SURGICAL COMPLICATIONS IN ORTHOPEDICS AND SOLUTIONS FOR THE PREVENTION OF COMPLICATIONS

Editor

Asst. Prof. M.D. Murat SAYLIK

LIVRE DE LYON

2022

Health Sciences

SURGICAL COMPLICATIONS IN ORTHOPEDICS AND SOLUTIONS FOR THE PREVENTION OF COMPLICATIONS

Editor
Asst. Prof. M.D. Murat SAYLIK

LIVRE DE LYON
Lyon 2022

SURGICAL COMPLICATIONS IN ORTHOPEDICS AND SOLUTIONS FOR THE PREVENTION OF COMPLICATIONS

Editor
Asst. Prof. M.D. Murat SAYLIK

LIVRE DE LYON
Lyon 2022

Surgical Complications in Orthopedics and Solutions for the Prevention of Complications

Editor • Asst. Prof. M.D. Murat SAYLIK • **Orcid:** 0000 0002 1023 4164
Cover Design • Motion Graphics
Book Layout • Mirajul Kayal
First Published • October 2022, Lyon

ISBN: 978-2-38236-464-2

Publisher • Livre de Lyon
Address • 37 rue marietton, 69009, Lyon France
website • http://www.livredelyon.com
e-mail • livredelyon@gmail.com

LIVRE DE LYON

FOREWORD

We are experiencing a rapid development in Orthopedics and Traumatology with the rapid progress of technology, as in all areas of the health sector. Recently, the tendency to switch from classical open surgery methods to minimally invasive procedures has increased. New designed implants and robotic surgery applications have more preferred in prosthetic surgery. The most important aim of this and similar surgeries is to reduce the complications arising from surgery. Complications cause serious economic and social losses to both the patient and the physician.

In recent years, the high-participation meetings of the congresses are in the halls where the complications are explained. The prestige of physicians who shared their complications and reported solutions has increased.

In this book, it is aimed to raise awareness by explaining the complications. Approaches and solutions to complications will be a resource that especially our young colleagues will benefit from.

We aimed to present our experiences to the reader by supporting them with current information in the literature. The authors are entirely responsible for the accuracy of the information in the texts. The views in each chapter are the views of the authors of that chapter. Some of the views may not be the consensus of all the authors in the book.

I would like to thank who contributed to the writing of the chapters and the preparation of the book for publication.

Asst. Prof. M.D. Murat SAYLIK

CONTENTS

CHAPTER I

COMPLICATIONS OF APPLICATION OF MINIMALLY INVASIVE UNICONDILAR KNEE PROSTHESIS

Murat SAYLIK

(Asst. Prof. M.D.), Orthopedics and Traumatology / İstinye University
e-mail: drmuratsaylikster@gmail.com
ORCID: 0000 0002 1023 4164

1. Introduction

Knee joint involvement (gonarthrosis), which is one of the most common sites of osteoarthritis, is one of the important causes of morbidity and mortality in elderly patients. The first treatment choice in gonarthrosis is conservative. Before surgical application, oral or topical analgesics support to reduce the load on the knee (walker, cane, knee pad, insole), physical therapy applications, and intra-articular injections (analgesic, corticosteroid, platelet-rich plasma, collagen, hyaline) are applied. Surgical treatment options should be considered in patients who have been treated conservatively for six months or more but no results have been obtained. The surgical method is decided according to the degree of involvement of the medial, lateral, and patellofemoral joints in the knee joint.

Unicondylar knee replacement (UKP) in isolated medial joint osteoarthritis (OA) has been applied since the 1950s. However, UKP applications were avoided due to the high failure and recurrence rates in the first surgical procedures. In recent years, successful results have been reported in which long-term prosthesis survival after UKP has been reported with the formation of consensus on the indications and contraindications of UKP, the development of surgical application sets and prostheses, the correct patient selection and the increase in surgical experience (1). Contraindications to UKP: Inflammatory diseases, morbid obesity, presence of more than 15 degrees of varus of the knee or flexion contracture of more than 10 degrees, anterior cruciate ligament damage (UKP with fixed inscription can be applied), and the presence of infection (2).

UKP can be implemented in two ways, mobile or fixed-face. There is a great deal of research investigating the superior aspects of these two designs (3). UKP can be performed by classical or minimally invasive surgical method (1,4). With miniinmal invasive unicondylar knee prosthesis (MIUKP), there is less blood loss, rehabilitation is started earlier after surgery, and the return to daily life is fast (5).

Although the revision results of short and medium period MIUKP were higher than TKP in the studies comparing MIUKP and total knee prosthesis (TKP), the MIUKP results are still satisfactory (6,7).

Component loosening, polyethylene abrasion, collapse of the tibial component and osteoarthritis in the opposite compartment are common complications in UKP. The most common complication in fixed inpatient prostheses is the collapse of the tibial component and the relaxation of the femoral component. The most common complication in mobile UKP is inst dislocation. Less common complications are metallosis, meniscal tear in the lateral joint and pseudomenial synovial impingement syndrome (8). In order to avoid complications in MIUKP, the kinematic arrangement of the knee should be provided with bone incisions without loosening the ligaments.

Most of the prosthetic problems due to complications in MIUKP can be revised with primary knee prosthesis without the need for stem or other implant support (9).

2. Complications due to long learning time of MIUKP

MIUKP surgery is completely different from TKP and is considered to be more difficult to perform. Therefore, the existence of a learning curve is inevitable for an orthopedist who starts to practice UKP (1,10). If the surgeon who will perform MIUKP has been applying arthroscopy and arthroplasty for a long time and with high intensity, it will be easier to provide bone block incisions and ligament balance (11). In a large-series study investigating the UKP learning curve, 445 knees with MIUKP were included in the study and revision and re-operation rates were investigated according to the number of cases. On average, 26 strings (5.8%) revisions were applied in 3.25 years, and the survival rate of prosthesis in year 2 was 96%+/-1.7%. As the number of MIUKP applications increased, it was seen that the number of revisions and re-surgeries decreased. Especially after the second year, revision rates decreased from 5% to 2.5% and re-surgery rates decreased from 8.1% to 5.4%, but there was no significant difference (10). In a study on the MIUKP learning curve in our country, a 40% shortening of the operation time after 4 years and 33 cases and successful results close to CIP in Knee Society Scoring were reported. In this study, it was seen that the operation time was significantly higher in the first 20 cases than in the subsequent cases, and there was no significant change in the amount of bleeding (2). In these two studies, it is seen that the time difference in the transition to the successful case is related to the intensity of MIUKP applied and the learning time decreases as the number of MIUKP increases.

In another study on the MIUKP learning curve, patients were followed for 52 months and it was reported that 7 revisions were made in the first 2-year period (12). Similarly, in a study in which 59 MIUKP were applied, all 6 cases with revision with CIP were reported in the first 2 years (13).

In the early period of MIUKP application, proximal tibial fracture may occur due to deep application of tibial bone vertical incision and inappropriate surgical technique. This complication can be prevented by controlled use of the saw, non-deterioration of cortical integrity and the use of insinört of appropriate thickness (14).

3. Mobile or Fixed MIUKP application complications

MIUKP mobile or fixed can be applied. The first UKP applications were mobile insertion weighted with the idea that the mobile insertion would provide more knee flexion opening than the fixed insertion. However, in the studies conducted, there was no significant difference in terms of knee flexion patency and clinical outcomes in both prosthesis types (12). The most common complication in cases where mobile insertion is applied is the protrusion of the insemination to the suprapateller region. The most common complication in fixed insemination is the collapse of the tibial component (12).

4. Complications of implant placement due to surgical application

The results of MIUKP depend on the experience of the surgeon and the surgical technique applied. Radiological inappropriate results are seen due to the improper placement of the prosthesis in the joint (15). Implant placement and leg alignment are evaluated with two-way knee X-ray and extremity length radiography taken after MIUKP. The suitability of the radiological location of the prosthesis is measured by the radiological evaluation criteria recommended by the Oxford group (4).

According to these criteria:

- Evaluation of femoral component: There should be varus - valgus angle below 10 degrees, flexion - extension angle below 5 degrees.
- Tibial component evaluation: varus below 10 degrees - valgus angle, overflow less than 2 mm on the medial plateau, posterior slope should be between 7 and -5 degrees.
- Initiation evaluation: Especially the mobile insemite should have a compatible placement with the femoral and tibial components.

In the evaluation of MIUKP 2-way knee X-ray according to Oxford group criteria, it was reported that incorrect position in the femoral or tibial component may cause early revision of the prosthesis (1,16,17). The application of prosthesis in varus was found to be more tolerable than the application in valgus. With the application of MIUKP in valgmus, there is a risk of fracture of the medial tibial condyle due to overload in the axial intercondylar region (18). In another study, it was reported that the application of tibial component up to 6 degrees in varus would not cause serious abrasion in polyethylene, but application in valgus would increase insectant wear (19, 20).

Location of the tibial component in the valgus and posteriority, and internal rotation of the femoral component significantly increases the risk of instuction in UKP with mobile inshirt (21). In addition, the posterior tibial slope of more than 8 degrees affects mobile insurrection dislocation (22).

Lateral application of the femoral component increases the contact load in polyethylene and wear in the insect, while close application to the medial reduces the contact load but may cause mobile insurrection dislocation. The optimal position to prevent premature wear of the polyethylene incisor and dislocation of the incisors is the placement of the component in the femoral condyle power plant (23). Application of the femoral component in flexion more than 15 degrees in the sagittal plane may cause a glass lesion due to the osteophyte remaining posteriorly in the femoral condyle gland. Glass lesion may cause flexion limitation and mobile insemination. If a glass lesion is noticed, osteophyte excision should be performed (24).

5. Chondral damage to the patellofemoral and lateral joint

Failure to achieve kinematic alignment of the knee (valgus and/or tight knee) after MIUKP is considered to be the cause of OA in the lateral joint and patellofemoral joint. The mechanical axle measured on the orthoroentgenogram is requested to be between 5-7 degrees, which is the acceptable limit of the mechanical axis after surgery.

If the degree of chondral damage to the patellofemoral joint develops as a complication before and after MIUKP, it should be evaluated. Chondral damage to the patellofemoral joint, which is often considered one of the contraindications to the administration of MIUKP, has not been accepted as an absolute contraindication in some studies (25). There are studies showing that moderate patellofemoral chondromalacia after MIUKP does not change clinical outcomes (25).

Patellofemoral joint complications occur in patients with quadriceps inhibition due to long tourniquet duration or improper closure of incisions during surgery (26). Squeezing the tourniquet just before surgery, applying the tourniquet pressure at a maximum pressure of 100 mmHg from the patient's blood pressure arterial pressure, separating the quadriceps muscle from the clivag in accordance with the minimally invasive technique and repairing it with 2 no vicril after surgery reduces the incidence of patellofemoral joint complications.

6. Thromboembolism

The most important differences that make MİUKP stand out compared to TKP are that the complications of venous trpmboembolism (VTE), deep vein thrombus (DVT) and pulmonary embolism (PE) due to thromboembolism are less. This is due to the fact that MIUKP is a less invasive method than TKP, it is a shorter-term surgery and it is generally applied in more active patients (27). However, the gold standard for the low molecular weight heparin dose to be administered to prevent VTE due to MIUKP has not been established and there is no consensus on the optimal treatment dose (28). Mobilization of the patient on the same day after MIUKP also reduces the risk of thromboembolism.

7. Conclusion

MIUKP is a completely different surgical application from TKP, which has a long learning time. It can be applied in gonarthrosis with isolated medial joint involvement. Compared to CIP, complication rates are significantly less. The most common complication in cases where mobile insertion is applied is the protrusion of the insert to the suprapateller region. In fixed insertion, the most common complication is collapse of the tibial component. In the evaluation of the suitability of the prosthesis, the radiological evaluation criteria recommended by the Oxford group are often used.

References

1- B Murat, S Akpınar, M Uysal, N Cesur, M A Hersekli, M Özalay, Özkoç G. Unicondylar knee artroplasty in medial unicompartmantal osteoarthritis: Technical faults and difficults. Joint disease andrelated surgery. 2010; 21(1): 31-37.

2- Saylık M, Şener N. Learning Curve UCA. Acıbadem üniversity health sciense magazine. January.2013

3- Bülbül AM, Kuyucu E, Kara A, Küçükdurmaz F, Erdil M . Orta Dönemde Hangi Unikondiler Protez Daha İyi, Fiks mi, Mobil mi?2016 Jarem, 6(1);31-34.doi:10.5152/jarem.2015.863.

4- Shakespeare D,Ledger M,Kinzel V,Accuracy of implantation of components in the Oxford knee using the minimal invasive approch.Knee 2005;12:405-9.

5- Berger RA, Cross MB, Sanders S. Outpatient Hip and Knee Replacement: The Experience From the First 15 Years. Instr CourseLect. 2016; 65: 547-5

6- Labek G, Thaler M, Janda W, Agreiter M, Stöckl B. Revision ratesafter total joint replacement: cumulative results from world widejoint register datasets. J Bone Joint Surg Br 2011; 93:293-7.

7- Lygre SH, Espehaug B, Havelin LI, Furnes O, Vollset SE. Pain andfunction in patients after primary unicompartmental and total kneearthroplasty. J Bone Joint Surg Am 2010; 92:2890-7.

8- Jung KA, Lee SC, Hwang SH. Pseudomeniscal synovial impingement after unicondylar knee arthroplasty. Orthopedics2009:32(5):361.

9- Springer BD, Scott RD, Sah AP, et al. McKeever hemiarthroplasty of the knee in patients less than sixty years old. J Bone Joint Surg Am 2006;88(2):366–71.

10- Hamilton WG, Ammeen D, Engh CA JR, Engh GA. Learning curvewith minimally invasive unicompartmental knee arthroplasty. JArthroplasty 2010; 25:735-40.

11- Boniforti F. Medial unicondylar knee arthroplasty: technical pearls.Joints. 2015 Nov 3;3(2):82-4. doi:10.11138/jts/2015.3.2.082.

12- Sun PF, Jia YH. Mobile bearing UKA compared to fixed bearingTKA: A randomized prospective study. Knee. 2011;21.

13- Heller S, Fenichel I, Salai M, Luria T, Velkes S. The Oxford unicompartmental knee prosthesis for the treatment of medial compartment knee disease: 2 to 5 year follow-up. Isr Med Assoc J2009;11:266-8.

14- Pandit H. Murray DW, Dodd CA. Medial tibia plateau fracture andthe Oxford unicompartmantel knee. Orthopedics 2007;30(5Suppl):28-31

15- Braito M, Giesinger JM, Fischler S, Koller A, Niederseer D,Liebensteiner MC . Knee Extensor Strength and Gait Characteristics After Minimally Invasive Unicondylar Knee Arthroplasty Minimally Invasive Total Knee Arthroplasty : A Nonrandomized Controlled Trial. J Arthroplasty. 2016 Feb 10. pii: S0883-5403 (16)00111-X . doi: 10. 1016/j.arth.2016.01.045

16- Kim KT, Lee S, Kim JH, Hong SW, Jung WS, Shin WS. TheSurvivorship and Clinical Results of Minimally Invasive Unicompartmental Knee Arthroplasty at 10 Year Follow up. Clin Orthop Surg. 2015 Jun; 7(2):199-206. doi: 10.4055/cios.2015.7.

17- Saylık M, Şener N. Common Errors in The Practice According toOxford Group Radiological Assesment Criteria in Minimally İnvasive Unicompartmental Knee Arthroplasty. AcıbademUnıversity Journal of Health Sciences.2021;12(2): 220-227. Doi:10.31067/acusaglik849564.

18- Inoue Akagi M, Asada S, Mori S, Zaima H, Hashida M. The Valgus Inclination of the Tibial Component Increases the Risk of Medial Tibial Condylar Fractures in Unicompartmental Knee Arthroplasty.J Arthroplasty. 2016 Feb 27. pii: S0883-5403(16) 00189-3.doi:10.1016/j.arth. 2016.02.043.

19- Koh YG, Hong HT, Kang KT. Biomechanical Effect of UHMWPEand CFR-PEEK Insert on Tibial Component in Unicompartmental Knee Replacement in Different Varus and Valgus Alignments.Materials (Basel). 2019 Oct 14;12(20).

20- Innocenti B, Pianigiani S, Ramundo G, Thienpont E. Biomechanical Effects of Different Varus and Valgus Alignments in Medial Unicompartmental Knee Arthroplasty. J Arthroplasty. 2016 Dec;31(12):2685-2691. doi: 10.1016/j.arth.2016.07.006. Epub 2016 Jul 15

21- Gulati A, Weston-Simons S, Evans D, Jenkins C, Gray H, Dodd CA,Pandit H, Murray DW. Radiographic evaluation of factors affectingbearing dislocation in the domed lateral Oxford unicompartmentalknee replacement. Knee. 2014 Dec; 21(6):1254-7.

22- Weber P, Schröder C, Schmidutz F, Kraxenberger M, UtzschneiderS, Jansson V, Müller PE. Increase of tibial slope reduces backsidewear in medial mobile bearing unicompartmental knee arthroplasty.Clin Biomech (Bristol, Avon). 2013 Oct;28(8):904-9. doi: 10.1016/j.clinbiomech.2013.08.006.

23- Kang KT, Son J, Koh YG, Kwon OR, Kwon SK, Lee YJ, Park KK.Effect of femoral component position on biomechanical outcomes ofunicompartmental knee arthroplasty. Knee. 2018 Jun;25(3):491-498.

24- Bozkurt M , Akmese R, Cay N, Isik Ç, Bilgetekin YG, Kartal MG, Tecimel O. Cam impimgement of the posterior femoral condyle inunicompartmental knee arthroplasty.Knee Surg Sports TraumatolArthrosc. 2013 Nov; 21(11):2495-500. doi. 10.1007/s00167

25- Song, Eun-Kyoo, et al. No difference in anterior knee pain after medial unicompartmental knee arthroplasty in patients with or without patellofemoral osteoarthritis. Knee Surgery, Sports Traumatology, Arthroscopy, 2016, 24.1: 208-213.

26- Erçin E,Çelen K, Kızılkaya C et AL. Patellofemoral pain after mobile bearing unicondylar knee arthroplasty. Mustafa Kemal paşa unıversty.2017:8(32):11-19. doi: 10.17944/mkutfd.291872.

27- Willis-Owen CA, Sarraf KM, Martin AE, Martin DK. Are current thrombo-embolic prophylaxis guidelines applicable to unicompartmental knee replacement? J Bone Joint Surg Br. 2011;93:1617–1620.

28- Duchman KR, Gao Y, Pugely AJ, Martin CT, Callaghan JJ. Differences in short-term complications between unicompartmental and total knee arthroplasty: a propensity score matched analysis. J Bone Joint Surg Am. 2014;96:1387–1394.

CHAPTER II

PULMONARY THROMBOEMBOLISM AND FAT EMBOLISM IN TOTAL HIP ARTHROPLASTY

Alikemal YAZICI[1,2]

[1]*Faculty of Medicine, Orthopedics and Traumatology Department, Near East University, Nicosia, Cyprus*

[2]*Buyuk Anadolu Hospital, Orthopedics and Traumatology Department, Samsun, Turkey, e-mail: alikemalyazici@hotmail.com*
ORCID: 0000-0001-7902-5734

1. Deep Vein Thrombosis

1.1. Definition and Epidemiology

Deep vein thrombosis (DVT) refers to the development of thrombosis, often in the lower extremity and pelvic deep veins and occasionally in the upper extremity veins, associated with the presence of acquired and/or hereditary factors leading to stasis, hypercoagulability and vascular damage, defined as the Virchow triad. DVT is seen in 40%-60% of total hip arthroplasty (THA) patients who are not administered thromboprophylaxis, and proximal DVT is seen in 20%. In THA patients who are administered thromboprophylaxis, these rates are 1.3%-10% DVT, 5% proximal DVT, and 0.4-2.7% symptomatic DVT. DVT is usually seen together with pulmonary thromboembolism and is usually asymptomatic. It has been reported that 30% of DVTs start during the operation, and of these, a third spontaneously recover within a few days, 40% progress, and 25% form proximal DVT. The risk of DVT is increased 2-24-fold in major orthopaedic surgery (1-5).

1.2. Risk Factors

Risk factors for DVT have been stated to include; Major orthopaedic surgeries such as THA, cemented femoral stem, immobilisation, diabetes mellitus, Cushing syndrome, hyperthyroidism, obesity, rheumatoid arthritis, ulcerative colitis, hypoalbuminemia, malignant diseases, stroke, varices, smoking, hypertension, metabolic syndrome, long operation time (>2 hrs), oral contraceptive use, advanced age, polycythemia vera, and several genetic risk factors (hereditary thrombophilia, antithrombin III, protein C and S deficiency, homozygote factor V Leiden disease, antiphospholipid syndrome) (3, 5).

1.3. Clinical Status and Diagnosis

Clinically, DVT is seen as swelling in the extremity, pain especially in the calf, heat and/or redness, a feeling of heaviness in the extremity and cramps. Findings determined in the physical examination are pain in the calf on dorsiflexion of the foot (Homans sign), increased pain in the popliteal region with knee extension, and an increase of >3cm in the leg circumference compared to the contralateral leg. However, these symptoms and clinical findings can only be determined in 50% of the patients who develop DVT (8). Although DVT is usually seen together with pulmonary embolism (PE), it may not lead to any symptoms, and these patients may present with symptoms of dyspnea, tachypnea, and tachycardia associated with PE (5-7). Diagnosis is made with compression ultrasonography of the lower extremity (9).

1.4. Prevention and Treatment

Mechanical thromboprophylaxis (hydration, foot pedal exercises, early mobilisation, elastic antiembolism stockings at 14-15mmHg pressure, intermittent pneumatic compression devices) and pharmacological thromboprophylaxis (generally low molecular weight heparin (LMWH), but also new oral anticoagulants, fondaparinux, and standard heparin) are recommended in the prevention of DVT (3, 5). The form of application of thromboprophylaxis is described in the guidelines of the American College of Chest Physicians (ACP) and the American Academy of Orthopaedic Surgeons (AAOS).

According to the ACCP guidelines, 1A use (strong recommendation, consistent with randomised controlled studies results) of LMWH is recommended as thromboprophylaxis in elective THA, starting at 12 hours preoperatively or 6-12 hours postoperatively according to the additional risks of the patient, and continued for at least 10 days and for as long as 35 days (4, 10). In the treatment of DVT, anticoagulant treatment (LMWH, Vitamin K antagonists [warfarin], standard heparin, fondaparinux, oral anticoagulants [rivaroxaban, dabigatran, apixaban, edoxaban]), mechanical venous thrombectomy, fibrinolytic drugs are recommended.

In the classic treatment approach of the ACCP and National Venous Thromboembolism Prophylaxis and Treatment Guidelines, it is recommended to start parenteral (LMWH or standard heparin or fondaparinux) and warfarin treatment together, then to stop the parenteral treatment after 5-7 days depending on the INR (INR value >2 measured twice at a 24-hour interval [target value 2.5; 2-3]) and to continue the warfarin treatment for at least 3 months (4, 11). In the acute pulmonary emboli diagnosis and treatment guidelines published by the European Cardiology Association, the use of new oral anticoagulant agents is recommended in the acute and long-term treatment of DVT (3).

2. Pulmonary Embolism Definition and Epidemiology

Pulmonary embolism (PE) is one of the most serious complications of THA, causing significant costs, morbidity and mortality. It usually occurs as a result of the thrombus formed in the deep veins of the lower extremity blocking the pulmonary artery and branches. When DVT and PE are seen together, it is referred to as pulmonary thromboembolism (PTE) or venous thromboembolism (VTE). In THA patients who are not administered thromboprophylaxis, PE is seen at 5-10%, and fetal PE at 1.8-3.3%, whereas in cases administered thromboprophylaxis, PE has been reported at 0.2-0.6%. Mortality rates vary between 0.2-1.2% in low -risk cases and 4.8-25% in high-risk cases (1-3, 12, 13).

2.1. Etiopathogenesis

Flexion and rotation movements of the hip during THA causing femoral vein torsion lead to venous stasis and endothelial damage. An increased tendency to clotting formed by the surgical trauma also causes the Virchow triad to form, which is necessary for thrombus. At the same time, fat and bone marrow pass to the venous system because of the increasing intramedullary pressure associated with cement and stem implantation. Therefore, there is an increased risk of both venous thromboemboolism and air and fat embolism in THA (14).

2.2. Clinical Status and Diagnosis

PTE is seen in 3 forms of low-risk without symptoms (non-massive), moderate-risk with moderate symptoms (sub-massive), and high-risk extending as far as sudden death (massive). The clinical status of patients varies according to the severity of PTE. In non-massive PTE, blood pressure and right ventricle functions are normal, and mild side chest pain may be observed. In sub-massive PTE, blood pressure is normal but there are findings of right ventricle failure (dialatation, hypokinesis) on echocardiography. These may be seen as dyspnea, tachypnea, tachycardia, pleurotic side chest pain, cough, hemoptysis, anxiety, and hypotension. In massive PTE, other findings are also seen of peresistent hypotension, a feeling of substernal pressure, mental confusion, acute right ventricle failure, syncope, and cardiopulmonary arrest. The most commnly seen findings are dyspnea, tachypnea, tachycardia, and pleurotic side chest pain (3, 5, 7, 8). DVT and PTE include the same risk factors, and major orthopaedic surgery is said to be the most important risk factor for PTE (3, 5). DVT has been reported to be determined in 16.9-56.4% of PTE cases, with approximately 50% of DVTs seen as asyymptomatic, 80% in proximal veins, and 20% in distal veins.

For diagnosis, patients should be evaluated according to the criteria of the Wells or Simplified Wells pulmonary embolism severity index (PESI) (Table 1). D-dimer should be examined, and lower extremity compression ultrasonography, echocardiography, and multidetector CT angio (≤4) should be performed. In early diagnosis, multidedector CT angio (≤4) should be performed first if the patient and hospital conditions are suitable, otherwise echocardiography should be the first method applied for diagnosis and differential diagnosis (5, 15-17).

Table 1. Wells and Simplified Wells pulmonary thromboembolism clinical prediction score

Finding	Wells (points)	Simplified Wells (points)
Presence of deep vein thrombosis symptoms and signs	3.0	1
Alternative diagnosis unlikely	3.0	1
Tachycardia (> 100/min)	1.5	1
History of immobilization or surgery in the last 4 weeks	1.5	1
Previous history of deep vein thrombosis or pulmonary embolism	1.5	1
Hemoptysis	1.0	1
Presence of cancer	1.0	1
Wells < 2.0 points: Low clinical probability, 2.0-6.0 points: Moderate clinical probability, > 6.0 points: High clinical probability, ≤ 4 points: PTE not likely, > 4 points: PTE possible		
Simplified Wells 0-1point: PTE not likely, ≥ 2 points: PTE possible		

2.3. Risk Factors

The PE risk factors include the risk factors stated for DVT (3, 5).

2.4. Treatment and Prevention

The treatment includes general support treatment, anticoagulants (LMWH, standard heparin, fondaparinux, new oral anticoagulants, danaparoid, Vitamin K anntagonists, parenteral direct thrombin inhibitors), thrombolytic treatment (recombinant human tisssue type plasminogen activator (alteplase), streptokinase and urokinase), embolectomy performed with surgical and percutaneous techniques, and vena cava inferior filter application (18-21). It has been reported that >50% of the deaths of patients with PTE occur within the first hour (5). Therefore, when there is sudden postoperative development of dyspnea, tachypnea, tachycardia, pleurotic side chest pain, cough, hemoptysis, anxiety, or mental confusion in a patient who has undergone THA, PTE should be the first pathology to come to mind, and admitting the patient to intensive care conditions, starting supportive treatment and effective treatment after definitive diagnosis can be life-saving. As stated for DVT, the priority for prevention of PTE should be mechanical and pharmacological thromboprophylaxis. In addition, excessive hip movements should be avoided during the operation, the extremity should be frequently relaxed in the appropriate position, and long operating times should be avoided.

3. Fat Embolism

3.1. Definition and Epidemiology

Fat embolism (FE) is defined as an accumulation of fat droplets and bone marrow components exceeding 8-10 microns forming a blockage, generally in the pulmonary capillaries (22). Clinically, it is classified as:

Grade 0: no hypoxia (SO_2% >94%), no decrease or decrease of <20% in systolic blood presssure, clinicallyy insignificant fat embolism,

Grade 1: moderate hypoxia(SO_2 % < 94%), or 20-40% decrease in systolic blood pressure,

Grade 2: severe hypoxia (SO_2 % < 88%) or >40% decrease in systolic blood pressure,

Grade 3: cardiovascular collapse requiring cardiopulmonary resucitation (23, 24).

The incidence of FE is common in THA patients, but is not noticed as the majority of cases are insidious and asymptomatic. Occasionally, massive FE and FE syndrome (FES) may be encountered. Generally, the incidence of FE during THA has been reported to be 24%, as 69% Grade 0, 22% Grade1, 8% Grade 2, and 0.4% Grade 3, with the probability of 30 day mortality reported as 3.46% in Grade 3 (23). Also in THA, cerebral microembolisation has been reported at the rate of 23%, the vast majority of which occur intraoperatively (25).

3.2. Etiopathogenesis and Physiopathology

Previous studies have investigated the presence of FE during femoral osteotomy, acetabular reaming and cup placement, reaming of the femoral canal and stem placement, and while FE has not been determined in femoral osteotomy, it has been shown at a minimal level in acetabular reaming and cup placement, increasing during femoral canal reaming and reaching a maximum during implant placement (25). In the physiopathology of FE, there is the mechanical phase in which pulmonary capillaries are blocked by fat droplets, and the biochemical phase in which lipase production is started in the pulmonary alveolar cells and the fat droplets in the pulmonary capillaries start to be hydrolised by lipase, and within approximately 3 days, the fat embolism is eliminated. Therefore, the majority have a silent course. The fatty acids emerging from the hydrolised fat are transported by albumin, and thus no symptoms are seen in the majority of patients exposed to FE. However, occasionally when there is a massive FE, in which approximately 80% of the pulmonary capillary network is blocked by the FE, or FES develops, this can lead to death through damage to several organs, primarily the lungs and brain. Despite dense emboli in the majority of patients with FE in orthopaedic operations, severe symptoms are not seen because the effect in these patients is only mechanical, and there is a simple and temporary blockage of the pulmonary capillary network (22-28).

Although the venous system and lungs are affected as in PTE, the fat droplets of FE are different and can affect other organs by passing into the arterial system through anatomic arterio-venous connections, pulmonary capillaries, and foramen ovale pathways (22, 25). The organs most affected through the arterial route are the brain, the skin, and the retina. In addition, the kidneys, spleen, liver, myocardium, and adrenal glands may also be frequently affected (29-31). Embolisation of fat tissue, bone marrow, air and acrylic cement microfragments has been described in THA cases. Previous studies have shown that during the drilling of the medullar canal, reaming, cementing, and placement of the prosthesis in THA cases, there is migration of heart small chambers full of blood with a sudden snowstorm appearance (air embolism) on transoesophageal echocardiography, and during cementing, the right atrium and ventricle are made opaque by the FE. Snowstorm associated with air embolism has been observed in 85% of cases and FE in 72% during femoral stem implantation in previous studies (26).

Intramedullar pressure (IMP) is 30-50 mmHg under anaesthesia, and when this is 50-100 mmHg, it has been shown that FE has formed. It has been reported that when IMP is>150mmHg, there is a 10-fold increase in FE. During reaming of the medullar canal, IMP has been shown to reach values of 650 mmHg, during cementing this pressure reaches 650-1500 mmHg and as high as 3190 mmHg, and fat embolisation continues for more than 20 minutes. However, with the use of uncemented prostheses, IMP has been reported to reach a maximum of 125 mmHg. Previous studies have reported that during cementing and femoral stem placement, IMP increases, air, fat, and bone marrow particles enter the venous system v)ia the linea aspera, a proportion of these particles cause an embolism, and a proportion cause the development of mixed thrombus by adhering to the vessel walls (22, 23, 26, 32-38).

Bone cement (polymethylmethacrylate=PMMA) may play a vasodilator and vasodepressor role in the myocardium, and it has been reported that bone cement implantation syndrome, characterised by hypoxia, hypotension, and loss of consciousness, may be seen during cementing. However, for this effect it would be necessary to use a dose 40-50-fold greater than the dose used in arthroplasty, and therefore, cement cannot be held responsible for FE. In the majority of patients followed up, PMMA has not been reported to have been determined in the circulation intraoperatively during arthroplasty. However, studies have shown that FE is seen because of increased IMP during cementing and prosthesis placement in all hip and knee arthroplasties, and most of these have been during prosthesis placement with cement. The temperature occurring during heating of the cement, and methyl-methacrylate monomers or dimethyl-p-toluidin released in the cement hardening process have been reported to cause hypotension during cement application, but studies have not determined any statistical correlation of these (22-26, 39-42).

FES must not be confused with FE and massive FE. They refer to two different conditions, and FES is atypical development or a complication of FE, in which FE or macroglobins in the circulation lead to multisystem failure. At this point, the fatty acids hydrolised in the pulmonary capillaries and neutralised by being transported with albumin remain free in an as yet unknown way. By directly damaging the alveolar and endothelial cells, the free fatty acids cause acute respiratory failure, findings of brain involvement (anxiety, agitation, confusion, delirium, coma), petechiae on the skin, retinal infarct, and hormonal, cellular, hemodynamic, immunological, and blood clotting changes which can lead to hemorrhage and death. The event here is not the FE and trauma itself, but the systemic inflammatory or traumatic metabolic response triggered by the fatty acids. Although FES is rarely seen (0.1-11%), the mortality rate is high (5.8-20%) (22, 43-51).

3.3. Clinical Status and Diagnosis

FE can be seen with a drop in blood pressure, bradycardia, confusion, respiratory failure, heart attack, cardiac arrest, and death. In most patients, hypotension, cardiac arrhythmia, and a decrease in partial oxygen pressure (PaO2) can be seen lasting for a few minutes or longer without symptoms. Clinical findings can vary according to the severity of FE, the heart and liver functions of the patient and comorbid diseases (heart, lungs, kidneys), cancer, age >75 years, and the American Society of Anesthesiologists (ASA) score of III and IV (22-24, 39-42).

The diagnosis of FE, massive FE, and FES is made from the clinical, laboratory, and radiological findings. However, there is no clear finding for diagnosis. The patient must be evaluated with all the data together. In cases with the sudden emergence of tachypnea, hypotension, cardiac arrhythmia, and a slight decrease in PaO2 pressure during the intraoperative placement of cemented or uncemented femoral stem, and these findings lasting several minutes (or sometimes they can continue after the operation), and in the postoperative period, pulmonary findings such as tachypnea, dyspnea, a fall in PaO2 pressure (<60mmHg), findings of brain involvement such as clouded consciousness, sudden loss of consciousness, agitation, confusion, and delirium, skin findings such as petechiae in the axillary region and upper section, and findings of retina involvement, massive FE and/or FES should come to mind. However, it must not be forgotten that this status can also form in pulmonary contusion and craniocephalic trauma.

In the laboratory tests, anemia, thrombocytopenia (seen in 30% of cases), coagulopathy findings (occur occasionally with an increase in fibrin destruction products, a decrease in fibrinogen in circulation, an increase in thrombocyte count and aggregation, and prolonged active partial thromboplastin and prothrombin time), complement increase, an increase in free fatty acids and lipase enzyme, and the presence of fat droplets in the blood and urine, are used to support the diagnosis. On imaging, widespread bilateral infiltration in the basal and perihilar region is generally determined on the pulmonary radiographs 12-24 hours after the event, but this may not be seen in 30-50% of cases. This appearance may also be seen in pulmonary contusion, tracheobronchial aspiration, and acute respiratory distress syndrome (ARDS). Thorax CT, and multidetector CT-angio (≤4) and echocardiography should be performed for diagnosis and differential diagnosis. Brain oedema can be observed on brain CT, and the damage caused by embolism can be determined early and specifcally on brain magnetic resonance imaging (MRI). Both oedema and infarct areas smaller than 2cm in diameter can be seen (22, 49-53).

3.4. Treatment and Prevention

Many methods have been attempted in treatment but have not found a place in practice. Symptomatic treatment forms the basis of treatment. The aim of supportive treatments is to provide the preservation of oxygen levels and cardiac flow. With the aim of maintaining cardiac flow, saline and Ringer solution, dextrane, blood and blood products can be administered. If this is not successful, vasoactive drugs are used (dopamine, dobutamine, noradrenalin). To keep oxygen saturation over 95%, nasal oxygen (3-6 litres/min) or mechanical ventilation methods are used. Although corticosteroid treatment has not been shown to be useful in treatment, it has been reported to have a protective role when applied before the establishment of massive FE and FES (6-90 mg/kg methylprednisolone).

In the treatment of FE, there is no routine application of anticoagulants, unlike for PE. However, they can be applied in selected suitable patients if there is an additional indication for another reason and this will not cause complications. By reversing the disseminated intravascular coagulation status, heparin reduces lipemia by stimulating lipase but causes an unwanted increase in fatty acids. It may also cause a high risk of bleeding in arthroplasty and polytrauma patients (22, 47, 50, 51).

All these results have been revealed to originate from FE increasing IMP during THA. Therefore, to prevent FE in THA, efforts should be directed to preventing increased pressure in the medullar canal. In IMP, the duration of pressurisation, the cement viscosity, stem design, the gap remaining between the stem-cement and bone, and the severity of the pressure applied are important.

To prevent FE (22, 49-57), fat, bone, blood, and bone marrow remnats must be cleaned by washing the region with pulsatile lavage (pressurised washing) at all the femur preparation stages (after the first drilling and rasping of the femur, placement of the distal tampon, before placement of the cement and femoral stem). At least one litre of sterile saline solution should be used for the washing. To obtain a strong bone-cement interface, strong washing is required to clean the spongious bone. Pulsatile lavage both achieves this and reduces the passage of fat and bone marrow to the venous system. In a previous study that compared manual lavage and pulsatile lavage, there was seen to be a higher rate of fat and bone marrow intravasion in cases where manual lavage was applied. By closing the canal, femoral distal intramedullar tampons cause the formation of higher IMP during implantation and increase the risk of embolism. However, it has been attempted to reduce this risk with new designs of distal tampon placed 2cm distal of the implant.

As conic prostheses and prostheses larger than the medullar cavity further increase IMP, prosthesis selection should be made accordingly.

The cementing procedure should be performed retrograde from distal to proximal with a cement gun. Ensuring expulsion of the blood and residues remaining inside the femoral channel during cementation is accepted as the safest way of cementation for the prevention of PE.

It has been reported that high viscosity cement increases IMP but provides better bone attachment, and low viscosity cement passes into the venous system before deeper penetration to the bone is obtained and could be a cause of death.

There has been shown to be a lower risk of FE with the use of uncemented prostheses. In experimental studies, the amount of fat passing into the system has been determined to be two-fold greater with the use of cemented prostheses compared to uncemented prostheses. Therefore, uncemented prostheses should be selected if possible.

References

1. Hyers TM. Venous Thromboembolism. Am J Respir Crit Care Med 1999;159:1-14.
2. Mula V, Parikh S, Suresh S, Bottle A, Loeffler M, Alam M. Venous thromboembolism rates after hip and knee arthroplasty and hip fractures. BMC Musculoskelet Disord. 2020 Feb 12;21(1):95.
3. Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC guidelines for the diagnosis and management of acute pulmonary embolism developed

in collaboration with the European Respiratory Society (ERS). Eur Heart J 2019;00:1-61.

4. Falck-Ytter Y, Francis CW, Johanson NA, Curley C, Dahl OE, Schulman S, Ortel TL, Pauker SG, Colwell CW Jr. Prevention of VTE in orthopedic surgery patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012 Feb;141(2 Suppl):e278S-e325S.
5. Türk Toraks Derneği Pulmoner Tromboemboli tanı ve tedavi uzlaşı raporu 2021.
6. Higuera CA, Krebs VE, Molloy RM, Piuzzi NS. An Update on Venous Thromboembolism Rates and Prophylaxis in Hip and Knee Arthroplasty in 2020. Medicina (Kaunas). 2020 Aug 19;56(9):416.
7. Tzoran I, Saharov G, Brenner B, et al. Silent pulmonary embolism in patients with proximal deep vein thrombosis in the lower limbs. J Thromb Haemost 2012;10:564-71.
8. Bauersachs RM. Clinical presentation of deep vein thrombosis and pulmonary embolism. Best Practice & Research Clinical Hae- matology 2012;25:243-51.
9. Goodacre S, Sampson F, Thomas S, et al. Systematic review and meta-analysis of the diagnostic accuracy of ultrasonography for deep vein thrombosis. BMC Med Imaging 2005;5:6.
10. Mont MA, Jacobs JJ. AAOS clinical practice guideline: preventing venous thromboembolic disease in patients undergoing elective hip and knee arthroplasty. J Am Acad Orthop Surg. 2011 Dec;19(12):777-8.
11. Güven Platformu. Ulusal Venöz Tromboembolizm Profilaksi ve Tedavi Kılavuzu. 2010.
12. Santana DC, Emara AK, Orr MN, Klika AK, Higuera CA, Krebs VE, Molloy RM, Piuzzi NS. An Update on Venous Thromboembolism Rates and Prophylaxis in Hip and Knee Arthroplasty in 2020. Medicina (Kaunas). 2020 Aug 19;56(9):416.
13. Warren JA, Sundaram K, Anis HK, Kamath AF, Higuera CA, Piuzzi NS. Have Venous Thromboembolism Rates Decreased in Total Hip and Knee Arthroplasty? J Arthroplasty. 2020 Jan;35(1):259-264.
14. Clarius M, Heisel C, Breusch S.J. (2005). Pulmonary Embolism in Cemented Total Hip Arthroplasty. In: The Well-Cemented Total Hip Arthroplasty. Springer, Berlin, Heidelberg.

15. Ishaaya E, Tapson VF. Advances in the diagnosis of acute pulmonary embolism. F1000Research 2020;44:1-10.
16. Bernal AG, Fanola C, Bartos JA. American Collage of Cardiology. Management of PE Jan 27, 2020.
17. Pollack CV, Schreiber D, Goldhaber SZ, et al. Clinical characteristics, management, and outcomes of patients diagnosed with acute pulmonary embolism in the emergency department: initial report of EMPEROR (Multicenter Emergency Medicine Pulmonary Embolism in the Real World Registry). J Am Cardiol 2011;57:700-6.
18. Hyers TM. Management of venous thromboembolism: past, present, and future. Arch Intern Med. 2003 Apr 14;163(7):759-68.
19. Paul JD, Cifu AS. Management of Acute Pulmonary Embolism. JAMA. 2020 Aug 11;324(6):597-598.
20. Blitzer RR, Eisenstein S. Venous Thromboembolism and Pulmonary Embolism: Strategies for Prevention and Management. Surg Clin North Am. 2021 Oct;101(5):925-938.
21. Keller K, Hobohm L, Ebner M, et al. Trends in thrombolytic treatment and outcomes of acute pulmonary embolism in Germany. Eur Heart J. 2020 Jan 21;41(4):522-529.
22. Silva F.M, Filho P.D, Eloy T, & Amatuzzi M. (2005). Fat Embolısm: a Revıew for Current Orthopaedıcs Practıce.
23. Rassir R, Schuiling M, Sierevelt IN, van der Hoeven CWP, Nolte PA. What Are the Frequency, Related Mortality, and Factors Associated with Bone Cement Implantation Syndrome in Arthroplasty Surgery? Clin Orthop Relat Res. 2021 Apr 1;479(4):755-763.
24. Donaldson AJ, Thomson HE, Harper NJ, Kenny NW. Bone cement implantation syndrome. Br J Anaesth. 2009;102:12-22.
25. Patel RV, Stygall J, Harrington J, Newman SP, Haddad FS. Cerebral microembolization during primary total hip arthroplasty and neuropsychologic outcome: a pilot study. Clin Orthop Relat Res. 2010 Jun;468(6):1621-9.
26. Clarius M, Heisel C, Breusch S.J. (2005). Pulmonary Embolism in Cemented Total Hip Arthroplasty. In: The Well-Cemented Total Hip Arthroplasty. Springer, Berlin, Heidelberg.
27. Wenda K, Degreif J, Runkel M, Ritter G. Pathogenesis and prophylaxis of circulatory reactions during total hip replacement. Arch Orthop Trauma Surg. 1993;112(6):260-5.

28. Byrick RJ, Mullen JB, Mazer CD, Guest CB. Transpulmonary systemic fat embolism: studies in mongrel dogs after cemented arthroplasty. Am J Respir Crit Care Med. 1994;150:1416–1422.
29. Bengtson A, Larsson M, Gammer W, Heideman M. Anaphylatoxin release in association with methylmethacrylate fixation of hip prostheses. J Bone Joint Surg Am. 1987;69:46-49.
30. Modig J, Busch C, Waernbaum G. Effects of graded infusions of monomethylmethacrylate on coagulation, blood lipids, respiration and circulation. An experimental study in dogs. Clin Orthop Relat Res. 1975;113:187-197.
31. Tryba M, Linde I, Voshage G, Zenz M. Histamine release and cardiovascular reactions to implantation of bone cement during total hip replacement. Article in German. Der Anaesthesist. 1991;40:25-32.
32. Hofmann S, Hopf R, Mayr G, Schlag G, Salzer M. In vivo femoral intramedullary pressure during uncemented hip arthroplasty. Clin Orthop Relat Res. 1999;360:136–146.
33. Kallos T, Enis JE, Gollan F, Davis JH. Intramedullary pressure and pulmonary embolism of femoral medullary contents in dogs during insertion of bone cement and a prosthesis. J Bone Joint Surg Am. 1974;56:1363–1367.
34. de Froidmont S, Bonetti LR, Villaverde RV, del Mar Lesta M, Palmiere C. Postmortem findings in bone cement implantation syndrome-rlated deaths. Am J Forensic Med Pathol. 2014;35:206-211.
35. Orsini EC, Byrick RJ, Mullen JB, Kay JC, Waddell JP. Cardiopulmonary function and pulmonary microemboli during arthroplasty using cemented or non-cemented components. The role of intramedullary pressure. J Bone Joint Surg Am. 1987;69:822-832.
36. Olsen F, Kotyra M, Houltz E, Ricksten SE. Bone cement implantation syndrome in cemented hemiarthroplasty for femoral neck fracture: incidence, risk factors, and effect on outcome. Br J Anaesth. 2014;113:800-806.
37. Bredbacka S, Andreen M, Blomback M, Wykman A. Activation of cascade systems by hip arthroplasty: no difference between fixation with and without cement. Acta Orthop Scand. 1987;58:231–235.
38. Schwarzkopf E, Sachdev R, Flynn J, Boddapati V, Padilla RE, Prince DE. Occurrence, risk factors, and outcomes of bone cement implantation syndrome after hemi and total hip arthroplasty in cancer patients. J Surg Oncol. 2019;120:1008-1015.

39. Modig J, Busch C, Olerud S, Saldeen T, Waernbaum G. Arterial hypotension and hypoxaemia during total hip replacement: the importance of thromboplastic products, fat embolism and acrylic monomers. Acta Anaesthesiol Scand. 1975;19:28-43.
40. Olsen F, Hård Af Segerstad M, Nellgård B, Houltz E, Ricksten SE. The role of bone cement for the development of intraoperative hypotension and hypoxia and its impact on mortality in hemiarthroplasty for femoral neck fractures. Acta Orthop. 2020;91:293-298.
41. Breed AL. Experimental production of vascular hypotension, and bone marrow and fat embolism with methylmethacrylate cement: traumatic hypertension of bone. Clin Orthop Relat Res. 1974;102:227–244.
42. Wheelwright EF, Byrick RJ, Wigglesworth DF, et al.. Hypotension during cemented arthroplasty. Relationship to cardiac output and fat embolism. J Bone J Surg Br. 1993;75:715-723.
43. Gossling HR, Pellegrini VD Jr. Fat embolism syndrome: a review of the pathophysiology and physiological basis of treatment. Clin Orthop Relat Res. 1982 May;(165):68-82. PMID: 7042168.
44. Baker PL, Paxel JA, Pettier LF. Free fatty acids, catecholamine and arterial hypoxia in patients with fat embolism. J Trauma. 1971;11:1026–30.
45. Gelinas JJ, Cherry R, MacDonald SJ. Fat embolism syndrome after cementless total hip arthroplasty. J Arthroplasty. 2000 Sep;15(6):809-13.
46. Issack PS, Lauerman MH, Helfet DL, Sculco TP, Lane JM. Fat embolism and respiratory distress associated with cemented femoral arthroplasty. Am J Orthop (Belle Mead NJ). 2009 Feb;38(2):72-6.
47. Colonna DM, Kilgus D, Brown W, Challa V, Stump DA, Moody DM. Acute brain fat embolization occurring after total hip arthroplasty in the absence of a patent foramen ovale. Anesthesiology. 2002;96:1027–1029.
48. Parvizi J, Holiday AD, Ereth MH, Lewallen DG. The Frank Stinchfield Award. Sudden death during primary hip arthroplasty. Clin Orthop Relat Res. 1999;369:39-48.
49. Kwiatt ME, Seamon MJ. Fat embolism syndrome. Int J Crit Illn Inj Sci. 2013 Jan;3(1):64-8.
50. He Z, Shi Z, Li C, Ni L, Sun Y, Arioli F, Wang Y, Ammirati E, Wang DW. Single-case metanalysis of fat embolism syndrome. Int J Cardiol. 2021 Dec 15;345:111-117.
51. T. Kainoh, H. Iriyama, A. Komori, D. Saitoh, T. Naito, T. Abe, Risk factors of fat embolism syndrome after trauma: a nested case-control study with

the use of a Nationwide trauma registry in Japan, Chest 159 (2021) 1064–1071.

52. Kambayashi J, Sakon M, Yokota M, Shiba E, Kawasaki T, Mori T. Activation of coagulation and fibrinolysis during surgery, analyzed by molecular markers. Thromb Res. 1990;60:157–167.
53. Gammer W, Bengtson A, Heideman M. Inhibition of complement activation by high-dose corticosteroids in total hip arthroplasty. Clin Orthop Relat Res. 1988:205-209.
54. Tronzo RG, Kallos T, Wyche MQ. Elevation of intramedullary pressure when methylmethacrylate is inserted in total hip arthroplasty. J Bone Joint Surg Am. 1974;56:714–718.
55. Griffiths R, White S, Moppett I, et al.. Safety guideline: reducing the risk from cemented hemiarthroplasty for hip fracture 2015: Association of Anaesthetists of Great Britain and Ireland British Orthopaedic Association, British Geriatric Society. Anaesthesia. 2015;70:623-626.
56. Pitto RP, Koessler M, Kuehle JW. Comparison of fixation of the femoral component without cement and fixation with use of a bone-vacuum cementing technique for the prevention of fat embolism during total hip arthroplasty. A prospective, randomized clinical trial. J Bone Joint Surg Am. 1999 Jun;81(6):831-43.
57. Sherman RM, Byrick RJ, Kay JC, Sullivan TR, Waddell JP. The role of lavage in preventing hemodynamic and blood-gas changes during cemented arthroplasty. J Bone Joint Surg Am. 1983 Apr;65(4):500-6.

CHAPTER III

SHOULDER ARTHROSCOPY COMPLICATIONS

Mesut TIKMAN[1] & Yılmaz ERGİŞİ[2]

[1](Orthopedics and Traumatology Specialist), Gölbaşı Şehit Ahmet Özsoy State Hospital, Ankara, e-mail: mesuttikman@gmail.com
ORCID: 0000-0003-1671-9661

[2](Asst. Prof. Dr.; Orthopedics and Traumatology Specialist,), Karabuk University Training and Research Hospital, Karabük
e-mail: yilmazergisi@gmail.com
ORCID: 0000-0003-2456-6802

1. Introduction

Shoulder arthroscopy has become a more common surgical intervention in recent years. With arthroscopic shoulder surgeries, shoulder anatomy and pathologies have become more understandable. However, as it has become a popular surgical procedure, its complications are becoming more familiar. It is known that the complication rate after shoulder arthroscopy is between 0.99% and 9.5%. Proper patient selection, appropriate techniques, and encouraging rehabilitation in the postoperative period can reduce the likelihood of complications[1,2].

2. Infections

Infection can occur after arthroscopic surgery, albeit a little. Infection can be seen at a rate of 0.16-0.8% after primary shoulder arthroscopy and 2.1% after revision surgeries [1]. The most common causative agents are Proprionobacterium acnes and staphylococci. It should be kept in mind that at least seven days of incubation is required for Propionibacterium acnes to grow in culture. To reduce the skin colonization of P. acnes, the combination of benzoyl peroxide, and clindamycin phosphate was applied to the skin. As a result, it decreased the colonization rates of P. acnes[1]. Patient-related and surgical causes of infection, obesity, male gender and advanced age were determined as patient-dependent risk factors. In addition, short surgical time and prophylactic antibiotic therapy are surgery-related risk factors known to reduce the incidence of postoperative infection[1,3]The most common complaint in the presence of infection after shoulder arthroscopy is pain. In addition to pain, hyperemia, swelling, and temperature increase in the shoulder are also seen. Among the diagnostic laboratory tests, increased white blood cell count, increased erythrocyte sedimentation rate, and high C-reactive protein is findings in favor of infection. Other tests used to diagnose infection include the investigation of microbial agents by gram staining in synovial fluid with a puncture from the glenohumeral joint and detecting white blood cell count. Imaging is rarely required for diagnosis. Magnetic resonance imaging can help determine the location of the abscess or when osteomyelitis is suspected. The diagnosis is made with clinical and laboratory findings. While treatment can be provided with antibiotics appropriate for the causative agent in superficial infections, surgical debridement with antibiotic therapy is required in septic arthritis or late infections. Debridement can be done open or arthroscopically. Implants associated with infection should be removed[4].

3. Anesthesia-related complications

General anesthetic complications associated with any surgical procedure are also applicable to shoulder arthroscopy. Under general anesthesia, the most important thing is to provide hypotensive anesthesia in a way that does not cause impaired cerebral perfusion. In addition, fatal air embolism and pneumothorax, pulmonary edema, and pneumomediastinum can be seen during arthroscopic shoulder procedures, possibly due to CO2 use. Shoulder arthroscopy can also be performed with regional anesthesia, especially for intraoperative management and postoperative pain control; the use of interscalene anesthesia has increased[5]. Regional anesthesia is safe and effective pain control. Still, there have been rare reports of seizures, cardiovascular collapse, and severe respiratory distress secondary to pneumothorax, secondary to systemic anesthetic toxicity. It also carries a risk of neurological complications secondary to regional blockades and temporary or permanent brachial plexus injuries.[2,3].

4. Deep vein thrombosis and pulmonary

The overall incidence of venous thromboembolism (VTE) reported in studies after shoulder surgery ranges from 0.02% to 13% [6]. Takahashi et al.found that when patients who underwent shoulder arthroscopy were examined by ultrasonography, deep vein thrombosis was found in 5.7,% and pulmonary embolism was observed in 0.6% [7]. Prolongation of shoulder arthroscopy, advanced age, male gender, and obesity increase the risk of VTE [6]. Unlike these, the general risk factors for VTE are dehydration, admission to intensive care, active malignancy or active cancer treatment, known history of thrombophilia, previous VTE, history of a first-degree family member with VTE, use of estrogen-containing contraceptives, hormone replacement therapy, and pregnancy or pregnancy. Having recently given birth is also among the risk factors. Unfortunately, there is no consensus in the literature on prophylaxis to prevent VTE. According to the risk-benefit analysis, mechanical prophylaxis is a universally accepted prophylaxis method. Chemical prophylaxis with low molecular weight heparin or heparin should be considered in patients with risk factors for VTE but who have taken necessary precautions against the risk of bleeding complications [8].

5. Complex regional pain syndrome

The most common symptom after shoulder arthroscopy is pain. While the pain is at its highest level in the early postoperative period, the severity of the pain decreases over time. In shoulder arthroscopy surgery, the most painful procedure is reported as rotator cuff repair, while the minor painful procedure is reported as instability surgery.

Complex regional pain syndrome (CRPS) is a chronic pain that usually affects an arm or leg. CRPS typically develops after an injury, surgery, stroke, or heart attack. CRPS findings; severe pain, allodynia, edema, local hyperthermia, trophic disorders, and skin dyschromia. Pain is not proportional to the severity of the initial injury. CRPS can be seen at a rate of 11-13%, secondary to the pain felt after shoulder arthroscopy, but the exact incidence is unknown due to the variability in the diagnostic criteria [9]. There is no specific treatment protocol for the treatment of CRPS. Despite the treatments applied, only 10% of the patients reported having complete resolution of symptoms.

6. Complications associated with shoulder arthroscopy

6.1. Patient position

The patient positions used in shoulder arthroscopy are beach chair and lateral decubitus. Because traction is applied to the arm in the lateral decubitus position, the glenohumeral joint and the subacromial space are easily observed. In addition, lateral distraction facilitates access to the posterior and inferior parts of the glenohumeral joint, and imbrication processes can be performed more efficiently. Complications can occur in any position. Their functional superiority over each other is debatable. The most frequently reported complication is nerve damage with varying rates. Nerve damage in the brachial plexus due to traction applied to the extremity, especially in the lateral decubitus position, can range from temporary paresthesia to permanent neuropraxia. While the rate of temporary paraesthesia in the lateral decubitus position is 10%, this complication is observed below 1% in the chaise longue position. This complication is thought to be caused by the tension in the brachial plexus due to the traction applied during the surgery and the arm's position[10]. A study showed that the best intra-articular visual field is provided in 45" flexion-90° abduction or 45° flexion-0' abduction positions, while the tension on the brachial plexus is at the lowest level [5]. The risk of traction-related nerve damage can be reduced by positioning the patient appropriately, using less than seven kilograms of weight, keeping the surgical time short, and using the correct portal position. Apart from neurapraxia, there are also rare complications related to patient position. In both positions, the peroneal nerve may be exposed to pressure. Regarding possible skin problems and peroneal nerve complications, the lower lateral malleolus and lateral knee should be supported in the lateral decubitus position. Airway obstruction is secondary to fluid extraction in the lateral decubitus position and musculocutaneous nerve injury. Cerebral hypo-perfusion, air embolism, pneumothorax, ophthalmoplegia, and cervical neuropraxia have also been reported in the beach chair position. The position of the head is critical in the beach chair position. As the amount of flexion, rotation, and lateral deviation increases, the risk of neurological complications also increases. C5-6 transient quadriparesis has been reported due to prolonged flexion of the cervical vertebrae in the beach chair position. Cerebral infarcts may occur due to decreased vertebral artery blood flow when the head is in rotation and extension. The pressure on the brachial plexus and artery should be reduced by placing axillary support on the thoracic wall. The risk of complications will be reduced by considering the potential complications associated with the patient's position and taking the necessary precautions[11].

6.2. Nerve damage

One crucial point to remember in the operations performed on the glenoid is that the suprascapular nerve is closer than 2 cm to the glenoid articular surface.

The probability of injury to this nerve due to arthroscopic surgery has been seen between 2-28%. Due to damage to this nerve, atrophy can be observed in the supraspinatus and infraspinatus muscles. It is located at least 2.3 cm medial from the upper edge of the glenoid and 1.4 cm medial from the scapula's base. The risk of this complication is significantly reduced by controlled drilling and using suture anchors away from these distances [12]. Another nerve to be considered during arthroscopy is the axillary nerve. The axillary nerve is at risk when performing a capsular incision in the axillary space during inferior capsular displacement applied in atraumatic or multidirectional instabilities. To minimize the risk of nerve damage, an incision should be made at the glenoid edge, where the axillary nerve is safest, in all arm positions[13].

6.3. Portal placement

Appropriate positioning of the portals from the right place is perhaps the most crucial step of shoulder arthroscopy. Both provide a good field of view, and the correct placement of the implants depends on this. One of the complications that can be observed during portal opening is neurovascular injury. In this situation, which can be observed most frequently during the lateral portal incision, cutaneous nerve damage and axillary nerve damage can occur in 7% of cases. Although the possibility of neurovascular injury during the posterior portal incision is very low, axillary nerve damage is possible if this portal is placed too low, and suprascapular nerve damage if set too high[12]. In a study, the most commonly used primary arthroscopy portals were evaluated with the outside-in technique. It was determined that all portals were at least 20 mm away from the neurovascular structures. It was reported that portal opening with the outside-in method is safe in shoulder arthroscopy. A cannula would be an excellent choice to reduce fluid extravasation and related complications. Another complication is an injury to the tendinous part of the supraspinatus due to the incorrect opening of the Wilmington portal used to repair posterosuperior labrum tears. All complications due to portal placement can be reduced to the minimum possible level with good anatomy knowledge[5].

6.4. Arthroscopic visual field

Deterioration of the field of view due to bleeding in the surgical area is the most common and perhaps the most annoying complication. As a result, the increased risk of additional complications is an important detail. The anticoagulant therapy used by the patients, while intervening with the coracoacromial ligament in the subacromial region, the injury of the coracoacromial artery may cause bleeding and should be kept in mind. Hypotensive anesthesia, appropriate pump pressure, and radiofrequency ablation of the bleeding tissue without wasting time are critical in maintaining the field of view.

6.5. Technical errors

In the first step of the shoulder arthroscopy procedure, osteochondral lesions may occur due to the failure of the trocar to be placed with the appropriate technique. Choosing a blunt-tipped trocar and the proper technique is essential to prevent this. Technical errors play an essential role in the failure of arthroscopic repair. Errors in implant selection and placement are the leading ones. It is essential to place the implant at the most appropriate angle to hold on to the placed tissue and withstand the tension created by the repair tissue. Another critical issue is the knotting technique. The knot should generate enough force in the tissue, be of proper tension, and have a low profile. The sliding knot should be slid towards the anchor, the first knot should be well tightened, and no excessive tension should be created in the soft tissue. Finally, it should not be forgotten that the subscapularis tendon may be damaged during anterior capsular release, which is preferred, especially in frozen shoulder cases. Complications observed during acromioplasty are inadequate decompression due to incomplete resection or instability due to excessive resection. The field of view must be provided with adequate bursectomy and hemostasis for appropriate acromioplasty. Similarly, in arthroscopic distal end resection of the clavicle applied in acromioclavicular joint degeneration, poor results can be observed due to incomplete resection or instability due to excessive resection. Therefore, keeping the resection amount at 10-15 mm and protecting the coracoclavicular and superior acromioclavicular ligament is crucial. Superomedial dislocation of the humeral head can be seen with coracoacromial arch excision performed in patients without a functional rotator cuff, and pseudo paralysis may develop. In these patients, preserving the coracoacromial arch and avoiding acromioplasty is vital for stability.

6.6. Implant failure

One of the complications that can be observed during or after arthroscopic shoulder surgery is implant failure. This may occur especially in elderly patients with poor bone quality, inability to master anatomy, and difficulty placing an implant without proper vision. Although metal implants have advantages in radiographic evaluation of implant placement and better attachment in osteoporotic patients, there is a risk of artifact formation and displacement in magnetic resonance imaging. In addition, complications such as osteolysis and chronic synovitis associated with free body formation after partial absorption and the resulting biological response can be seen in absorbable implants. Therefore, Bioinert and biocomposite implants have been developed to minimize the complications in absorbable implants [3]. It should be kept in mind that loose body formation and secondary cartilage damage may occur after using these radiolucent implants, and this complication should definitely be considered in patients with mechanical symptoms and pain.

A study shows that up to 22% of re-rupture cases after rotator cuff repair in 2-year follow-ups are one of the complications observed after rotator cuff repair. The tear size, the tissue's quality, the amount of retraction, and the patient's age are essential regarding the risk of re-tearing. Recurrence after arthroscopic instability surgeries is a more common complication than other surgical procedures. In the literature, recurrences between 0% and 60% have been reported with different techniques [5]. Implant failure, technical errors, presence of other pathologies, tissue quality, patient compliance, functional expectations, and contact sports are the factors affecting the frequency of recurrence.

6.7. Cartilage damage

It was previously mentioned that chondral damage might occur with a wrong approach during the first insertion of the joint during shoulder arthroscopy. In addition, cartilage damage can be observed in cases such as not placing the implants at the appropriate depth during the rotator cuff repair or placing them without paying attention to the cartilage border. Therefore, always using an appropriate portal and field of view is essential to avoid such situations.

Chondrolysis is another complication that can be seen after shoulder arthroscopy. As a result of the studies, it was stated that chondrolysis was observed at rates of up to one in ten.

Although the exact cause of chondrolysis cannot be clarified, it has been reported that it can be observed in cases where the use of local anesthetic for the joint exceeds 48 hours, in cases where the temperature of the intra-articular fluid exceeds 45 degrees during the use of thermal probes, in case of trauma, due to absorbable implants and in cases of infection[3].

6.8. Joint stiffnes

The most common complication after shoulder arthroscopy is joint stiffness/ arthrofibrosis, which causes severe morbidity and loss of function. In the literature, joint stiffness after shoulder arthroscopy is reported to be between 2.8% and 15%. In addition, temporary stiffness can be observed in 10% of cases after arthroscopic rotator cuff repair, and only 3% of these require capsular release[14]. There is a negative correlation between the rotator cuff tear size and the prevalence of joint stiffness. Joint stiffness clinically presents a decrease in active and passive range of motion, especially in external rotation. The most crucial cause of post-surgical joint stiffness is patient non-compliance and inadequate rehabilitation. Early range of motion exercises reduce the risk of joint stiffness [15]. It should be kept in mind that failure may occur in the repair area due to early and aggressive rehabilitation, especially in patients undergoing repair. If joint stiffness persists for six months despite postoperative rehabilitation, arthroscopic release can be planned. In the preoperative period, patients' range of motion should be well evaluated, and pathologies that directly affect the surgical outcome, such as adhesive capsulitis, should be kept in mind. Patients with preoperative limitations of joint movement are also more likely to have limitations after surgery. Rotator cuff and labrum repair should be avoided in patients with suspected adhesive capsulitis. Surgery should wait until the full passive range of motion is achieved to reduce the risk of this complication in acute traumatic tears. Since there is increased proteinase activity in the early period, the risk of arthrofibrosis is high. Middle and inferior glenohumeral ligament tension can result in the limitation of external rotation. It is essential to evaluate patients from this point of view before surgery, especially in instability surgery[16].

7. Reasons for failure

The shoulder joint is separated from other joints by its wide range of motion. The complexity of the anatomy may make it challenging to recognize pathologies during arthroscopic surgery. Some variations, such as the Buford complex and the Sublabral space, are essential to identify. Treatments that are not known may cause failure. It is crucial to detect pathologies with physical examination findings in the clinic and understand the macro anatomy. It is necessary to evaluate patients by combining them with radiological images. Otherwise, various pathologies may not be recognized, and failures may occur due to deficiencies in the treatment.

During shoulder arthroscopy surgery, the most appropriate adjustment of the patient's position is necessary so that the portals required for the surgery can be placed at the appropriate point. Portal placement, which is already difficult due to the wide soft tissue cover and few bone markers due to the inappropriate position, may become even more difficult.

Placing the implants used to repair shoulder pathologies in the right spot and mastery of knotting techniques are among the factors that affect success. When sufficient technical knowledge and skills are unavailable, situations such as stripping the threads from the anchor and pulling the anchor from the bone if it is not placed correctly can be encountered.

Appropriate patient selection, detailed pre-operative examination and evaluation, and the surgery's technical studies should be carefully made to encounter fewer complications after shoulder arthroscopy surgery. Taking the necessary precautions to provide the right equipment makes the surgery more manageable, and complications may occur due to missing equipment. In addition, conditions must be provided for effective physiotherapy after surgery. Inadequate post-operative rehabilitation can lead to failure.

References

1. Sircana G, Passiatore M, Capasso L, Saccomanno MF, Maccauro G. Infections in arthroscopy. *Eur Rev Med Pharmacol Sci*. 2019;23(2 Suppl):279-287. doi:10.26355/EURREV_201904_17502
2. Weber SC, Abrams JS, Nottage WM. Complications associated with arthroscopic shoulder surgery. *Arthroscopy*. 2002;18(2 Suppl 1):88-95. doi:10.1053/JARS.2002.31801
3. Noud PH, Esch J. Complications of arthroscopic shoulder surgery. *Sports Med Arthrosc Rev*. 2013;21(2):89-96. doi:10.1097/JSA.0B013E31829006F0
4. Bauer T, Boisrenoult P, Jenny JY. Post-arthroscopy septic arthritis: Current data and practical recommendations. *Orthop Traumatol Surg Res*. 2015;101(8 Suppl):S347-S350. doi:10.1016/J.OTSR.2015.09.004
5. Paxton ES, Backus J, Keener J, Brophy RH. Shoulder arthroscopy: basic principles of positioning, anesthesia, and portal anatomy. *J Am Acad Orthop Surg*. 2013;21(6):332-342. doi:10.5435/JAAOS-21-06-332
6. Ji L, Lyu C lei, Feng M, Qiang H. Asymptomatic Pulmonary Embolism After Shoulder Arthroscopy: Case Report and Literature Review. *Orthop Surg*. 2021;13(3):1119-1125. doi:10.1111/OS.12982
7. Takahashi H, Yamamoto N, Nagamoto H, Sano H, Tanaka M, Itoi E. Venous thromboembolism after elective shoulder surgery: a prospective cohort study of 175 patients. *J Shoulder Elbow Surg*. 2014;23(5):605-612. doi:10.1016/J.JSE.2014.01.054

8. Aibinder WR, Sanchez-Sotelo J. Venous Thromboembolism Prophylaxis in Shoulder Surgery. *Orthop Clin North Am*. 2018;49(2):257-263. doi:10.1016/J.OCL.2017.11.012
9. Martel M, Laumonerie P, Pecourneau V, Ancelin D, Mansat P, Bonnevialle N. Type 1 Complex Regional Pain Syndrome After Subacromial Shoulder Surgery: Incidence and Risk Factor Analysis. *Indian J Orthop*. 2020;54(Suppl 1):210-215. doi:10.1007/S43465-020-00174-8
10. Li X, Eichinger JK, Hartshorn T, Zhou H, Matzkin EG, Warner JP. A comparison of the lateral decubitus and beach-chair positions for shoulder surgery: Advantages and complications. *Journal of the American Academy of Orthopaedic Surgeons*. 2015;23(1):18-28. doi:10.5435/JAAOS-23-01-18
11. Rojas J, Familiari F, Bitzer A, Srikumaran U, Papalia R, McFarland EG. Patient Positioning in Shoulder Arthroscopy: Which is Best? *Joints*. 2019;7(2):46. doi:10.1055/S-0039-1697606
12. Scully WF, Wilson DJ, Parada SA, Arrington ED. Iatrogenic nerve injuries in shoulder surgery. *J Am Acad Orthop Surg*. 2013;21(12):717-726. doi:10.5435/JAAOS-21-12-717
13. Moen TC, Rudolph GH, Caswell K, Espinoza C, Burkhead WZ, Krishnan SG. Complications of shoulder arthroscopy. *J Am Acad Orthop Surg*. 2014;22(7):410-419. doi:10.5435/JAAOS-22-07-410
14. Cucchi D, Marmotti A, de Giorgi S, et al. Risk Factors for Shoulder Stiffness: Current Concepts. *Joints*. 2017;5(4):217-223. doi:10.1055/S-0037-1608951
15. Chang KV, Hung CY, Han DS, Chen WS, Wang TG, Chien KL. Early Versus Delayed Passive Range of Motion Exercise for Arthroscopic Rotator Cuff Repair: A Meta-analysis of Randomized Controlled Trials. *Am J Sports Med*. 2015;43(5):1265-1273. doi:10.1177/0363546514544698
16. Itoi E, Arce G, Bain GI, et al. Shoulder Stiffness: Current Concepts and Concerns. *Arthroscopy*. 2016;32(7):1402-1414. doi:10.1016/J.ARTHRO.2016.03.024

CHAPTER IV

COMPLICATIONS OF PRIMARY KNEE ARTHROPLASTY

Mesut TIKMAN

(Orthopedics and Traumatology Specialist), Gölbaşı Şehit Ahmet Özsoy State Hospital, Ankara, e-mail: mesuttikman@gmail.com

ORCID: 0000-0003-1671-9661

1. Introduction

Total knee arthroplasty (TKA) is a major surgical procedure that can be performed to relieve pain from end-stage osteoarthritis. In addition, TKA can be performed for rheumatoid arthritis, hemophilic arthropathy, traumatic arthritis, and neuropathic arthropathy. TKA is one of the safe, effective, and low-cost operations practiced worldwide (Lee & Goodman, 2008).

TKA applications have been increasing over the years with the increase in life expectancy, the advancement of surgical techniques, and the expanding range of indications. Although complications due to TKA are relatively rare, it is essential to know them in terms of causing mortality and morbidity in patients. Complications may occur during and after TKA. Complications of total knee arthroplasty (TKA) include anaesthesia-related risks, worsening of comorbid medical issues, drug therapies and allergic reactions, and complications related more specifically to TKA. Efforts should be made to minimize the risk of complications through appropriate patient selection and optimization, careful surgical technique, and thoughtful postoperative management. This topic discusses complications of total knee arthroplasty.

2. Early complications

2.1. Thromboembolism

Thromboembolism is one of the severe complications after TKA, with high mortality and morbidity. The main risk factors that increase the incidence of thromboembolism are; Previous venous thromboembolism, varicose veins, congestive heart failure, body mass index greater than 30, malignancy, hypertension, advanced age and female gender, previous myocardial infarction, chronic renal failure, prolonged immobilization, estrogen use, prolongation of the surgical time risk thromboembolism (Zhang et al., 2015). It usually originates from veins in the calf. Pulmonary embolism is the most dreaded and fatal complication of thromboembolism. [1].

It is known that when mechanical and pharmacological DVT prophylaxis is applied after TKA, the risk of thromboembolism is reduced. In pharmacological prophylaxis, low molecular weight heparin is the most preferred agent (Flevas et al., 2018).

When no prophylaxis for deep vein thrombosis is performed, 40-80% of deep vein thrombosis occurs after TKA (Friedman et al., 2011).

While mortality rates of DVT in the first month are 6%, it is approximately 12% in patients with pulmonary embolism (Flevas et al., 2018).

In the case of DVT or PE, thrombolytic and anticoagulant therapies come to mind as the first treatment. However, thrombolytic therapy has no place in major orthopedic surgeries such as TKA. Anticoagulant treatment can be applied for DVT and PE developing after TKA. However, anticoagulant use is also associated with a high risk of bleeding. Therefore, the prophylactic dose of anticoagulant therapy should be started 12–24 weeks after surgery. It is recommended to start in the first hour and not to switch to the treatment dose before 48-72 hours. Inferior vena cava filters can be applied in patients with proximal vein thrombosis who cannot take anticoagulants due to the risk of bleeding. (Streiff et al., 2016).

2.2. Peroneal nerve injury

Peroneal nerve injury after total knee arthroplasty is a rare complication. Peroneal nerve damage, seen in 0.01% to 4.3% of cases, adversely affects the functions of the related extremity. Although the factors that increase the risk of peroneal nerve injury are preoperative genu valgum deformity, epidural anesthesia, preoperative flexion contracture, high body mass index, and diabetes history, there is no consensus in the literature yet on risk factors(Carender et al. 2020). Lumbar spine disease (lumbar stenosis, previous lumbar surgery) and a tourniquet close to the knee has also been associated with peroneal nerve damage [3]. When drop foot develops due to peroneal nerve damage, 20% to 100% of patients can recover without intervention. The literature recommends prophylactic neurolysis in cases with valgus deformity requiring excessive correction. However, neurolysis is also recommended since hematoma is detected in the relevant area in the early postoperative period (Carender et al. 2020).

2.3. Arterial injury

After total knee arthroplasty, simple hemorrhages may occur, as well as severe bleeding that may lead to limb loss, although rarely. The most common vascular injuries are the popliteal artery, geniculate artery, superficial femoral artery, and anterior tibial artery. The incidence of iatrogenic popliteal artery injury after total knee arthroplasty was 0.057% [4,5]. Risk factors for arterial injury are revision surgery, peripheral vascular disease, weight loss (weakness), renal failure, coagulopathy, and malignancy. In the case of arterial injury, the length of hospital stay is prolonged, and morbidity increases [6]. Treatment options are primary repair, thrombectomy, bypass with a saphenous vein graft, endovascular stenting, or embolization(Gosslau et al. 2022).

2.4. Wound healing problem

Surgical site infections, which occur in a wide range from the separation of the wound lips to wound site discharge, are complications that can cause surgical wound ulcers and subsequent wound opening [8]. The presence of previous incisions in the surgical field, inadequate full-thickness skin flaps, inadequate bleeding control, and tight skin closure are predisposing factors for wound problems. Diabetes mellitus is a risk factor in itself. These problems can be avoided by applying appropriate techniques and close observation of the patient in case of discharge at the wound site or detecting areas of necrosis at the wound site. Care should be taken, and the cause of the problem should be determined and intervened. Otherwise, it will cause deep tissue infection and failure of TKA. When there is a problem in the wound site, the problem can be solved by local interventions, as well as cases requiring reconstruction with skin flaps or medial gastrocnemius muscle flaps can be seen [9](Garbedian, Sternheim, and Backstein 2011).

2.5. Surgical site infection

Wound infections are a severe condition that prolongs hospitalizations, delay healing, and leads to undesirable outcomes such as periprosthetic infection. The culprit of wound infection is sought in the operating room environment and skin flora. For this reason, a laminar flow, air-filtered operating room environment has been developed, and chemicals such as povidone-iodine and chlorhexidine are used before the patient takes a shower the night before to provide skin antisepsis[11]. In addition, factors such as the presence of bacteria in the patient's urinary system before surgery and being a methicillin-resistant staphylococcus carrier are also blamed [12,13].

2.6. İntraoperative fracture

During total knee arthroplasty, unintentional fractures may occur while osteotomies of the tibia, femur, and patella are performed. When the fracture occurs, the need for additional surgical intervention increases the patient's morbidity. Pre-existing osteoporosis, rheumatoid arthritis, chronic steroid use, advanced age, and female gender are the factors that increase the risk[14]. Anterior femoral notching of more than 3 mm during femoral cuts increases the risk of supracondylar periprosthetic femur fracture (Stamiris et al. 2022). Depending on the location and type of the fractures, they can be treated with fixation of the fracture or revision arthroplasty. [16]

2.7. Ligament injury

After total knee arthroplasty, the functional ligaments are medial and lateral collateral. The medial collateral ligament is the rarely injured ligament. The incidence of intraoperative medial collateral ligament injury is between 0.5-3%. Injury may occur while releasing the medial collateral ligament to correct the varus deformity, or injury may occur with saws. Depending on the extent of the injury, in the presence of instability, the ligament can be strengthened with a primary repair or tendon grafting. If sufficient stability cannot be achieved, polyethylene thickness is increased, and constrained TKA may be preferred if it is insufficient. There is no consensus yet on the treatment of medial collateral ligament injuries [17].

3. Intermediate-long term complication

3.1. Aseptic loosening

Aseptic loosening is defined as the failure of prostheses without the presence of a mechanical cause or infection, which could result in instability. It is mainly associated with osteolysis and inflammatory response within the knee. Loosening may occur between the prosthesis and the cement, as well as between the cement and the bone. Although the primary mechanism is not known precisely, it is thought that osteolysis secondary to inflammation caused by the wear particles of polyethylene or cement causes loosening. Aseptic loosening is seen in 20% of TKA's [18]. Loosening may manifest as a radiolucent line between the prosthesis and the bone in the radiological evaluation performed with direct radiography, and the primary diagnosis is made by excluding septic causes while there is pain and instability in the clinic [18]. Conditions that cause aseptic loosening can be classified as patient-related, implant-related, and surgery-related causes. It is known that male gender, hyperactive joint, and osteopenia are patient-related factors that increase loosening[19]. . Implant design can also be effective in loosening; It has been reported that there is more loosening in constrained prostheses and cementless designs that allow forward flexion. Among the surgical factors, it was determined that malalignment, especially in the knee, may cause early loosening of the varus alignment [20] The diagnosis is made in the presence of pain and instability in the clinic and radiologically in the presence of a radiolucent area between the implant and the bone. The single photon emission computed tomography has also gained importance in evaluating painful prostheses. Revision knee arthroplasty is performed in the treatment.

3.2. Instability

After total knee arthroplasty, component (femoral, tibial, patellar components of the knee brace and insert) loosening/breakage and polyethylene wear may cause instability. In some prosthetic designs, patients may have mid-flexion (between 0-90 degrees) instability, especially when getting up from a chair and climbing stairs. While evaluating instability, muscle weakness, implant infection, component breakage, and patellofemoral joint relationship should be considered. Instability is the cause of approximately 3% of revision surgery. It is treated with various revision knee arthroplasty surgeries[21].

3.3. Periprosthetic infection

Bacteria are most commonly responsible for the infective picture that develops in the joint after knee arthroplasty, followed mainly by staphylococci, streptococci, and gram-negative rods. As can be understood from the factors, most agents are members of the skin flora [22]. Previous knee septic arthritis, diabetes mellitus, rheumatoid arthritis, obesity, smoking, HIV, malnutrition, fungal infestation in distant organs, and organ transplantation increase the risk of periprosthetic joint infection (Lima et al. 2013). Periprosthetic joint infection can occur by direct contamination, hematogenous, and reactivation of the occult agent. Periprosthetic joint infection is seen in 1.55% of patients after primary knee arthroplasty. In the clinic, heat, hyperemia, pain, and limitation of movement can be seen in the joint. In order to diagnose with laboratory tests, the recommendations of the "periprosthetic joint infection guideline" are followed. Presence of the sinus tract from the joint, two positive cultures taken from the joint, and detection of the pathogen make a definitive diagnosis, while high serum CRP and sedimentation are systemic findings of infection. The examination of the synovial fluid sample provides valuable data in the diagnosis of periprosthetic joint infection. Synovial fluid sample culture should be cultured; the agent should be searched in gram staining, and biochemical analyses should be done; High leukocyte esterase, neutrophil count, IL-6 level, Alpha-defensin, and CRP elevation are in favor of infection. Factors with low virulence may not yield significant findings in these studies and clinically. When a low virulence agent is suspected, antibiotherapy should be discontinued for two weeks, and culture incubation periods should be extended before samples are taken to obtain more accurate results. Sonication is the most effective way to detect the causative agent. When periprosthetic joint infection is detected in the early period, treatment can be provided with antibiotherapy, polyethylene replacement, and severe debridement, while revision arthroplasty with 2-stage surgery is required in the late period(Izakovicova, Borens, and Trampuz 2019).

3.4. Periprosthetic fracture

Intraoperative fractures were reviewed above. This section will examine fractures occurring around the prosthesis in the postoperative period. Fractures in the tibia, femur, and patella may occur after knee arthroplasty. Periprosthetic fractures can also occur after high-energy trauma or in osteoporotic patients after low-energy trauma. Supracondylar femur fractures are frequently encountered in 0.2-1% of all THA patients. Female patients with osteoporosis are the most likely victims of a supracondylar femur fracture. Anterior femoral notching is also known to increase the risk of fracture. While conservative treatments (splint, rest, traction) are applied in patients with nondisplaced femur fractures, and the femoral component of TKA remains stable, osteosynthesis with open reduction and internal fixation is required in displaced fractures. Retrograde femoral intramedullary nails or plates can be used for osteosynthesis. Revision arthroplasty may be necessary for unfixable multi-component fractures or where bone loss is excessive. Tibial fractures are seen between 0.07-0.1%. Nonoperative treatments can be applied depending on the fracture size, shape, and stability of the prosthesis, as well as minimally invasive plate osteosynthesis or revision knee arthroplasty. Patellar fractures can be seen at a rate of 0.2-21%. Half of the cases are caused by direct trauma. Moreover, the patellar fracture may occur after resurfacing with the patellar component, obesity, rheumatoid arthritis, and exposure to corticosteroids. Depending on the fracture type, non-operative, open reduction internal fixation, partial or complete patellectomy, or excision of the patellar component can be applied(Lombardo et al. 2020; Yoo and Kim 2015).

3.5. Patellofemoral complications

Despite improvements in technique and implant designs, patellofemoral complications still occur in 10% of THA patients. These are patellar dislocation, patellar fracture, crepitation or patellar clunk, osteonecrosis, patellofemoral impingement, and anterior knee pain. Patella fracture can be treated non-operatively if the extensor mechanism is not impaired and the patellar component is stable, but surgical intervention is required otherwise. In case of patellar dislocation, it should be determined whether there is a misalignment, and if there is, a revision should be made. If there is patellar dislocation despite the absence of malalignment, the patella should be medialized with tibial tubercle osteotomy, or the medial patellofemoral ligament should be checked and reconstructed if necessary. Pathologies that may cause this in the case of patellar clunk or crepitation should be determined; nodular scar tissue on the patella and an intra-articular fibrous band are considered. Rehabilitation treatment can treat patellofemoral problems; in advanced cases, pathologies can be treated arthroscopically. If patellar impingement with TKA is present, malalignment status should be checked, and if there is, revision TKA should be performed. If there is no malalignment, lateral fasciectomy or lateral retinacular release may be considered. The way to prevent this is to make appropriate intraoperative incisions and carefully select the correct component size. If a cause of anterior knee pain cannot be found, it is best to choose conservative methods (Putman et al. 2019). Implant-related factors are also thought to affect patellofemoral problems. Various prosthesis manufacturers are trying to create a more compatible patellar component with the femoral groove by designing different patellar components. In addition, although the patellar component is designed in various ways, the dome-shaped polyethylene patellar component is most often used. Loosening or failure of the patellar component is attributed to the patient being obese, lateral retinacular release, and patellar osteonecrosis [28].

4. Late Complications

4.1. Arthrofibrosis

Arthrofibrosis is a well-known complication of injury or trauma characterized by excessive production of fibrous scar tissue due to an exaggerated inflammatory response in a joint. The clinical finding of arthrofibrosis is passive knee flexion of fewer than 90 degrees and inability to perform full extension. In addition, diffuse edema, the tension in the knee, and ambiguous temperature increase can also be seen. Weakness of quadriceps muscle strength and anterior knee pain may accompany this picture. Arthrofibrosis is vital in terms of causing disability in patients. Its etiology is multifactorial; preoperative, intraoperative, postoperative, and patient-related risk factors have been defined [29]. In the literature, joint stiffness varies between 1.2% and 17%. One-fourth of these patients may require surgical intervention to achieve a sufficient range of motion. There may be stiffness in the knee secondary to patient-induced surgical technique or postoperative complications. Preoperative range of motion of the patient, systemic rheumatological diseases, previous knee surgery, smoking, arthrosis after septic arthritis, or low pain tolerance can be counted as patient-related factors. In addition, keloid or hypertrophic scarring seen in the patient after surgery may also limit joint movements. Surgical risks are; are the factors that disrupt the kinematics of the knee, such as stiff knee, improper placement of components, and inability to maintain soft tissue balance. Early movement in the postoperative period is thought to reduce arthrofibrosis. Nonoperative treatment begins with preoperative patient education and continues with physiotherapy. Increased pain in the perioperative period is associated with decreased range of motion, and appropriate use of non-steroidal anti-inflammatory drugs may reduce the risk of arthrofibrosis. If a patient does not respond to conservative treatment, manipulation can be performed under anesthesia. Arthroscopic adhesiolysis is also a minimally invasive procedure, and it can debride adhesions in the knee and increase the joint's range of motion. Open scar excision and revision knee arthroplasty should be considered if adequate results cannot be obtained with these interventions [30].

4.2. Pain

After TKA, patients complain of persistent and unknown pain. Pain negatively affects postoperative recovery and life. The reason for this is not known precisely. Although the causes of persistent pain have not yet been entirely determined, some factors are suspected of causing permanent pain. It is thought that the high pain level, anxiety, depression, and pain catastrophizing in the preoperative period are directly proportional to the risk of persistent postoperative pain. In addition, female gender and young age are also risk factors [31]. The causes of persistent postoperative pain should be clarified, and if septic or aseptic loosening is detected, appropriate revisions should be made. Apart from this, it has been found that starting a bisphosphonate in the postoperative period, among the things that can be done for permanent pain, is effective in pain management and reducing periprosthetic bone loss [32].

4.3. Tibiofemoral Dislocation

Tibiofemoral dislocation is a rare but severe complication. The relationship of the tibial component with the femoral component is impaired; most cases are posteriorly dislocated; also, anterior dislocation can be seen [33]. Dislocation often occurs after trauma, but atraumatic dislocations cannot be underestimated. Obesity, severe preoperative alignment deformity, central or peripheral nerve damage, extensor mechanism defects (patella fracture, patellar or quadriceps tendon rupture), small insert post, and psychiatric diseases are the diseases accompanying tibiofemoral dislocation. In dislocation, the most crucial examination is neurovascular examination because the popliteal artery may be damaged in dislocations, and popliteal thrombosis may occur after a dislocation. Therefore, the treatment begins with the reduction of the dislocation and the re-evaluation of the neurovascular system, and the treatment is applied according to the cause of the dislocation. If there are ligament injuries, conservative and surgical reconstructive interventions may be required, while revision knee arthroplasty at varying levels from partial to total is considered due to a defect in the implants [34].

5. Conclusion

Total knee arthroplasty surgery is increasing in number with the aging population. Therefore, recognizing and treating the complications is essential for the patient to lead a quality life. It may be possible to provide a more comfortable life for patients by knowing that there are unresolved complications such as persistent pain, even if the surgery is performed under the most appropriate conditions, and by evaluating and treating each patient carefully and diligently, to detect and treat complications.

Keywords: Primary Knee Arthroplasty, Complication

References

1. Itou J, Kuwashima U, Itoh M, Okazaki K. No difference in the incidence or location of deep venous thrombosis according to use of pharmacological prophylaxis following total knee arthroplasty. *BMC Musculoskelet Disord*. 2021;22(1):1-7. doi:10.1186/S12891-021-04707-6/TABLES/5
2. Carender CN, Bedard NA, An Q, Brown TS. Common Peroneal Nerve Injury and Recovery after Total Knee Arthroplasty: A Systematic Review. *Arthroplast Today*. 2020;6(4):662-667. doi:10.1016/J.ARTD.2020.07.017
3. Shetty T, Nguyen JT, Sasaki M, et al. Risk factors for acute nerve injury after total knee arthroplasty. *Muscle Nerve*. 2018;57(6):946-950. doi:10.1002/MUS.26045
4. Sundaram K, Udo-Inyang I, Mont MA, Molloy R, Higuera-Rueda C, Piuzzi NS. Vascular Injuries in Total Knee Arthroplasty: A Systematic Review and Meta-Analysis. *JBJS Rev*. 2020;8(1). doi:10.2106/JBJS.RVW.19.00051
5. Padegimas EM, Levicoff EA, McGinley AD, Sharkey PF, Good RP. Vascular Complications After Total Knee Arthroplasty-A Single Institutional Experience. *J Arthroplasty*. 2016;31(7):1583-1588. doi:10.1016/J.ARTH.2016.01.013
6. Ko LJM, DeHart ML, Yoo JU, Huff TW. Popliteal artery injury associated with total knee arthroplasty: Trends, costs and risk factors. *Journal of Arthroplasty*. 2014;29(6):1181-1184. doi:10.1016/j.arth.2014.01.007
7. Gosslau Y, Warm TD, Foerch S, Zerwes S, Scheurig-Muenkler C, Hyhlik-Duerr A. Iatrogenic injury of the popliteal artery in orthopedic knee surgery: clinical results and development of a therapeutic algorithm. *Eur J Trauma Emerg Surg*. Published online 2022. doi:10.1007/S00068-022-01961-8
8. Sazegari MA, Mirzaee F, Bahramian F, Zafarani Z, Aslani H. Wound dehiscence after total knee arthroplasty. *Int J Surg Case Rep*. 2017;39:196-198. doi:10.1016/J.IJSCR.2017.08.032
9. Dennis DA. Wound complications in total knee arthroplasty. *Instr Course Lect*. 1997;46:165-169. doi:10.3928/0147-7447-19970901-26
10. Garbedian S, Sternheim A, Backstein D. Wound healing problems in total knee arthroplasty. *Orthopedics*. 2011;34(9). doi:10.3928/01477447-20110714-42
11. Cai Y, Xu K, Hou W, Yang Z, Xu P. Preoperative chlorhexidine reduces the incidence of surgical site infections in total knee and hip arthroplasty:

A systematic review and meta-analysis. *International Journal of Surgery*. 2017;39:221-228. doi:10.1016/J.IJSU.2017.02.004

12. Ribau AI, Collins JE, Chen AF, Sousa RJ. Is Preoperative Staphylococcus aureus Screening and Decolonization Effective at Reducing Surgical Site Infection in Patients Undergoing Orthopedic Surgery? A Systematic Review and Meta-Analysis With a Special Focus on Elective Total Joint Arthroplasty. *J Arthroplasty*. 2021;36(2):752-766.e6. doi:10.1016/J.ARTH.2020.08.014
13. Gomez-Ochoa SA, Espín-Chico BB, García-Rueda NA, Vega-Vera A, Osma-Rueda JL. Risk of Surgical Site Infection in Patients with Asymptomatic Bacteriuria or Abnormal Urinalysis before Joint Arthroplasty: Systematic Review and Meta-Analysis. *Surg Infect (Larchmt)*. 2019;20(3):159-166. doi:10.1089/SUR.2018.201
14. Alden KJ, Duncan WH, Trousdale RT, Pagnano MW, Haidukewych GJ. Intraoperative Fracture During Primary Total Knee Arthroplasty. doi:10.1007/s11999-009-0876-9
15. Stamiris D, Gkekas NK, Asteriadis K, et al. Anterior femoral notching ≥ 3 mm is associated with increased risk for supracondylar periprosthetic femoral fracture after total knee arthroplasty: a systematic review and meta-analysis. *Eur J Orthop Surg Traumatol*. 2022;32(3):383-393. doi:10.1007/S00590-021-02989-Z
16. Mak YF, Lee QJ, Chang WYE, Wong YC. Intraoperative femoral condyle fracture in primary total knee arthroplasty - a case-control study in Asian population. *Knee Surg Relat Res*. 2020;32(1):1-9. doi:10.1186/S43019-020-00043-6/FIGURES/5
17. Li J, Yan Z, Lv Y, et al. Impact of intraoperative medial collateral ligament injury on outcomes after total knee arthroplasty: a meta-analysis and systematic review. *J Orthop Surg Res*. 2021;16:686. doi:10.1186/s13018-021-02824-5
18. Revell PA. Biological causes of prosthetic joint failure. *Joint Replacement Technology*. Published online January 1, 2021:299-371. doi:10.1016/B978-0-12-821082-6.00018-2
19. Cherian JJ, Jauregui JJ, Banerjee S, Pierce T, Mont MA. What Host Factors Affect Aseptic Loosening After THA and TKA? *Clin Orthop Relat Res*. 2015;473(8):2700. doi:10.1007/S11999-015-4220-2
20. Lee BS, Cho HI, Bin S il, Kim JM, Jo BK. Femoral Component Varus Malposition is Associated with Tibial Aseptic Loosening After

TKA. *Clin Orthop Relat Res*. 2018;476(2):400-407. doi:10.1007/S11999.0000000000000012

21. Wilson CJ, Theodoulou A, Damarell RA, Krishnan J. Knee instability as the primary cause of failure following Total Knee Arthroplasty (TKA): A systematic review on the patient, surgical and implant characteristics of revised TKA patients. *Knee*. 2017;24(6):1271-1281. doi:10.1016/j.knee.2017.08.060
22. Nickinson RSJ, Board TN, Gambhir AK, Porter ML, Kay PR. The microbiology of the infected knee arthroplasty. *Int Orthop*. 2010;34(4):505. doi:10.1007/S00264-009-0797-Y
23. Lima ALL, Oliveira PR, Carvalho VC, Saconi ES, Cabrita HB, Rodrigues MB. Periprosthetic joint infections. *Interdiscip Perspect Infect Dis*. 2013;2013. doi:10.1155/2013/542796
24. Izakovicova P, Borens O, Trampuz A. Periprosthetic joint infection: current concepts and outlook. *EFORT Open Rev*. 2019;4(7):482. doi:10.1302/2058-5241.4.180092
25. Lombardo DJ, Siljander MP, Sobh A, Moore DD, Karadsheh MS. Periprosthetic fractures about total knee arthroplasty. *Musculoskelet Surg*. 2020;104(2):135-143. doi:10.1007/S12306-019-00628-9
26. Yoo JD, Kim NK. Periprosthetic fractures following total knee arthroplasty. *Knee Surg Relat Res*. 2015;27(1):1-9. doi:10.5792/KSRR.2015.27.1.1
27. Putman S, Boureau F, Girard J, Migaud H, Pasquier G. Patellar complications after total knee arthroplasty. *Orthopaedics and Traumatology: Surgery and Research*. 2019;105(1):S43-S51. doi:10.1016/J.OTSR.2018.04.028
28. Matz J, Lanting BA, Howard JL. Understanding the patellofemoral joint in total knee arthroplasty. *Can J Surg*. 2019;62(1):57-65. doi:10.1503/CJS.001617
29. Cheuy VA, Foran JRH, Paxton RJ, Bade MJ, Zeni JA, Stevens-Lapsley JE. Arthrofibrosis Associated With Total Knee Arthroplasty. *J Arthroplasty*. 2017;32(8):2604-2611. doi:10.1016/J.ARTH.2017.02.005
30. Thompson R, Novikov D, Cizmic Z, et al. Arthrofibrosis After Total Knee Arthroplasty: Pathophysiology, Diagnosis, and Management. *Orthop Clin North Am*. 2019;50(3):269-279. doi:10.1016/J.OCL.2019.02.005
31. Lewis GN, Rice DA, McNair PJ, Kluger M. Predictors of persistent pain after total knee arthroplasty: a systematic review and meta-analysis. *Br J Anaesth*. 2015;114(4):551-561. doi:10.1093/BJA/AEU441

32. Piscitelli P, Iolascon G, Innocenti M, et al. Painful prosthesis: approaching the patient with persistent pain following total hip and knee arthroplasty. *Clin Cases Miner Bone Metab*. 2013;10(2):97-110. doi:10.11138/ccmbm/2013.10.2.097
33. Jethanandani RG, Maloney WJ, Huddleston JI, Goodman SB, Amanatullah DF. Tibiofemoral Dislocation After Total Knee Arthroplasty. *Journal of Arthroplasty*. 2016;31(10):2282-2285. doi:10.1016/j.arth.2016.03.010
34. Rouquette L, Erivan R, Pereira B, Boisgard S, Descamps S, Villatte G. Tibiofemoral dislocation after primary total knee arthroplasty: a systematic review. *Int Orthop*. 2019;43(7):1599-1609. doi:10.1007/S00264-019-04287-0

CHAPTER V

THROMBOEMBOLIC COMPLICATIONS IN TOTAL KNEE ARTHROPLASTY

Gökhan PEKER

(Exp. Dr.), Istinye University, Trabzon Yıldızlı Medical Park Hospital.
e-mail: drgokhanpeker@gmail.com
ORCID: 0000-0002-6211-6645

1. Introduction

Although total knee replacemet is one of the most successful procedure today which improves quality of life, the risk of deep vein thrombosis (DVT) and/or venous thromboembolism (VTE) is high(1). After soft tissue dissection and bone reaming, thromboplastin is released and there is also stasis due to immobility. VTE events are provoked by them(2).

Female gender, older age, obesity, estrogen usage, cancer history, thrombophilia, immobility, history of thromboembolism , cardiovascular, renal and respiratory comorbidities such as myocardial infarction, atrial fibrillation/flutter, congestive heart failure, chronic obstructive pulmonary disease, nephrotic syndrome, peripheral artery disease, indwelling femoral vein catheter, varicose veins, smoking, hypertension, and diabetes mellitus are risc factors of DVT.

DVT prevelance after total knee arthroplasty (TKA) without any prophylaxis is between 40% to 80% with about %33 at deep proximal veins(3). Proximal thrombus occurring in 9% to 20% of patients has a greater risk of PE than thrombus in the calf veins, occurring in 40% to 60% of patients. Thrombus in the calf veins tend to spread proximally in 6% to 23% of patients. Asymptomatic PE is 10% to 20%, symptomatic PE can be seen in 0.5% to 3% of patients and the mortality rate is 2%. Because most clots form without signs or symptoms, clinical examination is not reliable in detecting DVT. Venography is the gold standard diagnostic method of DVT. Duplex ultrasound is an alternative diagnostic modality of DVT after total knee replacement, using venography for comparison, and its sensitivities range from 67% to 86%.. Because of cost-effectivity, repeatability and minimal morbidity, duplex ultrasound is useful as a screening test but it's radiologist's experience depended.

2. Prophylaxis Methods

Mechanical methods such as compression stockings, foot pumps, warfarin, low molecular weight heparin, fondaparinux, oral factor Xa inhibitors, and aspirin are available methods for DVT prophylaxis. The American Academy of Orthopaedic Surgeons (AAOS) and the American College of Chest Physicians (ACCP) are all recommend the use of mechanical compressive devices for VTE prophylaxis(4).

2.1. Mechanical Prophylaxis

Graduated compression stockings (GCS), intermittent pneumatic compression device (IPCD) and venous foot pumps (VFP) are mechanical methods for VTE prophylaxis (5, 6). The advantages of mechanical VTE prophylaxis are low bleeding risk, no need for blood tests for follow-up, and no risk of clinically significant side effects(7). In addition, the efficacy of anticoagulants can be supported by some mechanical methods. Endogenous fibrinolytic activity is stimulated by decreasing plasminogen activator inhibitor-1 levels by IPCDs(8). Disadvantages of mechanical VTE prophylaxis methods include adaptation problems related to reduced mobility, the need to be worn continuously for 72 hours before, during and after surgery, and the lack of strong evidence that they can reduce the risk of death or PE(5, 7). If GCS are not used in the appropriate size and manner of use, they may cause deterioration in tissue oxygenation(9). Contraindications for the use of mechanical VTE methods include open fractures, peripheral arterial insufficiency, severe heart failure, infection and ulceration of the lower extremities(6).

Early mobilization is the simplest and most feasible way of VTE prophylaxis(6). Although early walking reduces hospital stay, complications and six-month mortality rates, most symptomatic VTE events occur after mobilization. Mobilization alone is not sufficient for VTE prophylaxis and should be carefully considered(10-12).

The latest ACCP (2012) guideline recommends combining pharmacological VTE prophylaxis treatment with mechanical methods in hospitalized patients at high risk of VTE undergoing major orthopedic surgery(13).

2.2. Pharmacological Prophylaxis

2.2.1. Aspirin:

Sharda et al. found a higher risk of VTE with aspirin-only thromboprophylaxis treatment after arthroplasty in cohort studies compared to previous data(14). Contrary to this study, in Matharu et al.'s study, aspirin did not differ statistically significantly in terms of clinical efficacy and safety profile compared to other anticoagulants(15). Similar to the ACCP and AAOS guidelines, aspirin is recommended for VTE prophylaxis, according to a 2016 meta-analysis, but the evidence is of limited quality and the dose and duration of the drug remains unclear(16). Compared with enoxaparin, rate of symptomatic VTE defined as DVT above or below the knee, or pulmonary embolism is significantly increased with aspirin usage at 90 days after knee arthroplasty(17). Another study, in which more than a quarter of patients used only aspirin for prophylaxis after arthroplasty, did not show a higher risk of venous thromboembolism compared to other anticoagulants(18). As a result, the use of aspirin alone seems to be controversial in VTE prophylaxis(19).

2.2.2. Vitamin K Antagonists (VKA):

ACCP guidelines recommend VKAs for VTE prophylaxis in patients undergoing TKA(13). Prophylactic use of warfarin is started in the evening before or after surgery and adjusted according to daily prothrombin time. Using the international normalized ratio (INR) ensures standardization of the measured anticoagulant effect. It is recommended to keep the target INR between 2.0 and 3.0 with an average of 2.5. It is recommended to start INR monitoring after the first two or three doses(19). While the advantages of warfarin are that it can be administered orally and is inexpensive, its easy interaction with drugs, the need for monitoring with INR, the late onset of its effect and the risk of bleeding are disadvantages.

2.2.3. Low-molecular-weight heparin (LMWH):

LMWH is effective in thromboprophylaxis after TKA. LMWH exhibits more inhibitory activity against factor Xa than thrombin, and it binds to cells and proteins to a lesser extent than UFH(19). Compared to UFH, its pharmacokinetic and pharmacodynamic properties are more predictable, its half-life is longer, and the risk of non-hemorrhagic side effects is lower. Administration with a standard dose, lack of routine laboratory monitoring, less drug interactions are advantages, but the disadvantages are that it is expensive, administered subcutaneously, and may increase the incidence of bleeding. The clinician should be very careful while using LMWH with epidural or spinal anesthesia as hematomas with neurological complications have been reported. In patients undergoing TKA, prophylaxis with LMWH is recommended to be started either 12 hours or more pre-operatively or 12 hours or more post-operatively(13). Most VTEs are diagnosed after discharge, on average 7 days after TKA. Different studies have shown that both LMWH and adjusted dose warfarin are more effective than placebo in long-term prophylaxis (up to 35 days) after orthopedic surgery(3). The use of LMWH, fondaparinux, vitamin K antagonist (eg, warfarin) for DVT prophylaxis for at least 10 days (minimum 10 to 14 days and maximum 35 days) after TKA surgery is recommended in the American College of Chest Physicians (ACCP) 2008 guideline.

2.2.4. Unfractioned Heparin (UFH):

UFH is recommended by the last ACCP guidelines for thromboprophylaxis(13). Heparin should be administered parenterally as a continuous intravenous (IV) infusion or subcutaneously because it cannot be absorbed orally. Hemorrhage can be seen as a complication in the use of UFH(19). Heparin-induced thrombocytopenia (HIT) and osteoporosis can also be non-hemorrhagic side effects of UFH. Activated partial thromboplastin time (aPTT or APTT) is used to monitor the effect of UFH.

2.2.5. Fondaparinux:

It is a synthetic pentasaccharide with a higher specific anti-Xa activity and a longer half-life than LMWH. It is rapidly and abundantly absorbed after subcutaneous administration. The benefits of Fondaparinux over UFH and LMWH are that it does not inactivate thrombin (factor IIa), has no effect on platelets, and does not cross-react with the serum of patients with HIT(6). Fondaparinux is recommended by ACCP guidelines for VTE prophylaxis after TKA(13).

2.2.6. New Oral Anticoagulant Drugs:

They do not require laboratory monitoring and can be administered at specific doses(19).

2.2.6.1. Rivaroxaban

Rivaroxaban which is direct inhibitor of activated factor X (Xa) is used orally with daily dose of 10 mg for VTE prophylaxis after elective TKA(20). It was found to be more effective than enoxaparin in Phase III clinical studies(21, 22). Rivaroxaban is recommended by ACCP guidelines for VTE prophylaxis after TKA(13).

2.2.6.2. Dabigatran

Dabigatran is a selective, reversible, direct thrombin inhibitor. Since dabigatran itself is not absorbed from the gut, it is administered as an orally absorbable prodrug, dabigatran etexilate. Studies comparing dabigatran etexilate (220 or 150 mg daily) with enoxaparin reported that both are effective in thromboprophylaxis(19, 23). For VTE prophylaxis, dabigatran is administered immediately after surgery, starting at half a dose, at 150 mg and 220 mg per day(24). Although previous studies have reported an association between dabigatran and an increased risk of myocardial infarction or acute coronary syndrome, the latest ACCP guidelines recommend dabigatran(13, 25).

2.2.6.2.1. Apixaban

Apixaban which is a direct factor Xa inhibitor has been approved for VTE prophylaxis in studies comparing it to enoxaparine(26, 27). The It's suggested dosage is 2.5 mg twice daily starting 12 to 24 hours after TKA surgery until 12 days (27). Apixaban is recommended by ACCP guidelines for VTE prophylaxis after TKA (13).

3. Guidelines

Since, aspirin, low-dose UFH, LMWH, fondaparinux, VKA, apixaban, rivaroxaban and dabigatran are all recommended by current ACCP guidelines with Grade 1B level of evidence, IPCD is suggested with grade 1C level of evidence at least 10 to 14 days and up to 35 days(12, 13). The level of evidence for preferring LMWH over other recommended agents is 2B, whereas in the case of dose-adjusted VKA or aspirin it is 2C(13). It is recommended that the IPCD device be used together with another antithrombotic agent for at least 18 hours in the hospital. Imaging with Doppler ultrasonography is not recommended in asymptomatic patients(13).

The AAOS guidelines recommend the use of prophylactic pharmacological agents and/or mechanical compression devices in patients without a high risk of VTE or bleeding after elective TKA (moderate grade of recommendation).

According to the SIGN guideline, it is recommended to use pharmacological prophylaxis and mechanical prophylaxis together if there are no contraindications (Grade A)(28).

According to the NICE guideline, aspirin (75 mg or 150 mg for 14 days), LMWH (for 14 days until discharge with antiembolic stockings), rivaroxaban, apixaban or dabigatran is recommended for patients undergoing elective TKA.

4. Results

Mechanical and/or pharmacological VTE prophylaxis should be applied for TKA surgery because of VTE risk during hospitalization, during and after surgery. There are few published guidelines for VTE prophylaxis methods. Prophylaxis with LMWH seems to be more effective compared to other methods and should therefore be preferred first. There is still some controversy over the use of aspirin as the sole modality for VTE prophylaxis in TKA surgery.

References

1. Keller K, Hobohm L, Barco S, Schmidtmann I, Münzel T, Engelhardt M, et al. Venous thromboembolism in patients hospitalized for knee joint replacement surgery. Sci Rep. 2020;10(1):22440. Epub 20201231. doi: 10.1038/s41598-020-79490-w. PubMed PMID: 33384429; PubMed Central PMCID: PMC7775461.
2. Warwick D. Prevention of venous thromboembolism in total knee and hip replacement. Circulation. 2012;125(17):2151-5. doi: 10.1161/circulationaha.111.055731. PubMed PMID: 22547755.
3. <p5b8sample03.pdf>.
4. Box HN, Shahrestani S, Huo MH. Venous Thromboembolism Prophylaxis after Total Knee Arthroplasty. J Knee Surg. 2018;31(7):605-9. Epub 20180307. doi: 10.1055/s-0038-1636907. PubMed PMID: 29514375.
5. Sourmelis S. Guidelines for Prevention of Deep Vein Thrombosis in Orthopaedics. Athens: HAOST. 2009.
6. Leme LE, Sguizzatto GT. PROPHYLAXIS OF VENOUS THROMBOEMBOLISM IN ORTHOPAEDIC SURGERY. Rev Bras Ortop. 2012;47(6):685-93. Epub 20151104. doi: 10.1016/s2255-4971(15)30023-9. PubMed PMID: 27047885; PubMed Central PMCID: PMC4799480.
7. Geerts WH, Pineo GF, Heit JA, Bergqvist D, Lassen MR, Colwell CW, et al. Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest. 2004;126(3 Suppl):338s-400s. doi: 10.1378/chest.126.3_suppl.338S. PubMed PMID: 15383478.

8. Davis P. Venous thromboembolism prevention-an update. Journal of Orthopaedic Nursing. 2004;1(8):50-6.
9. Agu O, Hamilton G, Baker D. Graduated compression stockings in the prevention of venous thromboembolism. Br J Surg. 1999;86(8):992-1004. doi: 10.1046/j.1365-2168.1999.01195.x. PubMed PMID: 10460633.
10. White RH, Gettner S, Newman JM, Trauner KB, Romano PS. Predictors of rehospitalization for symptomatic venous thromboembolism after total hip arthroplasty. N Engl J Med. 2000;343(24):1758-64. doi: 10.1056/nejm200012143432403. PubMed PMID: 11114314.
11. Hoenig H, Rubenstein LV, Sloane R, Horner R, Kahn K. What is the role of timing in the surgical and rehabilitative care of community-dwelling older persons with acute hip fracture? Arch Intern Med. 1997;157(5):513-20. PubMed PMID: 9066455.
12. Geerts WH, Bergqvist D, Pineo GF, Heit JA, Samama CM, Lassen MR, et al. Prevention of venous thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008;133(6 Suppl):381s-453s. doi: 10.1378/chest.08-0656. PubMed PMID: 18574271.
13. Falck-Ytter Y, Francis CW, Johanson NA, Curley C, Dahl OE, Schulman S, et al. Prevention of VTE in orthopedic surgery patients: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e278S-e325S. doi: 10.1378/chest.11-2404. PubMed PMID: 22315265; PubMed Central PMCID: PMC3278063.
14. Sharda AV, Fatovic K, Bauer KA. Aspirin thromboprophylaxis in joint replacement surgery. Res Pract Thromb Haemost. 2022;6(1):e12649. Epub 20220124. doi: 10.1002/rth2.12649. PubMed PMID: 35106432; PubMed Central PMCID: PMC8785144.
15. Matharu GS, Kunutsor SK, Judge A, Blom AW, Whitehouse MR. Clinical Effectiveness and Safety of Aspirin for Venous Thromboembolism Prophylaxis After Total Hip and Knee Replacement: A Systematic Review and Meta-analysis of Randomized Clinical Trials. JAMA Intern Med. 2020;180(3):376-84. doi: 10.1001/jamainternmed.2019.6108. PubMed PMID: 32011647; PubMed Central PMCID: PMC7042877.
16. An VV, Phan K, Levy YD, Bruce WJ. Aspirin as Thromboprophylaxis in Hip and Knee Arthroplasty: A Systematic Review and Meta-Analysis.

J Arthroplasty. 2016;31(11):2608-16. Epub 20160413. doi: 10.1016/j.arth.2016.04.004. PubMed PMID: 27178011.

17. Sidhu VS, Kelly TL, Pratt N, Graves SE, Buchbinder R, Adie S, et al. Effect of Aspirin vs Enoxaparin on Symptomatic Venous Thromboembolism in Patients Undergoing Hip or Knee Arthroplasty: The CRISTAL Randomized Trial. Jama. 2022;328(8):719-27. doi: 10.1001/jama.2022.13416. PubMed PMID: 35997730.
18. Baumgartner C, Maselli J, Auerbach AD, Fang MC. Aspirin Compared with Anticoagulation to Prevent Venous Thromboembolism After Knee or Hip Arthroplasty: a Large Retrospective Cohort Study. J Gen Intern Med. 2019;34(10):2038-46. Epub 20190624. doi: 10.1007/s11606-019-05122-3. PubMed PMID: 31236894; PubMed Central PMCID: PMC6816584.
19. Flevas DA, Megaloikonomos PD, Dimopoulos L, Mitsiokapa E, Koulouvaris P, Mavrogenis AF. Thromboembolism prophylaxis in orthopaedics: an update. EFORT Open Rev. 2018;3(4):136-48. Epub 20180427. doi: 10.1302/2058-5241.3.170018. PubMed PMID: 29780621; PubMed Central PMCID: PMC5941651.
20. Kubitza D, Berkowitz SD, Misselwitz F. Evidence-Based Development and Rationale for Once-Daily Rivaroxaban Dosing Regimens Across Multiple Indications. Clin Appl Thromb Hemost. 2016;22(5):412-22. Epub 20160218. doi: 10.1177/1076029616631427. PubMed PMID: 26893445; PubMed Central PMCID: PMC4888194.
21. Eriksson BI, Borris LC, Friedman RJ, Haas S, Huisman MV, Kakkar AK, et al. Rivaroxaban versus enoxaparin for thromboprophylaxis after hip arthroplasty. N Engl J Med. 2008;358(26):2765-75. doi: 10.1056/NEJMoa0800374. PubMed PMID: 18579811.
22. Lassen MR, Ageno W, Borris LC, Lieberman JR, Rosencher N, Bandel TJ, et al. Rivaroxaban versus enoxaparin for thromboprophylaxis after total knee arthroplasty. N Engl J Med. 2008;358(26):2776-86. doi: 10.1056/NEJMoa076016. PubMed PMID: 18579812.
23. Eriksson BI, Dahl OE, Büller HR, Hettiarachchi R, Rosencher N, Bravo ML, et al. A new oral direct thrombin inhibitor, dabigatran etexilate, compared with enoxaparin for prevention of thromboembolic events following total hip or knee replacement: the BISTRO II randomized trial. J Thromb Haemost. 2005;3(1):103-11. doi: 10.1111/j.1538-7836.2004.01100.x. PubMed PMID: 15634273.

24. Ageno W, Gallus AS, Wittkowsky A, Crowther M, Hylek EM, Palareti G. Oral anticoagulant therapy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest. 2012;141(2 Suppl):e44S-e88S. doi: 10.1378/chest.11-2292. PubMed PMID: 22315269; PubMed Central PMCID: PMC3278051.
25. Uchino K, Hernandez AV. Dabigatran association with higher risk of acute coronary events: meta-analysis of noninferiority randomized controlled trials. Arch Intern Med. 2012;172(5):397-402. Epub 20120109. doi: 10.1001/archinternmed.2011.1666. PubMed PMID: 22231617.
26. Lassen MR, Raskob GE, Gallus A, Pineo G, Chen D, Portman RJ. Apixaban or enoxaparin for thromboprophylaxis after knee replacement. N Engl J Med. 2009;361(6):594-604. doi: 10.1056/NEJMoa0810773. PubMed PMID: 19657123.
27. Madan S, Shah S, Dale P, Partovi S, Parikh SA. Use of novel oral anticoagulant agents in venous thromboembolism. Cardiovasc Diagn Ther. 2016;6(6):570-81. doi: 10.21037/cdt.2016.11.17. PubMed PMID: 28123977; PubMed Central PMCID: PMC5220203.
28. Network SIG. Prevention and management of venous thromboembolism. Edinburgh: Healthcare Improvement Scotland. 2010.

CHAPTER VI

SURGICAL COMPLICATIONS IN BONE TUMORS

Fener ÇELEBİ

İzmir Bozyaka Training and Research Hospital, Department of Orthopaedics and Traumatology, drfenercelebi@hotmail.com
ORCID: 0000-0002-9605-098X

An understanding of the basic biology and pathology of bone and soft tissue tumors is essential for the appropriate planning of their treatment. Soft tissue and bone sarcomas are a rare and heterogeneous group of tumors. These neoplasms represent less than 1% of all adults and 15% of pediatric malignancies. Complications and functional outcomes of reconstruction with an osteoarticular allograft after intra-articular resection of the proximal aspect of the humerus.(1)

In benign bone tumors, the diagnosis of the lesion is usually attempted with conventional radiography and advanced imaging methods after clinical evaluation. Following the preliminary examination, staging and prognostic evaluations should be made according to histological type and anatomical localization. In the surgical treatment of benign bone tumors, treatment options according to staging were primarily considered. The stage, size, localization of the tumor, the risk of fracture, the need for reconstruction and the age of the patient determine the type of surgical treatment. The general characteristics of the tumor may necessitate the use of adjuvant treatment and/or surgical methods. Autogenous bone grafts, allografts, bone cement or graft analogs can be used for reconstruction.(2)

Patients with malignant primary bone tumors are usually in the most active period of their lives in terms of social and personal functioning. Therefore, the treatment in these patients focuses on a goal besides removing the tumor: In addition to helping these patients achieve maximum function, it is aimed to use a method with minimal cost and less surgical complications. (1)

Treatment of malignant bone tumors requires multidisciplinary interventions such as surgical tumor resection, chemotherapy and radiation therapy. Most bone defects after surgical tumor resection require reconstruction using endoprostheses, allografts, autografts, artificial bones, or distraction osteogenesis.(3)

Complications following any limb-sparing reconstruction are not uncommon. Most patients have a suppressed immune system due to chronic disease, chemotherapy, and malnutrition. Patients are often anemic and have coagulation abnormalities, including thrombocytopenia. The presence of long-term indwelling catheters for the administration of chemotherapy can lead to unrecognized bacteremia and potential infection of the operative site. When planning the final surgery, staging and diagnosis with biopsy constitute the first step of the process.(3)

Necrosis and Circulatory Disorders

The anatomical location of a tumor and necessary resection can lead to significant impairment of the venous and lymphatic drainage of the limb during resection, venousstasis, swelling, and lymphedema. This can quickly lead to flap necrosis, secondary infection and eventual amputation postoperatively.(4)

Surgical Wound Infection

Wound infection is one of the most serious complications of bone tumor surgery. It has been reported that the incidence of Surgical Site Infection following malignant bone tumor surgery is 3-10 times higher after general orthopedic surgery. It is a feared complication of minimally invasive prostheses. The recorded infection rate is 1% per lengthening procedure. Acute infection is treated with flushing and 6 weeks of intensive antibiotics. However, the chance of cure is only 20%. Two-stage surgery is applied for chronic infection. (2)

Loss of Extensor Mechanism and Delay in Recovery of Extremity Functions

The tibia is anatomically unique in its anterior subcutaneous border and patellar tendon insertion. Routine use of the gastrocnemius rotation flap has significantly reduced the incidence of postoperative complications, and combined reconstruction of tendon insertion and attention to postoperative rehabilitation may result in minimal extensor loss and recovery of function. Joint stability in the knee is achieved using the same hinge design used in distalfemoral replacements. (5)

Nonunion

Nonunion is one of the complications seen after bone tumor surgical operations. (2)

Distal femur non-union

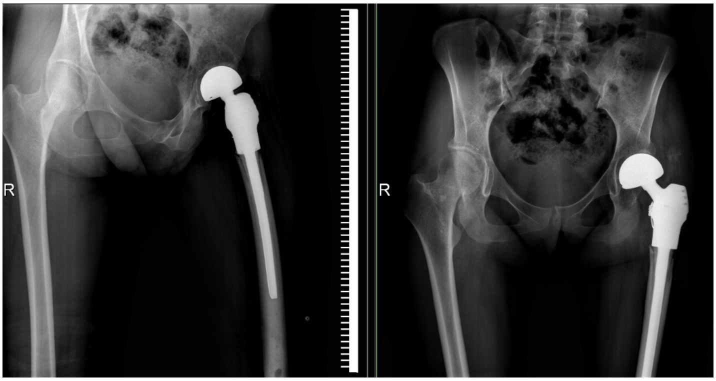
Complications Related to Reconstruction Material

Complications specific to endoprosthetic reconstruction may be related to mechanical or biological factors. Denture fracture, separation of modular components, fatigue failure, and polyethylene wear have been described. Improved implant designs, metallurgy, and manufacturing techniques can significantly reduce the incidence of these problems. Biological failure of an endoprosthesis or implant material can occur as a result of joint instability, aseptic loosening, or periprosthetic fracture of the bone surrounding the prosthesis. (6)

Fracture of the proximal Tumor resection prosthesis of the femur

Loosening of Prostheses and Implants

The use of hydroxyapatite collars has significantly reduced the incidence of aseptic loosening. Revision is usually quite simple and the prosthesis is changed to an adult model. It is important to keep in mind the possibility that the relaxation is due to a low-grade infection.(7)



Aseptic and rotational loosening of the proximal femur after 8 years.

Unplanned Lengthening or Shortening

It is an unusual complication resulting from mechanism failures, which often requires revision of the implant. Most extension procedures are done 10 mm at a time. Attempting to extend it much further than this can lead to complications such as fixed flexion deformity or sometimes the development of neuropraxia.(7)

Acute shortening of an adult prosthesis due to medial displacement of an extension ring requiring revision.

Restriction in Joint Movements

Stiffness is a common problem in young children who have a prosthesis around the knee or if the prosthesis placed to add extra length is longer than the bone removed. In some children, excessive scar tissue forms around the prosthesis; In such cases, removal of scar tissue along with intensive physical therapy may help. Intensive physical therapy, including serial plaster casts, may be helpful in fixed flexion deformities. Abductor failure and Trendelenburg gait are among the complications that can be seen due to prosthesis and wide resection around the hip. (8)

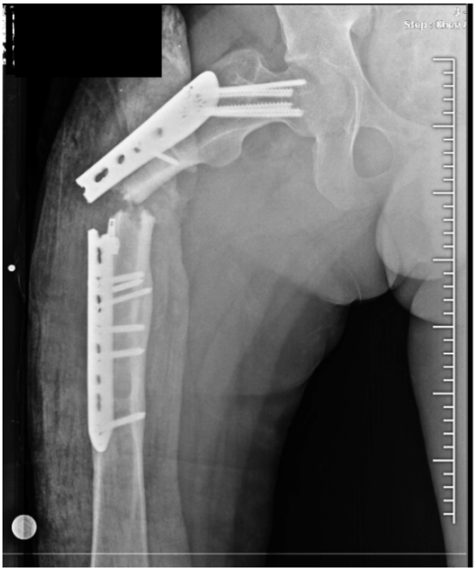
Hip or Shoulder Subluxation

Subluxation is one of the complications in femur proximal or humerus proximal tumor resection prostheses. Femoral head subluxation is a much greater problem in young children with proximal femur replacement. It has been reported that the tendency of the upper edge of the acetabulum to develop improperly increases in children younger than 12 years of age, and the frequency of subluxation of the femoral head is increased in these children. (8)

Hip subluxation has been a problem in young patients receiving proximalfemoral replacements.

Implant Fracture

Implant fracture is rare in patients who have reached maturity while the child prosthesis is still in place. The most common site for a fracture is where the thinner elongation joins the main component. In any case, revision is necessary. (4)



Implant fracture in femoral shaft ewing sarcoma

Periprosthetic Fractures

Periprosthetic fractures are rare, but there appears to be an increased risk of femoral fracture on a sliding femoral prosthesis used with a proximaltibial growing prosthesis. (8)

Periprosthetic fracture of the femoral shaft due to tumor resection prosthesis

References

1. Getty PJ, Peabody TD. Complications and functional outcomes of reconstruction with an osteoarticular allograft after intra-articular resection of the proximal aspect of the humerus. J Bone Joint Surg Am. 1999 Aug;81(8):1138-46.
2. Healey JH, Brown HK (2000) Complications of bone metastases: surgical management. Cancer 88(S12):2940–2951
3. Albergo JI, Gaston CL, Aponte-Tinao LA, Ayerza MA, Muscolo DL, Farfalli GL, Jeys LM, Carter SR, Tillman RM, Abudu AT, Grimer RJ. Proximal Tibia Reconstruction After Bone Tumor Resection: Are Survivorship and Outcomes of Endoprosthetic Replacement and Osteoarticular Allograft Similar? Clin Orthop Relat Res. 2017 Mar;475(3):676-682.
4. Gharedaghi M, Peivandi MT, Mazloomi M, Shoorin HR, Hasani M, Seyf P, Khazaee F. Evaluation of Clinical Results and Complications of Structural Allograft Reconstruction after Bone Tumor Surgery. Arch Bone Jt Surg. 2016 Jun;4(3):236-42.
5. Benedetti MG, Bonatti E, Malfitano C, Donati D. Comparison of allograft-prosthetic composite reconstruction and modular prosthetic replacement in proximal femur bone tumors: functional assessment by gait analysis in 20 patients. Acta Orthop. 2013 Apr;84(2):218-23.
6. Renard AJ, Veth RP, Schreuder HW, van Loon CJ, Koops HS, van Horn JR. Function and complications after ablative and limb-salvage therapy in lower extremity sarcoma of bone. J Surg Oncol. 2000 Apr;73(4):198-205.
7. Lapp MA, Bridwell KH, Lenke LG, Baldus C, Blanke K, Iffrig TM. Prospective randomization of parenteral hyperalimentation for long fusions with spinal deformity: its effect on complications and recovery from postoperative malnutrition. Spine (Phila Pa 1976). 2001 Apr 1;26(7):809-17; discussion 817.
8. Gharehdaghi M, Hassani M, Parsa A, Omidikashani F, Jarrahi L, Hosseini R. Short Term Complications and Functional Results of Sarcoma Limb Salvage Surgeries. Arch Bone Jt Surg. 2019 Mar;7(2):161-167.

CHAPTER VII

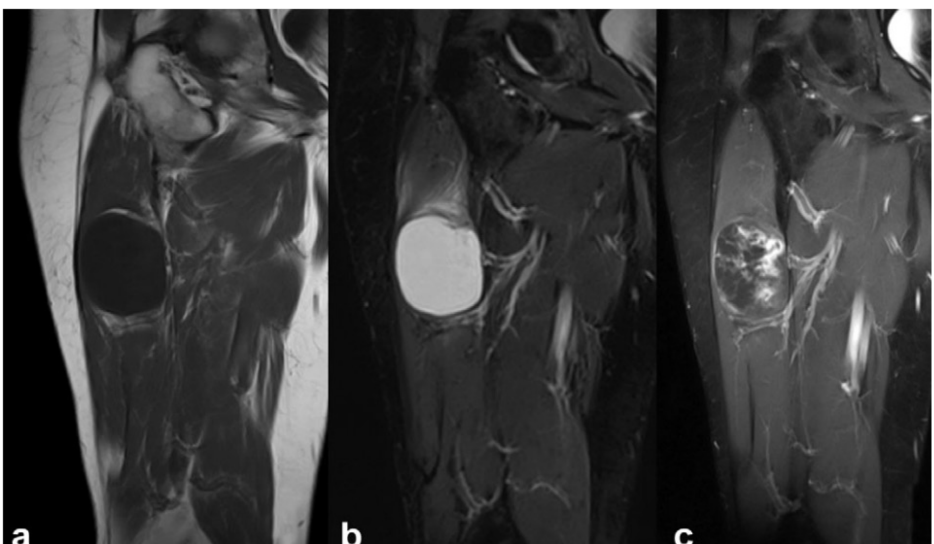
SURGICAL COMPLICATIONS IN SOFT TISSUE TUMORS

Mohamed Ali BEKIR

(Dr.) Department of Orthopaedics and Traumatology,
Dr. Nafiz Korez Sincan City Hospital, Ankara, Türkiye
e-mail: mbekir2705@yahoo.com
ORCID: 0000-0002-9040-322X

In benign soft tissue tumors, cellular growth is much slower than in malignant ones. Distant spread is often not seen and local recurrence rates are much less than in malignant lesions, according to the margins of the surgery (intralesional, marginal, large, radical).(1) They usually do not cause pain. Nodular fasciitis and fat necrosis, which are fast growing and painful, are excluded from these generalizations.(2)

Malignant soft tissue lesions usually show rapid growth, their local growth is aggressive and infiltrative. They have only pseudocapsules and may have satellite lesions. Therefore, they must be removed with a wide margin.



Coronal MR images of a 61-year-old woman with an intramuscular myxoma of the right vastus lateralis. a: T1w MR image shows a hypointense mass with well-defined/sharp hyperintense margins (pseudocapsule); there is a triangular area of low SI at the upper pole. b: STIR image reveals a hyperintense lesion surrounded by a hypointense pseudocapsule; the triangular area at the upper pole shows high signal intensity and ill-defined margins. c: On contrast-enhanced T1w MR image, the lesion presents an inhomogeneous but intense contrast enhancement.(3)

Otherwise, they encounter very high local recurrence and metastasis rates.(4) Low grade malignancies grow more slowly and show early infiltration, but their rate of metastasis is higher than that of local recurrence.(2)

The incidence of soft tissue masses in the general population in the USA is 300/100000 per year. Such masses are always encountered by the orthopedic specialist in daily practice. Soft tumors are approximately 100 times more common than sarcomas. This big difference is a point that misleads all orthopedists and general surgeons.(5)

Surgical Options İn Soft Tissue Tumors

- **Intralesional resection :**It is the removal of the tumor tissue piece by piece by entering it. Good this is the classical approach in benign tumors
- **Marginal resection**: Tumor tissue is intact. It is the removal of the capsule or pseudocapsule without entering the tumor.
- **Wide resection**: a tumor tissue around it amount without being seen with healthy tissue is to be removed.
- **Radical resection**: Total of the entire anatomical compartment (bone) where the tumor tissue is located. It is to be removed.
- **Amputation**: Surgery to remove part or all of an arm or leg. Amputation is rarely used to treat soft tissue sarcoma.

Complications İn Soft Tissue Tumor Surgery

- **Relapses, residual tumor and pseudotumors**

  Inflammation: Residual mass and local recurrence rates are high, especially in patients who do not wide resection and radical resection. Pseudotumor formation may be encountered due to postoperative changes.
- **Metastases and Lymphadenopathy**

  Soft tissue sarcomas most frequently metastasized to the lung and the musculoskeletal system including muscles (both ipsilateral and contralateral) and bone (skip and multiple distant bones of the axial and appendicular skeleton). Other metastases to lymph node and liver were synchronous with pulmonary and bone metastases

- **Second Malignancy**
  Although rare, it is possible to develop secondary malignancy after treatment. such as the development of melanoma in the left forearm in a patient who underwent resection for alveolar rhabdomyosarcoma in the right forearm, or leimyosarcoma in the local region of the same extremity.
- **Inflammatory Changes of Muscle, Subcutaneous Tissue and Skin**
  Depending on where the inflammatory change is, there can be many changes, from skin or subcutaneous necrosis to chronic or acute muscle atrophy.(6)
- **Seromas and Abscesses**
  These are changes that vary depending on the size of the tissue resected post-operatively, but can occur in any condition, may require debridement surgery.
- **Heterotrophic Ossification**
  It can be seen especially in patients who have undergone amputation, but after any kind of resection, even if they do not receive radiation therapy.(7)
- **Nerve Complication:**
  Complications such as neuritis and entrapment may occur due to compression caused by fibrosis and adhesions, and it is one of the mechanisms that play a role in muscle atrophy.

References:

1. Rochwerger A, Mattei JC. Management of soft tissue tumors of the musculoskeletal system. Orthop Traumatol Surg Res. 2018 Feb;104(1S):S9-S17. doi: 10.1016/j.otsr.2017.05.031. Epub 2017 Dec 2. PMID: 29203433.
2. Katenkamp K, Katenkamp D. Soft tissue tumors: new perspectives on classification and diagnosis. Dtsch Arztebl Int. 2009 Sep;106(39):632-6. doi: 10.3238/arztebl.2009.0632. Epub 2009 Sep 25. PMID: 19890408; PMCID: PMC2770206.
3. Stramare R, Beltrame V, Gazzola M, Gerardi M, Scattolin G, Coran A, Faccinetto A, Rastrelli M, Rossi CR. Imaging of soft-tissue tumors. J

Magn Reson Imaging. 2013 Apr;37(4):791-804. doi: 10.1002/jmri.23791. Epub 2012 Sep 12. PMID: 22972755.

4. Fornaciari G. Histology of ancient soft tissue tumors: A review. Int J Paleopathol. 2018 Jun;21:64-76. doi: 10.1016/j.ijpp.2017.02.007. Epub 2017 Mar 21. PMID: 29776878.
5. Choi JH, Ro JY. The 2020 WHO Classification of Tumors of Soft Tissue: Selected Changes and New Entities. Adv Anat Pathol. 2021 Jan;28(1):44-58. doi: 10.1097/PAP.0000000000000284. PMID: 32960834.
6. Murahashi Y, Iba K, Teramoto A, Emori M, Okada Y, Kamiya T, Watanabe K, Yamashita T. Clinical features of bone and soft tissue tumors of the foot and ankle: Results from a retrospective single-center case-series. J Orthop Sci. 2021 Sep;26(5):885-890. doi: 10.1016/j.jos.2020.08.016. Epub 2020 Oct 14. PMID: 33067084.
7. Noebauer-Huhmann IM, Chaudhary SR, Papakonstantinou O, Panotopoulos J, Weber MA, Lalam RK, Albtoush OM, Fueger BJ, Szomolanyi P, Grieser T, Bloem JL. Soft Tissue Sarcoma Follow-up Imaging: Strategies to Distinguish Post-treatment Changes from Recurrence. Semin Musculoskelet Radiol. 2020 Dec;24(6):627-644. doi: 10.1055/s-0040-1721464. Epub 2020 Dec 11. Erratum in: Semin Musculoskelet Radiol. 2020 Dec;24(6):e1. PMID: 33307581.

CHAPTER VIII

COMPLICATIONS OF SURGICAL TREATMENT IN ISOLATED ANTERIOR CRUCIATE LIGAMENT INJURY

Murat SAYLIK

(Asst. Prof. Dr.; Orthopedics and Traumatology Specialist,), İstinye University,
e-mail: drmuratsayliksterr@gmail.com
ORCID: 0000 0002 1023 4164

An anterior cruciate ligament (ACL) injury is seen in the active young-middle age group in almost every society, causing temporary loss of labor force and causing economic burden (1). The patient's complaints show the damage to the ACL to a great extent with careful physical examination findings and magnetic resonance (MR) visualization. While MR gives reliable results, especially in the diagnosis of acute ACL ruptures, MR images may not provide reliable results due to the development of synovial scar tissue proliferation in chronic ACL tears (2). The degree of damage to the ligament should be evaluated arthroscopically before ACL reconstruction.

In young patients and athletes with a complete ACL injury, ACL reconstruction has been the method used for many years for knee stability and functional improvement (3). There is no consensus on the best time for post-traumatic ACL reconstruction. After the regression of acute hemarthrosis and the reduction of edema in the tissues, the surgical application is recommended. Professional athletes may insist on early surgery because they want to return to the sport early. However, it has been reported that recurrence of ACL injury after early ACL reconstruction increased by 30%, arthrofibrosis increased by 22%, and range of motion was seen by 37% (4,5,6).

It has been reported that the physical condition of the knee during surgery is more important in the success of ACL surgery than in the time after trauma. Before ACL reconstruction, it is desirable that the quadriceps strength and control are good, the EHA is close to complete, and the swelling and inflammation in the knee are over (7).

In order to reduce the complications after ACL reconstruction, an appropriate rehabilitation method should be applied during the postoperative period and in the operation time suitable for the physical structure of the patient. The most commonly reported complication is graft failure (8). According to the complication rates that can be seen after knee surgery applications, the complication rate after ACL surgery is quite high. For example, while the postoperative complication rate for the meniscus was 1.5%, this rate was reported as 9% after ACL surgery (9).

Before ACL surgery, the level of injury, bone and soft tissue damage, hematoma, and other intra-articular pathologies (meniscus, posterior cruciate ligament, lateral ligaments, and cartilage) should be evaluated in detail. The presence of these pathologies causes limitation of movement in the knee after surgery. Especially due to the surgical treatment of pathologies in addition to an ACL injury, the operation time and the immobilization time are prolonged, thus prolonging the time of regaining joint movement (10).

When rehabilitation after ACL surgery is inadequately applied, the quadriceps weaken and the hamstrings remain relatively stronger, causing flexion contracture in the knee. In this case, the patient cannot stretch the patellar tendon sufficiently which causes patella inferia. In order to prevent this, early initiation of straight leg lifting and quadriceps stretching exercises prevent patellar tendon contracture and patella inference. The first goal is to ensure the extension. Patellar mobilization should be continued by the patient at regular intervals.

The complication rate in ACL reconstruction has been reported to be between 1.34% and 9% (8.9). Knowing the possible complications allows the surgeon to minimize the risk of complications by considering the risks.

1. Cyclops Lesion

It is the development of proliferative fibrous tissue in the intercondylar region that causes excretion limitation after ACL reconstruction. Anterior knee pain is diagnosed by the limitation of extension in the knee and a sound is heard when the knee is forced to an extension. It usually occurs because the tunnel debris cannot be adequately cleaned from inside the joint (11). Although medical treatment and physical therapy are the first treatment methods, arthroscopic debridement can be applied to patients who cannot get results from physical therapy.

2. Complications resulting from the surgical application

The most common cause of limitation in the range of motion of the knee after ACL reconstruction is the wrong placement of the graft. Placement of the femoral graft in the anterior according to the anatomical footprint leads to flexion limitation. Placement of the tibial graft in the anterior causes pain due to compression in the femoral intercondylar noc, extension shortage, and graft failure. Placement of the tibial graft to the posterior causes the graft to be located on a closer position to the vertical. This causes the loss of rotational stability, the impingement of ACL with posterior cruciate ligaments and early graft failure (12,13). The optimal knee angle during graft detection is 30-degree flexion and it has been reported that it provides more appropriate detection for rotational biomechanics of the knee (14).

Other complications related to the graft are the inability to remove the graft in full size and the formation of a hematoma in the area where the graft is taken. The meticulous surgical application can minimize these complications. In patients with persistent hematoma, after the drain is removed, the presence of infection should be considered, a culture should be taken from the hematoma site and appropriate antibiotic therapy should be initiated.

3. Infection

Although infection after ACL reconstruction is rare, it is the most important complication that can be seen in the early period. Infection in the knee joint after ACL surgery can be seen in 0.14-1.71% (15).

In a review investigating infections after ACL reconstruction, the frequency of septic arthritis was 0.5% at 53.6 months of follow-up and all of these patients received prophylactic antibiotics. In 78% of the patients, there was a reproduction in culture and the most common causative agent was staphylococcus epidermidis. It has been determined that hamstring grafts were used in 55% of these patients. It has been reported that if the suture material in the hamstring tendon graft is left in too many joints, inflammation will develop and the drainage that will occur may cause infection (16). In terms of infection, allograft preference is more reliable. The infection rate was reported to be 0.24%/0.59% in patients who underwent allograft in ACL reconstruction compared to patients who underwent autograft (17).

After ACL reconstruction, the degree of infection varies from superficial wound infection to septic arthritis and often the causative pathogen is bacteria. Septic arthritis is a complication that is difficult to treat as it causes serious problems with sudden and progressive pain, swelling, and fever. The determinant of the treatment is the detection of the microorganism and the severity of sepsis (15). Pain and swelling exacerbated after ACL reconstruction should be monitored for infection. If the C-reactive protein (CRP) value is not within the normal value of 0-5 in the first 2 weeks, an infection should be suspected (18). The general treatment approach is arthroscopic debridement with a specific antibiotic appropriate to the culture antibiogram result. During debridement, the graft should be protected as much as possible, but in resistant infections, the graft can be sacrificed for the health of the knee joint and the patient. While arthroscopic debridement is generally successful in acute infections, open debridement has been suggested in chronic infections. In patients where the graft was debrided due to infection, a revision of ACL was recommended 6-9 months after the infection completely disappeared (19).

In a study conducted for the most undesirable infection complication of ACL surgery, the graft was subjected to vancomycin solution without placing it in the knee and it was reported that no infection was found (20).

4. Arthrofibrosis

Painful limitation of movement that may develop after surgery in or around the joint is called arthrofibrosis. Limitation of movement, pain, and stiffness with effusion in the joint are known symptoms of arthrofibrosis (21). The prevalence of arthrofibrosis after ACL reconstruction has been reported to be between 2% and 35% (22). If arthrofibrosis is not noticed early after ACL surgery, the rehabilitation process is very much prolonged, and persistent pain or patient invulnerability due to limitation of movement may develop.

The arthrofibrosis classification (5), developed by Shelborn in 1996 according to the range of motion, was revised by Scott and Shelborn in 2018 (23).

Extension limitation has been reported to be caused by adhesion scar tissue and cyclops syndrome in the posterior capsule, while flexion limitation is caused by shortening of the quadriceps muscle, peripatellar scar tissue, medial-lateral ligament, and capsular stiffness (23).

There are two primary and secondary forms of arthrofibrosis. In primary arthrofibrosis, there is active proliferative massive and generalized fibrosis. In the primary form, an inflammatory reaction is observed, which causes resistant chronic synovitis. Secondary arthrofibrosis is local and benign. There are reasons such as limitation of joint movement before surgery, patellar tendon graft use, inadequate postoperative rehabilitation, additional intra-articular pathologies, wrong position of the graft, patella inferia, infrapatellar contracture syndrome, cyclops syndrome that can cause arthrofibrosis in the secondary form (23). However, a study reported that graft type, additional meniscus damage, and patient age were not linked to the development of arthrofibrosis, while female sex increased the risk of arthrofibrosis by 2.5 times. It has been reported that it may be linked to inappropriate postoperative rehabilitation due to poor tolerance of pain in women (22). While surgical application in the first week after ACL injury is thought to cause arthrofibrosis, it has recently been reported that the degree of inflammation in the knee is more valuable in determining the risk of arthrofibrosis (5,24).

The first treatment of arthrofibrosis is medical treatment and physical therapy. In cases where physical therapy is insufficient, manipulation under anesthesia, arthroscopic loosening of intraarticular adhesions or open arthroliz can be performed.

5. Deep Vein Thrombosis and Embolism

The frequency of venous thromboembolism (VTE) after ACL reconstruction has been reported to be between 0.2% and 14% (25,26). Clinical suspicion, diagnostic methods, demographic structure, surgical techniques, and thromboprophylaxis applications are thought to be effective in reporting these very different results. When ACL reconstruction is applied without low molecular weight heparin; The DVT rate was reported as 9.7%, the frequency of symptomatic DVT as 2.1%, and the pulmonary embolism rate as 0.1% (25). In patients who underwent heparin, DVT rate was reported as 0.55% and pulmonary embolism rate as 0.12% (8).

Risk factors for VTE are smoking, use of oral contraceptives, being over 30 years of age, being female, having a body mass index more than 30 kg/m, having chronic venous insufficiency, complex surgical application, tourniquet use for more than two hours and prolonged immobilization. Routine thromboprophylaxis is not recommended for patients who have undergone isolated ACL reconstruction and do not have VTE risk factors (25, 27). However, thromboprophylaxis will be appropriate in patients with risk factors with a previous history of DVT. Recurrent ACL damage may develop in 20% of patients after ACL reconstruction (28).

6. Vascular and Nerve Damage

Vascular complications after ACL reconstruction may cause serious legal problems and morbidity. Severe pain after surgery, hemarthrosis, and swelling in the popliteal region with pulsation are the first clinical signs. Pseudoaneurysm can occur due to drill sheaver or detection materials. The most commonly damaged arteries are; inferior-superior lateral genicular arteries, posterior tibial arteries, and popliteal arteries. It is not always safe to take pulsation in palpation of peripheral pulses or to see it intact with Doppler USG. Angiography is much more valuable in diagnosis. Depending on the degree of vascular damage, exploration or vascular repair can be performed (25).

Neurological complications that may develop in ACL reconstruction are often seen with sensory losses. Clinical findings include hypoesthesia, dysesthesia, neuroma, reflex sympathetic dystrophy, and pain. When the hamstring tendon graft is removed, the sartorial terminal and infrapatellar branch of the saphenous nerve may be injured (29). Sartorial branch injury can occur with proximal compression of the tendon scraper when the gracilis tendon graft is removed and can be seen in 23% (29). Infrapatellar branch damage can be seen during the skin incision applied to remove the hamstring tendon graft. This is the most commonly reported complication when taking a hamstring tendon graft and has been reported in 12%-84% (30,31). It has been reported that horizontal or oblique application of the skin incision can avoid iatrogenic infrapatellar branch damage (32). It has been reported that hypoesthesia and sensory loss are more common in patients with vertical skin incisions (33).

7. Femoral and Tibial Tunnel Complications

Failure to open the tunnels to anatomical footprints and femoral/tibial tunnels opened in an inappropriate position may cause graft failure and cause the failure of ACL reconstruction (34). Especially in the ACL reconstruction performed with the transtibial technique, opening the tibial tunnel in the wrong direction will cause the femoral tunnel to be misdirected. Opening the femoral tunnel to the posterior or opening the tunnel wide will cause a fracture in the posterior cortex and graft failure. When opening the femoral tunnel, the knee can be kept in flexion more than 90 degrees, and the posterior cortex and tunnel distance can be protected at a safe boundary to prevent the bursting of the femoral tunnel (blowout) (35). Tunnel expansion after ACL reconstruction can be seen in 68% of cases due to biological and mechanical factors. Biorelaxation;

swelling in the graft, and the presence of synovial fluid in the bone tunnel can occur due to increased cytokine levels. Mechanical loosening; movement of the graft in the tunnel, and tunnel/graft direction mismatch may develop due to early rehabilitation with the use of incorrect detection material (36). It has been reported that tunnel expansion will not have a negative effect on clinical outcomes, but fracture risk may be seen by joining the tunnels during revision (36).

8. Graft Complications

The most common complication of hamstring tendon graft, which is the most commonly used in ACL reconstruction, is the removal of the tendon as shorter than the standard. When semitendinous and gracilis tendon grafts are removed, tendon grafts may not be taken in sufficient length due to premature abrasion or anatomical variation in the tendons. In order not to strip the tendon grafts prematurely and not to escape to the proximal gland, it is very important that anesthesia provides sufficient muscle relaxation to the patient during the graft removal process. Tendons should be thoroughly removed from the surrounding soft tissues, the area should be wetted to provide lubrication, tendon scrapers should be kept parallel to the tendon, and the graft should be removed when the tendons are in sufficient tension and the knee is in flexion of about 45 degrees. If there is not enough graft length and thickness, graft removal from the opposite knee and bone-patellar tendon-bone graft removal options may be considered.

Complications of bone-patellar tendon-bone graft can be seen as patella fracture, patellar tendinitis, patellar tendon rupture, patella inferia syndrome, arthrofibrosis, anterior knee pain, and difficulty sitting above the knee (37). Patellar fracture risk should be avoided with an appropriate mini-saw, also incisions should not exceed graft block limits and graft should be removed in the form of trapezoids (38).

9. Complications resulted from Detection Materials

The most preferred fixing material for ACL reconstruction tendon graft detection is biodegradable screws. But since these screws are destroyed after a while, the pieces that fall into the joint can cause locking and cartilage damage in the joint. These biodegradable screws can cause osteolysis inside the tunnel, causing tunnel dilation, allergy or foreign body reactions, effusion in the joint, abscess, and encapsulation around the screw. When applying the screw, it may break, or screw migration may occur in the long term (39).

In the endobutton detection of the graft passed through the femoral tunnel, the tibial graft should be determined after making sure that the implant fits the lateral cortex completely. The most common complication with cross-screws is that the nails cannot detect the graft inside the tunnel.

10. Conclusion

In young patients and athletes with a complete ACL injury, ACL reconstruction has been the method used for many years for knee stability and functional improvement. According to the complication rates that can be seen after knee surgery applications, the complication rate after ACL surgery is quite high. The complication rate in ACL reconstruction has been reported to be between 1.34% and 9% . Knowing the possible complications allows the surgeon to minimize the risk of complications by considering the risks.

References

1- Öztürk S, Kılıç D. What is the economic burden of sports injuries? Eklem hastalık Cerrahisi 2013;24:108-11.

2- Atik, OŞ. Is anterior cruciate ligament surgery protective against osteoarthritis? Eklem hastalık Cerrahisi 2009;20(2):63.

3- Beynnon BD, Johnson RI, Abate JA, Fleming BC, Nichols CE. Treatment of anterior cruciate ligament injuries, part 1. AM J Sports Med 2005 ;33(10):1579-1602.

4- Wasilewski SA, Covall DJ, Cohen S. Effect of surgical timing on recovery and associated injuries after anterior cruciate ligament reconstruction. AM J Sports Med 1993;21(3):338-42.

5- Shelbourne KD, Patel DV, Martini DJ. Classification and management of arthrofibrosis of the knee after anterior cruciate ligament recontruction. Am J Sports Med 1996;24(6):857-62.

6- Harner CD, Irrgang JJ, Paul J, Dearwater S, Fu FH. Loss of motion after anterior cruciate ligament reconstruction. Am J Sports Med 1992;20(5):499-506.

7- Noyes FR. Knee Disorders, Surgery, Rehabilitation, Clinical Outcomes. Saunders Elsevier; 2009.

8- Cvetanovich GL, Chalmers PN, Verma NN, Cole BJ, Bach BR. Risk factors for short-term complications of anterior cruciate ligament reconstruction in the United States.Am J Sports Med 2016;44(3):618-24.

9- Salzler JM, Lin A, Miller CD, Herold S, Irrgang JJ, Harner CD. Complications after arthroscopic knee surgery. The Am J Sports Med. 2014;42(2):292-6.

10- Wasilewski SA, Covall DJ, Cohen S. Effect of surgical timing on recovery and associated injuries after anterior cruciate ligament reconstruction. AM J Sports Med 1993;21(3):338-42.

11- Jackson DW, Schaefer RK. Cyclops sendrome: loss of extension following intraarticular anterior cruciate ligament reconstruction. Arthroscopy 1990;6:171-8.

12- Romano VM, Graf BK, Keene JS, Lange RH. Anterior cruciate ligament reconstruction. The effect of tibial tunnel placement on range of motion. Am J Sports Med 1993;21(3):415–8.

13- Howell SM, Clark JA. Tibial tunnel placement in anterior cruciate ligament reconstructions and graft impingement. Clin Orthop Relat Res 1992;(283):187–95.

14- Debandi A, Maeyema A, Hoshino Y, Asai S, Goto B, Smolinski P, Freddie F. The Influence of Knee Flexion Angle for Graft Fixation on Rotational Knee Stability During Anterior Cruciate Ligament Reconstruction: A Biomechanical Study. Arthroscopy 2016;32(11):2322–8.

15- Judd D, Bottoni C, Kim D, Burke CPTM, Hooker MAJS. Infections following arthroscopic anterior cruciate ligament reconstruction. Arthroscopy 2006;22(4):375–84.

16- Binnet MS, Basarır K. Risk and outcome of infection after different arhroscopic anterior cruciate ligament reconstruction techniques. Arthroscopy 2007;23(8):862-8.

17- Kim SJ, Postigo R, Koo S, Kim JH. İnfection after Arthroscopic anterior cruciate ligament reconstruction. Orthopedics 2014;37:447-84.

18- Margheritini F, Camillieri G, Mancini L, Mariani PP. C-reaktive protein and erythrocyte sedimentation rate changes following arthroscopically assisted anterior cruciate ligament reconstruction. Knee Surg. Sports Traumatol Arthrosc 2001;9:343-5.

19- Matawa MJ, Evans TA, Wrigt RW, Shively RA. Septic arthritis of the knee following anterior cruciate ligament reconstruction: results of a survey of sports medicine fellowship directors. Arthroscopy 1988;14:717-25.

20- Schuster P, Schlumberger M, Mayer P, Eichinger M, Geßlein M, Reddemann F, Richter J. Soaking of the graft in vancomycin dramatically reduces the incidence of postoperative septic arthritis after anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc 2020.

21- Bosh U, Zeichen J, Lobenhoffer P, Albers İ. Arthrofibrose. Arthroskopie 1999;12(3):117-20.

22- Sanders TL, Kremers HM, Bryan AJ, Kremers WK, Stuart MJ, Krych AJ. Procedural intervention for arthrofibrozis after ACL reconstruction: trends over two decades. Knee Surg Sports Traumatol Arthrosc 2015. doi:10.1007/s00167-015-3799-x.

23- Lawrance SE, Shelbourne KD. Treatment and Rehabilitation of Arthrofibrosis of the Knee (Chap. 52). In: Giangara CE, Manske RC editörs. Clinical Orthopaedic Rehabilitation: A Team Approach, 4th ed. Elsevier; 2017. p. 353-8.

24- Kwoc C, Harrison T , Servant C. The optimal timing for anterior cruciate ligament reconstruction with respect to the risk of postoperative stiffness. Arthroscopy 2004;20:4-6.

25- Janssen RP, Reijman M, Janssen DM, Mourik van JBA. Vascular complications after ACL reconstruction and DVT prophylaxis. In: Prodromos CC(ed): The anterior cruciate ligament. Reconstruction and basic sciense. Philadelphia, USA: Saunders, an imprint of Elsevier, 2015, 592-B.

26- Gaskill T, Pullen M, Bryant B, Sicicnano N, Evans AM, DeMaio M. The Prevalence of symptomatic deep venous thrombosis and pulmonary embolism after anterior cruciate ligament reconstruction. Am J Sports Med 2015;43:2714-9.

27- Geerts WH, Bergqvist D, Pineo GF, Heit JA, Samama CM, Lassen MR, Colwell CW. Prevention of venous thromboembolism: American College of Chest Physicians Evidence-Based Clinical Practice Guidlines (8th Edition). Chest 2008;133:381-453.

28- Hewett TE, Di Sitasi SL, Myer GD. Current concepts for injury prevention in athletes after anterior cruciate ligament reconstruction. Am J Sports Med 2013;41:216-24.

29- Sanders B, Rolf R, Mc Clelland W, Xerogeanes J. Prevalence of saphenous nerve injury after autogenous hamstring harvest: an anatomic and clinical study of sartorial branch injury. Arthroscopy 2007;23:956-63.

30- Jameson S, Emmerson K. Altered sensation over the lower leg following hamstring graft anterior cruciate ligament reconstruction with transverse femoral fixation. Knee 2007;14:314-20.

31- Figueroa D, Calvo R, Vaisman A, Campero M, Moraga C. İnjury to the infrapatellar branch of the saphenous nerve in ACL reconstruction with the hamstring technique: clinical and electrophysiological study. Knee 2008;15:360-3.

32- Tavakoli Darestani R, Bagherian Lemraski MM, Hosseinpour M, Kamrani-Rad A. Electrophysiological assessment of injury to the infrapatellar branches of the saphenous nerve during anterior cruciate ligament recontruction using medial hamstring auto-grafts: vertical versus oblique harvest site incisions. Arch Trauma Res 2013;2:118-23.

33- Sabat D, Kumar V. Nerve injury during hamstring graft harvest: a prospective comparative study of three different incisions. Knee Surg Sports Traumatol Arthrosc 2013;21:2089-95.

34- Coobs BR, Spiridonov SI, LaPrade RF. Intra-artıcular lateral femoral condyle fracture following an ACL revision reconstruction. Knee Surg Sports Traumatol Arthrosc 2010;18(9):1290-3.

35- Keyhani S, Vaziri AS, Shafiei H, Mardani-Kivi M. Femoral Condyle Fracture during Revision of Anterior Cruciate Ligament Reconstruction: Case Report and a literatüre. Arch Bone jt Surg 2015;3(2):137-40.

36- Julien TP, Ramappa AJ, Rodriquez EK. Femoral condylar fracture through a femoral tunnel eleven years after anterior cruciate ligament reconstruction: a case report. J Bone Joint Surg Am 2010;92;963-7.

37- Erdil M, Aşık M, Şen C, Polat G. Heterotopic bone formation following anterior cruciate ligament reconstruction with BPTB autograft. Acta Orthop Traumatol Turc 2012;46(1):72-6.

38- McGuire DA, Hendricks SD. Anterior cruciate ligament reconstruction graft harvesting: pitfalls and tips. Sports Med Arthrosc 2007;15:184-90.

39- Pereira H, Correlo VM, Silva-Correia J, Oliveria JM, Reis RL, Esprequeira-Mendes J. Migration of ‘’bioabsorbable’’ screws in ACL repair. How much do we know? A systematic review. Knee Review of Surg Sports Traumatol Arhrosc 2013;21(4):986-94.