Imaging ACL reconstructions and their complications

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Abstract Examination of ligament reconstructions, particularly of the anterior cruciate ligament (ACL), are common situations in everyday knee imaging practice. Knowledge of normal appearances, the expected changes over time and the potential complications of these plasties are essential. MRI is the imaging method of choice. This article illustrates the main complications specific to this procedure: suboptimal positioning of the femoral or tibial tunnels, impingement between the graft and bony contours, rupture (partial or complete) of the plasty due to friction or injury, arthrofibrosis and particularly the “Cyclops” syndrome, fragmentation or migration of the fixation materials and a granulomatous reaction to biomaterials.

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Anterior cruciate ligament plasties occupy a significant proportion of everyday orthopedic procedures, particularly because of the increasing number of sports injuries [1,2].

Over the last 4 decades, plasty with autologous transplantation has become the method of choice to treat ACL rupture [3,4].

The aim of surgery is to prevent knee instability, which inexorably progresses, to meniscal rupture and chondropathy, and therefore to early osteoarthritis [5].

Postoperative laxity of the plasty is deemed to be a surgical failure and occurs in 1 to 8% of patients who undergo ACL ligament plasty [6,7], which has a complication rate of 10 to 25% [2]. These complications may occur early (during the first year after surgery, related to the surgical procedure, incomplete incorporation of the graft or revalidation errors) or may develop late (from one year after surgery and possibly due to a further injury) [7]. The common complications of ACL plasty include “shearing” of the graft due to mechanical impingement (roof impingement), development of cysts within the fixation tunnel,

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complications from the fixation materials (migration, fragmentation, bone reaction, with irritation for example of the tibial band, etc.) and finally, infection.

The surgical techniques and materials used are evolving and improving. Knowledge of these is essential in order to confirm their expected morphology on imaging and to identify their potential complications.

The imaging technique of choice to monitor ligament plasties and identify complications after ACL ligamentoplasty is MRI [8, 9]. If MRI is contraindicated or not readily available, CT-arthrography is a very satisfactory alternative [1, 2].

**Normal appearances of the ACL plasty**

**Techniques**

The choice of ACL replacement and fixation materials for the ACL is changing over time. After solutions using allografts, the method of choice has now become autologous transplantation either with a bone-patellar tendon-bone complex (median portion of the patellar tendon sampled with small portions of its bony insertions) or with the semitendinosus and gracilis tendons folded back on themselves two or four times (“DIDT”) [2, 10]. These two techniques have not been found to have any apparent difference in terms of success or complication rates [1]. Bone-patellar tendon-bone plasty may result in complications at the harvest site, with anterior knee pain; the complications of folded tendon plasty is usually loosening of the fixation or fraying of the graft, although complications from this technique are uncommon [11, 12]. Neurological complications may occur including damage to an infrapatellar branch where the excision site is made or to the saphenous nerve when dissecting or “stripping” the semitendinosus tendon.

Plasty fixation in the tibial tunnels is carried out in parallel to the roof of the notch (Kenneth-Jones method) using interference screws made either of metal, or biomaterials which are reputed to be degradable and/or pins also made of “resorbable” materials. The “endobutton” (a small fixation button on the cortical surface) can be used for the femoral fixation [1, 2].

Biomaterial screws are assumed to remain visible on MRI for 6 months to 3 years depending on the model and normally gradually change, fading and disappearing, to be replaced by fibrous tissue [2]. Metal screws produce artefacts, which may interfere considerably with examination of the plasty. CT arthrography is a good alternative in this situation to examine screw position and the morphology of the graft if any doubt remains on MRI about possible malpositioning [13].

**Positioning**

The key factor for correct and long-lasting function of the plasty is its correct position. The aim of positioning the plasty is to retain graft isometry independently of knee movements particularly in each intermediary position during flexion–extension movements [1, 14].

The optimal position of the femoral tunnel is as far posterior as possible in the intercondylar notch without breaching the posterior femoral cortical bone (preferably 1–2 mm residual cortical thickness). Coronally the femoral tunnel should lie above the lateral condyle [10] (Figs. 1 and 2).

If the femoral tunnel is too high or too posterior the plasty becomes excessively long and tene when the knee is extended. Conversely, if the femoral fixation tunnel is located too anteriorly, the plasty becomes loose on extension and too tense when the knee is flexed.

The tubial tunnel should be parallel to the Blumensaat line in the sagittal plane (the line of the roof of the notch), opening behind the intersection of this line with the tibial plateau, with the knee in extension. Corronaly it should open opposite the intercondylar notch [1] (Figs. 1 and 2).

If the tubial tunnel is positioned too posteriorily, residual laxity develops and the plasty is ineffective (Fig. 3).

If it is positioned too anteriorly, it causes impingement between the graft and the roof of the intercondylar notch, causing graft impingement (Fig. 4).

A further procedure known as “notchplasty” may be used in this situation, in which a small fragment of bone is resected from the medial wall of the tibial femoral condyle (anterolateral portion of the notch) [15]. In the same procedure an interference screw is often inserted in the anterior aspect of the tibial fixation tunnel in order to correct the excessively anterior tubial tunnel [1].

**Maturation**

The natural evolution of the plasty may result in changes in its MRI appearances during the first months after surgery. This “ligamentization” process represents maturation of the plasty and explains the heterogeneous and varied MRI appearances of the different tendon components of the graft [16, 17]. These may persist for up to 4 years after surgery and should not be interpreted as pathological [18].

An initial natural evolutionary phase of the plasty is the “neovascularization” phase, beginning at the synovium and endosteal vessels. This is seen after surgery and lasts for approximately 2 months. The MRI signal from the graft is high on T1 and T2 weighted images although it does not reach the intensity of fluid signal. The graft is relatively thickened in appearance.

The second phase is the “cellular proliferation and collagen production” phase, which extends over a period ranging from 2 months to one year after surgery. The graft signal remains high on T1 and T2 weighted images and it becomes thinner.

The third “graft maturation” phase extends between 1 and 3 years after surgery. During this phase the plasty becomes homogeneous in appearance and has a reduced signal on T1 and T2 weighted images. The graft appearances may, however, remain slightly heterogeneous and include small striae of hyperintensity disseminated through the continuous tendon fibers [2].

**Complications**

**Graft impingement**

Graft shearing may occur as a result of impingement between the distal portion of the graft and the
**Figure 1.** Correct position of the tibial and femoral graft fixation tunnels. Postero-anterior (a) and lateral (b) knee radiographs illustrating the correct position of the tunnels.

**Figure 2.** Correct positioning of the tibial and femoral tunnels and appearances of the normal plasty. Sagittal proton density MR image showing the correct position of the femoral tunnel lying very posterior (a) and opening onto the intercondylar notch where the plasty is homogeneous in appearance (b), before reaching its tibial tunnel (c).

**Figure 3.** Too posterior tibial tunnel with abnormal vertical direction of the ACL plasty. Sagittal proton density sections showing a tibial tunnel lying too far posteriorly and too vertically (a), diverging from the tangent to the roof of the intercondylar notch. This excessively vertical position of the tibial tunnel gives the plasty a vertical appearance on the coronal T1 weighted MR image (b).
anterior-inferior portion of the intercondylar notch, which may become trapped between the femur and tibia, particularly during complete knee extension. It often follows an arched path with an anterior concavity.

In this case image abnormalities are seen on MRI in the anterior and middle two-thirds of the graft, which may be curved or frayed in appearance [9]. Ultimately it may rupture (Fig. 4).

The most common cause of impingement is incorrect positioning of the tibial fixation tunnel, too anteriorly, in front of the intersection of Blumensaat’s line and the tibial plateau [15,19]. It is important in making the diagnosis of shearing to ensure that the impingement and anterior position of the tibial tunnel are not due to the tibia being too anterior overall because of postoperative thickening of the posterior capsule which may cause anterior tibial translation [1].

**Plasty rupture**

Besides “spontaneous” rupture due to impingement, partial or complete rupture may occur as a result of injury. Traumatic re-rupture is not particularly rare in the sporting population.

The MRI signs of partial rupture are liquid signal appearances infiltrating the plasty and lying between the continuous tendon fibers, possibly with thinning of the plasty [9]. Partial rupture should not be confused with the ligamentization process (a natural postoperative evolution in the new graft as described above).

Complete rupture of the plasty secondary to a further knee injury may be promoted by non-isometric graft position, exposing it to high forces.

In complete rupture MRI shows loss of continuity of the tendon fibers and fluid filling of the graft discontinuation. Other signs are a joint effusion and posterior or anterior tibial condylar bone marrow contusions, predominantly in the lateral compartment due to traumatic impaction from the anterior tibial drawer associated with the rupture [1]. Put simply, the features are the same on MRI as are seen in initial ACL ruptures (Fig. 5).

In an old rupture, the fixation tunnel may be filled with fluid or with bone.

**Arthrofibrosis and the “Cyclops” lesion**

One of the most common complications of ligament plasty is arthrofibrosis, which involves the development of fibrous scar tissue within and around the synovium in at least one compartment of the joint. This may also develop diffusely (Fig. 6).

One specific form of this is joint fibrosis located in the anterior part of the intercondylar space producing the “Cyclops” lesion because of its arthroscopic appearance
(a nodular structure is seen similar to a Cyclops eye on the line formed by the anterior femoral condyles), which affects up to 10% of patients who have undergone ACL ligament plasty [5,20,21]. This is a mass of nodular fibrous tissue, which is usually small in size (10—15 mm) and occasionally forms around the anterior portion of the ligament graft [22]. It may become trapped between the femur and tibia when the knee is completely extended and clinically results in limited extension or even pain in the knee. The Cyclops lesions can contain cartilaginous or bony tissues [22].

The prevalence of joint fibrosis is higher in patients who have had ACL reconstruction within 4 weeks after the injury and in those who had signs of inflammation (swelling, effusion, pyrexia) at the time of surgery [1].

The most popular pathogenic explanation is that the Cyclops lesion develops around the distal stump of the native ruptured ligament because of an inflammatory reaction [23].

On MRI, the Cyclops lesion appears as a nodular lesion on the anterior portion of the intercondylar notch, which has a moderately heterogeneous appearance on all sequences and occasionally extends along the roof of the notch. It enhances with gadolinium [2]. The differential diagnoses are villonodular synovitis, synovial chondromatosis and an intra-articular foreign body, although these are uncommon after ligament plasty. This complication requires surgery with arthroscopic debridement [1,2] (Fig. 7).

**Cyst in the fixation tunnel**

During the first 18 months after reconstruction of the ACL, a small amount of fluid may be seen in the graft fixation tunnel which may enlarge moderately (up to 13 mm) [24]. This is a common and harmless process, which does not necessarily develop into a true cyst in the tunnel or failure of surgery.

Expansion of the fixation tunnel after cyst formation may be due to incomplete graft incorporation. The development of necrotic tissue around the plastic then leads to accumulation of synovial fluid in the tunnel leading it to gradually widen (over 20 mm) and form as cyst in the tunnel [9,25,26]. In some cases the cyst formation process may extend beyond the cortex of the bone and clinically then often results in a subcutaneous nodule, which is painful on palpation [2] (Fig. 8).

Femoral tunnel cysts are less common than tibial tunnel cysts. They may cause pain or may remain asymptomatic and be discovered incidentally on MRI [1]. Some of these cysts appear to be due to a host bone reaction to the fixation materials (cf. below).

**Displacement, malposition and fragmentation of fixation materials**

Whether they are made of biomaterials or metal, the interference screws, pins and other types of graft fixation materials can cause complications or lead to failed surgery.

The materials can cause fractures of the underlying bone, fragment, move about the joint space or outside of the bony

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**Figure 5.** Post-traumatic rupture of the ACL plasty. T1 weighted coronal MRI (a) and sagittal DP views (b). The plasty is heterogeneous in appearance and interrupted (arrow) and associated with joint swelling.

**Figure 6.** Diffuse joint fibrosis: T2 weighted sagittal sections: very hypointense areas involving all, anterior and posterior compartments (arrows), the Hoffa fat pad in particular makes the ligament structures in the notch almost indiscernible.
contours into the subcutaneous soft tissues, causing pain or impingement with adjacent structures, or even severe damage to cartilage tendons, blood vessels or nerves (e.g. popliteal artery pseudoaneurysm) [2,27] (Fig. 9).

These complications of ACL ligamentoplasty are seen particularly with biodegradable pins. If they are malpositioned, move about or get fragmented, the materials may come into contact with the ilio-tibial band or tendon structures causing irritation as a result of friction, or thickening, or even rupture [1,28].

**Inflammatory reactions and rejection**

Gradual degradation of a biomaterial graft fixation screw and its progressive incorporation into the bone structure and replacement by bone tissue are the expected stages in the natural evolution of the material.

Some biomaterials used (e.g. Calaxo, Smith & Nephew, withdrawn from the market in 2007) cause a severe inflammatory reaction within the bone structures and also possibly in the neighbouring soft tissues because of
the immune reaction triggered, notably with excessive local cytokine production [29,30]. This process may lead to granuloma formation, either inside the bone around the screw or around its fragments, in the superficial opening of the fixation tunnel or in the neighbouring soft tissues [31]. This effect may progress, making the bone fragile and may be complicated by fracture [31] (Fig. 10).

Figure 9. A patient with pain a few months after ACL plasty. Fracture and migration of a femoral graft fixation pin outside of the bone. Comparison with MRI appearances on coronal and sagittal proton density views with fat saturation (a and b) and the coronal and sagittal plane reconstructions from the joint CT (c and d). Tubular structure seen migrating outside of the bone (arrows), close to a branch of the popliteal artery. Photograph of fragments recovered surgically (e).
Septic arthritis

Postoperative joint infection is a rare complication (0.1—0.9%) [1]. In the absence of typical local symptoms such as redness, warmth, pain or reduced mobility, early diagnosis is difficult in the acute postoperative phase. A warning sign is the combination of joint swelling and moderate pain, and in any doubt, tests should be carried out for laboratory indices such as a CRP or erythrocyte sedimentation rate remaining raised after the first week postoperatively [1,32].

Suggestive imaging signs of infection are appearances of synovitis, bony erosions, periarticular edema and fluid collections or abscesses [9].

Other complications

The other less common complications of ACL plasty include retractile capsulitis, rupture of the patellar tendon at the graft sampling site, fractures at the sampling site for the patellar tendon bone complex or shortening of the "donor" tendons by scarring, together with the more general complications inherent to surgery, such as deep vein thrombosis or bleeding [2,33].

Conclusion

Confirming that at ACL ligament plasty has been correctly performed and investigating for complications of the procedure are common situations in postoperative knee imaging.

MRI is the method of choice to examine and monitor ACL plasty.

This imaging modality can identify the main complications, which may be due to the surgical procedure and include incorrect positioning of the fixation tunnels, impingement between the graft and roof of the intercondylar notch, partial or complete rupture of the graft due to impingement, a cyst within the tunnel and localized or diffuse joint fibrosis. Others are associated with the fixation materials, including fragmentation, migration, fractures, granulomas and friction. Traumatic rupture of the plasty may occur. Finally, general postoperative complications may occur including joint infection, bleeding and venous thrombosis.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.
References


