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Complications after Total Knee Arthroplasty

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Abstract

Nowadays, the incidence of knee arthritis increases with the prolongation of human life and the increase in world population. As a result, total knee arthroplasty application rates increased and surgeons gained more experience. There have also been technical advances and total knee arthroplasty operations have been performed using better implants. However, despite these developments, the number and variety of complications are increasing. In addition to performing total knee arthroplasty correctly, it is now becoming more important to recognize complications that may or may develop. Variety of complications after total knee replacement; from minor skin problems to life-threatening complications. In this review article, we aimed to investigate early and late complications during and after total knee replacement surgery.

Keywords: knee, total knee arthroplasty, survival, complications

1. Introduction

Total knee arthroplasty is an effective treatment option which has been applied with increasing rates in recent years with its highly satisfactory results. Recently increased total knee arthroplasty (TKA) procedures increase the number of complications too. In addition to proper patient selection, an accurate surgical technique, early diagnosis, and proper management of complications are required. Complications of TKA have a wide range. Complications vary from small skin problems to mortality. The development of complications may be due to many factors. Some of these are listed below:

- Error in surgical technique.
- Medical error.
- Nurse error.
- Patient non-compliance.
- Trauma.
- Associated comorbid diseases.

Reviewing all the risk factors before surgery and being prepared for the complications that may occur may be lifesaving in TKA, which is currently applied frequently. It is important to recognize, identify, and classify the complications in a timely manner in the correct and effective management of complications. The ambiguity about the complications of TKA in the literature helped identify and classify the complications in a study conducted in 2013 by the knee community [1]. According to this study, 22 complications were described. These are [1]:

- Bleeding
- Wound problems
- Thromboembolism
- Neural deficit
- Vascular issues
- Medial collateral ligament injury
- Instability
- Malalignment
- Stiffness-toughness-contracture
- Deep wound infection
- Fracture
- Extensor mechanism injury
- Patellofemoral dislocation
- Tibiofemoral dislocation
- Bearing surface wear
- Osteolysis
- Implant loosening
- Implant breakage
- Reoperation
- Revision
- Re-hospitalization and mortality [1]

When the complications are examined, it is seen that some of them are simple and easy to overcome with a short-term solution, while some of them can be serious and can go to revision arthroplasty. The number of complications such as implant

Intraoperative	Early postoperative	Late postoperative
Vascular injuries	Bleeding	Instability
Neurological complications	Superficial skin problems	Joint stiffness
Extensor mechanism injury	Deep skin problems	Periprosthetic joint infection
Patellar tendon injury	Deep vein thrombosis	Periprosthetic fractures
Quadriceps tendon injury	Pulmonary embolism	Aseptic loosening
Patella fractures		Osteolysis
Medial collateral ligament injury		Patellofemoral joint problems

Table 1.
Intraoperative, early postoperative, and late postoperative complications.

fracture and polyethylene surface wear has been reduced due to the techniques and innovations in implant materials and designs. In a study, it was shown that the most common cause of revisions in the first 5 years postop was infection, and the reasons for revision in the next 5 years were polyethylene loosening [2]. Complications will be classified as intraoperative, early postoperative, and late postoperative complications (Table 1).

2. Intraoperative complications

2.1 Vascular injuries

Although arterial injury during knee replacement is rare, it may have serious results from limb loss to mortality. Arterial injuries can be seen as thromboembolism, direct vascular laceration, pseudoaneurysm, and arteriovenous fistula [3]. The incidence is reported to be 0.03–0.2% in the literature [4].

Vascular injuries may develop due to the thermal effect of cement polymerization, joint manipulations, dislocations, and excessive manipulation [5]. Considering the issue as specific to the total knee arthroplasty, care should be taken against vascular injury during posterior cruciate ligament and posterior capsular release during femoral condylar cutting. Atypical localization of vascular structures due to changes in adhesions and normal anatomy in revision cases increases the risk of vascular injury twice as compared to primary cases [6]. Nowadays, increasing procedures of TKA bring about the possibility of vascular injuries although they are rare. Therefore, it is necessary to take precautions against vascular injuries that may develop, to identify risky patients and to make an early diagnosis. For this, a good anamnesis and physical examination are essential. It is important to examine the presence of hypertension, diabetes, smoking, and vascular claudication. Coldness of the extremities to be operated during physical examination, skin atrophy and thinning, prominent vascular structures, ulcerative wound, and distal arterial pulse weakness are the findings that need attention. In addition to these findings, the presence of vascular calcifications in radiological scanning, a history of bypass, and an ankle-brachial index below 0.9 are other findings that should be considered. No tourniquet should be used in patients with the abovementioned conditions [7]. Embolism and arterial insufficiency may develop due to tourniquet effect in patients with vascular disease and atheroma plaque in the superficial artery [8]. It has been shown that during the manipulation of the superficial femoral artery fixed during tourniquet effect, intimal damage may occur [9]. Improper placement of retractors can also cause damage by direct mechanical trauma [10]. Particularly during insertion of the posterior retractor, a 1 cm area

in the lateral portion of the midline was identified as a risky area [11]. In a cadaver study, neurovascular structures on the tibial side were mapped on a clock diagram. Accordingly, the popliteal vein at 12 o'clock, the popliteal artery at 1 o'clock, and the anterior tibial artery at 2 o'clock for the left knee were shown as in place [12]. Cautious use of the saw between 11 and 3 o'clock defined in the tibial cutting is important in protecting vascular structures [12].

If vascular injury is suspected the tourniquet should be deflated, and bleeding control should be performed before the incision is closed. The possibility of arterial injury should be taken into consideration in the presence of excessive and pulsatile bleeding and in the absence of peripheral pulses. Although recent studies suggest bleeding control after routine tourniquet deflation prior to incision, its benefit is controversial [13]. The surgeon should perform a postoperative peripheral pulse examination routinely, suspect acute ischemia in the presence of cold and delayed distal capillary filling, and request cardiovascular consultation [14]. Acute ischemia cases with delayed diagnosis of 4–6 hours cause irreversible damage. Prophylactic fasciotomy is performed after revascularization [14].

Pseudoaneurysm may present with pulsatile swelling in the popliteal fossa due to direct damage to the popliteal artery during surgery. Doppler ultrasonography is useful in the diagnosis. In the treatment, excision of the lesion and repair with vascular graft is applied after embolization [15]. Arteriovenous fistula is less common. It usually occurs due to injury to the medial and lateral geniculate arteries and its branches. It may present with pulsatile swelling in the popliteal region that gives “trill.” Hemarthrosis or pseudoaneurysm may develop. Ultrasound and angiography are used for diagnosis [16]. The detected lesions should be evaluated together with cardiovascular surgery, and treatment should be planned. Embolization, lesion excision, and graft repair are treatment options [15].

2.2 Neurologic complications

Nerve injuries are rare during TKA. Peroneal nerve injury is the most common of these [17]. Sacral plexopathy and sciatica neuropathy are also seen, although rarely [18]. Risk factors for neurological injury are [19]:

- Flexion deformity
- Advanced valgus deformity
- Presence of an intra-articular hematoma

It has been shown that the risk of nerve injury is increased in patients with rheumatoid arthritis [20]. However, none of these risk factors is directly related to nerve injury [18]. Nerve injury is associated not only with the surgical procedure but also with the anesthesiologist-induced regional anesthesia [21]. Hypertension, diabetes, nerve compression history, presence of tethered cord, and rheumatoid arthritis in the patients increase the risk of neural complications secondary to regional anesthesia [22]. The duration of tourniquet use was associated with nerve injuries. According to this, in the tourniquet applications exceeding 2 hours, the risk of peroneal and tibial nerve injuries including 89% peroneal nerve was determined as 7%. All of these have been shown to get recovery. In procedures exceeding 2 hours, the 10–30-minute break and deflation of the tourniquet reduces the complication rate [19]. Although there is a minimal effect on the functional results of the patients effect on the functional results of the patients during the follow-up, paresthesia and numbness are seen in the distal and lateral site of incision due to the injury of the infrapatellar branch of

the saphenous nerve. It is seen in the literature at a rate of 25–76%, and most of these recover spontaneously [23]. Nerve injuries are difficult to detect intraoperatively. In the presence of postoperative nerve injury, physical therapy should be planned immediately. EMG examination is recommended after 3 months [20]. If no improvement is observed, nerve exploration may be planned in the future.

2.3 Extensor mechanism injuries

The extensor mechanism in the knee joint consists of quadriceps muscle group, quadriceps tendon, patella, patellar retinaculum, patellar tendon, and tuberositas tibia. Extensor mechanism integrity may be impaired during surgery [20]. Although extensor mechanism injuries occur more frequently postoperatively, they may also occur intraoperative. The incidence is reported to be between 1 and 12% [24]. The treatment of extensor mechanism injuries is quite difficult and the results are not satisfactory.

2.3.1 Patellar tendon rupture

Rupture usually occurs at the site of insertion to the tuberositas tibia. The risk of development is less than 1% [25]. Less frequently, intratendinous and infrapatellar tendon rupture may also occur [25]. The risk of injury increases when patellar tendon mobility decreases. These are [26]:

- Patella baja
- Previous surgery
- Severe limitation of movement in the knee

The risk of tendon injury especially on stiffness knees due to forced manipulations and during the tibial bone cutting increases during surgery. The most common injury mechanism after surgery is falling onto the knee while knee is flexed [27]. Patellar tendon injury without trauma is seen by weakening the tendon after repeated contact of the polyethylene insert [27].

In patients with patellar tendon rupture, pain, swelling, loss of extension, and a palpable defect at the infrapatellar side are detected.

Age, functional status, tendon rupture localization, and soft tissue status are the determinants of the treatment. Splitting and bracing are considered in patients who do not have functional expectations and are unsuitable for surgery [28]. Treatment of acute patellar tendon rupture intraoperative is primary repair [26]. Several techniques have been described using staple and suture anchors for this purpose [28]. Reconstruction techniques are used in patients with poor soft tissue quality. For this purpose, biological materials (hamstring tendon autograft, achilles, peroneal tendon autograft, and extensor mechanism allograft) and synthetic materials can be used [28–30].

2.3.2 Quadriceps tendon rupture

It is very rare. It is especially seen as a rupture from the intension side to the patella. Excessive patella cutting, previous quadriceps snip, or V-Y tipping are risk factors [28]. The clinical finding is similar to patellar tendon rupture.

Good results have been reported with plaster cast in partial tears [31]. Extensor loss greater than 20° is considered a complete tear and should be treated surgically.

It has unsatisfactory results due to high complication rates and tendency to re-rupture depending on tendon quality and soft tissue condition.

2.3.3 Patella fractures

Patellar fractures are the most common injury among the extensor mechanism injuries [24, 32]. In general, the risk increases with excessive bone cutting while preparing for patellar component. Patellar fracture may occur by direct trauma to the anterior knee or as an avulsion due to the pull of the quadriceps muscle [32].

For diagnosis, pain, swelling, and extensor insufficiency are detected in front of the knee. Lateral knee radiography and tomography in case of clinical suspicion are helpful imaging methods for the diagnosis.

A classification has been developed to assess implant stability and extensor mechanism continuity for periprosthetic patella fractures [33]. Type 1, a stable implant and continuous extensor mechanism; Type 2, a stable implant but a discontinuous extensor mechanism; and Type 3, which indicates instable implant and discontinuous extensor mechanism. Patellar bone stock is classified as 3A if good and 3B if poor. Treatment is also determined according to this classification. Conservative treatment methods are preferred for type 1 cases, while surgical treatments are preferred for types 2 and 3 [33]. In recent studies, it is reported that 40–50% of complications occur and more than half strength loss of extensor mechanism is observed [34].

2.4 Medial collateral ligament injury

During total knee replacement, medial collateral ligament (MCL) is important for soft tissue stabilization and coronal plan stability. The incidence of iatrogenic MCL injury is 2.2–2.7% [35]. In the case of surgical injuries, direct repair, constrained prosthesis use, and even revision at the same session are among the options [36]. Unrecognized MCL injuries during surgery cause early instability. This leads to early implant wear and consequently the need for early revision. Therefore, it is important to diagnose and repair the injury during surgery [37]. Sudden instability in the valgus stress test during knee stabilization indicates MCL injury. Injury may occur from femoral insertion, within the tendon or tibial insertion [38].

Primary repair technique varies according to injury level. Fixation with screw is recommended if MCL injury occurs from its femoral insertion site. Otherwise, if it is through tendon, repairing with insoluble suture technique is recommended. Finally, if MCL injury occurs from its tibial insertion site, both insoluble suture anchor technique and fixation with staple technic are recommended [39, 40]. Factors that increase the risk of medial collateral ligament injury during surgery are as follows [39]:

- Using a larger saw blade than femoral condyle
- Delayed excision of medial side osteophytes
- Performing challenging manipulations of varus-valgus
- Patients with flexion contractures [39]

Patient-related risk factors include obesity and severe deformities [41, 42].

A certain algorithm has not yet been established for the treatment of iatrogenic MCL injuries that occur intraoperative. Many treatment methods with

disadvantages and advantages have been used [39, 43, 44]. The traditional method is using constrained prosthesis. However, in this method, it was shown that the stress load on the implant increased and direct repair and treatment with non-constrained prosthesis were recommended instead. In addition, augmentation or increase in polyethylene thickness has been proposed [45]. In one study, it was shown that the risk of instability was 57% in the use of non-constrained prostheses independent of the repair technique after MCL injury [37]. In a 2016 study, four treatment modalities were compared after MCL injury. These are the use of non-constrained prosthesis only, the use of non-constrained prosthesis with primary repair, the use of non-constrained prosthesis only, and the use of constrained prosthesis with primary repair. In 23 patients, the most appropriate treatment method according to the knee community scoring was found to be the use of constrained prosthesis only [46]. However, due to the small number of patients, larger series of studies are needed to determine which treatment is most appropriate.

3. Early postoperative complications

3.1 Bleeding

Bleeding is seen in varying rates between 0 and 39% after TKA [47]. This naturally increases the need for blood transfusion. Intraoperatively, care should be taken about bleeding and good bleeding control is established. Thus, the amount of bleeding is reduced to a minimum. As a result, the risks of immunological reaction due to transfusion are reduced.

Bleeding tolerance is low in patients with comorbid disease and in patients with insufficient cardiac capacity, and the risk of complications increases even in small amounts of bleeding. Preoperative blood preparation before surgery and limitation of the use of anticoagulants are among the measures that can be taken. Precautions during and after TKA surgery can reduce the amount of bleeding. These methods are as follows:

- Use of femoral intramedullary plugs [48]
- Hypotensive anesthesia [49]
- Cryotherapy and Jones bandage [50]
- Use of fibrin tissue adhesive [50, 51]
- Clamping the drain [52–54]
- Application of tranexamic acid [55]

Fibrinolysis is activated by surgical trauma and tourniquet use [56]. Increased fibrinolytic activity causes increased bleeding during TKA. Tranexamic acid shows an anti-fibrinolytic effect by inhibiting the conversion of plasmin to plasminogen [57]. Tranexamic acid can be administered in four different ways: intravenous, oral, intramuscular, and intra-articular [55]. Transition to maximum plasma levels is 30 minutes for intramuscular use, 5–15 minutes for intravenous use, and 2 hours after oral use [58]. Patients with total knee arthroplasty may be treated with a fast-acting intravenous route. Many studies have shown that administration of tranexamic acid after tourniquet deflation and postoperative dose repeat reduces the amount of

bleeding and the need for transfusion [59–61]. However, many different protocols for the use of tranexamic acid have been implemented. Preoperative single dose and repeated dose every 8 hours for 3 days have been described in the literature and shown to be effective [62]. In a study conducted in 2011, tranexamic acid was administered at a dose of 10 mg/kg 10 minutes before the tourniquet was opened, and the same dose was repeated 3 hours postoperatively. Five hundred mg tranexamic acid was administered orally 3 times a day for 5 days. At the end of this study, it was shown that the amount of hemorrhage and the rate of transfusion decreased effectively [55].

3.2 Skin healing problems: superficial and deep infections

The incidence of wound problems after TKA is 1–25% [63]. The skin problems may be delayed wound healing, skin necrosis, traumatic or atraumatic separation of the lips of the wound, prolonged serous discharge at the wound site, formation of superficial or deep hematoma, allergic reaction to patch, suture material or dressing materials, bullae formation, fat necrosis, bleeding, keloid formation, and superficial or deep infection [64].

Etiologic reasons that may develop the problem before TKA should be determined in advance, and appropriate measures should be taken [65]. Presence of systemic diseases such as diabetes, hypertension, rheumatoid arthritis, and vascular insufficiency, which may adversely affect wound healing before TKA, should be questioned. Since the soft tissues around the knee are thinner than the other parts of the body, even the smallest problem that may occur at the wound site can cause serious complications. Incision planning should be made carefully in the case of a history of operation from the same place and scarring beforehand, and if necessary, plastic surgery assistance should be taken.

3.2.1 Skin healing problems

Factors adversely affecting wound healing are obesity, hypertension, diabetes, smoking, chronic drug use, steroid use, previous radiotherapy, scarring, inflammatory disease, malnutrition, albumin levels below 3.5 g/dl, and hemoglobin levels below 10 g/dl. Transferrin and lymphocyte levels may also contribute to wound healing problems [66]. Therefore, a detailed anamnesis and physical examination and laboratory examination before surgery give an idea about possible skin problems. Accordingly, measures are taken, replacement therapies are given, and surgery may be postponed until the current pathology is corrected, if necessary. Adjustment of fasting blood sugar levels below 200 g/dl and keeping HbA1C below 6.5 in patients with diabetes will reduce the risk of possible wound problems [67].

Patients with a body mass index above 30 kg/m² are 6 times more likely to have infection and wound problems [66]. In obese patients, dietician support should be given before surgery; unnecessary exclusion should be avoided during surgery, and soft tissue surgery should be applied carefully.

A study of smoking patients showed that there were 2 times more wound problems [65]. Because of the vasoconstrictor effect of nicotine in the cigarette, it is recommended to quit smoking 60 days before surgery due to decreased blood supply at the wound site.

Incision planning should be performed in the presence of scar after previous surgery. In the presence of a single longitudinal incision without problems, the same incision should be used. If the old incision cannot be used, a distance of at least 7–8 cm should be left. If there is more than one old incision scar in the anterior part of the knee, the most lateral scar is used considering that the anterior knee feeding is from the medial perforating artery. In addition, the lateral soft tissue flap should not be dissected too

much [65]. Unnecessary retractors and additional soft tissue damage should be avoided during surgery. The wound lips should be exactly opposite to each other. Overstretched closing should be avoided. This should be checked with capillary filling time.

Especially in patients with risk factors, it should be performed without tourniquet or at low pressures [65]. Difficult rehabilitation in the early postoperative period should be postponed if possible until it is ensured that there are no wound problems.

Hematoma formation increases the risk of infection [65]. Therefore, measures should be taken to prevent the formation of hematoma. These include no dead space during wound closure, good bleeding control, use of a Jones bandage, and avoidance of overdose of the prophylactic anticoagulants used [65, 68]. Once the hematoma has developed, a needle aspiration can be performed. However, if the hematoma is organized and the drainage cannot be achieved, discharge and debridement can be achieved by arthrotomy under operating room conditions.

The presence of necrosis in the wound leads to catastrophic consequences. Respect to soft tissue is the most important step to prevent necrosis development. The depth of necrosis is important. Superficial necrosis can be treated by local intervention. If larger, debridement and full-thickness skin grafts or fasciocutaneous flaps are required [69]. If necrosis includes full-thickness soft tissue, closure with fascial skin or muscular skin graft should be performed after urgent aggressive debridement [70].

3.2.2 Superficial and deep infections

Despite all current precautions, surgical site infections remain the most serious and feared complications of TKA. After TKA, patients should be followed up with daily dressings, and wound discharge should be evaluated carefully. Prolonged wound discharge is defined as a discharge that lasts more than 48 hours regardless of the amount of drainage [64]. Wet wounds greater than 2×2 cm are considered abnormal after 72 hours and are associated with fat necrosis, hematoma, necrosis, or poor closure of the fascia. They are reported as 1–10% after primary knee replacement [65]. In the early stage of treatment, usually dressing and immobilization for 3–5 days is recommended [71]. Continuous discharge for 72 hours is dangerous. If it exceeds 5 days, debridement should be applied in operating room conditions as it will increase the risk of superficial or deep infection [64].

Superficial infection: It is defined as infection of the soft tissue above the skin—subcutaneous and deep fascia that has not passed under the deep fascia, not opened into the joint cavity. It occurs most frequently in the first 30 days after surgery. The incidence of superficial infection after TKA has been reported as 10% [72]. It may occur through direct contamination or blood. Improper preparation of direct contamination sterilization environment, inadequate surgical field preparation, presence of sloppy surgical team, non-sterile dressing materials, and application may occur as a result of the presence of infected patients in the same environment [73]. The risk of direct contamination can be minimized by precautions. Hematogen contamination can occur if there is any other focus of infection in the body. Therefore, in the presence of a possible infection focus with detailed anamnesis and examination before the operation, the current focus treatment can be planned through detailed examination.

Infection after TKA can be evaluated as patient-related risk factors, surgical intervention-related factors, and postoperative factors [66, 68, 74–77].

- *Patient-related risk factors* include advanced age, previous knee surgery, previous knee infection, steroid use, presence of inflammatory disease, obesity, diabetes, smoking, intravenous drug use, hematologic diseases, oncologic

diseases, above ASA score 2, immunosuppressive use, regional skin problems, old incision scars, previous radiotherapy procedures, malnutrition, vascular insufficiency, albumin level below 3.5 g/dl, transferrin level below 200 mg/dl, hemoglobin level below 10 g/dl [78, 79].

- *Surgical intervention related risk factors* include prolonged surgical time of more than 2 hours, absence of laminar flow in the operating room, transfusion, use of hinged knee prosthesis, failure of surgical team to comply with asepsis, and sterility rules [80].
- *Operative period related risk factors* include prolonged hospital stay pre- and postoperative, lack of appropriate antibiotic prophylaxis, hematoma formation, and prolonged wound drainage for more than 5 days [81].

Superficial wound infection is considered with the presence of at least one of the following: discharge from the wound incision, culture of the wound from aseptic conditions, suspicion of infection in clinical evaluation, disproportionate pain, increased temperature, erythema, and localized swelling [79].

In superficial wound infection, unlike deep infection, there is no progressive change in erythrocyte sedimentation rate, C-reactive protein level, and peripheral leukocyte count; the increase is below 25% [82]. In addition, leukocytes in synovial fluid are detected less than 2000/ml, and polymorphonuclear leukocytes are detected under 50%. Alpha defensin and leukocyte esterase tests are negative [71].

When superficial wound infection is detected, the development of deep infection can be prevented by early intervention. Otherwise, it may develop into periprosthetic infection and cause catastrophic results. In the presence of superficial infection, local wound care due to the underlying cause and debridement should be performed if appropriate anti-therapy is required [80]. In the selection of antibiotics, consultation with infectious diseases should be requested. Antibiotherapy is continued after reproduction. If deep infection is excluded in surgical debridement, the joint should not be opened, and the implant should not be touched [83]. Hyperbaric oxygen therapy has a positive effect on appropriate patient selection [84].

3.3 Deep vein thrombosis and pulmonary embolism

Deep vein thrombosis is the general name of thrombosis in the venous circulatory system. It occurs most commonly in the deep veins of the lower extremity [85]. From asymptomatic deep vein thrombosis to pulmonary embolism, which can be fatal, it can be confused with clinical manifestations of varying degrees [85]. It is one of the important complications that increase morbidity and mortality after TKA [86]. Even with mechanical or pharmacological methods, the incidence of asymptomatic DVT is 5.1%, and the incidence of symptomatic DVT is 0.4% [87]. The mortality rate due to pulmonary embolism after TKA is 0.08% [88].

It is important to understand the Virchow triad in the pathogenesis of DVT development. There is a slowdown in blood flow (stasis), endothelial damage, and hypercoagulability [89]. The admixture of fat and bone marrow particles into the venous system after engraving of the femoral canal during TKA explains the hypercoagulability branch of the Virchow triad. Hyperflexion of the leg during surgery and anterior manipulation of the tibia with retractors explain endothelial damage. In addition, this manipulation causes obstruction of the popliteal veins and prolonged immobilization of the leg, leading to venous pooling and stasis [89].

3.3.1 Risk factors

- VTE risk increases after age of 40 and doubles every 10 years after that age [90]. Age increases the risk of VTE regardless of other risk factors.
- Genetic factors are also an important parameter that increases the risk of DVT. Factor V Leiden mutation that causes thrombophilia, as well as protein C, protein S, and antithrombin III deficiency are among the factors that increase the risk of DVT.
- Although tourniquet use has been reported to cause venous stasis, it has been shown that it does not significantly increase the risk of DVT because of its fibrinolytic effect [91, 92].
- The type of anesthesia also affects the risk of developing DVT. General anesthesia has been shown to increase the risk of DVT compared to neuraxial anesthesia (spinal or epidural). Neuroaxial blockade causes vasodilatation in the lower extremities and reduces venous pooling; therefore it explains the mechanism of action [93].
- Other risk factors that increase the risk of DVT are immobilization, smoking, oral contraceptive and hormone use, history of VTE, obesity, malignancy, and difficult knee manipulations.

3.3.2 Diagnosis

A painful, swollen, and reddened leg after TKA should suggest the possibility of DVT. Incomplete DVTs usually do not show signs. Incomplete DVTs are seen especially after arthroplasty. Clinical findings are seen in 1% of all DVT cases. Physical examination findings include redness, swelling, and Homan's sign test and Pratt test positivity. Clinical Wells risk score was established for the diagnosis of deep vein thrombosis [94]. Clinical Wells Scoring criteria are malignancy, paralysis (paresthesia or splinting lower extremity), immobilization for more than 3 days, localized tenderness in the deep venous system, swelling of the lower extremity, 3-cm-diameter differentiation from the other leg, pretibial gode positive edema, history of deep vein thrombosis, and collateral superficial veins. The presence of each risk factor was evaluated as 1 point, and clinical scoring of 3 and above was found to be a high risk for the development of deep vein thrombosis.

Clinical data are not sufficient for the diagnosis of DVT. Therefore, further examination with clinical risk scoring, D-dimer level, Doppler ultrasonography, contrast-enhanced venography, CT, and MRI should be performed. Venography is the best method for the diagnosis of DVT in the lower extremities. The accuracy rate was 97% in the lower extremity veins and 70% in the iliac veins [95]. Venography is not preferred as first-line imaging because it has a 3% risk of DVT and is an invasive method, and also it requires contrast matter that can be toxic to the kidneys. Doppler USG is the most commonly used first-line imaging method because of its cheapness, reproducibility, and patient comfort in the suspicion of DVT. Proximal DVT sensitivity was 96%, distal DVT sensitivity was 44%, and DVT specificity was 93% [96].

Pulmonary embolism should be suspected in the case of sudden shortness of breath, tachypnea, tachycardia, and chest pain after TKA. However, since there are many other diseases with these findings, risk factor assessment and effective differential diagnosis should be made. Wells pulmonary embolism clinical probability scoring was established [97]. Pulmonary angiography is the gold standard for the diagnosis of pulmonary embolism [85].

3.3.3 Prophylaxis

Primary treatment of DVT and related pulmonary embolism is very difficult and cost-effective. Therefore, it is more plausible to establish protocols that prevent the development of DVT and to give ideal prophylaxis. Many pharmacological and mechanical prophylaxis methods are available. The aim is to prevent the development of DVT and not to increase bleeding. Therefore the drug or method of choice should be patient-specific:

- *Mechanical prophylaxis*: The aim is to reduce venous stasis by compressing the lower extremity and to increase fibrinolysis. The risk of hemorrhage is very low, and, if applied correctly, there are almost no complications. Patient compliance is important in mechanical prophylaxis and is the only negative aspect of the method. Mechanical prophylaxis methods include early mobilization, in-bed exercise, use of antithromboembolic socks, and pneumatic compression devices. It has been shown that intermittent pneumatic compression devices provide as effective prophylaxis as chemical prophylactic agents, and the American College of Chest Physicians (ACCP) recommends the use of mechanical prophylaxis [98].
- *Chemical prophylaxis*: Many agents are used. They all have their own advantages and disadvantages. Risk factors are determined by patient-based evaluation and the most appropriate agent should be preferred:
 - *K vitamin antagonist warfarin*: It prevents the formation of fibrin by inactivating 2, 7, 9, and 10 of the clotting factors. It also inhibits the activation of fibrinolysis-causing protein C and S. Since this effect occurs earlier, it creates a temporary clotting condition. Patients with warfarin should therefore be heparinized until the effect on coagulation factors begins. The anticoagulant effects of warfarin are reversible and monitored by the international normalization rate (INR) measurement. Interaction with other drugs, narrow confidence interval, and dual effect have recently reduced the usage of post-TKA [99, 100].
 - *Heparin*: It acts by inactivating circulating antithrombin III. Antithrombin III also inactivates circulating factors 2, 9, 10, 11, and 12. The use of standard heparin has recently been restricted due to the low risk of bleeding due to low-molecular-weight heparin.
 - *Acetylsalicylic acid*: It acts as an anticoagulant by blocking thromboxane A₂, which is necessary for platelet aggression. Recent studies have shown that VTE can be used prophylactically [101].
 - Other oral anticoagulants that may be used: *rivaroxaban* (direct factor Xa inhibitor), *apixaban* (direct factor Xa inhibitor), and *dabigatran* (direct thrombin inhibitor).

4. Late postoperative complications

4.1 Instability

The development of instability after TKA is the third most common cause of revision (17%) after aseptic loosening and infection [102]. Patients present with signs of pain and swelling with movement and weight loss. There may also be pain, emptiness, or abnormal friction and rattling noise in some range of motion.

On the knee during walking, varus or valgus orientation and recurvatum can be seen. Anterior knee pain during sitting up is typical in flexion instabilities. The heaviest table is knee dislocation. The treatment of instability is revision surgery. However, the rate of recurrent instability after revision was 18–60% [103]. This high rate is usually due to the lack of correct identification of the cause of instability.

A clinical classification of knee instability was established. Components of this classification are flexion-extension gap mismatch, component alignment problem, isolated ligament failure, extensor mechanism failure, component loosening, and global instability [103].

4.1.1 Risk factors

The success of total knee replacement depends on the correct alignment of the lower limb mechanical axis. It is recommended that the postoperative lower limb mechanical axis should be in neutral alignment. The tibial cut surface in the coronal plane should be made perpendicular to the mechanical axis of the tibia. Similarly the femoral cut in the coronal plane should be made perpendicular to the mechanical axis of the femur. It is necessary for a stable knee to obtain a rectangular gap in both flexion and extension after bone incisions and soft tissue release in TKA. Balancing the gaps is important to ensure stability and for full range of motion. Flexion gap controlled by posterior femoral condylar cut and tibial cut. Extension gap controlled by distal femoral condylar cut and the tibial cut. If there is a symmetric gap problem, tibial bone cut is adjusted first; otherwise if there is asymmetric gap problem, adjust femoral bone cut first. For example, if the knee is tight both in extension and flexion, it is called symmetrical gap problem, and its solution is to cut more proximal tibia. The asymmetric gap is one of the most common causes of instability. In some patients, the underlying cause increases the risk of instability. These reasons can be listed as follows:

- Knee with advanced deformity.
- Regional muscle weakness.
- Neuromuscular disease.
- Internal side ligament or posterior cruciate ligament failure.
- Obesity and rheumatoid arthritis.
- Charcot arthropathy

4.1.2 Treatment

It is necessary for a stable knee to obtain a rectangular gap in both flexion and extension after bone incisions and soft tissue release in TKA. If the cavity is larger than the prosthesis, the term symmetrical discrepancy is used. The reason for this instability is that the distal femoral incision or the tibial incision is more than necessary [85].

If the tibial incision is excessive, both extension and flexion will be loose. If this condition is noticed intraoperatively, it is thought that the problem is solved with a thicker insert, but in fact, both the patellofemoral joint problems can arise as the joint line will go down more inferiorly and the early relaxation and fixation problems can arise because the tibial component will sit on the narrower surface.

If the distal femoral incision is excessive, there will be looseness in the extension range. The use of a thick insert during surgery will improve the looseness of the

extension, but there will be tightness in flexion [104]. In addition, as the joint line will increase, both the effective distance of collateral ligament will decrease, and patellofemoral joint problems will occur. Therefore, if the distal femoral incision is excessive, the use of distal femoral augment should be preferred instead of the use of a thick insert [105].

Asymmetric mismatches occur when the joint space is trapezoidal rather than rectangular. It occurs mostly during surgery after excessive loosening of the soft tissue and is most commonly seen in extension. In this case, the transition to the restrictive prosthesis should be considered [106].

4.2 Joint stiffness

One of the reasons that greatly affect patient satisfaction after TKA operations is the amount of joint range of motion. To achieve good results, a flexion range of at least 90° is required. Sixty-five degrees of flexion is required during walking; 106° of flexion is required when sitting on a chair and tying shoes. Postoperative limited and painful joint movements significantly reduce patient comfort. A flexion range of less than 90° for 6 weeks after TKA surgery is defined as a rigid knee [107].

4.2.1 Risk factors and causes

Hip osteoarthritis, heterotopic ossification, and reflex symptomatic dystrophy can be considered as independent factors. Inadequate posterior femoral incision and inadequate medial collateral ligament releasing of the knee with severe varus deformity may be among the causes for a rigid knee due to surgical technique [108, 109]. In one study, it was observed that joint stiffness occurred more frequently than unilateral knee arthroplasty in patients who underwent bilateral total knee arthroplasty in the same session, and manipulation was required under anesthesia [110].

Excessive tight extension and flexion gap, tight PCL, malrotation of components, and inadequate tibial slope angle may lead to joint stiffness [108].

One of the most important indicators of joint stiffness is the extremely limited range of motion in the knee before surgery [109]. The range of motion obtained within the surgery should be considered in the determination of joint stiffness. A sudden loss of motion should suggest a mechanical problem, loosening, and infection.

Arthrofibrosis is the most treatment-resistant cause of joint stiffness. It develops due to excessive increase of fibrous tissue in the joint [108].

4.2.2 Treatment

The strongest determinant of postoperative flexion movements is the degree of preoperative flexion. Other than that, age, preoperative diagnosis, and severity of deformity are other factors [111].

The efficacy of conservative treatment is limited in joint stiffness after TKA. Aggressive range of motion improvement of 3.1° was observed with aggressive physical therapy for almost 1 year [112]. It has been shown that the use of continuous passive motion device (CPM) in the early postoperative period reduces bleeding and is beneficial in preventing joint stiffness by reducing the formation of fibrosis [113].

Although there is no consensus in the literature, manipulation under anesthesia should be performed in cases where knee flexion is below 90° between 2 weeks and 3 months. Revision rates are lower in patients with early manipulation [114]. Manipulation is performed under general anesthesia using a muscle relaxant until the knee and hip reach at least 90°. After this procedure, an average gain of 30–47° was reported [115].

If the joint movement limitation continues despite these methods, surgical procedures are performed. These are arthroscopic release, open release and limited revision knee arthroplasty, and total revision knee arthroplasty [116].

4.3 Periprosthetic joint infections

Deep infection after TKA is the most common cause of revision. Systemic complications such as septicemia and cardiopulmonary insufficiency may also occur in patients with periprosthetic infection [117]. As a result, it increased mortality rates. Nowadays, the incidence of deep infection after TKA varies between 0.4 and 2% [74]. Factors that pave the way for infection in the postoperative period include the presence of rheumatoid arthritis, diabetes, hemophilia, malignancy, HIV, obesity, smoking, intravenous drug addiction, knee septic arthritis and osteomyelitis, prolonged surgical time, malnutrition, steroid use, and prolonged skin problems.

Antibiotic prophylaxis is the most effective method to prevent infection [118]. Prophylaxis should be administered 30–60 minutes before skin incision [119]. It has been shown that short postoperative antibiotherapy is more beneficial than the longer one [120].

Fewer people entering the operating room, using drapes to prevent superficial contamination, providing laminar air flow, effective sterilization of surgical instruments, and keeping the surgical time 150 minutes below are also necessary to prevent infection [121].

Risk groups of patients should be identified before the operation, and a separate planning should be made for each patient according to comorbid diseases. Antibiotic cement has been shown to reduce the infection rate in patients at risk [122]. However, it has been reported that the use of antibiotic cement in the patient group with no risk may cause premature loosening [123].

The most common organisms produced after infected knee arthroplasties are *Staphylococcus aureus*, coagulase negative *Staphylococcus*, and *Streptococcus* bacteria [124]. However, many microorganisms can also be active. Variations have occurred in microorganisms due to the unnecessary antibiotics used recently, and this has led to the development of resistance. Of these microorganisms, the most common isolates are methicillin-resistant *Staphylococcus aureus* (MRSA) and many antibiotics [125]. Fungal infections are not common, but the most common causative agents in these isolated are *Candida* species [126].

Bacteria that cause prosthetic infection form a biofilm layer on the implant. This biofilm layer increases the virulence of the agent. In addition, it forms resistance to treatment because of its limitation on antibiotic permeability. The best antibiotic to cross the biofilm layer is rifampicin [127]. There are studies suggesting the addition of rifampicin to antibiotic treatment specific for the reproductive bacteria [127, 128].

4.3.1 Diagnosis

Detailed anamnesis and detailed physical examination should be performed in the diagnosis of periprosthetic infection. In addition, the presence of a progressive radiolucent area around the prosthesis with direct radiographs, osteopenia, or osteolysis extending to the subchondral bone and the formation of new bone in the periosteal area can be evaluated in favor of infection [129]. The pain caused by rest is unique. However, increasing severity of pain and prolonged drainage at the wound site can also be evaluated in favor of infection. Arthrocentesis is then performed. In the case of active isolation, the necessary treatment is started. Empirical antibiotic therapy should be avoided. Wait until the agent is isolated.

Because empirical antibiotherapy will suppress a possible infection and may cause deep infection due to delayed diagnosis of prosthesis infection that may be saved by debridement and may require removal of the prosthesis [130].

CRP and sedimentation values should be evaluated in diagnosis. However, it should be remembered that CRP returns to its previous level after 14–21 days postoperatively [131]. Alpha defensin, lactoferrin, ELA-2, BPI, procalcitonin, and synovial CRP values are other parameters that can be used in diagnosis [132].

Current consensus has been reached in the diagnosis of periprosthetic infection [133]. Accordingly:

- Major criteria
 - Generation of the same agent in two positive cultures.
 - Presence of sinus mouth associated with prosthesis. In the presence of one of them, the diagnosis is established [133].
- Minor criteria
 - Calculated weights of high serum CRP (>1 mg/dL), D-dimer (>860 ng/mL), and erythrocyte sedimentation rate (>30 mm/h) are also 2, 2, and 1 points, respectively.
 - High synovial fluid white cell count (>3000 cells/ μ L), alpha defensin (signal cutoff ratio > 1), leukocyte esterase (++) , polymorphonuclear percentage (>80%), and synovial CRP (>6.9 mg/L) were arranged as 3, 3, 3, 2, and 1 points, respectively.

Patients with a total score equal to or greater than 6 were considered infected.

4.3.2 Treatment

The goal of infection treatment in total knee arthroplasty is eradication of the infection, pain relief, and maintenance of limb function. Treatment options are antibiotic pressure, debridement, single- or double-stage revision, arthrodesis, resection arthroplasty, and amputation. Revision surgery also has single-stage or double-stage revision options [134–136].

4.4 Periprosthetic fractures

Periprosthetic fractures around the knee are fractures that occur during or after surgery within 15 cm of the knee joint or within 5 cm of the intramedullary part of the prosthesis, if any [137]. The incidence of these fractures after TKA is 0.3–2.5% for femur and 0.4–1% for tibia [138, 139].

The main risk factor related to the patient is the age of the patient. This risk is due to an increased risk of falling due to the patient's age and osteoporosis associated with age [140]. Corticosteroid use, diseases that may increase the risk of falling with rheumatoid arthritis (epilepsy, Parkinson's, cerebellar ataxia, myasthenia gravis) can be counted as other patient-related risk factors [141].

Intraoperative diaphyseal femoral fractures may occur due to incorrect placement of the intramedullary guide and osteopenia [142]. Unsuitable bone incisions, aggressive impaction of the ligamentous posterior stabilized femoral component, and eccentric placement of trial components are also risk factors for femoral

fracture. It has been shown to increase the frequency of periprosthetic fractures due to increased resistance in flexion and rotation movements in anterior femoral notching [143]. The possibility of periprosthetic fracture is increased in revision TKA cases [144]. Periprosthetic fractures are more common due to the rotational forces of restrictive prosthesis using shear forces in the prosthesis [141].

Due to the stronger structure of the tibia, fracture development is rare.

For femoral periprosthetic fractures, there is a classification that questions fracture displacement and component fixation.

- Type 1 describes fractures with non-displaced and stable components.
- Type 2 refers to component stable fractures with displacement of more than 5 mm or angulation of more than 5°.
- Type 3 indicates loose fractures [145].

4.4.1 Treatment

4.4.1.1 Femoral fractures during surgery

The femur fractures vertically more than the metaphyseal region. A stable periosteum prevents displacement. It is followed conservatively without any additional intervention. For fractures penetrating the femoral cortex, whether or not a bone graft is used, the penetration level should be treated with a stem prosthesis that is at least twice the diameter of the femoral canal [146].

4.4.1.2 Postoperative femoral fractures

When non-displaced fractures and stable prosthesis occur after TKA, conservative treatment may be preferred. Four to six weeks of non-weight procedure, long leg plaster, or hinged orthosis is followed.

Displaced and unreducible supracondylar fractures are almost always treated surgically in the presence of adequate bone stock (**Figure 1**) [147].



Figure 1.
Supracondylar periprosthetic femur fracture treated with open reduction and internal fixation.

Locked compression plates are preferred for knee periprosthetic fractures [144]. Prosthetic revision should be performed in fractures that cause prosthesis loosening and malposition. In these cases, stented prosthesis of sufficient length should be placed to obtain a stable fixation of the intact bone [138]. Knee replacement revision after periprosthetic fractures is often associated with the loss of range of motion (ROM) [148].

4.4.1.3 Tibia fractures

The majority of tibial periprosthetic fractures during surgery involve the plateau region and are generally non-displaced [146, 149]. If prosthetic loosening is present, revision surgery using a stem component long enough to cross the fracture line is required [150].

Postoperative tibial fractures can be examined in four groups. In type 1 fracture, revision is recommended because tibial component will be in varus alignment. The medial defect should be closed with bone graft or metal support [150]. Type 2 fractures are treated with nonsurgical treatment if the component is stable and there is minimal displacement [149]. Displaced type 2 fractures are treated with internal fixation. If the component is unstable, it must be revised using a long tibial stem to cross the fracture line [149]. Internal fixation should be performed for type 3 and 4 fractures [145].

4.5 Aseptic loosening

The deterioration of the relationship between prosthesis and bone is defined as loosening. The loosening may be between the prosthetic cement and the cement bone. Loosening is inevitable in long-term prostheses. It is useful to distinguish the concepts of osteolysis and loosening. Without prosthesis osteolysis, loosening of the cement may occur. The mechanisms that cause loosening are micromotion, component collapse, and periprosthetic osteolysis [151].

Overuse and osteopenia are the causes of patient-related loosening. Implant design may also be the cause of loosening. According to this, loosening is more likely in cementless prosthesis and constrained prosthesis. One of the most important causes of aseptic loosening is malalignment. It has been shown that a 4 mm medial collapse of the tibial component and varus deformity of more than 2° increases the likelihood of loosening [152]. In the early period, a radiolucent line is seen between the component and bone on radiography, and a collapse occurs as the loosening progresses. Loosening is more common around the tibial component [152]. In the presence of loosening around the whole component, septic loosening should be considered, and differential diagnosis should be performed.

In the case of loosening, the treatment is decided according to symptoms and progress. If pain is associated with instability and there are X-ray findings, early revision surgery is recommended for bone stock preservation.

4.6 Osteolysis

Osteolysis usually occurs due to inflammatory reactions caused by worn polyethylene particles or in the presence of infection. Metal particles can also cause osteolysis. Titanium causes more osteolysis than cobalt and chromium. Giant cells that develop against abrasive particles act by forming a membrane [153]. Particle size is important for this mechanism. The particle sizes range from 1 to 100 micrometers under the electron microscope. Large parts do not cause osteolysis [153]. There is no osteolysis if the parts are not spread to the cancellous bone, so osteolysis is not seen when

the cancellous bone is properly covered with cement [154]. On the other hand, the incidence of osteolysis increases when pres-fit prosthesis is applied; screw fixation without cement is used or cement breaks [155]. Osteolysis is closely related to prosthetic design. Osteolysis usually occurs after 2 years of TKA. Occurrence is rare before 2 years [156]. Osteolysis is mostly seen in the tibia [157]. Diagnosis includes pain, joint effusion, and synovitis due to joint instability. Focal bone destruction may be seen on radiolucent line and X-ray. It can be seen that there is no continuity of trabeculae and bone cortex in cancellous bone. Therefore, control X-rays are very important in patient follow-up and must be compared with old radiographs in controls. CT and MRI can be used for osteolysis that cannot be detected on direct radiography [158].

If the lesion is small in treatment and the prosthesis is stable, observation is sufficient. Bisphosphonate and calcium supplementation can be initiated [159]. If the prosthesis is unstable, two options can be applied. The first one is debridement, polyethylene replacement, and curettage, followed by impaction of the defect with bone graft. The second is revision [159].

4.7 Patellofemoral joint problems

Patellofemoral joint problems after TKA generally cause anterior knee pain. Patients' ability to tolerate this pain rarely causes patellofemoral joint problems to be revised [160]. It should be kept in mind that not only patellar component-related procedures but also procedures involving the tibiofemoral joint may cause this problem. Even in revision surgery due to a problem of patellofemoral origin, it is often caused by a component in the tibia and femur [161]. In a study, patella and malrotation were among the eight most common causes of failed TKA [162].

Advanced valgus alignment, previous high tibial osteotomy, or tuberositas tibia osteotomy increases the rate of patellofemoral joint problems in TKA [160].

There are many points to be considered in the surgical technique to prevent patellofemoral joint problems. These are [163]:

- *Component placement:* If the femoral component is placed medially, anteriorly, or flexed, or if there is internal rotation and if the component is excessive in size, patellofemoral problems may occur finally. Likewise, the medialization and internal rotation of the tibial component increases the risk.
- *Surgical approach type:* Midvastus and subvastus interventions that protect the extensor mechanism more can reduce PF joint problems.
- *Lateral release:* The need for lateral retinacular release increases PF joint problems.
- *Patella resection amount:* When patellar component is used, resection of the patella with anterior–posterior reduction of 12 mm increases the risk of PF joint problems [164, 165].

Patellar surface replacement is controversial today. However, in a recent study, it was found that anterior knee pain was less common in patients who underwent patellar surface change than those who did not. In the same study, the causes of PF joint revision were more common in patients without patella surface changes [166].

The results in patients with patellar articular surface alteration due to persistent anterior knee pain after TKA are not as successful as those with surface replacement during primary TKA [167]. In the treatment of anterior knee pain after TKA, mechanical causes should be investigated after the exclusion of an underlying infection.

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References

- [1] Healy WL, Della Valle CJ, Iorio R, Berend KR, Cushner FD, Dalury DF, et al. Complications of total knee arthroplasty: Standardized list and definitions of the knee society. *Clinical Orthopaedics and Related Research*. 2013;**471**:215-220. DOI: 10.1007/s11999-012-2489-y
- [2] Dalury DF, Pomeroy DL, Gorab RS, Adams MJ. Why are total knee arthroplasties being revised? *The Journal of Arthroplasty*. 2013;**28**:120-121. DOI: 10.1016/j.arth.2013.04.051
- [3] Langkamer V. Local vascular complications after knee replacement: A review with illustrative case reports. *The Knee*. 2001;**8**:259-264. DOI: 10.1016/S0968-0160(01)00103-X
- [4] Abularrage CJ, Weiswasser JM, DeZee KJ, Slidell MB, Henderson WG, Sidawy AN. Predictors of lower extremity arterial injury after total knee or total hip arthroplasty. *Journal of Vascular Surgery*. 2008;**47**:803-807. DOI: 10.1016/J.JVS.2007.11.067
- [5] Shoenfeld NA, Stuchin SA, Pearl R, Haveson S. The management of vascular injuries associated with total hip arthroplasty. *Journal of Vascular Surgery*. 1990;**11**:549-555. DOI: 10.1016/0741-5214(90)90301-P
- [6] Calligaro KD, Dougherty MJ, Ryan S, Booth RE. Acute arterial complications associated with total hip and knee arthroplasty. *Journal of Vascular Surgery*. 2003;**38**:1170-1175. DOI: 10.1016/S0741-5214(03)00918-2
- [7] Smith DE, McGraw RW, Taylor DC, Masri BA. Arterial complications and total knee arthroplasty. *The Journal of the American Academy of Orthopaedic Surgeons*. n.d.;**9**:253-257
- [8] Rand JA. Vascular complications of total knee arthroplasty. Report of three cases. *Journal of Arthroplasty*. 1987;**2**:89-93
- [9] Inomata K, Sekiya I, Otabe K, Nakamura T, Horie M, Koga H, et al. Acute arterial occlusion after total knee arthroplasty: A case report. *Clinical Case Reports*. 2017;**5**:1376-1380. DOI: 10.1002/ccr3.1075
- [10] Saleh KJ, Hoeffel DP, Kassim RA, Burstein G. Complications after revision total knee arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 2003;**85-A**(Suppl 1):S71-S74. DOI: 10.2106/00004623-200300001-00013
- [11] Ninomiya JT, Dean JC, Goldberg VM. Injury to the popliteal artery and its anatomic location in total knee arthroplasty. *The Journal of Arthroplasty*. 1999;**14**:803-809
- [12] Rubash HE, Berger RA, Britton CA, Nettrour WS, Seel MJ. Avoiding neurologic and vascular injuries with screw fixation of the tibial component in total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1993:56-63
- [13] Rama KRBS. Timing of tourniquet release in knee arthroplasty: Meta-analysis of randomized, controlled trials. *The Journal of Bone and Joint Surgery*. 2007;**89**:699. DOI: 10.2106/JBJS.F.00497
- [14] Holmberg A, Milbrink J, Berqqvist D. Arterial complications after knee arthroplasty: 4 cases and a review of the literature. *Acta Orthopaedica Scandinavica*. 01 Feb 1996;**67**(1):75-78. DOI: 10.3109/17453679608995616
- [15] Ibrahim M, Booth RE, Clark TWI. Embolization of traumatic pseudoaneurysms after total knee arthroplasty. *The Journal of Arthroplasty*. 2004;**19**:123-128

- [16] Kapetanos GA, Papavasiliou KA, Makris V, Nikolaidis AP, Kirkos JM, Symeonides PP. Recurrent spontaneous hemarthrosis after total knee arthroplasty successfully treated with synoviorthesis. *The Journal of Arthroplasty*. 2008;**23**(6):931-933. DOI: 10.1016/j.arth.2007.07.012
- [17] Knutson K, Leden I, Sturfelt G, Rosén I, Lidgren L. Nerve palsy after knee arthroplasty in patients with rheumatoid arthritis. *Scandinavian Journal of Rheumatology*. 1983;**12**:201-205
- [18] Rose HA, Hood RW, Otis JC, Ranawat CS, Insall JN. Peroneal-nerve palsy following total knee arthroplasty. A review of the hospital for special surgery experience. *The Journal of Bone and Joint Surgery. American Volume*. 1982;**64**:347-351
- [19] Horlocker TT, Hebl JR, Gali B, Jankowski CJ, Burkle CM, Berry DJ, et al. Anesthetic, patient, and surgical risk factors for neurologic complications after prolonged total tourniquet time during total knee arthroplasty. *Anesthesia and Analgesia*. 2006;**102**:950-955. DOI: 10.1213/01.ane.0000194875.05587.7e
- [20] Black R, Green C, Sochart D. Postoperative numbness of the knee following total knee arthroplasty. *Annals of the Royal College of Surgeons of England*. 2013:565-568. DOI: 10.1308/003588413X13629960049009
- [21] Idusuyi OB, Morrey BF. Peroneal nerve palsy after total knee arthroplasty. Assessment of predisposing and prognostic factors. *The Journal of Bone and Joint Surgery. American Volume*. 1996;**78**:177-184. DOI: 10.2106/00004623-199602000-00003.
- [22] Brull R, McCartney CJL, Chan VWS, El-Beheiry H. Neurological complications after regional anesthesia: Contemporary estimates of risk. *Anesthesia and Analgesia*. 2007;**104**:965-974. DOI: 10.1213/01.ane.0000258740.17193.ec
- [23] Hopton BP, Tommichan MC, Howell FR. Reducing lateral skin flap numbness after total knee arthroplasty. *The Knee*. 2004;**11**:289-291. DOI: 10.1016/j.knee.2003.09.004
- [24] Nam D, Abdel MP, Cross MB, LaMont LE, Reinhardt KR, McArthur BA, et al. The management of extensor mechanism complications in total knee arthroplasty: AAOS exhibit selection. *The Journal of Bone and Joint Surgery*. 2014;**96**(6):e47. DOI: 10.2106/JBJS.M.00949
- [25] Parker DA, Dunbar MJ, Rorabeck CH. Extensor mechanism failure associated with total knee arthroplasty: Prevention and management. *The Journal of the American Academy of Orthopaedic Surgeons*. 2003;**11**:238-247
- [26] Rand J, Morrey B, Bryan R. Patellar tendon rupture after total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1989;**246**:233-238
- [27] Schoderbek RJ, Brown TE, Mulhall KJ, Mounasamy V, Iorio R, Krackow KA, et al. Extensor mechanism disruption after total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 2006;**446**:176-185. DOI: 10.1097/01.blo.0000218726.06473.26
- [28] Putman S, Boureau F, Girard J, Migaud H, Pasquier G. Patellar complications after total knee arthroplasty. *Orthopaedics and Traumatology, Surgery and Research*. 2019;**105**:S43-S51. DOI: 10.1016/j.otsr.2018.04.028
- [29] Browne JA, Hanssen AD. Reconstruction of patellar tendon disruption after total knee arthroplasty: Results of a new technique utilizing synthetic mesh. *The Journal of Bone*

and Joint Surgery. American Volume. 2011;**93**:1137-1143. DOI: 10.2106/JBJS.J.01036

[30] Ayas MS, Gül O, Okutan AE, Turhan AU. Extensor mechanism reconstruction with peroneus longus tendon autograft for neglected patellar fracture, report of 2 cases. *Journal of Clinical Orthopaedics and Trauma*. 2019;**10**:S226-S230. DOI: 10.1016/j.jcot.2019.05.020

[31] Dobbs RE, Hanssen AD, Lewallen DG, Pagnano MW. Quadriceps tendon rupture after total knee arthroplasty: Prevalence, complications, and outcomes. *The Journal of Bone and Joint Surgery*. 2005;**87**(1):37-45. DOI: 10.2106/JBJS.D.01910

[32] Hozack WJ, Goll SR, Lotke PA, Rothman RH, Booth RE. The treatment of patellar fractures after total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1988;**236**:123-127

[33] Ortiguera CJ, Berry DJ. Patellar fracture after total knee arthroplasty. *Journal of Bone and Joint Surgery*. 2002;**84**(4):532-540

[34] Chalidis BE, Tsiridis E, Tragas AA, Stavrou Z, Giannoudis PV. Management of periprosthetic patellar fractures. A systematic review of literature. *Injury*. 2007. DOI: 10.1016/j.injury.2007.02.054

[35] Leopold MSS, McStay C, Klafeta K, Jacobs JJ, Berger RA, Rosenberg AG. Primary repair of intraoperative disruption of the medial collateral ligament during total knee arthroplasty. *The Journal of Bone and Joint Surgery*. 2001. DOI: 10.2106/00004623-200101000-00012

[36] Healy WL, Della Valle CJ, Iorio R, Berend KR, Cushner FD, Dalury DF, et al. Complications of total knee arthroplasty: Standardized list and definitions of the knee society knee.

Clinical Orthopaedics and Related Research. 2013. DOI: 10.1007/s11999-012-2489-y

[37] Lee GC, Lotke PA. Management of intraoperative medial collateral ligament injury during TKA. *Clinical Orthopaedics and Related Research*. 2011. DOI: 10.1007/s11999-010-1502-6

[38] Wang X, Liu H, Cao P, Liu C, Dong Z, Qi J, et al. Clinical outcomes of medial collateral ligament injury in total knee arthroplasty. *Medicine (United States)*. 2017. DOI: 10.1097/MD.00000000000007617

[39] Kenneth Della Torre P, Stephens A, Lii H. Management of medial collateral ligament injury during primary total knee arthroplasty: A systematic review. *Reconstructive Review*. 2014;**4**:17-23. DOI: 10.15438/rrv4i2.69

[40] Adravanti P, Dini F, Calafiore G, Rosa MA. Medial collateral ligament reconstruction during TKA: A new approach and surgical technique. *Joints*. 2015;**3**(04):215-217. DOI: 10.11138/jts/2015.3.4.215

[41] Whiteside LA. Correction of ligament and bone defects in total arthroplasty of the severely valgus knee. *Clinical Orthopaedics and Related Research*. 1993;**288**:234-245

[42] Dimitris K, Taylor BC, Steensen RN. Excursion of oscillating saw blades in total knee arthroplasty. *The Journal of Arthroplasty*. 2010;**25**(1):158-160. DOI: 10.1016/j.arth.2008.09.021

[43] Zheng X, Li T, Wang J, Dong J, Gao S. Medial collateral ligament reconstruction using bone-patellar tendon-bone allograft for chronic medial knee instability combined with multi-ligament injuries: A new technique. *Journal of Orthopaedic Surgery and Research*. 2016;**11**:85. DOI: 10.1186/s13018-016-0416-8

- [44] Heller K-D. Intraoperative damage to the medial collateral ligament (MCL)—what is to be done? *Zeitschrift für Orthopädie und Unfallchirurgie*. 2013;**151**:580-584. DOI: 10.1055/s-0033-1350932.
- [45] Koo MH, Choi CH. Conservative treatment for the intraoperative detachment of medial collateral ligament from the tibial attachment site during primary total knee arthroplasty. *The Journal of Arthroplasty*. 2009;**24**(8):1249-1253. DOI: 10.1016/j.arth.2009.06.007
- [46] Siqueira MBP, Haller K, Mulder A, Goldblum AS, Klika AK, Barsoum WK. Outcomes of medial collateral ligament injuries during total knee arthroplasty. *The Journal of Knee Surgery*. 2016;**29**:68-73. DOI: 10.1055/s-0034-1394166
- [47] Kotzé A, Carter LA, Scally AJ. Effect of a patient blood management programme on preoperative anaemia, transfusion rate, and outcome after primary hip or knee arthroplasty: A quality improvement cycle. *British Journal of Anaesthesia*. 2012;**108**(6):943-952. DOI: 10.1093/bja/aes135
- [48] Raut VV, Stone MH, Wroblewski BM. Reduction of postoperative blood loss after press-fit condylar knee arthroplasty with use of a femoral intramedullary plug. *The Journal of Bone and Joint Surgery. American Volume*. 1993;**75**:1356-1357. DOI: 10.2106/00004623-199309000-00010
- [49] Juelsgaard P, Larsen UT, Sørensen JV, Madsen F, Søballe K. Hypotensive epidural anesthesia in total knee replacement without tourniquet: Reduced blood loss and transfusion. *Regional Anesthesia and Pain Medicine*. n.d.;**26**:105-110. DOI: 10.1053/rapm.2001.21094
- [50] Gibbons CE, Solan MC, Ricketts DM, Patterson M. Cryotherapy compared with Robert Jones bandage after total knee replacement: A prospective randomized trial. *International Orthopaedics*. 2001;**25**:250-252. DOI: 10.1007/s002640100227
- [51] Wang GJ, Hungerford DS, Savory CG, Rosenberg AG, Mont MA, Burks SG, et al. Use of fibrin sealant to reduce bloody drainage and hemoglobin loss after total knee arthroplasty: A brief note on a randomized prospective trial. *The Journal of Bone and Joint Surgery. American Volume*. 2001;**83**:1503-1505. DOI: 10.2106/00004623-200110000-00007
- [52] Prasad N, Padmanabhan V, Mullaji A. Comparison between two methods of drain clamping after total knee arthroplasty. *Archives of Orthopaedic and Trauma Surgery*. 2005;**125**:381-384. DOI: 10.1007/s00402-005-0813-7
- [53] Roy N, Smith M, Anwar M, Elsworth C. Delayed release of drain in total knee replacement reduces blood loss. A prospective randomised study. *Acta Orthopaedica Belgica*. 2006;**72**:34-38
- [54] Ryu J, Sakamoto A, Honda T, Saito S. The postoperative drain-clamping method for hemostasis in total knee arthroplasty. Reducing postoperative bleeding in total knee arthroplasty. *Bulletin/Hospital for Joint Diseases*. 1997;**56**:251-254
- [55] Charoencholvanich K, Siriwattanasakul P. Tranexamic acid reduces blood loss and blood transfusion after TKA: A prospective randomized controlled trial. *Clinical Orthopaedics and Related Research*. 2011;**469**:2874-2880. DOI: 10.1007/s11999-011-1874-2
- [56] Janssens M, Joris J, David JL, Lemaire R, Lamy M. High-dose

aprotinin reduces blood loss in patients undergoing total hip replacement surgery. *Anesthesiology*. 1994;**80**:23-29. DOI: 10.1097/00000542-199401000-00007

[57] Dubber AH, McNicol GP, Douglas AS. Amino-methyl-cyclohexane-carboxylic acid: AMCHA; a new patent inhibitor of the fibrinolysis. *British Journal of Haematology*. 1965

[58] Sano M, Hakusui H, Kojima C, Akimoto T. Absorption and excretion of tranexamic acid following intravenous, intramuscular and oral administrations in healthy volunteers. *Rinsho Yakuri/Japanese Journal of Clinical Pharmacology and Therapeutics*. 1976. DOI: 10.3999/jscpt.7.375

[59] Molloy DO, Archbold HAP, Ogonda L, McConway J, Wilson RK, Beverland DE. Comparison of topical fibrin spray and tranexamic acid on blood loss after total knee replacement. *Journal of Bone and Joint Surgery. British Volume (London)*. 2007. DOI: 10.1302/0301-620x.89b3.17565

[60] Veien M, Sørensen JV, Madsen F, Juelsgaard P. Tranexamic acid given intraoperatively reduces blood loss after total knee replacement: A randomized, controlled study. *Acta Anaesthesiologica Scandinavica*. 2002;**46**:1206-1211

[61] Orpen NM, Little C, Walker G, Crawford EJP. Tranexamic acid reduces early post-operative blood loss after total knee arthroplasty: A prospective randomised controlled trial of 29 patients. *The Knee*. 2006;**13**:106-110. DOI: 10.1016/j.knee.2005.11.001

[62] Jansen AJ, Andreica S, Claeys M, D'Haese J, Camu F, Jochmans K. Use of tranexamic acid for an effective blood conservation strategy after total knee arthroplasty. *British Journal of Anaesthesia*. 1999;**83**(4):596-601. DOI: 10.1093/bja/83.4.596

[63] Feng B, Lin J, Jin J, Qian W-W, Wang W, Weng X-S. Thirty-day postoperative complications following primary total knee arthroplasty: A retrospective study of incidence and risk factors at a single Center in China. *Chinese Medical Journal*. 2017;**130**:2551-2556. DOI: 10.4103/0366-6999.213071

[64] Parvizi J, Mui A, Purtill JJ, Sharkey PF, Hozack WJ, Rothman RH. Total joint arthroplasty: When do fatal or near-fatal complications occur? *The Journal of Bone and Joint Surgery. American Volume*. 2007;**89**:27-32. DOI: 10.2106/JBJS.E.01443

[65] Simons MJ, Amin NH, Scuderi GR. Acute wound complications after total knee arthroplasty: Prevention and management. *The Journal of the American Academy of Orthopaedic Surgeons*. 2017;**25**:547-555. DOI: 10.5435/JAAOS-D-15-00402

[66] Galat DD, McGovern SC, Larson DR, Harrington JR, Hanssen AD, Clarke HD. Surgical treatment of early wound complications following primary total knee arthroplasty. *The Journal of Bone and Joint Surgery*. 2009;**91**(1):48-54. DOI: 10.2106/JBJS.G.01371

[67] Stryker LS, Abdel MP, Morrey ME, Morrow MM, Kor DJ, Morrey BF. Elevated postoperative blood glucose and preoperative hemoglobin A1C are associated with increased wound complications following total joint arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 2013;**95**:808-814, S1-2. DOI: 10.2106/JBJS.L.00494

[68] Simons MJ, Nirav HA, Giles RS. Acute wound complications after total knee arthroplasty: Prevention and management. *Journal of the American Academy of Orthopaedic Surgeons*; 2017;**25**(8):547-555

[69] Osei DA, Rebehn KA, Boyer MI. Soft-tissue defects after

total knee arthroplasty: Management and reconstruction. *The Journal of the American Academy of Orthopaedic Surgeons*. 2016;**24**:769-779. DOI: 10.5435/JAAOS-D-15-00241

[70] Ries MD. Skin necrosis after total knee arthroplasty. *The Journal of Arthroplasty*. 2002;**17**(4):74-77. DOI: 10.1054/arth.2002.32452

[71] Vince KG, Abdeen A. Total knee replacement after valgus tibial osteotomy. Technical problems. *Clinical Orthopaedics and Related Research*. 1992;**78**(7):438-448

[72] Gaine WJ, Ramamohan NA, Hussein NA, Hullin MG, McCreath SW. Wound infection in hip and knee arthroplasty. *Journal of Bone and Joint Surgery. British Volume (London)*. 2000;**82**:561-565

[73] Wu C-T, Chen I-L, Wang J-W, Ko J-Y, Wang C-J, Lee C-H. Surgical site infection after total knee arthroplasty: Risk factors in patients with timely administration of systemic prophylactic antibiotics. *The Journal of Arthroplasty*. 2016;**31**:1568-1573. DOI: 10.1016/j.arth.2016.01.017

[74] Blom AW, Brown J, Taylor AH, Pattison G, Whitehouse S, Bannister GC. Infection after total knee arthroplasty. *Journal of Bone and Joint Surgery. British Volume (London)*. 2004;**86**(5):688-691

[75] Namba RS, Paxton L, Fithian DC, Lou SM. Obesity and perioperative morbidity in total hip and total knee arthroplasty patients. *The Journal of Arthroplasty*. 2005;**20**:46-50. DOI: 10.1016/j.arth.2005.04.023

[76] Bongartz T, Halligan CS, Osmon DR, Reinalda MS, Bamlet WR, Crowson CS, et al. Incidence and risk factors of prosthetic joint infection after total hip or knee replacement in patients with rheumatoid arthritis. *Arthritis Care and*

Research. 2008;**59**(12):1713-1720. DOI: 10.1002/art.24060

[77] Papavasiliou AV, Isaac DL, Marimuthu R, Skyrme A, Armitage A. Infection in knee replacements after previous injection of intra-articular steroid. *Journal of Bone and Joint Surgery. British Volume (London)*. 2006;**88**:321-323. DOI: 10.1302/0301-620X.88B3.17136.

[78] Hijas-Gómez AI, Lucas WC, Checa-García A, Martínez-Martín J, Fahandezh-Saddi H, Gil-de-Miguel Á, et al. Surgical site infection incidence and risk factors in knee arthroplasty: A 9-year prospective cohort study at a university teaching hospital in Spain. *American Journal of Infection Control*. 2018;**46**:1335-1340. DOI: 10.1016/j.ajic.2018.06.010

[79] Saleh K, Olson M, Resig S, Bershadsky B, Kuskowski M, Gioe T, et al. Predictors of wound infection in hip and knee joint replacement: Results from a 20 year surveillance program. *Journal of Orthopaedic Research*. 2002;**20**:506-515. DOI: 10.1016/S0736-0266(01)00153-X

[80] Teo BJX, Yeo W, Chong HC, Tan AHC. Surgical site infection after primary total knee arthroplasty is associated with a longer duration of surgery. *Journal of Orthopaedic Surgery*. 2018;**26**(2). DOI: 10.1177/2309499018785647

[81] Scuderi GR. Avoiding postoperative wound complications in total joint arthroplasty. *The Journal of Arthroplasty*. 2018;**33**:3109-3112. DOI: 10.1016/j.arth.2018.01.025

[82] Löwik CAM, Wagenaar F-C, van der Weegen W, Poolman RW, Nelissen RGHH, Bulstra SK, et al. LEAK study: Design of a nationwide randomised controlled trial to find the best way to treat wound leakage after primary hip and knee arthroplasty. *BMJ*

Open. 2017;7:e018673. DOI: 10.1136/bmjopen-2017-018673

[83] Haleem AA, Berry DJ, Hanssen AD. Mid-term to long-term followup of two-stage reimplantation for infected total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 2004;**428**:35-39. DOI: 10.1097/01.blo.0000147713.64235.73

[84] Özkurt B, Utkan A. Primer total diz artroplastisi sonrası yara yeri sorunları ve yüzeysel enfeksiyon. *TOTBID Dergisi*. 2019;**18**:128-137. DOI: 10.14292/totbid.dergisi.2019.15

[85] Segal JB, Eng J, Tamariz LJ, Bass EB. Review of the evidence on diagnosis of deep venous thrombosis and pulmonary embolism. *Annals of Family Medicine*. 2003;**5**:63-73. DOI: 10.1370/afm.648

[86] Shahi A, Chen AF, Tan TL, Maltenfort MG, Kucukdurmaz F, Parvizi J. The incidence and economic burden of in-hospital venous thromboembolism in the United States. *The Journal of Arthroplasty*. 2017;**32**:1063-1066. DOI: 10.1016/j.arth.2016.10.020

[87] Dorr LD, Gendelman V, Maheshwari AV, Boutary M, Wan Z, Long WT. Multimodal thromboprophylaxis for total hip and knee arthroplasty based on risk assessment. *The Journal of Bone and Joint Surgery. American Volume*. 2007;**89**:2648-2657. DOI: 10.2106/JBJS.F.00235

[88] Ogonda L, Hill J, Doran E, Dennison J, Stevenson M, Beverland D. Aspirin for thromboprophylaxis after primary lower limb arthroplasty: Early thromboembolic events and 90 day mortality in 11,459 patients. *The Bone and Joint Journal*. 2016;**98-B**:341-348. DOI: 10.1302/0301-620X.98B3.36511

[89] Tun NM, Oo TH. Prevention and treatment of venous thromboembolism

with new oral anticoagulants: A practical update for clinicians. *Thrombosis*. 2013;**2013**:10. Article ID: 183616. DOI: 10.1155/2013/183616

[90] Anderson FA, Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A population-based perspective of the hospital incidence and case-fatality rates of deep vein thrombosis and pulmonary embolism. The Worcester DVT Study. *Archives of Internal Medicine*. 1991;**151**:933-938

[91] Fukuda A, Hasegawa M, Kato K, Shi D, Sudo A, Uchida A. Effect of tourniquet application on deep vein thrombosis after total knee arthroplasty. *Archives of Orthopaedic and Trauma Surgery*. 2007;**127**:671-675. DOI: 10.1007/s00402-006-0244-0

[92] Aglietti P, Baldini A, Vena LM, Abbate R, Fedi S, Falciani M. Effect of tourniquet use on activation of coagulation in total knee replacement. *Clinical Orthopaedics and Related Research*. 2000;**371**:169-177. DOI: 10.1097/00003086-200002000-00021

[93] Lieberman JR, Huo MM, Hanway J, Salvati EA, Sculco TP, Sharrock NE. The prevalence of deep venous thrombosis after total hip arthroplasty with hypotensive epidural anesthesia. *The Journal of Bone and Joint Surgery. American Volume*. 1994;**76**:341-348. DOI: 10.2106/00004623-199403000-00004

[94] Ho WK, Hankey GJ, Lee CH, Eikelboom JW. Venous thromboembolism: Diagnosis and management of deep venous thrombosis. *The Medical Journal of Australia*. 2005;**182**:476-481

[95] Geerts W, Heit J, Clagett G, Pineio G, Colwell C, Anderson F, et al. Prevention of venous thromboembolism: 6th ACCP consensus conference on antithrombotic

therapy. *Chest Journal*. 2001. DOI: 10.1378/chest.126.3_suppl.338S

[96] Goodacre S, Sampson F, Thomas S, van Beek E, Sutton A. Systematic review and meta-analysis of the diagnostic accuracy of ultrasonography for deep vein thrombosis. *BMC Medical Imaging*. 2005. DOI: 10.1186/1471-2342-5-6

[97] Wells PS, Anderson DR, Rodger M, Stiell I, Dreyer JF, Barnes D, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: Management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. *Annals of Internal Medicine*. 2001

[98] Pierce TP, Cherian JJ, Jauregui JJ, Elmallah RK, Lieberman JR, Mont MA. A current review of mechanical compression and its role in venous thromboembolic prophylaxis in total knee and total hip arthroplasty. *The Journal of Arthroplasty*. 2015;**30**:2279-2284. DOI: 10.1016/j.arth.2015.05.045

[99] Cafri G, Paxton EW, Chen Y, Cheetham CT, Gould MK, Sluggett J, et al. Comparative effectiveness and safety of drug prophylaxis for prevention of venous thromboembolism after total knee arthroplasty. *The Journal of Arthroplasty*. 2017;**32**:3524-3528.e1. DOI: 10.1016/j.arth.2017.05.042

[100] Bala A, Huddleston JI, Goodman SB, Maloney WJ, Amanatullah DF. Venous thromboembolism prophylaxis after TKA: Aspirin, warfarin, enoxaparin, or factor Xa inhibitors? *Clinical Orthopaedics and Related Research*. 2017;**475**:2205-2213. DOI: 10.1007/s11999-017-5394-6

[101] Gutowski CJ, Zmistowski BM, Lonner JH, Purtill JJ, Parvizi J. Direct costs of aspirin versus warfarin for venous thromboembolism prophylaxis after total knee or hip arthroplasty. *The*

Journal of Arthroplasty. 2015;**30**:36-38. DOI: 10.1016/j.arth.2015.04.048

[102] Dalury DF, Pomeroy DL, Gorab RS, Adams MJ. Why are total knee arthroplasties being revised? *The Journal of Arthroplasty*. 2013;**28**:120-121. DOI: 10.1016/j.arth.2013.04.051

[103] Song SJ, Detch RC, Maloney WJ, Goodman SB, Huddleston JI. Causes of instability after total knee arthroplasty. *The Journal of Arthroplasty*. 2014;**29**:360-364. DOI: 10.1016/j.arth.2013.06.023

[104] Vince K. Mid-flexion instability after total knee arthroplasty woolly thinking or a real concern? *The Bone and Joint Journal*. 2016;**98**(1 SuppleA):84-88

[105] Matsuda S, Ito H. Ligament balancing in total knee arthroplasty - medial stabilizing technique. *The Asia-Pacific Journal of Sports Medicine, Arthroscopy, Rehabilitation and Technology*. 2015;**2**(4):108-113. DOI: 10.1016/j.asmart.2015.07.002

[106] Romero J, Stähelin T, Binkert C, Pfirrmann C, Hodler J, Kessler O. The clinical consequences of flexion gap asymmetry in total knee arthroplasty. *The Journal of Arthroplasty*. 2007;**22**:235-240. DOI: 10.1016/j.arth.2006.04.024

[107] Schiavone Panni A, Cerciello S, Vasso M, Tartarone M. Stiffness in total knee arthroplasty. *Journal of Orthopaedics and Traumatology*. 2009;**10**(3):111-118. DOI: 10.1007/s10195-009-0054-6

[108] Nelson CL, Kim J, Lotke PA. Stiffness after total knee arthroplasty. *JBJS Essential Surgical Techniques*. 2005;**os-87**:264-270. DOI: 10.2106/JBJS.E-00345

[109] Ritter MA, Harty LD, Davis KE, Meding JB, Berend ME. Predicting range

of motion after total knee arthroplasty. Clustering, log-linear regression, and regression tree analysis. *The Journal of Bone and Joint Surgery. American Volume*. 2003;**85**:1278-1285. DOI: 10.2106/00004623-200307000-00014

[110] Meehan JP, Monazzam S, Miles T, Danielsen B, White RH. Postoperative stiffness requiring manipulation under anesthesia is significantly reduced after simultaneous versus staged bilateral total knee arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 2017;**99**:2085-2093. DOI: 10.2106/JBJS.17.00130

[111] Schurman DJ, Parker JN, Ornstein D. Total condylar knee replacement. A study of factors influencing range of motion as late as two years after arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 1985;**67**:1006-1014

[112] Esler CNN, Lock K, Harper WMM, Gregg PJJ. Manipulation of total knee replacements. Is the flexion gained retained? *The Journal of Bone and Joint Surgery. British volume*. 1999;**81**(1):27-29. DOI: 10.1302/0301-620X.81B1.8848

[113] O'Driscoll SW, Giori NJ. Continuous passive motion (CPM): Theory and principles of clinical application. *Journal of Rehabilitation Research and Development*. 2000;**37**(2):179-188

[114] Scranton PE. Management of knee pain and stiffness after total knee arthroplasty. *The Journal of Arthroplasty*. 2001;**16**:428-435. DOI: 10.1054/arth.2001.22250

[115] Fitzsimmons SE, Vazquez EA, Bronson MJ. How to treat the stiff total knee arthroplasty? A systematic review. *Clinical Orthopaedics and Related Research*. 2010;**468**(4):1096-1106. DOI: 10.1007/s11999-010-1230-y

[116] Mont MA, Seyler TM, Marulanda GA, Delanois RE, Bhave A. Surgical treatment and customized rehabilitation for stiff knee arthroplasties. *Clinical Orthopaedics and Related Research*. 2006;**446**:193-200. DOI: 10.1097/01.blo.0000214419.36959.8c

[117] Hebert CK, Williams RE, Levy RS, Barrack RL. Cost of treating an infected total knee replacement. *Clinical Orthopaedics and Related Research*. 1996:140-145. DOI: 10.1097/00003086-199610000-00019

[118] Hill C, Flamant R, Mazas F, Evrard J. Prophylactic cefazolin versus placebo in total hip replacement. Report of a multicentre double-blind randomised trial. *Lancet (London, England)*. 1981;**1**:795-796. DOI: 10.1016/s0140-6736(81)92678-7

[119] Leigh DA, Griggs J, Tighe CM, Powell HD, Church JC, Wise K, et al. Pharmacokinetic study of ceftazidime in bone and serum of patients undergoing hip and knee arthroplasty. *The Journal of Antimicrobial Chemotherapy*. 1985;**16**:637-642. DOI: 10.1093/jac/16.5.637

[120] Mauerhan DR, Nelson CL, Smith DL, Fitzgerald RH, Slama TG, Petty RW, et al. Prophylaxis against infection in total joint arthroplasty. One day of cefuroxime compared with three days of cefazolin. *The Journal of Bone and Joint Surgery. American Volume*. 1994;**76**:39-45. DOI: 10.2106/00004623-199401000-00006

[121] Peersman G, Laskin R, Davis J, Peterson M. Infection in total knee replacement: A retrospective review of 6489 total knee replacements. *Clinical Orthopaedics and Related Research*. 2001:15-23

[122] Jämsen E, Huhtala H, Puolakka T, Moilanen T. Risk factors for infection after knee arthroplasty. A register-based analysis of 43,149 cases. *The Journal of*

Bone and Joint Surgery. American Volume. 2009;**91**:38-47. DOI: 10.2106/JBJS.G.01686

[123] Hanssen AD. Prophylactic use of antibiotic bone cement: An emerging standard--in opposition. *Journal of Arthroplasty*. 2004;**19**:73-77

[124] Wasielewski RC, Barden RM, Rosenberg AG. Results of different surgical procedures on total knee arthroplasty infections. *The Journal of Arthroplasty*. 1996;**11**:931-938

[125] Kilgus DJ, Howe DJ, Strang A. Results of periprosthetic hip and knee infections caused by resistant bacteria. *Clinical Orthopaedics and Related Research*. 2002;**404**:116-124. DOI: 10.1097/00003086-200211000-00021

[126] Phelan DM, Osmon DR, Keating MR, Hanssen AD. Delayed reimplantation arthroplasty for candidal prosthetic joint infection: A report of 4 cases and review of the literature. *Clinical Infectious Diseases*. 2002;**34**:930-938. DOI: 10.1086/339212

[127] Zimmerli W, Widmer AF, Blatter M, Frei R, Ochsner PE. Role of rifampin for treatment of orthopedic implant-related staphylococcal infections: A randomized controlled trial. Foreign-body infection (FBI) study group. *JAMA*. 1998;**279**(19):1537-1541

[128] Arizono T, Oga M, Sugioka Y. Increased resistance of bacteria after adherence to polymethyl methacrylate. An in vitro study. *Acta Orthopaedica Scandinavica*. 1992;**63**:661-664

[129] Morrey BF, Westholm F, Schoifet S, Rand JA, Bryan RS. Long-term results of various treatment options for infected total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1989:120-128

[130] Schoifet SD, Morrey BF. Persistent infection after successful arthrodesis

for infected total knee arthroplasty: A report of two cases. *Journal of Arthroplasty*. 1990;**5**(3):277-279. DOI: 10.1016/S0883-5403(08)80083-6

[131] White J, Kelly M, Dunsmuir R. C-reactive protein level after total hip and total knee replacement. *Journal of Bone and Joint Surgery. British Volume (London)*. 1998;**80**:909-911

[132] Tahta M, Simsek ME, Isik C, Akkaya M, Gursoy S, Bozkurt M. Does inflammatory joint diseases affect the accuracy of infection biomarkers in patients with periprosthetic joint infections? A prospective comparative reliability study. *Journal of Orthopaedic Science*. 2019;**24**:286-289. DOI: 10.1016/j.jos.2018.08.022

[133] Parvizi J, Tan TL, Goswami K, Higuera C, Della Valle C, Chen AF, et al. The 2018 definition of Periprosthetic hip and knee infection: An evidence-based and validated criteria. *The Journal of Arthroplasty*. 2018;**33**:1309-1314.e2. DOI: 10.1016/j.arth.2018.02.078

[134] Shaikh AA, Ha CW, Park YG, Park YB. Two-stage approach to primary TKA in infected arthritic knees using intraoperatively molded articulating cement spacers. *Clinical Orthopaedics and Related Research*. 2014. DOI: 10.1007/s11999-014-3545-6

[135] Juul R, Fabrin J, Poulsen K, Schroder HM. Use of a new knee prosthesis as an articulating spacer in two-stage revision of infected total knee arthroplasty. *The Knee Surgery and Related Research*. 2016. DOI: 10.5792/ksrr.2016.28.3.239

[136] Ha C-W. Treatment of infected total knee arthroplasty. *The Knee Surgery and Related Research*. 2017. DOI: 10.5792/ksrr.17.301

[137] Dennis DA. Periprosthetic fractures following total knee arthroplasty. *Instructional Course Lectures*. 2001;**50**:379-389

- [138] Ricci WM. Periprosthetic femur fractures. *Journal of Orthopaedic Trauma*. 2015. DOI: 10.1097/BOT.0000000000000282
- [139] Haller JM, Kubiak EN, Spiguel A, Gardner MJ, Horwitz DS. Intramedullary nailing of tibial shaft fractures distal to total knee arthroplasty. *Journal of Orthopaedic Trauma*. 2014. DOI: 10.1097/BOT.0000000000000096
- [140] Canton G, Ratti C, Fattori R, Hoxhaj B, Murena L. Periprosthetic knee fractures. A review of epidemiology, risk factors, diagnosis, management and outcome. *Acta Biomedica*. 2017. DOI: 10.23750/abm.v88i2 -S.6522
- [141] Culp RW, Schmidt RG, Hanks G, Mak A, Esterhai JL, Heppenstall RB. Supracondylar fracture of the femur following prosthetic knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1987;222:212-222
- [142] Lombardi AV, Mallory TH, Waterman RA, Eberle RW. Intercondylar distal femoral fracture. An unreported complication of posterior-stabilized total knee arthroplasty. *The Journal of Arthroplasty*. 1995;10:643-650
- [143] Gujarathi N, Putti AB, Abboud RJ, MacLean JGB, Espley AJ, Kellett CF. Risk of periprosthetic fracture after anterior femoral notching: A 9-year follow-up of 200 total knee arthroplasties. *Acta Orthopaedica*. 2009;80(5):553-556. DOI: 10.3109/17453670903350099
- [144] Parvizi J, Jain N, Schmidt AH. Periprosthetic knee fractures. *Journal of Orthopaedic Trauma*. 2008;22:663-671. DOI: 10.1097/BOT.0b013e31816ed989
- [145] Rorabeck CH, Taylor JW. Periprosthetic fractures of the femur complicating total knee arthroplasty. *The Orthopedic Clinics of North America*. 1999;30:265-277
- [146] Engh GA, Ammeen DJ. Periprosthetic fractures adjacent to total knee implants: Treatment and clinical results. *Instructional Course Lectures*. 1998;47:437-448
- [147] Merkel KD, Johnson EW. Supracondylar fracture of the femur after total knee arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 1986;68:29-43
- [148] Mortazavi SMJ, Kurd MF, Bender B, Post Z, Parvizi J, Purtill JJ. Distal femoral arthroplasty for the treatment of periprosthetic fractures after total knee arthroplasty. *The Journal of Arthroplasty*. 2010;25:775-780. DOI: 10.1016/j.arth.2009.05.024
- [149] Felix NA, Stuart MJ, Hanssen AD. Periprosthetic fractures of the tibia associated with total knee arthroplasty. *Clinical Orthopaedics and Related Research*. 1997;345:113-124
- [150] Rand JA, Coventry MB. Stress fractures after total knee arthroplasty. *The Journal of Bone and Joint Surgery. American Volume*. 1980;62:226-233
- [151] Işık C, Emre F, Ertaş SE. Aseptik gevşeme. *TOTBİD Dergisi*. 2019;18:163-169. DOI: 10.14292/totbid.dergisi.2019.19
- [152] Lee B-S, Cho H-I, Bin S-I, Kim J-M, Jo B-K. Femoral component varus malposition is associated with tibial aseptic loosening after TKA. *Clinical Orthopaedics and Related Research*. 2018;476:400-407. DOI: 10.1007/s11999.0000000000000012
- [153] Tírico LEP, Pasqualin T, Pécora JO, Gobbi RG, Pécora JR, Demange MK. Estudo da estabilidade dos componentes na artroplastia total do joelho sem cimento. *Acta Ortopédica Brasileira*. 2012;20(4):230-234. DOI: 10.1590/s1413-78522012000400008
- [154] Fraser JF, Werner S, Jacofsky DJ. Wear and loosening in total knee

arthroplasty: A quick review. *The Journal of Knee Surgery*. 2015;**28**:139-144. DOI: 10.1055/s-0034-1398375

[155] Naudie DDR, Ammeen DJ, Engh GA, Rorabeck CH. Wear and osteolysis around total knee arthroplasty. *The Journal of the American Academy of Orthopaedic Surgeons*. 2007;**15**:53-64

[156] Bozic KJ, Kurtz SM, Lau E, Ong K, Chiu V, Vail TP, et al. The epidemiology of revision total knee arthroplasty in the United States. *Clinical Orthopaedics and Related Research*. 2010;**468**:45-51. DOI: 10.1007/s11999-009-0945-0

[157] Peters PC, Engh GA, Dwyer KA, Vinh TN. Osteolysis after total knee arthroplasty without cement. *The Journal of Bone and Joint Surgery. American Volume*. 1992;**74**:864-876

[158] Robinson EJ, Mulliken BD, Bourne RB, Rorabeck CH, Alvarez C. Catastrophic osteolysis in total knee replacement. A report of 17 cases. *Clinical Orthopaedics and Related Research*. 1995;**321**:98-105

[159] Callaghan JJ, O'Rourke MR, Liu SS. The role of implant constraint in revision total knee arthroplasty: Not too little, not too much. *Journal of Arthroplasty*. 2005;**20**:41-43

[160] Doolittle KH, Turner RH. Patellofemoral problems following total knee arthroplasty. *Orthopaedic Review*. 1988;**17**:696-702

[161] Bozic KJ, Kamath AF, Ong K, Lau E, Kurtz S, Chan V, et al. Comparative epidemiology of revision arthroplasty: Failed THA poses greater clinical and economic burdens than failed TKA. *Clinical Orthopaedics and Related Research*. 2015;**473**:2131-2138. DOI: 10.1007/s11999-014-4078-8

[162] Vince KG. The problem total knee replacement: Systematic, comprehensive and efficient evaluation. *The Bone*

and Joint Journal 2014;**96**-B:105-111. doi:10.1302/0301-620X.96B11.34531

[163] Doolittle KH, Turner RH. Patellofemoral problems following total knee arthroplasty. *Orthopaedic Review*. 1988

[164] Young SW, Saffi M, Spangehl MJ, Clarke HD. Unexplained pain following total knee arthroplasty: Is rotational malalignment the problem? *The Knee*. 2018. DOI: 10.1016/j.knee.2018.01.011

[165] Czurda T, Fennema P, Baumgartner M, Ritschl P. The association between component malalignment and post-operative pain following navigation-assisted total knee arthroplasty: Results of a cohort/nested case-control study. *The Knee Surgery, Sports Traumatology, Arthroscopy*. 2010. DOI: 10.1007/s00167-009-0990-y

[166] Longo UG, Ciuffreda M, Mannering N, D'Andrea V, Cimmino M, Denaro V, et al. Patellar resurfacing in total knee arthroplasty: Systematic review and meta-analysis. *Journal of Arthroplasty*. 2017. DOI:10.1016/j.arth.2017.08.041 LK. Available from: <http://sfxit.ugent.be/ugent?sid=EMBASE&issn=15328406&id=doi:10.1016%2Fj.arth.2017.08.041&atitle=Patellar+Resurfacing+in+Total+Knee+Arthroplasty%3A+Systematic+Review+and+Meta-Analysis&stitle=J.+Arthroplasty&title=Journal+of+Arthroplasty&volume=&issue=&spage=&epage=&aulast=Longo&aufirst=Umile+G.&auinit=U.G.&aufull=Longo+U.G.&coden=JOARE&isbn=&pages=-&date=2017&auinit-1=U&auinitm=G>

[167] Petersen W, Rembitzki IV, Brüggemann GP, Ellermann A, Best R, Koppenburg AG, et al. Anterior knee pain after total knee arthroplasty: A narrative review. *International Orthopaedics*. 2014;**38**(2):319-328. DOI: 10.1007/s00264-013-2081-4