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Posterolateral Corner Injuries: Epidemiology, Anatomy, Biomechanics and Diagnosis

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Abstract

Increased internal and external rotational laxity of the knee may result from a wide range of pathologies in or around the knee. However, the principal cause of increased external
rotational laxity is damage to the posterolateral corner (PLC). The aim of the review is to discuss the epidemiology, anatomy, biomechanics and diagnosis of PLC injuries.

Keywords: knee
pct
plc
posterior cruciate ligament
postero lateral corner

Epidemiology
The prevalence of PLC injuries is difficult to quantify, partly due to difficulty in diagnosis. An MRI based prospective study [18], analysed patients presenting acutely with knee haemarthrosis following injury. Ligament injuries [4,18] occurred in 187 (56%) of 331 patients. Of these 187 patients, 126 (67%) had isolated ligament injuries and 61 (33%) had combined multiple ligament injuries. Of the 187 patients, 4 (2.1%) had isolated PLC tears. The overall prevalence of posterolateral knee injuries was 16% (30/187) of all knee ligament injuries and 9.1% (30/331) of all acute knee injuries with a haemarthrosis. Of the 30 patients with PLC injuries, 26 (87%) had multiple ligament injuries. Of the 27 patients with PCL tears, 16 (52%) had combined multiple ligament injuries. The rate of isolated PLC injuries was reported as 5% of all knee ligament injuries [5] presenting to a sports injury clinic, which compares to the 2% reported in the MRI study [18]. Using combined examination under anaesthesia and arthroscopy of 61 consecutive patients with acute knee injuries and haemarthrosis presenting to a tertiary referral trauma centre, Fanelli [19] reported that 43 (70%) had an ACL injury and 27 (44%) had a PCL injury. Of the 27 PCL injuries, only 2 (7%) were isolated. The reported frequencies of combined injuries were: PCL and ACL 1
(4%), PCL/ACL/MCL 8 (30%), PCL/ACL/PLC 4 (15%), PCL/PLC 11 (41%), PCL/MCL 1 (4%). Therefore it is commoner for PLC injuries to be part of a multiligament injury pattern with the peripherally located PLC usually injured with a central cruciate ligament [6,7,18,19].

**Mechanism of injury**

Isolated rupture of the PLC can be induced in the laboratory by a direct impact onto the proximal anteromedial tibia, causing combined hyperextension and varus [20]. Clinically, PLC injuries can be due to contact and non-contact hyperextension injury, non-contact varus injury or coupled hyperextension and external rotation [ER] [6,7]. The PLC can be injured in complete knee dislocations [21], often in combination with other ligament injuries [19]. The PLC was injured in over 50% of PCL injuries presenting to a tertiary trauma centre with the majority involving motor vehicle accidents, motorcycle accidents and pedestrians being hit by cars [19]. A retrospective analysis of 494 patients found that the most common injury mechanisms for PCL injuries were dashboard injuries, affecting 173 (35%) patients, and falls on the flexed knee with the foot in plantar flexion, affecting 119 (24%) [22].

**Anatomy**

Understanding of PLC anatomy has been hampered by its complexity and variability [20]. A three layered description (illustration 1 and 2 [78]) of the soft tissue structures on the lateral aspect of the knee has been proposed [23].

**The superficial layer**
The ilio-tibial band (ITB) is connected to the intermuscular septum down to the femoral supracondylar region via Kaplan’s fibers [24]. The ITB continues distally and inserts into Gerdy’s tubercle on the anterolateral aspect of the proximal tibia [24,25]. The thin anterior fibers of the ITB attach to the lateral patella and patellar tendon, joining with fibres from vastus lateralis to form the lateral retinaculum [24,25,26]. Biceps femoris lies posterior to the ITB and has a layered attachment to the styloid process and head of the fibula, and an insertion to Gerdy’s tubercle [25].

The middle layer

The middle layer consists of the vastus lateralis obliquus, deep transverse fibres of the ITB, lateral patellofemoral and the patellomeniscal ligaments [28]. The anatomy of this region, and this particular layer, has had varied descriptions, interpretations and nomenclature [24, 28, 29]. The tendon vastus lateralis obliquus has an oblique direction, passing medially, anteriorly and distally to attach to the lateral border of the patella at an average angle of 38°, providing a posterolateral force vector on the patella [28]. The deep transverse fibres of the ITB can be found between the underside of the ITB proper and the tendon of the vastus lateralis obliquus [28]. The joint capsule in this region shows thickenings or condensations. A thickening connecting the widest part of the lateral border of the patella to the lateral femoral epicondyle is described as the lateral patellofemoral ligament [28] which corresponds to the superior retinaculum or the epicondylopatellar ligament described elsewhere [24, 29]. There is a similar thickening of the capsule connecting the inferior lateral patellar border of the patella to the anterolateral aspect of the lateral meniscus, termed the patellomeniscal ligament [28], which corresponds to the inferior patellar retinaculum described by Kaplan [24].
The deep layer

The deep layer is subdivided into superficial and deep lamina [21,23]. The lateral, or fibular, collateral ligament (LCL) and the fabellofibular ligament are located in the superficial lamina laterally, and the arcuate ligament spreading across the posterior aspect of the lateral femoral condyle [21,23]. The deepest layer contains the popliteus muscle-tendon unit [21,23]. The popliteofibular ligament (PFL) passes distally from the popliteus tendon to the styloid process of the fibula.

Lateral collateral ligament (LCL)

The LCL is a strong, rounded, fibrous cord, attaching to the posterior aspect of the lateral femoral epicondyle, proximal to the groove for the tendon of the popliteus [20,21,23]. Distally the LCL inserts into the anterolateral side of the head of the fibula, anterior and distal to the styloid process and, hence, distal to the insertions of the arcuate ligament and PFL [17,20,21,23].

Fabello-Fibular ligament

The fabella is a sesamoid bone found in the lateral head of the gastrocnemius [23], approximately at the most prominent posterior bulge of the lateral femoral condyle. It is an inconsistent structure which, when present, always has a fabello-fibular ligament running parallel to the LCL, from the fabella to the fibula [23].

3.3.3 Popliteus muscle and tendon complex
This has a dual static and dynamic effect on the knee. The tendon’s attachment is at the proximal fifth of the popliteus sulcus of the distal femur, just antero-distal to the lateral epicondyle. The muscle arises from the proximal posterior aspect of the tibia, above the soleal line [8,9,30,31]. The tendon runs postero-distal from its femoral attachment, passing lateral to the lateral meniscus and then curving down the posterior aspect of the tibia, medial to the fibular head, where it becomes muscular. The tendon gives off three fascicles to the lateral meniscus, which are thought to be important for lateral meniscal stability [20,21]. It also acts as the attachment of the PFL.

*Popliteofibular ligament (PFL)*

The PFL originates from the popliteus tendon superior to the musculotendinous junction [10,32,33,34]. The PFL connects the popliteus tendon to the styloid process at the postero-proximal corner of the head of the fibula. The PFL has been reported as being present in 94% [7] and 98% [34] of cadaver knees, but these figures should be viewed with caution because it can be difficult to see the PFL when it lies deep to the arcuate ligament complex. The PFL is tensed by tibial external rotation, which moves the fibular head posteriorly [20,30].

*Arcuate ligament*

The arcuate ligament arises from the posterior part of the capsule overlying the femoral condyles, the fascia then condenses to become the ligament, which passes laterally and distally over the popliteus tendon and inserts into the posterior aspect of the fibular head [23]. Reported incidence is 80% [23] and 24% [33] in cadaver dissections.
Summarising, the LCL and popliteal muscle complex are present in all knees [11,23]. The reported prevalence of the PFL, arcuate and fabello-fibular ligaments are variable and are summarised in table 1. Seebacher et al [23] suggested that the arcuate ligament and the fabello-fibular ligament have an inverse relationship; the presence of a bony fabella with a ligament greater than 5mm wide was accompanied by an absence of the arcuate and, conversely, the presence of a thin and ill-defined fabello-fibular ligament was accompanied by a well-defined arcuate ligament in over 60% of knees.

The reasons and consequences of the anatomical variability are not well understood. A CT-based study showed that the fibula has a variable position around the tibial plateau [34]. This has been shown to correlate with the orientations of the posterolateral ligaments [31]. Thus, a posteriorly-sited fibula leads to the LCL and PFL complex having postero-distal orientations, so that they will resist tibial external rotation efficiently. Conversely, a laterally-sited fibula will provide efficiently oriented ligaments to resist varus moments [33].
Biomechanics

LCL

The mean length of the LCL has been reported as 70mm [10] and 59.2mm [33]. LCL strength has been measured in vitro at 750N [10] and 309N [33], with the lower reported strength due perhaps to the higher age of specimens tested. The reported [10] cross-sectional area of the LCL is 7.2mm$^2$.

The LCL is the primary restraint to varus of the knee [7,8,20]. Sectioning the LCL increased varus angulation significantly at low flexion angles, compared to intact knees [7,8,20]. Measurement of forces across the LCL during application of varus moments showed that while there were increases at all knee flexion angles, the increase was greatest at 30° flexion [35]. Force measurements for ER moments showed the highest amount of force seen on the LCL was at 0° knee flexion, with the response to ER moments significantly less at 90° [35]. The reducing contribution with knee flexion reflects both the length change pattern - the LCL slackens with knee flexion – and the changing orientation with roll-back of the lateral femoral condyle [33]. Summarising, the LCL is the primary restraint to varus angulation at all flexion angles, and is a primary restraint to ER close to extension.

PFL

The strength of the PFL has been measured from cadaveric specimens as 425N [10] and 186N [33]. The reported [10] cross-sectional area is 6.9mm$^2$, similar to the LCL. Posterolateral damage caused external rotational laxity, maximal at low flexion angles [3,36]. The PFL is the primary restraint to ER at all ranges of flexion [10,32,37], and displays
isometric behaviour throughout flexion [33]. Direct measurement of forces across the popliteus tendon and the PFL during an ER moment found mean popliteus tendon and PFL load response at 0° was significantly less than at 30°, 60° and 90° flexion. In contrast, the LCL showed a higher response at 0° and 30° flexion, matching the length changes noted above [33]. The sectioning and force measurement studies point to a reciprocal relationship between the LCL and the PFL & popliteus tendon complex: the LCL is a primary restraint to ER at low flexion angles, while at greater knee flexion the PFL and popliteus tendon complex becomes the primary restraint.

**PCL**

Isolated sectioning of the PCL produces increased tibial posterior translation at all knee flexion angles, which is small near extension and maximal at 90° flexion. The PCL tends to slacken and becomes a secondary restraint near knee extension, and so the PLC then becomes the primary restraint [20,30,36]. Thus there is a reciprocal action between the PLC near extension and the PCL in the flexed knee [36]. Combined sectioning of both PLC and PCL increases posterior translation at 90° flexion when compared to isolated sectioning of either structure [9,20,30,37,38,39].

Isolated sectioning of the PCL, which is sited centrally, has a negligible effect on tibial ER and varus laxity [20,36,37,39,40,41]. Conversely, isolated sectioning of the PLC increases both ER and varus laxity at both 30° and 90° flexion, but maximally at 30° flexion [20,38,39,41]. Sectioning of both the PLC and PCL produces significantly greater increases in tibial ER at 90° flexion [20,40]. This is a crucial finding which is the basis for the clinical ‘dial test’.
**Clinical Diagnosis**

**History**

Early diagnosis can mean the PLC can be repaired (or primarily reconstructed) with better prognosis, compared to reconstruction in chronic cases [14-16]. PLC injuries are often combined with injuries to other ligaments, particularly the PCL [1,6,7]. The ‘dashboard’ injury which occurs in car crashes is a classic example of this, when the impact onto the flexed knee displaces the tibia posteriorly. In multiligament injuries, the possibility of knee dislocation increases. This can be associated with neurovascular complications and the clinician needs to assess the function of the common peroneal nerve and the functional integrity of the popliteal artery. Associated peroneal nerve damage occurs in 12-29% of PLC injuries [5]. In an acute injury, ecchymosis and haemarthrosis, with posterolateral pain and tenderness may be present [21,39]. The patient may describe instability or giving way, which would be prominent in extension when weightbearing [1,20]. Medial knee pain may be present due to traction of the saphenous nerve from increased tibial ER [39].

**Varus Thrust Gait**

PLC injuries can lead to a ‘varus thrust’ gait during the stance phase of gait [6,42,43]. The ‘triple varus knee’ describes the stepwise increase in knee varus severity which occurs, from normal anatomical tibiofemoral varus to weightbearing through to varus thrust gait [13, 42,43] Primary varus refers to the anatomical tibiofemoral varus seen on standing. Double varus refers to the subsequent opening of the lateral compartment during the stance phase, as the subject’s weight is transferred through the medial compartment. This causes an increase
in the tension of the lateral ligamentous structures. Triple varus or hyperextension varus thrust indicates recurvatum of the knee joint during the stance phase due to the inability of the lateral ligamentous structures to withstand the increased tension in the lateral compartment [42,43].

**PCL**

*Posterior Drawer Test*

The posterior drawer test is carried out with the knee flexed 90° [12,13,15,20]. Normally, at 90° flexion, the distal aspects of the femoral condyles are posterior to the anterior rim of the tibial plateau by approximately 10mm. This is often referred-to as a ‘step-off’. With increasing severity of injury to the PCL, the tibia progressively comes to lay posterior to the femur. In grade I injuries, a palpable step-off remains (5mm) but is smaller than the normal side. In grade II, the anterior tibia and femur are level and the step-off has disappeared. Grade III injuries are when the anterior tibia comes to lie posterior to the femoral condyles, leading to a reverse step-off [12,15]. With a torn PCL, the reverse step-off is greatest at 90° flexion [12,20,36,40]. With combined injuries, posterior step-off will be increased at 30° and 90° flexion [12,20,36,40]. Following sectioning of the PCL in ten paired knees, the posterior drawer increased from grade 0 to grade 2. Additional sectioning of the PLC increased this to grade 3 [44]. Stress radiography of posterior drawer showed laxity of average 2.9mm in intact knees, 12.7mm in PCL-sectioned and 22.3mm in combined PCL-PLC deficient knees [44]. Hewett et al [45] showed posterior translation of >8mm on stress radiography suggests PCL tears in cadavers and clinical subjects. Schulz et al [46] utilised stress radiography in 1041 patients to conclude >8mm posterior translation is indicative of complete PCL injury.
However, the clinical data is difficult to extrapolate as it wasn’t possible for both sets authors to be certain the patients had isolated PCL injuries, and not combined PCL and PLC injuries [46,47]. Apsingi et al [47] found in 8 cadaver knees using an 80N posterior drawer force, PCL transection increased from 4mm to 6mm at 0° knee flexion and from 6mm to 17mm at 100°. Additional transection of the PLC resulted in significantly increased posterior translation at all angles of knee flexion.

*Reducibility of posterior step-off*

In chronic PCL injuries, Larson [15] emphasised the importance of assessing whether the posterior position of the tibia in relation to the femur can be reduced. Although this can be done clinically, Larson [15] suggested that radiographic confirmation, perhaps using a Telos or similar loading fixture, is prudent because a fixed posteriorly placed tibia needs correction prior to PCL reconstruction, otherwise the knee will be forced to articulate abnormally. The chronically contracted soft tissues may be stretched by progressive splinting prior to reconstructive surgery [48].

*Godfrey Test*

This test is conducted with the subject supine, with the hips and knees flexed 90°, and the weight of the foot supported to allow the muscles to relax. In the presence of PCL deficiency, the tibia sags posteriorly in comparison to the normal side, due to gravity [49].
**Quadriceps active test**

The quadriceps active test is carried out with the knee flexed at 90° with the patient supine and the feet flat on the examination couch. The examiner asks the patient to contract their quadriceps (either voluntarily or by extending the knee to 60° by sliding the foot forward while keeping it on the examination couch). In a PCL-deficient knee the posteriorly translated tibia [5,20,40] reduces anteriorly [50]. This test has been validated using the KT-1000 arthrometer [51] which showed significant anterior translation on active quadriceps contraction in 97% of patients with known PCL deficiency [50]. Additionally, this anterior translation on active quadriceps contraction was absent in normal and ACL-deficient knees. This is important, because the anterior drawer test for ACL deficiency can show false-positive results in the presence of PCL deficiency. In painful knees, quadriceps inhibition can make the test difficult to undertake [52].

**Posterolateral corner (PLC)**

**Varus rotation**

The varus stress test is carried out at 0° (extension) and 30° knee flexion [7,15,17,20], corresponding to the role of the lateral collateral ligament (LCL) as the primary restraint to tibial adduction at these flexion angles [7, 8,20,33]. Isolated PLC injuries result in greatest varus rotation laxity at 30° flexion. Apsingi *et al* [47] found in 8 cadaver knees that isolated PCL sectioning does not increase varus rotation compared to intact knees at 30° or 90° knee flexion. Combined sectioning of PCL and PLC structures, including the LCL, resulted in significant increases in varus rotation; from 4° to 15° at 30° knee flexion and from 4° to 22°
at 90° flexion. Grood et al. [3] used 9 paired cadaver knees to show isolated PCL sectioning did not increase varus laxity. Isolated sectioning of the PLC significantly increased varus laxity from 8° to 16° at 30° knee flexion and from 9° to 16° at 90° flexion. Varus laxity in extension usually indicates an associated ACL and posterior capsular injury [3,11], corresponding to their tightness in extension and their role as a secondary restraint to varus rotation [4,30].

Injuries to individual structures of the PLC, namely the PFL or the popliteus complex, may show an absence of increased varus laxity at either extension or 30° flexion [10,37,40], because their injury is masked by the action of the intact LCL, which is the primary restraint [4].

*External Rotation Recurvatum*

With the patient lying supine, bilateral hallux are grasped to suspend the lower legs clear of the examining couch. It has been suggested that in the presence of a PLC injury, the pathological knee will hyperextend, externally rotate and fall into relative varus angulation [15,53,54]. However, although it has been suggested that an associated cruciate ligament injury is needed for this test to be positive [6,55], this test may be confusing in limbs with more generalised laxity.

*Posterolateral Drawer Test*

The knee is put in 80° flexion and, with the foot externally rotated 15°, a posterior force is applied. A positive test exhibits combined ER and posterior displacement of the tibia, and so the posterior motion of the lateral aspect of the tibial plateau is magnified [53,54]. Although a
study of 100 normal knees examined under anaesthesia (EUA) reported difficulty in quantifying the degree of laxity in this test [55], EUA findings and clinic tests may not directly correlate. Contemporary evidence suggests the posterolateral drawer is likely to be testing, partially at least, for PCL integrity [20,37,39]. The PCL is a secondary restraint to ER at 90° flexion, with the PLC the primary restraint at all flexion angles [37,39]. Therefore, for the posterolateral drawer test to be positive, combined PLC and PCL injury would have to be present [20,37,39]. The test should be performed at 30° and 90° flexion as isolated PLC injuries would have increased laxity at 30° flexion, and compound injuries would have increased laxity at both flexion angles [12,15,20].

Reverse pivot-shift test

The aim of the test is to use the different positions of the iliotibial band (ITB) in variable degrees of knee flexion to cause reduction of a subluxed tibia due to a PCL and PLC injury [11,24,26,56,57]. The reverse pivot-shift is performed with the patient in a supine position and the knee at 90° flexion [1,53,55,57]. The knee is then passively extended under a valgus moment with ER of the foot [1,53,55,57]. At 20-30° flexion, the ITB changes from being a flexor to an extensor [56], and its passive elastic tension causes a sudden reduction of a posteriorly subluxed tibia [1,53,57]. This is essentially a dynamic posterolateral drawer test and for the tibia to be posteriorly subluxed at 90° flexion, a combined PLC and PCL injury would need to be present [20,37,39,76].
**Dial (posterolateral external rotation) test**

The dial test involves the comparison of ER, under an induced moment, at 30° and 90° flexion. Biomechanical evidence suggests that the PLC structures are the primary restraints to ER at all knee flexion angles, but particularly at low flexion angles [20,32,33,35,36,37,39,40]. Additionally, the PCL is a secondary restraint to ER at 90° flexion, hence PCL sectioning, in the presence of a PLC injury, leads to further increases in ER [20,37,39]. In contrast isolated PCL injuries do not show any significant effect on tibial ER because of the intact PLC, the primary restraint [37,39,47]. Therefore, increased ER at all angles indicates PLC injury. Further increased ER at both 30° and 90° knee flexion suggests injury to both the PLC and the PCL. While it is suggested that a left to right difference in ER of $\geq 10^\circ$ is considered pathological [12,13,58], cadaveric data suggests that isolated PLC injuries leads to an average increase in ER of $13^\circ$ at 30° flexion, with an increase of $21^\circ$ at 90° flexion with combined PLC and PCL injuries [20,37,39,47]. In combined PCL and PLC injuries, reducing the posteriorly displaced tibia to an anatomical position, prior to the dial test being undertaken, increases tibial ER, reducing the possibility of misdiagnosing PCL-PLC injuries [59,60]. The dial test can be performed either supine or prone. In the supine position, the thighs can be stabilised individually using the side edge of an examination couch, while an ER torque is applied to the tibia via the foot. The clinician looks for an increase in the amount of external rotation of the foot in relation to the thigh in comparison to the normal side [thigh-foot angle, TFA], or at the amount of ER at the tibial tubercle [patellar-tubercle angle, PTA], while simultaneously feeling for an end-point [61,62]. In the prone position, the examiner can only utilise the side-to-side differences in the TFA [13, 55,62]. However, rotation of the foot, in comparison to rotation of the tibia, is significantly higher [62,63,64], poorly correlated [62,64] and has poor intra- and inter-observer correlation,
meaning side-to-side differences can be inaccurate [62]. Additionally, because most knee reconstruction and arthroscopic procedures are carried out with the patient supine, the prone method for the dial test cannot be easily utilised intra-operatively [62].

The reader should note that, although a positive dial test is usually diagnostic of PLC injury, that may not always be the case. The medial soft tissues, particularly the superficial and deep bands of the medial collateral ligament (MCL), also restrain tibial external rotation [65]. Thus, with a positive dial test, the examiner must confirm whether there is an abnormal posterior motion of the head of the fibula, or anterior motion of the medial aspect of the proximal tibia. The latter situation, an anteromedial laxity, can occur in conjunction with an ACL injury.

**Roentgenograms**

Plain Roentgenograms can occasionally aid the diagnosis of posterolateral corner injuries, and can also rule out other injuries. Possible Roentgenograms include the standard anteroposterior (AP) and lateral views and AP long-leg weight-bearing views. In patients with PLC injuries, as with most ligamentous injuries of the knee, plain radiographs are often normal.

**Knee alignment**

Knee misalignment on standard projections can be diagnostic. Dislocations are obvious on X-ray and can be due to multiple ligamentous injuries. Dislocations may have severe
neurovascular associations as discussed above which require further investigations and treatment. Knee subluxations may be subtle and require a high index of suspicion. The posterior sag of the tibia in relation to the femur, an indication of possible PCL injury, should be looked for on the lateral view [12,15]. A long-leg AP weight-bearing view will allow simultaneous assessment of lateral joint line opening as well as the overall lower limb alignment. This is of particular significance in chronic injuries, where failure to correct bony varus misalignment can lead to early failure of soft tissue reconstruction [48,61].

*Varus thrust*

The varus thrust gait pattern due to PLC injuries can be confused with medial compartment degenerative changes [42], particularly in genu varus and in chronic injuries. An AP weight-bearing Roentgenogram can help to differentiate between normal bony architecture in patients with PLC injuries and those with medial compartment arthritis. However, chronically injured knees may show features of secondary arthritis [42], not limited to one knee compartment. In patients with varus thrust gait patterns, the weight-bearing AP view can also depict the degree of lateral joint line opening [6,66].

*Segond fracture*

The Segond fracture was described in the late 19th century, based on cadaveric studies, by Paul Segond [67]. He initially described a cortical avulsion at the tibial insertion of the lateral capsule in its mid-portion. The Segond fracture is currently taken to include any avulsion fracture involving the proximal tibia in the immediate vicinity of the lateral edge of the tibial plateau [6]. The Segond fracture has been described as a ‘small avulsion that reflects major
ligamentous damage’ [68], due to its presence on radiographs being associated with major knee ligamentous injury. A Segond fracture is strongly associated with ACL injuries, meniscal tears and damage to the posterolateral knee structures [68,69]. Indeed Segond [67] himself described the cortical avulsion occurring due to a combination of internal rotation of the femur and varus stress, a mechanism that can lead to ACL and LCL injuries. The presence of a Segond fracture should alert the clinician to consider the presence of significant ligamentous damage [69,70,71].

Arcuate sign

The arcuate sign is an avulsion fracture of the fibula head [71,72], seen on a standard AP radiograph. The avulsed fracture can be attached to components of all three layers [23] of the soft tissues of the lateral compartment, including the LCL, the biceps femoris and the PFL [71,72]. The arcuate sign is therefore an indicator of posterolateral corner injury and knee instability requiring further investigation. When the traction causes an avulsion of the proximal fibular head, the peroneal nerve may be drawn into the fracture gap [73].

Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging (MRI) is the imaging modality of choice in knee ligament injuries. Yu et al [74] developed thin-slice coronal oblique MRI sequences through the fibular head to supplement their standard MRI knee protocols to aid PLC injury diagnosis. This was developed from cadaveric studies. However their clinical MRI findings could not be validated operatively. LaPrade et al [5] prospectively analysed 20 knees with PLC injuries using MRI. These results were then verified at surgical reconstruction. They found that using
a high powered MRI (>1.5 Tesla) and standard images, supplemented with thin-slice coronal oblique T1-weighted images through the entire fibular head provided high sensitivity and specificity for the identification of individual injured PLC components. Bone bruises in the anteromedial femoral condyle in particular, but also the posterolateral tibial plateau, have been found to be associated with isolated and combined PLC injuries [75].

MRI is used to further evaluate ligamentous injuries where radiographs indicate likely PLC injuries, for example in Segond fractures [68,69] and Arcuate signs [71,72]. Additionally, as PLC injuries are often associated with other injuries, MRI can be used to assess the cruciates and menisci.

Summary

The anatomy and biomechanics of the posterolateral aspect of the knee are complex. Injuries to the PLC often go undiagnosed. They are usually accompanied by ACL or PCL injuries and are often treated only as isolated cruciate injuries. This leads to late presentations and the need for reconstruction, as opposed to early diagnosis and repair or primary reconstruction with better prognosis. Many diagnostic tests are described, but confusion surrounds their significance and purpose. A sound understanding of the epidemiology, anatomy and clinical diagnosis can aid early diagnosis and improved outcomes. The most important diagnostic points are that the PCL controls posterior translation of the centre of the tibial plateau, while the PLC controls tibial rotations, both ER and varus.

Conflict of interest: None
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Illustration 1: Cross-sectional view demonstrating the three layers of the knee and associated anatomic structures of the posterolateral aspect of the knee.
Illustration 2: Lateral view of the posterolateral corner demonstrating relationships of the anatomic structures within the three layers of the knee
Table 1  Variability in the prevalence of structures of the PLC of the knee

<table>
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<tr>
<th></th>
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<th>Sudasna et al [38]</th>
<th>Watanabe et al [31]</th>
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<td>Prevalence of Ligament in Study</td>
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