Case Report

Arthroscopic Roofplasty: A Method for Correcting an Extension Deficit Caused by Roof Impingement of an Anterior Cruciate Ligament Graft

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Summary: Intercondylar roof impingement should be suspected in any patient having difficulty regaining knee extension following an anterior cruciate ligament (ACL) reconstruction. An arthroscopically performed roofplasty can eliminate the clinical complaints. The extent of bone removal can be planned from studying a lateral radiograph taken with the knee in terminal extension. Extension exercises or passive extension devices should not be used in patients with extension deficits caused by roof impingement because they may further damage the graft. Key Words: Roofplasty—Roof impingement—Graft, anterior cruciate ligament—Extension deficit.

Intercondylar notch impingement of an anterior cruciate ligament (ACL) graft is a perplexing problem confronting the reconstructive knee surgeon. Skilled knee surgeons, despite their keen awareness of this problem, continue to report late complications of pain, limited knee extension, synovitis, graft abrasion, and late graft rupture attributed to graft impingement (1-4).

At least three anatomic causes of ACL graft impingement have been documented: (a) The volume of the intercondylar notch may be congenitally small, resulting in stenosis of the ACL by a narrow notch (5). (b) Chronically unstable knees acquire osteophytes that stenose the aperture of the intercondylar notch. These osteophytes, if not removed during the ACL reconstruction, can result in late graft abrasion (6,7). (c) Tibial tunnel placement anterior to the slope of the intercondylar roof can result in graft impingement as the knee is extended (2, 8-10).

This report describes the clinical course of a patient with roof impingement of an ACL graft caused by an anteriorly placed tibial tunnel. Serial magnetic resonance (MR) findings of the graft are presented and successful arthroscopic correction of the impingement by a delayed roofplasty is described.

CASE REPORT

A 30-year-old man who is a recreational basketball player suffered an acute ACL rupture during a noncontact deceleration maneuver while playing basketball. Six weeks following the injury he reported complaints of pain, effusion, and limited knee extension to the orthopaedic clinic. Physical examination revealed a moderate knee effusion, range of motion from 15 to 115°, a positive Lachman test, no joint line pain, and no varus/valgus or posterior instability. Pivot shift testing was prevented by pain. KT-1000 ligament laxity testing (MedMetric, San Diego, CA, U.S.A.) revealed an increase in anterior tibial translation of 2.5 mm at 89 newton (N) (20#) and 5 mm during a maximal manual drawer test compared
to the normal side.

An arthroscopically assisted patellar tendon bone reconstruction was performed 8 weeks postinjury. Knee motion under anesthesia, before reconstruction, was from 10 to 135°. Pivot shift was grade 2. There were no meniscal lesions blocking extension. Femoral and tibial pilot pins were inserted by using a front entry guide system (Acufex Microsurgical, Norwood, MA, U.S.A.). The tibial guide pin was placed 5 mm anterior and medial to the center of the ACL insertion. A tensiometer (Acufex Microsurgical, Norwood* MA, U.S.A.) confirmed that the tibial-femoral separation distance decreased 1 mm during knee flexion from 10 to 90°. A 10 mm wide patellar tendon graft was harvested and secured in the femoral and tibial tunnels with the medial-lateral dimensions of the tendon rotated 90° and oriented in the sagittal plane. Side wall impingement was corrected by a wallplasty that removed -5 mm of the medial aspect of the lateral femoral condyle. Limited knee extension, caused by preoperative soft tissue contracture, prevented complete assessment of the potential for intercondylar roof impingement.

A roofplasty did not appear to be needed. An extraarticular augmentation was performed by securing the posterior 12 mm of the iliotibial band to the posterior femur with a screw and washer.

Postoperatively, the knee was placed in a continuous passive motion machine for 4 days until 90° of flexion was regained. A long leg hinged brace with an extension stop at 35° of flexion was worn full time for the first 5 weeks. Full weight bearing was begun at 7 weeks. A derotation brace with an extension stop at 35° of flexion was used only during strengthening exercises (.5 h/day) for the next 4 1/2 months. A brace was not worn for walking and activities of daily living. At 6 months the extension stop was removed from the derotation brace and progressive return to athletics was permitted. The patient was enrolled in a daily supervised physical therapy program for the first 6 months.

As part of a separate ongoing study, serial sagittal MR scans were obtained at weeks 1, 6, 11, 24, and 48 postoperatively to follow any time-related changes in the ACL graft appearance. The MR scans were performed using a 0.35 Tesla superconducting magnet with a dedicated quadrature detection knee coil (Diasonics, San Francisco, CA, U.S.A.). Imaging was confined to ten 2.5 mm thick sagittal sections (0.625 mm² pixels) centered about the intercondylar region of the knee. The knee was externally rotated 10 to 15° to align the graft optimally in the sagittal plane. Image acquisition was performed with the standard spin-echo technique using a 1500-msec TR and 50-msec TE. Encoding and reconstruction was performed with the standard two-dimensional Fourier transformation technique using 256 phase encoding steps and two excitations (15 min acquisition time).

The patient’s progress in physical therapy plateaued at 12 weeks postoperatively. He complained of pain, an effusion, and a persistent 10° flexion contracture. Physical exam findings supported these complaints. A review of the first three MR scans revealed a step-wise deterioration of the appearance of the graft. At 1 week the graft was of low signal intensity, black and homogeneous in appearance from origin to insertion, and indistinguishable from the MR appearance of the posterior cruciate ligament and the quadriceps tendon (Fig. 1). The knee was in -20° of flexion during the imaging procedure, with the graft and the intercondylar roof in direct contact. By week 6 the graft began to thin in its sagittal dimension due to the impingement of the intercondylar roof as the knee regained extension (Fig. 2). The MR scan at 11 weeks revealed regionalized edema confined to the distal one-third of the graft. The graft almost

FIG. 1. One-week graft of low signal intensity, and homogeneous in appearance from origin to insertion, and indistinguishable from the magnetic resonance (MR) appearance of the posterior cruciate ligament and the quadriceps tendon. The graft was aligned adjacent to the intercondylar roof in the partially flexed knee (20°) (arrow).
Because of the MR relationship of the graft to the intercondylar roof, a lateral radiograph was obtained with the knee in maximal extension (Fig. 4). The tibial tunnel was noted to be located partially anterior to the intercondylar roof verifying the impingement noticed on MR scanning (2,8).

A repeat arthroscopy was performed at 14 weeks. Adhesions in the suprapatellar pouch were minimal and did not require removal. The fat pad was firmer in consistency than normal, but not fibrosed. Limited removal of the fat pad was required to view the graft. A roofplasty was performed. The inclination of the intercondylar roof was made more horizontal by removing 8 mm of bone. The postroofplasty notch view displays the location and extent of bone removal (Fig. 5). A manipulation using anesthesia was not performed.

The patient’s complaints of pain, effusion, and extension deficit disappeared by 3 weeks after the roofplasty. The knee regained 10° of extension with a motion arc of 0 to 135°. An MR scan of the ACL graft at 24 weeks (10 weeks after the delayed roofplasty) revealed restoration of a normal MR signal.
The increased MR signal under the distal edge of the intercondylar roof regained the low signal intensity identical to the 1 week appearance of the graft (Fig. 6).

The clinical result of the reconstruction was excellent at 25 months. The range of motion in the operated knee equaled that of the normal knee (0 to 135°). KT-1000 ligament laxity testing revealed equal anterior translation during an 89 N and manual maximum test (MMT) test. The involved knee achieved 100% of the distance jumped by the normal knee in the single-leg hop test (11). The Lysholm score was 98 (12). An MR scan at 48 weeks confirmed that the ACL graft maintained a normal MR signal.

DISCUSSION

The MR appearance of impinged and unimpinged ACL grafts has been studied over time and is markedly different (2,13). Grafts placed in tibial tunnels anterior to the slope of the intercondylar roof suffer from roof impingement (2,8-10). Characteristically, roof impingement results in an increase in signal intensity in the distal portion of the graft by 3 months postimplantation (2,8-10). An impinged graft directly contacts the intercondylar roof and may be seen to bow posteriorly if the scan is obtained with the knee maximally extended (2,13). This regionalized signal increase does not spontaneously revert to a low signal intensity (13). The signal increase persists during the first 3 years of implantation (2).

In contrast, grafts placed posterior to the slope of the intercondylar roof are not impinged (2,8,10). Unimpinged grafts maintain a low signal intensity during the entire first year of implantation (2). They do not acquire a regionalized MR signal increase. Clinically, knees with unimpinged grafts are more likely to regain full extension than impinged grafts (2).

Clearly, the graft in this patient was subjected to roof impingement. The lateral radiograph confirmed that the tibial tunnel was anterior to the slope of the intercondylar roof. The signal increase of the distal one-third of the graft on the 11-week MR scan was diagnostic. Direct contact between the intercondylar roof and the graft was seen on all the MR scans.

There was no interposed soft tissue on the MR scan to suggest that a “cyclops syndrome” was the cause of the impingement (3). Therefore, the extension deficit was caused by the graft impinging against the bone of the intercondylar roof.

The delayed roofplasty eliminated the flexion contracture within 3 weeks of raising the intercondylar roof.
The MR signal of the graft returned to the original low signal intensity within 10 weeks after eliminating the roof impingement. The MR signal of impinged grafts has been shown to remain elevated for 1 to 3 years after implantation (2,13). Therefore, the roofplasty was responsible for returning the MR appearance of the graft to the low signal intensity characteristic of an unimpinged graft.

REFERENCES