficult in patients who have zonular separation. It may be necessary to use iris hooks or a capsule tension ring to stretch the anterior capsule depending on the degree of instability of the anterior

capsule (12). These rings provide support by distributing the tension on the zonulas and may work in cases of zonular separation of less than 180°. It may be necessary to use a stitched Cionni Ring in case of separation greater than 180 degrees. This ring is more difficult to place, but excellent surgical results can be achieved. The Cionni Ring is similar to the modified capsule tension ring and has 2 holes through which the suture can pass to fix the ring to the sclera (13). If such a ring or segment is not present, the IOL may not be centralized and an anterior chamber lens may need to be placed, which can be fixed by attaching the iris anteriorly or posteriorly to the iris.

Zonular separation may not be noticed preoperatively in most cases or may have occurred as a result of surgical trauma. Such cases are the result of pseudoexfoliation syndrome, trauma, or natural zonular weakness in very elderly patients. Intraoperative zonular separation may result from excessive traction on the zonulas, such as during unintentional direct vacuuming/aspiration of the peripheral capsule from the phacoemulsification probe or irrigation/aspiration probes. In such cases, management is the same as described earlier depending on the extent of the separation and the stage at which it occurs. A capsule tension ring is contraindicated if the posterior capsule is not intact. Any vitreous protruding into the anterior chamber must of course be recognized and removed with anterior vitrectomy. After the stabilization of the capsule and the removal of any vitreous, the IOL can usually be inserted safely into the capsule.

Suprachoroidal Hemorrhage

Suprachoroidal hemorrhage has an incidence of approximately 0.04% in small-incision cataract surgery (14). It usually occurs with sudden rupture of vessels in the suprachoroidal space as a result of cataract surgery with larger incisions. The ocular contents protrude out of the surgical wound, which can have devastating consequences for both the surgeon and the patient with the sudden increase in intraocular pressure. The risk factors for suprachoroidal bleeding include advanced age, high intraocular pressure, history of systemic cardiovascular disease, systemic hypertension, and complicated cataract surgery (15). When it occurs, it manifests with rapid and progressive loss of red reflex and iris prolapse and sudden narrowing of the anterior chamber associated with increased intraocular pressure. Open wounds must be sutured immediately and intraocular pressure must be reduced with systemic Acetazolamide or Mannitol. Experienced surgeons can drain blood through scleral flaps. It was reported that

visual outcome was 20/40 or better in 40% of patients, and less than 20/200 in another 40% (14).

However, there is no evidence of an increased risk of significant intraocular bleeding as a result of the use of systemic anticoagulants in routine uncomplicated cataract surgery (16).

Post-operative complications

Posterior capsule opacification

Posterior Capsule Opacification (PCO) is the most common postoperative complication of cataract surgery. Migration of the residual lens epithelial cell remnants to the posterior capsule during the surgery causes opacification and results in decreased visual acuity, contrast sensitivity, and glare in the patient (17, 18). PCO is associated with age, intraocular lens material, intraocular lens design, amount of residual lens, presence of ocular inflammation history, and size of capsulorhexis (19). The incidence of posterior capsule opacification was reported as 11.8% in the first 1 year, 20.7% in 3 years, and 28.4% in 5 years (20).

A posterior capsulotomy is most often performed with a Neodymium YAG Laser Capsulotomy (Nd:YAG) device to eliminate visual complaints in the treatment in case of postoperative posterior capsular opacification. YAG laser capsulotomy rates were reported in 20.4% of pseudophakic cases (21). The procedure is generally simple and gives excellent results in improving the visual quality of the patient. Although it was shown that this therapy is safe and effective, it also includes complications such as transient elevation of intraocular pressure (15-30%), subluxation of the IOL (9-20%), transient iritis or vitritis (0.4-0.7%), CME (1.2%), and retinal rupture or decollement (2%) (22). For this reason, most physicians do not prefer to perform a YAG laser capsulotomy before three months following the surgery. The increased risk of retinal rupture and related decollement is more controversial. Young patients who have a higher risk of retinal decollement than the population and those with high myopia have a higher risk (23). Although the exact mechanism of how a YAG laser capsulotomy might be associated with a retinal rupture or hole is not fully known, the mobilization and deterioration of the vitreous structure because of the procedure are blamed (24). However, anterior vitreous decollement during phacoemulsification might also cause a hole or rupture in the retina. With posterior capsulotomy performed in such cases, the vitreous might displace anteriorly and the retinal rupture area may enlarge, and vitreous

leakage might occur under the retina from this growing rupture, and this may cause retinal decollement by separating the retina from its adherence (23).

The incidence of PCO in infants and children is markedly higher than in adults because of the higher proliferation ability of lens epithelial cells. Jensen et al. (25) suggested that PCO occurred in 40% of the eyes 22 months after the surgery, and for this reason, they recommended primary posterior capsulotomy in children younger than 6 years old.

Elevated intraocular pressure (IOP)

It was shown that there is a temporary postoperative increase in intraocular pressure (IOP) in eyes with previous cataract surgery. When the viscoelastic materials used during the surgery cannot be completely cleaned from the eye, the IOP rises temporarily after the surgery. Also, the higher the molecular weight of the viscoelastic material, the higher the possibility of postoperative high IOP. This unwanted situation can be prevented by administrating oral acetazolamide or topical antiglaucomatous agents in the early postoperative period. A previous survey study reported that one-third of surgeons routinely used a topical antiglaucomatous medication after surgery (26). Since glaucoma patients may have a higher risk of developing a postoperative increase in IOP, it is more likely that surgeons use such agents.

Another cause of ocular hypertension following cataract surgery is pigment dispersion occurring secondarily to accidental sulcus placement, especially of the one-piece acrylic lens used (10). Intensive use of postoperative steroid eye drops is also among the causes of secondary IOP increase. Steroid-induced IOP usually recovers after the discontinuation of topical steroids, but IOP because of pigment dispersion may require more intense topical anti-glaucoma medication use, and may even lead to glaucoma surgery if ocular hypertension does not improve with medical treatment.

Corneal Decompensation

Approximately 5-8% of corneal endothelial cell loss was reported after cataract surgery (27). Risk factors include a shorter axial length and longer phacoemulsification time (28). Postoperative corneal edema occurs immediately after surgery and usually recovers within 2-4 weeks. Endothelial traumas that result from prolonged surgical manipulations, persistent postoperative inflammation, high intraocular pressure, intraoperative or postoperative complications (e.g. posterior capsule rupture, toxic anterior segment syndrome,

or Descemet's membrane decollement) can irreversibly damage corneal endothelial cells, resulting in corneal endothelial damage, which can then cause the development of bullous keratopathy, which is a common indication for a cornea transplant. The presence of pre-existing corneal endothelial disease, such as Fuchs Endothelial Dystrophy, is an important risk factor for persistent corneal edema following the surgery (29).

To avoid this undesirable condition, it may be necessary to use a dispersive viscoelastic combination (e.g. Viscoat, Alcon Laboratories) to avoid excessive intraoperative phacoemulsification power and to protect the corneal endothelium by coating during the surgery (30). In the case of postoperative corneal decompensation, a corneal graft (endothelial graft) may be needed to improve the vision of the patient. Pseudophakic bullous keratopathy is currently the second most common indication for corneal graft surgery. For this reason, the timing of cataract surgery needs to be carefully adjusted according to the endothelin status, corneal thickness, and cataract grade in patients with Fuch Endothelial Dystrophy. Since a dense cataract requires much higher phacoemulsification power and consequently increased endothelial cell loss, early surgery can prevent such undesirable situations.

Cystoid macular edema

Cystoid Macular Edema (CME), which is also called Irvine-Gass Syndrome, is a clinical condition occurring approximately 4-6 weeks after the cataract surgery and can cause decreased vision. Intraretinal fluid accumulates in the outer plexiform and inner nuclear layers and forms cystic spaces. The incidence of asymptomatic cystoid macular edema, which can be demonstrated by fluorescein angiography in the absence of Optical Coherence Tomography, was approximately 2% after uncomplicated cataract surgery (31). It was reported that this rate can reach up to 5-14% with OCT (32, 33). However, its prevalence was reported as 1.2-11.0 even in the absence of other complications and risk factors (19, 34). Preoperative risk factors include diabetes, uveitis, previous retinal vein occlusion, epiretinal membrane, and the use of topical prostaglandin analogs (35). Also, the development of PCR increases the risk of CME at significant levels (34, 36).

Most patients recover spontaneously within 6 months, and therefore, it is difficult to evaluate the effect of therapeutic agents for cystoid macular edema (19, 36). Topical corticosteroids, topical NSAIDs, and periocular corticosteroid injections are commonly used in the treatment of CME (37). Although there is

no consensus, some ophthalmologists add topical NSAIDs after the surgery for patients who are considered to have greater risks for macular edema, such as those with diabetic retinopathy.

Optical Coherence Tomography (OCT) facilitates the diagnosis of cystoid macular edema by evaluating macular morphology in an accurate and non-invasive manner. OCT studies reported that clinically asymptomatic cystoid macular edema shows an asymptomatic increase in macular thickness even 12 weeks after the surgery (38). CME fluorescein angiography findings have a "flower petal-like" appearance, specifically, because the stain accumulates in the cystic spaces of the outer plexiform layer.

Although CME usually resolves spontaneously, it was shown that topical steroids and nonsteroids accelerate this improvement in the clinical manifestation (35). If topical treatment fails, intravitreal steroids can be used to resolve the edema. CME may also occur because of the traction of the vitreous in the anterior camera with insufficient vitreous cleaning after intraoperative PCR. In the treatment of this clinical manifestation, the traction can be improved by separating the adherent vitreous in the anterior camera with the YAG laser, but in cases where the YAG laser is not adequate, it may be necessary to re-operate the patient for anterior or posterior vitrectomy. The prognosis is generally good, with more than 90% of cases recovering within two postoperative years (39).

Retinal Decollement

The risk of retinal decollement increases after cataract surgeries. Posterior vitreous decomposition induced by a loss of internal volume that results from the removal of the cataractous lens and the implantation of a thinner intraocular lens was blamed in this respect. The increased risk persists for many years after the surgery. Cumulative risk of 0.9% was reported four years after the surgery, and this risk increases each year after the surgery (40). A previous study reported that the increased risk persisted for at least six years, and the overall eight-year incidence of retinal decollement increased almost nine times in operated eyes (41).

Preoperative patient-related factors were also found to increase the risk of retinal decollement. These include a history of retinal decollement in the other eye, greater axial length (especially for eyes longer than 23 mm), male gender, and surgery at younger ages (42). The risk may persist up to 10 years after cataract surgery in high myopia (43). Young patients who have advanced

myopia are particularly at risk. This must be kept in mind, especially in patients who underwent clear lens extraction to correct the refractive error.

Intraoperative complications such as PCR and zonular separation were reported to increase the risk at significant levels (42). It was also found that the falling of the lens parts into the vitreous increases the risk even more (44).

Pseudophakic patients must be carefully examined when they present with symptoms suggestive of retinal rupture or posterior vitreous decollement as existing peripheral retinal ruptures may not be evaluated clearly because of anterior or posterior capsular fibrosis or decentralization of the intraocular lens.

Endophthalmitis

Endophthalmitis is among the most feared complications of cataract surgery because of its devastating results with a prevalence of 0.006-0.04% (45). Among the most common risk factors, there are PCR, prolonged surgery time, leakage from the main incision site, less experience of the surgeon, advanced age, corneal incision site, and wound configuration (46). Other reported risk factors include immunosuppressive diseases (e.g. Type 2 Diabetes), silicone intraocular lenses, clear corneal incision, and being male (4, 45, 47).

Endophthalmitis can occur in acute or chronic form. Acute cases occur in the days after the surgery and manifest with pain, redness, prominent anterior chamber cells, hypopyon, and vitritis. It was reported that it is most commonly caused by gram-positive microorganisms and the valve flora of the patient (48). Chronic cases may appear weeks later (approximately 6 weeks) and present as persistent inflammation. Early recognition of the clinical manifestation with rapid anterior chamber and vitreous biopsies is vital. In acute treatment, intravitreal antibiotics, usually Ceftazidime and Vancomycin, and primary vitrectomy are performed depending on the severity of the case. In chronic cases, Proprioniobacterium acnes emerge as a cause of inflammation. Since the microorganism is classically located between the capsule and the IOL, swollen white posterior capsular plaque is seen in the biomicroscopic examination. Other than intravitreal antibiotics (usually Vancomycin) and vitrectomy, a capsulectomy with or without IOL extraction may be required (49, 50).

In cataract surgery, many risk factors have been extensively investigated for endophthalmitis. To reduce the risk, it is necessary to use an appropriate antiseptic solution (povidone-iodine) to cover the surgical area sufficiently to exclude the eyelashes of the patient, and to employ intracameral antibiotics such as cefuroxime at the end of the surgery. The European Society of

Cataract and Refractive Surgeons (ESCRS) study showed that intracameral administration of Cefuroxime 1 mg per 0.1 ml at the end of surgery reduced the risk of endophthalmitis approximately five times (from 0.34% to 0.07%) (51). However, preservative-free antibiotics that are formulated in doses suitable for intracameral injection are not commercially available in most countries, and therefore, the prophylactic use of intracameral antibiotics has not yet been adopted. The postoperative treatment of endophthalmitis includes rapid assessment and the use of intensively supplemented topical broad-spectrum antibiotics and intravitreal injections of antibiotics. The Endophthalmitis Vitrectomy Study suggested that immediate *Pars Plana Vitrectomy* is indicated when the baseline visual acuity of the patients decreases to light perception. In contrast, intravenous antibiotics are of no benefit in this regard (52).

In endophthalmitis prophylaxis, the clinical manifestation of severe blepharitis that causes inflammation, especially in the lids, must also be treated before the surgery because most of the organisms that cause infection originate from the patient's flora. The use of topical antibiotics before the surgery may also be beneficial in preventing endophthalmitis.

Endophthalmitis is a clinical condition that has a high morbidity rate and a poor prognosis. Poor prognostic factors include old age, corneal edema, or initial presentation with a hypopyon greater than 1.5 mm, detection of bacterial species other than coagulase-negative Staphylococcus species, and inability to obtain fundus images. In general, 49-57% of patients will have no better vision than 20/40 (6/12) (53, 54).

Refractive errors

The IOL Master is generally considered the most accurate and gold standard in biometrics, but it cannot be used in very dense or mature cataracts and patients who have a poor fixation. In contact lens users, soft lenses must be discontinued 24 to 48 hours before biometry and hard lenses 2-3 weeks before biometry is performed to stabilize keratometry (55). Once the corneal curvature and axial length measurements are determined, several IOL formulas are available each with its indication. Some of the formulas are better at estimating IOL power in extremely short or long eyes.

With current IOL formulas, target refraction within one diopter is achieved in 96% of cases (56). Biometry must always be examined for any irregular results before the surgery, such as asymmetry in axial lengths or keratometric values not reflected in refraction. In case axial lengths <21.3 and >26.6 mm,

keratometric values <41.0 D and >47 D, >2.5 D astigmatism, >0.7 mm axial length asymmetry, and mean keratometric findings greater than 0.90 diopters, it is necessary to be more careful so that there is no measurement error (57). In cases there is a postoperative refractive error, biometrics must be carefully evaluated again before the second eye surgery. Where ultrasonic applanation is used instead of optical alignment, one of the most common causes is incorrect axial length measurement. Pressing the ultrasound probe too hard on the eye may cause eye compression and thus shorter axial length measurement. Incorrect axial length measurement may occur in high myopia, especially in the presence of staphyloma. Another increasingly common cause of erroneous refractive results is previous laser refractive surgery. Changes might occur in corneal curvature as a result of laser refractive surgery, which might change the curvature of the cornea leading to changes in the keratometric findings necessary for the biometric measurement to be accurate. Better results can be achieved if refraction and K values are available before and after the LASIK, or by employing new technologies such as Pentacam (Oculus) and specially developed formulas such as the BESSt Formula to measure corneal strength (58).

Another cause of patient dissatisfaction with the refractive outcome may be monovision. It is important for patients to try to stimulate monovision before the surgery. Monovision can be done by using a trial contact lens before the surgery on both eyes or after the first eye surgery. It may be less important whether the dominant eye is corrected for distance or near.

For patients who are not satisfied with postoperative refractive error, whatever the cause, surgical treatment options include IOL replacement, and supine IOL (an IOL placed in front of the existing IOL in a capsule and into the sulcus), or refractive laser surgery.

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CHAPTER XI

MANAGEMENT AND COMPLICATIONS IN PEDIATRIC UROLOGY

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Pediatric urology surgeries are the most frequently performed operations in pediatric surgical practice. We will detail the most frequently performed surgeries in these operations under 4 main headings.

1. Circumcision

Circumcision is the most common elective surgical operation performed on men today. Circumcision; It can be defined as the process of surgically cutting the foreskin, called the prepuce, covering the glans penis, in a certain shape and length, and exposing the tip of the penis. It is known that an average of 25% of men all over the world are circumcised due to religious, cultural, medical, or familial choices. Circumcision is recommended in children with recurrent urinary tract infections, in the presence of congenital urological anomalies such as vesicoureteral reflux, after pathological phimosis, paraphimosis, recurrent balanoposthitis attacks, after zipping of the prepuce or other traumatic injuries, apart from religious and traditional preferences.

It is most ideal to perform the circumcision procedure under operating room conditions, with the support of the anesthesia team, by pediatric surgery, pediatric urology, or urology specialists. However, in our country, non-physician health personnel have been performing this duty for years under unsuitable conditions such as mass circumcision feasts and with high complication rates.

Most of the complications seen after circumcision are minor complications and are seen in the early period. However, it can be seen in early serious complications such as glans amputation. Complications seen after circumcision can be divided into 2 groups as early and late complications.

In the early period; Methemoglobinemia can be seen due to bleeding, wound infection, urethra or glans injuries, urinary retention, allergic reactions due to local anesthesia, and use of prilocaine.

In the late period; We can list deformities due to excessive or undercutting of the foreskin, stenosis of the meatus, adhesions between the glans and the body of the penis, skin bridges, inclusion cysts, urethrocutaneous fistula, bad scar tissue, and psychological problems (1).

In a retrospective study conducted by Geçit and Benli, it was stated that complications occurred in 89% of these patients after circumcision, mostly by non-physicians. Penile hematoma (8%) was observed (2).

Turkan et al. In another retrospective study conducted by specialist physicians, it was reported that while 1.2% was seen in the circumcision procedure performed by specialist physicians, 15% complications were observed after circumcision performed by non-specialists physicians. Another remarkable finding in this study was that while the rate of simultaneous urological anomaly detection was high (100%) in circumcision procedures performed by specialist physicians, this rate was reported to be very low 17% in non-physician personnel (3).

Early complications after circumcision

Bleeding: Bleeding is the most common complication. The majority of post-circumcision bleeding can be halted with easy techniques (applying pressure, cauterization, suturing). Inappropriately performed circumcisions or trauma after circumcision can cause bleeding from the frenular artery, the epidermis, or mucous membranes. A vitamin K treatment should be administered right away following delivery to stop the onset of bleeding issues brought on by coagulation disorders, especially in newborn infants. In children who are not prone to bleeding, pressure should be applied for 10-20 minutes and then a tight bandage should be applied, if this is not enough, suturing should be preferred in ongoing bleeding. It is recommended to perform good bleeding control in the circumcision process in patients with a blood disease, to replace the missing factor or use fresh frozen plasma for children with factor deficiency, to use local fibrin glue (fibrin glue) after the circumcision procedure, and to wrap with an elastic bandage at the end of the circumcision (1, 4).

Wound infection: Infection is very rare if circumcision surgery is performed under ideal conditions. However, serious wound infections can be seen in mass circumcision ceremonies and circumcisions performed by non-physician personnel, since asepsis is not followed in our country (4).

In a retrospective study conducted by Geçit and Benli, the rate of wound infection was found to be 20% after a mass circumcision festival, while this rate was reported to be 0.8% after circumcision performed in a hospital (2).

If a wound infection is detected, the patient should be managed with anti-inflammatory medications, local or systemic antibiotics, and daily dressings.

Glandular and penile shaft complications: Amputation and incision of the glans or penile shaft are rare complications of circumcision, which can be life-threatening and cause serious functional problems for the patient.

Urethral injuries: Urethral injury may occur as a result of compression of the urethra in circumcision procedures performed with Gomco clamps and similar devices. When the ventral penis sutures are stitched together to include the urethra for bleeding control, urethral erosion or urethrocutaneous fistulas may develop (1).

Uretrocutaneous fistulas: It can be repaired 6 months after circumcision. Generally, successful results are obtained in terms of functional and cosmetic.

Urinary retention: Urinary retention after circumcision is mostly due to the pressure dressing in the early period, and the problem is solved by loosening the dressing. It should be kept in mind that late urinary retention may be due to severe meatal stenosis or balanitis xserotica obliterans.

Anesthesia-related complications: Methemoglobinemia is a well-known side effect that may occur after the use of drugs containing prilocaine, which is preferred for local anesthesia in newborn circumcision. Methemoglobinemia is a potentially fatal complication of local anesthetic agents. Since the cytochrome b5 enzyme level, which helps to prevent methemoglobinemia, is low in the first months of life, it is thought that other local anesthetic agents should be used instead of prilocaine as a local anesthetic agent in children younger than six months. Asymptomatic patients with methemoglobin blood levels below 20% can be followed with conservative methods. If the methemoglobin blood level is higher than 30%, 1-2 mg/kg 1% methylene blue can be administered intravenously. The same dose can be repeated if the cyanosis does not resolve within one hour. If the methemoglobin blood level is above 70%, additional hyperbaric oxygen and blood exchange may be required (1,4).

Late period complications after circumcision

Mea stenosis: It is mostly seen after newborn circumcision. It can be seen after cutting the frenulum artery during circumcision or meatal irritation, especially after newborn circumcision. Children with meatal stenosis may have difficulty urinating, fringed urination, or voiding with a long and low flow rate. The

choice between the use of locally effective steroids and meatal dilatation and meatoplasty options is decided according to the patient and the clinical picture (4,5).

Incomplete or excessive cutting of the foreskin: These are complications that can be seen after circumcision performed by people who are not experts in the technique or the job. Circumcision revision may be required, since the penis will not have a circumcised appearance in the situation that occurs as a result of undercutting the foreskin (1).

Scar tissue complications: After circumcision procedures, light, thin and dense adhesions can be seen between the glans penis and the penile skin. Denser adhesions can form skin bridges. Mild adhesions can be treated with gentle retraction and topical steroid creams. Revisions may be required in the treatment of skin bridges formed as a result of dense adhesions. After cautery circumcision procedures, secondary phimosis may be encountered due to insufficient separation of the prepuce from the glans or due to inadequate and poor post-circumcision care.

Epidermal inclusion cysts: It can be seen if skin tissue, smegma residue or foreign material remains in the penile shaft after circumcision. Total excision is sufficient in the treatment of those who are mostly asymptomatic and cause cosmetic problems.

Psychological problems: Ideally, circumcision is performed under general anesthesia with the support of the anesthesia team. Contrary to popular belief, circumcision is a painful procedure for infants as well.

2. Hypospadias Complications

Hypospadias is a male sex anomaly with a rate of 1/300 and is in the first 3 rank among the causes of surgery in boys, along with undescended testis and circumcision. Although more than 300 surgical techniques have been reported, the overall complication rate is 20-30%. Severe chordee and penoscrotal skin transposition, which increases complications, is generally observed in more proximal hypospadias. However, tissue necrosis due to the formation of ischemic tissues or compression during the operation and the inexperience of the surgeon are very effective in the development of complications. Technical applications such as protection of the urethral plate, dorsal midline plication, covering the new urethra with de-epithelialized tissues, and alternative two-session techniques in appropriate cases, together with these standard steps, make a significant contribution to the reduction of complications. The use of an algorithm that

includes a small number of surgical techniques by hypospadias surgeons enables the experience of each technique to increase faster, the assistants to learn easier, and the hypospadiologist to better follow the non-technical factors affecting the result. The reasons for the occurrence of complications are largely known. However, it is still difficult to compare the success of scientific methods with standard techniques. Treatment of complications specific to hypospadias surgery, such as persistent hypospadias, urethral stricture, urethrocutaneous fistula, diverticulum, urethral calculus, residual chordee, hair growth in the urethra, and stone formation, involves various difficulties due to repeated surgeries. Patients with primary proximal hypospadias and those requiring reoperation are significantly more dissatisfied in the late follow-up. For this reason, it is important to reduce complications and to treat emerging complications without the need for reoperations as much as possible (6, 7, 8).

3. Ureteral Reimplantation Surgery

The relationship between reflux nephropathy and vesicoureteral reflux (VUR) has been known for a long time. On the other hand, the surgical treatment of VUR in the modern sense begins with an article by Hutch in 1952. However, the surgical treatment method described by Politano and Leadbetter in the journal J Urol in 1958 became widespread all over the world and became one of the most frequently applied surgical methods in children in the 1960s. Politano and Leadbetter's surgery offered a solution to this problem. The ureter is separated from the bladder by intravesical dissection, it is taken back into the bladder through a detrusor defect created more cranially and laterally from its original location, and a submucosal tunnel is formed, and the ureter is transported from the new orifice to its old place between the mucosa and detrusor, and anastomosis is applied again. Thus, the term "ureteroneocystostomy" was coined. Although we cannot draw its boundaries with precise definitions, VUR can take three forms (9).

1. A congenital, structural-anatomical ureterovesical junction anomaly
2. Secondary VUR due to bladder dysfunction
3. Coexistence of the above two conditions

The most obvious complication in the surgical treatment of vesicoureteral reflux is obstruction, not recurrence of the disease. Side effects of a real obstruction, which we know very clearly today, such as loss of renal function and hypertension

can occur after a ureteroneocystostomy. The most commonly used criteria for comparing surgical treatments with each other are the length of hospital stay, the need for hospital readmission, the need and duration of catheter use, and short-term complications. Long-term complications, such as rates of renal failure secondary to obstruction, have been rarely reported (10).

The two most commonly used methods of ureteroneocystostomy are: Politano-Lead better and Cohen operations. Comparing both techniques with early and partially late complications, most studies found that Cohen's surgery had fewer complications shown (9,10).

Üreteral reimplantation surgery: Complications

In the literature, there are 5 main surgical methods with which the above subjects can be compared. Apart from these, comparative literature is sparse.

When the most commonly used methods Politano-Leadbetter and Cohen techniques were compared, it was reported that the Politano-Leadbetter method was better in terms of recurrence and obstruction.

Complications of Lich-Gregoir surgery are usually post-operative urinary retention. The most important advantages of this method are that the bladder is not opened during the operation, there is no need for post-operative catheterization and its high success rates.

Although most of the comparative literature gives high success rates, a publication comparing success with respect to reflux grades has shown that success is reduced at high grades. Most reports for Lich-Gregoir surgery have been published for (usually) transient bladder dysfunction after surgery. This is probably due to the effect of the wound in the detrusor on the bladder nerves. The need for clean intermittent catheterization may arise after surgery. For these reasons, although retention is mostly temporary, unilateral surgery is recommended even in bilateral cases.

Post-surgery obstruction: The symptoms of obstruction can be very different. While the symptoms may appear early or very late, there are also those who develop asymptomatic obstruction. There is usually loss of renal function in silent cases, and we think this is the most morbid complication of ureteroneocystostomy. The most common symptoms in early cases are hypertension and decreased urine output (11).

We can list the reasons as follows:

1. Hematoma at the site of ureteroneocystostomy. It is usually temporary and rarely requires hematoma drainage.

2. Distal ischemia and ischemic anastomosis after careless or excessive dissection of the ureter. Often requires reoperation.
3. Angulation of the ureter at the entrance to the bladder. It is mostly seen in Politano-Leadbetter and Lich-Gregoir operations.
4. Bladder dysfunction not noticed during the operation.

It is often the cause of failed operations. As we mentioned above, some phenomena are silent. In most cases, even in the absence of obstruction, some hydronephrosis or hydroureter may be observed in the early post-operative period. The dilation usually disappears within a few weeks. If upper urinary dilatation lasts longer than a few weeks, catheterization of the ureters with cystoscopy is usually the first attempt.

Postoperative opposite reflux: After unilateral ureteroneocystostomy, recurrent urinary tract infection may be due to unrecognized bladder dysfunction or contralateral reflux.

Postoperative urinary tract infection: This problem is usually secondary to recurrence of reflux. However, it should be kept in mind that it may be due to bladder dysfunction or it may be uncaused. An important element here is upper urinary tract infection and its permanent effects (9,10,11).

4. Complications of Laparoscopic Procedures in Pediatric Urology

In the last decade, laparoscopic techniques in pediatric surgery have developed very rapidly and have been widely accepted by pediatric surgeons. With the use of minimally invasive surgery in urological surgical procedures in children, many complications that are not frequently encountered in open surgery have begun to be seen (12).

Complications of laparoscopic orchidopexy: Laparoscopy was first used in the treatment of undescended testicles in 1976. It has gradually increased its popularity as it causes minimal morbidity. The success rate of laparoscopic orchidopexy in abdominally located testicles is more than 90%. Laparoscopy is effective not only in determining the localization of the nonpalpable testis but also in surgical treatment. Its advantages are that it is fast and safe and that the surgical procedure is performed with a minimally invasive procedure. However, there is a risk of damage to the vas deferens, vessels and surrounding tissues during dissection in surgery. Due to the close proximity of the iliac artery and vein, there is a possibility of injury during dissection. In addition, there are studies showing the long operation time and high cost as disadvantages (12,13).

Complications of laparoscopic varicocelectomy: Varicocele is seen with a frequency of 15% in the adolescent age group. Laparoscopic varicocelectomy is one of the most common procedures in children. The laparoscopic approach is a safe, fast, and effective technique compared to other methods. The complication rate in laparoscopic approaches has been reported as 0-14%. During this procedure, the internal spermatic vessels are ligated. Although serious complications are not observed during the operation, vas deferens, inferior epigastric artery, iliac artery, and vein may be damaged during dissection, and subcutaneous emphysema may develop. In cases where lymphatic vessels cannot be dissected well, lymphocele can be seen after surgery. It is known that some surgeons consider having more than one incision as a disadvantage of laparoscopy (12,14).

Complications of laparoscopic pyeloplasty: Laparoscopic pyeloplasty has been widely used in patients with ureteropelvic stenosis. Although the success rate of this technique is similar to open surgery, it has lower morbidity. It can be done transperitoneally or retroperitoneoscopically. The most important early complications are urinary leakage from the anastomosis site, infection and complications related to the intestines. Paralytic ileus and severe hematuria cases have been reported after surgery. During the operation, blood loss may occur after difficult dissection due to periureteral fibrosis, the operation time may increase and dissection may be difficult.

Injury of the renal capsule with laparoscopic instruments, injury to the renal pelvis, migration of the ureteral stent, the passage of the pyeloplasty suture through the stent, injury to the colon, injury to the upper pole artery, and rupture of the double J stent are among the reported complications. Urinoma and hematoma can be seen in children after surgery. As a result, good presentation of the anatomy, optimal field of view, wide dissection rather than deep dissection, and providing a good image reduce the risk of complications (12).

Complications of laparoscopic nephrectomy and partial nephrectomy: Laparoscopic nephroureterectomy can be performed with a transperitoneal or retroperitoneal approach. The transperitoneal approach provides safe nephroureterectomy dissection because it provides a large area. However, intra-abdominal organ damage increases the risk of intestinal obstruction. Bleeding may occur during dissection from the renal pedicle, aorta, or vena cava. The most common complications are vascular injury, bowel injury, hematoma, pancreatic fistula, urinary fistula, urinoma, peritoneal perforation, and herniation in the port area. In addition, complications such as pneumothorax, duodenal perforation,

loss of function of the remaining kidney in partial nephrectomy, urinary leakage, the opening of the calyces, and collection in the renal lodge have been reported. The complication rate is 3% in the transperitoneal approach and 4% in the retroperitoneal approach (12).

5. Urogenital Trauma in Children: Complications and Treatment

Genitourinary system injuries constitute a small part of all pediatric traumas and most often (90%) develop after blunt trauma. Penetrating injuries are less common. In addition, iatrogenic injuries may occur during surgical interventions. It is aimed to evaluate the genitourinary trauma cases primarily for general body trauma and to perform systemic evaluation and supportive treatments, then to evaluate the case specifically for the organ and to program the treatment (15).

Children's kidney injuries are most frequently caused by blunt trauma, such as falls, auto accidents, and play ground mishaps. Gunshot and knife wounds that penetrate the skin are less frequent but can be far more dangerous. Additionally, some medical procedures can result in iatrogenic kidney damage (kidney and liver biopsies, percutaneous nephrostomy insertion, laparoscopy and abdominal open surgeries). (16-24).

Presence of macroscopic or microscopic hematuria suggests the possibility of renal injury. USG is a good screening tool in blunt abdominal traumas and the main purpose is to detect fluid (perisplenic, perihepatic and pelvic) in the abdomen. Contrast-enhanced computed tomography (CT) is now accepted as the gold standard in the diagnosis of renal injuries and detection of additional organ injuries. There is no diagnostic method other than contrast-enhanced CT in renal artery injuries. The main aim of treatment in kidney traumas is to protect kidney functions, reduce morbidity and mortality rates. There are two types of treatment for kidney injuries, conservative and surgical. Conservative treatment gives very good long-term results in most of the cases. USG, complete urinalysis and TA follow-up 6-8 weeks after conservative treatment provides valuable information about the development of complications in post-traumatic follow-ups. Depending on the severity of the trauma, renal scintigraphy to be performed 6-12 months later is important for the follow-up of parenchymal injuries (18-22).

In very few kidney injuries, there is an indication for emergency surgery. Nephrectomy should be performed if there is unstoppable bleeding during surgery, the patient's general condition is unstable, and there is extensive kidney injury.

Management of ureteral traumas and complications: In children, the ureters are rarely exposed to trauma because they are well protected by the surrounding tissues, small and mobile. The most common cause of ureteral injuries is iatrogenic (laparoscopic surgery, rigid ureteroscope, stone surgery). This is followed much less frequently by blunt trauma and penetrating injury. The diagnosis of ureteral injury is difficult because there are no specific symptoms and signs. For this reason, there is a delay in the diagnosis of ureteral injury and the diagnosis is often missed in the acute period. Abdominal pain, abdominal tenderness, fever, leukocytosis, ileus due to irritation and vomiting may be seen. Suspecting ureteral trauma is the most important diagnostic step. There is no specific diagnostic method for ureteral injury. CT can be used for diagnosis. The most specific finding on CT is the extravasation of the contrast material outside the ureter. Apart from this, abdominal mass, inability to see the ureteral trace, hydronephrosis in some cases and displacement of surrounding tissues are the main findings. If there is ureteral injury but delayed diagnosis, successful results can be obtained with urinary diversion by placing a ureteral stent (double J) or percutaneous nephrostomy (20-27).

Complications that can be seen in the early period after ureteral trauma are extravasation outside the ureter, urinoma, and abscess. In the late period, ureteral stenosis, hydronephrosis, urinary infection and urinary stones may occur. Percutaneous drainage and percutaneous nephrostomy catheter can be inserted for urinoma and abscess that occur in the early period, and a surgical treatment plan can be made according to the location of the injury.

For ureteral stenosis that may develop, successful results can be obtained with a long-term ‘double J’ stent and balloon dilatation. Urine cultures required for urinary infection should be taken and treatment should be arranged according to the antibiogram.

Bladder trauma and management of complications: Because the bladder is more intra-abdominal in children than in adults, they are more vulnerable to trauma. 90% blunt trauma is the most frequent cause of bladder injury. Injuries to the bladder that are iatrogenic and penetrating are also common. Pelvic fractures frequently accompany bladder injuries. Due to physical trauma, the bladder may become destroyed both intra- and extraperitoneally. Again, urological endoscopic operations may result in iatrogenic bladder damage. When performing an orchiopexy or hernia repair on young children, the bladder’s protrusion from the internal inguinal ring can easily result in harm.

In penetrating injuries, the bladder wall is directly affected. Penetrating injury of the bladder is often seen in conjunction with other abdominal organ

injuries. Retrograde cystogram is considered as a standard procedure in a case with suspected bladder injury. Extravasation of contrast indicates bladder injury. While treatment for extraperitoneal rupture caused by blunt trauma can be achieved in 90% of cases by draining the bladder by catheterization, primary surgical repair of the bladder is absolutely necessary in all intraperitoneal ruptures and penetrating injuries. Abscess may occur in the abdomen after urinary leakage from the bladder due to trauma or after surgery. This condition is treated with antibiotics and/or percutaneous drainage(20-27)..

Management of urethral trauma and complications: Impotence, stricture and incontinence in the long-term follow-up of urethral injuries increase the importance of choosing the treatment after injury in urethral traumas. The most common cause of urethral injuries in children is blunt trauma. In addition, the urethra can be injured penetratingly and iatrogenically. Urethral injuries are divided into two groups as anterior and posterior urethral injuries according to the anatomy of the urethra. Posterior urethral injuries are most common in children.

The presence of blood in the urethral meatus in a traumatized child suggests urethral injury. Urethral injury is supported by difficulty in voiding, painful voiding, full bladder, presence of perineal and penoscrotal hematoma, swelling and edema in the penis, and tenderness in the pelvis. The urethral injury should be considered in a patient presenting with such symptoms and a urinary catheter should not be inserted. Because the catheter can turn an incomplete urethral injury into a complete one. In such a patient, first retrograde urography should be performed. Extravasation of contrast outside the urethra on this radiograph indicates urethral injury. Retrograde urethrography can also be performed in unstable children with other organ injuries due to trauma after suprapubic bladder drainage is provided and the patient's general condition improves.

There can be two types of complications of urethral injury as early and late period.

Early period: Infection, stricture and complete urethral closure are the most common early complications. Blood or urine leaking out of the urethra as a result of injury can cause an abscess after infection. The infection can be alleviated with appropriate antibiotic therapy. Here, urethrocutaneous fistula and periurethral diverticula can be seen. After the development of stricture, urethrotomy and urethral dilatation can be performed, and if there is no response, primary repair can be performed with open or endoscopic surgical treatment.

Late period: Fistula and strictures can also be seen in this period. Their treatment is like early complications. Impotence and incontinence, which are late complications and can significantly affect the child's future life, have an important place here. These complications are proportional to the severity of the injury(20-27)..

External genital organ traumas and management of complications:

Extragenital organ injury is more common in children than in adults. It is mostly seen as a result of blunt traumas, falling off a bicycle, animal bites, traffic accidents, penile interventions such as circumcision, and sexual abuse. There are more extragenital organ injuries in boys than girls. In males, the testicles and scrotum are more affected. It is often seen as a result of blunt trauma. Significant edema and ecchymosis may occur in the scrotum after simple trauma. USG can be done to rule out testicular torsion. In simple injuries, analgesic therapy, hydration, and elevation of the scrotum and testicles are sufficient for treatment.

In more severe traumas, sometimes the scrotum skin can be opened and the testis can come out. In this case, very good wound care and cleaning should be done, the integrity of the testis should be ensured, and antibiotic treatment should be started (20-27).

5. Conclusıon

It should be known that complications in surgical treatment in pediatric urology can be so morbid as to lead to kidney loss. Surgical indications should be given to the patients correctly and they should be intervened at the right time. The maintenance and quality of the devices should be increased. Surgeons who are new to this business can start operations with experienced surgeons for the first time, which can reduce complications.

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Lütfen yazınızı kopyalayıp bu boş dosyaya yapıştırınız.

CHAPTER XII

AS A DENTAL IMPLANT COMPLICATION; INFERIOR ALVEOLAR NERVE INJURY

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1. Introduction

The perioral region is one of the most sensory regions in the body. Any sensory impairment in this area can cause speech and chewing disorders, nutritional deficiencies and difficulties in social relationships. Even minor nerve injuries can become a handicap in some individuals. In such cases, the quality of life of the patients is highly affected and the iatrogenic contribution of this situation further increases the negative psychological state of the patients(1, 2).

Sensory innervation of the face, mucous membranes and other structures in the head region is provided by 3 branches of the trigeminal nerve. These branches of the trigeminal nerve are the ophthalmic, maxillary and mandibular branches. Inferior alveolar nerve (IAN), mental nerve and lingual nerve (LN) are the most injured terminal branches of the mandibular nerve during oral and maxillofacial treatments(3, 4). Injury of terminal branches of the IAN is unfortunately a common problem after oral and maxillofacial surgery and sometimes even during routine dental treatments(5-7).

Injuries of IAN, mental nerve and LN branches may occur due to various reasons. Trigeminal nerve damage during dental treatments may occur due to nerve contact with various chemical stimuli. Injection of local anesthetic directly into nerve branches(2, 8) and contact of canal filling pastes and irrigation

solutions such as calcium hydroxide used during endodontic treatment to nerve branches may cause chemical damages (6, 9). Manipulation of nerve branches and surrounding tissues during orthognathic surgery for the correction of maxillofacial deformities is another cause of nerve damage. Direct mechanical nerve damage may also occur during extraction of third molars or removal of cyst or tumor tissues (10-12). Terminal branches of the IAS may be injured after maxillofacial traumas such as mandible fractures involving the mandible body and parasymphysis region(13, 14).

While direct manipulation of the nerve may cause elongation, crushing, compression or disruption of its integrity, manipulations to the surrounding tissues may damage to the nerve as a result of temporary edema, infection or ischemia. The accompanying complication of nerve damage depends on the severity of the damage. Complications can range from mild complications such as transient hypoesthesia to all life-altering conditions such as neuropathic pain or trigeminal neuralgia(4, 15).

Dental implants are a widely used treatment for the treatment of missing teeth. Due to its ability to effectively restore aesthetics and function, it has become the preferred option for replacing hopeless and missing natural teeth. High success rates of 94.6% also increase the application of dental implants (16) However, despite the high success rate, many complications related to its use can be encountered. In 1995, Worthington said, 'the number of practitioners performing implant surgery has increased dramatically over the past fifteen years. As clinicians grow in self-confidence, they tend to accept more and more challenging cases, and the incidence of problems and complications is expected to increase.' (17) One of the most common complications related to implants today is altered sensation in the posterior mandible. (18). The most traumatized nerve during implant surgeries is the IAN. (19).The incidence of implant-related inferior alveolar nerve (IAN) injuries varies from 0 to 40 % (20). However, studies evaluating the incidence of nerve injuries also have many inconsistencies in terms of injury classification. There is a large variation from study to study in terms of sample sizes, use of different evaluation methods, and retrospective nature of these studies (21)

2. Causes and Prevention of Implant-Related IAS Injury

Implant-related nerve damage is often the result of inadequate planning. These injuries are mainly due to unawareness of the amount of bone available for implant placement or lack of knowledge of the location of the neurovascular

canal. At the same time, insufficient evaluation of bone quality in the implant area is compatible with this situation. Pre-treatment planning and adequate radiographic evaluations will not only provide successful restorative results, but will also help minimize damage to surrounding tissues(22). In most cases, clinical evaluation, 2D radiographs, and intraoperative evaluations are sufficient(23). In advanced cases or in the presence of uncertainty in two-dimensional radiographs, 3-dimensional radiographic evaluation with CT (computed tomography) scanning will be useful. Although it is not necessary for all cases, performing the surgical procedure with a surgical guide prepared in accordance with the anatomical structures is also very convenient in terms of not damaging the neurovascular structures.

Neurosensory impairments can occur at all stages of dental implant surgery, including local anesthetic administration, incisions, soft tissue release, osteotomy preparation, bone augmentations, implant placement, suturing, or soft tissue swelling after surgery(19, 24). Injury to the IAN as a consequence of bone preparation or implant placement can be caused by errors in radiographic planning, drilling, or direct contact of the implant with the nerve.

Drill injuries to the IAN can be difficult to diagnose. Despite the distance between the nerve canal and the implant on the postoperative radiograph of the implant, the drill preparation may have been performed beyond the planned implant depth causing injury to the nerve (25). This error can be avoided by accurate radiographic measurement, the use of drilling equipment with predetermined depth stops, and careful technique to avoid drilling beyond the planned depth (25).

Indirect postoperative trauma may occurs as a result of partial perforation of the mandibular canal while drilling. Secondary ischemia due to hemorrhage and scar formation is the cause of this condition. Nerve injury occurs indirectly rather than from direct mechanical trauma as in implant preparation or placement(20). Thermal stimuli may also cause peri-implant bone necrosis and postoperative secondary IAN injury. It is thought that nervous tissue is more sensitive to thermal stimuli than bone tissue and may cause primary IAS damage. (26). The temperature increase produced by excessive drilling speed causes necrosis, fibrosis, osteolytic degeneration and an increase in osteoclastic activity. The thickness of the necrotic area is directly proportional to the amount of heat generated during surgery(27). In a study, it was shown that the maximum temperature at which the bone can be heated without necrosis is 47 °C and staying at 47 °C for 5 minutes will cause 20% bone resorption(28).

Usually the implant drill is long*er (in y dimension)* than the corresponding implant to facilitate slightly more subcrestal placementSimilarly, implant drills are often smaller than the diameter of the corresponding implant to provide primary stability(18). It is important for the Clinician to know size of the implant drills in terms of planning and implementation. Lack of knowledge on this subject can cause preventable complications.

The height and width of the bone at the surgical site should be adequate, as well as bone quality should be evaluated for implant placement and avoiding damage to surrounding tissues. In some cases with insufficient bone density, twist drills may fall into large intratrabecular spaces, resulting in deeper drilling than planned(29). Awareness for bone quality can be increased using a 2mm initial twist drill. When transitioning from compact bone to cancellous bone, the applied pressure should be reduced accordingly. Otherwise, the tissues around the nerve may be damaged. The use of third-party CT planning software before the procedure can give an idea about bone density(30). In the results of a study in which Başa and Dilek measured bone density and thickness using CT, average bone thickness in the premolar and molar regions was 0.87 ± 0.18 and 0.86 ± 0.18 mm, respectively, whereas the bone density in the premolar and molar regions was 649.18 ± 241.42 and 584.44 ± 222.73 Hounsfield units (HU). As a result of this study, it was concluded that the average density and thickness of the bone surrounding the mandibular canal was not sufficient to resist in implant drilling. The risk of IAN injury can be avoided by accurately determining the bone density around the canal and avoiding excessive force when approaching the canal(31).

In addition to drilling injury, the extent of IAN damage caused by the implant itself is related to the state of the implant entering the canal or its direct contact with the IAN (25). Despite proper bone preparation, nerve damage from implant placement can occur when the implant is placed beyond the vertical boundaries of the prepared bone, compressing or breaking the upper wall of the IAC and forcing the bone into the canal (22, 25, 29). Also, drilling deeper than planned may facilitate insertion of the implant beyond its intended depth and the implant may come into direct contact with the IAN.

Another cause of nerve injuries is immediate implant applications in areas closely related to neurovascular structures. In immediate implant applications, adequate drilling is required at the apical part of the extraction socket.. If the neurovascular bundle is close to the extraction socket, excessive crossing of the apical border may cause damage to the nerve structures(32). When there are concerns about the proximity of the neurovascular bundle apical to the extraction

socket, the implant can be performed with the delayed protocol without the need for excessive drilling, avoiding the immediate implant.

The mental nerve is located in the mandibular buccal soft tissue and there is a risk of injury during incisions. As the patient ages, the alveolar bone in an edentulous area resorbs and the position of the mental foramen approaches the apex of the alveolar crest (25). Therefore, this anatomical structure should be taken into account when positioning the incision. It is possible to apply sustained excessive pressure on the underlying MN during retraction of a mucoperiosteal flap. Gentle soft tissue retraction is recommended.

Located in the mandibular retromolar region, the lingual nerve is very close to the lingual plate below the ridge of the lingual crest. The position of the lingual nerve in anatomical dissections shows various variations. In a magnetic resonance (MR) study, it was reported that 10% of patients were actually on the retromolar pad(33). In these cases, the lingual nerve may be traumatized during flap elevation, release or suturing. Incisions in the ramus region remain on the buccal side and avoiding the over lingual approach can prevent possible nerve damage.

3. Classification of Nerve Injuries and Mechanism of Injury

IAN injury is mainly diagnosed by the patient's complaints and clinical examination findings. Depending on the size of the IAN injury, the sensory changes that the patient feels can range from a moderate paresthesia to anesthesia. Some situations can be temporary, manageable, or in some cases permanent. The mental region and the skin of the lower lip, mucous membranes, the area extending posteriorly to the second premolar teeth and gums may be affected(34). This can affect the patient's nutrition, speech, make-up, shaving, and even face washing(2). The altered sensation may present as paresthesia, dysesthesia, analgesia, or anesthesia(18). Paresthesia can be defined as a not unpleasant sensation such as numbness or tingling that is felt due to stimulus or spontaneously, while dysesthesia can be defined as an uncomfortable abnormal sensation that occurs either spontaneously or due to stimulus. Analgesia, on the other hand, means the disappearance of the sense of pain, and anesthesia means the total loss of sensation and perception against all harmful or non-harmful stimuli. Hypoesthesia describes decreased sensation to stimulus, while hyperesthesia describes increased sensation to stimulus (35). After bilateral sagittal split osteotomy, endodontic treatment, local anesthesia injection and post-procedure inflammatory processes, the most common sensory impairment

has been shown as hypoesthesia. In implant-related nerve damage, the sensory change varies according to the extent of the damage (4, 36). In a study by Agbaje et al., they showed that the most common sensory change was hypoesthesia, followed by neuropathic pain (4). The Association for the Study of Pain has standardized the most commonly used neurosensory definitions with a nomenclature (Table 1) (35).

Clinically, nerve injuries are described as beingeither “open” or “closed” (37). An open injury isone in which the oral and maxillofacial surgeonwitnesses the nerve injury (for example, seeing a severed IAN in a third-molar socket). Such injuries should be repaired promptly by an oral surgeon with microneurosurgical expertise. If that is not possible, the patient should be referred to a nerve injury specialist for nerverepair without further delay (37). A closed injury is an injury that is not visible to the surgeon at the time it occurs. In such cases, the patient applies to the dentist to complain of numbness a day or more after the procedure. These closed injuries are the most problematic for the dentist (37).

Seddon classifies nerve injuries as neuropraxia, axonotmesis, and neurotmesis. In neuropraxia, axon continuity is preserved and the injury is generally transient. There is a more serious injury to the axonotmesis. Although there was deterioration in the axons, the structural integrity of the neural tube and surrounding tissues remained intact. Neurotmesis is the most serious form of nerve injury, and neural tube structural integrity has also been compromised(38). Another classification was made by Sunderland in 1951 according to the degree of tissue damage, emphasizing the importance of each structural component of the nerve trunk. There are 3 types of first-degree injury in this system, similar to Seddon neuropraxia. Type 1 results from manipulation of the nerve trunk, mild traction, or mild compression, and is thought to result from transient ischemia. Nerve function usually returns to normal if blood flow improves; permanent injury and anesthesia may occur with longer ischemia.(39)

Table 1: Definitions of neurosensory disorders

Anesthesia	Total loss of feeling and sensation
Dysesthesia	An unpleasant feeling that occurs spontaneously or due to a stimulus
Allodini	Pain due to a stimulus that does not normally provoke pain
Hyperpathia	Increased painful response to stimulus
Causalgia	A burning sensation that persists after a traumatic nerve lesion
Anaesthetic dolorosa	Pain felt in the anesthetic area
Paresthesia	A not unpleasant feeling that occurs spontaneously or due to a stimulus
Hypoesthesia	Decreased sensitivity to stimuli
Hyperesthesia	Increased sensitivity to stimuli
Hypoalgesia	Decreased response to a stimulus that is normally painful
Hyperalgesia	Increased response to a stimulus that is normally painful
Synesthesia	Sensation felt in an area when another area is stimulated

Referrance*: Merskey, H. and N. Bogduk, International Association for the Study of Pain. Classification of chronic pain. 1994, IASP Press Seattle.*

Type 2 results from intrafascicular edema, decreased blood flow, and more specific traction or compression creating a conduction block, recovery variable. Type 3 injuries result from severe nerve traction or compression causing segmental mechanical disruption of myelin sheaths and demyelination. Recovery is delayed and loss of sensation may be permanent(39). Second, third, and fourth degree injuries correspond to axonotmesis in Seddon's classification. Afferent or efferent fibers are damaged, but the endoneurium, perineurium, and epineurium remain intact. Surgical decompression may be necessary and recovery requires axonal regeneration. Fifth degree injury means nerve transection. Surgical approach may be necessary(39).

In a histological study, it was shown that clinical signs and symptoms are the most important data in the evaluation of nerve injuries. For example, if there is complete numbness at the beginning and this tends to improve over time, this indicates a Sunderland first and second degree injury. The presence of complete numbness at the beginning does not mean that the continuity of the nerve is completely disrupted(29, 40).

Clinicians should be familiar with these types of nerve injuries and be able to perform standard neurosensory examinations to determine the extent of sensory alteration, be aware of the likely consequences, and decide when to refer the patient to a specialist. The patient's neurosensory functions should be evaluated

as part of the initial examination when starting implant therapy, particularly in patients with a history of sensory changes from previous dental procedures.

There are many neurosensory tests that can be used to detect neurosensory disturbances in the IAN and its terminal branches. These tests range from tests that can be easily performed with simple instruments in the clinic to advanced tests that require high-tech equipment. Simple clinical neurosensory tests are the most widely used and can be classified as mechanoceptive and nociceptive tests. (Table 2). Each test should be performed with the patient closing their eyes, in a relaxed position, away from distractions. Clinician should use contralateral side as control side and results should be recorded accurately(18, 41). Mechanoceptive tests include static light touch, two-point discrimination, and brushstroke direction. Pin tactile discrimination and thermal discrimination are nociceptive tests. Each test evaluates specific categories of receptors and axons.

In addition to objective tests such as clinical neurosensory tests, there are also tests that allow subjective examination. Trigeminal somatosensory evoked potentials are an electrophysiological method used to evaluate the trigeminal pathway. It is based on the detection of cerebral origin potential changes in the scalp after electrical stimulation of peripheral nerves (42). Another method used to monitor the function of the IAN is the recording of the orthodromic sensory nerve action potential.It is routinely used in conjunction with electromyography to evaluate peripheral nerve function (29, 43). *Blinx test*, in which the function of the trigeminal nerve is evaluated by placing electrodes on different parts of the face, is one of the sensory tests that can be used in the evaluation of IAN damage.

Table 2: Clinical neurosensory tests

Name of Tese	**Description**
Mechanoceptive tests	
Static light touch	The patient is asked to say when she feels a light touch on his/her face and point to the exact location.
Brushstroke direction	The patient is asked to tell when she feels the brush and to determine the movement direction
Two-point discrimination	The patient is asked to locate the single and 2 contact points. The examiner uses any 2 instruments with which he can change the distance between them.
Nociceptive tests	
Pin tactile discrimination	The patient's awareness when the needle is inserted is examined.
Thermal discrimination	The patient's feeling of cold or heat is evaluated.

Referances*:Akal, Ü.K., et al., Evaluation of the neurosensory deficiencies of oral and maxillofacial region following surgery. International journal of oral and maxillofacial surgery, 2000. 29(5): p. 331-336.*

Poort, L.J., J.W. van Neck, and K.G. van der Wal, Sensory testing of inferior alveolar nerve injuries: a review of methods used in prospective studies. Journal of Oral and Maxillofacial Surgery, 2009. 67(2): p. 292-300 (41, 44)

In cases where the nerve continuity is preserved, individuals can blink bilaterally, but if the continuity is lost, blinking does not occur on the affected side (45). In addition, electrical pulp tests and carbon dioxide snow tests can be done to test the viability of the nerve (4). In rare cases, biopsies, fMRI, and teleradiographies are also used in the literature(4). There is an opinion that patients' subjective reports may provide a more sensitive indication of the presence of post-traumatic nerve damage than neurosensory test results, but tests are clearly needed to determine the nature of the injury, eg. Whether sensory functions mediated by small versus large diameter fibers are mainly affected (46). Clinical judgments regarding nerve injury-associated sensory dysfunction should not be based on threshold testing results only without the consideration of patients' subjective reports of altered sensation.

4. Considerations According to the Characteristics of the Regions

The IAN is one of the terminal branches of the mandibular nerve that carries both motor and sensory fibers. It enters the mandibular canal through the mandibular foramen in the ramus of the mandible. The average thickness in the mandibular canal is usually 3 mm, although there may be different variations. After the IAN completes its course in the mandibular canal, it emerges from the mental foramen as the mental nerve. It can follow a slight inclination towards the mental foramen within the mandibular canal, as well as an ascending or descending course (34). Anatomical differences such as being more superficial or deep in some areas create points to be considered in different parts of the mandible in implant surgery**.**

4.1. First Premolar Region, Anterior Loop of Mental Nerve

The term anterior loop refers to an extension of the IAN in front of the mental foramen before exiting the canal. It is also defined as the anterior loop of the mental nerve (32). Radiographic and anatomical evaluations show that the mental nerve makes an anterior loop. It is not clear how often this formation appears and its size. Greenstein and Tarnow concluded in their study that the use of radiographs in the evaluation of the anterior loop of the mental nerve can lead to false positive or false negative evaluations (32). In another study, the mean width of the anterior loop was found to be 2.39 mm (47). In both studies,

a 2 mm coronal distance from the suspicious area was defined as the safe area in implant planning.

The first premolar region, and the region between the first and second premolar regions, is one of the regions that requires careful planning when placing the implant. This should also be taken into account for angled implants for planning hybrid fixed prosthesis. In these cases, distally placed implants are placed at an angle to provide antero-posterior load distribution. In addition to radiographic evaluation, it may be useful to direct visualition of the foramen during surgery and to determine the safe area with a probe (22). In cases where there is an anterior loop, nerve damage may occur in the apical parts of the angled implant, although it is seen that there is a distance from the nerve in the coronal region. All implants in this area should be made after accurate and adequate radiographic evaluations prior to the procedur***e.***

4.2. Second Premolar Region, Mental Foramen

The mental foramen is located in the buccal cortex of the mandibular bone, just below the corners of the lips (chelion) on both sides and in close association with the root of the second premolar tooth (48). It moves posteriorly during the development of the mandible and usually makes a posteriorly directed ascent in adults (48, 49). Variations in the location of the mental foramen have been reported within and between various adult population groups. In cadaver studies, it has been shown that the mental foramen is in contact with the mandibular second premolar root at a very high rate of 73.8%. The second most common spot is between the roots of the first and second premolars (22). It can be said that these findings are especially important in immediate implant applications to be made in the second premolar region. In these applications, a deeper osteotomy is needed in the apical extraction socket to provide primary stability. In the presence of a mental foramen closely related to the root, nerve damage may be inevitable. In order to avoid nerve damage in surgeries in this region, the application of the delayed implant protocol if necessary, detailed planning with appropriate radiographic images before the procedure, and the visualization and measurement of the upper border of the mental canal with extra flap elevation should be performed.

4.3. Atrophic Posterior Mandible

Many patients have a toothed anterior segment and edentulous posterior segments in the mandible. In most cases, atrophy is observed in the mandible due to the effect of the time of edentulousness. This makes it difficult to use

partial dentures as well as making implant placement difficult. Considerations for safe implant placement in the atrophic posterior mandible will be explained in Chapter 5.

4.4. Anterior Mandibula Region, Mandibular İncisive Nerve and Canal

The mandibular incisive nerve (MIN) is defined as the terminal branch of the IAN and provides innervation to the incisor and canine teeth. MIN and its canal are located between the interforaminal regions. Although numerous studies report IAN damage during implant placement (29), few reports describe sensory changes related to MIN damage (50, 51). However, this nerve has been recognized as an important cause of such complications when removing bone from the interforaminal region (52). MIS is increasingly recognized as an important anatomical structure to be considered when planning surgery in the anterior mandible. However, there are still debates among clinicians regarding the clinical importance of the MIS in implant surgery (51).

In this region, it is often difficult to pinpoint the exact location of MIS with radiographs. Its location can be determined by CT or magnetic resonance techniques. Although not as common as IAS, it should be kept in mind that there is a risk of nerve damage in the anterior mandible as well. It may be beneficial to use CT in the planning to be made in this region, and to avoid excessively long implants.

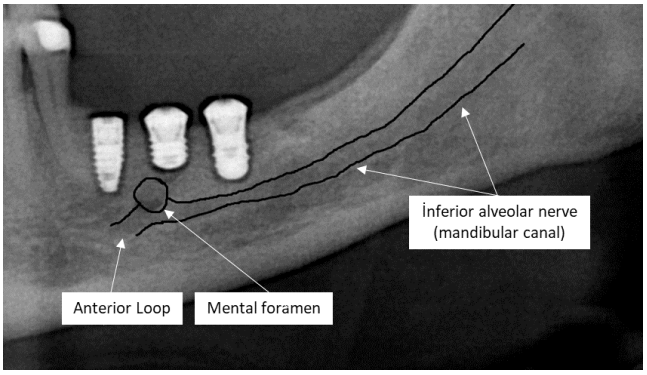
5. Avoid An Injury

Most injuries can be prevented with appropriate patient selection, adequate planning, and proper implementation of procedures (18, 20, 22, 29). Many clinicians plan the location of the IAS canal and the place where the implant will be made by making preoperative planning with software programs. There are 3 basic methods to avoid nerve injuries. These methods include maintaining a safe distance of at least 2 mm between the coronal of the nerve canal and the implant seen on periapical or panoramic radiographs, taking a CT (cone beam CT) if there are doubts about the location of the nerve on the radiographs and evaluation with appropriate software, direct visualization of the nerve during surgery, the location of the mental nerve and the anterior loop could not be detected on radiographs (20, 22**).**

5.1. Short Implants

There is no consensus on which implant should be considered as a short implant. While it is evaluated as <10 mm short implant in some studies (53),

it is evaluated as <6mm in some studies (54). Short implants are one of the ways to avoid nerve injuries in areas where the bone over the mandibular canal is minimal. Studies examining the success and survival rates of short implants have conflicting results. Overall, survival rates of short implants (≤6 mm) ranged from 86.7% to 100%, whereas the survival rates for longer implants (>6 mm) ranged from 95% to 100% with a follow□up from 1 to 5 years (54).



Figüre 1; Rehabilitation of the atrophic mandibular molar region with short implants (<6mm)

Insufficient bone thickness in posterior partial edentulism in the mandible and maxilla will be the first of the correct indications for short implants. Longer implants can be placed by performing sinus floor elevation using bone grafts in the maxilla. However, short implant placement is still more advantageous because of less morbidity and less biological complications (54). Vertical bone augmentations in the mandible are less predictable and more challenging (55). Therefore, under suitable conditions, short implants can be applied as an alternative. In the posterior mandible, short implantation is an effective way to avoid IAN injury if primary stability is achieved (22).

Although the crown-to-implant ratio does not appear to be associated with crestal bone loss or risk of failure of short implants (56), splinting short implants is advantageous in final fixed prostheses. In cases with two or more adjacent implants, splinting the short implants and providing the patient with mutually protected or canine guided occlusion will reduce mechanical forces on the individual implants and components (54).

5.2. Local Anesthesia Applications

In implant applications in the mandible, infiltration anesthesia is usually sufficient for the anesthesia of the region, without the need for IAN regional block. At

the same time, the lack of block anesthesia helps the clinician by causing the patient to react when the neurovascular structures in the mandibular canal are approached too closely during drilling or implant placement (22). At the same time, not applying block anesthesia will also protect the IAN from possible needle trauma. Sensory changes after implant surgery with block anesthesia will cause confusion if there is no evidence of direct implant-related nerve injury.

Local infiltration anesthesia provides adequate anesthesia in both flap and flapless implant surgery. There was no difference in terms of patient comfort between the application of block anesthesia or infiltration anesthesia during implant surgery (57). This is mainly due to the placement of implants in healed or grafted extraction sockets, which are typically poorly innervated, and sensory innervation to the mandible is primarily from the periosteum. Deep pulpal anesthesia required for restorative tooth preparation or tooth extraction is not required for implant placement (22, 57).

5.3. Bone Augmentation Techniques

During implant placement, increasing bone thickness with bone augmentation techniques can create a safe area for implant placement in atrophic cases where there is concern that it will come too close to the nerve canal. There are many augmentation techniques that will allow the creation of a safe area in the bone at least 2 mm from the nerve canal. Examples of these applications are bone distraction technique, onlay block graft application, interpositional sandwich technique, guided hard tissue applications using membranes such as titanium mesh (22). For this purpose, autogenous bone grafts, xenogenous grafts, allograft, alloplasts and bone morphogenetic protein can be used. Although it is not discussed in this section which of these materials and techniques are superior, increasing the bone thickness above the nerve canal in the atrophic mandible is an effective way to prevent possible nerve injuries. In bone augmentation procedures, sufficient interarch distance should be considered for proper prosthetic planning. It should be noted that improper interarch distances will cause difficulties in prosthetic abutment and crown placement.

5.4. Nerve Lateralization Procedures

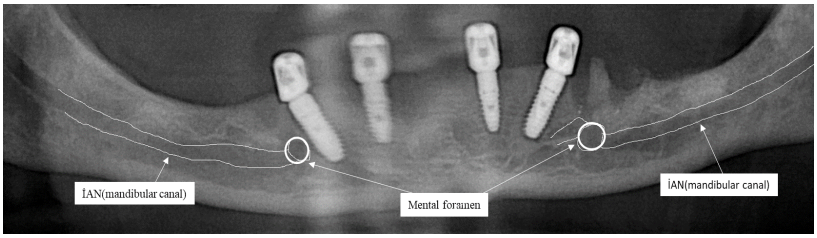
In situations where bone augmentation would cause interarch distance problems as described above, nerve lateralization is a useful procedure to facilitate placement of implants in atrophic posterior mandibles. With this procedure, cortical bone inferior to the mandibular canal can be used, which can increase implant stability.

Although useful, high experience and practice is needed for nerve manipulation. This need limits its routine application. Nerve lateralization requires decortication of the lateral cortex along the mandibular canal. In order to reveal the mandibular incisive branch of the IAN, decortication of the mental foramen region is also required. The incisive branch is usually released; and this allows the inferior alveolar branch and mental branch to make lateralization and elongation movements. When the nerve is lateralized, the implant is inserted into the alveolar bone medial to the nerve. The implant can be inserted deep enough to provide primary stability, even up to the lower border of the mandible. Bone graft is placed in the defect area caused by decortication. Some surgeons prefer to protect the exposed nerve with a type I/III collagen or polyglycolic acid tube (22, 58).

Most patients experience altered sensation after nerve lateralization. In most cases this is temporary. Besides being difficult to perform, sensory changes are another reason most clinicians avoid this technique. Although some cases of prolonged weak hypoesthesia have been reported in the literature, nerve lateralization is an effective technique that can prevent permanent nerve damage in atrophic mandibles (22, 58, 59).

5.5. Avoiding Implant Placement in the Atrophic Mandible

Many patients have early loss of mandibular molars; and even the use of partial prosthesis can be difficult in these patients who have alveolar crests that become atrophic over time. Implant treatment becomes complex in patients with atrophic mandibular crests for the same reasons that complicate prosthetic stability. In patients with total edentulous atrophic posterior mandible, the All-on-four concept can be applied where 4 implants are applied to the interforaminal region. The all-on-four concept is actually inspired by the work of Branemark et al (60). In this study, fixed prosthesis was made using 4 or 6 implants in edentulous mandible and maxilla crests. Although the 10-year results of the study show good success (78.3-80.3% for the maxilla and 88.4-93.2% for the mandible), a very long cantilever length is needed to provide sufficient posterior teeth. In atrophic jaws, before implant placement, bone grafting, augmentation techniques and sinus lifting surgeries for the upper jaw can be an alternative to this method. These procedures come with additional surgeries, additional costs, long treatment time and comorbidities experienced by the patients. As mentioned earlier, the inferior alveolar nerve lateralization has high success rates, but the high rate of postoperative paresthesia and the high technique and skill requirement of the method limit its use (61).



Figüre 2; Angled implant placement with the *All-on-Four* concept in a patient with bone deficiency above the upper border of the mandibular canal

In the All on four concept, distal angled implants were studied to improve implant position and reduce posterior cantilever length. Distal angled implants; Although it is a less invasive technique than conventional axial implants placed with bone graft applications, it has many biomechanical and clinical advantages. In 2000, Krekmanov et al. demonstrated that posterior angled implant-on-implant prostheses are possible (62). Mainly combined with antero-posterior force distribution, shortening of cantilever size, and cross-arch stabilization, prosthesis/implant results will be similar to those of conventional axial implants. Also angled implants; Allows posterior displacement of the implant support and placement of longer implants, and improves prosthetic load distribution. In addition, the amount of load on the implants is reduced with a rigid prosthesis. A rigid prosthesis combined with improved load distribution helps to minimize any significant movement and eliminates coronal stress at the marginal bone level. Angled distal implants at 30° to 45° positions relative to the occlusal plane allows 10 to 12 teeth to be placed. In addition, the implants can be spread over a more suitable distance for cleanability (61, 62) (Figüre 1). This treatment plan involves less time, less surgery, and fewer implants. IAN damage can be eliminated by evaluating the relationship of the implants with the nerve adequately. It is recommended to evaluate the location of the mental foramen well and to provide direct vision during surgery by releasing the flap elevation.. For these reasons, it is a useful strategy to restore an atrophic posterior region and avoid potential nerve injuries.

6. Management Of İmplant Related Injury

If there is concern about intraoperative nerve damage, it should be recorded and a comprehensive neurosensory examination performed as soon as the effect of local anesthetic wears off (63). During follow-up visits, clinical Examination results and the patient's complaints of sensory changes should be recorded.

Clinicians should suspect intraoperative nerve injury; Abnormal pain or change in sensation during drilling/implant placement, drill/implant slipping deeper than planned, and presence of pulsating excessive bleeding, especially if nerve proximity is suspected (18).

IAN injuries are a very disturbing condition that greatly affects patients' quality of life, and the iatrogenic origin of the injury makes this even more disturbing (1). Treatment of nerve injuries begins with the psychological treatment of the patient. Patients need accurate information, explanation, support, and realistic information about treatment outcomes quickly (29). Psychological treatment includes removing the implant that is pressing or in contact with the mandibular canal within 36 hours (20) to reduce the risk of permanent nerve (18, 29). In addition, there should be no irritants (bone debris, hematoma) in the area to allow bleeding and debris to disperse faster. (64). No bone graft should be placed in the area; because in this case, the graft material may enter the nerve repair area and interfere with healing. If the implant in the problem area is already osteointegrated, it can be removed using trephane drills. If there is concern about further damage to the surrounding tissues, if appropriate conditions are provided, an apicoectomy can be performed to relieve the nerve (65).

Even if no problems are encountered during the procedure, patients may experience sensory changes. The management of this condition varies depending on the cause of the IAN injury. This situation can develop due to many reasons as mentioned before. The cause should be determined by taking radiographs from the area where the implant is located. If it is seen on radiographs that the implant is compressing or in contact with the nerve, the implant should be removed (18). Immediate decision must be made, whatever the case, to prevent permanent nerve damage. Many authors recommend referral for injuries before four months, but this may be too late for many peripheral sensory nerve injuries (64). 3 months after injury; Persistent central and peripheral changes appear in the central nervous system that are unlikely to respond to surgical intervention (2, 20). Early removal of implants associated with IAN injury (less than 36 hours after injury) can help minimize or even resolve IAN neuropathy. In addition, corticosteroids and high-dose nonsteroidal anti-inflammatory drugs are effective in treatment (20).

Clinicians may also encounter situations where there are sensory changes but no nerve contact with the implant on radiographs. In this case, it can be suspected that nerve damage occurred during drilling. This should be considered

in cases where the implant is very close to but not in contact with the nerve canal. Although rarer, it should be noted that nerve damage may also occur during local anesthesia or aggressive release of the buccal flap (18, 25).

Medical treatment of nerve injuries is with the use of corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs). Administration of high-dose adrenocorticoids within the first week following injury has been shown to minimize the development of neuropathy (66). In addition, adrenocorticosteroids have been shown to inhibit axon sprouting centrally and ectopic discharges from injured axons and prevention of neuroma formation (67). If nerve damage occurs during surgery (such as nerve trunk transection or compression), topical application of dexamethasone is recommended.. One to two milliliters of the intravenous form of dexamethasone (4 mg/ml) can be administered topically for 1 to 2 minutes. Topical application of adrenocorticosteroids will reduce inflammation and reduce compression from edema, which may accelerate recovery from neurosensory disorders (29, 68). To control inflammatory reactions in the injured nerve, a course of oral steroids can be prescribed. Oral dexamethasone 4 mg 2 tablets AM for 3 days and 1 tablet AM for next 3 days or oral prednisolone 1 mg per kg per day (maximum 80 mg) can be indicated (29). An alternative would be a largedose of nonsteroidal anti-inflammatory drugs(eg, 800 mg ibuprofen) 3 times daily for 3 weeks. If the situation improves, the clinician can prescribe another course of anti-inflammatory drugs. In addition, vitamins and food supplements are believed to improve nerve health (4). Vitamin B supports nerve cell survival and remyelination (69). Montava et al in their animal study, showed that vitamin D increased functional recovery and myelination in injured nerves (70). Anti-epileptic and neuropathic pain drugs stabilizeneural membranes, which leads to suppression of hyperexcitabilityand reduction of neural discharges (71). Usually the first 2 senses to return are pain and temperature, and others may take longer to recover (72).

Cryotherapy should be applied extraorally to bone grafted and implanted areas, especially in areas where nerve injury is suspected. Ice should be applied to the paraneural tissues intensively for the first 24 hours, and then intermittently in the first week (73). It has been shown that cryotherapy reduces secondary nerve damage caused by edema-induced compression, decreases the metabolic degeneration rate of degenerating trigeminal ganglion cells, and slows potential neuroma formation. The application of ice compress to tissues after surgery has been shown to significantly improve recovering (29, 73).

Many patients respond well to this treatment sequence. Any improvement in the patient's condition and the results of the neurosensory examination should be recorded in the patient's own words. If the situation does not improve within 2 months, referral to a specialist should be made. Early referral will allow early management before permanent degeneration of the nerve (18). Examples of advanced microsurgery applications include removal of neuroma formation, external decompression by removing the affected bone, foreign material and soft tissue structures, neurorrhapy in which the transected nerve is anastomosed, and application of allogeneic or autogenous nerve grafts to provide nerve reconstruction (13, 25). Permanent damage usually occurs within 4-6 months after the nerve injury occurs. For this reason, it is very important to perform microsurgical procedures in the first months after the injury (72). Strauss et al concluded that 50% of patients who underwent microsurgical repair of IAN had significant improvement. In this study, 42.9% reported mild improvement and only 7.1% no improvement. In addition, after 1 year of microsurgical intervention, there was significant improvement (72). Generally, recovery of nerve function is assessed by symptoms of nerve damage. In the literature; It is seen that nerve injuries due to trauma, local anesthetic and endodontic reasons are usually temporary and heal over time(4, 36, 74). While it has been reported that there is an improvement in hypoesthesia as high as 80%, there is little improvement in neuropathic pain(4). Once neuropathic pain begins, it seems difficult to improve the patient's condition with late surgical intervention (eg, surgical trigeminal nerve repair) (4, 15). Pollitis et al. also found that the diagnosis of "neuropathic pain" was often not made on time in the records they reviewed; They say that reaching this diagnosis usually takes time. Abgaje et al. reported in their study that one-third of patients with injuries to the branches of the trigeminal nerve never fully recovered (4). They also concluded that it is actually difficult for patients to communicate unfamiliar symptoms and for surgeons to view neuropathic pain as a working diagnosis (15). Clinicians should be aware of symptoms and should not forget the power of early treatment. The clinician should be self-aware and refer to a specialist when there are no early signs of recovery. Doing so will give patients the best chance of restoring beneficial sensory function. It should be noted that Seddon said, "If a purely anticipatory policy is followed, the optimal time for operative intervention will always be missed (75).

7. Conclusion

IAN injuries are a very difficult situation for both the patient and the clinician and should be avoided. It can occur for many reasons, but in most cases, it can be prevented by appropriate patient selection, proper planning, and proper understanding of the anatomy of the area, surgical procedures, and implant systems. Once it occurs, the appropriate treatment protocol should be followed, starting with the psychological treatment of the patient. It should be remembered that when nerve damage occurs, early and appropriate treatment is the most important key to recovery.

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CHAPTER XIII

COMPLICATIONS IN PERIODONTAL FLAP SURGERY

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1. Introduction

Periodontal diseases caused by dental plaque are inflammatory diseases that are formed by specific microorganisms or specific groups of microorganisms in the tissues supporting the tooth, and are observed as a result of the effect of tooth support tissues at various levels. (1)

Periodontal disease is characterized by the formation of an inflammatory response in the supporting bone and connective tissue against the microbial dental plaque, and the nature of the resulting inflammatory response determines the course of periodontal disease. (2,3) The development of periodontal disease largely depends on the virulence factors of microorganisms and their concentrations in the periodontal pocket. As a result of the interactions of microorganisms living in the biofilm with each other, their virulence characteristics increase and antimicrobial agents can gain resistance against host defense responses. (4) Tissue damage and loss occur as a result of gram-negative anaerobic or facultative bacteria in the microbial dental plaque, which is the main etiological factor in the formation of the disease, and the response of the host defense system to these microorganisms and their products. (5)

Gingivitis is an inflammatory gingival disease that can be localized or generalized, developing following dental plaque accumulation, characterized by erythema, edema, loss of gingival surface roughness and bleeding. (6)

Gingivitis is the mildest form of gingival inflammation and periodontitis is the advanced stage of gingivitis. (7-10) Periodontitis is a chronic, infectious disease caused by bacteria and their products in dental plaque, characterized by

inflammatory destruction of the tooth-supporting periodontal ligament, alveolar bone and soft tissues. (2) Their treatment comprises of non-surgical and surgical approach provided depending on the severity of the disease. (11)

As a result of non-surgical periodontal treatment, it is aimed to control the microbial periodontal infection by periodically cleaning the bacterial biofilm, dental calculus and bacterial toxins on the root surface. (12)

Flap operations have been developed as a result of new searches for the treatment of cases with advanced periodontal disease. (13)

In this section, the complications that may occur during and after periodontal flap surgery to obtain a healthy periodontal tissue, and the strategies to eliminate these complications, and more importantly, the rules to be followed in order to minimize or prevent them are discussed.

2. Periodontal Flap Operation*s*

2.1. What is a Periodontal Flap?

Periodontal flap is the part of the gingival and oral mucosa that is separated from the teeth and alveolar bone with the help of horizontal and/or vertical incisions and is in contact with the alveolar mucosa in at least one area. (14)

The periodontal flap operation is a basic periodontal surgical procedure that is utilized to eliminate periodontal pockets, to allow access for the treatment of osseous defects, to treat mucogingival problems, and to treat mucogingival problems, and to treat combinations of the above. (15)

A periodontal flap is a section of gingiva, mucosa, or both that is surgically separated from the underlying tissues to provide for the visibility of and access to the bone and root surface. The flap also allows the gingiva to be displaced to a different location in patients with mucogingival involvement. (16)

Periodontal flaps are removed to see the root surfaces of the bone and tooth, to increase accessibility or to change the position of the gingiva (in the presence of mucogingival problems). (13)

Flaps may be classified as full thickness or partial thickness. The decision of which to utilize is dependent upon the periodontal problem, the anatomical relationship between the tooth, alveolus and gingival tissues, and an understanding of the healing of both types of flaps. (15)

In a full-thickness flap, all of the soft tissue, including the periosteum, is reflected to expose the underlying bone. This complete exposure of and access to the underlying bone is indicated when resective osseous surgery is

contemplated.The partial-thickness flap includes only the epithelium and a layer of the underlying connective tissue. The bone remains covered by a layer of connective tissue that includes the periosteum. This type of flap is also called the split-thickness flap. The partial-thickness flap is indicated when the flap is to be positioned apically or when the operator does not want to expose bone. (1***6)***

2.2. Indications of Flap Operations

It is a technique applied mainly for the elimination of real pockets. It performs the reduction and/or elimination of pathological pockets. It provides access to root surfaces and thus removal of local irritants and visualization and treatment of alveolar bone margins and defects. It allows for more accurate prognosis estimation. Regenerative periodontal procedures such as bone grafting and guided tissue regeneration require flap removal. It is also used in implant surgery. In addition, in the presence of mucoingival problems, the flap is removed in surgeries performed to cover the root surfaces. It provides the appropriate periodontal environment for restorative dentistry practices. (crown upgrades and similar transactions) Reducing the pocket depth is important in facilitating the patient's plaque elimination.

Flap operation facilitates accessibility for oral hygiene and supportive periodontal treatments. It allows the formation of gingival morphology, which facilitates plaque control and enables the patient to perform it. It enables periodontal instruments to reach inflamed areas more easily. It provides regeneration and reattachment (reattachment) of new tissues.It protects periodontal support.Trauma, pain, arm tenderness and aesthetic shock are minimal after periodontal surgery.Form in dentistry means function. Flap operation will reveal the gingiva with better form and function.

Local factors and inflamed tissues eliminated as a result of flap operation,high sedimentation, leukocyte increase, bruxism, It will be able to relieve back, neck and chewing muscles pain, and enable them to wake up more vigorously and vigorously. (1***3)***

2.2. Contraindications of Flap Operations

1. Poor oral hygiene: It is contraindicated to perform flap surgery for patients who do not brush their teeth 3 times a day, who cannot use dental floss, super-flos, or interdental brushes. Patients with inadequate oral hygiene should be included in a palliative program such as six-month calculus removal.

2. Flap operation and similar surgical interventions should not be applied to pregnant women.
3. Patients with malignant tumor cases and cerebrovascular attack or patients with limited life expectancy,
4. Patients with hemorrhage problems (leukemia, thrombocytopenia, hemophilia and anticoagulant drug use)
5. Uncontrolled diabetes,
6. Patients consuming more than 50 g of sucrose or more than 100 g of glucose per day, (See: Nutrition and periodontal disease)
7. Chronic alcoholism, cirrhosis,
8. Cigarette consumption: Delayed wound healing and various post-operative problems are encountered in those who smoke 1 pack or more. Smoking style makes the treatment unsuccessful depending on the patient's body structure.
9. Low resistance of the patient to infections,
10. Presence of sensitivity to local anesthetic agents and antibiotics to be used for the operation
11. The patient's psychological state is not suitable
12. Patients under severe mental stress
13. Malnutrition, bulimia,
14. Patients with chemotherapy and radiotherapy,
15. Patients with hyperparathyroidism, cardiovascular and cerebrovascular problems,
16. It is contraindicated in patients using corticosteroids, Addisson's disease, Multiplesclerosis, Parkinson's disease. (1***3)***

2.3. Anatomical Structures

The anatomical features of the region where flap operation will be performed should be considered. The areas that require the most attention are foramen incisivum, foramen palatinum majus, foramen infraorbitale and tuber maxilla in the maxilla. In the mandible, there are Linea oblique, foramen mentaleler, fovea submandibularis and fovea sublingualis.

The foramen mandible is the area that should be considered the most and the dentist will act most carefully throughout his life. Muscle and mucous adhesions of the alveolar bone, maxilla and mandible, and the area and features of the gingiva will affect the duration and course of the flap operation.

Before the flap operation, the dentist has to consider the neighborhood of the maxilla and mandible, the vestibule and lingual regions, muscle adhesions and morphological changes with light palpation without disturbing the patient. (13, 17)

2.4. Application of Flap Operation

The first and most important rule of good surgery is to keep the living tissue alive and to conclude the surgery with minimum trauma.

Instruments to be used elegantly in surgical intervention will help the tissue wound, which is composed of many cells, to fully heal. The ruptured tissue loses its vitality and becomes necrotic. This condition causes infection and delays healing. In order to keep the trauma to a minimum, the surgical operation should be planned very well. Different flap techniques and shapes to be used in periodontal surgery form the basis of general practice. Three principles should be considered while performing flap operations.

1. The flap forms the basis of blood circulation.
2. The flap should allow the working area to be seen easily.
3. The flap should be able to cover the surgical area while closing after the operation. (13)

The operative site is anesthetized with local anesthetic. (15) A periodontal flap may be elevated in full thickness or partial thickness. Bir periodontal flep tam kalınlıkta veya kısmi kalınlıkta kaldırılabilir. (18)

2.4.1. Full Thickness Flap

After probing and sounding to determine the pocket depth and morphology of the defects, an inverse beveled incision is made about 1 milimeter apical to the free gingival margin, with the blade of the scalpel directed toward the alveolar crest. The incision follows the form of the gingival margin and the result should be a carefully scalloped incision. If relaxing incisions are to be made to enhance Access and visibility, they are made at the mesial and distal boundaries and should be made at the mesial or distal aspects of the teeth. A relaxing incision should not be made over radicular bone, since this would create unnecessary trauma to the labile radicular bone. (15)

In a full thickness flap, all of the soft tissue, including the periosteum, is reflected to expose the underlying bone. This complete exposure of and access

to the underlying bone are indicated when resective or regenrative osseous surgery is contemplated. This type of flap is also called the mucoperiosteal flap. Elevation of a full thickness flap requires the internal bevel incision to penetrate the periosteum, the last tissue overlying the bone. Once the periosteum has been completely incised along the length of the flap, a full-thickness flap is reflected by elevating the periosteum off the bone via blunt dissection. (18)

2.4.2. Partial Thickness Flep

The partial-thickness flap includes only the epitehelium and a layer of the underlying connective tissue. The bone remains covered by a layer of connective tissue that includes the periosteum. This type of flap is also called the split-thickness flap. The partial thickness flap is indicated when the flap is to be positioned apically or when exposure of bone is not desired. Elevation of a partial-thickness flap is completed by sharp dissection with a surgical scalpel.

The partial thickness flap is elevated by sharp disection. Depending upon the thickness of keratinized tissue present, the initial incision is made either in the sulcus or just apical to the sulcus with the scalpel held at a steep angle so as to create an internal bevel. Relaxing incisions may be made and the flap is very carefully disected and elevated, taking great care not to perforate the flap. It is frequently possible to begin the disection at the corner of the flap at the area of the relaxing incision and it is sometimes possible to insert the blade into the mucosa through the relaxing incision and carefully disect in a coronal direction. (18)

2.4.3. Goals in Incision Techniques

1. Preservation of attached gingiva.
2. Absence of gingival in the form of steps after flap operation, sticking of gingival to the tooth surface with a thickness close to the blade edge by thinning the gingiva.
3. Narrow attached gingiva Using the sulcular incision technique in the anterior region.
4. While performing incisions in the anterior region, applying the papilla-sparing incision technique with the help of 11, 12 and 15 numbered scalpels.
5. Vertical incisions should be avoided. If a vertical incision is required, it should be used 1-2 mm mesial or distal to the apex of the interdental

papilla. If vertical incisions to the palatal region are required, they should be parallel to the marginal gingiva.

6. Incisions should be planned so that the gingiva covers the alveolar bone in general. Resorption and pain in the exposed alveolar bone are very high.
7. Generally, full thickness flap incision is preferred. In cases where the alveolar bone of the gingiva is thin, split thickness flap operation may be preferred for flap shifts. The flap thickness after incision should not be less than 1.5 mm. Necrosis may occur in flaps thinner than 1.5 mm.
8. Incisions to be made in the palatal region should cover the alveolar bone. The open alveolar bone can go into necrosis.
9. Scalpels should be sharp and should be changed immediately as they become dull. Blind scalpels cause unnecessary tissue damage. After starting the incision, it should be continued until it is completed; The scalpel should not be inserted and removed frequently to create an irregular appearance. Smooth incisions heal quickly and the patient feels less discomfort. It should not leave dead space in the incisions.
10. Attention should be paid to the base of the flap, the incision should be planned to ensure blood flow, and the post-operative volume of the flap should be considered. Flap incisions should not create tension on the flap. Liberating incisions can be used to mobilize the flap.
11. The lingual flap in the mandible is thin and fragile.Vertical incisions should be avoided. Vertical incisions in the lingual region a. Since it is related to the sublingual chamber, the infection spreads easily and painful conditions may occur. b. Vertical incision in the second molar region will allow the infection to spread to the parapharyngeal chamber; It can cause severe pain, abscesses, swallowing difficulties and bleeding. c. Vertical incisions cause lingual gaps between the bone and the flap. (13)

2.4.4. Removal of the Flap

Flap lifting should be done as needed, as inflamed tissues are eliminated, flap lifting should be increased as more vision is needed; Care should be taken to never exceed 10-15 mm.

The more it is exposed that the alveolar bone is fed from the flap, the more bone biology can be affected, the longer the full thickness or half thickness is removed, the more bleeding and blood loss will be, the more anesthetic solution It should be taken into account that more pain and infection may occur in the post-operative period.

After removing the flap, if there are purplish colored inflammatory granulation tissues on the inner surface of the gingiva, it is tried to be cleaned with a small scissors. It should be shown not to pierce and tear the gums during the procedure. (13)

2.4.5. Subgingival Curettage

After the flap is removed, the subgingival region is curetted with various crepitus and curettes. The most important issues regarding curettage are:

a. Not tearing the gingiva,
b. Avoiding subgingival attachments (dental calculus, bone fragments, sclerotic tissues) to the palatal, vestibular and lingual apical regions,
c. Complete elimination of inflamed tissues and hard appendages in the subgingival region,
d. Using treatment tools starting from the thickest cusps and curettes to the thinnest curettes.

Several blows with curettes on the bone surface Bone curettage is performed by striking.After root straightening and bone curettage are finished, The area is irrigated with physiological saline with the help of a 20 gauge injector, and it is visually checked again. (13)

2.4.6. Closure of the Flap and Suturing

The use of appropriate techniques and materials in the closure of the flap after periodontal surgical procedures allows the flaps to be fixed in the desired position, to achieve primary healing, to shrink the wound surface, to control bleeding, and to increase patient comfort. (19)

After the buccal, palatal and lingual flaps are aligned, gentle pressure is applied with sponges on the flaps to ensure that the appropriate amount of clot is formed between the soft tissue and bone. Especially when procedures such as GDR are applied, there should be no tension in the tissues when the flap is sutured. (13)

It should not be forgotten that suture threads used after surgical procedures may cause tissue reaction or susceptibility to infection as well as contributing to wound healing. Passing the suture through the soft tissue and the presence of foreign material may increase the susceptibility to infection. Knowing the properties of the material used, choosing it according to the type of operation

performed, and using the right suture technique are extremely important in terms of reducing complications and increasing surgical success. In the selection of the suture material, the type and thickness of the tissue, its location in the mouth, the structural properties of the material, the ease of use, the price and how long it is planned to remove the suture are important. (20)

The thickness of the suture materials varies according to the purpose of the operation. In cases where thick gingival tissues are present, 3-0 silk sutures can be used. Generally, the standard suture of flap operations is 4-0 silk suture. The important feature to keep in mind in periodontal surgery is that the sutures used in the oral mucosa should not be thicker than 5-0.

While suturing, the needle is held with a porthole near the center of the body near the junction with a few millimeters of thread. However, if it is kept too close to the tip, it causes bending at the needle tip, and if it is kept close to the connection point with the thread, it causes bending in the needle body. (13)

Generally, suturing is started from the distal of the last tooth. The needle tip should be sharp, if it is dull, it should be replaced. While passing the needle through the tissue, force should be applied to the needle in the direction of its curvature. While suturing, the needle is first passed through the most mobile tissue flap.

Since edema can be seen in the first 48 hours of wound healing, the needle should be passed through the tissue at least 2-3 mm away from the wound edges. If this distance is less than 2-3 mm, the tension in the tissue will increase as a result of edema and the thread may cause the tissue to rupture and impair healing.

While pulling the needle out of the tissue, holding and pulling the tip with the portegu will damage the needle tip, this should be avoided. More than the tip should be taken out of the tissue by pushing the back part of the needle in accordance with its curvature as much as possible. In order for this to be possible, the size of the needle should be chosen according to the area to be sewn, short needles should not be tried to be passed from a long distance. (21)

While suturing, care should be taken to ensure that the sutures are tight enough to provide sufficient tension without leaving dead spaces between the wound edges, but loose enough not to cause tissue ischemia or necrosis. If the wound edges are not fixed by bringing them together, or if the sutures do not apply sufficient tension to the flap edges, bleeding may continue, causing blood and serum to accumulate under the flap, moving the flap away from the bone, and ultimately delaying healing. (19)

2.4.6. Precautions to be Taken After Periodontal Surgery and Explaining to the Patient

1. Put ice in the nylon bag and wrap it with a thin towel. It is said that it should be applied to the operation area of your face for 5 minutes and removed for 5 minutes. If the nylon bag is applied without being wrapped in a cloth or towel; bare ice burns the face. If it is applied continuously, paresthesia occurs. The ice pack can be applied until the patient goes to bed in the evening.
   Ice application
   a. Decreases bleeding
   b. It reduces the feeling of pain
   c. It prevents or reduces swelling in the cheek area
2. The patient should not drive after the operation. Local anesthetic solutions reduced reflexes. The patient should avoid walking; He should go home and rest. If the patient will go down the stairs after the operation, it is appropriate to be accompanied by a person.
3. After the operation, hard and hot foods should be avoided.
4. The patient can use painkillers and antibiotics depending on the doctor's prescription. However, he should not use aspirin or painkillers that contain analgesics.
5. He should not take a bath for 24 hours after the surgical procedure. After 1 day, the body can be washed with warm water. In order to wash the head with warm water, it is necessary to wait for the slight swelling on the face to completely disappear. Washing the head with hot water or taking a shower can cause bleeding or facial swelling. The patient should not pull his cheeks and lips in front of the mirror to see the operation area.
6. The teeth in the area where the operation was not performed can be brushed.
7. The patient should be told that a drop of blood turns a glass of water red.
8. After the operation, the patient's temperature of 1-1.5 °C may increase, and if it exceeds 38°C, the doctor should be informed. (13)

3. Complications in Periodontal Flap Surgery

Often these non-surgical and surgical approaches yield positive results without any downsides, but in a few cases they can lead to certain complications that can alter these predictable results. Mostly these non-surgical and surgical approaches result in favorable outcome without any untoward events but in few cases, they

might lead to certain complications that could alter these predictable outcomes. (22)

Complications occurring after periodontal surgery include postoperative pain, bleeding, swelling, root hypersensitivity, delayed healing, trismus, bruising, taste changes. These complications can alter the outcome of periodontal therapy. Therefore, for a clinician it becomes mandatory to acknowledge their etiology and management in order to achieve successful periodontal therapy. (23)

The following complications may be encountered after periodontal surgery. (13)

1. Shock
2. Bleeding (Hemorrhage)
3. Pain
4. Swelling and hematoma
5. Sensitivity to the neck (hypersensitivity in the root)
6. Increased tooth mobility
7. Post-operative bacteremia
8. Delays in wound healing
9. Trismus
10. Taste Changes

3.1. Shock

It can generally develop against local anesthetic solutions. The patient may experience restlessness, difficulty in breathing, nausea, pale face and a picture that turns into cyanosis. The patient is immediately placed in the Tradelenburg position and oxygen is administered. If the patient's blood pressure is low and his heartbeat is weak, necessary emergency procedures are initiated. If the patients who usually come without eating, add 2 cubes of sugar to 1 glass of water and mix, the state of fainting can pass. The patient's tie should be loosened and the shirt collar should be opened. The patient's pale face should remain in the tradelenburg position until it regains its normal color. If the patient does not improve despite all the applications, help should be sought from the emergency department. (13)

3.2. Bleeding (Hemorrhage)

Bleeding problem is encountered from time to time during the operation. In some cases, the physician may encounter bleeding even though the patient's blood findings are normal. If a tendency to bleeding is felt in the first incision at

the beginning of the operation, the operation area should be narrowed and the operation should be planned to include 3-4 teeth (mini flap).

If bleeding is encountered after flap removal, the bleeding site is determined by applying pressure beforehand. Applying pressure (with a gas pad or index finger) and suturing around the vessels leading to the bleeding site prevents bleeding. (13)

Post operative bleeding may be present immediately (primary hemorrhage),within 24hrs or as delayed post operative bleeding (reactionary hemorrhage). It can be due to slippage of suture, dislodgement of clots, cessation of reflex vasos pasm, normalization of blood pressure.Bleeding after 7-14 days is secondary to trauma or surgery. (26) In these cases patient should be examined for the causative factors such. (24)

The likelihood of this may be attributed to many factors, like the

- tissues of mouth and jaw are highly vascular
- infection
- intrinsic trauma
- presence of foreign bodies
- Even after repeated instructions patients tend to play with the area of surgery with their tongue and dislodge the blood clot, which initiates secondary bleeding.
- The tongue may also cause suction of blood by creating small negative pressures that cause secondary bleeding.
- salivary enzymes may lyse the blood clot before it gets organized. (25)

For the management of bleeding it is very important to find the source of bleeding and then the approach for its management should be planned. In case of mild bleeding a pressure pack can be applied for 15- 20 minutes. Still if bleeding is persistent then haemostatic agents like surgicel, gelfoam, microfibrillar collagen (Avitene) etc. can be used. If the bleeding is arterial, then ligating the vessel is considered as the best option. (24)

If bleeding is due to residual granulation tissue or liver clot type then it should be removed by high speed suction or currettage. Bony bleeding can be controlled by crushing the bone with appropriate instrument. (26)

Soft tissue bleeding may be treated by clamping it with hemostat, if it still persists vessel ligation with sutures, laser coagulation or electrocautery may be necessary. (24)

3.2. Pain

Periodontal surgery t should produce only minimal pain and discomfort. A study of 304 consecutive periodontal surgical interventions revealed that 51.3 % of the patients reported minimal or no postoperative pain, and only 4.6 % reported severe pain. Of these, only 20.1 % took five or more doses of analgesic. (27)

In general, pain begins 1.5-2 hours after the flap and gingivectomy operation, after the effect of local anesthetic substances has passed. (13)

Eighty percent of patients experience acute pain after surgery; of these patients, 86% experience moderate, severe, or extreme pain.Postoperative pain experienced within the first 3 days after surgery is considered normal and should progressively diminish throughout the healing phase. (24)

After the operation, the painkillers given to the patient will relax the patient. Rarely, it may be necessary to resort to pain medications for 24 hours. (13)

For relieving pain initially certain medications like nonsteroidal anti-inflammatory drugs (NSAIDs), such as diclofenac (1 mg/kg) and ibuprofen, paracetamol (15 mg/kg) can be prescribed. (24)

Postoperative pain can occur as a result of extensive and long surgical procedures; poor tissue handling (including incising with a dull instrument, tissue trauma, and poor local anaesthesia); poor infection control (which increases the risk of postoperative infection); poor knowledge of surgical anatomy (which increases the risk of complications, such as nerve injury and edema) ;patients who underwent the procedures that involved mucogingival/ bone or surgeries with large wounds; Patients whose healing process might be delayed (e.g. immunosuppressed people, those with uncontrolled diabetes, smokers, those taking bisphosphonates, those with a history of radiotherapy in the head and neck area) ;patients with a past history of high analgesic intake after periodontal surgery ;patients experiencing preoperative anxiety. (24)

If infection, mild fever and lymphadenopathy are associated with the pain, it may be necessary to put the patient under antibiotic pressure. The duration of antibiotic use may be 5-7 days, and 14 days if osteomyelitis is suspected. (13)

3.4. Swelling and Hematoma

Excessive work on soft tissues and bones may cause hematoma or swelling. (13) The swelling becomes apparent after the day following surgery and will reach its maximum within 2-3 days post-operatively. (24)

Type of the incision, its extension, tissue manipulation and duration of surgery are some factors that can affect swelling. Smaller incisions usually cause less postoperative swelling and pain. (26)

Ice application, usually wrapped in a thin towel, prevents hematoma and swelling. If the patient has lymphodenopathy swelling and pain, it may be necessary to put under antibiotic pressure for 7-14 days. It usually occurs as a result of abduction of bone particles into the apical region of the flap while performing bone correction after the flap has been lifted. Swelling in the upper jaw and abscess formations in the lower jaw that can progress to difficulty in swallowing may be encountered in the lingual region. Abscess events antibiotic administration; If necessary, it can be controlled by performing abscess drainage. (13)

Antibiotic as prophylaxis therapy to prevent distant site infection or to control postoperative sequalae or to treat an established infection in periodontal surgery is a well accepted indication with proved efficacy. (26)

Corticosteroids reduces inflammation, fluid transudation and edema. Various surgical strategies like piezosurgery have also shown to minimise discomfort after the periodontal sur geries. Beneficial effects of ice applied on a surgical wound are due to changes of blood flow which causes vasoconstriction and reduced metabolism thus reducing bacterial growth. (26)

3.5. Sensitivity to the Neck (Hypersensitivity in the Root)

Dentin or root hypersensitivity is a relatively common problem in periodontal practice. It may occur spontaneously when the root becomes exposed as a result of gingival recession or packet formation, or it may appear after scaling and root planning and other periodontal surgical procedures. (27)

Post-operative recession of soft tissue further exposes the dentinal tubules. Patient inability to maintain plaque control in the healing phase further complicates the problem. (24)

Though the sensitivity decreases around 2 weeks but if it doesn't then it is recommended to use desensitizing agents like sodium fluoride, stannous fluoride, calcium sodium phosphosilicate bioactive glass (NovaMin®); resins, varnishes, toothpastes (occlusion of dentinal tubules); iontophoresis, lasers and gingival grafts. (24)

3.6. Increased Tooth Mobility

Tooth mobility usually increases immediately after surgery. This results from edema in the periodontal ligament space from the inflmmation that occurs

postsurgically. The mobility diminishes to the pretreatment level by the fourth week. The patient should be reassured before surgery that the mobility is temporary. (27)

After 30- 45 days if mobility persists then the etiological factor for mobility should be identified and corrected through occlusal adjustment and finally splinting should be done to stabilize the teeth. Although if the mobility is still progressive then extraction can be considered as an option. (28)

3.7. Post-operative Bacteremia

There is huge microbial challenge to the patient during periodontal surgery. The occurrence of post-surgical bacteraemia depends on amount of trauma imposed during surgery. (24, 26)

It is documented that 88% of all blood cultures are positive after periodontal therapy. Okel and Elliot in their study found Staphylococcus albuscoagulase negative as most common pathogens involved in postoperative bacteremia. Similarly, Mc Entegart and Porterfield in their study concluded Staphylococcus albus as the most frequently isolated micro-organism occurring six times whereas Psedomonas aerugenosa, Streptococcus viridans, Alpha hemolytic streptococcus occurring more than once and Neisseria catarrhalis, the least isolated, occurring only once in postoperative infection after periodontal surgery. (24)

Transient bacteremia can be effectively treated by giving antibiotic prophylaxis before surgery. Amoxicillin is considered to be highly effective in reducing post-operative bacteremia in periodontal flap surgery as well as in preventing the possible sequelae (infective endocarditis and other systemic maladies) insusceptible patient. (24)

The other antibiotics prescribed post surgically included amoxicillin-clavulanate (3.1%), azithromycin, ciprofloxacin, metronidazole, and trimethoprim sulfamethoxazole (each <1%). (24)

Antiseptic mouthwashes are widely used before routine dental treatment, especially as a standard pre-operative measure (29) and have an important role in reducing the number of microorganisms in the oral cavity. A recent systematic review showed moderate evidence that mouth rinsing with antiseptics before the procedure can reduce dental-derived aerosolization of live microbes. (30)

3.8. Delays in Wound Healing

Wound healing, as a normal biological process in the human body, is achieved through four precisely and highly programmed phases: haemostasis,

inflammation, proliferation, and remodelling. By 7 days surface epithelisation gets completed following periodontal surgery. (23)

The flap operation is a reattachment (reconnection) operation. Wound healing in full thickness flap operation begins with clot formation between 0-24 hours after the flap is closed and sutures are applied. The clot contains fibrin reticulum, polymorphonuclear leukocytes, and capillaries. It has been stated that the clot is effective in stabilizing the flap. The clot starts to resorb after 3-4 days. Clot resorption ends on the 6th-7th days. 12 days after the flap operation, the flap reattached to the tooth (reattachment) and the periosteal vascular structure began to function. In the periodontal ligament part of the alveolar bone, resorption, which starts in 3-4 days, reaches its highest level in 8-10 days and starts to decrease after 10-12 days. Antibiotics do not affect reattachment. 14 days after the operation, the collagen fibers are parallel to the tooth surface and the flap area appears clinically normal, although the association of the flap with the tooth is weak due to immature collagen fibers. Wound healing in the third week conforms to the general histological picture. Regenerating connective tissue thickens and covers the alveolar crest, and the periosteal surface shows osteoblastic and minimal osteoclastic activity. In the fourth and fifth weeks, wound healing is completed with the repair (regeneration) and maturation of all tissues. The gingival pocket has a fully epithelialized epithelial attachment. From the 2nd to the 3rd month of wound healing, the flap adheres firmly to the tooth and cementum, and apposition begins in the bone. After flap operation and curettage of the defect in intraosseous defects the bone can regenerate and fill the defect.If the alveolar bone is not covered by the gingiva during the flap operation, significant bone resorptions occur on the open bone surface. (13)

The most probable cause of delayed wound healing is infection which results in dead necrotic tissue which promotes bacterial growth. Other causes include wound dehiscence (unapproximated flap margins), hematoma, Stitch abscess (infection of suture track), foreign substances (like calculus, tooth fragments, periodontal pack), allergic reactions to graft material, suture material, periodontal pack, tight closure via suturing. Thorough debridement and irrigation followed by prescription of antibiotics and analgesics usually lowers down the symptoms and accentuates wound healing. (23)

3.9. Trismus

Trismus is an inability to open the mouth.Treatment of trismus varies depending on the aetiological factor. (29)

Trismus after periodontal surgery can occur due to trauma, infection, infection of masticatory space, inaccurate positioning of needle. The degree of discomfort and dysfunction varies, but is usually mild when it is due to incorrect positioning of needle in superior alveolar or inferior alveolar nerve block. (29)

For reducing it heat therapy, soft diet and muscle relaxants can be used. (31)

Aspirin because of its antiinflammatory properties is beneficial and given in managing the pain associated with trismus. (29) If the pain is intense then analgesics can be given. If required, diazepam (2.5–5 mg three times daily) and other benzodiazepines may be given for muscle relaxation. (31)

3.10. Taste Changes

Taste change is also one of the complication after periodontal surgery. It can be due to any infection, trauma to any nerve, invasive procedures or idiopathic. It can also be due to any surgery requiring insertion of a periosteal elevator, section ing of tooth, lingual flaps etc. (26)

Nerve damage has also been linked to the experience of the operator and procedures performed under various forms of sedation. It can also be associated with the use of local anes thetic. Sometimes needle directly contact the tissues and can traumatizes the nerve which can alter the sensation. Damage to smaller intraneural blood vessels can cause intraneural he matoma. Healing process can also be impede by compres sion of the nerve. (26)

Taste change could be described in the terms of dysgeusia: disgusting oral taste or altered taste sensation; Hypogeusia: reduction in all 4 taste modalities i.e. sweet, salty, sour and bitter; Ageusia: no taste sensation is perceived; Phantogeusia: spontaneous, continuously altered, often metallic taste which is usually drug related. (24)

Management includes administration of zinc (gluconate or sulfate) as it plays an important role in the regeneration of taste bud cells. Taste function is also affected by amount of saliva. (24)

Matsuo and Yamamoto in their study showed a significant association between saliva and taste. Thus, low salivary flow may also alter taste, which require the use of a sialogogue. (pilocarpine -30mg/day). (24)

Repair of nerve damage can also be done to manage taste disturbances. In a review, Ziccardi and Steinberg found that trigeminal nerve microsurgery was one treatment modal ity option for patients with nerve injury. The articles

reviewed suggested that injuries should be repaired within the first 90 days to increase the chances of improvement. (26)

4. Conclusion

Successful Periodontal therapy is necessary for providing better dental care. For which the selection of the most suitable technique for treatment, evaluation of the complications associated with it is considered crucial for paving way towards favourable outcomes.

As any periodontal surgery can be followed by occurrence of these complications, a thorough knowledge of their etiology and management is required to achieve maximum results with reduced patient discomfort.

Periodontal flap surgery is the most widely used surgical procedure to reduce the pocket depth. Periodontal flaps are designed to preserve gingival integrity and to gain access to root surfaces for residual calculus removal and to thoroughly remove granulation tissue so that bone defects can be visualized and treated. Gentle and efficient procedures result in optimum healing and minimal postoperative pai**n.**

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CHAPTER XIV

IMPORTANCE OF CANALIS SINOUSUS AS A RARE ANATOMICAL STRUCTURE IN DENTAL IMPLANT SURGERY

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1. Introduction

Canalis sinuosus was first described by Frederic Wood Jones in 1939 as a bony canal with a diameter of approximately 2 mm, which separates from the infraorbital nerve at the posterior part of the infraorbital foramen, runs alongside the nasal cavity, and passes through nerves and blood vessels. This canal, called the CS because of its double arcuate course, runs forward and downward on the inferior wall of the orbit, lateral to the infraorbital canal. It then passes under the infraorbital foramen and curves medially towards the anterior wall of the maxillary sinus. After reaching the edge of the anterior nasal apertura in front of the anterior end of the inferior concha, it follows the lower edge of the nasal apertura and ends lateral to the nasal septum in front of the incisive canal. (1) This canal, which has a course of approximately 55 mm in the maxilla, carries the anterior superior alveolar nerve and the vessels of the same name. (2)

It is important to be aware of neurovascular structures and variations in the planning of surgical operations in order to avoid complications. Many surgical operations (endodontic surgery, extraction of impacted teeth, cyst treatment, orthognathic surgery, implant placement) are commonly performed in the anterior region of the maxilla. (3) While vascular damage that may occur during surgical operations creates a risk of bleeding, nerve damage can significantly affect the patient's quality of life due to hyperesthesia, paresthesia or pain. (4,5)

In addition, there are some researches reporting that the contact of the implant with the neural tissue during the placement of dental implants in the jaws may lead to failure in osseointegration. (6,7)

Therefore, the anatomical variations in this region should be well known as well as the major neurovascular structures in the anterior maxilla. One of the anatomical structures that have not been known sufficiently in this region is canalis sinuosus. Although the CS is a normal anatomical formation, the accessory channels continuing in the anterior maxilla are not well known. These structures may not be adequately visualized on two-dimensional radiographs. These canals may be superposed on tooth roots, mimicking periapical inflammation or root resorption causing the physician to misdiagnose. (8,9,10,11)

In the literature, there are also case reports of traumatic neuromas originating from the anterior superior alveolar nerve and pain after implant applications in contact with the CS. (12,13,14,15,16)

The terminal portion of the CS ends at the anterior maxilla and may terminate in the alveolar bone in the region of the canine teeth. In addition, this canal is surrounded by a thin bone, which exposes the anterior superior alveolar nerve within it to trauma. Therefore, it is important for clinicians to know the presence of accessory canals associated with this canal in the CS and anterior maxilla for accurate diagnosis and treatment planning.

2. Imaging Methods of Canalis Sinuosus

Radiological evaluation is an important diagnostic method in dentistry. Appropriate imaging method should be selected in order to diagnose, treat and follow up dental and maxillofacial diseases wel*l.*

2.1 Periapical radiography

Periapical radiographs are used in many cases, such as in the diagnosis of apical infections, evaluation of periodontal tissues, dental traumas, examination of impacted teeth and root morphology, during endodontic treatment, determination of alveolar bone lesions, and evaluation of deeply carious teeth. (17)

In case reports in which well-defined radiolucent areas suggesting resorption on the root surface were observed on periapical radiographs taken from the anterior region of the maxilla, researchers observed that radiolucent areas were displaced in radiographs repeated with different horizontal angles. When CBCT imaging was performed in these cases, external root resorption was not observed in the roots as suspected. Neurovascular bone channels, which are thought to be

the terminal part of the CS in the anterior maxilla, were observed on the palatal side of these teeth. These canals, running palatal to the roots, were superposed to the root surface on two-dimensional periapical radiographs, forming radiolucent areas on the root surface that aroused suspicion of resorption. (9,10,11)

In the case report presented by Leven and Sood, the diameter of the neurovascular bone canal, which runs palatal to the right maxillary lateral incisor and mimics external root resorption on periapical radiograph, was found to be approximately 1.5 mm. (9) Insufficient examination of superpositions, incomplete knowledge of anatomical structures such as CS and their variations may lead to incorrect diagnosis and treatment planning. Shelley et al. presented a case of canalis sinuosus, manifested as a periapical radiolucency on an maxillary canine in a periapical radiograph. (***8)***

2.2 Panoramic Radiography

In the panoramic radiography technique, a tomographic image is created that selectively displays a certain part of the body part. Scarfe et al. evaluated the appearance of the infraorbital canal and anterior dental plexus on 246 panoramic radiographs.(18) They followed the infraorbital canal in 81.3% of 246 panoramic radiographs. They also mentioned a subtle but distinct radiographic feature consisting of a single or a series of linear relative radiolucency in close association with the infraorbital canal, which can only be discerned with high-intensity illumination. Researchers defined this radiolucent structure as the anterior dental plexu***s.***

2.3. Cone Beam Computed Tomography

Cone-beam computerized tomography (CBCT) is a medical image acquisition technique based on a cone-shaped X-ray beam centered on a two-dimensional (2D) detector. The source-detector system performs one rotation around the object producing a series of 2D images. The images are reconstructed in a three-dimensional (3D) data set using a modification of the original cone-beam algorithm developed by Feldkamp et al. in 1984. (19)

CBCT is a widely used tool for several dental applications, such as implant planning, endodontics, maxillofacial surgery and orthodontics. (20,21)

Common uses of CBCT imaging in the head and neck are impacted teeth, evaluation, planning of implant treatment, TMJ evaluations, orthodontic and surgical planning, diagnosis of dento-alveolar pathologies, paranasal evaluation of the sinuses and pharyngeal airway. (17)

In recent years, there are many studies in which the canalis sinosus imaging was performed with CBCT in the literature. Some of these studies are shown in the table below. (Table 1**)**

Table 1: Recent Studies Examining Canalis Sinousus with CBCT

Article name	**Author(s)**	**Year**
An extremely rare anatomical variation bilateral canalis sinuosus and nasopalatine duct cyst cyst and role of cbct in diagnosis.	Kose, E, Sekercı, A. E., Soylu, E., & Nazlım, S.	2014
Canalis Sinuosus and radiographic procedures in the region of anterior maxilla	Kim, J. H., Júnior, R. A., Aoki, E. M., Baladi, M. G., Cortes, A. R., Watanabe, P. C., & Arita, E. S.	2015
Branch of the canalis sinuosus: a rare anatomical variation—a case report.	Torres, M. G. G., de Faro Valverde, L., Vidal, M. T. A., & Crusoé-Rebello, I. M.	2015
Location and classification of Canalis sinuosus for cone beam computed tomography: avoiding misdiagnosis	Manhães Júnior, L. R. C., Villaça-Carvalho, M. F. L., Moraes, M. E. L., Lopes, S. L. P. D. C., Silva, M. B. F., & Junqueira, J. L. C.	2016
Accessory branch of canalis sinuosus mimicking external root resorption: A diagnostic dilemma	Shah, P. N., Arora, A. V., & Kapoor, S. V.	2017
Cone-beam tomographic analysis of canalis sinuosus accessory intraosseous canals in the maxilla.	Ghandourah, A. O., Rashad, A., Heiland, M., Hamzi, B. M., & Friedrich, R. E.	2017
Evaluation of the morphology of the canalis sinuosus using cone-beam computed tomography in patients with maxillary impacted canines	Gurler, G., Delilbasi, C., Ogut, E. E., Aydin, K., & Sakul, U.	2017
An anatomical variant: evaluation of accessory canals of the canalis sinuosus using cone beam computed tomography.	Orhan, K., Gorurgoz, C., Akyol, M., Ozarslanturk, S.,& Avsever, H.	2018
Radiological and morphometric features of canalis sinuosus in Russian population: cone-beam computed tomography study.	Anatoly, A., Sedov, Y., Gvozdikova, E., Mordanov, O., Kruchinina, L., Avanesov, K., ... & Darawsheh, H. M.	2019

Article name	Author(s)	Year
Canalis sinuosus: anatomical variation or structure?	Aoki, R., Massuda, M., Zenni, L. T. V., & Fernandes, K. S.	2020
Assesment of accessory branches of canalis sinuosus on CBCT images.	Tomrukçu, D. N., & Köse, T. E.	2020
Misinterpretation of a periapical radiograph: the canalis sinuosus mimicking a root resorption	Bliggenstorfer, S., Chappuis, V., & von Arx, T	2021
Evaluation of location of canalis sinuosus in the maxilla using cone beam computed tomography.	Şalli, G. A., & Öztürkmen, Z.	2021
Analysis of canalis sinuosus prevalence by cone beam computed tomographs (CBCT).	Brücker, M. R., Pohren, D., & Cantarelli, A. R.	2021
Evaluation of canalis sinuosus in individuals with cleft lip and palate: a cross-sectional study using cone beam computed tomography.	Ferlin, R., Pagin, B. S. C., & Yaedú, R. Y. F.	2021
Prevalence, Radiographic Features and Clinical Relevancy of Accessory Canals of the Canalis Sinuosus in Cypriot Population: A Retrospective Cone-Beam Computed Tomography (CBCT)	Beyzade, Z., Yılmaz, H. G., Ünsal, G., & Çaygür-Yoran, A.	2022
Examination of canalis sinuosus using cone beam computed tomography in an Australian population.	Yeap, C. W., Danh, D., Chan, J., & Parashos, P.	2022

3. Common Complications of Dental Implants

Dental implants are defined as an artifical tooth root replacements and used to support restorations that resemble a natural tooth or group of natural teeth. (22)

Any number of complications can occur during or after the placement of dental implant. Most are immediately apparent; however, some can occur much later. Most complications can be traced to treatment planning and execution and are therefore preventable. Bleeding, postoperative infections, nerve injury, malpositioning of implants, injury to adjacent teeth, fracture of jaws, displacement or infringement on adjacent spaces may occur after implant

placement as common complications. (23) Nerve damage may be encountered due to insufficient knowledge of the anatomy of neurovascular structures and insufficient preoperative radiological examination. Prevention can be simplified to careful preoperative planning. 3D examination allows the evaluation of anatomical structures and their variations, thus preventing injuries.

4. Importance Of Canalis Sinousus In Dental Implant Surgery

There are many studies in the literature associated with canalis sinousus after implant surgery in the anterior maxilla region. Shintaku et al (12) reported unexpected chronic neurosensory disturbances without any clinical signs supportive of implant failure. The retrospective study showed that tomographic assessment using CBCT revealed the invasion of the CS and its accessory canals by dental implants.

Volberg and Mordanov (14) described a case of 45 year old female patient who underwent upper left lateral incisor extraction and immediate implant placement and implant removal in 16 days secondary to pain and paresthesia in the maxillary left region. The case report presented complaining about the pain and paresthesia in the left maxilla in a few hours after dental implant placement and increased in a week located in the area of the maxillary left canine. After 3 dimensional examination via CBCT, neurovasculer damage occured and the implant was extracted in in about 2 weeks. After th,s procedure, the pain and paresthesia started slowing down.

Lopes dos Santos et al. (24) describes a case of a patient who suffered pain due to exposure of the CS and also the review of the literature revealed six cases. It is reported that five out of seven cases were related to dental implant placement and resulted in postoperative pain and/or paresthesia. Similar to previous study, the dental implant was removed in 4 out of the 5 cases due to neurovascular damage.

Torres et al. (25) found anatomical variation of CS in the CBCT image taken for preoperative implant surgery evaluation and presented it in their case, drawing attention to the importance of preoperative 3 dimensional radigraphic evaluation.

Rosano at al. (26) described a case of 62 year old female patient with severe bone atrophy. Graft procedure applications and eight implants replacements were described. It is mentioned in the study that the patient had complained from increasing pain. As a complication the CBCT images had demonstrated that a CS on the right side was compressed by the apex of the implant in position #11.

Kim et al. (27) reported a case that implant was positioned close proximity of CS. The CBCT image demonsrated the relationship between this anatomical structure and dental implant. It is recommended that even though there is no signs of complications or symptoms. surgeons should consider possible anatomical structure.

Okumuş et al. (28) reported two cases of the canalis sinuosus that were detected by CBCT done for the dental implant treatment planing in a 46-year-old male and a 40-yearold female patients. Similar to previous studies, it is recommended that identification of these anatomical variations may help the surgeon to avoid injuries to nerves during surgical processes**.**

5. Conclusion

Persistent increasing pain and paraesthesia after maxillary anterior implant surgery are associated with invasion of the CS. Clinicians should be very careful about Canalis Sinousus and its possible anatomical variations when planning implants in the maxilla anterior region and during implant surgery. Preoperative examinations via CBCT prevent possible neurovascular damage. Unanticipated neurosensory symptoms after implant placement in the anterior maxilla justify the use of CBCT to rule out an injury to this neurovascular bundle. In the evaluation of possible post-operative complications, it should be considered that the symptoms of neurovascular damage in the maxilla anterior region may be related to the canalis sino**usus.**

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CHAPTER XV

VENTRICULOPERITONEAL SHUNT COMPLICATIONS

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HYDROCEPHALUS

Hydrocephalus is a disorder characterized by excessive cerebral and spinal fluid (CSF) in the enlarged ventricles and in the subarachnoid space (1,2). In the pediatric age group, hydrocephalus is always associated with increased intracranial pressure (IICP). This is due to the excessive amount of increased CSF in the brain ventricles, which causes disruption in the cerebrospinal fluid circulation (occlusive or non-occlusive hydrocephalus). Disruptions in absorption (communicated hydrocephalus), which are less common, also lead to an increase in cerebrospinal (3). The incidence of congenital hydrocephalus has been reported to be between 1-1.5 per 1000 live births (3). Together with other congenital neurological problems and hydrocephalus occurring due to many acquired intracranial pathologies, the rate increases to 3-4 per 1000 live births (3).

CSF circulation is generally explained by the Monro-Kellie doctrine. Accordingly, there are four basic structures in the brain. These include the brain, vascular structure, CSF, and blood circulation that keep the entire volume of CSF under control (4). Choroid plexuses produce 80% of CSF. 20-30% is produced by extrachoroidal and ependymal cells (5,6,7). In adults, up to 500 cc of CSF is produced in a day. There is a circadian rhythm that is activated 3-4 times a day. There are ongoing animal experiments investigating the circulation of CSF and hydrocephalus (7).

Three main themes in animal experiments: Production, absorption, and circulation.

Current knowledge of CSF hydrodynamics states that after CSF is produced from the ventricles, it circulates through the system, reaches the subarachnoid space in the cortical region, and is then absorbed from the sinuses (7). In 1919, Dandy identified this basic information and revealed that hydrocephalus occurs as a result of obstruction (8). Recent studies have uncovered new information regarding CSF hydrodynamics (9,10,11). Greitz, in his study in 2004, revealed that CSF is mainly produced everywhere in the central nervous system, especially in the choroid plexus, and the absorption of CSF occurs from the capillary circulation in the central nervous system (12). Greitz also determined that the rapid passage of CSF in the subarachnoid space occurs with vascular pulsation. Apart from protecting the brain from mechanical traumas by acting as a cushion, CSF also carries out the transport of different neurotransmitters and other metabolites produced in the brain (12).

SHUNT

Ventriculoperitoneal (V/P) shunt application is the most commonly used treatment method for hydrocephalus (13). The first ventriculoperitoneal (VP) shunt application was performed by Kausch in 1908 but the patient died the next day after surgery (14). In 1949, Nulsen and Spitz of the University of Pennsylvania connected a valve system consisting of a stainless steel ball to a tube and provided drainage from the lateral ventricle of the brain to the jugular vein (14). Shunt complications that occurred since then created significant problems for physicians as well as the patients, thus causing the continuous development and renewal of the designs used for the shunt. Despite the development in shunt technology, of the patients who undergo shunt surgery, 33%, 50%, and 70% are diagnosed with shunt dysfunction within a year, within two years, and within ten years, respectively (15). Over- or under-discharge of the shunt or shunt infections play a role in the development of shunt dysfunctions (14). The most common reason for the slow operation of the shunt is the obstruction of the catheter placed in the ventricle (16). Choroid plexus, blood, tissue debris, inflammatory cells and tumor cells may play a role in the obstruction of the ventricular catheter. At the same time, catheter-related occlusion may also occur if the ventricular tip remains in the parenchyma due to improper placement or shrinkage of the ventricles (17).

Among all mechanical complications in the literature, the incidence of ventricular catheter occlusion is 63.2% (18). It was previously thought that occlusion of the pump occurred rarely and due to the tissue residues filling the

pump in the early period (19). In another study, it was reported that chlorine, calcium and sodium precipitated in the pump and formed a layer that narrowed the outlet area of the pump, and the layer forming could be seen in the electron microscope after the second week (20).

One of the reasons for the decrease in shunt drainage is occlusion in the peritoneal catheter. The most common causes of intraperitoneal catheter occlusions are placement of the catheter in the pre-peritoneal space during surgery and pseudocysts formed in the abdomen (21). Abdominal ultrasonography should be performed in patients who present with abdominal discomfort and have a shunt placed previously. Since the cerebrospinal fluid (CSF) sample to be obtained with the help of a needle from the inside of the pump or from the ventricle can often be in a sterile form, the removed end of the peritoneal catheter should be sent to cultural examination to determine the infectious agent (22).

Separation or breaking of the parts forming the shunt is one of the factors that cause a decrease in the discharge of the shunt. Breakage of the shunt is a late complication. Even if the shunt is broken, drainage from the surrounding fibrous sheath continues (23). Overdrainage of the shunt is a problem that is generally associated with the pump. The reason for overdrainage is wrong pump selection or the effect of the siphon. When the patient stands up, the negative pressure created by the hydrostatic fluid column in the peritoneal catheter connected to the downstream end of the pump may cause the pump to overwork. This effect is called the siphoning effect (24). Subdural hematoma or fluid collection may occur as a result of shunt overdrainage (25). Isolated 4th ventricular syndrome, slit ventricle syndrome or craniosynostosis may also occur due to overdrainage (17,26,27). In long-term studies, the reported rate of shunt dysfunction is 32% (28).

Infections, which are frequent complications of shunting, are serious problems (29,30,31,32). Various studies report shunt infections in 20% and 5-10% of patients (19,30,32). Complications that occur in shunt surgeries can be categorized under 3 main headings: mechanical failure, shunt-related infection, functional failure of shunt operation.

Complications that occur in shunt applications are generally due to the patient, surgeon, or the shunt. These are usually different combinations of the factors listed. If the problem is directly related to the shunt, it is often defined as a shunt complication. If the problem is related to the function of the shunt, it may be difficult to evaluate it as a shunt complication (33,34). Mechanical complications in the shunt are usually time-dependent. Likelihood of shunt

failure in the patient is highest in the first months. Follow-up data shows that this rate generally decreases to 25-40% within a year. The risk decreases by 4-5% with each passing year. Studies show that the average lifetime of a shunt is 5 years (35).

SHUNT OCCLUSIONS

Shunt occlusions can occur at three different levels.

1. Proximal level occlusion (Ventricular or proximal side occlusion): In shunt surgery, occlusion occurs most commonly at the ventricular end and is the most common cause of failure. It accounts for 63.2% of all mechanical occlusions (19,32).
2. At the level of the valve system (valve occlusion).
3. At the distal level (distal occlusion).

Proximal Occlusions

There are different amounts of tissue residue and debris in the CSF. In addition, it provides the contraction of the ventricular cavity together with the wall (36). The choroid plexus floats in the CSF and surrounds the proximal catheter with the CSF flow. Together with the lumen, cellular debris and blood in the CSF may block the holes in the ventricular catheter. At the same time, leptomeninges, glial tissue, ependymal cells and connective tissue together with the choroid plexus in the ventricle may occlude the catheter (37,38,39,40). There is no ideal point to insert the ventricular catheter tip. The most suitable and lowest risk region is the largest region remaining in the ventricle after ventricular decompression, which differs from person to person (41,42). If the catheter is adhered to the ventricular wall and subsequently occluded, withdrawal of this adhered catheter increases the risk of major bleeding. Sometimes a metal wire can be sent to the tip of the catheter and coagulated, or the occlusion can be removed using the coagulation wire of the endoscope. It is also possible to remove a catheter wrapped with the choroid plexus in this way.

The tip design of ventricular catheters specifically aims to distance the catheter from the choroid plexus or ventricular wall (36). Ventricular measurements are an important factor for late occlusions of the proximal catheter (43,44,35). Regardless of how and where the proximal catheter is placed, the ventricular wall and the holes of the catheter may come into contact with each other as a result of overdrainage or go further and be wrapped by the choroid

plexus. A slit-like ventricle is a common condition in patients operated on with normal shunts. In published pediatric series, the incidence of this condition may be as high as 40% (35).

Valve Occlusions

Valve occlusions generally occur in three different ways:

1. While the shunt is being placed, the brain tissue or a clot from the ventricle may occlude the valve.
2. Valve occlusion may occur due to bacterial growth (40).
3. Immune response occurring at the cellular level may cause valve occlusion in the late period (38).

Distal Occlusions

The main causes of distal catheter occlusions are the distal tip coming out of the peritoneal cavity, the distal tip breaking off, and the separation of the valve and the distal tip from each other as young pediatric patients grow. The risk in distal end occlusions differs according to the shape of the catheter in which the CSF is drained. Catheters with a notched distal side and a closed tip have a high risk of occlusion. Occlusion may occur due to tissue build-up in the dead spaces between the two closed ends in the notches. Such occlusions do not occur in tubes with open distal ends.

In ventriculoatrial shunts, occlusion risk occurs when the tip of the drainage catheter protrudes from the right atrium. This condition, caused by thrombus, does not occur in peritoneal catheters. Although it is thought that partial occlusions occurring in the distal end may be caused by the decrease in the absorption capacity due to ascites in the peritoneal cavity or abdominal pseudocyst, the reason could not be understood in some of the cases (45). Intraperitoneal problems such as abdominal pseudocyst or peritonitis are also among the causes of distal end occlusions. In addition, distal occlusion can also occur by the peritoneal end coming in contact with and protruding into internal organs such as the bladder, bowel, thorax, stomach, vagina, scrotum, etc. (46,30,47,48,49).

Shunt Rupture and Separation

The second most common cause of shunt failure in the pediatric patient group is separation of the shunt after rupture. Main factors facilitating shunt rupture

are the quality of the material together with the shunt design and surgical technique used. The tension in the shunt, which occurs at any fixation point, may cause rupture. In addition, calcifications and dissolutions that occur as a result of immune reaction may lead to failure in shunt material and cause ruptures (50,51). During the surgical placement of the shunt, loose binding or the use of an absorbable suture material on the connection piece may create tension in the system and ultimately lead to separation. Hard, especially metal surgical instruments used during the procedure may cause tears on the shunt, which may cause ruptures subsequently.

There is risk of separation only if there are connection apparatuses in the shunt. There is no risk of separation in one-piece shunts (52,53). A catheter with its distal tube adhered to the valve and covered with pure silicon on the outer wall is the most correct option if its ventricular ends are properly connected.

Skin Problems

As with other types of shunt complications, skin problems mostly occur as a result of the interaction between the surgical techniques, shunt material, and the patient. Problems that occur due to the weakness of the surgical technique used may only be at the scar level or in the form of necrosis of the skin over the shunt. Such complications pose a great risk for contamination of the shunt. Pressure on the skin over the shunt (such as tight bandages or the patient lying down on the valve system) should be avoided. The optimum surgical technique is to place the valve under the galea with a small skin incision and suture the incision in two layers. Large valves, valves with sharp and hard plastic edges can cause skin trauma and necrosis.

Subcutaneous Fluid Collections

CSF accumulation under the skin is usually the result of shunt occlusion. Such a complication may sometimes occur in functioning shunts. Even if the shunt works normally, complications may occur due to reasons such as large ventricles, use of variable resistance or high valves, large incisions, young patients having thin skin, or excessive dissection under the skin.

Shunt Overdrainage

Shunt overdrainage is one of the problems encountered after the activation of valves (54,55). Complications that occur as a result of overdrainage include

craniosynostosis, subdural collections, ventricular loculation, and slit ventricle syndrome (33,56,57,58,59,54). The risks of these complications are very high in elderly patients due to neck length and postural changes.

Subdural Collections

In shunted patients, widening of the subarachnoid space occurs with good functioning of the shunt. Sometimes rupture of the arachnoid membrane and stretching of the veins in the subarachnoid region can cause hygroma or subdural hematomas. This is seen spontaneously in most of the shunted cases. Such complications can be managed with endoscopic alternative methods such as shunt avoidance or third ventriculostomy. Devices that prevent overdrainage prevent these complications from occurring. Most of the subdural collections that occur after overdrainage do not require treatment (52,25).

Slit Ventricular Syndrome

Slit ventricle syndrome occurs as a result of excessive CSF drainage. It is described as a triad of recurrent headaches, small ventricles on brain images, and slow refilling of the shunt pump. Classically, it is often seen in childhood, when brain development and excessive CSF drainage are concurrent. It has a certain pattern in the form of relapse and recovery periods, and its duration can be spread over months and years.

Craniosynostosis

If the intracranial pressure (ICP) is constantly below the physiological value in shunted pediatric patients, this rare complication is an indication for surgery. Premature closure, which usually occurs in the cranial sutures and brain growth in infants, along with continuous overdrainage of CSF, create slit ventricles with possible side effects (27,60).

Loculated Ventricles

In patients with hydrocephalus, loculation of the ventricles often occurs after an inflammatory condition such as meningitis or bleeding. However, in some cases, excessive CSF drainage on its own may cause loculation in the ventricular system. After shunting, ventricular asymmetry usually occurs and the ventricle where the shunt is placed remains small. Loculation of the ventricular system is a complication that needs to be treated with multiple drainage systems (61,62).

Orthostatic Hypotension

Clinical symptoms (nausea, headache) after orthostatic hypotension generally occur in elderly patients and often after shunt placement (33,58,59,54). These symptoms usually disappear after a short time and the patient adapts to the new conditions hydrodynamically. Vomiting, headaches, unconsciousness, nausea, and seizures may be seen in patients who have undergone shunt surgery, but these symptoms are not always related to shunt dysfunction (34). In such cases, procedures such as CSF flow measurement or ICP monitoring are recommended instead of unnecessary surgery (33, 63,64,65,66).

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