Chronic extensor mechanism insufficiency in total knee arthroplasty (TKA)

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Abstract Chronic insufficiency of the knee extensor mechanism is a very challenging pathology to treat. An insufficient extensor mechanism negatively affects implant survival and patient outcomes. The causes of insufficiency can be several, and the surgeon has to be prepared to manage them intraoperatively and postoperatively. Various reconstructive techniques have been proposed, but traditional results in patients with a total knee arthroplasty are usually inferior to those patients with native knee joints. It is of primary importance to understand the anatomy, and tailor the correct treatment to the patient.

Keywords Total knee arthroplasty (TKA) · Extensor mechanism disruption · Extensor mechanism reconstruction · Marlex mesh reconstruction · Synthetic reconstruction

Introduction

Extensor mechanism disruptions of the knee are a feared complication after total knee arthroplasty (TKA). The incidence is reported to range from 1 to 12 % [1]. Extensor mechanism

failure is more commonly observed in the chronic settings [1–3]. Management requires meticulous planning, finite execution, and a conservative postoperative rehabilitative protocol.

In general, complications of the extensor mechanism can be divided into six categories [4•]:

- Patellar tendon ruptures
- Quadriceps tendon ruptures
- Patellar crepitus and soft-tissue impingement
- Periprosthetic patellar fractures
- Patellofemoral instability
- Osteonecrosis of the patella

Anatomy and risk factors

From distal to proximal, the extensor mechanism is composed of the tibial tubercle, patellar tendon, patella, and quadriceps muscle group. Other important structures include the patellar retinaculum and adjacent soft tissues. Blood supply is from the recurrent branch of the anterior tibial artery and the descending genicular artery branches (superior lateral, inferior lateral, superior medial, and inferior medial). Both lateral and medial structures can be damaged by surgical approaches [1, 5]. Occasionally, a medial parapatellar arthrotomy and lateral release can lead to disruption of the blood supply to the patella. This combination of exposure and release increases the risk of patellar avascular necrosis by fourfold compared to medial parapatellar arthrotomy alone [6]. Even patellar maltracking or impingement can cause injury to the knee extensor mechanism. For instance, patellar maltracking can injure the quadriceps tendon and damage the patella, leading to a patella fracture [7].

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Of important note, prior surgical interventions can alter the anatomy and also produce fibrotic lesions [4•]. While prior surgical procedures can affect the integrity of the extensor mechanism, so can systemic pathologies and medications [8]. Predisposing factors for rupture are as follows:

- Local corticosteroids injections [8]
- Diabetes [8–10]
- Microtrauma [9]
- Inflammatory disorders (e.g., rheumatoid arthritis) [3]
- Metabolic disorders (e.g., diabetes) [3]
- Iatrogenic lesions (e.g., ACL reconstruction with patellar tendon graft) [11]
- Fluoroquinolone use [12, 13]

Patellar tendon ruptures

Patellar tendon ruptures are rare, with an estimated prevalence less than 1 % in those patients undergoing TKA [1, 2, 14]. Rupture is more common at the tibial tubercle level. Postoperative ruptures can be traumatic or atraumatic. Traumatic ruptures occur secondary to direct trauma (such as a fall on a flexed knee). Intra-substance lesions normally are not traumatic and occur in degenerated tendons. Intraoperative patellar tendon ruptures are typically a result of a difficult exposure in stiff knees, such as those with patella baja or prior surgeries leading to abundant scar tissue formation.

A typical clinical presentation includes knee pain and swelling, with an extensor lag and a palpable defect in the subpatellar portion of the knee with a patella that is felt high in the knee. If an arthrocentesis is performed, usually a hematoma is evacuated. However, in chronic or atraumatic lesions, the aspiration may be unremarkable and the clinical findings may be under-appreciated. In partial extensor mechanism ruptures, while a lag is not present, extension of the knee may be painful.

From an imaging standpoint, plain radiograph in orthogonal projections (anteroposterior (AP) and lateral) are essential. They can show patella alta, an indirect sign of a tendon rupture [15]. A magnetic resonance (MRI) or an ultrasonographic (US) investigation may be helpful in better understanding the level of the rupture, as well as the surrounding soft tissues. A CT scan is normally not helpful and rarely indicated.

Treatment should be tailored to the patients' functional requests and activity level. Patients with partial ruptures, who are low demand or who are not fit for surgical intervention, can be treated conservatively with a hinged knee brace locked in extension while ambulating. In most other cases, surgical intervention is required. Given the high failure rate of primary repair without reconstruction, it is almost always advisable to complete a reconstruction with augmentation.

Various options include hamstring autograft, fresh-frozen or freeze-dried Achilles tendon with bone block, complete extensor mechanism allograft, and synthetic grafts such as Marlex mesh (C.R. Bard, Murray Hill, NJ) [16•] [6, 17]. Synthetic augmentation with Marlex mesh was initially described in 2011 by Browne and Hanssen [16•] and is the preferred technique at our institution. Brown and Hanssen [16•] reported excellent functional and subjective improvements, with <10° extensor lag in almost all patients, normal knee flexion, and statistically improved KSSs.

Quadriceps tendon ruptures

Quadriceps tendon ruptures are much less common than patellar tendon injuries and reported to occur in 0.1 % of all TKAs [18]. Most patients with a quadriceps tendon rupture are elderly [19]. Other risk factors include systemic disorders such as autoimmune inflammatory diseases, diabetes mellitus, renal insufficiency, obesity, and hyperthyroidism or prior surgical procedures (e.g., patellar over-resection, quadriceps snip, V-Y turndown) [3]. Normally, quadriceps tendon ruptures in TKAs are non-traumatic, and an underlying degeneration of the tendon is already present [18]. The patient may not report a history of trauma. However, the clinical presentation is very similar to that of a patellar tendon rupture with pain, swelling, an extensor lag, and the presence of a palpable gap proximal to the patella. In a portion of cases, the clinical presentation is not obvious and the diagnosis can be missed [19].

As with patellar tendons, plain knee radiographs in the AP and lateral planes are paramount in order to evaluate patella position and associated lesions. Again, a MRI or a US to confirm the diagnosis and to study the tissue around the ruptured tendon can be considered.

With partial lesions, treatment is cast immobilization in extension for 4–6 weeks [19, 20]. Traditional repair has yielded poor results (35 % re-rupture rate) with a high complication rate (33 %) [3, 15, 18, 21].

When considering surgical intervention, augmentation is mandatory [18], and the available grafts are the same as for patellar tendon reconstructions: complete extensor mechanism, Achilles tendon allograft, or synthetic augmentation.

Periprosthetic patellar fractures

Patellar fractures are infrequent but can occur with resurfaced and un-resurfaced patellas [22]. Periprosthetic fractures occur more commonly in men than in women, likely due to the higher weight and physical activity level [23]. A reported

greater risk is also known for resurfaced patellas, especially if an excessive resection is performed, or if an implant with a large plug or an uncemented metal-back patella is implanted [4•]. Malpositioning of the prosthesis leading to a patellar maltracking can increase mechanical loading on the patella, enhancing the risks of fracture as well. Some studies have also shown that patella eversion can increase the risk of fracture [24]. In addition, obesity and low bone mass are patient-related risk factors [4•].

Patellar fractures can be a consequence of direct trauma on the anterior aspect of the knee or secondary to a strong contraction of the quadriceps muscle with the foot fixed on the ground. The clinical presentation is the same as other extensor mechanism failures (i.e., pain, swelling, and presence of an extensor lag). Again, AP and lateral radiographs of the knee, as well as a Merchant's view, are helpful. In this scenario, a CT scan may be considered to determine the grade of bone fragmentation and to evaluate component stability.

Ortiguera and Berry [25] classified patellar fractures according to three different parameters: integrity of the extensor mechanism, fixation status of the patellar implant, and quality of the remaining bone. Well-fixed implants and an intact extensor mechanism can lead to a conservative approach with a good result. On the other hand, the need for surgical intervention comes with increased complications, and failure rates that are high [25].

Fractures of the patella can be classified as one of three types using the Ortiguera and Berry classification [25]:

- Type I: Stable implant with an intact extensor mechanism
 - Type II: Intact implant with a disrupted extensor mechanism
 - Type III: Loose implant
- A: Good patellar bone stock (≥ 10 mm of remaining bone stock)
 - B: Poor patellar bone stock (< 10 mm of remaining bone stock or comminution)

The treatment varies based upon location, fracture pattern, remaining bone stock, and extensor mechanism status. Type I defects typically are treated non-operatively with a long-leg cast and immobilization in extension for 4–6 weeks [7, 25].

Type II periprosthetic fractures are traditionally treated with open reduction and internal fixation (ORIF). Of note, 42 % of such patients require additional surgical intervention, and 50 % have complications [4•].

Type III periprosthetic fractures have a loose patella component in 20 % of cases and have a complication rate of 45 % [4•]. In addition, anatomical fracture reduction is not a positive predictor for surgical outcomes, with a high complication rate as well [1, 7, 26–28].

Patellar avascular necrosis

Avascular necrosis (AVN) of the patella is a rare complication after TKA. The incidence is reported to be from 0 % [29] to 1.4 % [30]. However, it is important to note that this is historical data, and contemporary implant designs and techniques that are less invasive and with better instrumentation, may change the risk. Excessive releasing around the patella can affect bone vascularization by damaging blood vessels, thus producing osteonecrosis and patellar complications [31]. Patellar necrosis can also be heat derived from cement polymerization or button impaction on the femoral component during knee flexion over 95° [31]. In some patients, AVN of the patella can be asymptomatic, limiting our ability to determine the true incidence. In symptomatic cases, the presentation is typically anterior knee pain, and it can also be correlated with patellar instability, fracture, or implant loosening [4•]. Again, radiographs should be completed, and occasionally, a MRI is required in order to evaluate AVN extension and soft tissue. MRI is also useful in uncertain diagnoses because it can detect necrosis in an earlier stage than conventional radiographs.

The treatment of patellar AVN is individualized and based on clinical presentation and local factors. If asymptomatic, a conservative approach is appropriate. When symptomatic, revision TKA is required. Patellar revision can be attempted if the bone stock is adequate. Excessive bone loss can affect extensor mechanism integrity and may require a reconstruction or an augmentation.

Fig. 1 The figure above demonstrates two steps in Marlex Mesh reconstruction: graft preparation at the *top* and Marlex Mesh placement in the *bottom* image

Reconstructive surgical techniques

Whole extensor mechanism allograft reconstruction

Allograft reconstructions can be completed with an Achilles tendon with calcaneal bone block or a whole knee extensor mechanism. Allograft choice is based on the anatomy of the residual knee extensor mechanism with particular attention on residual patellar bone. If the patella is not present or the bone loss is significant, a whole mechanism allograft should be considered. If the patella is present and can be mobilized, an Achilles tendon allograft is indicated [6]. Allograft reconstruction grants a better anatomical reconstruction and good incorporation into the host tissue [2]. However, the tissue usually loses tension when in place. For this reason, it is important to fully tension the grafts during surgery [10, 32•]. A reported downside of extensor allograft technique is loss of tension with time leading to recurrence of extensor lag.

The surgical technique involves exposing the residual extensor mechanism, and creating a tibial through a small burr distally and medially to the tibial tubercle. The allograft, meanwhile, is prepared on the back table, and the bone block is trimmed and proximally dovetailed like the through in the tibia in order to prevent proximal migration. The bone block is secured to the tibia with three 16-gauge wires that have to be passed through the tibial shaft and above the bone graft. The proximal part of the allograft is then tensioned and secured on the distal part of the residual extensor mechanism with heavy non-absorbable sutures stitched in a Krachow fashion [4•]. The Achilles allograft can be alternatively passed under the remnant patellar tendon scar tissue and then passed anteriorly through a slit laterally to it. After that, the allograft can be secured to the quadriceps tendon with tension [32•].

Marlex mesh reconstruction

Reconstruction with Marlex mesh is our preferred technique and was previously described by Browne and Hanssen in 2011 [16•]. With this technique, we utilize a knitted monofilament polypropylene mesh. A sheet of 10×14 in. Marlex mesh is folded into a tube of nearly 2.5 cm wide and stitched together with a non-absorbable suture. The mesh is typically secured behind the anterior tibial cortex and in front of the tibial stem through a slot created proximal to the anterior tibial tubercle. During knee revisions in which the tibial component is revised, the mesh can be secured to the tibia with the methylmethacrylate cement used for tray fixation. In both scenarios, a transfixing screw is utilized. The mesh is then passed under the patellar tendon scar tissue and then anterior through an opening lateral to the residual patella. The quadriceps complex is then brought distally, and the mesh is secured to the vastus lateralis with the patella positioned in the trochlea at the level of the anterior flange of the TKA with the knee in full

extension. The mesh has then to be covered with the vastus medialis muscle in a so-called pants-over-vest fashion (Fig. 1) [4•].

Conclusion

Chronic disruption of the extensor mechanism is rare and often devastating to patients. Accurate and timely diagnosis is essential. In low demand patients who are unfit for surgical intervention, those with partial ruptures, or those with non-displaced patellar fractures and patellar AVN, conservative management is the standard of care. With complete ruptures, surgical reconstruction with augmentation is mandatory. Patients should be informed of the severity of the pathology and the increased complexity of the management.

Compliance with Ethics Guidelines

Conflict of Interest Dr. Hanssen has received royalties from Stryker. Drs. Cottino and Abdel have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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