The aim of this study was to evaluate the tunnel enlargement phenomenon after ACL reconstructions performed with hamstrings tendons fixed using the cross pin technique. Sixty-two knees in 62 patients were followed for two years to evaluate the possible clinical implications of the femoral and tibial tunnel enlargements noted after ACL reconstruction. The reconstructions were done with hamstring tendons using the cross-pin technique. Evaluation was based on calculated clinical scores (IKDC and Lysholm knee scores) and quantified by KT-1000 measurements. Sagittal and coronal plane computed tomography and conventional radiography were performed 3 days after operation and were repeated after 3 and 6 weeks, 6, 12 and 24 months, to assess early tunnel enlargement.

Although it seems that tunnel enlargement after ACL reconstruction has no impact on the clinical results, long-term implications and potential need for revision surgery must be assessed. In this study, tunnel enlargement was noted fairly early after operation and was thought to be related with drilling of the tunnels. A possible solution to this problem may be drilling the tunnels to a diameter 1 mm smaller than the measured graft diameter, then to enlarge the tunnels to the graft diameter with the appropriate tunnel dilator.

**Keywords**: ACL reconstruction; hamstring tendon; tunnel enlargement.

**INTRODUCTION**

The most popular techniques for primary ACL reconstruction currently use bone-patellar tendon-bone or hamstring tendon autografts. Tunnel enlargement after operations using both types of grafts and various fixation techniques have been described in the literature (4,9). No definite causes have been demonstrated for tunnel enlargement, and the aetiology is thought to be multifactorial (7, 13).

Biplanar conventional radiographs have been used to measure femoral and tibial tunnels diameters. In conventional radiographs, measurements are usually made from the sclerotic edges of the largest part of the tunnel, but the sclerosis is
thought to be prominent no earlier than three months after operation. It has been shown however that CT scans measurements were in discordance with measurements on plain radiographs. Tunnel enlargement was noted earlier and appeared more marked on CT measurements compared to the conventional radiographs. In this prospective study, CT scans were used to measure tunnel enlargement immediately after operation, 3 weeks and 6, 12 and 24 months after ACL reconstructions using hamstring tendons fixed using the cross-pin technique, with a view to disclose the possible aetiology for tunnel enlargement.

PATIENTS AND METHODS

Sixty-two knees in 62 consecutively operated patients (12 female, 50 male) with a mean age of 26.4 ± 4.5 years (range: 18 to 42) were enrolled in the study. All patients had positive unilateral anterior drawer and Lachman test, no previous knee surgery and closed physes. Patients with axial malalignment, additional ligament injury or arthritis were excluded from the study. All the patients were clinically evaluated during scheduled follow-up visits, biplanar radiographs were taken, IKDC and Lysholm knee scores and measurements of sagittal laxity with the KT-1000 (Med-Metric, San Diego, CA) were recorded (1, 6). Tibial and femoral tunnels measurements were performed after consenting the patients for the CT examinations with Hitachi Whole body X-Ray CT system CT-W800. The system specifications were as follows: density resolution: 2 mm (0.5% contrast) at 10 mm slice; Slice thickness: 10.5 mm (+10%), 3 mm (+15, -10%), 2 mm (+30, -10%); radiation dose to skin: 1 to 10 rad; effective FOV: 160, 200, 250, 300, 350, 420 mm. The scans were performed first within the first three days following surgery, then after 3 and 6 weeks and after 6, 12 and 24 months.

CT scans were standardised by scanning the operated knee with 5 mm slices that were made perpendicular to the tibial and femoral tunnel respectively, from the cross pins to the lowest level of the tibial tunnel. Digital measurements were made independently by two orthopaedic surgeons. Measurements were made on the slices showing the largest tunnel diameter on the tibial tunnel exit on the tibial plateau and similarly the largest tunnel diameter on the tunnel entry point into the lateral femoral condyle for the femoral side. If any sclerosis was encountered in the tunnels, the outer margins of the sclerosis were measured and recorded. Student’s t-test was used to compare the tunnel measurements at different follow-up visits. Pearson’s chi squared test was used to evaluate the changes in clinical scores and tunnel enlargements.

SURGICAL TECHNIQUE

The gracilis and semitendinosus autografts were harvested and each was made into a double strand graft.

Standard anterolateral and anteromedial portals were used to confirm absence of the ACL and to assess additional pathologies such as meniscal tears and/or chondral lesions. Tibial and femoral tunnel entry points were cleaned with a shaver and RF probe. Tibial and femoral tunnels were drilled to the exact diameter of the autograft, using the appropriate guides. The four-strand tendon autograft was placed inside the tunnels using a flexible guide wire over which the transfixing cross-pin (Artrex OrthoTech, München-Germany, Florida-USA) was inserted. On the tibial side, the autograft was fixed in the tibial tunnel using a soft screw and two staples for extra stability.

Patients were allowed to weightbear as tolerated using an extension brace. Patellar mobilisation, terminal extension and closed chain kinetic exercises were initiated after the drain was removed on the 1st postoperative day. All patients were discharged after 4 ± 1 days. Cooperation to rehabilitation was evaluated every 10 days during the first month postoperatively.

RESULTS

The mean diameter of the femoral and tibial tunnels drilled at operation depended on the thickness of the harvested tendon graft: it was 8.6 mm on average (range: 7.0 to 10.0). The one-year follow-up measurements showed enlargement of the femoral and tibial tunnels: in the coronal plane, there was a mean tunnel enlargement of 1.1 mm (range: 0.9 to 1.3) and 0.85 mm (range: 0.7 to 1.0) respectively; in the sagittal plane, there was a mean enlargement of 1.0 mm (range: 0.7 to 1.2) and 0.7 mm (range: 0.6 to 1.0) respectively (table I).
On the CT scans made on the first postoperative day, the diameters of the femoral and tibial tunnel were observed to be 0.23 mm (range : 0.2 to 0.4) and 0.21 mm (range : 0.2 to 0.3) larger than the diameter of the drill used to open the tunnels (fig 1). A dense rim which was thought to be due to a possible bone necrosis and compacted bony debris created during drilling, was also observed in the periphery of the tunnels.

These radiopaque rims were no longer visible on the CT scans made after three weeks, leading to further enlargement of both tunnels. The tunnel enlargements on the femoral and tibial tunnels were then 0.64 mm (range : 0.5 to 0.8) and 0.43 mm (range : 0.4 to 0.6) respectively (fig 2). On the measurements made after 6 weeks, the tunnel enlargements on the femoral and tibial tunnels were 0.84 mm (range : 0.7 to 1.1) and 0.61 mm (range : 0.5 to 0.8) respectively. On the measurements made after 6 months, the tunnel enlargements on the femoral and tibial tunnels were 1.1 mm (range : 0.9 to 1.2) and 0.83 mm (range : 0.7 to 0.9) respectively. The one year measurements were similar to those at 6 months, and the tunnel enlargements on the femoral and tibial tunnels were 1.2 mm (range : 0.9 to 1.4) and 0.85 mm (range : 0.7 to 1.0) respectively (fig 3) (table II).

The measurements made after two years did not show any further tunnel enlargement; on the contrary, they showed a decrease in diameter due to thickening of the sclerotic rim. The tunnels decreased in diameter 0.2 mm (range : 0.1 to 0.3) on the femoral side, and 0.14 mm (range : 0.1 to 0.2) on the tibial side, compared to the measurements of the one year follow-up (fig 4).

In the follow-up clinical evaluations, no correlation ($p \geq 0.05$) was found between the clinical scores and the tunnel enlargements. Table III summarises the preoperative and follow-up clinical scores.

**DISCUSSION**

Tunnel enlargements on both sides of the joint were found to be a fairly early process produced first by the pendulum movement of the drill bit and heat necrosis, enhanced by a non specific inflammatory reaction. These findings are supported by other authors’ work (3,5,10,13). Although no correlation has been noted between the clinical scores and the tunnel enlargement values, the long term implications are still unknown. To lessen the potential risks of tunnel enlargement, we propose drilling the tunnels with a drill 1 mm less in diameter than the harvested graft, then enlarging the tunnels with the exact size dilator to minimise the early enlargement.
which might be produced by the pendulum movement of the drill and the necrosis.

Tunnel enlargement after ACL reconstruction was first described with allograft use (2). With techniques fixing the graft far from the joint, like the Endobutton system, tunnel enlargements were reported more often but the same phenomenon was also reported subsequently with bone – patellar tendon – bone (BTB) autografts (4,9).

Tunnel enlargement after ACL reconstructions is thought to be multifactorial (7,13). There are authors blaming biological factors and others blaming mechanical factors (12,14). Heat necrosis, graft movement inside the tunnel, early and aggressive rehabilitation and non-specific inflammatory response have all been considered possible factors in the pathophysiology of tunnel enlargement (9,11). Synovial fluid presence inside the tunnels is reported to facilitate graft movement, creating the so-called windshield wiper effect, leading to enlargement of the tunnel (8,9,14).

Many studies discussed tunnel enlargement, but none of these could clarify when this process of tunnel enlargement started. In our study, tunnel diameters were measured right after the operation (postoperative day 1 to 3) and found to be larger.

Table II. — Serial CT measurements (mean and range) of the diameters of the tibial and femoral tunnels

<table>
<thead>
<tr>
<th></th>
<th>1-3 days</th>
<th>3rd week</th>
<th>6th week</th>
<th>6th month</th>
<th>12th month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral tunnel</td>
<td>0.23 mm (0.2-0.4 mm)</td>
<td>0.64 mm (0.5-0.8 mm)</td>
<td>0.84 mm (0.7-1.1 mm)</td>
<td>1.2 mm (0.9-1.3 mm)</td>
<td>1.1 mm (0.9-1.2 mm)</td>
</tr>
<tr>
<td>Tibial tunnel</td>
<td>0.21 mm (0.2-0.3 mm)</td>
<td>0.43 mm (0.4-0.6 mm)</td>
<td>0.61 mm (0.5-0.8 mm)</td>
<td>0.83 mm (0.7-0.9 mm)</td>
<td>0.85 mm (0.7-1.0 mm)</td>
</tr>
</tbody>
</table>

Table III. — Clinical scores and KT–1000 measurements (n = 62)

<table>
<thead>
<tr>
<th></th>
<th>Postoperative</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Preoperative</td>
</tr>
<tr>
<td>IKDC</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>0</td>
</tr>
<tr>
<td>Nearly normal</td>
<td>0</td>
</tr>
<tr>
<td>Abnormal</td>
<td>45.4%</td>
</tr>
<tr>
<td>Severely abnormal</td>
<td>54.6%</td>
</tr>
<tr>
<td>LYSOLM</td>
<td>60.2 ± 11.6</td>
</tr>
<tr>
<td>KT-1000(89N)</td>
<td>4.9 ± 1.3</td>
</tr>
</tbody>
</table>

Fig. 3. — CT scan of the same patient, one year after operation. Femoral tunnel : 9.4 mm, Tibial tunnel : 9.4 mm.

Fig. 4. — Two years after operation: Thickening of the sclerosis.
than the drill size used. Also, a sclerotic rim was observed in all patients, possibly due to necrotic tissue and compacted bony debris created during drilling. These sclerotic rims were found to have disappeared after three weeks, leading to further tunnel enlargement. Fahey et al. (3) referred to resorption of the necrotic bone as one of the potential aetiologies of tunnel enlargement.

The tunnel enlargements noted in the measurements after three weeks were thought to occur due to circumferential heat necrosis of the cancellous bone during tunnel drilling. After the third week, this necrotic rim is resorbed with a giant cell reaction, leaving a larger diameter tunnel.

Webster et al. (15) postulated that the tunnel enlargements occurring in the first 4 months postoperatively do not decrease during the first and second postoperative years, and aggressive rehabilitation was considered to be the aetiologic factor for tunnel enlargement.

In this study, tunnel enlargement was an early postoperative finding, as 50% of the total enlargement occurred in the first three months. The enlargement was progressive until the 6th month and especially on the femoral side the tendon movement due to distant fixation of the graft was blamed as the causative factor. After the sixth month, the enlargements stopped progressing and after two years tunnel diameters were found to slightly decrease, which was attributed to sclerosis formation.

A possible solution to this problem may be to drill the tunnels to a diameter 1 mm less than the graft, then to enlarge the tunnels with the graft diameter dilator. This is what we have been doing after completion of this study.

REFERENCES


