Anatomy and Biomechanics of the Posterior Cruciate Ligament and Their Surgical Implications

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Anatomy

The knowledge and understanding of the complex anatomy and biomechanical function of the native posterior cruciate ligament (PCL) is vitally important when evaluating PCL injury and possible reconstruction. Many studies have been performed looking specifically at the PCL and its unique relationships to the surrounding structures in the knee. The first section of this chapter serves as a foundation for understanding the complex origin and insertional anatomy, the relationships of the distinct bundles of the PCL within the knee, as well as detailing the neurovascular anatomy as it pertains to the PCL. Advanced imaging of the PCL is also covered in detail.

Gross Anatomy

The PCL is the largest of the intraarticular ligaments and travels from the lateral aspect of the medial femoral condyle to the posterior tibia. The PCL plays an integral role in knee joint stability. The PCL is named due to its insertion on the posterior aspect of the proximal tibia. The PCL originates from a broad, concave, semicircular area along the medial femoral condyle within the intracondylar notch. The PCL inserts into a depression just inferior to joint line, between the two tibial plateaus, and posterior to the tibial spine (Figs. 2.1 and 2.2). This depression is known as the posterior intercondyloid fossa or PCL fossa [1]. The PCL consists of longitudinally oriented collagen fibers which is narrowest in its middle portion and fans out superiorly and to a lesser extent inferiorly [2]. The fibers of the PCL attach to the femoral footprint in a lateral to medial orientation and to the tibial footprint in an anterior to posterior orientation. The average length of the PCL as measured in 44 cadaver knees was 38 mm and the average width within the middle portion was 11 mm [2, 3]. The range of PCL width measurements was higher than that of the PCL length measurements due to variation in intercondylar notch size. Utilizing comparative data from anatomic dissection with radiologic correlation, an Austrian group obtained geometric anatomic data characterizing the dimensions of the anterolateral and posteromedial bundles and their footprints. Using 15 cadaver specimens, they found the overall average length and diameter of the PCL was 37 and 11 mm, respectively [4].

The PCL has a wide variation in shape and size of its femoral attachments, whereas the tibial attachments size and shape are more consistent [5]. The substance of the ligament is made up of two distinct but inseparable bundles that allow for resistance of posterior translation in both extension and flexion. The bundles are named by their position within the femoral footprint/attachment: anterolateral bundle and posteromedial bundle (Fig. 2.3).To help identify these bundles during dissection or arthroscopy, other anatomical landmarks have been identified.

On the femoral side, the medial intercondylar ridge defines the proximal limit of the insertion of the PCL, whereas the medial bifurcate ridge separates the insertion sites of the two bundles (Figs. 2.4 and 2.5) [6]. There is a change in slope as each bundle approaches the femoral insertion site, putting the bundles in different planes when the knee is flexed. The PCL footprint on the femur is made up of approximately

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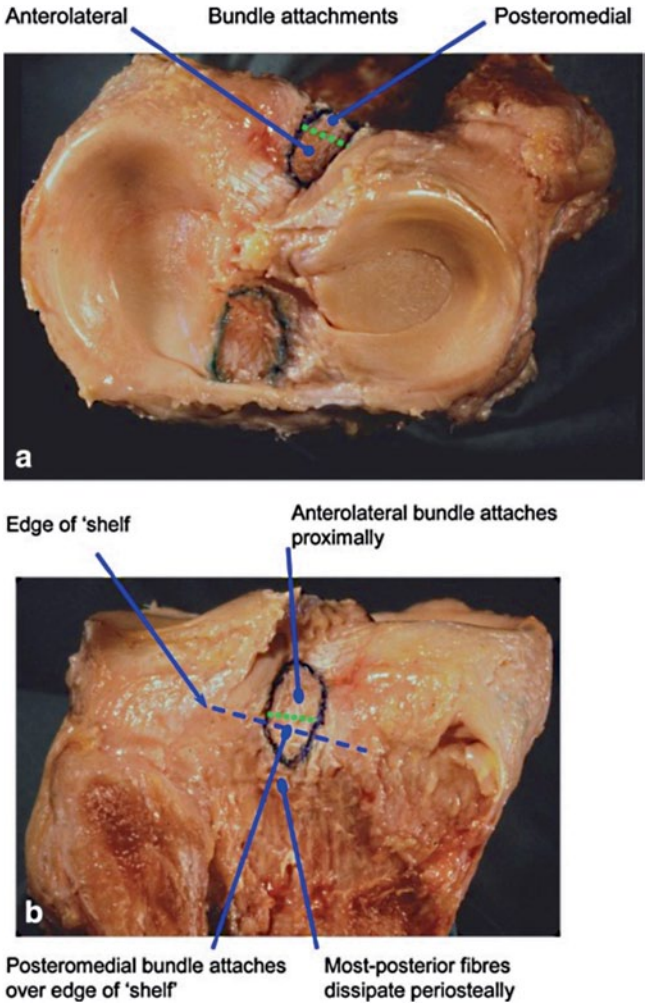

Fig. 2.1 **a** Posterior–anterior view of tibial plateau with PCL attachment marked. Note how the attachment area passes 'over the back. **b** Proximal-distal view of tibial plateau with PCL attachment marked [62]. *PCL* posterior cruciate ligament

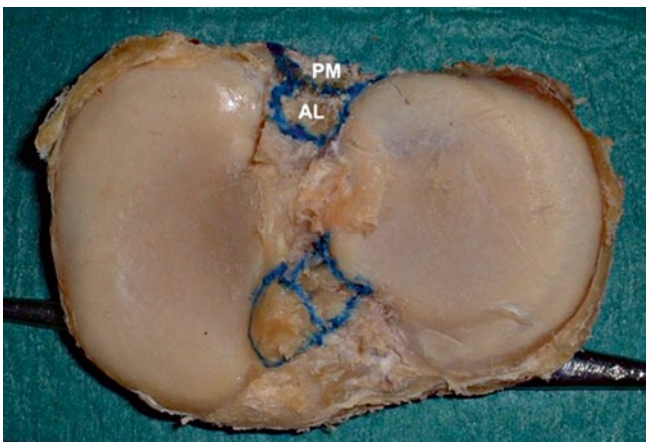

Fig. 2.2 Tibial footprints of the insertions of anterolateral (AL) and posteromedial (PM) bundles of the PCL [63]. *PCL* posterior cruciate ligament

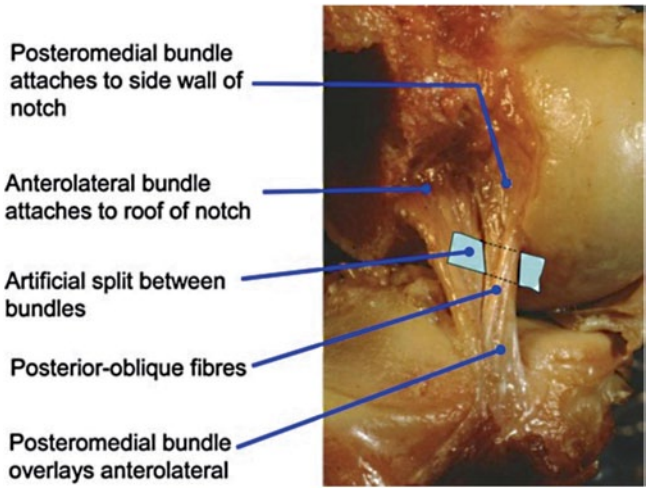

Fig. 2.3 The PCL fibers have been separated into the ALB and the PMB. Posterolateral view of left knee after removal of the lateral femoral condyle [62]. *PCL* posterior cruciate ligament, ALB anterolateral bundle, PMB posteromedial bundle

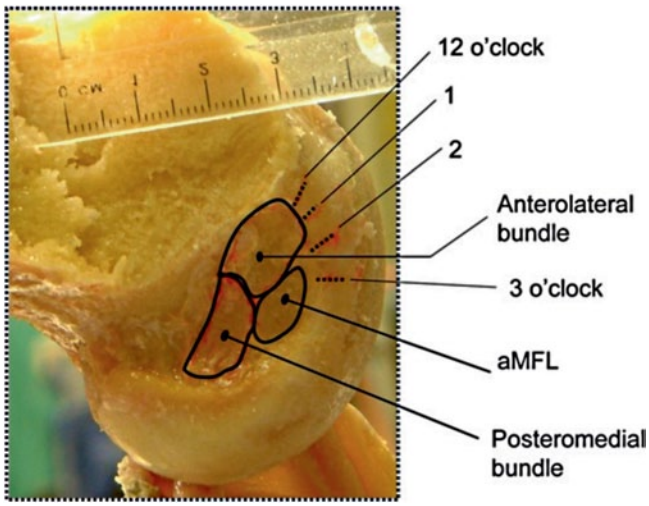

Fig. 2.4 The femoral attachment of the PCL. Lateral–medial view in a left knee after removal of the lateral femoral condyle. The anterolateral and posteromedial bundles of the PCL, plus anterior meniscofemoral ligament attachments are outlined [62]. PCL posterior cruciate ligament

55% anterolateral bundle and 45% posteromedial bundle. The mean distance between the centers of the anterolateral and posteromedial bundles on the femur is 12.1 mm. The distal margins of the anterolateral and posteromedial bundles are a mean 1.5 and 5.8 mm proximal to the notch articular cartilage, respectively [7]. While the femoral footprint size is nearly equal between the two bundles (Fig. 2.6), the anterolateral bundle's crosssectional area is significantly larger than the posteromedial bundle. The anterolateral bundle is the major contributor to PCL strength.

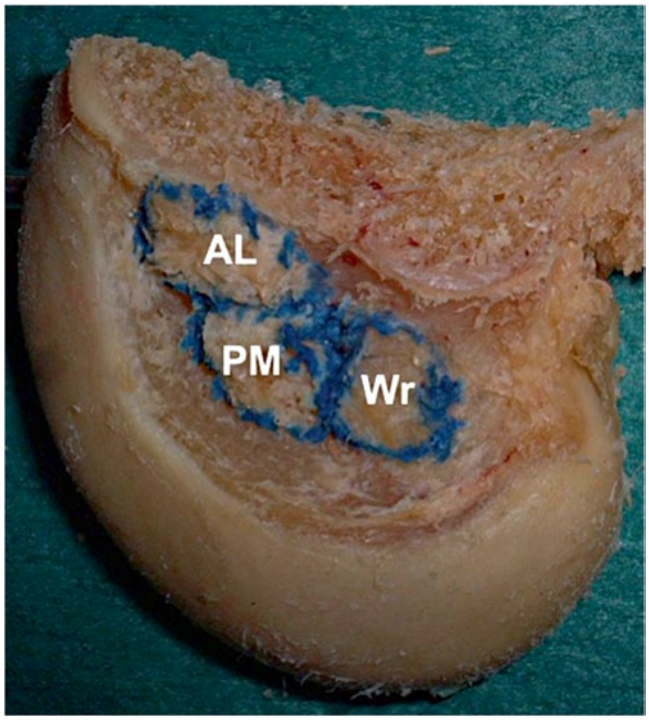

Fig. 2.5. Femoral footprints of the insertions of anterolateral (AL), posteromedial (PM) bundles of the PCL, and Wrisberg ligament (Wr) [63]. *PCL* posterior cruciate ligament

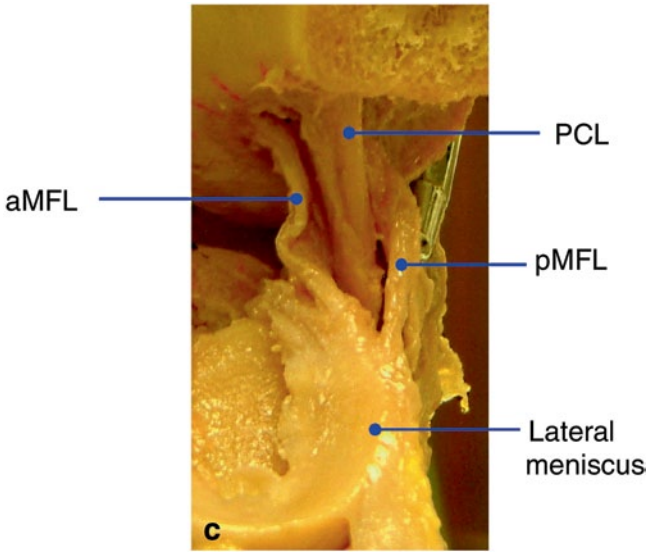

Fig. 2.7 Posterior view of knee showing the PCL attachment on the tibia and accessory ligaments located posteriorly [65]. *PCL* posterior cruciate ligament

The tibial insertions of the anterolateral and posteromedial bundle occur within the PCL fossa which is trapezoidal in shape and becomes wider inferiorly. The anterolateral bundle is attached at the superolateral aspect of the footprint and the posteromedial bundles are seen in the inferomedial portion of the fossa. The identification of each bundle is made easier with each bundle attachment having separate slopes. Across 21 knees, this change in slope angle was found to be an average of 14.5°. Also, an extensive portion of the posteromedial bundle is below the posterior part of the tibial rim, whereas none of the anterolateral bundle attachment is below the tibial rim. The superolateral and superomedial corners of the footprint were both represented by depressions and a reproducible ridge represented the inferior border, all of which could be identified with arthroscopy [8].

Johannsen et al. analyzed the posterior root attachments of the medial and lateral menisci, quantifying their position in relation to the PCL (Fig. 2.7). The lateral meniscus posterior root attachment center was 4.3 mm medial to the lateral tibial plateau articular cartilage edge and directly 12.7 mm to the most anterior edge of the PCL tibial attachment. The medial meniscus posterior root attachment center was 9.6 mm posterior and 0.7 mm lateral from the medial

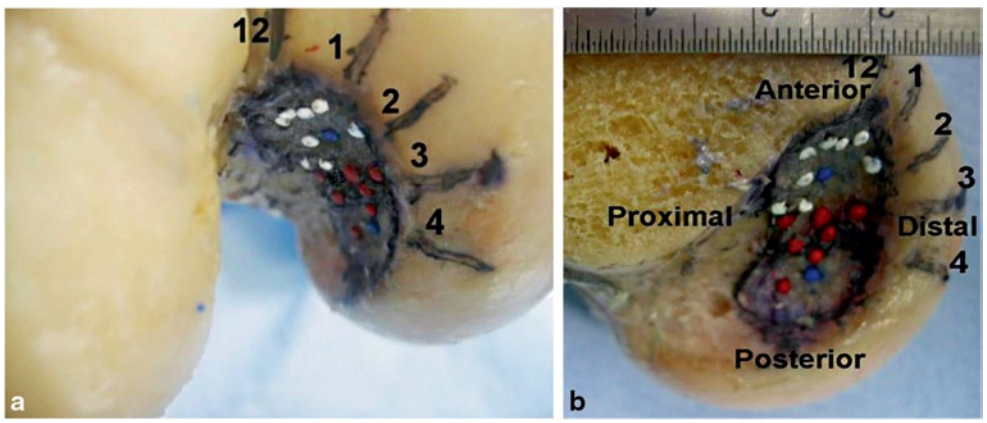

Fig. 2.6. **a** Positions of the anterolateral bundles (*white*) and the posteromedial bundles (*red*) and the anatomical centers of the two bundles of the PCL (*blue*) when viewed from anterior to lateral direction into the intercondylar notch in the knee flexed to 90°. **b** Positions of the anterolateral bundles (*white*) and the posteromedial bundles (*red*) and the anatomical centers of the two bundles of the PCL (*blue*) as seen in the sagittal section to view the medial femoral condyle [64]. *PCL* posterior cruciate ligament

tibial eminence, and 8.2 mm anteromedial from the PCL. This anatomy is very important during PCL reconstruction as nonanatomic tunnel placement may compromise the meniscal root attachment.

The meniscofemoral ligaments are two distinct structures with variable incidence that run from the posterior horn of the lateral meniscus to the lateral aspect of the medial femoral condyle. The ligaments are named based on their location in relation to the PCL. The anterior meniscofemoral ligament is also known as the ligament of Humphrey, while the posterior meniscofemoral ligament is also known as the ligament of Wrisberg. The anterior meniscofemoral ligament is sometimes confused for the PCL during arthroscopy, albeit less than one-third the diameter of the PCL. The posterior meniscofemoral ligament can be nearly half the size of the PCL. Tugging on either of the meniscofemoral ligaments should reveal obvious motion of the lateral meniscus and thus will help you to identify it from the PCL. Multiple research studies looking at cadaver knees found the presence of either the anterior or posterior meniscofemoral ligaments in ~70% of the time. Anderson et al. found that in those knees where both meniscofemoral ligaments were present, the posterior meniscofemoral ligament, posteromedial bundle, and anterior meniscofemoral ligament were aligned parallel to each other, proximally to distally [7]. The posterior meniscofemoral ligament is located directly proximal to the medial intercondylar ridge, proximal to the posteromedial bundle. There are no attachments from the PCL to the medial meniscus.

The posterior joint capsule runs in near continuation with the PCL. The posterior joint capsule originates above the femoral condyles and extends distally to the posterior margin of the tibial plateau. The posterior capsule is within 1–2 mm of the posterior aspect of the tibial attachment of the PCL. The anterior wall of the popliteal artery lies approximately 7–10 mm from the posterior border of the PCL at 90° of flexion [3]. Matava et al. found the distance between the PCL and popliteal artery was maximal at 100° of knee flexion, with measurements of 9.9 mm in the axial plane and 9.3 mm in the sagittal plane, using magnetic resonance imaging (MRI) [9]. There is an anterior septum between the capsule and PCL that is made up of fatty tissue wrapped in a thin synovial membrane which creases a triangular thickening. In the upper third of this tissue is the entry point for the bundle of the middle genicular artery, above the oblique popliteal ligament. Ahn et al. advocate for release of the posterior capsule. They believe it increases the distance between the insertion of the PCL and the popliteal artery, providing an increase in the volume of the posterior compartment during arthroscopy through expansion of this septal tissue. Greater posterior compartment volume enables better viewing of the insertion of the PCL and lowers the risk of neurovascular complication [10].

The blood supply to the PCL comes from the middle genicular artery, a branch of the popliteal artery. The middle genicular artery shows variations in its origin off the popliteal artery. The thin synovial sheath vessels that surround the cruciate ligaments are also seen in the fat pad have been found to be major contributors. These end arteries appear to branch from the middle genicular artery [11]. Capsular vessels supply distal portions of the PCL via branches from the inferior genicular and popliteal arteries [12].

Nerve fibers from the popliteal plexus supply the PCL and its synovial sleeve. The popliteal plexus is derived from the posterior articular nerve and the terminal branches of the obturator nerve [13]. The posterior articular nerve branches from the tibial nerve. A histologic study of mechanoreceptors performed by Katonis et al. observed three types of nerve endings in the PCL: Ruffini corpuscles (type I, pressure receptors), Vater–Pacini corpuscles (type II, velocity receptors), and free nerve endings (type IV, pain receptors) [14]. Golgi tendon organlike structures are found in the PCL origins beneath the synovial sheath and likely play a role in proprioceptive function in the knee [13]. Thus, injury to the PCL creates not only a mechanical disturbance but also a neurologic one by severing the afferent signals to the central nervous system [1].

Congenital deficiency of the PCL is described in association with various congenital longitudinal deficiencies of the lower limb. Albeit rare, PCL deficiency is often seen in conjunction with ACL deficiency. Congenital absence of the cruciate ligaments can result in anterior or posterior dislocation, specifically when undergoing surgery for leg length discrepancy. It is not uncommon for these patients to complain of retropatellar pain as they lock the knee in extension to help provide stability during gait increasing patellofemoral pressure during ambulation [15]. Arthroscopy of these patients often finds a shallow, hypoplastic intercondylar notch, poorly developed or absent tibial spines, and in some, fibular hypoplasia. There has not been good evidence to show that agenesis of the cruciate ligaments is associated with changes in the menisci (i.e., meniscal agenesis or discoid meniscus). Chomiak et al. reported there was little clinical significance to the cruciate deficiency in patients with proximal femoral focal deficiency (PFFD) as the majority of patients did not complain of knee instability during normal daily activities. Prevention of posterior dislocations of the knee is recommended in all lengthening procedures in patients with PFFD, as this complication can be anticipated [16].

Radiographic Anatomy

MRI is widely used to image internal derangements of the knee. To obtain good quality images, an appropriately sized field of view should be used to maximize the resolution. The

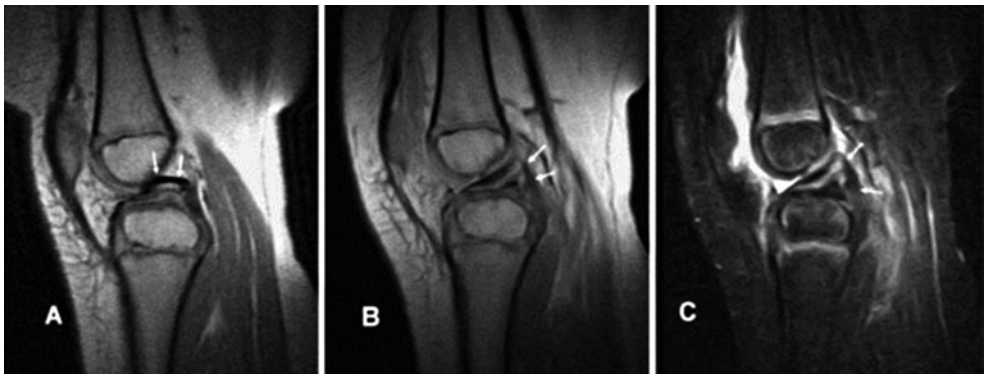

Fig. 2.8 **a** Normal PCL (*arrows*) in the left knee on TSE PD sagittal MR image (TR/TE 1840/21 ms). **b** TSE PD (TR/TE 1840/21 ms) and **c** fat-suppressed TSE T2-weighted sagittal MR images (TR/TE 4430/27, TI 90 ms) shows a thickened, poorly defined PCL with abnormal internal high signal intensity (*arrows*) [66]. PCL posterior cruciate ligament, TSE PD turbo spin-echo proton density

knee should be imaged in three orthogonal planes: axial, coronal, and sagittal planes (Fig. 2.8). On sagittal T2-weighted images, the normal PCL appears as a well-defined uniform band of very low signal intensity. When the knee is in extension, the PCL is lax and it has a gentle posterior convex curvature (Fig. 2.9). The PCL should be found near the midline of the joint in at least 2–3 consecutive images. The meniscofemoral ligaments can often be seen as a small round or oval structure of low signal intensity just anterior or posterior to the PCL. On coronal images, the posterior vertical portion of the PCL is seen in the intercondylar notch, adjacent to the lateral aspect of the medial femoral condyle. The ligament curves forward anteriorly and the horizontal portion appears as a circular or ovoid area of low signal intensity within the intercondylar notch. Axial images are useful in visualizing the vertical portion of the PCL from its tibial insertion [15]. Coronal and axial images can be complementary in the evaluation of the femoral and tibial attachments of the PCL.

The majority of PCL tears are associated with other injuries due to the high level of force necessary to tear the strong PCL fibers. Tears occur most frequently at the middle portion of the PCL. Uncommonly, the PCL may avulse from its tibial attachment. Injuries are best evaluated using sagittal T2-weighted images. A normal intact PCL is a well-defined continuous band of low signal intensity in all pulse sequences. In a complete tear of the PCL, the fibers are discontinuous, with high-intensity fluid signal completely traversing the fibers (Fig. 2.10). Amorphous high signal intensity would also be seen on T1-weighted imaging. In a partial tear, the PCL is thickened, with an increased signal (Fig. 2.11). Avulsion fracture of the PCL is uncommonly seen but would reveal increased signal on T2-weighted images through the posterior portion of the tibia at the level of the PCL attachment. Common associated injuries with PCL tear include ACL tear, anterior bone contusion, collateral ligament injury, and menisci tears [17]. T1-weighted or short tau inversion recovery (STIR) images are valuable in the assessment of associated bone marrow injury [18]. The

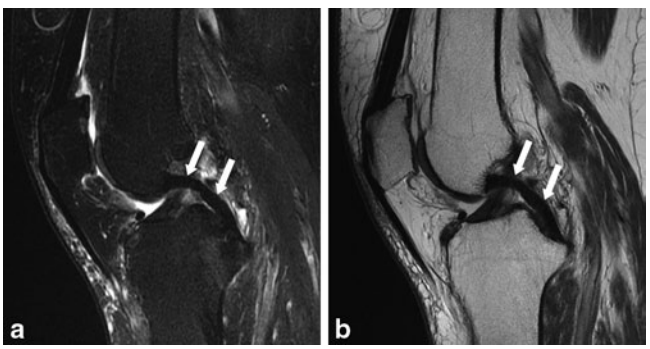

Fig. 2.9 **a** and **b** Demonstrate the normal appearance of the PCL (*arrows*) on T2 and PD images, respectively. Note the normal curved appearance and the homogeneously low signal on both sequences [67]. PCL posterior cruciate ligament, PD proton density

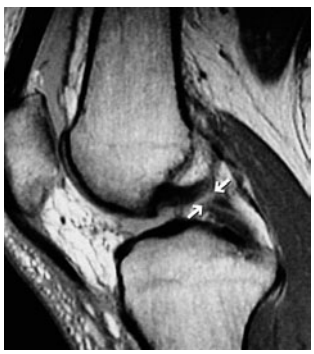

Fig. 2.10 Complete PCL tear. PDWI of a woman in her thirties shows complete tear of PCL at its mid-portion (*arrow*) [68]. PCL posterior cruciate ligament, PDWI proton-density-weighted image

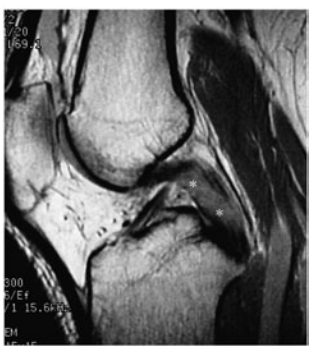

Fig. 2.11 Partial tear of PCL (intrasubstance injury) (1). PDWI of a man in his thirties shows partially torn PCL, which is swollen and shows hyperintensity (*) for the entire length, representing intrasubstance injury. Periphery of the PCL retains continuity of fibers [68]. PCL posterior cruciate ligament, PDWI proton-density-weighted image

sensitivity and specificity for diagnosing a PCL tear by MRI approaches 100 %. MRI has proven to be an accurate modality for evaluating the integrity of the PCL [19].

Biomechanics

Over the past 30 years, the biomechanical role of the PCL has been investigated by many authors. Cadaveric sectioning studies of the PCL, comparison studies with PCL deficiency in one knee and a normal contralateral knee, as well as contact pressure studies and kinematic analysis have all been performed, yielding insight into the multitude of functions the PCL provides in the normal and pathologic knee. We have found that attempting to understand the literature available is best done by collating the studies based on their findings to specific questions. As such, this chapter has been subdivided into various subtopics of PCL biomechanics, each of which examines the present literature and based upon the best available evidence attempts to draw conclusions. The subdivisions are as follows: posterior tibial translation, rotational and medial/lateral stability, joint contact pressure studies, morphological/meniscal/chondral degeneration, strength, proprioception, and compensatory mechanisms: electromyography and muscle activation.

Posterior Tibial Translation

In vitro studies reaffirm the role the PCL has in preventing posterior tibial translation; however, controversy still exists as to the relative translational stability provided by the PCL at varying angles of knee flexion. Kumagai et al. conducted translational measurements in the anterior to posterior plane on five cadaveric specimens before and after PCL sectioning [20]. They found no difference in posterior translation below 25° of knee flexion, while displacement increased significantly between 25° and 90° of flexion, averaging >10 mm of posterior translation at 90° of flexion [20]. In contrast, Li et al. examined eight cadaveric knees and found that in PCL-deficient knees, posterior tibial translation only occurred above 60°; this was reaffirmed by Pearsall et al. where eight cadaveric knees were examined with strain gauges in either meniscus to measure strain in the menisci in varying degrees of flexion in the PCL intact, ruptured, and reconstructive states [21, 22]. They found that in scenarios where the PCL was cut, the total anterior–posterior translation was >18 mm and was statistically significant compared to the intact and reconstructive PCL states at knee flexion angles of 60° and 90° [22].

However, other authors have shown increased posterior tibial displacement in PCL-deficient knees throughout the arc of motion (0–120°) [23–28]. Gollehon et al. published their work in the *Journal of Bone and Joint Surgery* (JBJS) in 1987 where they tested the static stability of the PCL and the posterolateral corner structures in a sectioning study of 17 human cadaveric knees at knee flexion angles between 0° and 90° [23]. They found that at all angles of flexion, the PCL was the principle restraint to posterior translation and that at all angles tested, isolated sectioning of the PCL did not affect varus or external rotation stability [23]. Similarly, Li et al. examined 12 fresh-frozen cadaveric knees using a robotic testing system and applied a 130 N posteriorly directed load at 30° increments between 0° and 150°; they found statistically significant posterior tibial translation at all knee flexion angles except 150°, indicating the role of the PCL in posterior stability except for at extreme flexion angles [24]. Hagemeister et al. reaffirmed the importance of the PCL in providing posterior stability at low flexion angles when they looked at five pairs of fresh-frozen cadaveric knees with a mean age of 73.2 years and used electromagnetic sensors to effectively examine the translation caused by sectioning of the PCL and posterolateral corner structures [25]. Statistically significant differences in anterior–posterior translation existed at all 15° intervals measured from 0° to 75° [25]. Furthermore, Harner et al. examined the effect of the popliteus tendon in conjunction with the PCL in ten cadaveric specimens and found that with a simulated posterior drawer test of 110 N and a simulated popliteus contraction of 44 N in the PCL-intact knees did not result in increased posterior tibial translation; however, when the PCL was sectioned, not only did the posterior tibial translation increase at knee flexion angles of 0°/30°/60°/75°/90° but also a statistically significant decrease in posterior tibial translation was found with the addition of a simulated popliteus muscle contraction [26]. As such, they concluded that the popliteus muscle is an important stabilizer against posterior tibial translation in the PCL-deficient knee [26]. Grood et al. also looked at the effect of tibial translation at low flexion angles of the knee using 15 whole lower limb cadavera and found that sectioning of the PCL did produce posterior tibial sag even at full extension; however, the posterior translation was most apparent at 90° of flexion (the highest flexion angle they measured) where it averaged 11.4 mm of posterior displacement [27]. Finally, Hoher et al. examined eight cadaveric knees using a universal force-moment sensor (UFS) testing system to apply a 110-N load to the knee at 0°/30°/60°/75°/90° [28]. They found that with the application of the load, even at full extension there was a difference in posterior tibial translation of 3.0 mm, which progressively increased to 14.1 mm at 90° of knee flexion [28].

In addition, in vivo studies examining the posterior tibial translation of PCL-deficient knees also exist. Castle et al. examined posterior tibial translation at varying degrees of knee flexion in ten patients with unilateral PCL deficiency using lateral radiographs [29]. They found that at knee flexion

angles between 70° and 90°, the mean displacement of the tibia posteriorly was 7.4 mm, while knee flexion angles between 30° and 50° resulted in a mean displacement of just 2.1 mm [29]. Furthermore, displacement at lower flexion angles was not uniform. Logan et al. also looked at in vivo analysis of the PCL in their series of six patients with unilateral PCL deficiency who underwent MRI evaluation while squatting as well as while a posterior drawer test was performed [30]. Their findings were suggestive of altered kinematics in the medial compartment, with approximately 5 mm of posterior tibial translation measured on the sagittal image throughout all weight-bearing flexion angles [30]. Additionally, in the stress sagittal images obtained at 90° of flexion, a mean of 10.1 mm of posterior tibial translation occurred [30].

Assimilation of the data available in all of these studies would suggest that the PCL is the primary restraint to posterior translation of the knee and that although it likely has an effect throughout the functional arc of motion, its effect is likely greatest at high angles of knee flexion (60–120°). Additionally, the popliteus muscle may act as a restraint to posterior translation in the PCL-deficient knee.

Rotational and Medial/Lateral Stability

The role that the PCL plays in providing rotational stability to the knee is still unclear. Many reports exist throughout the literature that show isolated sectioning of the PCL does not significantly affect tibial external rotation. Gollehon et al. sectioned the PCL in 17 cadaveric specimens and showed that external rotation or varus stress did not increase at any position of knee flexion [23]. Grood et al. performed sectioning of the PCL and posterolateral structures in 15 cadaveric specimens and measured displacement in varying degrees of knee flexion with a electrogoniometer [27]. Their results showed no increased tibial external rotation or varus/valgus angulation compared to the intact state [27]. Nielsen et al. evaluated 25 osteoligamentous-intact cadaveric knees and performed sectioning of the PCL along with the medial and lateral structures [31]. They found that axial rotatory instability was only detectable when the PCL lesion was combined with either a medial or lateral side ligamentous injury; furthermore, a reverse pivot shift was only elicited when the PCL/lateral collateral ligament (LCL)/popliteus tendon were sectioned [31]. Finally, Kaneda et al. looked at 15 cadaveric fresh-frozen knees and performed sequential sectioning of the PCL, LCL, and posterolateral structures and found that in isolated sectioning of the PCL, there was no increase in tibial external rotation; however, they did find that isolated sectioning of just the anterolateral bundle of the PCL shifted the axis of external rotation of the knee [32].

This is in contrast to four other studies, all which showed that isolated sectioning of the PCL does lead to increased tibial rotation. Li et al. examined eight cadaveric knees using a robotic testing system with applied simulated quadriceps and hamstring loads (400 and 200 N, respectively) at knee flexion angles from 0° to 120° and found that at angles above 60° significant external tibial rotation occurred [21]. Harner et al. performed an investigation using ten cadaveric knee specimens where simulated popliteus muscle contraction was performed in both the presence and absence of the PCL [26]. They found that simulated popliteus muscle contraction resulted in an internal tibial rotation of 2° and 4° at 60° and 90° of knee flexion, regardless of whether or not the PCL was intact [26]. Gupte et al. used eight cadaveric knees to evaluate the role of the meniscofemoral ligaments in providing sagittal plane and rotational stability in the posterior cruciate ligament-deficient knee [33]. They found that although isolated sectioning of the PCL does increase rotational instability between 60° and 90°, further sectioning of the meniscofemoral ligaments still does not lead to increased rotational instability [33]. Finally, Ogata et al. performed sequential sectioning of the PCL and collateral ligaments and found increasing posterior sag and internal rotation of the tibia with increasing degrees of knee flexion in specimens in which only the PCL was deficient [34].

Additionally, three in vivo studies examined the varus/valgus and rotational stability provided by the PCL. Fontbote et al. examined ten patients with unilateral grade II (6–10 mm posterior displacement) PCL insufficiency and found objective clinical and radiographic evidence of posterior tibial displacement; however, although differences in gait and vertical landing existed, they concluded that minimal biomechanical and neuromuscular differences were found between PCL-intact and PCL-insufficient knees [35]. Hooper et al. examined nine patients with PCL deficiency compared to a control group in walking and ascending and descending stairs [36]. They found a direct correlation between subjective patient outcome measures (Flandry) and higher peak knee extensor torque during stance phase [36]. Finally, Jonsson and Karrholm looked at eight patients with unilateral PCL deficiency and performed radiostereometric measurements in patients performing a step-up test while a posterior stress test was applied to the tibia at 30° of flexion; this study was unable to show any kinematic differences in the knee during the step-up test [37]. In addition, the study suggested that of the eight patients with known isolated PCL deficiency, six of them were found to show abnormalities in the other ligaments of the knee [37].

In conclusion, the role the PCL plays in the rotational control of the knee is still unclear, with many contradictory studies published in the literature. It may act as a secondary stabilizer to rotational forces when other ligaments are

compromised and other ligaments may provide control to rotation when the PCL is deficient. Further work both in the in vivo and biomechanical arenas may provide further insight into the exact role the PCL plays in providing rotational stability to the knee.

Joint Contact Pressure Studies

To further delineate the biomechanical alterations in the medial compartment after PCL injury, two in vitro studies have been performed to look specifically at this. In the first study, Skyhar et al., in their 1993 paper, looked at the contact pressures using pressure-sensitive film in ten cadaveric knees with sequential sectioning of the PCL and posterolateral corner structures (posterolateral capsule, popliteus muscle and tendon, and the lateral collateral ligament) [38]. They found a mean pressure increase of 52% in the medial compartment, regardless of the angle of knee flexion in specimens with isolated PCL deficiency [38]. Furthermore, pressure increases were also noted in the patellofemoral compartment with progressive sectioning of the PCL and posterolateral corner from an intact state measurement of 23.2 Pa to measurements of 28.0 and 34.8 Pa with subsequent sectioning of the PCL and posterolateral corner, respectively [38]. In the second study, MacDonald et al. used nine fresh-frozen cadavers under the age of 45 years to study the biomechanical changes that occur in the absence of the PCL under physiologic loads [39]. They used pressure-sensitive film inserted into the medial and lateral compartments and measured loads up to 1.5 kN at angles of 0°/30°/60° of knee flexion. Their results did show significant posterior subluxation of the tibia at 60° of flexion in the PCL-deficient specimen, which resulted in increased contact pressure and pressure concentration in the medial compartment [39]. They concluded that this increased contact pressure in the medial compartment of PCL-deficient knees might explain the long-term degenerative changes observed in the medial compartment in PCL-deficient states [39].

Based on these studies, it appears that the deficiency of the PCL results in increased joint contact pressures in the medial and patellofemoral compartments. This contact pressure increase correlates with natural history studies of untreated PCL deficiency where over time, greater incidence of medial and patellofemoral compartment degeneration has been observed.

Morphological/Meniscal/Chondral Degeneration

Hamada et al. looked at 61 patients with acute, isolated PCL tears characterized as grade 2+ or higher and found that 28% of these patients had meniscal tears (with the anterior horn of the lateral meniscus being the most common site of pathology) and 52% of these patients had chondral injuries (most commonly in the medial femoral condyle) [40]. They recommended for the clinician to have a high index of suspicion for concomitant pathology in the menisci or cartilage when evaluating patients with presumed isolated, high-grade PCL injuries.

Ochi et al. have evaluated the ultrastructural changes that occur in the anterior cruciate ligament (ACL) in response to chronic PCL deficiency. They examined 14 patients at a mean of 22.1 months from their isolated PCL injury by obtaining biopsy specimens from the anteromedial and proximal one-third of their knees arthroscopically and compared these via electron microscopy to PCL-intact knees that were obtained secondary to amputation [41]. Interestingly, they found that in the PCL-deficient knees, the ACL had decreased number of collagen fibrils, increased collagen fibril diameter, and decreased collagen packing density compared to controls (PCL-intact knees) [41]. They concluded that isolated PCL deficiency can have adverse effects on other ligamentous structures of the knee.

Shelbourne et al. examined the natural history of the isolated PCL-deficient knees in 68 patients with a mean age at the time of injury of 25.2 years, and obtained subjective, objective, functional, and radiographic data at a mean of 5.4 years postinjury [42]. They found no difference in subjective knee scores and the amount of time from the initial injury; furthermore, laxity did not increase with time and laxity did not correlate with radiographic changes [42]. In addition, regardless of laxity, 50% of this cohort returns to sports at the same level or higher [42]. Radiographic interpretation suggested that medial tibiofemoral compartment arthrosis was more prevalent in the PCL-injured knee compared to the contralateral (normal) knee; however, this did not quite reach statistical significance in this series ($p=0.077$) [42]. Shelbourne et al. most recently provided longer-term follow-up (minimum 10 years) in their 2013 paper published in the *American Journal of Sports Medicine* (AJSM) [43]. Here, they had subjective and objective outcomes data on 44 patients at a mean follow-up of 14.3 years (range 10–21) from the time of injury [43]. Although radiographic changes and progressive degeneration of the knee was seen in 41% of patients, patients maintained quadriceps strength compared to the contralateral side (97% of normal), and had subjective knee scores in the form of International Knee Documentation Committee (IKDC) and modified Cincinnati Knee Rating System (CKRS) scores of 73.4 and 81.3, respectively [43].

Parolie et al. treated 25 patients with isolated PCL tears without surgical reconstruction and followed them up for a mean of 6.2 years (range 2.2–16 years); they found that although 36% had radiographic changes, 80% of patients were satisfied with their knees and 84% had returned to their previous sport [44]. In their study, quadriceps strength

seemed to correlate with patient satisfaction [44]. They concluded that the majority of athletes with PCL-deficient knees who maintain strength in their quadriceps can predictably return to sports without disability.

Keller et al. examined 40 patients with isolated PCL injuries (75 % were sports related) who were treated nonoperatively at a mean of 6 years from the initial injury [45]. On the modified Noyes knee questionnaire, 65 % of patients noted limitations in their activities and 49 % noted that their knee had not fully recovered despite adequate rehabilitation [45]. In contrast to other studies, they did find a correlation between the length of time since the injury and worse knee score and progression of radiographic degenerative changes [45]. Furthermore, 90 % of patients complained of activity-associated knee pain and 43 % had pain with basic activities such as walking—despite having strength measurements essentially the same as the contralateral, uninjured extremity [45].

Boynton et al. examined 38 patients with isolated PCL injuries both subjectively (questionnaire) and objectively (physical exam and radiographs of both knees) at a mean follow-up of 13.4 years (range 5–38 years) [46]. They found that 21 % of patients had to have additional surgery for meniscal pathology and that those patients had statistically significant worse subjective scores than those without meniscal pathology [46]. In addition, 81 % of patients with normal menisci had at least occasional knee pain and 56 % had occasional swelling [46]. Radiographic examination did demonstrate articular degeneration which seemed to increase with time from the injury [46]. They concluded that a bimodal distribution of patients exists, with some having significant symptoms and radiographic degeneration and others remaining essentially asymptomatic with no loss of function.

Strobel et al. published their series of 181 patients with a known PCL injury who had undergone arthroscopy to assess chondral damage in the Arthroscopy journal in 2003 [47]. They found that patients with a duration of PCL deficiency greater than 5 years had an incidence of nearly 78 % for lesions of the medial femoral condyle and nearly 47 % had chondral damage of the patella [47]. Furthermore, they also found that degenerative changes in the medial femoral condyle set in fairly quickly with a threefold increase in the number of lesions within the first year of becoming PCL-deficient; they also found that medial degeneration increased significantly with the presence of a combined PCL/PLC injury [47]. They recommended that the early and continuous increase in both medial compartment and patellofemoral degeneration be taken into account when counseling patients about options for conservative versus reconstructive treatments.

Assimilation of the literature on both acute and chronic PCL tears would suggest that clinicians examining the acute, high-grade PCL tear should have a high index of suspicion for concurrent diagnosis of lateral meniscus tear or medial femoral condyle chondral injury and that failure to diagnose these conditions may miss an opportunity for potentially natural history-altering intervention. Furthermore, deficiency of the PCL may lead to ultrastructural changes in other knee ligaments as they are required to assume additional roles as posterior stabilizers. Predictable sequences of degeneration occur in both the medial and patellofemoral compartments with untreated PCL deficiency. With regard to the outcomes of nonoperative treatment, the literature is mixed with some series providing very compelling evidence for nonoperative treatment of PCL deficiency with high subjective outcomes and return to sports, and other series showing activity-associated knee pain in 90 % of patients with PCL deficiency. We believe that patients with PCL deficiency should be evaluated for concomitant injuries and counseled about the natural history of nonsurgical treatment so that the patient can make an informed decision regarding their care.

Strength

Many authors have examined the effect that PCL injury has on strength of the ipsilateral and contralateral knee. Both Inoue et al. in 1998 and Fontbote et al. in 2005 examined patients with PCL deficiency and found no difference in strength compared to the contralateral, uninjured extremity [35, 48]. In contrast, Hooper et al. in their study on gait adaptations in patients with chronic PCL deficiency showed that peak knee extension torque at 60°/s was significantly less in both the PCL-deficient and contralateral uninjured knee than the control group, leading one to believe that the loss of the strength in the PCL-deficient knee also leads to decreased strength in the contralateral knee [36]. Shirakura et al. have also examined the effect of strength on the PCL-deficient knee and found that a significant decrease in quadriceps eccentric and concentric torque in the PCL-deficient knee occurred only above 36° of flexion [49]. Additionally, MacLean et al. examined 17 patients with isolated PCL injuries that were treated conservatively and found that the PCL-deficient limb was weaker for both the quadriceps and hamstrings compared to the contralateral, normal side [50]. Finally, Tibone et al. looked at isokinetic and isometric quadriceps strength of patients with conservatively treated PCL injuries and found that the PCL-deficient knees exhibited significantly lower quadriceps peak torque at 60°/s but not at 120°/s [51].

However, there have been many prospective studies on quadriceps strength of conservatively treated PCL-deficient knees which found no difference compared to the contralateral, uninjured side [42, 44, 45, 52]. Still, Torg et al. examined 43 patients who had either isolated PCL deficiency or multidirectional knee ligament injuries at a mean of 6.3 years after the injury and did find that 53 % of subjects had

quadriceps strength deficits between 22 and 30% compared to their contralateral, uninjured limb [53].

In conclusion, the present data regarding strength in the PCL-deficient and contralateral normal knee is mixed in the literature, with some studies suggesting significant differences in both lower extremities compared with controls and others suggesting no difference in strength between the PCL-deficient and normal contralateral knee. Further studies will likely need to be performed to draw any definitive conclusions regarding strength.

Proprioception

It has long been postulated that both of the cruciate ligaments play a role in the proprioception of the lower extremity. Clark et al. sought to further delineate the role the PCL has in this when they examined eight patients with PCL deficiency using a motorized apparatus that flexed or extended the knee at a rate of 0.5°/s in a randomized fashion, using the contralateral normal knee as a control [54]. Subjectively, all eight patients noted greater difficulty in perceiving movement in the PCL-deficient knee. In addition, significant differences were found in the threshold of perception to passive movement (TPPM) with the normal knee exhibiting values of 0.93°±0.32° and the PCL-deficient knees having mean values of 1.19°±2.7° [54]. They concluded that this loss of proprioception may play a role in knee instability and be part of the constellation of degenerative changes that occur as part of the natural history of the PCL-deficient knee.

Safran et al. also examined the role that the PCL has in proprioception of the knee. They examined 18 patients with isolated PCL deficiency between 1 month and 19.5 years after injury and evaluated kinesthesia and joint position sense using the threshold to detect passive motion (TTDPM) and the ability to reproduce passive positioning (RPP) [55]. They did find statistically significant differences in TTDPM and RPP between the PCL-deficient and PCL-intact knees; however, this was dependent on the starting position of each joint and whether the knee was flexed or extended [55]. They concluded that there may be proprioceptive mechanoreceptors within the PCL that play a role in proprioception.

In conclusion, there is some evidence that the PCL has a minor role in proprioception of the knee, with small but statistically significant differences noted in the above studies. Although the loss of proprioception has been postulated to be a potential etiology leading to the predictable pattern of medial compartment and patellofemoral degeneration, this has not yet been substantiated in the literature. Finally, to our knowledge there are no studies suggesting that the reconstruction of the PCL-deficient knee restores this proprioceptive role.

Compensatory Mechanisms: Electromyography and Muscle Activation

Inoue et al. performed electromyogram (EMG) studies on the quadriceps, hamstrings, and gastrocnemius muscles on both the PCL-deficient side and the contralateral normal side in 12 patients, while having them perform concentric isokinetic contractions at 30°/s and 60°/s [48]. There were no differences observed in either the quadriceps or hamstring activation between the deficient and control knees; however, prior to generation of flexion torque, EMG revealed a significantly earlier activation of the gastrocnemius muscle at each velocity in the PCL-deficient knees, suggesting that the gastrocnemius may play a role in compensatory stabilization during flexion in PCL-deficient knees [48].

Cain and Schwab published a case study of a football player with PCL deficiency that was subjected to EMG evaluation while running [56]. Their findings suggested that quadriceps contraction occurs 20% earlier in the gait cycle in the lower extremity with PCL deficiency [56]. Tibone et al. also evaluated the compensatory mechanisms involved in a PCL-deficient knee by examining 20 patients (10 with PCL deficiency and 10 with PCL reconstructions) during activities such as walking, running and stair climbing; although they did observe differences such as early activation of the gastrocnemius–soleus complex, the results were not statistically tested [51]. Although relevant to the discussion, it is difficult to draw conclusions from case reports and studies where statistical analysis was not performed.

Finally, Fontbote et al. used surface EMG on ten patients with unilateral PCL deficiency to obtain data on six muscles (vastus medialis and lateralis, medial and lateral hamstring, and both heads of the gastrocnemius) during gait (ten trials) and vertical drop landing on one leg from a height of 30 cm (five trials performed) [35]. The contralateral, normal extremity was used as the control. They found no difference in EMG values for either activity on all of the muscles tested [35].

The relative compensatory contributions from other muscles in the setting of PCL deficiency remain an area where further study needs to be performed to draw any real conclusions. Although one study did show earlier activation in the gastrocnemius muscle during walking, this has not yet been validated by other studies.

Clinical Relevance

Although the biomechanical function of the PCL is known to be primary as a restraint to posterior tibial translation, the true clinical relevance lies in the assessment of whether or not operative reconstruction will allow for more predictable

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