Knee Injuries in Football

3

Jacob B. Stirton, Burak Altintas, and Darren L. Johnson

Knee Dislocation

The top priority of the team physician and athletic training staff is always to rule out a medical emergency. Regarding the knee, these emergencies involve vascular injuries which result in a compromise of blood flow to the limb. This is typically associated with knee dislocations or multiligamentous injuries with a large zone of injury. This requires timely recognition with subsequent transport to a hospital for definitive work-up and treatment. The key is to quickly recognize the injury as one that cannot be treated on the sideline and that needs emergent transport to a high-level trauma center where a vascular surgeon is on call.

A typical scenario involves a player being tackled from multiple directions with a planted leg. Observers may even see the knee "bend in the wrong direction" (Fig. 3.1a). It is an injury that will cause most people in the stadium to gasp and instantly know something is very wrong. The player will be writhing in pain – much more so than other knee injuries that we will address later on in this chapter.

Our job is to immediately diagnose, stabilize, and transport our athlete to a higher level of care. A delay in care can result in a loss of limb. The gravity of this statement cannot be overstated.

When approaching the player on the field, an immediate assessment of the knee is required. If the knee is "locked" in a dislocated position, remove the player's shoes and socks and check for a posterior tibial pulse prior to reduction. Once

J. B. Stirton (✉)
Department of Orthopedics and Sports Medicine, Union General Hospital, Blairsville, GA, USA

B. Altintas · D. L. Johnson
Department of Orthopedics and Sports Medicine, University of Kentucky, Lexington, KY, USA
e-mail: dljohns@uky.edu

K. W. Farmer (ed.), *Football Injuries*,
https://doi.org/10.1007/978-3-030-54875-9_3

Fig. 3.1 Traumatic knee dislocation (**a**) and on-field evaluation, stabilization, and transport (**b**)

neurovascular status has been assessed, longitudinal traction/countertraction will reduce the joint. This is a multi-person effort, especially with a player who is likely in significant pain. Once reduced, which you will recognize by a more "normal"-appearing knee as well as an ability to now passively range the knee, reassess the pulse. Regardless of the pulses, this is still an emergency that must be transported to the hospital immediately. EMS should be joining you on the field at this point. The assessment of the pulses allows for an accurate description of the timing of events to the treating physicians at the hospital. Limb salvage and vascular repair are time sensitive.

After the knee has been reduced and the neurovascular status has been assessed, there is no need for further examination. Stabilize the knee in extension and immediately transport to the hospital (Fig. 3.1b).

A second scenario (and more common) is the knee dislocation or multiligamentous knee injury that is already reduced when you arrive at the player's side on the field. Most of us can recognize a dislocated knee and know that it is an emergency. The much more dangerous situation is one in which we fail to recognize the dislocation because the knee is already reduced. A failure to recognize the gravity of the situation can cause (and has caused) players to lose their leg due to inadequate treatment. There are three keys to recognizing this injury: have a high index of suspicion based on the violent nature of the injury (knee bent the wrong way or people gasping and looking away), a good examination on the field, and a low threshold for transporting to the hospital immediately.

The first key should be easy. We all know a bad injury when we see it. If you see it, do not second guess yourself, and go onto the field thinking the worst. It is a dislocation until proven otherwise.

Once you are on the field, your assessment is the next key. First document pulses. Any pulse that is absent or diminished when compared to the contralateral side is an emergency. Second, any knee injury that opens to a valgus or varus stress in full extension is unstable and represents either a now reduced knee dislocation or a multiligamentous knee injury. After the neurovascular exam, we would argue that this is the most important on the field examination maneuver of the knee. It will instantly tell you what is the emergency and what can be worked up further on the sideline.

The last key is that if your index of suspicion was high going onto the field and you cannot definitively rule out a knee dislocation and multiligamentous knee injury, then transport the athlete to the hospital. Do not let your pride put their health at risk. Let someone with more expertise, access to advanced imaging, and the ability to definitively treat make the call. No one will fault you for putting your student athlete's health first.

Meniscus Injury

Meniscal injuries, although not as common as MCL or anterior cruciate ligament (ACL) injuries, still account for almost 18% of all knee injuries in the football athlete [1]. Expanding evidence that the meniscus is not only a shock absorber but also an important secondary stabilizer of the knee has led to an even more important role in early diagnosis and treatment of these injuries as it is paramount to the long-term health of the knee joint.

The medial and lateral menisci are c-shaped fibrocartilaginous structures that attach to the tibial plateau anteriorly and posteriorly via their respective roots. They are wedge shaped in cross section and act to increase the congruency between the femoral condyles and tibial plateaus. The difference in shape of the two menisci can be explained by the difference in shape of the bony components of the lateral and medial compartments. The medial meniscus is more c-shaped while the lateral meniscus is more circular. The lateral meniscus is also more mobile than the medial meniscus due to fewer peripheral attachments and the need for greater excursion during knee range of motion. This may also explain the lower incidence of lateral meniscal injuries versus medial meniscus injury [2]. The biomechanical properties of the menisci arise from the arrangement of collagen fibers in a circumferential fashion that disperses the hoop stresses of weight-bearing as well as the radial fibers that resist longitudinal stresses. The lateral compartment of the knee is more dependent on its respective meniscus than the medial compartment due to the difference in shape and size between the lateral and medial meniscus. It is very important to understand that return to play differs in the knee with a simple medial meniscectomy versus lateral meniscectomy. This is because the lateral meniscus transmits more load and covers more surface area of the articular cartilage on the lateral side than the medial meniscus does on the medial side. Both menisci not only provide congruency for load transmission but also add stability to the knee. [3, 4]

The focus of this discussion is on acute meniscus tears in the football player; therefore the typical mechanism of an *acute* injury to the meniscus is a twisting motion of the flexed knee while the foot is planted. The highest-risk athletes therefore are those in cutting sports such as football and soccer with men being almost four times as likely to tear their meniscus as women [5]. And injury to the ACL significantly increases the risk of meniscal pathology (up to 60–70% prevalence in acute ACL ruptures), likely due to their insufficient efforts as secondary stabilizers to the knee after loss of the ACL [6].

On-field examination of the knee should include a brief explanation of injury mechanism, followed by a focused examination to localize tenderness to palpation (usually at the joint line for meniscal pathology) as well as a thorough ligamentous and neurovascular exam. Once ruled stable, the player can be transferred to the sideline or locker room for a more in-depth evaluation.

Once the player is off the field of play, he may be more thoroughly examined by the team physician. Once again, confirm the mechanism of injury with the player. Proceed with a lower-extremity exam focusing on the knee in the usual manner: inspection, palpation, range of motion, assessment of strength, and neurovascular exam, followed by any special tests. In this case, focus on palpation of the joint line as this is the most sensitive and specific physical examination finding for the diagnosis of a meniscus injury [7]. However, it is less so in the setting of a concomitant ACL injury [8]. Special tests for meniscal pathology include the McMurray, Apley grind, and Thessaly tests. The player should also be evaluated for any concomitant ligamentous injury. Over the following 24–48 hours, care should be taken to monitor for any joint effusion that may develop indicating an intraarticular pathology. An isolated meniscus tear in the white-white zone typically does not result in an effusion in the first 48 hours.

If there is high suspicion for meniscal pathology found on physical examination (e.g., effusion, joint line tenderness, positive McMurray test, etc.), advanced imaging is warranted. As with most soft tissue injuries, a non-contrast MRI is the study of choice (Fig. 3.2a), although most typically a complete set of standing plain films of the knee will be ordered first to rule out any osseous pathology or malalignment issues. The menisci should be evaluated on all three planes of the MRI – coronal, sagittal, and axial. The coronal and sagittal views will elucidate the majority of tears, but the axial can be helpful in certain cases of radial or bucket-handle tears if it happens to catch the right cut.

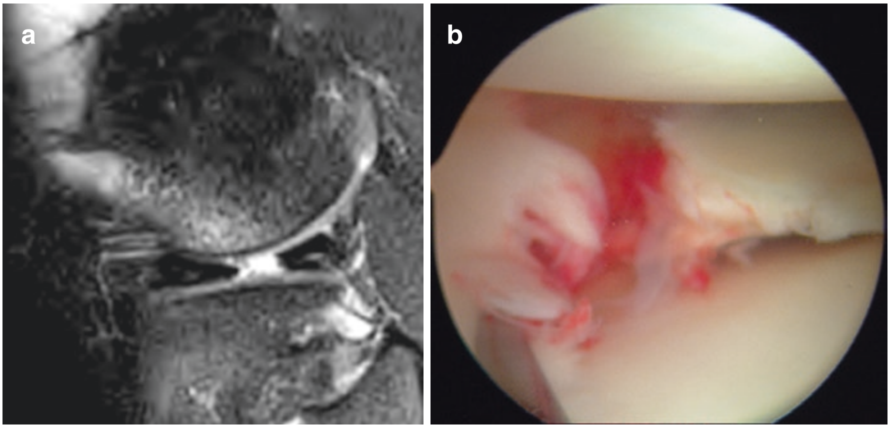

Fig. 3.2 T2-weighted sagittal MRI image of an acute lateral meniscus radial tear (**a**) and corresponding arthroscopic image in preparation of repair (**b**)

The differentiation between exact tear types is beyond the scope of this chapter, but different variations do exist – vertical, horizontal, oblique, radial, etc. For the purpose of nonoperative versus operative treatment, the focus of this chapter will be on stable versus unstable tear types.

Small, stable meniscal tears with no mechanical symptoms (catching, locking) can be treated nonoperatively with rest, activity modification until asymptomatic, NSAIDs, and possibly intraarticular injections of corticosteroids with/without a local anesthetic. The main issue with nonoperative management of meniscal tears, specifically in the high-level athlete, is their risk for re-injury and tear propagation leading to an irreparable tear. This would necessitate the need for partial meniscectomy which has deleterious effects on pressure distribution, load sharing, and stabilization as discussed earlier. Therefore, the authors suggest every effort to preserve meniscal tissue for the overall long-term health of the knee. A non-displaced meniscal tear without effusion or significant mechanical symptoms may be allowed to participate in football until the season is over after a lengthy educational discussion with all involved.

Operative management is necessary in large and unstable tears, patients with mechanical symptoms, and those who have failed to improve with nonoperative treatment. Operative interventions include either repair (Fig. 3.2b) or excision of the torn tissue or a combination of both. While this chapter will not go into an in-depth explanation of operative techniques, it is the authors' opinion that meniscal tissue should be salvaged if at all possible, especially in the young athlete and particularly on the lateral side. The post lateral meniscectomized knee is one of the most common "career-ending" situations seen at the collegiate and NFL level. A truly isolated bucket-handle tear of the medial meniscus is very abnormal, while seeing this injury on the lateral side is quite common. We often repair this lateral bucket-handle tear but the player's season is done. The medial-sided bucket-handle tear is much more controversial with high failure rates of isolated repair. Unless it is the entire medial meniscus, we would recommend meniscectomy on the medial side in the normal knee, while repair of the lateral side is required because of the biomechanical characteristics of each menisci. While very small oblique tears can likely be debrided back to stable tissue, most other tear patterns will necessitate an attempt for repair. Meniscus repair techniques depend on the tear pattern and surgeon preference, but all include repairing a tear with good vascularity and potential for healing in a stable way, whether that be inside-out, outside-in, or all-inside repair technique. It should also be mentioned that evidence has shown that meniscal repairs in the setting of concomitant ACL reconstructions have a higher rate of healing than meniscus repairs performed in isolation. This is possibly secondary to bone marrow stimulation and subsequent influx of bone marrow cells into the joint during the ACL reconstruction [9]. Therefore, the authors advocate microfracture of the intercondylar notch for all meniscus repairs done in isolation in order to potentiate that same healing response.

Rehabilitation for nonoperative treatment of meniscus injuries includes rest, activity modification until asymptomatic, NSAIDs, and possibly intraarticular injections of corticosteroids with/without a local anesthetic. The rehabilitation process for meniscal pathology treated surgically is dependent on the stability of the tear pattern as well as the operative intervention – repair versus debridement. A small stable tear that is simply debrided to a stable border can begin weight-bearing immediately after surgery with therapy to address swelling, ROM, and strengthening. Impact and loaded squatting past 90 degrees of knee flexion is avoided for the first 4 weeks, but initiated soon after. Meniscal tears that are stable but require a repair are treated more conservatively. The authors prefer a 4-week period of weight-bearing as tolerated in a hinged knee brace locked in full extension with immediate postoperative therapy to address swelling, ROM, and strengthening as well. Similarly, loaded knee flexion past 90 degrees and impact are avoided for 8 weeks and then gradually reintroduced. Strengthening and straight-line exercises are continued for the next 2 months at which time cutting drills can be initiated. The most unstable tears (bucket handle, radial) requiring surgical repair are placed on crutches and made toe-touch weight-bearing in a hinged knee brace locked at 30 degrees of knee flexion for the first 4 weeks. They are then transitioned to weight-bearing as tolerated in a hinged knee brace locked in full extension for the following 4 weeks. Therapy to address swelling, ROM, and strengthening are still initiated immediately after surgery with the caveat that ROM not exceed 90 degrees of flexion, even non-weight-bearing, for the first 4 weeks after surgery. They are then progressed similarly to above (only on a 4-week delay). It should be noted that regardless of initial tear stability, all patients are encouraged to come out of their brace while sitting or lying down to work on range of motion immediately after surgery and throughout recovery.

While there is no data supporting strict return to play criteria postoperatively, most surgeons would agree that the patient must have full range of motion and at least 80% strength compared to the contralateral side [10]. Many protocols employ functional testing and single leg hop or crossover hop tests to demonstrate appropriate strength and proprioception. A simple meniscus debridement could return to play within a month (lateral takes longer to return than medial), a stable meniscus repair by 3–4 months, and an unstable meniscus repair by 4–6 months, although each athlete is treated on an individual basis.

MCL and Posteromedial Corner Injuries

Medial collateral ligament (MCL) injuries are the most common ligamentous knee injury in football players as well as the general population (40% of all ligamentous knee injuries). The majority of these are low grade and can be treated nonoperatively, but a thorough examination for concomitant injuries is paramount in protecting our athletes and safely returning them to play.

Valgus forces at the knee are resisted by static stabilizers of the medial knee including the posterior oblique ligament (POL), superficial MCL, and deep MCL. Valgus forces are also resisted by dynamic forces such as the medial head of the gastrocnemius, vastus medialis, pes anserinus, and semimembranosus [11]. The superficial and deep MCLs provide resistance to valgus stress at the knee while the posteromedial corner provides rotational stability to the medial side of the knee. The MCL has a native strength of 4000 N [12].

An MCL injury typically occurs after a contact injury resulting in a valgus force to the knee joint with the foot planted on the ground. This mechanism often leads to a disruption of the deep and superficial MCL. If this is combined with an external rotational force, a disruption of additional restraints such as the ACL and the posteromedial corner is possible. This injury is most commonly seen when a ball carrier is tackled into the lateral aspect of an offensive lineman's knee while he is engaged with a defender [13]. The most commonly associated injuries with a non-isolated MCL tear are injuries to the ACL (95%) followed by the meniscus (5%) [14].

Like most knee injuries, an acute evaluation of an MCL is ideal, prior to any muscle spasms occurring. This initial cursory physical exam and history can take place on the field by the athletic trainer/team physician. A description of the mechanism of injury must be elicited. The examination includes inspection for peripheral hematoma along the medial side of the knee, palpation of the joint line, hamstrings, and the femoral origin and tibial insertion of the MCL, as well as ligamentous stability exams. A thorough evaluation of the neurovascular status of the extremity is necessary to avoid missing a limb-threatening vascular injury. Once it is concluded that the player is stable, he may be removed to the sideline for further in-depth examination.

Once the player is off the field of play, he may be more thoroughly examined by the team physician. Once again, the mechanism of injury needs to be confirmed with the player. Afterward, a lower-extremity examination focusing on the knee in the usual manner should proceed: inspection, palpation, range of motion, assessment of strength, and neurovascular examination, followed by any special tests. In this case, a valgus stress test is performed at full extension and 30 degrees of knee flexion to diagnose MCL injuries: grade 1 (0- to 4-mm medial-sided opening), grade 2 (5- to 9-mm opening), and grade 3 (10- to 15-mm opening). Of note, any medial-sided opening at full extension should alert the examiner to a concomitant ACL or posteromedial corner injury and a possible knee dislocation. Consideration for urgent triage to a medical facility for a definitive work-up is appropriate. Slocum-modified anterior drawer test and an anterior drawer may be considered in external rotation test to evaluate the deep MCL and posteromedial corner, respectively. A thorough examination of the knee should also assess the meniscus with joint line palpation and a McMurray or Thessaly test, the ACL with a Lachman or anterior drawer, the posterior cruciate ligament (PCL) with a posterior drawer, and the patella with the apprehension test. The MCL is most commonly injured on the femoral side of the MCL and is easily diagnosed by simple palpation. Distal MCL tears/avulsions are much less common but tend to be more severe. Any grade 3 opening at 30 or any effusion seen in the first 24 hours requires an urgent MRI.

Isolated grades 1 and 2 MCL injuries can be diagnosed clinically; and generally, an MRI is not necessarily indicated unless the patient has an intra-articular effusion or signs of other ligamentous injuries. If the player has been examined and further imaging is required to confirm the diagnosis, then standing X-rays of the knees can be obtained. The authors typically prefer at least three views – AP, lateral, and sunrise. Plain radiographs are usually not helpful for the diagnosis of an acute MCL injury but can rule out any bony abnormality such as an osseous avulsion or fracture. MRI without contrast is warranted to assess the extent and location of the MCL injury and to rule out any other pathology such as an ACL, posteromedial corner, or meniscus injury.

Acute isolated MCL injuries usually are treated nonoperatively with protective weight-bearing and bracing for 2 to 6 weeks (Fig. 3.3a). In particular, partial tears of the MCL (grade 1 or 2) heal well with conservative treatment [15]. Complete MCL injuries (grade 3 injuries) initially can be treated nonoperatively if they are femoral-based ligament ruptures (Fig. 3.3b) [16].

A complete tibial-sided MCL avulsion with POL extension (knee opens in full extension) is less likely to tighten up with nonoperative management and may require repair or reconstruction. Grade 3 MCL injuries in combination with other ligament injuries of the knee may require acute surgical repair in case of a complete tibial avulsion with POL extension [17]. The rehabilitation and return to play are dictated by the extent of the injuries (e.g., isolated MCL repair could be out 4–6 months while an ACL/MCL reconstruction could be out over 1 year).

Rehabilitation for grade 1 and 2 MCL injuries that are treated nonoperatively consists of a short period of protective weight-bearing and bracing followed by progressive strengthening with quad sets, straight leg raises, and hip adduction exercises. Athletes can work on range of motion and endurance by cycling and adding progressive resistance exercises as tolerated.

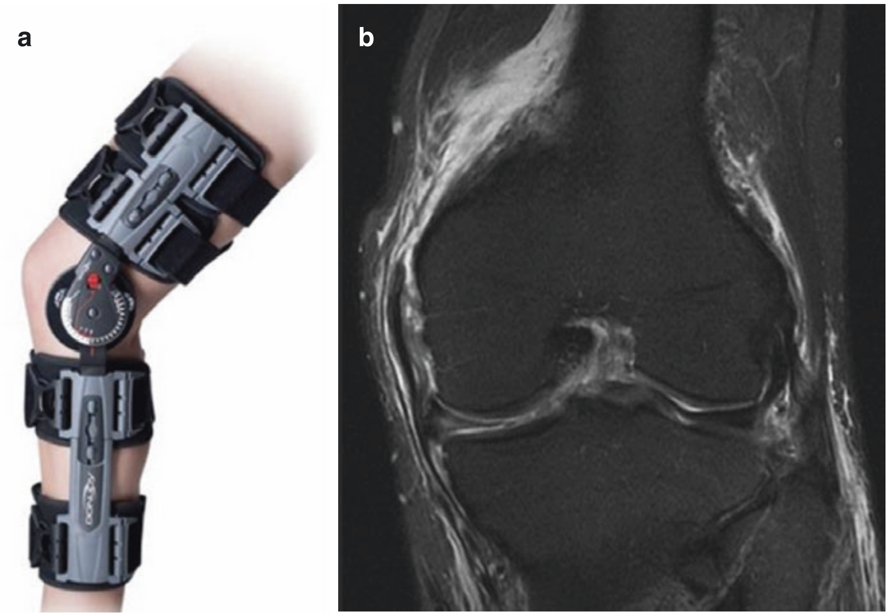

Fig. 3.3 Example of an MCL protecting knee brace from DonJoy (**a**) and T2-weighted coronal MRI image of a femoral-based MCL avulsion injury (**b**)

Grade 1 MCL sprains typically can return to play in 5–7 days, grade 2 in 2–4 weeks, and grade 3 in 4–8 weeks. But these return to play times are completely position dependent. Skill players may require much longer than those who play in the interior of the field. Functional MCL braces can be helpful for offensive linemen and are shown to help prevent MCL injuries in this particular group.

ACL Injury

In the 1970s, Dr. J.C. Kennedy would say that the ACL tear is the most common cause of the ex-athlete [18] meaning and that this was a career-ending injury. While still devastating, this absolutist view is no longer the case. A complete lateral meniscectomy is our most common unsolvable problem in this patient population today. With modern surgical techniques and the current view of rehabilitation, many athletes return to level I sports after ACL injuries.

The ACL's primary function is to resist anterolateral translation of the tibia relative to the femur [19]. It also acts to resist varus displacement of the knee in full extension along with the PCL. The ACL has a native strength of 2200 N [20].

The typical mechanism for an ACL rupture is a noncontact pivoting injury. Although ACL tears are up to 4.5 times as likely in women as in men, they are an extremely common injury in the athlete with over 400,000 reconstructions taking place every year [21]. The most commonly associated injury with a non-isolated ACL rupture is a meniscus tear (up to 75% in acute tears) [6].

As always, an acute evaluation of the knee is ideal as muscle spasm and guarding have often yet to set in. This is the optimal time to assess the stability of the knee and the integrity of the ACL. While on the field, confirm the mechanism of injury with the athlete to determine where to focus your exam. Noncontact pivoting injuries described by an audible "pop" with immediate pain and inability to bear weight are a diagnosis of ACL rupture until proven otherwise. The athlete will typically complain of immediate lateral-sided knee pain (this is because of the pivot mechanism and the large lateral bone bruise that occurs). The player needs to relax as much as possible for a reliable Lachman or anterior drawer test (making sure to compare to the uninjured contralateral side). It is then appropriate to remove the athlete from the field of play and continue a more thorough examination on the sideline or in the locker room. Often the knee effusion (hemarthrosis) is not immediate within 4 hours as opposed to a patellar dislocation where it develops immediately. The ACL-torn knee often "swells up" overnight and the next morning presents to the training room with a large effusion.

Imaging of the knee should consist of three views of plain radiographs to rule out any bony injury and should be followed by an MRI without contrast to confirm the ACL rupture (Fig. 3.4a) and diagnose any other intraarticular pathology such as a meniscus tear or cartilage injury.

ACL ruptures in the athlete are almost certainly treated with surgery. Any high-demand-cutting athlete will be unable to perform to their preinjury level without complete stability of the knee. There is also a high risk of injury to other structures

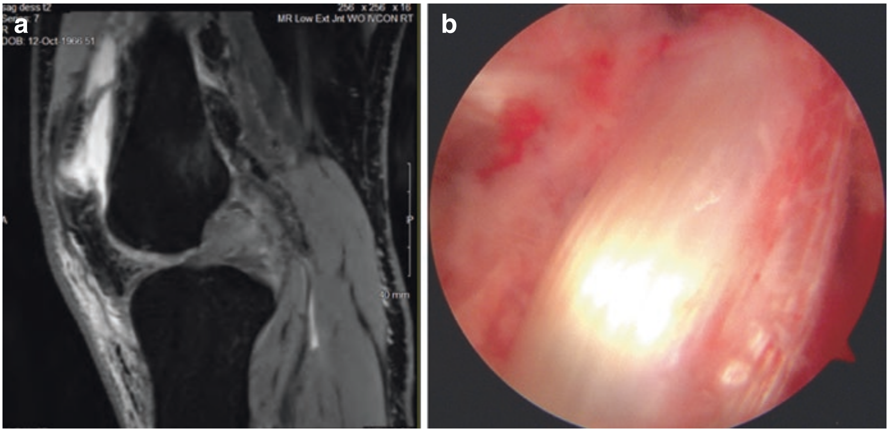

Fig. 3.4 T2-weighted sagittal MRI image of an ACL rupture (**a**) and arthroscopic image of a newly reconstructed ACL using BTB autograft (**b**)

in the ACL-deficient knee such as the medial meniscus which acts as a secondary stabilizer to anterior tibial translation and will endure abnormally high loads of force without an ACL [22]. Nonsurgical management may be considered in a player with a "partial ACL tear" or ACL sprain, which shows a majority of intact fibers on MRI but with some increased edema. If the physical exam confirms a solid endpoint and intact ACL, the athlete can opt fora course of nonoperative treatment which includes a period of rest and progressive rehabilitation. All other unstable ACL ruptures in high-level athletes require surgical treatment for restoration of stability.

The authors' preferred surgical treatment of ACL rupture in the football athlete is reconstruction with bone patella bone (BTB) autograft (Fig. 3.4b). There are varying opinions on the type of graft ranging from hamstring or BTB allografts to hamstring or quadriceps tendon autografts. The longest and most successful track record belongs to the BTB autograft which is why all other grafts are compared to it in high-level studies – e.g., the gold standard – and is this author's preferred technique. Other yet-to-be-proven techniques include ACL repair with new bioscaffolding or "internal bracing" techniques. These lack long-term outcome data and are not advocated by these authors (or senior author). [23, 24]

Rehabilitation from an ACL sprain is treated with activity modification to avoid cutting, pivoting, and impact for a period of 4–6 weeks. During that time focus should remain on range of motion and quad strengthening exercises. Gradual return to activity can resume starting at about 4 weeks from injury.

Postoperative rehabilitation from ACL reconstruction is still a hotly debated topic, but most surgeons agree that early range of motion and quad strengthening are critical components. Disagreement still remains on brace wear and exact return to play criteria and timelines. This author prefers his postoperative patients to begin physical therapy three days after surgery where they will focus on range of motion, patellar mobility, and quad activation. They are placed into a hinged knee brace locked in full extension any time they are ambulating for the first month after surgery. However, the brace comes off when they are sitting or lying down to encourage range of motion. They can wean off their crutches as soon as they are stable and no longer walk with a limp. At 4 weeks postoperatively, the hinged knee brace is unlocked during ambulation and they continue to progress their quad strengthening in physical therapy and at home. At 8 weeks postoperatively, they transition to a low-profile hinged knee sleeve which is worn any time they are in therapy, out of the house in crowded areas, or walking on uneven ground. This knee sleeve is worn for the next several months.

Therapy consists of phases: the immediate postop phase (1–2 weeks), ROM and early strengthening phase (2–4 weeks), the middle strengthening phase (4–12 weeks), the pre-impact and transition to late strengthening phase (3–4 months), and the impact and late strengthening phase (4–9 months).

Return to play is patient specific, but typically is between 9 months to 1 year from surgery. Each athlete will first undergo functional testing and then need to pass all sport-specific drills before being released to return to play. Each patient is counseled regarding the significant risk of graft re-rupture (~15%) as well as rupture of the contralateral ACL (~15%) with return to high-level sport [25].

PCL Injury

The PCL is recognized as an essential stabilizer of the knee. However, the complexity of the ligament has generated controversy about its definitive role and the recommended treatment after injury. A proper understanding of the functional role of the PCL is necessary to minimize residual instability, osteoarthritic progression, and failure of additional concomitant ligament graft reconstructions or meniscal repairs after treatment [26].

The PCL is composed of two bundles, the larger anterolateral bundle (ALB) and the smaller posteromedial bundle (PMB). Classically, the two bundles of the PCL were believed to function independently, with the ALB primarily functioning in flexion and the PMB in extension. However, in light of recent biomechanical investigations, a more synergistic and codominant relationship between the two bundles has been validated [22].

An anterior blow to a flexed knee, which can occur when an athlete falls directly on a hard surface with the knee flexed and foot in plantar flexion, may result in PCL tear [27]. As PCL injuries may be accompanied by concomitant ACL or collateral ligament injuries, leading to a knee dislocation, the on-field examination should focus on joint congruity to start with. If the knee is dislocated, it needs to be reduced on the field prior to transportation as it can be accompanied by a neurovascular injury putting the limb in danger. It is of utmost importance to check the neurovascular status before and after reduction in those cases followed by immobilization of the knee in a brace. The off-field evaluation should assess posterior translation of the knee. Tests used to evaluate the PCL are usually done with the knee flexed 90° since the PCL is the main structure preventing posterior translation of the tibia on the femur in this position [28]. Thus, when the PCL has been torn, posterior translation is increased. If the PCL is injured, a step-off deformity of the proximal tibia will be apparent (positive posterior sag sign). This test can be followed by the quadriceps active test. If the PCL is partially or completely torn, contracting the quadriceps will pull the posteriorly translated proximal tibia forward into a more normal position. Finally, the posterior drawer test (Fig. 3.5a) is done with the examiner sitting on the patient's foot to hold it in a neutral position. The amount of posterior translation is assessed by gently pushing the knee backward with the foot held in neutral rotation with the athlete's quadriceps and hamstrings completely relaxed. Imaging studies include plain radiographs as mentioned above to rule out osseous involvement. Stress radiographs can provide additional diagnostic information to help grade PCL tears [29]. MRI has become the study of choice in acute PCL injuries (Fig. 3.5b). A large prospective study found that MRI was 99% accurate in diagnosing the presence of PCL injury, confirmed by arthroscopy [30]. MRI is useful as it can also assess menisci, articular surfaces, and other ligaments of the knee.

Typically, acute, truly isolated PCL injuries (grades I and II) are treated nonsurgically. Most of these patients are able to return to sports within 4–6 weeks of nonoperative treatment [31]. The goal of a comprehensive rehabilitation program should be to strengthen the musculature about the knee while minimizing forces across the patellofemoral and tibiofemoral compartments. Exact rehabilitation protocols have been very nonspecific and varied. It has been shown that tibiofemoral compression forces are reduced with closed kinetic chain exercises, and open kinetic chain quadriceps exercises exert an anterior pull on the tibia [32].

Operative management (fairly uncommon) is usually indicated in isolated grade 3 PCL tears that remain symptomatic despite an adequate course of conservative therapy and for PCL injuries in the setting of concomitant-associated injuries. These include PLC injury or in the presence of a repairable meniscal body/root tear or MCL insufficiency [27]. There are two prevalent techniques that exist for PCL reconstruction based on the tibial insertion site: the transtibial and the tibial inlay techniques [27]. While there are multiple graft options, a recent systematic review did not show a significant difference in postoperative functional outcomes between patients treated with autograft and those treated with allograft [33]. There is no

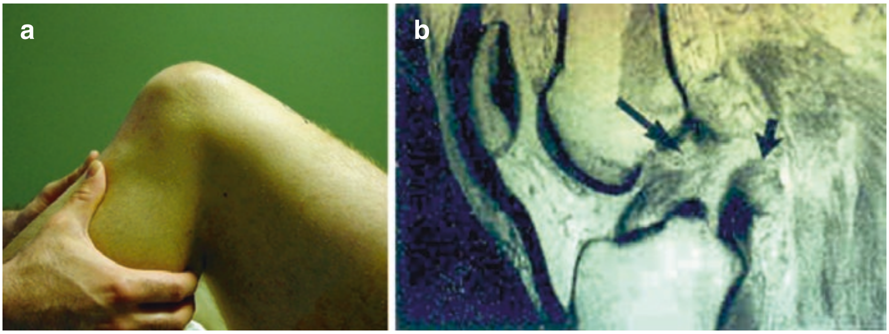

Fig. 3.5 Posterior drawer test signifying a PCL rupture (**a**) T1-weighted sagittal MRI image of a proximal PCL avulsion (**b**)

consensus in the postoperative rehabilitation in the current literature. The senior author of this article prefers toe-touch weight-bearing for four weeks with the brace locked in 30 degrees of flexion for the first four weeks after the surgery but enforces ROM as tolerated with the athlete in supine or sitting position. Afterward the athlete can be transitioned into weight-bearing as tolerated with the brace locked in extension for another four weeks. Starting in the ninth postoperative week, the athlete may continue weight-bearing with the brace unlocked for another month. The expected return to play after PCL reconstruction is similar to ACL reconstruction depending on the athlete's symptoms, ROM, stability, and strength. There are no clear guidelines regarding return to play after PCL reconstruction [34].

LCL and Posterolateral Corner Injuries

A varus load or blow inside of the knee may result in lateral collateral ligament (LCL) tears [35]. The majority of posterolateral knee injuries are caused by a blow to the anteromedial aspect of the knee, a contact or noncontact hyperextension injury, or a varus noncontact injury [36]. Approximately 60% of PCL injuries are associated with tears of the PLC structures, including the LCL, coronary ligament, popliteo-fibular ligament, popliteus tendon, and arcuate ligament [31]. An overseen LCL or PLC injury may lead to varus-thrust gait pattern which may be debilitating.

The LCL is the primary static stabilizer to varus opening of the knee [32]. The popliteus muscle and tendon complex on the posterolateral aspect of the knee has many components that provide a static and dynamic stabilizing effect to the posterolateral rotation of the knee. The midthird lateral capsular ligament is a thickening of the lateral capsule of the knee. The superficial layer consists of the iliotibial band and the biceps femoris.

The on-field examination of the injured athlete should again as stated above focus on ruling out a knee dislocation. If this is the case, the knee should be reduced immediately, and the athlete should be sent to the closest emergency room for further management. The delay in the ER presentation may result in serious long-term vascular complications which may even lead to loss of limb. Thus, these injuries should be taken very seriously. Most PLC injuries present with concomitant ACL or PCL injuries and isolated cases are rare. The off-field examination of the athlete should focus on determining the extent of the injury and understanding concomitant injuries. Especially the peroneal nerve can be injured. The correct diagnosis and documentation of this involvement are crucial for further management of the athlete as a severe peroneal nerve involvement with foot drop will necessitate an ankle-foot orthosis (AFO) to prevent long-term flexion contracture of the foot.

The severity of collateral ligament injury is generally indicated by the amount of joint-line opening with varus stress: less than 5-mm opening is indicative of grade 1 (minor sprain) injury while 6–10-mm opening is suggestive of a grade 2 (partial tear) injury. If the knee opens more than 1 cm without solid endpoint, a grade 3 (complete tear) is expected. However, the amount of joint-line opening in a varus stress test varies among patients, so it is essential to compare the injured knee with the uninjured knee. The dial test assesses posterolateral rotation of the tibia on the femur and injuries to the posterolateral knee. While the test is often described as being done with the patient prone, it can be performed in a supine position as well. This is especially more convenient for off-field examination. As the athlete's feet are externally rotated, the amount of rotation of the two limbs is compared by observing the tibial tubercles. An increased passive external rotation in 30 degrees of knee flexion is indicative of a PLC injury while a positive dial test in 90 degrees of knee flexion shows a combined PLC and PCL injury.

The imaging consists of radiographs of the injured knee. Special attention should be paid to Segond fracture which is a cortical avulsion at the tibial insertion of the lateral capsule in its mid-portion and is indicative of severe ligamentous damage. Moreover, the arcuate sign may be present on standard AP radiograph indicating avulsion fracture of the fibular head (Fig. 3.6a) [31]. Ultimately, an MRI of the knee should be obtained to assess the extent of the damage.

The recommended treatment for patients with grade 1 to 2 isolated PLC/LCL injuries (partial injuries) initially is nonoperative. This involves RICE, weight-bearing as tolerated, temporary bracing, and closed chain quadriceps exercises. The athlete may ambulate off crutches when he/she can walk without a limp. The estimated return to play is approximately 1 week for grade 1 injuries while it may take 1–4 weeks for grade 2 injuries.

Grade 3 injuries usually require surgical treatment. Of note, it is extremely unusual to have an isolated grade 3 injury of the PLC and is much more common to be combined with an ACL tear, PCL tear, or both. Based on the time from injury to surgery as well as injury severity, the surgical treatment may range from direct

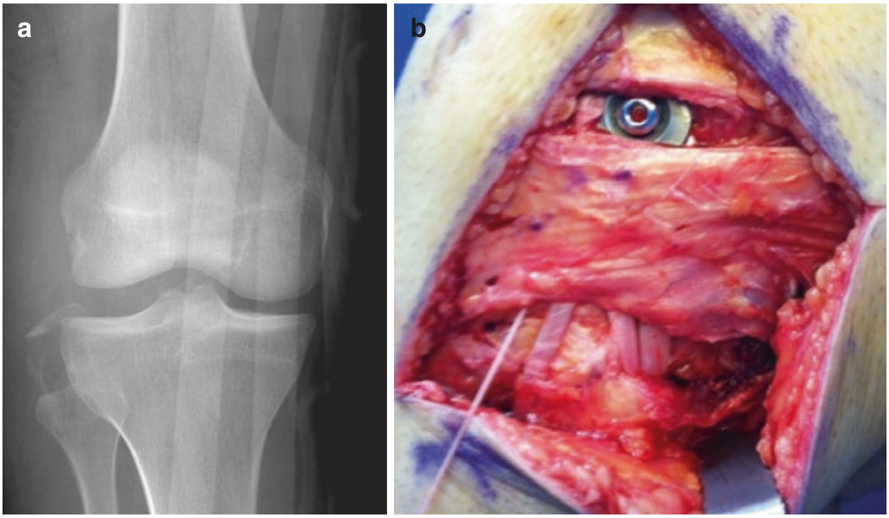

Fig. 3.6 AP plain film illustrating the fibular avulsion fracture, or "arcuate sign," as well as lateral joint space widening (**a**) and clinical image of a newly reconstructed PLC using a modified Larson technique using a hamstring allograft (**b**)

repair to reconstruction using auto- or allografts. The senior author prefers the modified Larson technique using a hamstring graft (Fig. 3.6b). The postoperative rehabilitation is the same as in PCL injuries. Regarding the return to play, there are no high-level evidence-based guidelines. The postoperative timeline is dependent on the athlete's subjective and objective assessment as mentioned above for the PCL injuries.

Patellar Dislocation Injury

Fifty to 60% of initial first-time lateral patellar dislocations will occur secondary to a sports-related injury and will involve a compromised medial patellofemoral ligament (MPFL) and medial retinaculum at least 80% of the time. [37, 38] The MPFL is a thin fascial band approximately 53 (ranging 45–64) mm long that links from the region of the medial epicondyle of the femur to the proximal part of the medial border of the patella. It is the primary passive restraint that resists lateral translation of the patella [39].

Lateral patellar dislocations may occur after lateral blow to the knee (Fig. 3.7a). The on-the-field management relies on the correct recognition and diagnosis followed by reducing it using a medial reduction maneuver while moving the knee into full extension; this will always reduce the patella if it is still dislocated in flexion "on the field." The off-field evaluation includes a more thorough examination with palpation along the MPFL and retinacular structures followed by performing the patellar apprehension test. With the athlete seated or supine and the knee flexed to 45°, the patella is pushed laterally. Apparent increased laxity and apprehension indicate the possibility of patellar subluxation or dislocation. Although rare, other injuries such as quadriceps tendon tears should be evaluated as well. A single leg raise test is usually sufficient for this. A large effusion hemarthrosis will develop in the first 12 hours which is typically much quicker than an ACL tear.

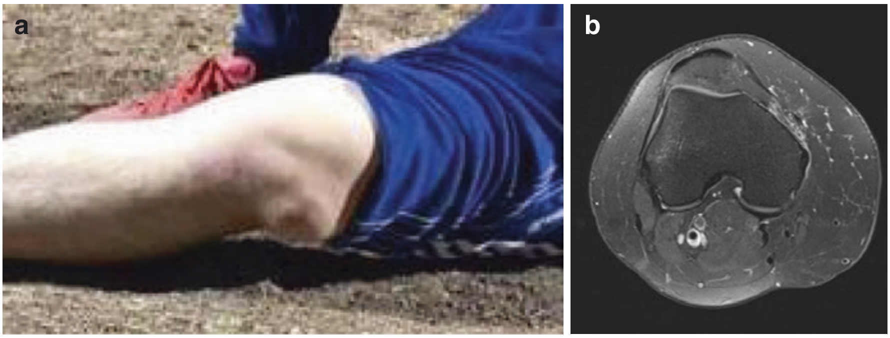

Fig. 3.7 Acute lateral patellar dislocation (**a**) and T2-weighted axial MRI image demonstrating an MPFL rupture (**b**)

The imaging should include radiographs to rule out fractures or osteochondral lesions. Moreover, an MRI of the knee needs to be obtained to assess the MPFL integrity as well as any chondral lesions with or without loose bodies or concomitant ligamentous damage (Fig. 3.7b). While recurrent lateral patellar dislocation is known to lead to degenerative process, a single first-time or infrequently recurring traumatic lateral patellar dislocation also seems to be associated with gradual cartilage deterioration [40]. Measurement of the Caton-Deschamps index and the tibial tubercle trochlear groove distance as well as the assessment of the trochlear groove is important to understand the anatomy and guide treatment. The bone bruise of the medial patella along with the lateral trochlea may be signs of patellar instability.

First-time patellar dislocations are commonly treated nonoperatively. Once this decision is made, aspiration of the hemarthrosis and injection of corticosteroid are performed to eliminate the knee effusion as quickly as possible and to allow for early range of motion and rehabilitation. Rehabilitation of the major muscle group of the thigh simply cannot move forward with a knee effusion. The biology of the knee, or an effusion, dictates how fast we can move in therapy and eventually return to play. However, a recent study suggests that the surgical MPFL reconstruction achieved better clinical outcomes than nonsurgical treatment for the acute primary patellar dislocation in the skeletally mature patients with the presence of abnormal patellofemoral anatomy. Thus, surgery may be considered as the better choice for these specific patients. [41, 42] Trochlear dysplasia, elevated TT–TG distance, patella alta, complete MPFL tear at its isolated femoral side, and complete combined MPFL tear in the first-time dislocation are independently associated with a higher incidence rate of the second-time dislocation [37]. Thus these factors should be weighted in the decision making. The nonoperative treatment includes mobilization with a brace locked in extension with weight-bearing as tolerated for one month with moving the knee as tolerated up to 90 degrees of flexion. If the patella remains stable after one month, the brace may be transitioned into a hinged patellar knee sleeve without any ROM restrictions. The sleeve can be discontinued by symptom relief and clinical patellar stability after 2 months.

Operative treatment includes the repair versus reconstruction of the MPFL. A recent systematic review showed that a double-bundle MPFL reconstruction seems to provide more favorable outcomes than a single-bundle MPFL reconstruction [43]. The senior author prefers reconstruction using a hamstring autograft for the treatment of recurrent dislocators. The postoperative rehabilitation is similar to the ACL reconstruction and involves one month of bracing locked in extension with weight-bearing as tolerated followed by gradual progression. The best available studies to date would suggest high return to play rates (84%–100%) and a highly variable timeframe for return (3–12 months) after patellar stabilization surgery [34].

References

1. Loughran GJ, et al. Incidence of knee injuries on artificial turf versus natural grass in National Collegiate Athletic Association American Football: 2004-2005 through 2013-2014 seasons. Am J Sports Med. 2019;47(6):1294–301.
2. Thompson WO, Thaete FL, Fu FH, et al. Tibial meniscal dynamics using three-dimensional reconstruction of magnetic resonance images. Am J Sports Med. 1991;19:210–6.
3. Greis PE, Bardana DD, Holmstrom MC, et al. Meniscal injury: I. Basic science and evaluation. J Am Acad Orthop Surg. 2002;10:168–76.
4. McDermott ID, Amis AA. The consequences of meniscectomy. J Bone Joint Surg Br. 2006;88:1549–56.
5. Baker BE, Peckham AC, Pupparo F, et al. Review of meniscal injury and associated sports. Am J Sports Med. 1985;13:1–4.
6. Spindler KP, Schils JP, Bergfeld JA, et al. Prospective study of osseous, articular, and meniscal lesions in recent anterior cruciate ligament tears by magnetic resonance imaging and arthroscopy. Am J Sports Med. 1993;21:551–7.
7. Fowler PJ, Lubliner JA. The predictive value of five clinical signs in the evaluation of meniscal pathology. Arthroscopy. 1989;5:184–6.
8. Shelbourne KD, Martini DJ, McCarroll JR, et al. Correlation of joint line tenderness and meniscal lesions in patients with acute anterior cruciate ligament tears. Am J Sports Med. 1995;23:166–9.
9. Krych AJ, et al. Surgical repair of meniscal tears with concomitant anterior cruciate ligament reconstruction in patients 18 years and younger. Am J Sports Med. 2010;38(5):976–82.
10. Kozlowski EJ, Barcia AM, Tokish JM. Meniscus repair: the role of accelerated rehabilitation in return to sport. Sports Med Arthrosc Rev. 2012;20:121–6.
11. LaPrade RF, Engebretson AH, Ly TY, et al. The anatomy of the medial part of the knee. J Bone Joint Surg Am. 2007;89(9):2000–10.
12. Smeets K, et al. Mechanical analysis of extra-articular knee ligaments. Part one: native knee ligaments. Knee. 2017;24(5):949–56.
13. Peterson L, et al. Incidence of football injuries and complaints in different age groups and skill-level groups. Am J Sports Med. 2000;28(5 Suppl):S51–7.
14. Fetto JF, Marshall JL. Medial collateral ligament injuries of the knee: a rationale for treatment. Clin Orthop. 1978;132:206–18.
15. Andrews K, Lu A, Mckean L, Ebraheim N. Review: medial collateral ligament injuries. J Orthop. 2017;14(4):550–4.
16. Giannotti BF, Rudy T, Graziano J. The non-surgical management of isolated medial collateral ligament injuries of the knee. Sports Med Arthrosc Rev. 2006;14:74–7.
17. Wilson TC, Satterfield WH, Johnson DL. Medial collateral ligament "tibial" injuries: indication for acute repair. Orthopedics. 2004;27:389–93.
18. Fowler PJ, Snyder-Mackler L. Commentary. J Orthop Sports Phys Ther. 2001;31(11):661.
19. Brandsson S, Karlsson J, Sward L, et al. Kinematics and laxity of the knee joint after anterior cruciate ligament reconstruction: pre- and postoperative radiostereometric studies. Am J Sports Med. 2002;30:361–7.
20. Dienst M, Burks RT, Greis PE. Anatomy and biomechanics of the anterior cruciate ligament. Orthop Clin North Am. 2002;33:605–20.
21. Gans I, et al. Epidemiology of recurrent anterior cruciate ligament injuries in National Collegiate Athletic Association Sports: the injury surveillance program, 2004–2014. Orthop J Sports Med. 2018;6(6).

22. Hirschman HP, Daniel DM, Miyasaka K. The fate of unoperated knee ligament injuries. Knee ligaments: structure, function, injury, and repair. New York: Raven Press; 1990.
23. Andersson C, Odensten M, Gillquist J. Knee function after surgical or nonsurgical treatment of acute rupture of the anterior cruciate ligament: a randomized study with a long-term follow-up period. Clin Orthop Relat Res. 1991:255–63.
24. Grontvedt T, Engebretsen L, Benum P, Fasting O, Molster A, Strand T. A prospective, randomized study of three operations for acute rupture of the anterior cruciate ligament. Five-year follow-up of one hundred and thirty-one patients. J Bone Joint Surg Am. 1996;78:159–68.
25. Wiggins AJ, et al. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction a systematic review and meta-analysis. Am J Sports Med. 2016;44(7):1861–76.
26. Laprade CM, et al. Emerging updates on the posterior cruciate ligament. Am J Sports Med. 2015;43(12):3077–92.
27. Laprade RF, Wentorf F. Acute knee injuries; on-the-field and Sideline evaluation. Phys Sportsmed. 1999;27(10):55–61.
28. Grood ES, Stowers SF, Noyes FR. Limits of movement in the human knee: effect of sectioning the posterior cruciate ligament and posterolateral structures. J Bone Joint Surg Am. 1988;70(l):88–97.
29. Fanelli GC, Beck JD, Edson CJ. Current concepts review: the posterior cruciate ligament. J Knee Surg. 2010;23(2):61–72.
30. Gross ML, Grover JS, Bassett LW, Seeger LL, Finerman GA. Magnetic resonance imaging of the posterior cruciate ligament. Clinical use to improve diagnostic accuracy. Am J Sports Med. 1992;20:732–7.
31. Johnson P, Mitchell SM, Görtz S. Graft considerations in posterior cruciate ligament reconstruction. Curr Rev Musculoskelet Med. 2018;11(3):521–7.
32. Lutz GE, Palmitier RA, An KN, Chao EYS. Comparison of tibiofemoral joint forces during open-kinetic-chain and closed-kinetic-chain exercises. J Bone Joint Surg Am. 1993;75:732–9.
33. Ansari AS, et al. Influence of graft source on postoperative activity and joint laxity in posterior cruciate ligament reconstruction: a systematic review. Arthroscopy. 2019;35(1):262–74.
34. Bedi A, Musahl V, Cowan JB. Management of posterior cruciate ligament injuries: an evidence-based review. J Am Acad Orthop Surg. 2016;24(5):277–89.
35. Nannaparaju M, et al. Posterolateral corner injuries: epidemiology, anatomy, biomechanics and diagnosis. Injury. 2018;49(6):1024–31.
36. LaPrade RF, Wentorf F. Diagnosis and treatment of posterolateral knee injuries. Clin Orthop Relat Res. 2002;402:110–21.
37. Laidlaw MS, Diduch DR. Current concepts in the management of patellar instability. Indian J Orthop. 2017;51(5):493–504.
38. Sherman SL, et al. Return to play after patellar stabilization. Curr Rev Musculoskelet Med. 2018;11(2):280–4.
39. Amis AA, et al. Anatomy and biomechanics of the medial patellofemoral ligament. Knee. 2003;10(3):215–20.
40. Salonen EE, et al. Traumatic patellar dislocation and cartilage injury a follow-up study of long-term cartilage deterioration. Am J Sports Med. 2017;45(6):1376–82.
41. Zheng X, et al. Surgical medial patellofemoral ligament reconstruction versus non-surgical treatment of acute primary patellar dislocation: a prospective controlled trial. Int Orthop. 2019;43(6):1495–501.
42. Longo UG, et al. Treatment of primary acute patellar dislocation: systematic review and quantitative synthesis of the literature. Clin J Sport Med. 2017;27(6):511–23.
43. Lee DY, et al. Which technique is better for treating patellar dislocation? A systematic review and meta-analysis. Arthroscopy. 2018;34(11):3082–93.