Atraumatic bilateral patellar tendon rupture in a patient receiving steroid therapy

Steroid tedavisi görmekte olan bir olguda travmaya bağlı olmayan iki tarafı patellar tendon kopması

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Bilateral concurrent patellar tendon rupture is a rarely seen disorder with higher morbidity involving extensor mechanism of knee. Atraumatic injury usually develops as a result of risk factors as rheumatological diseases,[1,2] metabolic abnormalities,[3] hormonal disorders,[4,5] and local steroid injection therapy.[6] However traumatic rupture of patellar tendon develops in association with overloading imposed on extensor mechanism of the knee, and it is more frequently seen in young athletes.

In this paper a patient with systemic lupus erythematosus (SLE) whom we performed surgery for atraumatic bilateral concurrent patellar tendon rupture is presented. Reconstructive repair of patellar tendon using semitendinosus and gracilis tendons is remarked with special emphasis on diagnostic challenges inherent to bilateral atraumatic patellar tendon rupture.

Case presentation

A 31 year-old woman who had been receiving prednisone (20 mg/d) regularly for 7 years referred to our clinics with complaints of pain in both knees and inability to walk. The patient fell down on a ground while walking with a sharp and sudden pain and couldn’t stand up anymore. In a healthcare center where he consulted two times, synovial effusion involving both knees was detected. The patient couldn’t extend his knees anymore. Though magnet-
Magnetic resonance imaging (MRI) revealed obvious signs of patellar tendon rupture, she was given non-steroidal anti-inflammatory drug therapy with diagnosis of knee sprain.

On physical examination effusion in both knees, tenderness, hypermobile patellas and pain on palpation without any marked skin lesions were detected. The patient could not extend her knees actively, despite presence of active quadriceps muscle contractions. Passive knee movements were restricted because of pain. A soft tissue defect was palpated on the inferior edge of the patella (Figure 1). While knee radiograms did not demonstrate any signs of malalignment, bilateral patella alta was observed (Figure 2). Any sign of bone avulsion was not seen on the inferior edge of patella or tibial tubercle. Magnetic resonance imaging revealed patellar tendon ruptures, degenerative changes and patella alta bilaterally (Figure 3). Eleven days after the injury patellar tendon reconstruction with semitendinous and gracilis tendon transfer was performed, and the repair was reinforced with eight wire tension bands.

**Surgical technique**

A midline incision was made starting from the midpoint of patellar bone proximally up to 2 cm beneath tibial tubercle distally. During operation

![Figure 1. Palpable soft tissue defect on the inferior edge of patella and proximally slid patella in the patient with atraumatic bilateral patellar tendon rupture.](image1)

![Figure 2. Bilateral patella alta detected on (a) anteroposterior and (b) lateral radiograms.](image2)

![Figure 3. Sagittal T1 weighted magnetic resonance imaging of both knees preoperatively. Advanced degeneration, thinning and occasional disruptions in continuity (arrows) of both tendons especially marked on left patellar tendon were detected; SAG, Right; SOL, Left.](image3)
both patellar tendon were observedly thinned and ruptured in the middle portion.

Any cartilageous, meniscal, cruciate ligament lesion on patella and other articular facets of the knee were not observed. After scarce local debridement torn fragments were revitalized (Figure 4a). As allografts, semitendinosus and gracilis tendons were used. Two parallel transverse holes from distal tip of the patella and also a hole on tibial tubercle were drilled. To maintain normal length of patella the knee was held at 60° flexion, and patella was anchored to the tibia with a figure of “8” circular wire sutures. Then semitendinosus tendon was transferred from lateral to medial through a hole drilled on tibial tubercle, and distal patellar hole. Gracilis tendon was passed from medial to lateral through the proximal hole. Ends of semitendinosus and gracilis tendons were sutured to the other tendon, and reconstruction was achieved (Figure 4b). Ends of patellar tendon reinforced with reconstruction were sutured (modified Kessler technique). Finally medial and lateral retinaculum were repaired with no. 2 monofilament sutures, and exposed layers were closed.

The knee was immobilised for 6 weeks with cylindrical cast extending from groin to ankle. During this period isometric quadriceps exercises five times a day each lasting for 15 minutes were applied, and the patient was allowed to walk with weight-bearing on crutches. Six weeks after the operation the cast was removed. Hinged braces whose knee flexion angles could be adjusted upward starting from 0°-45° were applied. At the end of three months, range of motion (ROM) of the knee joint reached 0°–100°. The patient could walk without the aid of crutches. Bilateral patella alta were not detected in radiograms (Insall-Salvati ratio 0.9) (Figure 5),
and three months later anchoring wires were removed under local anesthesia, and intensive physiotherapy was instituted. Six months later both knees had achieved full active ROM, and the patient could walk without crutches, and braces (Figure 6). Extensor deficits were absent in both knees, and the patient could fulfill all activities she achieved before the traumatic event.

**Discussion**

Patellar tendon rupture is the least seen disruptive cause of extensor mechanism of the knee. Zernicke et al. reported that forces 17.5 times the body weight are required for the rupture of the patellar tendon. Giblin et al. stated that bilateral patellar tendon rupture develops secondary to two pathogenetic mechanisms. Firstly, patellar injury is due to overloading imposed on extensor mechanism during sportive activities of patients younger than 40 years of age. Recurrent microtraumas in young athletes result in tendon degeneration and microtears. This degeneration leads to patellar tendon rupture at its last stage (jumper’s knee). These types of ruptures usually occur at osteotendineous junction. Secondly, patellar tendon ruptures occur spontaneously without any evidence of trauma due to predisposing systemic conditions. The most frequently reported causes are SLE, rheumatoid arthritis, chronic renal failure, diabetes mellitus, hyperparathyroidism, systemic and/or local steroid administration, and surgical intervention. Histological examinations performed on spontaneously ruptured tendons, have demonstrated structural deformities due to degenerative changes.

Chronic degenerative changes in connective tissue delay healing of the tendon and recurrent microtears predispose to tendon rupture. Middle portion of the tendon frequently ruptures bilaterally.

Our patient had been using steroids for seven years for her SLE, and bilateral concurrent tendon rupture developed spontaneously without any evidence of trauma. The presumed mechanism of patellar tendon rupture involves forced quadriceps contraction against the ground while the knee is partially extended. This contraction might impose intolerable impact on patellar tendon. In our patient this pathogenetic mechanism might contribute to the rupture formation.

Diagnosis of patellar tendon rupture might be sometimes delayed and challenging. Siwek and Rao examined 36 patellar tendon ruptures in 33 patients. They reported delay in making a definite diagnosis for more than two weeks in seven (19%), and erroneous diagnosis on the first examination in 10 (28%) patients. A case with delayed diagnosis, where loss of muscular strength was attributed to steroid usage was reported. Our patient was diagnosed on the 10th day of her referral.

The most striking feature of patellar tendon rupture is sharp and local pain. Edema follows trauma.
In bilateral ruptures comparative evaluations can not be done because of contralateral involvement. In differential diagnosis meniscal tears, quadriceps tendon rupture, patella fractures and anterior cruciate ligament rupture must be considered. Magnetic resonance imaging demonstrates patellar tendon rupture, while radiograms reveal only high insertion patella. Meticulous history taking and physical examination are essential for correct diagnosis. Besides, in suspected cases MRI can be helpful in avoiding erroneous diagnoses.

Early diagnosis is essential for early surgical intervention. It is difficult to lyse adhesions, repair the defect, and achieve better long-term functional outcomes with delayed surgical intervention. If surgery is delayed more than 6 weeks, poor functional outcomes will be obtained, and duration of rehabilitation will be prolonged because of m. quadriceps atrophy.[9,10] We conceive that early intervention have contributed to our improved outcomes.

For repair of patellar tendon rupture, primary repair, Müller and Krackow techniques, fixation with nonabsorbable synthetic Dacron tape or anchor wire sutures passed through transverse tunnels drilled in patella and tibial tubercle, semitendinosus tendon transfer, fixation of patellar tendon to patella with stitch hooks, gracilis and semitendinosus tendon transfer together with figure 8 tension band wire support (Ecker et al.) techniques have been defined.[2,8,14-20] In atraumatic spontaneous ruptures, tendon healing is poor due to predisposing systemic diseases. Therefore, end to end suture techniques can become inadequate due to weakened patellar tendon. In our patient semitendinosus and gracilis tendons have been used for the reconstruction of the degenerated tendon secondary to SLE.[10,17] To preserve the continuity of patellar tendon and to resist stress forces against extensor mechanism, the tendon was reinforced with anchor wires, and patella infera was not observed. Insall-Salvati ratio[14] was found to be 0.9. Therefore perfect functional results characterized with full return to pre-injury daily activities without any sequelae of m. quadriceps atrophy, recurrent patellar tendon ruptures, and any deficiency in extension have been obtained in our patient.

In patients with collagen metabolism abnormalities such as systemic lupus erythematosus, and a history of chronic steroid usage, risk of spontaneous patellar tendon rupture should not be overlooked. Early diagnosis and treatment play an important role in the success of the treatment. We think with reconstruction of patellar tendons with hamstring tendons, and fixation of patella to tibia with anchor wire sutures optimal results ensuring early functional recovery of patellar functions can be obtained.

References
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