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MRI of the Cruciate Ligaments

Anterior Cruciate Ligament (ACL)

Anatomy and normal appearance

The cruciate ligaments are intracapsular and extrasynovial structures. Both ligaments are enveloped by a fold of synovium that originates from the posterior intercondylar portion of the knee.[1] The anterior cruciate ligament provides restraint against anterior subluxation of the tibia. It is composed of an anteromedial bundle and a posterolateral bundle that twist on each other, thus providing reciprocal tautness as the knee moves from extension to flexion. The ACL extends from the roof of the intercondylar notch to the tibial plateau anterior to the lateral tibial spine. It has a straight course paralleling, or slightly steeper than, the intercondylar roof (known as Blumensaat's line on lateral radiographs) (Fig. 1).

On sagittal images the ligament has a linear striated appearance with intermediate or high signal intensity due to fat and connective tissue between fascicles (Fig. 2).The anterior margin may be darker and thicker than the rest of the ligament, corresponding anatomically to the anteromedial bundle (AMB). The longer and stronger anteromedial bundle tightens during flexion of the knee, whereas the smaller and shorter posterolateral bundle (PMB) tightens during extension of the knee.[3] Although it is not generally possible to distinguish between the two ACL bundles on sagittal sequences, they can be distinguished on other imaging planes (Fig. 3).

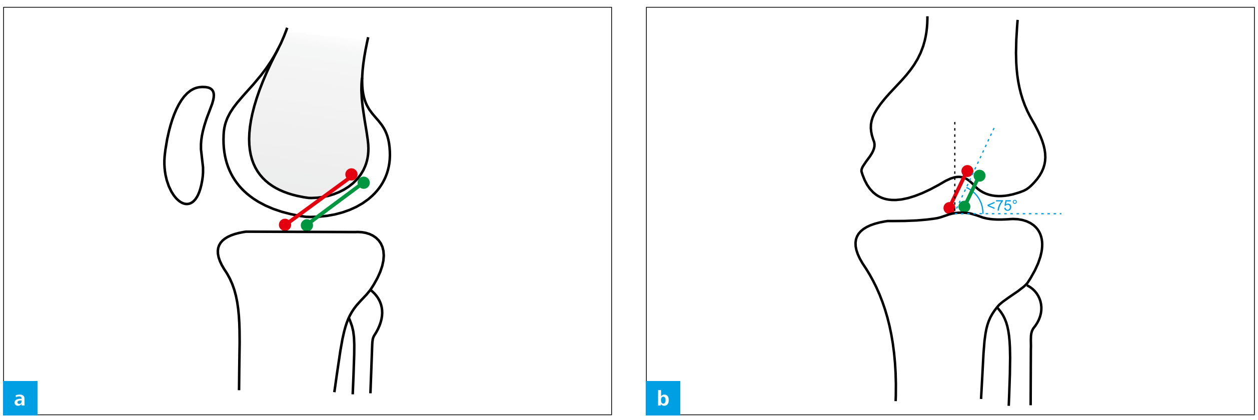


Fig. 1: Blumensaat line on a schematic diagram. The anteromedial bundle is red and the posterolateral bundle is green. In the sagittal plane (a) the ACL graft should course along the Blumensaat line, and the tibial tunnel should open posterior to the intercondylar region. In the coronal plane (b) the ACL graft should be oriented less than 75° relative to the tibial plateau.

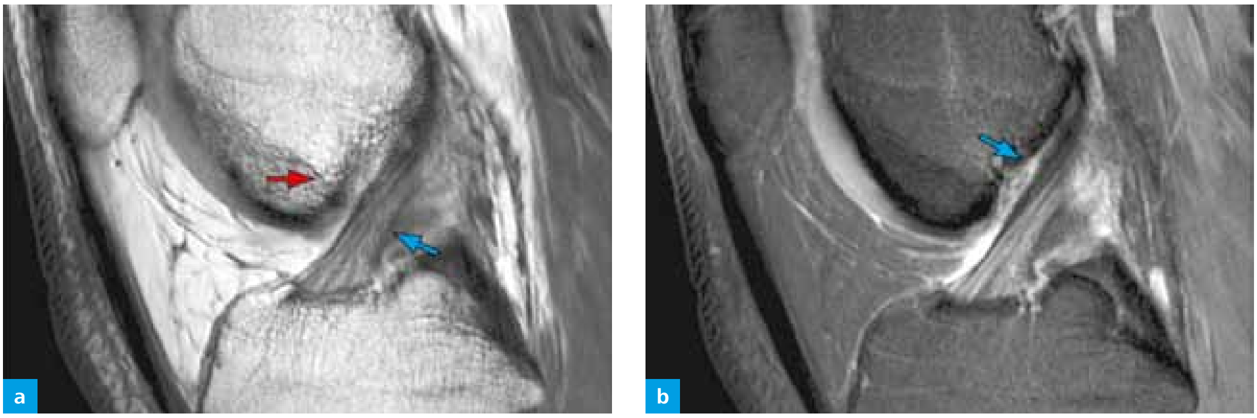


Fig. 2: Normal ACL. Sagittal proton density (a) and sagittal with fat saturation (b), (a) normal ACL (arrow) shows the typical striated appearance. Blumensaa's line (red arrow) representing the roof of the intercondylar notch is also seen. The ACL courses parallel to the line. (b) The anteromedial bundle is sometimes darker and thicker (arrow).

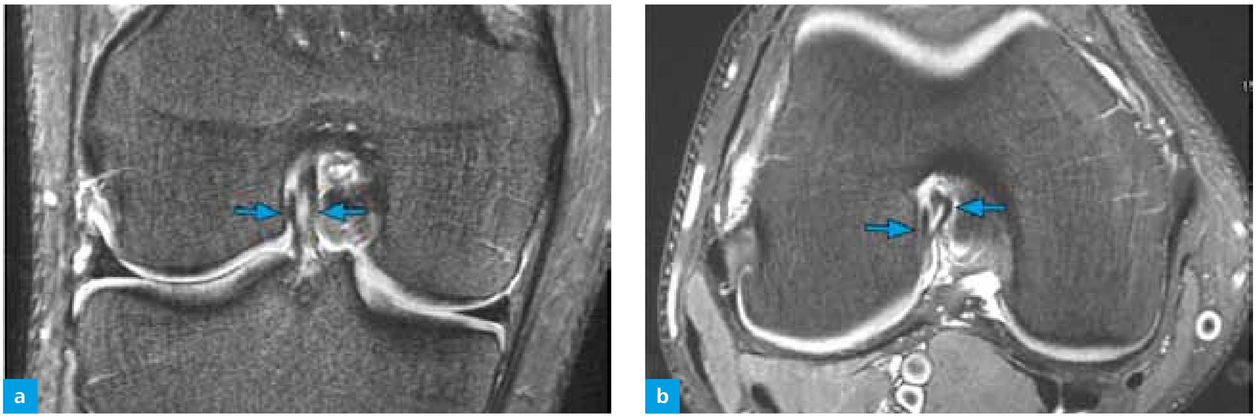


Fig. 3: Normal ACL. Coronal (a) and axial (b) proton density with fat saturation. The two bundles of the ACL are well differenciated (arrows).

Mechanism of injury

In sports the large majority of ACL injuries are caused by non-contact trauma. High-risk sports include downhill skiing, soccer, gymnastics, and American football. The ACL is especially at risk during landing, twisting and deceleration, particularly when the knee is in near full extension.[4]

Pivot shift injury

It is a type of non-contact injury among skiers and American football players. The classical mechanism is seen in downhill skiing, when the skier falls forward and catches the inside edge of the ski, forcing the tibia to rotate externally in valgus stress. The injury is marked by valgus load, flexion and external rotation of the tibia, or internal rotation of the femur. This mechanism characteristically causes contusions of the lateral compartment and is associated with meniscal and posterolateral capsular injury (Fig. 4, 5). However it should be noted that, in children, this pattern of offset lateral bone bruises may not be associated with an ACL tear because of the normal laxity of ligaments in children.[19]

Fig. 4: Sagittal proton density image of the lateral aspect of the knee showing characteristic bone bruises in the lateral compartment (arrows).

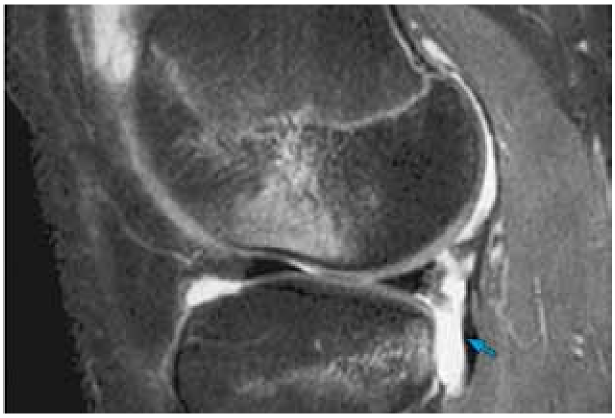


Fig. 5: Sagittal proton density with fat saturation.Posterolateral capsular injury (arrow).

Hyperextension injury

It is caused by direct force applied to the anterior tibia with a planted foot. A typical example is tackling injury in soccer. Indirect force is exerted by a forceful kicking motion. Kissing contusions are seen in the anterior femoral condyle and the anterior tibial plateau. Associated soft tissue injuries include meniscal and PCL lesions.

Clip injury

A clip injury is observed particularly among American football players. It is a contact injury secondary to pure valgus stress on a partly flexed knee. A large number of patients have medial collateral ligament and meniscal injuries.

Dashboard injury

It is caused by force on the anterior proximal tibia with the knee in flexion, and is associated with anterior tibial and posterior patellar edema as well as rupture of the PCL and the posterior joint capsule.

MR findings

Primary signs (Fig. 6) of an ACL rupture on sagittal MRI include replacement of the normal linear striated appearance by an amorphous cloud-like appearance of high signal intensity, and a discrete discontinuity of the ligament with fibers that do not run parallel to the intercondylar roof (Fig. 7, 8). Loss of continuity or an abnormal intra-substance signal are clear signs of ligament tear. The sensitivity of MR is reported to range from 92–96 % and its specificity from 89–99 %.[5, 6, 7] Rupture can also be assessed on T2-weighted axial images with a sensitivity of 92 % and a specificity as high as 100 % using the criterion of high signal intensity (indicating edema and hemorrhage) at the expected location of the ACL, or frank non-visualization of the ligament at its expected location[8] (Fig. 9). Some authors have reported that the use of an oblique coronal sequence, oriented parallel to the plane of the ACL, may enhance the diagnostic accuracy of MRI[9] (Fig. 10). Discontinuity of the ACL, especially on sagittal and axial planes, and failure of ACL fascicles to parallel the Blumensaat line, are considered to be primary and accurate signs of a ligament tear (Fig. 11).[10] Using quantitative measurements of the ACL angle, Mellado et al.[11] and Murao et al.[12] found that an ACL angle of 45 degrees was associated with a sensitivity of 93–100 % and a specificity of 84–100 %.The distal stump may become displaced anteriorly and cause locking or blockage of full extension. It appears as a nodular mass in the anterior aspect of the intercondylar notch or a tongue-like free edge folded on itself[13] (Fig. 12).

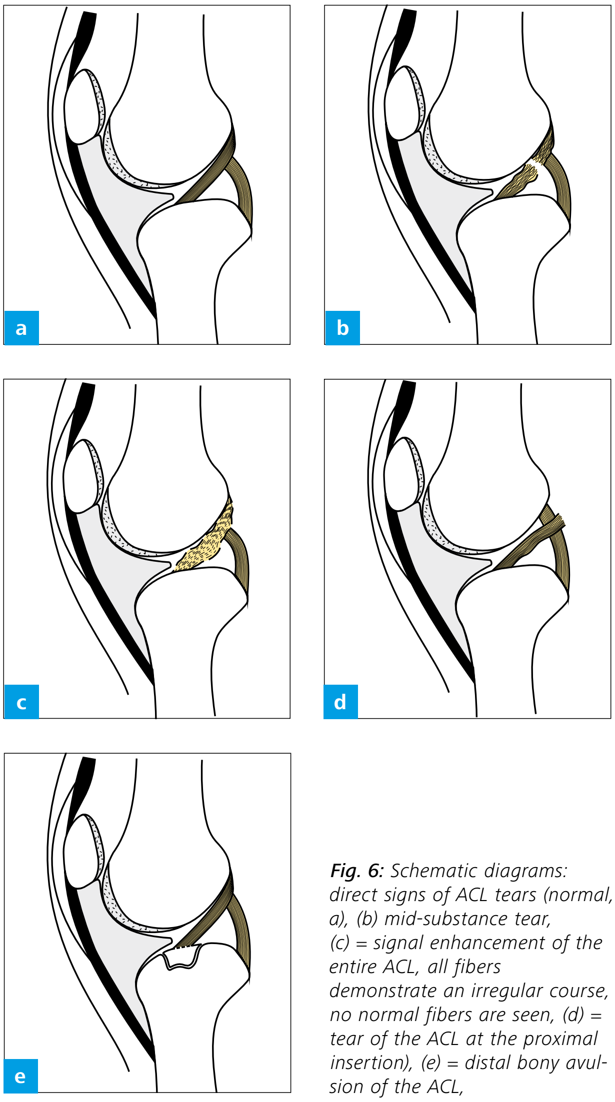


Fig. 6: Schematic diagrams: direct signs of ACL tears (normal, a), (b) mid-substance tear, (c) = signal enhancement of the entire ACL, all fibers demonstrate an irregular course, no normal fibers are seen, (d) = tear of the ACL at the proximal insertion), (e) = distal bony avulsion of the ACL,

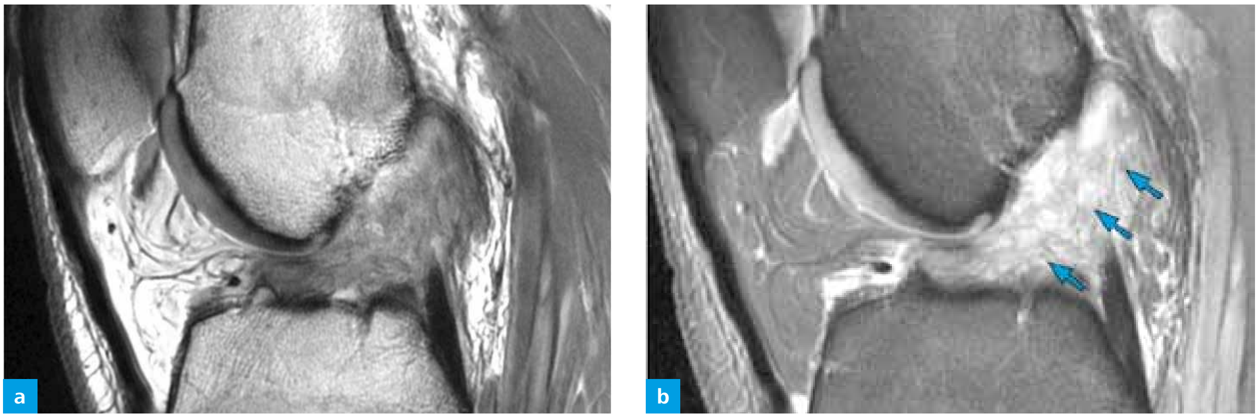


Fig. 7: *Anterior cruciate ligament rupture. Sagittal proton density without (a) and with (b) fat saturation shows waviness and a cloud-like appearance with an abnormal course of the fibers of the ACL and frank discontinuity (arrows).*

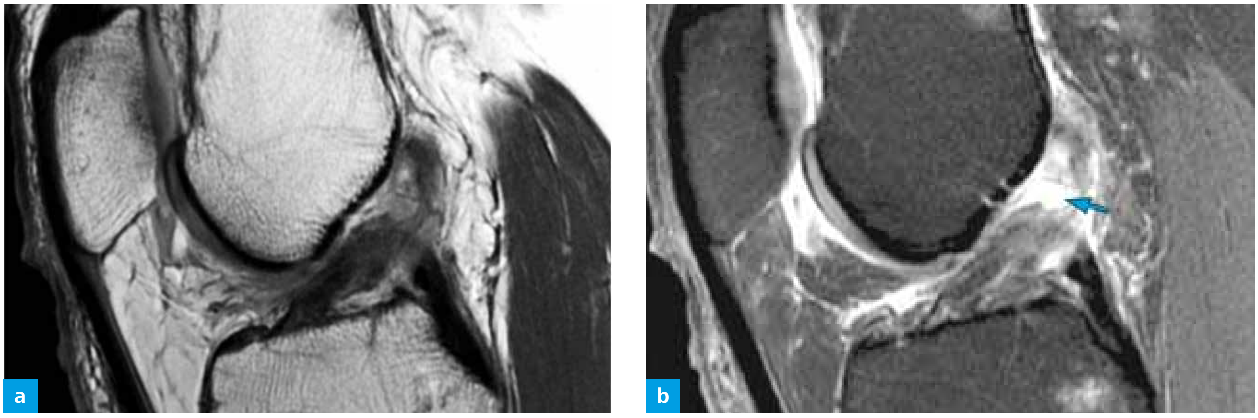


Fig. 8: *ACL tear. Sagittal proton density with (b) and without (a) fat saturation shows a high signal tear (arrow) in the proximal substance with loss of the continuity in the ligament fibers.*

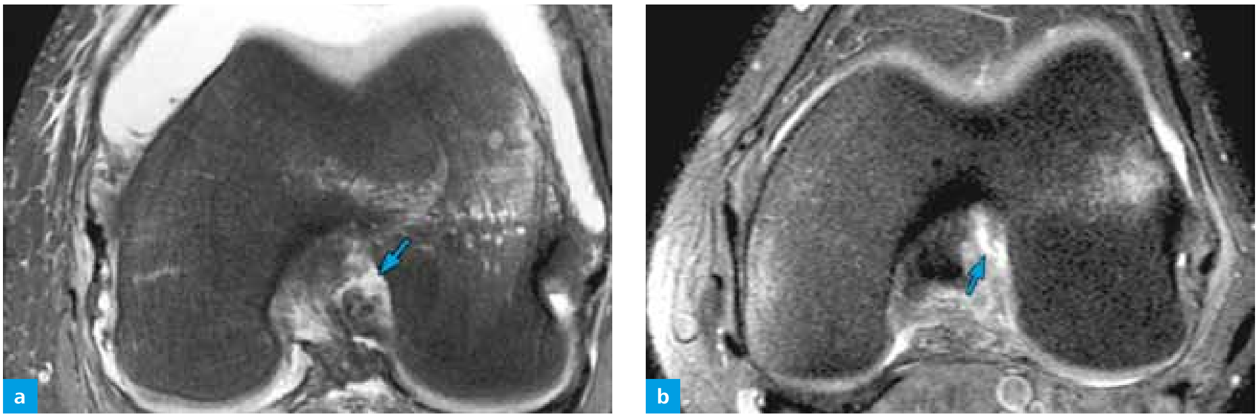


Fig. 9: *ACL tear. Axial proton density with fat saturation (a) and high signal-intensity tears (b) (arrows).*

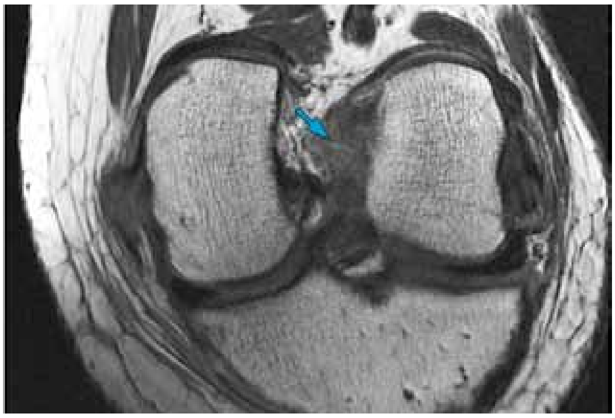


Fig. 10: ACL tear (arrow). Coronal oblique proton density image parallel to the ACL plane.

complete ruptures on the sagittal plane was associated with a low sensitivity of 40–75 % and a specificity of 62–89 % in one study[15], and with a very low accuracy of 19 % in another.[16] The difficulties in detecting partial ACL tears are related to the non-specificity of changes in the signal intensity of the ligament substance with concomitant alterations of morphology, such as attenuation or acute angulation of the ACL components.[15] The anteromedial band is most commonly involved in incomplete or partial ACL tears (Fig. 15). As the AMB constitutes the primary (96 %) restraining force to anterior drawer at 30° of knee flexion, a rupture of the AMB is believed to be functionally equivalent to a complete ACL tear.[23] Pitfalls in the interpretation of acute ACL injury

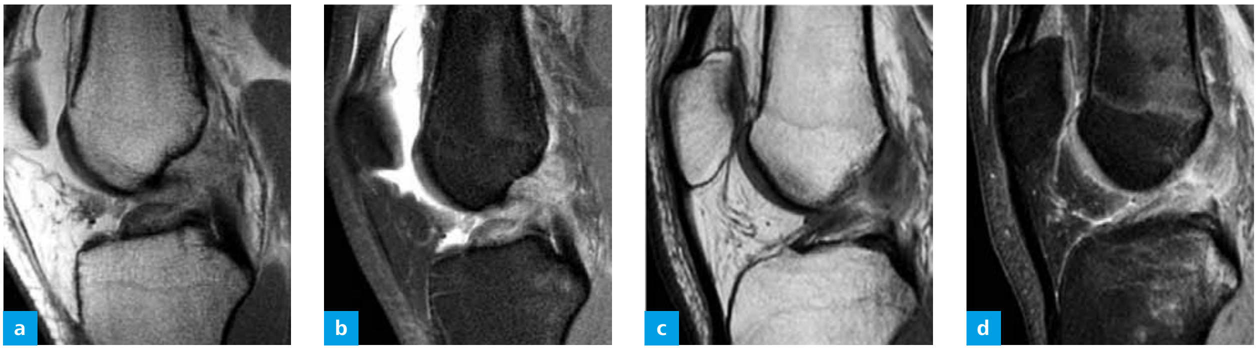


Fig. 11: Acute ACL tear. Sagittal proton density images without (a, c) and with (b, d) fat saturation.Discontinuity of the ACL fascicles in (a) and (b).Failure of the ACL to parallel the Blumensaat line (c) and (d).

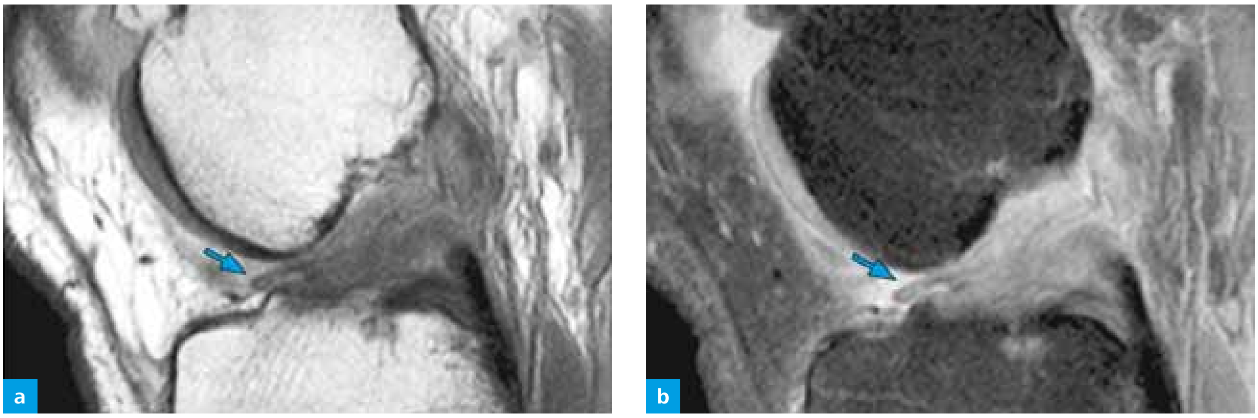
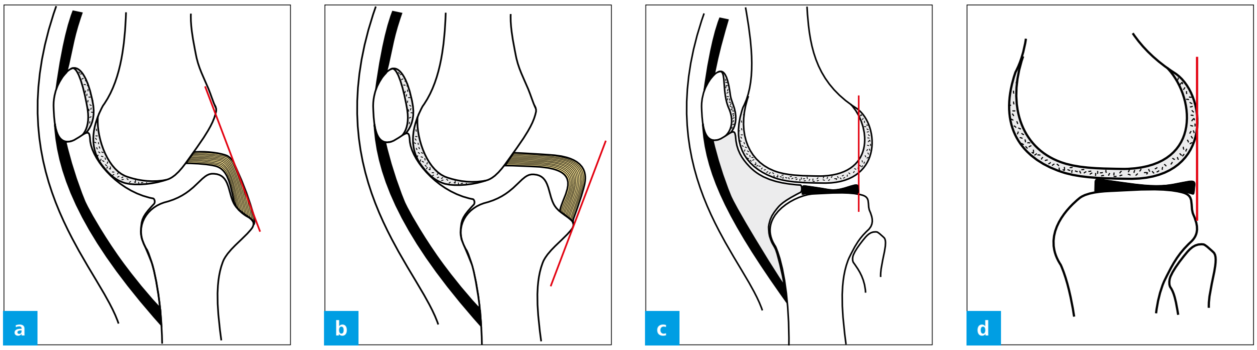


Fig. 12: Nodular mass appearance of the distal stump of the torn ACL (arrows). Sagittal proton density images with (b) and without (a) fat saturation.

Secondary signs (Fig. 13) are useful, but their absence does not rule out an ACL tear. Secondary signs with high specificity for ACL injury include pivot-shift bone bruises/ osteochondral fractures, anterior translocation of the tibia (the "uncovered meniscus" sign), and Segond fractures. Other less useful secondary signs with low specificity for ACL tears are buckling of the PCL and "kissing" bone bruises. Partial tears of the ACL may appear as focal loss of the normal striated appearance, not involving the entire diameter of the ligament and leaving intact fibers on at least one slice, or increased signal intensity on T2-weighted images with mild swelling of the ligament[14] (Fig. 14). Distinguishing partial tears from are intraligamentous ganglion cysts[17] and mucoid degeneration of the ACL[18], both of which result from remote trauma and may cause swelling and increased signal intensity, yet with an intact ACL and thus mimicking an acute injury (Fig. 16 and 17). The cysts are located in the middle and proximal portions of the ACL. They are attributed to mucoid degeneration or a herniation of synovial tissue through a capsular defect.[28] Cysts may be septated, show uniformly increased signal intensity on fluid-sensitive pulse sequences, and do not enhance after intravenous contrast injection. The "celery stalk" appearance of the ACL is marked by mucoid degeneration and fusiform enlargement of the ligament. It may occur in isolation or be associated with a well defined intraligamentous cyst (Fig. 18 and 19). Mucoid degeneration and cysts may coexist as a response of the ACL to degeneration and/or chronic trauma (Fig. 20).



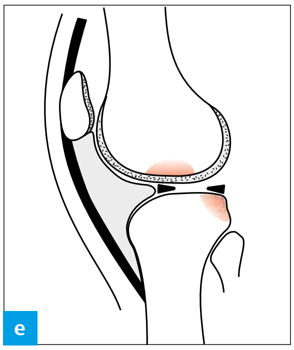


Fig. 13: *Schematic diagrams: indirect signsof ACL tears, (a) = normal, (b) = buckling of the PCL (steep course of the distal portion of the PCL) (c) = normal position of the lateral meniscus, (d) = posterior subluxation of the lateral meniscus, (e) = bony contusions at the outer aspect of the lateral femur condyle and the dorsolateral aspect of the proximal tibial condyle.*

Fig. 14: *ACL partial tear. Focal loss of the normal striated pattern with at least one fascicle intact (arrow). Proton density image (a) and proton density with fat suppression (b, c).*

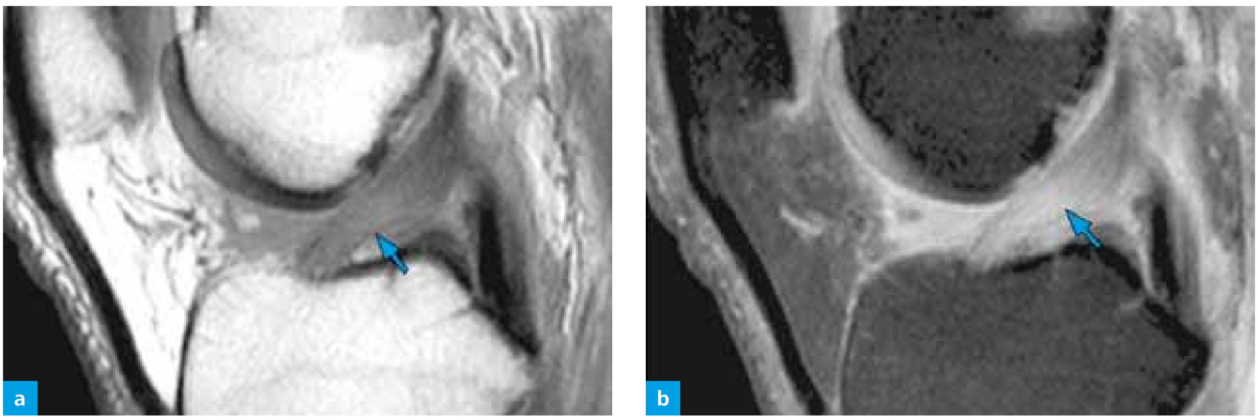


Fig. 15: *ACL partial tear of the anteromedial band (arrow). Increased signal intensity and mild swelling of the ligament on the sagittal proton density image (a) and in proton density with fat saturation (b).*

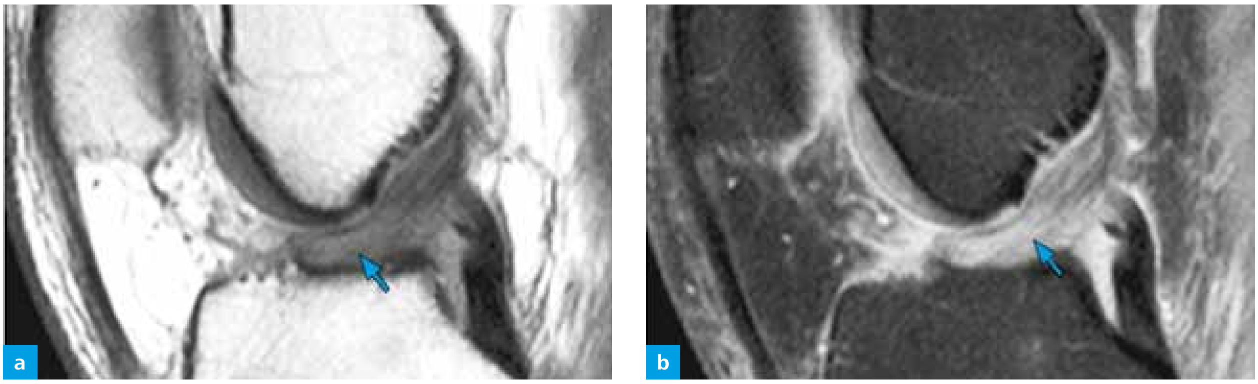


Fig. 16: Mucoid degeneration of the ACL. Swelling and increase in signal intensity on both sagittal proton density images without (a) and with (b) fat saturation (arrow).

Fig. 17: ACL cyst in continuity with proximal ACL fibers (arrows). Sagittal and axial proton density images with fat saturation (b, c) and sagittal proton density image (a).

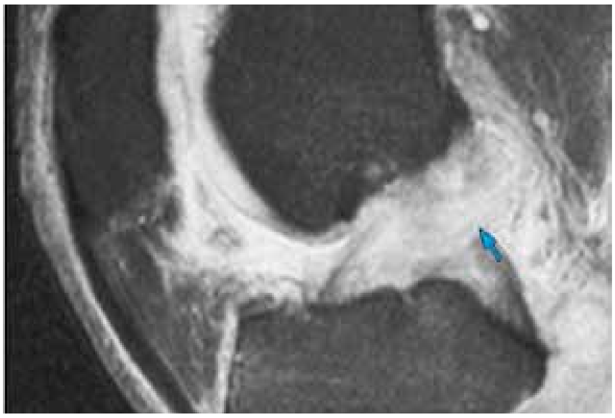


Fig. 18: Mucoid degeneration or "celery stalk" of the ACL (arrow). Sagittal proton density image with fat supression.

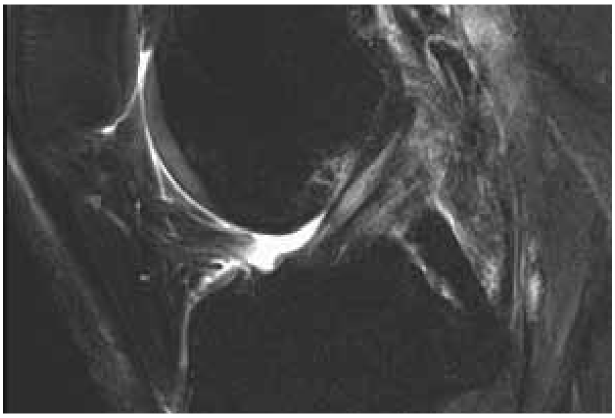


Fig. 20: Degenerative changes and a beginning ganglion of the ACL.

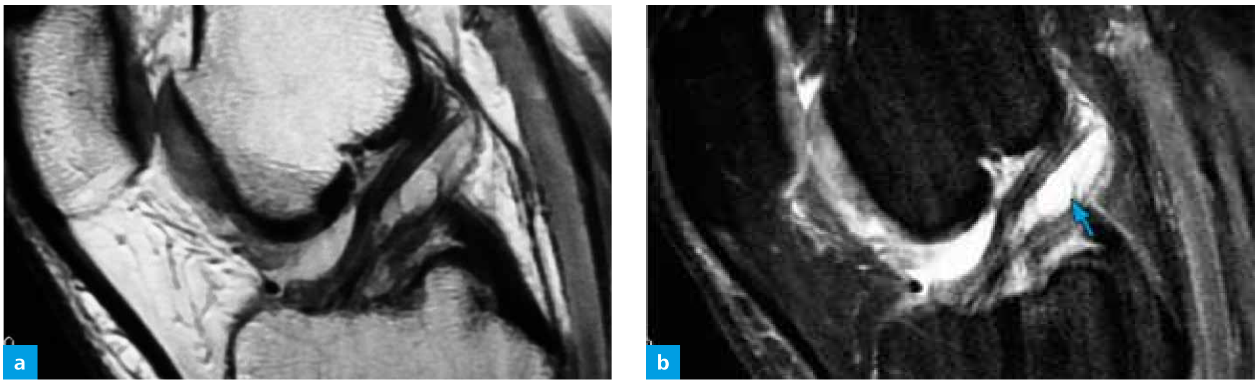


Fig. 19: ACL cyst. Sagittal proton density images without (a) and with (b) fat saturation show an intraligamentous septated cyst in the proximal ACL (arrow) with integrity of the ligamentous fibers.

Associated lesions

Anterior cruciate ligament tears may be part of a more complex injury, such as the well known triad of O'Donoghue, consisting of medial collateral ligament (MCL) injury, tear of the medial meniscus, and an ACL tear (Fig. 21).

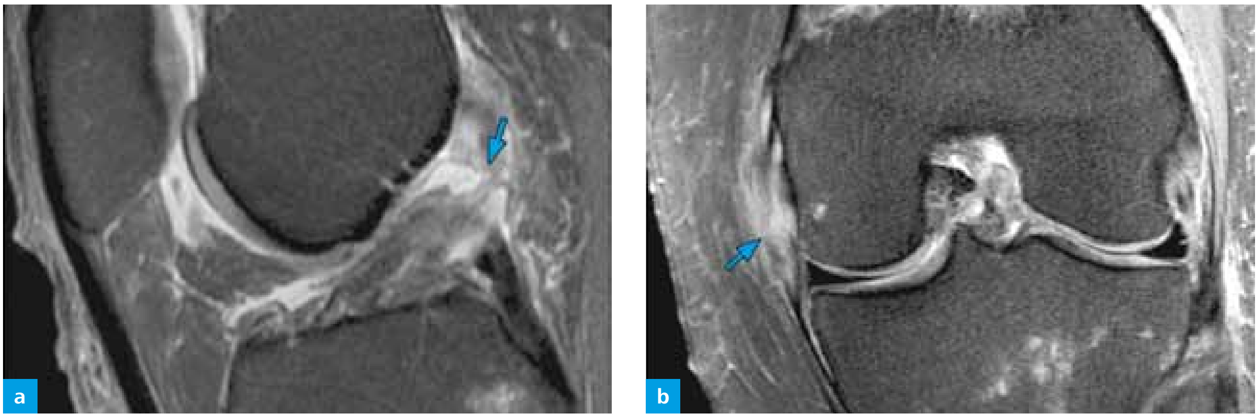


Fig. 21: *Acute tear ACL and MCL. Twisting injury with rupture of the proximal ACL (a) and middle MCL (b) on proton density with fat saturation coronal and sagittal images.*

The Segond fracture is a small vertically oriented fracture due to avulsion of the lateral joint capsule from the anterolateral aspect of the proximal tibia. It is always associated with ACL rupture and quite frequently with meniscal tear[20] (Fig. 22). Several other common types of bone injury may be associated with ACL rupture, depending on the mechanism of injury. The most common ones involve the weight-bearing portion of the lateral femoral condyle and the posterior aspect of the lateral tibial plateau (Fig. 23). Bone bruises constitute a spectrum of trabecular injury, but the overlying hyaline cartilage may also be injured. These were accompanied by thinning and degenerative changes in studies performed after recovery of the bone bruise.[21] "Kissing" bone bruises may occur in the anterior aspect of the tibial plateau and the anterior aspect of the femoral condyle due to hyperextension injury (Fig. 24).

Fig. 23: *Lateral femoral notch sign. Sagittal proton density image with fat saturation shows a deepened lateral condyle (arrow).*

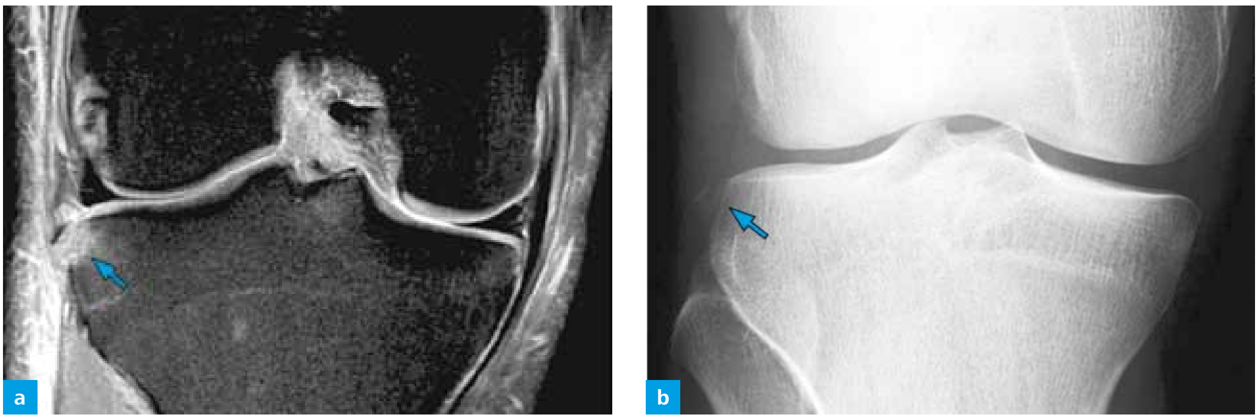


Fig. 22: *Acute Segond fracture associated with ACL tear (arrows). Coronal proton density with fat saturation (a) and AP view of the rigt knee (b).*

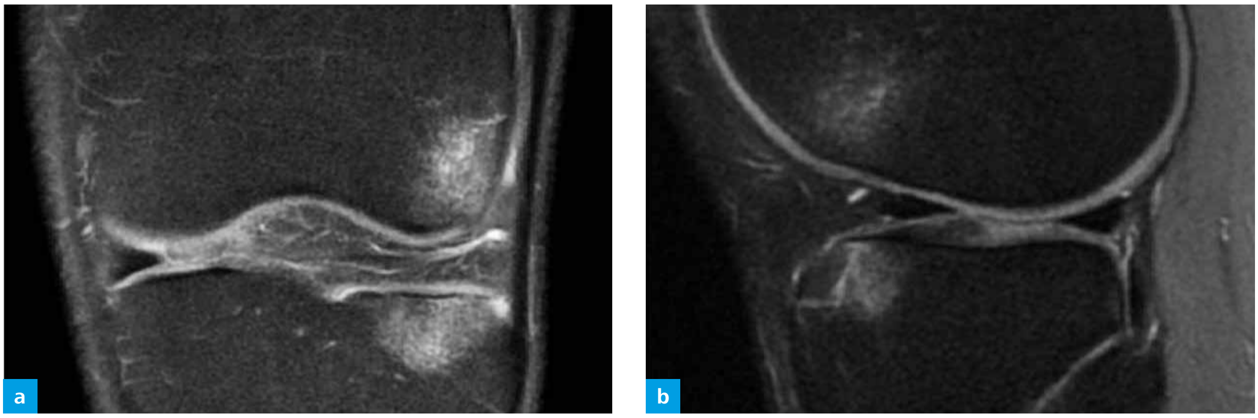


Fig. 24: "Kissing" bone bruises. Coronal (a) and sagittal (b) proton density images with fat saturation shows bone contusions secondary to ACL rupture in a hyperextension injury.

A less common site of bone injury is the posterior aspect of the medial tibial plateau. It consists of bone bruise or impaction fracture and is almost always accompanied by a peripheral meniscal tear at the meniscocapsular junction of the posterior horn of the medial meniscus[22] (Fig. 25).

This structure is rarely injured in isolation. The posterolateral corner or the arcuate complex includes the following structures:the lateral collateral ligament, the arcuate ligament with the medial and lateral limb, the popliteus muscle and tendon, the popliteofibular and fabellofibular ligaments, and the posterolateral capsule.

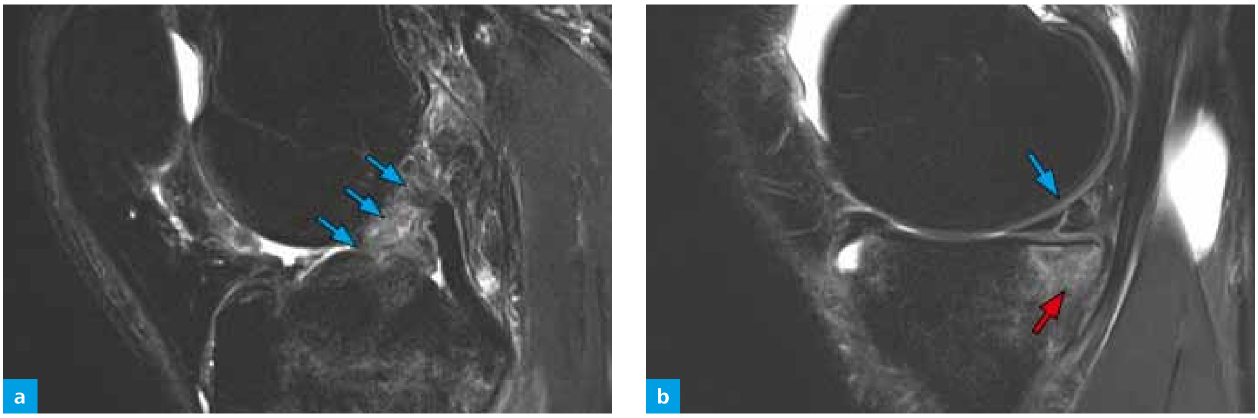


Fig. 25: Tear (blue arrows) of the ACL. Bone contusion region at the posterior aspect of the medial tibial condyle (red arrow) and rupture of the posterior horn of the meniscus (blue arrow).

Injuries to the posterolateral corner (posterolateral or arcuate complex) are frequently associated with acute ACL and also with PCL tears.[29] (Fig. 26).

Hyperextension is the most common mechanism in patients with a combined ACL and posterolateral injury.[30,31] MR findings include edema, fluid around the popliteus tendon, tearing at the popliteal musculotendinous junction, and disruption of the arcuate

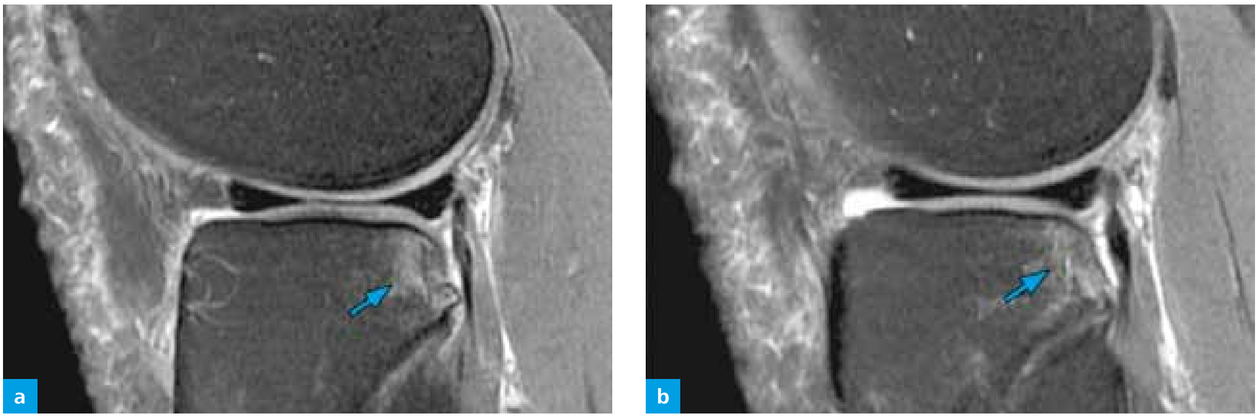


Fig. 26: Posterolateral corner injury associated with acute tear of the ACL. Contiguous sagittal proton density images with fat saturation (a, b) shows a compression fracture of the posterolateral aspect of the tibial plateau (arrow) and extravasation of fluid around the politeus tendon.

ligament. Extravasation of fluid along the course of the popliteus muscle and tendon is a sign of trauma to the attachment of the arcuate complex.

Other secondary signs of ACL injury include anterior subluxation of the tibia relative to the femur and consequent unveiling of the posterior horn of the lateral meniscus with buckling of the posterior cruciate ligament (Fig. 27).

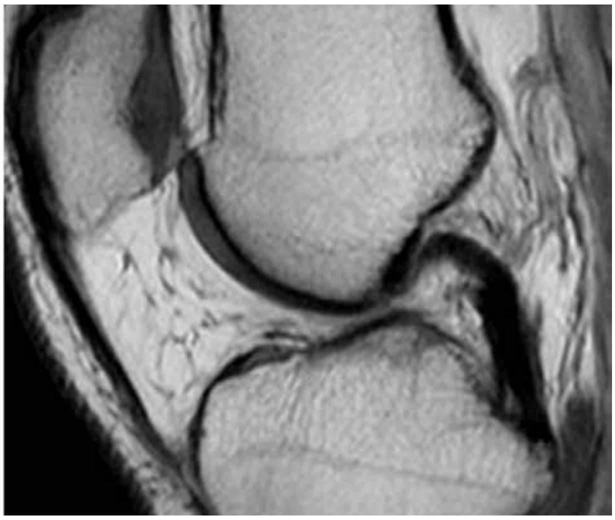


***Fig. 27:** Buckling of the PCL. Sagittal proton density image showing the abnormal morphology of the PCL secondary to anterior subluxation of the tibia.*

In skeletally immature patients, the tibial attachment of the ACL is weaker than the ligament itself. Therefore, ACL injuries occur more frequently as tibial avulsions rather than in the mid-substance of the ligament.

The injury is usually caused by forced flexion of the knee with internal rotation of the tibia. It is not associated with other knee injuries[24] (Fig. 28).

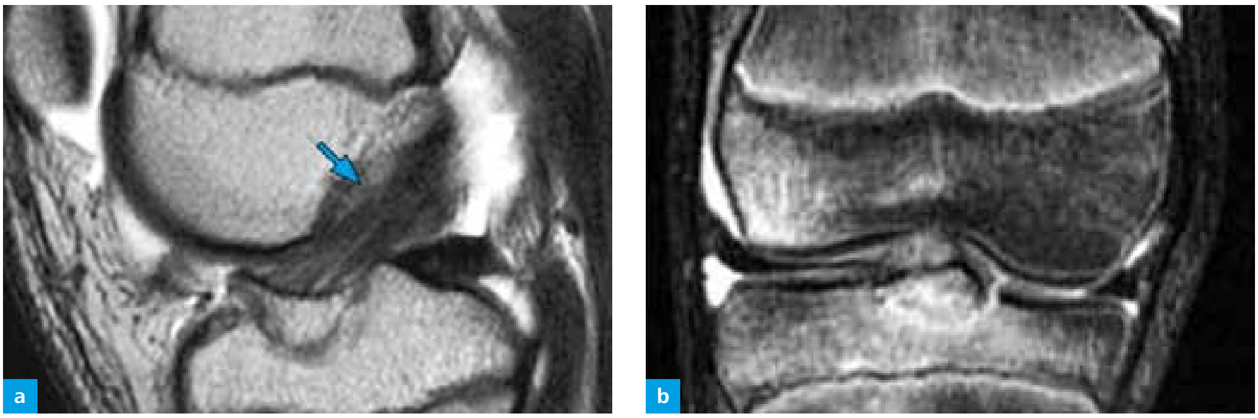


***Fig. 28:** Avulsion of the anterior tibial spine. Sagittal proton density (a) and coronal proton density images with fat saturation (b) showing an incomplete avulsion of the anterior tibial spine with integrity of tha ACL (arrow).*

Chronic tears of the ACL are manifested as non-visualization of the ligament or the so-called empty notch sign on coronal MR images (empty lateral wall), angulation of the ligament instead of a straight course due to scarring and fibrosis, or the ligament may be shallow instead of paralleling the intercondylar roof[25,26] (Fig. 29). The chronic torn ACL is often seen on the horizontal axis, although it may be oriented vertically and show discontinuity or retraction of its normal proximal condylar attachment site.

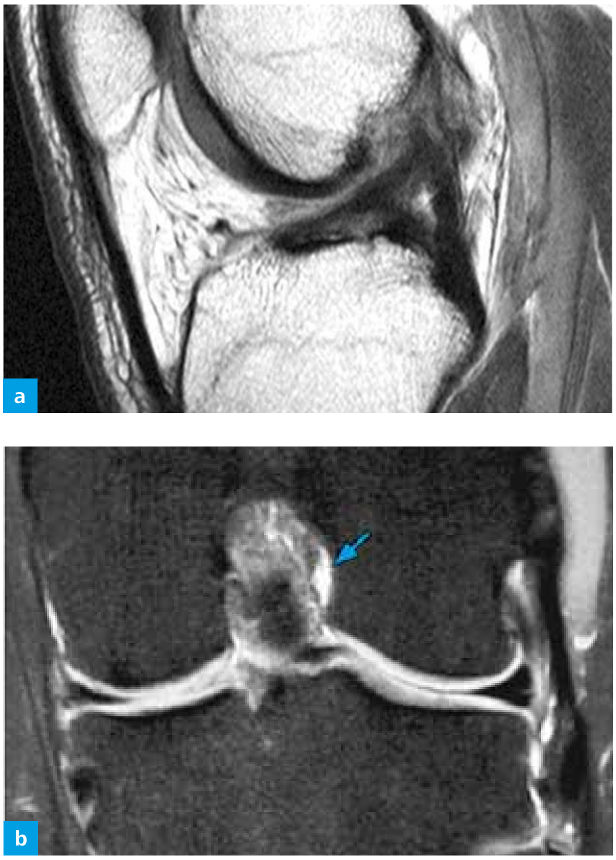


***Fig. 29:** Chronic tear of the ACL. Sagittal proton density image (a) showing angulation of the ligament and loss of its parallel orientation with the intercondylar roof. Coronal proton density image with fat suppression (b) and the so called "empty notch " sign (arrow).*

It is not uncommon to see a torn ACL adherent to the PCL (Fig. 30). Bone bruises are typically not present in a chronic ACL tear, but may be accompanied by anterior displacement of the tibia and buckling of the PCL due to residual joint laxity. The so-called lateral femoral notch sign is a deepened sulcus and indicates a chronic insufficiency of the ACL, but may also be seen in acute ACL injury due to traumatic impaction.[27]

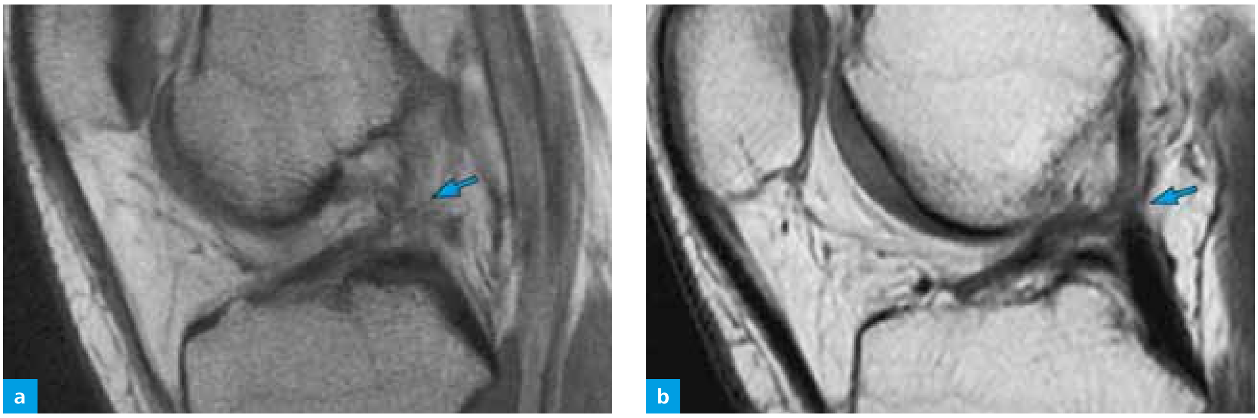


Fig. 30: Chronic ACL tear. Sagittal proton density images (a, b). Abnormal orientation of the ACL with scarring and fibrosis, adherned to the PCL (arrow).

Posterior Cruciate Ligament (PCL)

Anatomy and normal appearance

The PCL is the primary restraint against posterior subluxation of the tibia. It consists of an anterolateral and posteromedial bundle similar to the ACL, but the collagen fibers of the PCL are more tightly grouped. This is the reason for its rather uniformly low signal intensity on all MR pulse sequences. It also makes the PCL two- to four-fold stronger than the ACL (Fig. 31) The PCL is intra-articular but extrasynovial, and enveloped by a fold of synovium reflected from the posterior capsule. On sagittal images the PCL is curved. Occasionally the meniscofemoral ligaments may be seen in the anterior-to-posterior aspect of the PCL. The PCL originates in the lateral aspect of the medial femoral condyle, crosses the ACL, and attaches to the posterior intercondyloid fossa of the tibia.

Mechanism of injury

The PCL is stronger than the ACL. It has a larger cross-sectional area and greater tensile strength. These features account for the lower frequency of PCL rupture compared to ACL rupture. Injuries to the PCL constitute a mere 5–20 % of all injuries to knee ligaments. Tears of the PCL most commonly occur in the mid-portion (76 %), followed by avulsions from the femur and the tibia.[32] Tears may be caused by excessive rotation, hyperextension, dislocation, or direct trauma with the knee flexed. "Dashboard injuries" and contact sport injuries such as football are the most common causes of damage to the PCL (Fig. 32)

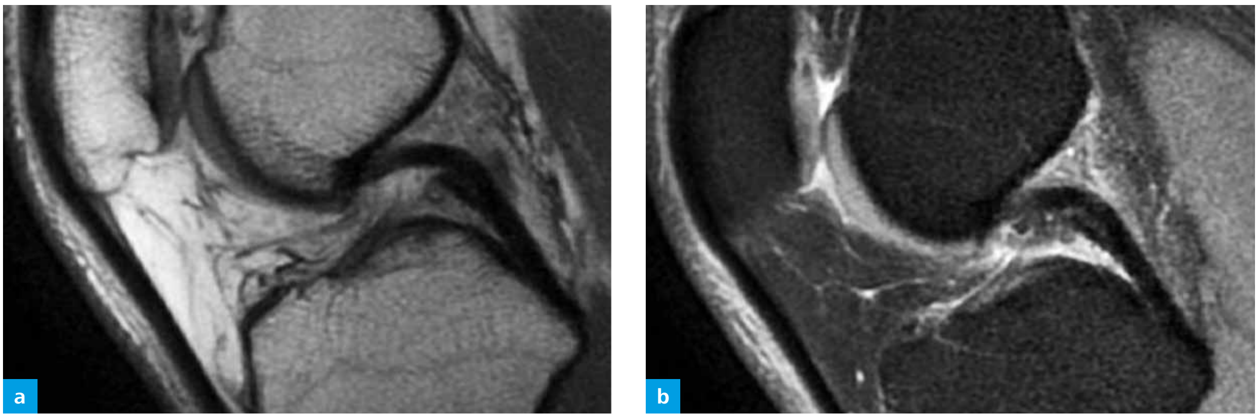


Fig. 31: Normal PCL. Sagittal proton density images without (a) and with (b) fat saturation.