Traumatic Knee Injuries

5

Steffen Sauer and Mark Clatworthy

5.1 Patella Dislocation

5.1.1 Background

The patella represents the largest sesamoid bone of the human body and is incorporated into the quadriceps tendon. Patella dislocations are a common sports-related knee injury and defined as the complete disengagement of the patella from the trochlear groove. According to the consistency and associated trauma mechanism, patella dislocations may be subdivided into traumatic, recurrent and habitual dislocations. Traumatic (inaugural/single) dislocations are usually the result of a relevant trauma, typically a pivoting manoeuvre with a twisting movement about the flexed knee [1]. Recurrent (several) patella dislocations usually occur during normal activities and are facilitated by dysplastic changes in the patellofemoral joint [2, 3]. Habitual patella dislocation is defined as the consistent dislocation of the patella whenever the knee is flexed. Predisposing factors of patella dislocation include genu valgum, patella alta, increased distance between the tuberositas tibia and the trochlear groove (TTTG distance) as well as increased internal rotation and anteversion of the femur [1, 4]. The patella usually dislocates laterally, leading to a rupture of the medial patellofemoral ligament (MPFL). Subsequently, the lack of medial retrain supported by the MPFL may lead to patellar instability and recurrent dislocation, especially in cases with associated bony dysplasia or muscle weakness. Spontaneous reposition is usually seen, otherwise emergency reduction is required.

5.1.2 Symptoms

Symptoms arising from patella dislocation are frequently related to the preceding trauma and type of dislocation. Traumatic first-time patella dislocation usually provokes medial para-patellar pain as a result of capsular disruption which is often followed by rapid onset of effusion. However, effusion may be absent, especially in recurrent dislocations where giving way and locking may be the leading symptoms.

5.1.3 Diagnosis

The diagnosis is typically made with the patella reduced as the vast majority of patella dislocations undergo spontaneous reduction. Clinical

S. Sauer (✉)
Department of Sports Traumatology, Aarhus University Hospital, Aarhus, Denmark
e-mail: stefsaue@rm.dk

M. Clatworthy
Department of Orthopaedic Surgery, Middlemore Hospital, Auckland, New Zealand

F. Margheritini et al. (eds.), *Complex Knee Ligament Injuries*,
https://doi.org/10.1007/978-3-662-58245-9_5

examination will usually reveal pain or a depression upon palpation of the medial retinaculum, usually disrupted during the dislocation. Pain during patellofemoral compression or palpation of the lateral condyle is usually present as a result of the patellofemoral collision during the dislocation. A positive apprehension sign when the examiner attempts to manually reproduce the luxation may be present and is thought to be pathognomonic for patella dislocation. In patients with recurrent patella luxation, patellar maltracking may become evident with a positive J-sign when the knee is extended. Massive effusion is typically seen in association with osteochondral lesions.

5.1.4 Imaging

Radiographs may visualize irregularities in the contour of the lateral femoral condyle and/or the medial patella facet consistent with osteochondral fractures. CT scanning should be performed if osteochondral lesions are suspected. MRI is used to visualize bone bruising as a result of the patellofemoral collision (Fig. 5.1) and identify eventual concomitant ligamentous or meniscal lesions. Furthermore, MRI is used to identify the degree of patellofemoral malalignment including the TTTG distance which has implications for the indication of surgical realignment procedures, such as tibial tubercle osteotomies.

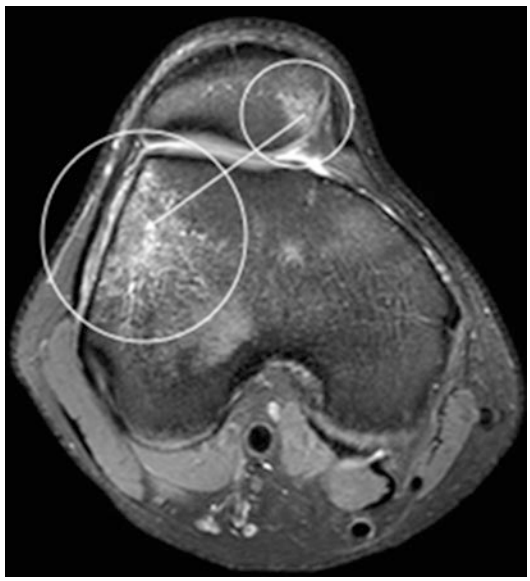

Fig. 5.1 Bone bruising following patellofemoral collision during luxation

5.1.5 Treatment

If the patella is dislocated, emergency reduction in indicated; the knee is hereby extended and the patella is reduced centrally. Following spontaneous or manual reduction of first-time patella dislocation, a conservative treatment approach is indicated [4]. This may entail RICE (rest, ice, compression, elevation), initial knee immobilization and progressive muscle strengthening. Knee arthroscopy may be indicated in the setting of chondral damage or osteochondral lesions that require refixation [1]. In recurrent cases of patella dislocation, surgical stabilization of the patella by either repairing or reconstructing the MPFL is indicated. However, there is no reliable data regarding joint stability or patient satisfaction after either conservative or operative treatment. Procedures that aim for correction of dysplasia as trochleoplasties should be reserved for cases in which basic surgery has failed.

5.1.6 Take-Home Message

The majority of first-time patella dislocations can be treated conservatively. However, acute CT scan is indicated in cases with massive knee effusion to identify eventual osteochondral lesions which are suitable for subacute refixation [1].

5.1.7 Treatment Algorithm

First-time patella dislocation	Conservative treatment
Recurrent patella dislocation	MPFL reconstruction
First-time patella dislocation with osteochondral lesion	Osteochondral refixation if possible and MPFL reconstruction

5.1.8 Facts

- Common injury.
- The vast majority of patella dislocations relocate spontaneously.
- MRI may be useful to identify typical bone bruising from patellofemoral collision when anamnesis is inconclusive.

5.2 Meniscus Injury

5.2.1 Background

Meniscal lesions are among the most common injuries of the knee joint and the most frequent indication for knee arthroscopy. Meniscal lesions are more often degenerative than traumatic in origin and classified after location (medial/lateral, anterior/posterior) and by morphology (longitudinal, radial, horizontal, flap tear type, bucket handle type). Bucket-handle tears are further classified in accordance to location and associated meniscal blood supply from the geniculate artery which has implications for healing potential (white-white zone, red-white zone, red-red zone) [5]. Meniscal lesions of the menisco-capsular junction area (ramp lesions) and the meniscal root area are further classified in accordance to specific injury pattern, which has implications for treatment procedures [6]. The medial meniscus is more frequently injured than the lateral meniscus due to its more rigid tibial and capsular attachment [7]. However, in association with ACL injures, lateral meniscal tears including root injuries are reported to be more frequent compared to medial meniscal tears [8]. The latter, however, may be easily missed during arthroscopy if the posterior menisco-capsular junction area of the medial meniscus is not thoroughly inspected [6]. The medial meniscus does not only account for half of the shock capacity of the medial compartment but does also restrain the tibia from anterior translation [9]. This is the reason why a large number of patients with ACL injuries have concomitant tears of the medial meniscus [7]. The lateral meniscus is more mobile compared to the medial meniscus. As the knee moves, the lateral meniscus moves back and forth across the tibia and is hereby stabilized by menisco-femoral ligaments. As the lateral meniscus can absorb up to 70% of the shock to the lateral compartment of the knee, total lateral meniscectomy is commonly associated with rapid onset of OA. Recently, the role of the lateral meniscus for rotational knee stability has become more evident which has emphasized the importance of meniscal root repairs for better ACLR outcomes [10]. Over time, menisci become more rigid and the incidence of degenerative meniscal tears increases. Especially in association with osteoarthritis, degenerative lesions of the posterior horn of the medial meniscus are frequently seen. There is an emerging body of literature challenging the beneficial effect of partial meniscectomy as a treatment strategy of these lesions [11, 12]. Traumatic meniscal lesions usually arise from a twisting movement about the flexed knee with the ipsilateral foot planted; the resulting compressive and rotational forces cause the meniscus to tear. Degenerative lesions usually show a subtle debut without a history of preceding trauma.

5.2.2 Symptoms

Symptoms arising from meniscal tears are frequently related to location, morphology and origin of the lesion. Purely traumatic meniscal lesions are associated with a sudden onset of focal pain after a rotational knee trauma. However, the degree of instant posttraumatic pain is variable. Patients sustaining small tears without tissue displacement usually proceed with sports activities. Severe tears are associated with more significant pain, and especially in bucket-handle meniscus lesions, hemarthrosis and reduced range of knee motion is usually present. However, a complete bucket-handle lesion may be luxated anteriorly without compromising range of knee motion. Other symptoms of meniscal injury include click sensation, catching and instability due to proprioceptive misinformation caused by the interference of meniscal tissue. Instability may eventually be aggravated by cruciate or collateral ligament injuries. Degenerative

lesions often show a subtle debut of diffuse pain without preceding trauma. The ability to squat is usually compromised. A serous effusion and quadriceps atrophy may be encountered.

5.2.3 Diagnosis

The diagnosis of meniscal tears is based on anamnesis followed by clinical examination. Partial, horizontal and anterior meniscal tears without mechanical interference may present without clinical findings. Meniscal injuries are typically associated with pain upon palpation of the respective joint line and may be aggravated by a variety of meniscal provocation tests. The accuracy of the physical examination is dependent on the type of injury and the observer [13]. Among the most important tests are McMurray's test (pain or popping sensation over the joint line during external tibial rotation under repeated passive flexion/extension) [13], the Steinmann I sign (pain during passive knee rotation) and the Thessaly test [14]. The latter has recently been popularized as loading forces on the menisci are simulated; the patient stands hereby on one leg with the knee flexed while actively rotating the knee and body. Pain or locking constitutes a positive test. As no isolated test is highly conclusive, a combination of meniscal provocation tests is recommended, and multiple positive findings with a history of relevant trauma suggest a meniscal injury. A negative test does not exclude a meniscal lesion. Hemarthrosis and reduced range of motion are commonly seen in association with displaced meniscal tears. However, a displaced bucket-handle tear extending into the anterior horn of the meniscus is associated with little or no loss of extension.

5.2.4 Imaging

MRI represents the main imaging modality for the diagnosis of meniscal tears and shows high sensitivity and specificity. Especially when range of knee motion is compromised, MRI should be performed to visualize meniscal injuries that require immediate attention [15]. Generally, however, MRI findings need to be interpreted in relation to clinical findings. Mucoid degeneration of the meniscus, which is associated with an increased signal from the centre of the meniscus, is a common finding and should not be misinterpreted as a traumatic tear. Recently, the role of MRI in the diagnosis of degenerative meniscus injuries has been challenged as consecutive arthroscopic procedures are rarely indicated.

5.2.5 Treatment

The treatment of meniscal tears should depend on location, morphology, origin as well as the correlation of meniscal injuries with clinical symptoms and associated lesions. Other factors affecting treatment procedures include patient age and activity level. In general, treatment of meniscal lesions should always aim to restore the best possible function of the meniscus, consequently reducing pain [9]. Bucket-handle lesions (Fig. 5.2a, b) with high healing potential should be reinserted, regardless of patient age. Different methods are hereby used including all-inside, inside-out and outside-in suturing techniques, which are mostly dependent on lesion accessibility [16, 17]. Isolated meniscal lesions without mechanical symptoms and chondral erosion may initially be treated conservatively. Partial meniscectomy is indicated for cases in which resection of disfunctional meniscal tissue is believed to optimize meniscal pressure distribution by restoring a sharp meniscal rim, e.g. in flap-tear lesions or radial tears. However, it must be kept in mind that the loss of functional meniscal tissue may enhance chondral degeneration [18, 19] (Fig. 5.3). Unstable meniscal lesions that affect the meniscal root or ramp areas should be addressed, especially in the setting of concomitant ACL injury. Meniscal root avulsions should be reattached through a transtibial tunnel [20, 21]. Unstable meniscal ramp lesions are best addressed with an all-inside or inside-out suturing technique through an additional posteromedial portal [22]. In contrast, degenerative lesions with underlying OA without meniscal displacement and mechanical symptoms

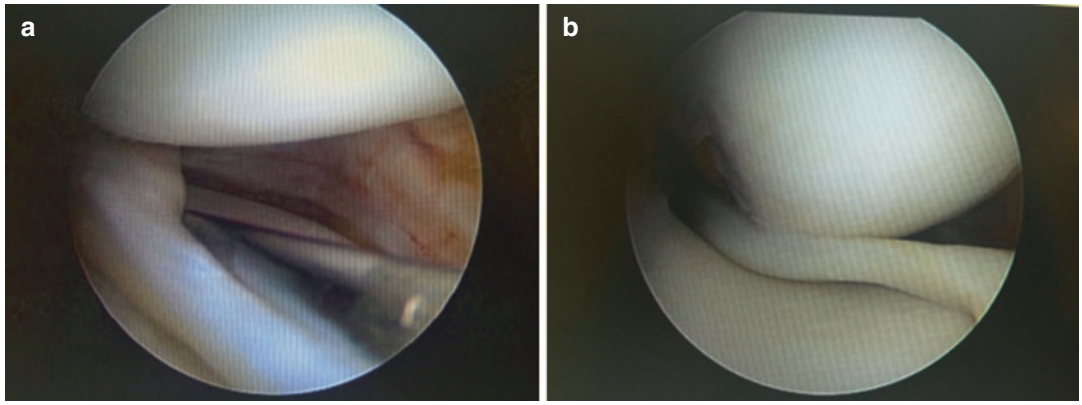

Fig. 5.2 Acute bucket-handle meniscus lesion. (**a**) (left): a small meniscal rim can be seen where the meniscus has been detached from the capsule. (**b**) (right): interfering meniscal tissue lying between the femur and tibia may cause an extension deficit

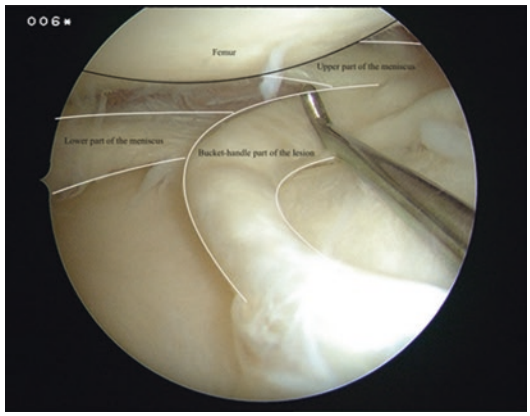

Fig. 5.3 Complex meniscus injury. The central part of the meniscus lies between the femur and tibia as the bucket-handle part of the injury. The peripheral part of the meniscus is torn transversally leaving a lower and upper meniscal portion

should be treated conservatively as no evidence is supporting the beneficial effects of partial meniscectomy [5, 23–26]. Meniscus allograft transplantation is an option for special cases.

5.2.6 Take-Home Message

Patients presenting with the inability to fully extend the knee after a relevant trauma should undergo subacute MRI to identify meniscal injuries that require immediate attention such as bucket-handle lesions.

5.2.7 Diagnosis Algorithm for Acute Meniscal Injuries

Hemarthrosis and reduced range of knee motion	MRI to exclude fracture, patella dislocation, ligament lesions and bucket-handle meniscus injury
Normal range of motion without instability	RICE and re-evaluation after 2–3 weeks

5.2.8 Diagnosis Algorithm for Chronic Meniscal Injuries

Plain radiographs show OA	Conservative treatment
Plain radiographs without OA	MRI to visualize meniscal injuries suitable for surgery

5.2.9 Treatment Algorithm

Degenerative meniscal lesions	Conservative treatment
Bucket-handle meniscal lesions	Subacute arthroscopy and meniscal reinsertion
RAMP lesions	Inside-out or all-inside suture where indicated
Root lesions	Transtibial root refixation

5.2.10 Facts

- The meniscus should always be preserved if possible [15, 27].
- Meniscal surgery is specialist surgery demanding special techniques, especially for root and ramp lesions.
- There is no evidence supporting the beneficial effect of partial meniscectomy in patients with underlying OA.

5.3 ACL Injury

5.3.1 Background

The anterior cruciate ligament (ACL) enables stable knee kinematics by limiting internal tibial rotation and anterior tibial translation. As the ACL has migrated anteriorly during embryologic development to its more central position, it has preserved synovial coverage. The ACL has been thought to be comprised of two distinguishable bundles: an anteromedial and a posterolateral bundle. Recent and revisited anatomical studies, however, have emphasized the ribbon-like structure of the ACL [28], proposing a new nomenclature in which no longer bundles are distinguished but direct from indirect fibres according to their insertional morphology [29]. Acute ACL ruptures are predominantly the result of a non-contact rotational or hyperextensional trauma (Fig. 5.4) [30, 31]. Altered biomechanics of the ACL-deficient knee may cause symptoms of instability, subsequent meniscal and chondral injury as well as osteoarthritis [32, 33]. ACL reconstruction (ACLR) is preformed to improve knee stability and shows overall satisfactory results and low revision rates. The therapeutic approach to ACL injuries has historically been a dynamic process, entailing repair procedures, augmentations, open and arthroscopic reconstructions as well as a variety of graft choices. Even though arthroscopic procedures are currently chosen over open approaches and reconstructions over repair procedures, the optimal graft choice is still controversially discussed. Meta-analysis of studies comparing the outcomes of ACLR depending on graft choice including auto- and allografts and bone-tendon versus pure tendon grafts have not emerged a specific superior graft [34–41]. The reconstruction of both ACL bundles (double-bundle ACLR) has been proposed by some authors [42, 43]. However, its necessity has been challenged as the procedure is technically more demanding without clearly improving patient outcomes [44, 45]. In single-bundle ACLR, graft positioning has been a matter of debate, especially after the transportal ACLR technique has been established as the gold standard [46–48]. Patients with high grade pivot-shift and habitual ligament laxity have shown higher ACL failure rates. In these patients, additional extra-articular stabilizing procedures as the lateral tenodesis or anterior lateral ligament (ALL) reconstruction have currently been popularised and thought to lower ACLR failure and revision rates [49–52]. In general, the susceptibility of ACL lesions is higher among women compared to men engaged in the same pivoting sports [53–55]. Physiological factors such as neuromuscular control and quadriceps-dominant deceleration, the geometrical shape of the knee joint and hormonal factors are thought to be the explanation of this disparity [47, 48, 56–61].

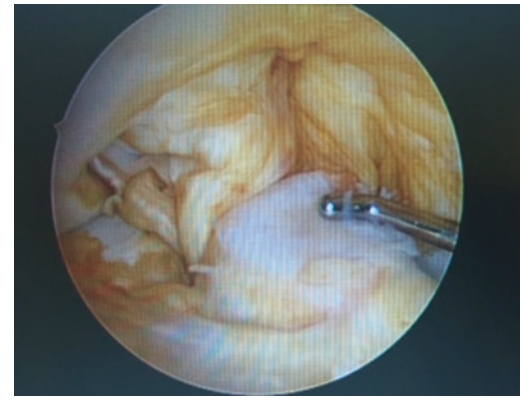

Fig. 5.4 ACL injury: arthroscopic view

5.3.2 Symptoms

Symptoms associated with ACL injury include a hearable snap at the time of injury followed by the inability to continue sports activity and rapid onset of knee effusion. Other symptoms including

pain and reduced range of knee motion may be present and are usually aggravated by concomitant meniscal injuries. Subjective instability with or without giving way phenomenon is usually reported. However, subjective instability may first be evident when pain is resolved and the knee is fully loaded.

5.3.3 Diagnosis

Clinical assessment of the knee is best performed shortly after ACL injury before the onset of muscular guarding. The Lachman's test is referred to as the gold standard [62, 63]; the knee is hereby flexed 20–30°, and the amount of anterior tibial translation and the quality of the translation endpoint are evaluated by pulling the lower leg in a forward direction [64]. The test usually induces less pain and muscular guarding than the anterior drawers test in which the knee is flexed 90° or the pivot-shift test, in which a dynamic subluxation of the tibia is induced [65]. Meta-analysis of the efficacy of these tests finds the Lachman test to be the most useful with sensitivity and specificity of 85% and 94%, respectively [62]. Especially regarding the anterior drawers test, PCL injury may mimic ACL insufficiency as the posterior sag will give the impression of increased anterior tibial translation, when in fact the knee is reduced to its neutral position. The clinical examination should include assessment of concomitant injuries that require immediate attention as bucket-handle meniscal lesions, acute patellar dislocations, MCL lesion, rupture of the popliteus tendon as well as PCL lesions. Hemarthrosis may be evacuated by percutaneous aspiration for pain relief.

5.3.4 Imaging

MRI is used to confirm the diagnosis and visualize concomitant injuries. Plain radiographs may visualize an avulsion fracture of the lateral tibial plateau referred to as the Segond fracture which is usually associated with ACL injury [66, 67]. In addition, plain radiographs may visualize fractures of the intercondylar eminentia which may be suitable for ORIF to avoid ACL insufficiency.

5.3.5 Treatment

Acute management of ACL injury include RICE and oral analgesics. Crutches may initially be indicated to avoid full weight-bearing in cases of severe instability. Further management including the necessity and timing of eventual ACL reconstruction is dependent on several factors as patient age and functional level, degree of instability, the condition of the knee and concomitant injuries. In general, patients with concomitant ligament injury or unstable meniscus lesions usually need surgical reconstruction due to increased instability of the knee [68]. Furthermore, patients who experience significant knee instability wishing to resume high-demand sports or occupation usually benefit from ACL reconstruction. Isolated ACL lesions with stable meniscal lesions may be treated conservatively, especially if return to pivoting sports is not desired [69]. As a general rule, ACL surgery is scheduled 6–8 weeks after the injury when normal range of motion is restored and peripheral structures including MCL lesions have healed. Injuries that need immediate surgical attention (e.g. bucket-handle injuries) should be addressed subacutely. Even though evidence is inconclusive, ACL reconstruction is often postponed until normal range of knee motion is restored to avoid complications including arthrofibrosis [70, 71]. However, there is no consensus among knee surgeons regarding optimal timing of ACLR. According to a systematic review of 69 studies including 7556 participants, 90% of patients undergoing ACL reconstruction achieve normal or near normal knee function. However, only 55% of patients resume their preinjury level of competition [72]. This suggests that psychological factors like fear of reinjury may play an important role in the treatment of ACL injury [72, 73]. There is no cutoff age for ACL reconstruction, and based on observational studies, it shows overall satisfactory results in patients over 40 years of age [74]. Even though rigorous prospective studies are rare, ACL deficiency is thought to be associated with increased risk of chondral and meniscal degeneration [75]. It remains a matter of debate how much the initial trauma itself contributes to progressive joint degeneration and to what extend ACL

reconstruction may modulate this risk [76–78]. In addition, the severity of the initial trauma, extend of meniscal injury, knee biomechanics and subsequent patient activity level may affect the development of joint degeneration.

5.3.6 Take-Home Message

A relevant knee trauma with early onset of effusion is highly suspicious for ACL injury. Muscular guarding may conceal instability, especially concerning the anterior drawers test.

5.3.7 Treatment Algorithm

If ACLR is indicated	Usually 6–8 weeks after injury when range of motion is normalized
ACL rupture with concomitant bucket-handle meniscal injury and fixed flexion deformity	Subacute meniscal suture if indicated, delayed ACLR until range of motion is normalized

5.3.8 Facts

- Up to seventy-seven percent of patients presenting with traumatic knee hemarthrosis have an ACL injury [79].
- ACLR delay may enhance meniscus and chondral degeneration [80].
- ACL deficiency is associated with increased risk of further injury (e.g. meniscal tear), chronic pain and decreased level of activity.
- OA may develop regardless of treatment approach [76, 81].
- No graft choice has been shown to be superior.
- Geometrical features of the knee joint affect ACL injury and ACL graft failure risk [47].

5.4 PCL Injury

5.4.1 Background

The posterior cruciate ligament (PCL) is the strongest of the intra-capsular ligaments of the knee and primary restrain to posterior tibial translation [82]. As the PCL has migrated anteriorly during embryologic development to its more central position, it has preserved synovial coverage. This extra-synovial location accounts for limited effusion in isolated PCL ruptures. The proximity to the posterior capsule, however, ensures blood supply to the PCL after complete rupture, which allows satisfactory results of primary PCL repair and conservative treatment in some cases [83–85]. The femoral and tibial insertion sites of the PCL are approximately three times larger than its mid-portion diameter. The PCL is inconsistently accompanied by two ligamentous structures that stretch between the medial condyle and posterior horn of the lateral meniscus, referred to as the Humphrey and Wrisberg ligament. PCL ruptures may be classified based on timing (acute versus chronic) and severity (isolated versus multi-ligament). Isolated PCL ruptures are rare and usually the result of a fall onto the flexed knee or forced hyperextension. PCL lesions are more likely found in the setting of complex multi-ligament injuries after high-velocity trauma mechanisms. In these cases, thorough assessment of the knee including the neurovascular status is crucial for satisfactory outcomes [86]. Isolated PCL lesions usually show subtle clinical appearance and may therefore go frequently undetected, leading to chronic insufficiency, a flexion deficit or generalized anterior knee pain [87]. The majority of athletes with isolated PCL injuries may continue to function at a high level [83, 84].

5.4.2 Symptoms

Clinical findings of acute PCL ruptures are highly dependent on the preceding trauma and thereof resulting concomitant injuries. Especially lesions of the posterolateral corner may severely aggravate knee instability. In isolated PCL lesions, effusion is uncommon as the blood usually drains into the posterior soft tissues and lower leg. In addition, muscle guarding may conceal mild instability. Pain in the fossa poplitea may therefore be the only perceptible symptom. A popping sensation is rarely reported, and patients with isolated PCL lesions are usually able to instantly resume

sporting activities. In contrast, PCL lesions in the setting of multi-ligament lesions are usually associated with hemarthrosis, severe instability, inability to bear weight as well as reduced range of knee motion. Patients presenting with chronic PCL deficiency suffer from a fixed anterior subluxation of the medial femoral condyle in relation to the tibia, which may cause generalized anterior knee and symptoms related to degeneration of the medial tibiofemoral joint compartment [87, 88].

5.4.3 Diagnosis

Acute and especially isolated PCL lesions may be challenging to diagnose and are frequently overlooked. Spontaneous posterior drawer sign is rare and primarily present with concomitant injury of the posterolateral corner [89]. Anterior-posterior translation is frequently seen in the chronic phase when muscular guarding is overcome [90]. In these cases, a positive posterior drawer test and positive posterior drawer palpation test are typically found (metacarpal joints of the observer's hand react sensitive to hyperextension and may help to identify a spontaneous posterior drawer test). Lesions of the posterolateral corner with rotational instability are assessed with the dial test; the patient is hereby lying prone, and both knees are externally rotated and compared.

5.4.4 Imaging

Plain radiographs may visualize posterior tibial displacement or an avulsion fracture of the tibial PCL insertion site. Subacute MRI is indicated when PCL lesions are suspected. However, MRI does not reveal the functional status of the PCL and degree of instability of the lesion. Treatment is therefore based on anamnesis and clinical findings including the degree of tibial displacement and associated functional instability.

5.4.5 Treatment

Displaced avulsion fractures of the tibial insertion without comminution should undergo ORIF to prevent PCL insufficiency. Arthroscopically assisted procedures and fracture fixation with suspension devises have also shown promising results. Ligamentous lesions should primarily undergo conservative treatment including rehabilitation with a dynamic brace that supports anterior reposition of the tibia during flexion [91]. Results after conservative treatment with mild instability are usually good [84, 92]. PCL reconstruction (PCLR) is indicated in cases of chronic instability [93, 94]. Surgical results after PCLR, especially after severe instability, are worse compared to ACLR outcomes [95–97]. PCL lesions in the setting of a multi-ligament injury should be treated operatively while addressing all injuries in a single operation to ensure early mobilization which is thought to be of paramount importance for satisfactory outcomes [98].

5.4.6 Take-Home Message

Isolated PCL lesions are easily overlooked. Pain in the fossa poplitea after a relevant trauma may be the only symptom.

5.4.7 Treatment Algorithm

Acute isolated PCL	Dynamic bracing
Chronic PCL lesions with mild instability	Physiotherapy
Chronic PCL lesion with distinct instability	PCL reconstruction
PCL injury in the setting of multi-ligament lesions (e.g. PLC)	Multi-ligament reconstruction in a single operation

5.4.8 Facts

- PCL lesions with spontaneous posterior drawer sign are usually associated with lesions of the posterolateral corner (PLC) [89].
- Massive effusion after PCL injury is uncommon as the blood usually drains into the posterior soft tissues and lower leg.
- Isolated PCL lesions show good outcomes when treated conservatively.

5.5 MCL Injury

5.5.1 Background

The medial collateral ligament (MCL) is one of the most commonly injured structures of the knee joint [99, 100]. Partial or complete MCL ruptures are typically the result of a direct valgus trauma, less frequently the result of indirect injury mechanisms including abduction and rotation of the lower leg [101]. MCL injuries usually carry low morbidity, in spite of the complex three-layered medial anatomical structure of the knee [102–104]. The superficial MCL (sMCL) is located within the second layer between the deep MCL and the sartorial facia. The superficial MCL is considered the main static retrain against valgus stress and rotational forces. The deep MCL lies in the third and deepest layer and forms the middle third of the medial capsule [104]. The deep MCL is not ascribed a significant joint-stabilizing function [105]. From the second and third layer originates a conjoint ligamentous structure referred to as the posterior oblique ligament (POL).The deep MCL and posterior oblique ligament have attachments to the medial meniscus which explains why MCL injuries may be associated with medial meniscal tears. MCL injuries are classified into minor stable injuries (grade I), partial injuries with mild instability (grade II) and complete tears with severe instability (grade III). MCL injuries carry low morbidity and tend to heal without complications undergoing the following stages: haemorrhage, inflammation, repair and remodelling. Most patients will resume pre-injury level of competitive sports following conservative treatment. Even though most MCL injuries occur isolated, they may be associated with both ACL and medial meniscus injury, referred to as the unhappy triad. Patients with chronic symptomatic MCL insufficiency should undergo MCL reconstruction [106, 107]. Patients with acute MCL lesions in a severe multi-structural injury setting should undergo repair or reconstruction to ensure early mobilization [106, 108, 109].

5.5.2 Symptoms

Isolated MCL injury is usually associated with pain and periarticular swelling along the course of the MCL. Joint effusion is usually absent and typically indicates concurrent intra-articular structural injury. Concomitant ACL and meniscal injury may aggravate instability and impair range of motion.

5.5.3 Diagnosis

Clinical findings are tenderness along the MCL, predominantly at the femoral insertion site. Instability may be present in 20–30° of flexion indicating MCL insufficiency. Instability in both 20–30° of flexion and extension is usually a sign of combined MCL and ACL injury.

5.5.4 Imaging

Plain radiographs may visualize a bony MCL avulsion. In chronic cases, an osseous irregularity at the femoral insertion site is seen as a result of repetitive trauma, referred to as the Pellegrini-Stieda complex [67, 110]. MRI and ultrasound are used to confirm the diagnosis. Stress radiographs comparing both knees where manual valgus stress is applied may be used to quantify the extent of instability [111, 112].

5.5.5 Treatment

Isolated MCL sprains without valgus instability may be treated conservatively after the RICE principle (rest, ice, compression and elevation) [108]. Early remobilization is encouraged. Grade II and III lesions associated with valgus instability require a coronal stabilizing brace for 5–6 weeks; free range of motion is usually granted. Surgical intervention is rarely indicated as conservative treatment usually shows good results [107]. However, a grade III lesion is often associated with multi-ligament lesions where repair procedures or reconstruction may be

indicated in a multi-ligament reconstruction setting [108, 113, 114]. In chronic cases with ongoing instability, MCL reconstruction is usually indicated [100].

5.5.6 Take-Home Message

Even though isolated MCL injury is frequently seen, thorough assessment of the knee is crucial to correctly identify concomitant meniscal or ligamentous injury.

5.5.7 Treatment Algorithm

Grade I	RICE following early remobilization
Grade II	Coronal stabilizing brace with free range of motion for 6 weeks
Grade III	Often associated with multi-ligament lesions where repair procedures or reconstruction may be indicated

5.6 LCL Injury/Posterolateral Corner Injury

5.6.1 Background

The lateral (fibular) collateral ligament (LCL) stretches from the lateral femoral epicondyle to the anterolateral aspect of the fibular head. Due to its tubular shape and the fact that the axial rotational axis of the knee lies within the medial compartment [115], complete injuries of the LCL usually lead to significant instability and poor conservative healing potential. If undetected or untreated, chronic instability is usually seen, frequently associated with a thrust gait [116, 117]. Isolated LCL lesions are rare and the result of a direct varus trauma. Most frequently, LCL lesions are present in the setting of multi-ligament injuries following high-energy trauma mechanisms [118]. The most common associated injuries are the posterior cruciate ligament, the popliteus tendon and the popliteo-fibular ligament [119]. The latter are referred to as the main static stabilizing structures of the posterolateral corner (PLC) in conjunction with the LCL. Other structures forming the posterolateral corner include the lateral capsule and iliotibial band, the biceps tendon and lateral head of the gastrocnemius muscle as well as variable structures as the arcuate and fabellofibular ligament. Especially following high-energy trauma mechanisms, a fibular avulsion fracture (arcuate fracture) or a common peroneal nerve injury may be present [120].

5.6.2 Symptoms

Pain and swelling along the lateral aspect of the knee is usually found. Complete LCL lesions and associated ACL/PCL or PLC injuries lead to instability near full knee extension [121], typically compounding stair climbing and pivoting manoeuvres.

5.6.3 Diagnosis

Ecchymosis and lateral joint line tenderness may be present and may be aggravated by concomitant meniscal injuries. Coronal stability of the knee is assessed in 20–30° of flexion and extension. Instability in extension and 20–30° of flexion is usually associated with injuries of the posterior capsule and cruciate ligaments. Rotational stability is assessed with the dial test; the patient is hereby lying prone, and both knees are externally rotated at 30 and 90° of flexion. The extent of external rotation is compared to the non-affected side. Rotational asymmetry at 30° but not in 90° indicates an isolated PLC injury. Rotational asymmetry at 30 and 90° indicates a combined PLC and PCL injury. Chronic instability may become evident in a thrust gait.

5.6.4 Imaging

MRI is used to confirm the diagnosis. Plain radiographs may visualize a fibular avulsion fracture of the biceps tendon suitable for subacute refixation. Stress radiographs comparing both knees where manual varus stress is applied may be used to quantify the extent of joint opening and instability.

5.6.5 Treatment

Isolated partial LCL lesions with no varus instability in full knee extension may initially be treated conservatively. This entails RICE (rest, ice, compression, elevation) and functional rehabilitation with a coronal stabilizing brace for 6 weeks. Conservative treatment of complete LCL lesions may result in ongoing varus instability. In these cases, LCL reconstruction is usually indicated. Patients with rotational instability should undergo LCL/PLC reconstruction. Patients with combined ACL/PCL and LCL/PLC insufficiency should undergo multi-ligament reconstruction [118].

5.6.6 Take-Home Message

LCL lesions are frequently associated with lesions of the posterior cruciate ligament and structures of the posterolateral corner (PLC).

5.6.7 Treatment Algorithm

Grade I and II LCL lesion	RICE and coronal stabilizing brace for 6 weeks
Grade III LCL lesion with coronal instability	LCL reconstruction
LCL/PLC lesion with rotational instability	LCL/PLC reconstruction
Multi-ligament setting	Acute reconstruction ±(ACL/PCL/LCL/PCL)

5.7 Multi-Ligament Injury

Multi-ligament injuries are predominantly the result of high-energy trauma mechanisms, and their management require high specialist expertise [122]. Ongoing development in the field of sports traumatology has uncovered a much higher incidence of multi-ligament injuries than initially thought. Thorough assessment of the knee after relevant trauma is crucial to correctly identify the extent of complex multi-ligament injuries [86, 123]. Multi-ligament injuries are typically addressed in a single operation to ensure early mobilization which is thought to be of paramount importance for satisfactory outcomes. In chronic situations, malalignment of the lower limb needs to be addressed before collateral ligament reconstruction is performed. In acute initial management of multi-ligament injuries, the neurovascular status needs to be assessed in accordance to ATLS principles, and CT angiography should always be considered in order to identify vascular injury, especially after knee dislocation [124, 125]. Overlooked vascular injuries are associated with high morbidity and may lead to amputation of the lower limb [120].

5.8 Quadriceps Tendon and Patellar Ligament Rupture

5.8.1 Background

Quadriceps tendon and patellar ligament ruptures may be partial or complete and commonly affect the non-dominant knee of male patients beyond 30 years of age. In younger patients, ruptures are usually the result of a direct trauma. In older patients, the rupture usually represents the final stage of prolonged underlying tendon degeneration. Associated factors which are thought to increase the susceptibility of these ruptures include diabetes, connective tissue disorders, renal failure and the use of intra-articular injections or fluoroquinolone antibiotics [126, 127].

5.8.2 Symptoms

Patients with acute ruptures present with pain and swelling at the rupture site. A popping sensation may be noted at the time of injury, especially in complete ruptures, followed by the inability to continue with sports activity.

5.8.3 Diagnosis

Tenderness and a palpable defect at the rupture site of the quadriceps tendon or patellar ligament is usually found. Knee extension against resistance and a

straight leg rise is usually not possible. Quadriceps tendon rupture is associated with reduction of the patella height, while patellar ligament rupture is associated with elevation of the patella height.

5.8.4 Imaging

Ultrasound and MRI are used to confirm the diagnosis. Plain radiographs may show patella alta in patellar ligament ruptures and patella baja in quadriceps tendon ruptures [128].

5.8.5 Treatment

Partial quadriceps tendon and patella ligament ruptures may be treated conservatively with short-term brace immobilization in full extension with a progressive range of motion and weight-bearing protocol. Complete quadriceps tendon and patellar ligament ruptures should undergo primary end-to-end, trans-osseous or suture anchor repair depending on rupture site location. Tendon reconstruction with auto- or allografts may be necessary in special cases. The use of NSAID for pain management after acute tendon rupture is still a matter of controversy as both beneficial and deleterious effects of NSAID on tendon healing have been reported [129, 130].

5.8.6 Treatment Algorithm

Partial ruptures	immobilization with progressive ROM and weight-bearing
Complete ruptures	repair

References

1. Duthon VB. Acute traumatic patellar dislocation. Orthop Traumatol Surg Res. 2015;101:S59–67.
2. Arendt EA, Fithian DC, Cohen E. Current concepts of lateral patella dislocation. Clin Sports Med. 2002;21:499–519.
3. Zaffagnini S, et al. The patellofemoral joint: from dysplasia to dislocation. EFORT Open Rev. 2017;2:204–14.
4. Frosch S, et al. The treatment of patellar dislocation: a systematic review. Z Orthop Unfall. 2011;149:630–45.
5. Mordecai SC, Al-Hadithy N, Ware HE, Gupte CM. Treatment of meniscal tears: an evidence based approach. World J Orthop. 2014;5:233–41.
6. Chahla J, Dean CS, Moatshe G, Mitchell JJ, Cram TR, Yacuzzi C, LaPrade RF. Meniscal ramp lesions. Orthop J Sports Med. 2016;4(7):232596711665781.
7. Ahn JH, Bae TS, Kang K-S, Kang SY, Lee SH. Longitudinal tear of the medial meniscus posterior horn in the anterior cruciate ligament-deficient knee significantly influences anterior stability. Am J Sports Med. 2011;39:2187–93.
8. Nikolić DK. Lateral meniscal tears and their evolution in acute injuries of the anterior cruciate ligament of the knee. Knee Surg Sports Traumatol Arthrosc. 1998;6(1):26–30.
9. Tscholl PM, Duthon VB, Cavalier M, Menetrey J. Current treatment strategy of meniscal lesions in athletes. Rev Med Suisse. 2016;12:1284–7.
10. Shybut TB, et al. Effect of lateral meniscal root tear on the stability of the anterior cruciate ligament-deficient knee. Am J Sports Med. 2015;43:905–11.
11. Sihvonen R, et al. Arthroscopic partial meniscectomy versus placebo surgery for a degenerative meniscus tear: a 2-year follow-up of the randomised controlled trial. Ann Rheum Dis. 2018;77:188–95.
12. Thorlund JB, Juhl CB, Roos EM, Lohmander LS. Arthroscopic surgery for degenerative knee: systematic review and meta-analysis of benefits and harms. Br J Sports Med. 2015;49:1229–35.
13. Scholten RJ, et al. The accuracy of physical diagnostic tests for assessing meniscal lesions of the knee: a meta-analysis. J Fam Pract. 2001;50:938–44.
14. Karachalios T, Hantes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN. Diagnostic accuracy of a new clinical test (the thessaly test) for early detection of meniscal tears. J Bone Joint Surg Am. 2005;87(5):955–62.
15. Helmark IC, Neergaard K, Krogsgaard MR. Traumatic knee extension deficit (the locked knee): can MRI reduce the need for arthroscopy? Knee Surg Sports Traumatol Arthrosc. 2007;15:863–8.
16. Barber FA, McGarry JE. Meniscal repair techniques. Sports Med Arthrosc Rev. 2007;15:199–207.
17. Fok AWM, Yau WP. Early results of all-inside meniscal repairs using a pre-loaded suture anchor. Hong Kong Med. 2013;J19:124–8.
18. Roemer FW, et al. Partial meniscectomy is associated with increased risk of incident radiographic osteoarthritis and worsening cartilage damage in the following year. Eur Radiol. 2017;27:404–13.
19. van Meer BL, et al. Which determinants predict tibiofemoral and patellofemoral osteoarthritis after anterior cruciate ligament injury? A systematic review. Br J Sports Med. 2015;49:975–83.
20. Bhatia S, LaPrade CM, Ellman MB, LaPrade RF. Meniscal root tears: significance, diagnosis, and treatment. Am J Sports Med. 2014;42:3016–30.

21. LaPrade RF, Matheny LM, Moulton SG, James EW, Dean CS. Posterior meniscal root repairs: outcomes of an anatomic Transtibial pull-out technique. Am J Sports Med. 2017;45:884–91.
22. DePhillipo NN, Cinque ME, Kennedy NI, Chahla J, Geeslin AG, Moatshe G, Engebretsen L, LaPrade RF. Inside-out repair of meniscal ramp lesions. Arthrosc Tech. 2017;6(4):e1315–20.
23. Herrlin S, Hållander M, Wange P, Weidenhielm L, Werner S. Arthroscopic or conservative treatment of degenerative medial meniscal tears: a prospective randomised trial. Knee Surg Sports Traumatol Arthrosc. 2007;15:393–401.
24. Khan M, Evaniew N, Bedi A, Ayeni OR, Bhandari M. Arthroscopic surgery for degenerative tears of the meniscus: a systematic review and meta-analysis. CMAJ. 2014;186:1057–64.
25. Kise NJ, et al. Exercise therapy versus arthroscopic partial meniscectomy for degenerative meniscal tear in middle aged patients: randomised controlled trial with two year follow-up. BMJ. 2016;354:i3740.
26. Ménétrey J, Siegrist O, Fritschy D. Medial meniscectomy in patients over the age of fifty: a six year follow-up study. Swiss Surg. 2002;8:113–9.
27. Stein T, Mehling AP, Welsch F, von Eisenhart-Rothe R, Jäger A. Long-term outcome after arthroscopic meniscal repair versus arthroscopic partial meniscectomy for traumatic meniscal tears. Am J Sports Med. 2010;38:1542–8.
28. Śmigielski R, Zdanowicz U, Drwięga M, Ciszek B, Ciszkowska-Łysoń B, Siebold R. Ribbon like appearance of the midsubstance fibres of the anterior cruciate ligament close to its femoral insertion site: a cadaveric study including 111 knees. Knee Surg Sports Traumatol Arthrosc. 2015;23(11):3143–50.
29. Moulton SG, Steineman BD, Haut Donahue TL, Fontboté CA, Cram TR, LaPrade RF. Direct versus indirect ACL femoral attachment fibres and their implications on ACL graft placement. Knee Surg Sports Traumatol Arthrosc. 2017;25(1):165–71.
30. Alentorn-Geli E, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: mechanisms of injury and underlying risk factors. Knee Surg Sports Traumatol Arthrosc. 2009;17:705–29.
31. Koga H, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. Am J Sports Med. 2010;38:2218–25.
32. Lohmander LS, Englund PM, Dahl LL, Roos EM. The long-term consequence of anterior cruciate ligament and meniscus injuries: osteoarthritis. Am J Sports Med. 2007;35:1756–69.
33. Mihelic R, Jurdana H, Jotanovic Z, Madjarevic T, Tudor A. Long-term results of anterior cruciate ligament reconstruction: a comparison with non-operative treatment with a follow-up of 17-20 years. Int Orthop. 2011;35:1093–7.
34. Carey JL, Dunn WR, Dahm DL, Zeger SL, Spindler KP. A systematic review of anterior cruciate ligament reconstruction with autograft compared with allograft. J Bone Joint Surg Am. 2009;91:2242–50.
35. Cavaignac E, et al. Is quadriceps tendon autograft a better choice than hamstring autograft for anterior cruciate ligament reconstruction? A comparative study with a mean follow-up of 3.6 years. Am J Sports Med. 2017;45:1326–32.
36. Dopirak RM, Adamany DC, Steensen RN. A comparison of autogenous patellar tendon and hamstring tendon grafts for anterior cruciate ligament reconstruction. Orthopedics. 2004;27:837–42; quiz 843–844.
37. Foster TE, Wolfe BL, Ryan S, Silvestri L, Kaye EK. Does the graft source really matter in the outcome of patients undergoing anterior cruciate ligament reconstruction? An evaluation of autograft versus allograft reconstruction results: a systematic review. Am J Sports Med. 2010;38:189–99.
38. Goldblatt JP, Fitzsimmons SE, Balk E, Richmond JC. Reconstruction of the anterior cruciate ligament: meta-analysis of patellar tendon versus hamstring tendon autograft. Art Ther. 2005;21:791–803.
39. Hu J, Qu J, Xu D, Zhou J, Lu H. Allograft versus autograft for anterior cruciate ligament reconstruction: an up-to-date meta-analysis of prospective studies. Int Orthop. 2013;37:311–20.
40. Laoruengthana A, Pattayakorn S, Chotanaputhi T, Kosiyatrakul A. Clinical comparison between six-strand hamstring tendon and patellar tendon autograft in arthroscopic anterior cruciate ligament reconstruction: a prospective, randomized clinical trial. J Med Assoc Thail. 2009;92:491–7.
41. Xie X, et al. A meta-analysis of bone-patellar tendon-bone autograft versus four-strand hamstring tendon autograft for anterior cruciate ligament reconstruction. Knee. 2015;22:100–10.
42. Li X, Xu C, Song J, Jiang N, Yu B. Single-bundle versus double-bundle anterior cruciate ligament reconstruction: an up-to-date meta-analysis. Int Orthop. 2013;37:213–26.
43. Siebold R, Branch TP, Freedberg HI, Jacobs CA. A matched pairs comparison of single- versus double-bundle anterior cruciate ligament reconstructions, clinical results and manual laxity testing. Knee Surg Sports Traumatol Arthrosc. 2011;19(Suppl 1):S4–S11.
44. Kondo E, Yasuda K, Azuma H, Tanabe Y, Yagi T. Prospective clinical comparisons of anatomic double-bundle versus single-bundle anterior cruciate ligament reconstruction procedures in 328 consecutive patients. Am J Sports Med. 2008;36:1675–87.
45. Murawski CD, van Eck CF, Irrgang JJ, Tashman S, Fu FH. Operative treatment of primary anterior cruciate ligament rupture in adults. J Bone Joint Surg Am. 2014;96:685–94.
46. Brophy RH, et al. Changes in the length of virtual anterior cruciate ligament fibers during stability testing: a comparison of conventional single-bundle reconstruction and native anterior cruciate ligament. Am J Sports Med. 2008;36:2196–203.

47. Sauer S, English R, Clatworthy M. The ratio of tibial slope and meniscal bone angle for the prediction of ACL reconstruction failure risk. Surg J. 2018;04(03):e152–9.
48. Clatworthy M, Sauer S, Roberts T. Transportal central femoral tunnel placement has a significantly higher revision rate than transtibial AM femoral tunnel placement in hamstring ACL reconstruction. Knee Surg Sports Traumatol Arthrosc. 2018; https://doi.org/10.1007/s00167-018-5036-x.
49. Guenther D, et al. The role of extra-articular Tenodesis in combined ACL and anterolateral capsular injury. J Bone Joint Surg Am. 2017;99:1654–60.
50. Ramesh R, Von Arx O, Azzopardi T, Schranz PJ. The risk of anterior cruciate ligament rupture with generalised joint laxity. J Bone Joint Surg Br. 2005;87:800–3.
51. Slette EL, et al. Biomechanical results of lateral extra-articular Tenodesis procedures of the knee: a systematic review. Art Ther. 2016;32:2592–611.
52. Weber AE, et al. Lateral augmentation procedures in anterior cruciate ligament reconstruction: anatomic, biomechanical, imaging, and clinical evidence. Am J Sports Med. 2018:363546517751140. https://doi.org/10.1177/0363546517751140.
53. Mountcastle SB, Posner M, Kragh JF, Taylor DC. Gender differences in anterior cruciate ligament injury vary with activity: epidemiology of anterior cruciate ligament injuries in a young, athletic population. Am J Sports Med. 2007;35:1635–42.
54. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. Art Ther. 2007;23:1320–5.e6.
55. Toth AP, Cordasco FA. Anterior cruciate ligament injuries in the female athlete. J Gend Specif Med. 2001;4:25–34.
56. Hewett TE, Myer GD, Zazulak BT. Hamstrings to quadriceps peak torque ratios diverge between sexes with increasing isokinetic angular velocity. J Sci Med Sport. 2008;11:452–9.
57. Hewett TE, Zazulak BT, Myer GD. Effects of the menstrual cycle on anterior cruciate ligament injury risk: a systematic review. Am J Sports Med. 2007;35:659–68.
58. LaPrade RF, Burnett QM. Femoral intercondylar notch stenosis and correlation to anterior cruciate ligament injuries. A prospective study. Am J Sports Med. 1994;22:198–202; discussion 203.
59. Shelbourne KD, Davis TJ, Klootwyk TE. The relationship between intercondylar notch width of the femur and the incidence of anterior cruciate ligament tears. A prospective study. Am J Sports Med. 1998;26:402–8.
60. Wordeman SC, Quatman CE, Kaeding CC, Hewett TE. In vivo evidence for tibial plateau slope as a risk factor for anterior cruciate ligament injury: a systematic review and meta-analysis. Am J Sports Med. 2012;40:1673–81.
61. Sauer S, Clatworthy M. The effect of medial tibial slope on anterior tibial translation and short-term ACL reconstruction outcome. Surg J. 2018;04(03):e160–3.
62. Benjaminse A, Gokeler A, van der Schans CP. Clinical diagnosis of an anterior cruciate ligament rupture: a meta-analysis. J Orthop Sports Phys Ther. 2006;36:267–88.
63. van Eck CF, van den Bekerom MPJ, Fu FH, Poolman RW, Kerkhoffs GMMJ. Methods to diagnose acute anterior cruciate ligament rupture: a meta-analysis of physical examinations with and without anaesthesia. Knee Surg Sports Traumatol Arthrosc. 2013;21:1895–903.
64. König DP, Rütt J, Kumm D, Breidenbach E. Diagnosis of anterior knee instability. Comparison between the Lachman test, the KT-1,000 arthrometer and the ultrasound Lachman test. Unfallchirurg. 1998;101:209–13.
65. Lane CG, Warren R, Pearle AD. The pivot shift. J Am Acad Orthop Surg. 2008;16:679–88.
66. Goldman AB, Pavlov H, Rubenstein D. The Segond fracture of the proximal tibia: a small avulsion that reflects major ligamentous damage. AJR Am J Roentgenol. 1988;151:1163–7.
67. Lee CH, et al. Osseous injury associated with ligamentous tear of the knee. Can Assoc Radiol. 2016;J67:379–86.
68. Lorbach O, et al. The influence of the medial meniscus in different conditions on anterior tibial translation in the anterior cruciate deficient knee. Int Orthop. 2015;39:681–7.
69. Monk AP, et al. Surgical versus conservative interventions for treating anterior cruciate ligament injuries. Cochrane Database Syst Rev. 2016;4:CD011166.
70. Duquin TR, Wind WM, Fineberg MS, Smolinski RJ, Buyea CM. Current trends in anterior cruciate ligament reconstruction. J Knee Surg. 2009;22:7–12.
71. Mayr HO, Weig TG, Plitz W. Arthrofibrosis following ACL reconstruction--reasons and outcome. Arch Orthop Trauma Surg. 2004;124:518–22.
72. Ardern CL, Taylor NF, Feller JA, Webster KE. Fifty-five per cent return to competitive sport following anterior cruciate ligament reconstruction surgery: an updated systematic review and meta-analysis including aspects of physical functioning and contextual factors. Br J Sports Med. 2014;48:1543–52.
73. Czuppon S, Racette BA, Klein SE, Harris-Hayes M. Variables associated with return to sport following anterior cruciate ligament reconstruction: a systematic review. Br J Sports Med. 2014;48:356–64.
74. Legnani C, Terzaghi C, Borgo E, Ventura A. Management of anterior cruciate ligament rupture in patients aged 40 years and older. J Orthop Traumatol. 2011;12:177–84.
75. Sri-Ram K, Salmon LJ, Pinczewski LA, Roe JP. The incidence of secondary pathology after

anterior cruciate ligament rupture in 5086 patients requiring ligament reconstruction. Bone Joint J. 2013;95-B:59–64.
76. Barenius B, et al. Increased risk of osteoarthritis after anterior cruciate ligament reconstruction: a 14-year follow-up study of a randomized controlled trial. Am J Sports Med. 2014;42:1049–57.
77. Frobell RB, et al. Treatment for acute anterior cruciate ligament tear: five year outcome of randomised trial. Br J Sports Med. 2015;49:700.
78. Gillquist J, Messner K. Anterior cruciate ligament reconstruction and the long-term incidence of gonarthrosis. Sports Med. 1999;27:143–56.
79. Hardaker WT, Garrett WE, Bassett FH. Evaluation of acute traumatic hemarthrosis of the knee joint. South Med J. 1990;83(6):640–4.
80. Anderson AF, Anderson CN. Correlation of meniscal and articular cartilage injuries in children and adolescents with timing of anterior cruciate ligament reconstruction. Am J Sports Med. 2015;43:275–81.
81. Culvenor AG, et al. Accelerated return to sport after anterior cruciate ligament reconstruction and early knee osteoarthritis features at 1 year: an exploratory study. PM R. 2017;10(4):349–56. https://doi.org/10.1016/j.pmrj.2017.09.005.
82. Wind WM, Bergfeld JA, Parker RD. Evaluation and treatment of posterior cruciate ligament injuries: revisited. Am J Sports Med. 2004;32:1765–75.
83. Agolley D, Gabr A, Benjamin-Laing H, Haddad FS. Successful return to sports in athletes following non-operative management of acute isolated posterior cruciate ligament injuries: medium-term follow-up. Bone Joint J. 2017;99-B:774–8.
84. Parolie JM, Bergfeld JA. Long-term results of nonoperative treatment of isolated posterior cruciate ligament injuries in the athlete. Am J Sports Med. 1986;14:35–8.
85. Patel DV, Allen AA, Warren RF, Wickiewicz TL, Simonian PT. The nonoperative treatment of acute, isolated (partial or complete) posterior cruciate ligament-deficient knees: an intermediate-term follow-up study. HSS J. 2007;3:137–46.
86. Woodmass JM, et al. Poly-traumatic multi-ligament knee injuries: is the knee the limiting factor? Knee Surg Sports Traumatol Arthrosc. 2017;26(9):2865–71. https://doi.org/10.1007/s00167-017-4784-3.
87. Logan M, Williams A, Lavelle J, Gedroyc W, Freeman M. The effect of posterior cruciate ligament deficiency on knee kinematics. Am J Sports Med. 2004;32:1915–22.
88. Allen CR, Kaplan LD, Fluhme DJ, Harner CD. Posterior cruciate ligament injuries. Curr Opin Rheumatol. 2002;l14:142–9.
89. Sekiya JK, Whiddon DR, Zehms CT, Miller MD. A clinically relevant assessment of posterior cruciate ligament and posterolateral corner injuries. Evaluation of isolated and combined deficiency. J Bone Joint Surg Am. 2008;90:1621–7.
90. Rosenthal MD, Rainey CE, Tognoni A, Worms R. Evaluation and management of posterior cruciate ligament injuries. Phys Ther Sport. 2012;13:196–208.
91. Jansson KS, Costello KE, O'Brien L, Wijdicks CA, Laprade RF. A historical perspective of PCL bracing. Knee Surg Sports Traumatol Arthrosc. 2013;21:1064–70.
92. Fowler PJ, Messieh SS. Isolated posterior cruciate ligament injuries in athletes. Am J Sports Med. 1987;15:553–7.
93. Bedi A, Musahl V, Cowan JB. Management of Posterior Cruciate Ligament Injuries: an evidence-based review. J Am Acad Orthop Surg. 2016;24:277–89.
94. Lopez-Vidriero E, Simon DA, Johnson DH. Initial evaluation of posterior cruciate ligament injuries: history, physical examination, imaging studies, surgical and nonsurgical indications. Sports Med Arthrosc Rev. 2010;18:230–7.
95. Hammoud S, Reinhardt KR, Marx RG. Outcomes of posterior cruciate ligament treatment: a review of the evidence. Sports Med Arthrosc Rev. 2010;18:280–91.
96. Pierce CM, O'Brien L, Griffin LW, Laprade RF. Posterior cruciate ligament tears: functional and postoperative rehabilitation. Knee Surg Sports Traumatol Arthrosc. 2013;21:1071–84.
97. Voos JE, Mauro CS, Wente T, Warren RF, Wickiewicz TL. Posterior cruciate ligament: anatomy, biomechanics, and outcomes. Am J Sports Med. 2012;40:222–31.
98. Harner CD, Höher J. Evaluation and treatment of posterior cruciate ligament injuries. Am J Sports Med. 1998;26:471–82.
99. Craft JA, Kurzweil PR. Physical examination and imaging of medial collateral ligament and posteromedial corner of the knee. Sports Med Arthrosc Rev. 2015;23:e1–6.
100. Duffy PS, Miyamoto RG. Management of medial collateral ligament injuries in the knee: an update and review. Phys Sportsmed. 2010;38:48–54.
101. Schein A, et al. Structure and function, injury, pathology, and treatment of the medial collateral ligament of the knee. Emerg Radiol. 2012;19:489–98.
102. Lundberg M, Messner K. Long-term prognosis of isolated partial medial collateral ligament ruptures. A ten-year clinical and radiographic evaluation of a prospectively observed group of patients. Am J Sports Med. 1996;24:160–3.
103. Lundberg M, Messner K. Ten-year prognosis of isolated and combined medial collateral ligament ruptures. A matched comparison in 40 patients using clinical and radiographic evaluations. Am J Sports Med. 1997;25:2–6.
104. Warren LF, Marshall JL. The supporting structures and layers on the medial side of the knee: an anatomical analysis. J Bone Joint Surg Am. 1979;61:56–62.
105. Narvani A, Mahmud T, Lavelle J, Williams A. Injury to the proximal deep medial collateral ligament: a problematical subgroup of injuries. J Bone Joint Surg Br. 2010;92:949–53.
106. Fanelli GC, Harris JD. Surgical treatment of acute medial collateral ligament and posteromedial

corner injuries of the knee. Sports Med Arthrosc Rev. 2006;14:78–83.
107. Menzer H, Treme G, Wascher D. Surgical treatment of medial instability of the knee. Sports Med Arthrosc Rev. 2015;23:77–84.
108. Smyth MP, Koh JL. A review of surgical and nonsurgical outcomes of medial knee injuries. Sports Med Arthrosc Rev. 2015;23:e15–22.
109. Stannard JP. Medial and posteromedial instability of the knee: evaluation, treatment, and results. Sports Med Arthrosc Rev. 2010;18:263–8.
110. Wang JC, Shapiro MS. Pellegrini-Stieda syndrome. Am J Orthop. 1995;24:493–7.
111. Kurzweil PR, Kelley ST. Physical examination and imaging of the medial collateral ligament and posteromedial corner of the knee. Sports Med Arthrosc Rev. 2006;14:67–73.
112. Laprade RF, Bernhardson AS, Griffith CJ, Macalena JA, Wijdicks CA. Correlation of valgus stress radiographs with medial knee ligament injuries: an in vitro biomechanical study. Am J Sports Med. 2010;38:330–8.
113. Bollier M, Smith PA. Anterior cruciate ligament and medial collateral ligament injuries. J Knee Surg. 2014;27:359–68.
114. Jiang KN, West RV. Management of Chronic Combined ACL medial posteromedial instability of the knee. Sports Med Arthrosc Rev. 2015;23:85–90.
115. Blaha JD, Mancinelli CA, Simons WH, Kish VL, Thyagarajan G. Kinematics of the human knee using an open chain cadaver model. Clin Orthop Relat Res. 2003;410:25–34.
116. Lunden JB, Bzdusek PJ, Monson JK, Malcomson KW, Laprade RF. Current concepts in the recognition and treatment of posterolateral corner injuries of the knee. J Orthop Sports Phys Ther. 2010;40:502–16.
117. Ranawat A, Baker CL, Henry S, Harner CD. Posterolateral corner injury of the knee: evaluation and management. J Am Acad Orthop Surg. 2008;16:506–18.
118. Malone AA, Dowd GSE, Saifuddin A. Injuries of the posterior cruciate ligament and posterolateral corner of the knee. Injury. 2006;37:485–501.
119. Davies H, Unwin A, Aichroth P. The posterolateral corner of the knee. Anatomy, biomechanics and management of injuries. Injury. 2004;35:68–75.
120. Johnson ME, Foster L, DeLee JC. Neurologic and vascular injuries associated with knee ligament injuries. Am J Sports Med. 2008;36:2448–62.
121. Frank JB, Youm T, Meislin RJ, Rokito AS. Posterolateral corner injuries of the knee. Bull NYU Hosp Jt Dis. 2007;65:106–14.
122. Helgeson MD, Lehman RA, Murphy KP. Initial evaluation of the acute and chronic multiple ligament injured knee. J Knee Surg. 2005;18:213–9.
123. Tay AKL, MacDonald PB. Complications associated with treatment of multiple ligament injured (dislocated) knee. Sports Med Arthrosc Rev. 2011;19:153–61.
124. Peskun CJ, et al. Diagnosis and management of knee dislocations. Phys Sportsmed. 2010;38:101–11.
125. Seroyer ST, Musahl V, Harner CD. Management of the acute knee dislocation: the Pittsburgh experience. Injury. 2008;39:710–8.
126. Kim BS, Kim YW, Song EK, Seon JK, Do Kang K, Kim HN. Simultaneous bilateral quadriceps tendon rupture in a patient with chronic renal failure. Knee Surg Relat Res. 2012;24(1):56–9.
127. Omar M, Haas P, Ettinger M, Krettek C, Petri M. Simultaneous bilateral quadriceps tendon rupture following long-term low-dose nasal corticosteroid application. Case Rep Orthop. 2013;2013:1–5.
128. Hockings M, Cameron JC. Patella baja following chronic quadriceps tendon rupture. Knee. 2004;11(2):95–7.
129. Hanson CA, Weinhold PS, Afshari HM, Dahners LE. The effect of analgesic agents on the healing rat medial collateral ligament. Am J Sports Med. 2005;33:674–9.
130. Warden SJ, et al. Low-intensity pulsed ultrasound accelerates and a nonsteroidal anti-inflammatory drug delays knee ligament healing. Am J Sports Med. 2006;34:1094–102.