Patellar Tendon Ruptures

Matthew J. Matava, MD

Abstract

Rupture of the patellar tendon is a relatively infrequent, yet disabling, injury, which is most commonly seen in patients less than 40 years of age. It tends to occur during athletic activities when a violent contraction of the quadriceps muscle group is resisted by the flexed knee. Rupture usually represents the final stage of a degenerative tendinopathy resulting from repetitive microtrauma to the patellar tendon. This injury may also occur during less strenuous activity in patients whose tendons are weakened by systemic illness or the administration of local or systemic corticosteroid medications. The diagnosis is made on the basis of the presence of a painful, palpable defect in the substance of the tendon; an inability to completely extend the knee against gravity; and the existence of patella alta confirmed by lateral radiographs. Ultrasonography and magnetic resonance imaging are useful in identifying a neglected rupture, as well as when the diagnosis is in question or an intra-articular injury is suspected. The prognosis after a patellar tendon rupture depends in large part on the interval between injury and repair. Surgery soon after the injury is recommended for optimal results. This is best accomplished by accurate reapproximation of the ruptured tendon ends, repair of the torn extensor retinacula, and placement of a reinforcing cerclage suture. An aggressive rehabilitation program, emphasizing early range-of-motion exercises, protected weight bearing, and quadriceps strengthening, will enhance the results of surgery. Patients who undergo delayed repair are at risk for a compromised result secondary to loss of full knee flexion and decreased quadriceps strength, although a functional extensor mechanism is likely to be reestablished.

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Rupture of the patellar tendon is a relatively infrequent injury and is thought to occur less frequently than rupture of the quadriceps tendon. It is usually seen in active patients under the age of 40[1]; with the increased emphasis on participation in athletic activities by all age groups, however, it is now not uncommon to see this injury in older patients.

The vast majority of these injuries are unilateral. Cases of bilateral ruptures have been described in association with systemic disease states known to weaken collagen structures.[2] In these instances, rupture may occur during less strenuous, nonathletic activity.

Anatomy

Technically, the term "patellar tendon" is a misnomer, since the "tendon" is actually a ligament connecting one bone (the patella) to another (the tibia). The patella is merely a sesamoid bone within a tendinous complex. For the purposes of this discussion, however, the term will be used, because the structure represents the thickened condensation of the quadriceps tendon that continues over the patella before inserting onto the tibial tubercle.

The extensor mechanism of the knee includes the quadriceps femoris muscle group, the quadriceps tendon, the patella, the patellar tendon, and the tibial tubercle (Fig. 1). The quadriceps femoris group consists of four muscles: the rectus femoris, the underlying vastus intermedius, the vastus medialis, and the vastus lateralis. The tendinous expansion of the vastus lateralis to the superolateral border of the patella and proximal tibia is referred to as the lateral retinaculum. The tendon of the vastus medialis inserts into the superomedial border of the patella and proximal medial tibia as the medial retinaculum. Fibers of the rectus femoris tendon traverse the anterior surface of the patella to form a thickened, fibrocartilaginous con-

Dr. Matava is Assistant Professor, Department of Orthopaedic Surgery, Section of Sports Medicine, Washington University, St Louis.

Reprint requests: Dr. Matava, Washington University Department of Orthopaedics, One Barnes Hospital Drive, Suite 11300, West Pavilion, St Louis, MO 63110.

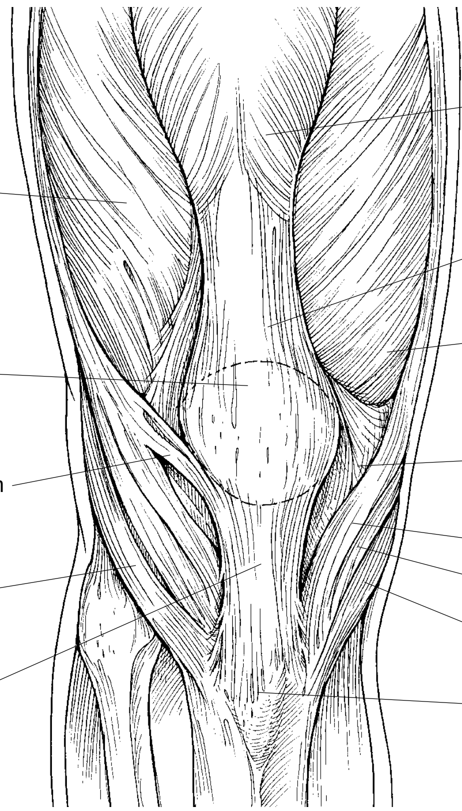


Fig. 1 Normal anatomy of the extensor mechanism of the knee.

densation inferior to the patella before inserting onto the tibial tubercle.

Proximally, the width of the tendon approximates that of the adjacent patella. Its thickness in the sagittal plane usually averages 4 mm and does not exceed 7 mm.[3] The tendon thickens and narrows distally, with an average thickness of 5 to 6 mm at the tibial tubercle. The patellar tendon merges medially and laterally with the medial and lateral retinacula, respectively. Injuries to the tendon usually involve the adjacent retinacula, causing dysfunction of the entire extensor hood.

Water constitutes 60% to 70% of the wet weight of the patellar tendon, and collagen comprises 70% to 80% of the dry weight. The patellar tendon is composed of large-diameter (greater than 100 nm) collagen fibers, of which 90% are type I collagen and fewer than 10% are type III. Elastin, proteoglycan, and other noncollagenous glycoproteins make up the remaining tendon matrix.[4]

The patellar tendon receives its blood supply primarily from the infrapatellar fat pad and retinacular structures.[5] The infrapatellar fat pad contributes vessels to the posterior aspect of the tendon via anastomoses from the inferior medial and inferior lateral genicular arteries. The retinaculum supplies blood vessels to the anterior portion of the tendon from branches of the inferior medial genicular and recurrent tibial arteries. These vessels originate at the proximal and middle portions of the tendon and travel distally. Both the proximal and distal attachments of the patellar tendon are relatively avascular.[6] These avascular regions, composed histologically of fibrocartilage, are the most common sites of rupture.

Biomechanics of the Extensor Mechanism

During active knee extension, forces generated in the quadriceps muscle group are transferred in a convergent fashion via the patellar tendon and retinacula to the proximal tibia. The greatest forces in the tendon occur with the knee at approximately 60 degrees of flexion.[7] The ratio of patellar tendon force to quadriceps tendon force has been found to be greater than 1.0 at knee flexion angles less than 45 degrees. At these smaller flexion angles, the patellofemoral contact area is located at the distal end of the patella, giving the quadriceps tendon a mechanical advantage during active knee extension.[7] At flexion angles greater than 45 degrees, the force ratio is less than 1.0. With increasing knee flexion, the patellofemoral contact area shifts proximally, giving the patellar tendon a mechanical advantage during active extension.[7] It is then that the tendon sustains relatively greater stress compared with the quadriceps tendon. Consequently, most ruptures tend to occur with the knee in a more flexed position.[8]

It has been shown that the deformation (strain) due to tensile load is much greater in the insertion sites of the patellar tendon than in the midsubstance of the tendon.[9] At peak load prior to failure, this end-region strain is approximately three to four times that in the midsubstance.[9] In addition, there is a corresponding decrease in collagen-fiber stiffness at the insertion site compared with the midsubstance region. It is theorized that this differential in peak strain exists because the collagen-fiber crimp period of the end region is shorter, and the crimp angle is larger, than that of the midsubstance.[9] These differences in in vivo force transmission may explain why the patellar tendon most commonly ruptures near its proximal insertion site, rather than in its midsubstance.

Forces generated in the patellar tendon during normal activities of daily living have been estimated with the use of geometric and trigonometric methods. The force generated while ascending stairs is believed to be approximately 3.2 times body weight.[10] A detailed cinematographic analysis by Zernicke et al[8] estimated that the force necessary to cause a patellar tendon rupture in a weight lifter approached 17.5 times body weight. It is presumed that the force required to rupture a tendon weakened by systemic disease is much lower, although no data are available to substantiate this hypothesis.

Causes of Tendon Rupture

It is generally accepted that healthy tendons do not rupture. Tensile overload of the extensor mechanism usually leads to a transverse fracture of the patella, which is considered the weakest link.[2] Patellar tendon rupture due to indirect trauma has been considered the end stage of a long-standing chronic tendon degeneration secondary to repetitive microtrauma.[11] Kannus and Józsa[12] evaluated 891 biopsy specimens of spontaneously ruptured tendons, including 53 patellar tendons. All of the ruptured tendons exhibited pathologic changes, whereas such changes were detected in only 35% of intact tendons from age-matched control subjects. Ninety-seven percent of the pathologic findings were degenerative in nature, including hypoxic tendinopathy, mucoid degeneration, tendolipomatosis, and calcifying tendinopathy.

Kelly et al[11] reviewed the data on 13 athletes with chronic symptoms of jumper's knee resulting in tendon failure. Ten of these athletes had patellar tendon ruptures (1 had bilateral ruptures), and 3 had quadriceps tendon ruptures. Those patients younger than 25 years of age demonstrated more severe symptoms of preexisting patellar tendinitis before rupture. Older patients complained of less severe symptoms, presumably due to a history of less severe microtrauma. The authors hypothesized that more advanced degeneration is required to critically weaken a younger, healthier tendon, whereas less advanced microtrauma, combined with the natural degeneration associated with aging, leads to rupture in the older athlete.

Patients with preexisting systemic disorders, such as systemic lupus erythematosus, rheumatoid arthritis, chronic renal failure, and diabetes mellitus, which are known to weaken collagen structures, are susceptible to patellar tendon ruptures during nonstrenuous activity.[2] This represents a significant injury because the disability of the tendon rupture is exacerbated by the comorbid condition, making tendon repair and its rehabilitation especially difficult.

This condition has also been seen in patients who were being treated with long-term systemic corticosteroid therapy.[13] Bilateral rupture may occur in these patients.[2,13] Rupture may also occur after the local injection of a corticosteroid near the tendon as treatment for chronic patellar tendinitis or jumper's knee.[14] Corticosteroid medications are known to weaken collagen structures by causing necrosis of the fibrils and disorganization of the collagen arrangement.[15] In the series by Kelly et al,[11] 8 of 13 patients treated for jumper's knee received an average of two to three steroid injections in or around the patellar tendon before rupture. Because of this potential complication, local steroid injection near the patellar tendon or its bone insertions is strongly discouraged.

Patellar tendon rupture has occurred after some surgical procedures that disturb the midsubstance or insertion sites of the patellar tendon. Although an infrequent complication of total knee arthroplasty, rupture has been associated with hinged designs, component malalignment, tendon impingement, removal of excess patellar bone, and devascularization secondary to overly aggressive lateral release.[16] In these instances, treatment has been universally discouraging, with no single technique providing consistently good results. Primary repair, autogenous hamstring-tendon augmentation, and extensor-mechanism allografts have been performed with some success.[16] It is recommended that primary suturing of the torn tendon ends be attempted if adequate local tissue is present. Allografts may be considered as a later method of salvage. Generally, the prosthesis does not require revision, nor is this recommended, due to the increased risk of wound morbidity.

Less commonly, patellar tendon rupture has been described after anterior cruciate ligament reconstruction performed with use of the central third as a reconstructive graft.[17] Proximal tendon rupture or avulsion of the distal pole of the patella usually occurs when the patient becomes too active before complete healing of the graft harvest site has occurred. Primary tendon repair is again recommended. This tends not to compromise the result of the initial surgery, as may occur with a patellar tendon rupture after total knee replacement.

Clinical Evaluation

History and Physical Examination

The history is often very helpful in diagnosing a patellar tendon rupture. Almost invariably, the patient has sustained a forceful quadriceps contraction against a fixed or sudden load of the full body weight, placing the knee in a flexed position (e.g., landing after a rebound, tripping up the stairs). Sudden pain with an associated tearing or popping sensation is a hallmark of the injury. The patient is unable to continue the activity, and weight bearing is impossible without assistance.

The patient usually presents with a tense hemarthrosis of the knee and inability to bear weight on the involved extremity. Intra-articular injury (e.g., an anterior cruciate ligament or meniscal tear) must be ruled out during the initial evaluation because of the similar mechanisms of injury. The most significant finding supporting a diagnosis of patellar tendon rupture is a lack of active knee extension or inability to maintain the passively extended knee against gravity. If the rupture extends completely through the tendon and retinacula, there will be total loss of active extension. Less commonly, if the rupture involves only the tendon and most of the retinacular fibers remain intact, some extension will be possible, although several degrees will still be lacking.[1] Soon after the injury, there is often a palpable gap at the level of the rupture, and the patella may feel proximally displaced compared with the contralateral side. Passive knee flexion is markedly diminished because of pain.

When the diagnosis is delayed, the tendon defect may be obscured by consolidation of hematoma and early formation of scar tissue. Some active knee extension usually is possible, although an extensor lag will be present. Quadriceps atrophy and an elevation in patellar height are usually seen. Weight bearing may be possible, but often with considerable weakness, leading to a forward flinging motion of the affected leg in the swing phase and feelings of knee instability during single-leg stance.[18] Activities such as stair climbing and rising from a chair are exceedingly difficult.

Radiologic Studies

Plain Radiography

At least anteroposterior and lateral plain radiographs should be obtained in all patients presenting with an acute significant traumatic injury to the knee. Special views (e.g., Merchant and tunnel) may also be helpful in ruling out a patellar dislocation or an osteochondral fracture. Weight-bearing views are unnecessary and are often not even feasible because of pain. Plain x-ray films are the most cost-effective radiographic means by which to diagnose a patellar tendon rupture and are sufficient in most cases. On the lateral view, patella alta is identified on the basis of the presence of the patella superior to Blumensaat's line (Fig. 2). One or more bone fragments may be attached to the tendon if the injury is an avulsion.

Ultrasonography

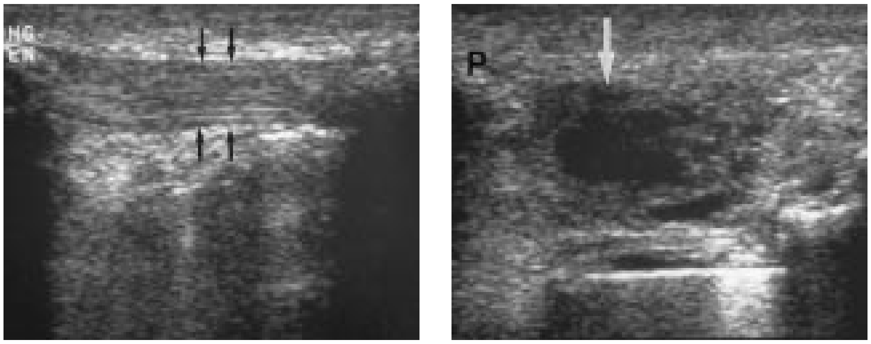
High-resolution ultrasonography has been recognized as an effective means of examining the patellar tendon in both acute and chronic injuries.[19] Sagittal images obtained with linear-array transducers at center frequencies of 7 to 10 mHz are recommended to allow full identification of the tendon and its bone insertions. Compared with the underlying fat pad, the tendon itself is usually hyperechoic (Fig. 3, A). With an acute rupture, a confluent area of hypoechogenicity is noted traversing the entire thickness of the tendon (Fig. 3, B). With chronic tears, thickening of the tendon at the rupture site is seen, along with disruption of the normal echo pattern.

Ultrasonography is advantageous in cases of chronic patellar tendinitis and chronic patellar tendon rupture because it is quick and relatively inexpensive and does not involve ionizing radiation. Unfortunately, this modality is both operator- and reader-dependent, which may make it less effective for some clinicians.

Magnetic Resonance Imaging

Magnetic resonance (MR) imaging has emerged as an effective, al-

Fig. 2 Lateral radiograph of the knee demonstrates a high-riding patella (patella alta), consistent with rupture of the patellar tendon. The entire patella is superior to Blumensaat's line (indicated by rule).



A B

Fig. 3 **A,** Sagittal ultrasound image of a normal patellar tendon (arrows). Note the shadowing effect of the patella and tibia. **B,** Acute patellar tendon rupture (arrow). Note the confluent region of hypoechogenicity extending completely through the tendon at the site of rupture. P = patella.

beit expensive, method of diagnosing chronic patellar tendinitis and acute and chronic patellar tendon ruptures.[20] The normal patellar tendon demonstrates a homogeneous low signal intensity throughout much of its course on proton-density images. The anterior and posterior margins are typically smooth and distinct (Fig. 4, A). With rupture, there is discontinuity of tendon fibers, waviness of the ends of the tendon, and an increase in signal intensity on sagittal T2-weighted images. Hemorrhage and edema may also be seen to extend posteriorly to the infrapatellar fat pad.

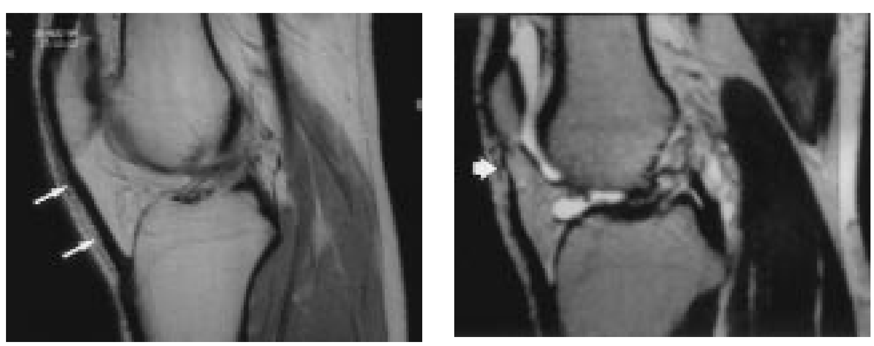
Magnetic resonance imaging is not recommended in the evaluation of most suspected patellar tendon ruptures. However, it may be helpful in patients with a neglected tear (Fig. 4, B) when the diagnosis is in question or when associated intra-articular injury is a possibility.

Classification

No widely accepted classification system for patellar tendon ruptures currently exists. Various authors have categorized these injuries on the basis of the location, configuration, and chronicity of the rupture. Hsu et al[21] grouped their series of 35 ruptures into three categories based on the location of the disruption: distal pole of the patella, tendon midsubstance, or tibial tubercle. Kelly et al[11] categorized their series of 11 ruptures by the anatomic configuration of the resulting tears: transverse, Z type (medial patellar avulsion with a lateral tubercle avulsion), and inverted-U type (medial and lateral portions ruptured from the tibial tubercle and the midportion avulsed from the patella). In neither of these series was any correlation made between the type of rupture and the method of repair or clinical outcome.

Giblin et al[13] classified bilateral patellar tendon ruptures into two groups: (1) midsubstance ruptures and (2) tendon avulsions from the proximal or distal end. Most cases of midsubstance rupture were noted to occur in patients with a chronic disease state. Proximal or distal tendon avulsions were usually seen in otherwise healthy patients with no signs of systemic or local disease. Again, no distinction was made between the type of tear and the clinical result.

Siwek and Rao[1] grouped their series of 36 repaired patellar tendon ruptures into two categories based on the interval between injury and repair: immediate repair and delayed repair (more than 2 weeks after injury). With this simple classification, the authors found a distinct advantage to immediate repair. Primary tendon repair was usually feasible in



A B

Fig. 4 **A,** Sagittal proton-density MR image of a normal patellar tendon (arrows). **B,** Sagittal T2-weighted MR image of a subacute partial proximal patellar tendon rupture (arrow). Note the attenuated proximal tendon and lack of hematoma, which is indicative of a subacute injury.

patients who were operated on immediately, whereas preoperative patellar traction and fascia lata autografts were commonly required for patients with a neglected rupture. Patients who underwent a delayed repair exhibited greater loss of flexion and higher incidences of persistent quadriceps atrophy and strength loss. The classification of Siwek and Rao is most helpful in assisting the clinician to determine the optimal method of treatment and to predict the final outcome after repair.

Treatment

Immediate Repair

Surgical restoration of the extensor mechanism is required for optimal return of function after a complete patellar tendon rupture. Nonoperative treatment is ineffective. This applies to the athlete as well as the nonathlete, regardless of the age of the patient. Surgical repair should be performed as soon after the injury as possible, which necessitates the timely diagnosis of the condition.

A number of methods for immediate surgical repair have been described in the orthopaedic literature. Simple end-to-end repair, with or without a reinforcing cerclage suture of wire or nonabsorbable suture material or tape, has been the most commonly used method.[11,13,21-24] Previously described techniques[1] employing a Bunnell pull-out wire connected to a Steinmann pin through the patella or the tibia (or both) have been abandoned because of the risk of pin-tract infection and the necessity of a second procedure for hardware removal.

Technique

Under tourniquet control, a longitudinal midline incision is made from the midpatella to the tibial tubercle. Thick skin flaps are created to expose the ruptured tendon and adjacent retinacula, which are usually involved in the tear. The tendon ends are debrided of frayed, nonviable tissue and hematoma. If the injury involves an avulsion off the distal pole of the patella or tibial tubercle, the tendon is cleared of loose bone fragments too small for internal fixation.

It is essential to identify both the medial and the lateral extent of the retinacular tear. Two No. 5 nonabsorbable sutures are woven in Bunnell fashion and passed proximally through one of three transpatellar holes with use of either a Keith needle or a Beath pin. A transverse 3.2-mm drill hole is made approximately 1 cm posterior to the tibial tubercle. A heavy nonabsorbable suture or piece of umbilical tape is passed through this hole, brought proximally, and passed transversely through the quadriceps tendon close to the superior border of the patella. A less bulky material, such as No. 5 nonabsorbable suture, can also be used to prevent a foreign-body reaction.

Tension is applied to the sutures, which are then clamped but not tied. A lateral radiograph of the knee is obtained to assess the patellar height compared with that on preoperative radiographs of the contralateral knee. Suture tension is either increased or reduced to reestablish the normal length of the patellar tendon. Once this has been accomplished, the sutures are tied over the superior pole of the patella, and the cerclage suture is tied adjacent to the patellar tendon (Fig. 5).

The knee is flexed to 90 degrees to test the repair. The patellar tendon suture line is oversewn with interrupted No. 2 absorbable sutures. The retinacular tear is similarly closed. When the ligament has been avulsed from the tibia, the Bunnell sutures are passed through a second transverse drill hole through the tibia. The wound is closed in layers, and the knee is placed in a well-padded dressing. A hinged brace is applied with the knee locked in 20 degrees of flexion.

Postoperative Rehabilitation

Isometric quadriceps- and hamstring-strengthening exercises are begun on the first day after surgery. Active flexion and passive extension of the knee is initiated 2 weeks after surgery, starting at 0 to 45 degrees and advancing 30 degrees per week. Active knee extension is permitted at 3 weeks postoperatively. Toe-touch weight bearing is initiated immediately after surgery and is advanced to full weight bearing without crutches

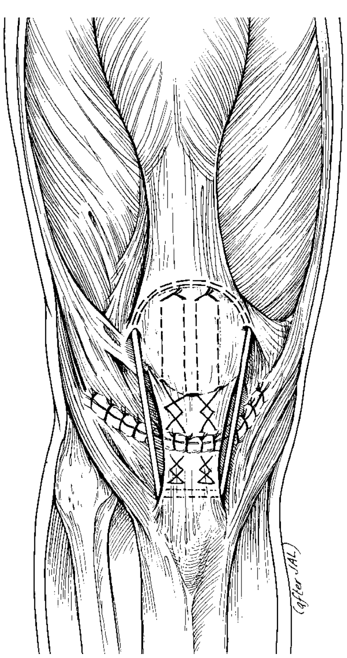


Fig. 5 Bunnell-type repair of an acute rupture with transosseous sutures through the patella. A reinforcing cerclage suture has been placed between the tibial tubercle and the quadriceps tendon.

by 6 weeks postoperatively. The brace and crutches are discarded when the patient is able to ambulate with good quadriceps control. A supervised isokinetic strengthening program is then initiated, and the patient is advanced to a sport-specific functional rehabilitation program. Resumption of strenuous athletic activity is prohibited for 4 to 6 months postoperatively, until the patient demonstrates full range of motion of the knee and 85% to 90% of the strength of the contralateral extremity on isokinetic strength testing.

Delayed Repair

Simple reapproximation of the torn tendon ends is often difficult when repair has been delayed more than 6 weeks.[1,18,25] The longer the delay between injury and repair, the greater the likelihood of quadriceps contraction and proximal patellar migration. Fibrous adhesions may form between the patella and the underlying femur, and degenerative changes may ensue in the nonarticulating patella and the trochlear groove. Distally, the ruptured tendon ends shrivel and become encased in scar tissue, and calcific deposits develop after long-standing injury.

Patients with a neglected rupture of several months' duration who present with superior patellar migration, loss of passive patellar mobility, and lack of full passive knee motion may require preoperative patellar traction.[1] This can be accomplished over the course of several days to weeks with the use of a 5-lb weight pulled distally through a Steinmann pin placed transversely through the midportion of the patella. Passive range-of-motion exercise of the knee is performed during the course of traction. Sequential lateral radiographs of the knee are taken until the patella is seen to be anatomically reduced compared with the contralateral knee.

A number of reconstructive options have been espoused for repair of the neglected rupture. Primary repair combined with autogenous graft augmentation using the fascia lata[1] or hamstring tendons[25] has been most commonly used and should be attempted if sufficient tendon is left for repair (Fig. 6, A). Inert materials, such as carbon fiber[26] and nonabsorbable tape suture material,[1] have also been advocated for the chronic tear. Extensor mechanism allografts consisting of an Achilles tendon or an intact patellar tendon unit have been used, albeit rarely, in salvage situations (Fig. 6, B).[27] Allografts are stronger, allowing earlier, more vigorous rehabilitation; however, they are associated with the risk of disease transmission. An external fixation device consisting of two transverse Steinmann pins (one through the patella and one through the tibia) connected by a Charnley compression clamp may be useful for reducing tension across the repair in the case of a long-standing rupture (Fig. 6, C).[18]

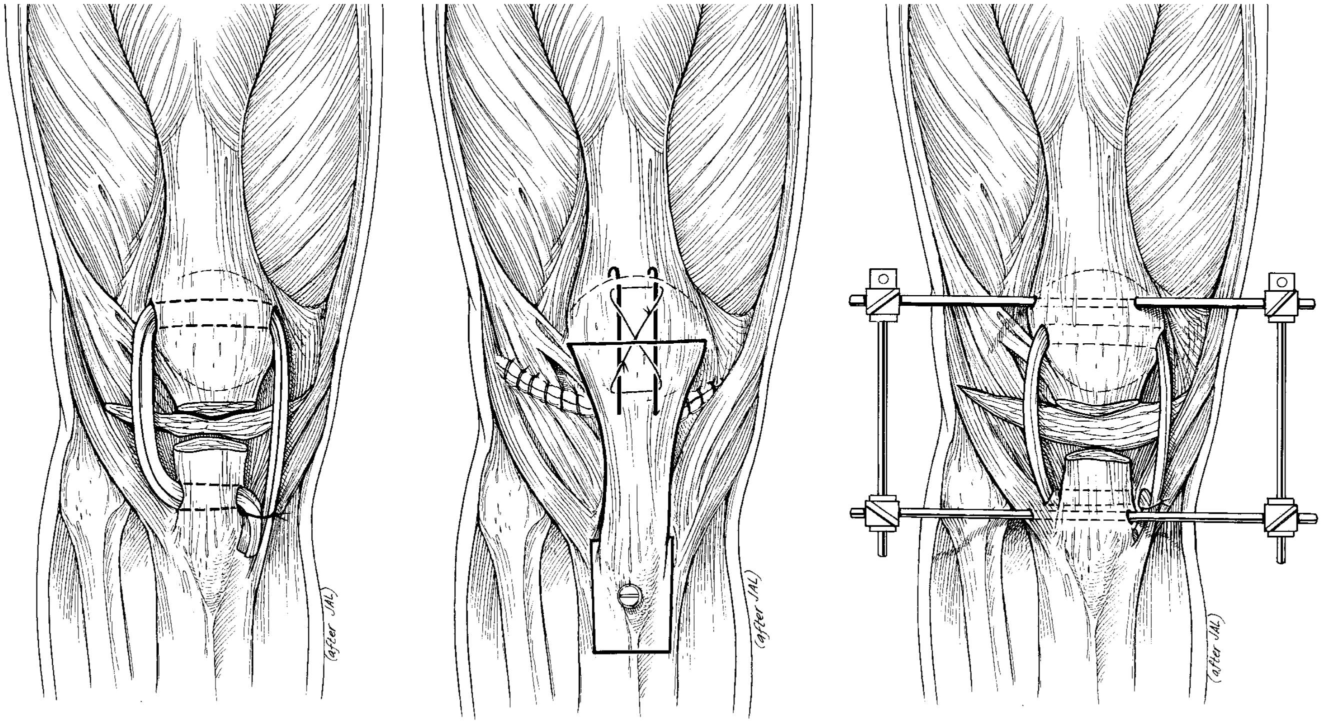
In general, the rehabilitation after a neglected rupture is considerably more conservative than that after repair of a fresh rupture. A bivalve cylinder cast is recommended for 6 weeks, during which time gentle passive-motion exercises may be considered, depending on the stability of the repair. This is followed by active range-of-motion exercises emphasizing full extension. Closed manipulation of the knee is occasionally required after casting to obtain greater knee flexion.

Results

The vast majority of patients who undergo primary repair in a timely fashion achieve nearly full return of knee motion and quadriceps strength. Persistent quadriceps atrophy commonly occurs, but tends not to affect the return of strength.[1,21] There does not appear to be any relationship between the configuration of the rupture, the method of repair, and the clinical outcome. The only factor that appears to correlate with clinical outcome is the timing of repair. In the series of Siwek and Rao,[1] 20 (80%) of the 25 patients who underwent immediate repair (within 7 days of injury) had excellent results, and 4 (16%) had good results. In contrast, of the 6 patients who underwent delayed repair (more than 2 weeks after injury), only 2 had excellent results, 3 had good results, and 1 had an unsatisfactory outcome. Patients who had undergone delayed repair also exhibited a greater degree of persistent quadriceps atrophy.

Hsu et al[21] treated 35 traumatic patellar tendon ruptures acutely with primary repair and a neutralization wire. Despite immediate repair, only 20 (57%) results were rated as excellent and 10 (29%) as good; 5 (14%) were rated unsatisfactory. The authors believed that their results were compromised by the fact that 34% of their patients had multiple injuries (27 of 35 ruptures were caused by motorcycle accidents).

Larsen and Lund[22] noted excellent or good results in 7 of 10 acute patellar tendon repairs. Radiographic comparison with the contralateral knee demonstrated incongruity on the Merchant and lateral radiographs in 7 cases. Patients with residual patellofemoral symptoms all had incongruity, but two thirds of the patients with incongruity were asymptomatic. Therefore, despite the accepted recommendations of accurate reestablishment of the normal patellofemoral articulation, articular incongruity alone may not be the cause of per-



A **B** **C**

Fig. 6 **A,** Neglected rupture reconstructed with a semitendinosus-gracilis autograft woven through the patella and tibial tubercle. **B,** Use of a patellar tendon allograft, with attached patellar and tibial bone blocks, for end-to-end repair of a chronic rupture with inadequate local tissue. **C,** Use of an external fixator made of two Steinmann pins and a Charnley clamp connecting the patella and tibial tubercle. This may be added to prevent proximal patellar migration while protecting the reconstruction of a neglected rupture.

sistent anterior knee pain in patients who undergo repair.

There are no large series evaluating the results of reconstruction of a neglected rupture. Isolated case reports describing the use of various reconstructive grafts have appeared in the literature.[18,25,27] Again, it appears that the most reliable indicator of prognosis is the interval between the injury and reconstruction. Patients who require preoperative traction and the use of autogenous or allograft tendon substitutes may have a compromised result compared with patients with sufficient tissue for an end-to-end repair.[1]

Complications

Decreased quadriceps strength and loss of full knee flexion are the most common reasons for a suboptimal result after a patellar tendon repair. These two complications are associated more with the initial injury than with the surgery itself. To overcome this, an aggressive postoperative rehabilitation program emphasizing early range-of-motion and quadriceps-strengthening exercises is recommended. Closed manipulation under anesthesia may be considered if at least 120 degrees of flexion is not obtained by 6 to 8 weeks postoperatively. Arthroscopic debridement should be reserved for patients who are capable of less than 15 degrees of passive extension despite a focused rehabilitation program. Quadriceps atrophy is noted in many patients after acute repair and in most patients after delayed repair. Nevertheless, atrophy does not appear to compromise the final return of strength as measured by subjective and objective evaluation.[1,21]

Complications of surgery are relatively infrequent. A persistent hemarthrosis necessitating either aspiration or formal drainage occasionally develops. This risk may be reduced by the use of a closed-suction drain postoperatively. Wound infection or skin dehiscence may occur; when it does, it is usually over the distal aspect of the wound, where skin coverage is thin. This complication can be reduced by placing the initial skin incision lateral to the tibial tubercle, where the skin is thicker and has a better vascular supply from the underlying anterior compartment musculature. There is also less tension at the suture line after skin repair.

Rerupture is occasionally seen in patients who attempt to return to running or jumping activities before complete healing of the repaired tendon.[1] Revision is usually successful in reestablishing knee motion and strength, as long as the repair is performed in a timely fashion.

Wire breakage may occur (often into multiple fragments) in repairs that have been reinforced with a cerclage wire. This often necessitates removal because of skin irritation and wire extrusion. For this reason, nonabsorbable suture, rather than wire, is recommended as the reinforcing suture material.

Patella baja may occur, with resultant motion loss and the risk of patellofemoral degeneration if excessive tension on the sutures causes inaccurate coaptation of the tendon ends. Intraoperative lateral radiographs obtained before final suture tensioning will assist in determining the optimal length of the repaired tendon.

Summary

Rupture of the patellar tendon can be a disabling injury in the active person. It is most commonly caused by a violent contraction of the quadriceps muscle against the fixed load of the patient's body weight with the knee in a flexed position. Rupture is thought to represent the end stage of a degenerative tendinopathy resulting from repetitive microtrauma to the fibers of the patellar tendon. In patients with systemic diseases that weaken collagen structure, rupture often occurs during nonathletic activities of daily living and may be bilateral.

The hallmark of a patellar tendon rupture on physical examination is the patient's inability to actively extend the knee against gravity. This finding, along with a painful, palpable defect in the substance of the tendon and demonstration of patella alta on a lateral radiograph, makes the diagnosis of this condition relatively straightforward.

Immediate surgical repair of the ruptured patellar tendon is recommended for optimal return of function. A Bunnell-type repair with the use of heavy nonabsorbable sutures through transosseous tunnels with a reinforcing cerclage suture is recommended for a secure repair. Patients who present with a neglected rupture occasionally require preoperative patellar traction to overcome the contracted quadriceps muscle so that reapproximation of the tendon ends is feasible. In rare instances, autogenous grafts or allograft tendons are required to span the defect when local tissue is unavailable. An aggressive postoperative rehabilitation program emphasizing early knee motion, quadriceps strengthening, and sport-specific functional rehabilitation will increase the likelihood of an excellent outcome with full return to an active lifestyle.

Outcome after repair has been found to be most closely related to the length of time between injury and repair. Patients who undergo an immediate repair usually achieve a full return of knee motion and quadriceps strength. Patients who undergo a delayed repair have been noted to have limitation of knee flexion, quadriceps weakness, and persistent quadriceps atrophy. Nevertheless, reasonable function can still be obtained in most cases.

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