Ruptures of the Quadriceps and Patellar Tendons of the Extensor Mechanism: A Review

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Abstract

Damage to the quadriceps and patellar tendons of the extensor mechanism can be devastating and often life-changing injuries that require prompt diagnosis and treatment. A sound understanding of anatomy, biomechanics, and degenerative changes of both tendons and the extensor mechanism of the knee can help guide surgical repair and postoperative rehabilitation of patients. Immediate primary repair has often resulted in improved postoperative results compared with delayed reconstruction, and the avoidance of gap formation by use of careful techniques and augmentation has been critical for successful treatment. Additionally, patients with extensor mechanism injuries frequently have medical comorbidities or notable tendon degeneration; subsequently, careful consideration of systemic diseases and appropriate medical treatment has been vital to success of operative treatment. I reviewed the anatomy of quadriceps and patellar tendons; biomechanics of the extensor mechanism and tendons; mechanisms of injury; clinical and biomechanical studies on use of surgical techniques for treatment; and postoperative rehabilitation protocols and possible complications. Early diagnosis, awareness of comorbidities, prompt surgical treatment using a careful approach, and a thorough postoperative rehabilitation program may allow patients to return to previous levels of activity, with promising long-term results.

Introduction

Injuries of the extensor mechanism occur relatively infrequently but often require timely diagnosis and treatment. Ruptures of the quadriceps tendon are more common than the patellar tendon in patients aged 40 years and older who have notable medical comorbidities, whereas healthier patients aged 40 years or younger typically have patellar tendon ruptures. Extensor mechanism injuries are much more common in men than women, with a reported ratio of 8:1. This may be explained by the reported earlier onset of osteoporosis in women, which weakens the patella bone. Subsequently, tensile overload of the extensor mechanism has resulted in transverse patella fractures rather than tendon ruptures. Furthermore, systemic diseases have been strongly correlated to bilateral, spontaneous, and low-energy rupture.[1,2]

An understanding of the anatomy of quadriceps and patellar tendons can considerably help a surgeon in caring for patients with these complex injuries. To help investigate successful methods of treatment, I reviewed notable anatomical locations of the patellar and quadriceps tendons; biomechanics of the extensor mechanism; mechanisms of injury and other possible causes of tendon rupture; methods used to help appropriately diagnose the injury during clinical evaluation; various operative techniques; process of rehabilitation; and possible postoperative complications.

Tendon Anatomy

Tendons are relatively avascular and rely on intrinsic and extrinsic systems for nutritional and healing support. The level of function of the extrinsic and intrinsic systems decreases with age, presence of systemic disease, and mechanical loading of force, resulting in a weakened tendon with decreased regeneration and healing potential.[3]

Quadriceps Tendon Anatomy

The quadriceps tendon is a coalescence of the tendinous portion of quadriceps muscles that form three to four distinct layers, ranging from most anteriorly located to deepest within the tissue: rectus femoris, vastus lateralis, vastus medialis, and vastus intermedius, respectively.

In 2008, Yepes et al[4] published an anatomical study with a proposed subdivision of the quadriceps tendon into three zones based on vascular supply and resultant common sites of rupture. By distance from the superior pole of the patella, zone 1 was at 0 to 1 cm; zone 2, 1 to 2 cm; and zone 3, greater than 2 cm. The most common site of rupture was zone 2 (41%), followed by zone 1 (37%) and zone 3 (12%). The high amount of zone 1 ruptures may have been explained by subperiosteal bone resorption caused by systemic diseases, which had weakened the site of attachment between tendon and bone.

Patellar Tendon Anatomy

The patellar tendon consists of 90% and less than 10% of types 1 and 3 collagens, respectively. Elastin, proteoglycan, and glycoproteins compose the remaining dry weight. Water makes up between 60% and 70% of the wet weight of the tendon, and collagen composes from 70% to 80% of the dry weight. The proximal width of the tendon approximates the width of the patella, which narrows and thickens as it extends distally. The average length of the patellar tendon spans 50 mm (SD, 5 mm).

The infrapatellar fat pad provides the main blood supply of the patellar tendon, which occurs by anastomoses from the genicular and recurrent tibial arteries through the patellar retinaculum. Similar to the distal attachment of the quadriceps tendon, the proximal and distal attachments of the patellar tendon are relatively avascular and prone to rupture.[1]

Biomechanics of the Extensor Mechanism

The function of the extensor mechanism allows for standing, bipedal ambulation, rising from a chair, and performing stairs. The force necessary to extend the knee against gravity to accomplish these tasks is called torque. In comparison with bringing the knee from a fully flexed position to less than 15° of extension, twice as much torque is needed to extend the knee to the final 15° of terminal extension.

The patella provides the constant torque and mechanical advantage needed by two separate mechanisms: linking and displacement. The linking mechanism generates torque from the quadriceps muscle to the tibia, whereas displacement helps generate the extra torque needed to reach full extension. As the knee extends, the patella slides out of the groove and onto the anterior cortex of the femur, which displaces the patella from the knee axis of rotation and generates 605 N of torque to gain the last 15° of full extension. This mechanical advantage indicates an important relationship between the patella and femur during arthroplasty and reconstruction of the extensor mechanism.[5]

The relative force applied to each tendon depends on the degree of knee flexion and ability of the patella to tilt in the sagittal plane in relationship to the trochlear groove. The greatest force undertaken by the extensor mechanism occur with the knee at 60° of flexion. At 30° or greater than 90° of flexion, the quadriceps-tendon force is 30% less or greater than the patella-tendon force, respectively.[2]

Mechanism of Injury

The most common mechanism of injury of quadriceps and patellar tendons has been forceful quadriceps contraction (ie, sudden force of the patient's body weight, with the knee in a flexed position).[1] Eccentric loading of the extensor mechanism often occurs when the foot and knee are planted and slightly bent, respectively, such as in an attempt to regain balance for avoiding a fall. This time-dependent, sudden strain with the knee in the position of greatest force (60° of flexion) causes tensile overload of the extensor mechanism, resulting in failure to extend the knee. Because the patella is considered to be the weak link of the structure, this mechanism commonly results in a transverse patella fracture in patients with intact quadriceps and patellar tendons. If damage occurs at the tendons, the cause of extensor mechanism failure becomes tendon rupture and avulsion.

In younger patients, the mechanism of injury has often been direct trauma, either blunt (ie, football helmet to the knee) or penetrating (ie, laceration from edges of ski blades). The three typical patterns of extensor mechanism injury, ranging from most common to least, are avulsion with or without bone fragments from the poles of the patella; midsubstance rupture; and distal avulsion from the tibia tubercle.[1]

Systemic Diseases and Degenerative Changes

Possible factors that affect tendon health have been grouped into two general categories: systemic diseases and degenerative changes. Systemic diseases include chronic renal failure; uremia; diabetes; chronic inflammation and synovitis of rheumatoid arthritis; hyperparathyroidism; and connective-tissue disorders. Degenerative changes result from microscopic injury to the tendon owing to repetitive force applied in the most severe "plastic phase" of tendon deformation (≤ 10% strain).[1]

A study published in 2011 by Wani et al[6] detailed the importance of simultaneous medical treatment in patients with long-term hemodialysis of chronic kidney disease and tendon ruptures. Concerning degenerative changes, Kannus and Józsa[7] evaluated 891 biopsy specimens with spontaneously ruptured tendons (of which 53 were patellar tendons) and noted that 97% had degenerative characteristics, mainly hypoxic tendinopathy, mucoid degeneration, tendolipomatosis, and calcifying tendinopathy. To note, multiple studies have reported on "jumper's knee," or quadriceps-patella tendinitis, which results from repetitive overloading of force on the extensor mechanism.[8-10] The injury has been shown to be a significant risk factor for ruptures of the patellar tendon.[10]

Clinical Evaluation

After initial injury of quadriceps and patellar tendons, patients present with the inability to actively extend the knee or maintain extension against gravity. Findings of radiographs may be essential in confirming diagnosis of extensor mechanism injuries and should be obtained before other imaging procedures are performed.

Physical Examination

Missed diagnosis of injury to the quadriceps and patellar tendons is relatively common and has been reported in 10% to 15% of patients without and 30% of patients with concomitant injuries.[1,11] Signs of limited active extension of the knee have been confused with an intact sartorius muscle.[11] An intact patellar retinaculum may allow for the limited active extension, in which considerable weakness and extensor lag exist compared with the uninjured leg when patients maintain extension against gravity. Use of knee aspiration and intra-articular anesthetic injection may improve the sensitivity of the examination to health of tendons. The presence of suprapatellar gap and palpable depression has been an indicator for possible disease of quadriceps tendons with ruptures. Furthermore, hemarthrosis can prevent the palpation of a gap and aspiration of the knee joint. Additionally, flexion at the hip to shorten the length of the rectus femoris (crosses hip joint) and increase the gap length may improve the sensitivity of the examination.

Imaging Procedures

Radiographs should be critically evaluated for the presence of patella alta and possible bone fragments that may be attached to the tendon after avulsion-type injuries. Patella alta or baja can be identified by the relationship of the patella to the Blumensaat line on a lateral radiograph, showing the knee flexed to 30°. Additionally, several standardized measurements can be used to assess patella position.

Ultrasonography has been a relatively cost-effective and timely imaging procedure, with acceptable specificity for ruptures of the quadriceps and patellar tendons. Disadvantages have included operator- and reader-dependent natures of the modality. Results of magnetic resonance imaging (MRI) are useful in diagnosing chronic injuries and partial ruptures and determining location of the disruption. The major disadvantages of MRI procedures are the cost and limited availability at some centers.

Treatment

Immediate primary repair within 72 hours after initial injury is the standard for successful operative treatment of quadriceps and patellar tendon injuries. Surgical treatment after 72 hours can result in difficulty with apposition of tendon edges and increase tension on suture lines. Notably, nonoperative treatment is associated with limited results in patients with incomplete rupture and should be reserved for patients with low levels of activity, who may not successfully undergo operative procedures. Findings of MRI help justify the use of nonoperative techniques because most views of the extensor mechanism should be intact. Nonoperative treatment typically consists of immobilization for 6 weeks, followed by limited activities involving high range of motion of the knee.

Operative Procedures

Several well-accepted techniques used during primary repair have helped minimize gap formation, including use of Krackow stitches,[12-14] suture anchors,[15,16] and augmentation to reduce tension placed on the suture.[17]

Although the gold standard of primary repair for treating extensor mechanism injuries has involved use of Krackow stitches, a biomechanical study by McKeon et al[13] reported no difference in maximum strength compared with use of a whipstitch, in which results were attributed to suture rupture rather than suture pull out. Petri et al[15] compared results of using theoretically improved suture anchors for repairing quadriceps and patellar tendon ruptures in patients, in which significantly high levels of maximum strength before failure that resulted in decreased gap formation were noted.[16] In 2011, Massoud[17] found promising results with use of augmented suture cerclage, with no cases of re-rupture or radiographic evidence of patella alta, patella baja, and patella-femoral degenerative changes in the tendons.

Postoperative Rehabilitation and Long-Term Outcomes

In rehabilitation of patients with injuries of the quadriceps and patellar tendons who were treated with operative repair, use of strict immobilization in a cylinder cast or knee immobilizer has been favored, particularly for treating quadriceps tendon ruptures in patients with systemic diseases. The other method of rehabilitation has involved early controlled motion, in which controlled tensile force promotes increased collagen production of the tendons, improved tendon architecture, and decreased scar formation. No consensus exists on the most effective method of postoperative rehabilitation because studies on either protocol have reported similar results.[18-21]

In general, the long-term results of immediate primary surgical repair for treating acute ruptures of quadriceps and patellar tendons have been promising. In 2014, Boudissa et al[18] reported a series of 50 acute quadriceps tendon ruptures, with a mean follow-up of 6 years, in which 97% of patients had regained full extension of the knee and mean flexion of 125°. No significant difference in level of function was noted when the results were compared with those of studies on early range of motion methods. Concerning patients with chronic ruptures and systemic diseases, Malta et al[19] reported that results of treatment were significantly worse with acute ruptures in healthy patients, although biochemical alterations of the tendon structure and delayed time to operative treatment may have affected findings. Finally, patients with patellar tendon ruptures tend to be young and healthy, and subsequent long-term results of postoperative treatment have been promising.[20,21]

Complications

Loss of range of motion of the knee and particularly active terminal extension (extensor lag) have been common postoperative complications after treating extensor mechanism injuries. Many techniques and modifications to rehabilitation protocols have been used to avoid postoperative extensor lags. The combination of tensioning the repair in full extension of the knee and protected methods of controlled motion has improved results by decreasing the degree of extensor lag while achieving acceptable knee flexion. Atrophy and weakness of the quadriceps muscle have been recognized as considerable postoperative complications. In 1981, Siwek and Rao[22] reported quadriceps atrophy of 2 to 4 cm at final follow-up but did not find clinical significance because the level of weakness still allowed for adequate strength of normal knee function.

Furthermore, because most patients with extensor mechanism injuries have comorbidities and low levels of activity, wound complications and postoperative infections occur more frequently than noted with other operative procedures for treating knee injuries.[23] The subcutaneous positioning of wires and large caliber sutures may contribute to this high rate of wound complications and infections, which can be minimized with avoidance of placing sutures in line with the incision, careful placement of knots, and tension-free closures of the wound. Patella baja, resultant loss of motion, and potential for patellofemoral degeneration should be carefully considered at the time of operative procedure because careful anatomical restoration of patella position and biomechanics can reduce complications. Finally, re-rupturing of tendons has required revision surgery, which has been reported in between 1% and 8% of cases.[10,20,23]

Conclusion

A general knowledge of quadriceps and patellar tendon structure, anatomy, and biomechanics may greatly improve the ability to diagnose, surgically treat, and appropriately direct postoperative rehabilitation of extensor mechanism injuries. Special attention to comorbidities of patients is critical to successful treatment. Additionally, many operative procedures and techniques warrant consideration (ie, type of suture, type and number of throws, and pre-loading methods). Use of augmentation methods and base-level rehabilitation plans should be employed for treating individual patients, which vary depending on patient characteristics and type and degree of injury. Time to operative procedure after initial injury is crucial, with the universally accepted notion that chronic injuries worsen quickly and considerably. A timely diagnosis and immediate yet careful primary repair, followed by a thoroughly directed physical therapy program, may result in a reliable return to previous levels of daily activities, work, and sports of patients.

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Conflict of Interest

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References

1. Matava MJ. Patellar tendon ruptures. J Am Acad Orthop Surg 1996;4(6):287-296.
2. Ilan DI, Tejwani N, Keschner M, Leibman M. Quadriceps tendon rupture. J Am Acad Orthop Surg 2003;11(3):192-200.
3. Sharma P, Maffulli N. Tendon injury and tendinopathy: healing and repair. J Bone Joint Surg Am 2005;87(1):187-202.
4. Yepes H, Tang M, Morris SF, Stanish WD. Relationship between hypovascular zones and patterns of ruptures of the quadriceps tendon. J Bone Joint Surg Am 2008;90(10):2135-41.
5. Browner BD, Jupiter JB, Levine AM, Trafton PG. Skeletal Trauma: Basic Science, Management, and Reconstruction. Vol 2. 3rd ed. Philadelphia, PA: Saunders, 2003.

6. Wani NA, Malla HA, Kosar T, Dar IM. Bilateral quadriceps tendon rupture as the presenting manifestation of chronic kidney disease. Indian J Nephrol 2011;21(1):48-51.
7. Kannus P, Józsa L. Histopathological changes preceding spontaneous rupture of a tendon: a controlled study of 891 patients. J Bone Joint Surg Am 1991;73(10):1507-25.
8. Zwerver J, Bredeweg SW, van den Akker-Scheek I. Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. Am J Sports Med 2011;39(9):1984-8.
9. Lian OB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. Am J Sports Med 2005;33(4):561-7.
10. Boublik M, Schlegel T, Koonce R, Genuario J, Lind C, Hamming D. Patellar tendon ruptures in National Football League players. Am J Sports Med 2011;39(11):2436-40.
11. Brunkhorst J, Johnson DL. Multiligamentous knee injury concomitant with a patellar tendon rupture. Orthopedics 2015;38(1):45-8.
12. Hahn JM, InceoÄŸlu S, Wongworawat MD. Biomechanical comparison of Krackow locking stitch versus nonlocking loop stitch with varying number of throws. Am J Sports Med 2014;42(12):3003-8.
13. McKeon BP, Heming JF, Fulkerson J, Langeland R. The Krackow stitch: a biomechanical evaluation of changing the number of loops versus the number of sutures. Arthroscopy 2006;22(1):33-7.
14. Krushinski EM, Parks BG, Hinton RY. Gap formation in transpatellar patellar tendon repair: pretensioning Krackow sutures versus standard repair in a cadaver model. Am J Sports Med 2010;38(1):171-5.
15. Petri M, Dratzidis A, Brand S, et al. Suture anchor repair yields better biomechanical properties than transosseous sutures in ruptured quadriceps tendons. Knee Surg Sports Traumatol Arthrosc 2015;23(4):1039-45.
16. Ettinger M, Dratzidis A, Hurschler C, et al. Biomechanical properties of suture anchor repair compared with transosseous sutures in patellar tendon ruptures: a cadaveric study. Am J Sports Med 2013;41(11):2540-4.
17. Massoud EI. Repair of fresh patellar tendon rupture: tension regulation at the suture line. Int Orthop 2010;34(8):1153-8.
18. Boudissa M, Roudet A, Rubens-Duval B, Chaussard C, Saragaglia D. Acute quadriceps tendon ruptures: a series of 50 knees with an average follow-up of more than 6 years. Orthop Traumatol Surg Res 2014;100(2):213-6.
19. Malta LM, Gameiro VS, Sampaio EA, Gouveia ME, Lugon JR. Quadriceps tendon rupture in maintenance haemodialysis patients: results of surgical treatment and analysis of risk factors. Injury 2014;45(12):1970-3.
20. Roudet A, Boudissa M, Chaussard C, Rubens-Duval B, Saragaglia D. Acute traumatic patellar tendon rupture: early and late results of surgical treatment of 38 cases. Orthop Traumatol Surg Res 2015;101(3):307-11.
21. West JL, Keene JS, Kaplan LD. Early motion after quadriceps and patellar tendon repairs: outcomes with single-suture augmentation. Am J Sports Med 2008;36(2):316-23.
22. Siwek CW, Rao JP. Ruptures of the extensor mechanism of the knee joint. J Bone Joint Surg Am 1981;63(6):932-7.
23. Negrin LL, Nemecek E, Hajdu S. Extensor mechanism ruptures of the knee: differences in demographic data and long-term outcome after surgical treatment. Injury 2015;46(10):1957-63.