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Review article

Extensor tendon ruptures after total knee arthroplasty

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ABSTRACT

Extensor tendon rupture is a rare but serious complication after total knee arthroplasty (TKA) that impairs active knee extension, thereby severely affecting knee function. Surgery is usually required. Surgical options range from simple suturing to allograft reconstruction of the entire extensor mechanism and include intermediate methods such as reconstruction using neighbouring tendons or muscles, synthetic ligament implantation, and partial allograft repair. Simple suturing carries a high failure rate and should therefore be routinely combined with tissue augmentation using a neighbouring tendon or a synthetic ligament. After allograft reconstruction, outcomes are variable and long-term complications common. Salvage procedures for managing the most severe cases after allograft failure involve reconstruction using gastrocnemius or vastus flaps. Regardless of the technique used, suturing must be performed under tension, with the knee fully extended, and rehabilitation must be conducted with great caution. Weaknesses of available case-series studies include small sample sizes, heterogeneity, and inadequate follow-up duration. All treatment options are associated with substantial failure rates. The patient should be informed of this fact and plans made for a salvage option. Here, the main techniques and their outcomes are discussed, and a therapeutic strategy is suggested.

1. Introduction

Extensor apparatus rupture is a rare but serious complication after total knee arthroplasty (TKA) that causes a deficit of active knee extension, thereby severely impairing knee function. The rupture creates a breach in the extensor mechanism of the knee, which includes the quadricipital tendon, the patella, the patellar tendon, and the attachment of the patellar tendon to the anterior tibial tuberosity. After TKA, extensor apparatus rupture occurs in 0.17% to 2.5% of cases [1]. The patellar tendon is involved in 0.17% [2] to 1% [1,3,4] of cases and the quadricipital tendon in 0.1% [5] to 1.1% [6] of cases.

The many available treatment options range from simple suturing to various allograft techniques and include reconstruction using neighbouring tendons or muscles and implantation of synthetic ligaments. The small sample sizes, heterogeneity, and inadequate follow-up in published case-series are obstacles to a meaningful analysis [4]. Thus, whether any technique is superior over the others remains unknown.

2. Background

The rupture may be located in the body or at the distal insertion of either tendon [7]. Distal rupture or avulsion of the patellar tendon is the most common form [3,4,8]. In chronic tendon ruptures, retraction of the quadriceps severely compromises the feasibility of direct repair.

The rupture often seems to occur during a traumatic event, which may be obvious (fall on the flexed knee) or trivial (e.g., arising from a chair or carrying a heavy weight). This event merely reveals a chronic abnormality or abnormal fragility of the tendon [3].

The division of arteries during TKA can weaken the extensor mechanism by compromising its blood supply. The following arteries are at risk:

- medial and descending genicular arteries during medial arthrotomy;
- lateral-inferior genicular artery and anterior tibial recurrent artery during fat pad excision and lateral meniscectomy;
- lateral superior genicular artery during release of the lateral retinaculum [1,3,6,9].

Extensive dissection consistently worsens the compromise to the blood supply (Fig. 1).

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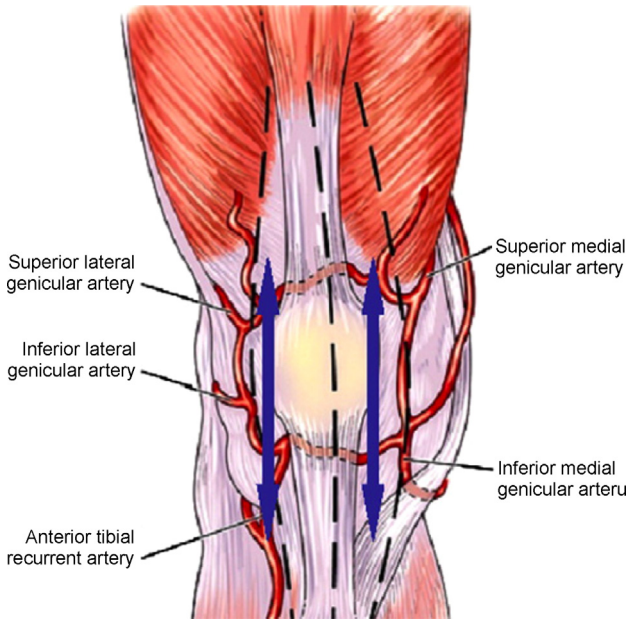

Fig. 1. Blood supply to the extensor mechanism. From Pawar et al. J Arthroplasty. 2009;24:636-640. With kind permission from Springer Science and Business Media. Superior lateral genicular artery; inferior lateral genicular artery; anterior tibial recurrent artery; superior medial genicular artery; inferior medial genicular artery. Note that whatever the skin incision (dotted lines) a medial parapatellar arthrotomy damages arteries 4 and 5 and that lateral arthrotomy damages arteries 1, 2 and 3 (black arrows).

Excessive traction on the patellar tendon may cause either a complete rupture or a partial rupture that may become complete over time. Thus, all factors that complicate the approach to the joint increase the risk of extensor tendon rupture, including stiffness [1,2,10,11], previous surgical procedures (tibial tuberosity osteotomy, extensor mechanism re-alignment [1,3,6,8,10], proximal tibial osteotomy [3,10], TKA revision [1]), post-infection sequelae [1,8], patella infera [1,3], and obesity. Knee manipulation under general anaesthesia can disrupt the extensor mechanism, particularly when performed with delay [10,11].

Other predisposing factors have been suggested: overhang of the tibial or patellar prosthesis [3,8,11]; patellar over-resection [1,3,4,11,12]; excessively proximal joint line level causing the tibial plateau to impinge on the patellar tendon [1,3,4,11,12]; excessively distal joint line level; and any prosthetic malposition, particularly in rotation, leading to an increase in patellofemoral stresses [1,3,11,13].

When technical difficulties or errors are contributing factors, the rupture often occurs shortly after the primary or revision TKA [14].

Many comorbidities have been implicated in extensor tendon ruptures, including inflammatory joint disease, diabetes mellitus, hyperthyroidism, lupus, long-term glucocorticoid therapy, chronic renal failure [1,3,5,6,8], and repeated local glucocorticoid injections [5]. When these factors are involved, several years may elapse between the TKA and the tendon rupture [14].

An active knee extension deficit frequently causes severe functional impairment, as the knee cannot be locked. In some patients, the deficit is apparent only when exercising or going up or down stairs. In more severe cases, level walking is not possible without a walking aid and falls occur frequently. Many patients force the knee into hyperextension to allow stable weight bearing, thereby placing stress on the prosthesis fixation [2,15,16]. Finally, extensor mechanism deficiencies increase the risk of dislocation of posterior-stabilised implants [17,18].

3. Diagnosis

The diagnosis may be obvious if a trauma is followed by an inability to lock the knee while walking, complete loss of active knee extension, and a palpable gap in the quadricipital or patellar tendon. A patient who presents with a feeling of insecurity or instability in the absence of any known trauma raises greater diagnostic challenges. Suggestive signs include loss of knee extension strength, extension lag of less than 20° [3,19], inability to fully extend the knee from the fully flexed position [5], and hyperextension to lock the knee while walking.

A comparison of successive weight bearing lateral radiographs may show progressive patellar migration, in the proximal direction if the patellar tendon is distended and in the distal direction if the quadricipital tendon is distended.

Ultrasonography is valuable, particularly in partial ruptures when the clinical findings are not convincing. Furthermore, this investigation provides information on tissue quality [3]. Magnetic resonance imaging (MRI) may be helpful, although artefacts hinder the interpretation of the images.

A neurological cause of quadriceps muscle weakness should be sought by a thorough physical examination and, if needed, by MRI of the lumbar spine and/or electrophysiological testing.

4. Surgical treatments

Several surgical techniques are available. Selection of the best technique is based on local factors (site and duration of the rupture, whether the rupture is partial or complete, and history of surgery and/or infection), the patient's general health status, the degree of discomfort, the functional demands, and the availability of allografts.

The surgical techniques can be classified into five categories:

- simple repair;
- repair with augmentation by a neighbouring tendon;
- reconstruction using an artificial ligament;
- replacement by an allograft;
- salvage techniques.

Table 1 reports the outcomes of these techniques.

4.1. Simple repair

The tendon can be repaired by direct or trans-osseous suturing, using non-absorbable sutures, anchors, or staples [2]. The suture should be protected by placing a cerclage wire (or thick non-absorbable suture) around the patella and tibia, taking care to position the patella at the normal height. The knee is usually immobilised in extension for 6 weeks. The rehabilitation programme should be conducted with great caution until 12 weeks after surgery.

The outcomes are mediocre. In a study by Rand et al. [2], suturing failed in 6 of 9 cases and stapling in 2 of 4 cases. Lynch et al. [6] reported that direct repair performed in 4 cases failed consistently and indicated that chronic rupture may increase the risk of failure. Similarly, in quadricipital tendon ruptures, Dobbs et al. reported a high failure rate [5].

Thus, simple repair has an extremely limited role. The indication is an intra-operative or very recent rupture, in a patient with good-quality yielding tissue. The best indication may be stapling or trans-osseous suturing or an intra-operative distal avulsion [2,8]. Nevertheless, as a general rule, simple repair should always be combined with tissue augmentation.