Case Report

Bilateral Patellar Tendon Rupture in an Adolescent Patient with the Presence of Osgood-Schlatter Disease: A Case Report and Review of the Literature

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ABSTRACT

Background: Bilateral patellar tendon rupture is an extremely rare injury in children and adolescents. Predisposing factors include repetitive microtrauma, systemic metabolic or rheumatic diseases, and corticosteroid or fluoroquinolone usage. The coexistence of Osgood-Schlatter disease and patellar tendon rupture has been described very rarely in the literature.

Case Report: We presented a 14-year-old male patient who had been previously followed-up with the diagnosis of Osgood-Schlatter disease and operated for bilateral patellar tendon rupture by using primary repair to the anatomical locations with U nails and suture anchors. At the end of the fifth-year follow-up, the patient had no symptoms and achieved full range of motion in both knees without terminal extension lag.

Conclusion: Bilateral patellar tendon rupture is considered a severe injury and necessitates early diagnosis and surgical intervention. Structural alterations resulting from inflammation and tendon degeneration due to existing Osgood-Schlatter disease can be a causative factor for bilateral patellar tendon rupture in adolescents.

INTRODUCTION

Patellar tendon rupture is a rare injury that necessitates early diagnosis and surgical intervention in children and adolescents [1,2]. It represents 7% of all acute traumatic injuries of the knee extensor mechanism in the pediatric population [2]. However, there are limited information's in the literature about the frequency, treatment modality, complications, and outcomes of this injury in childhood. Patellar tendon rupture often results from strong eccentric contraction of the quadriceps muscle while the knee joint is partially flexed [2]. In the immature skeleton, bony avulsion fractures such as tibial tubercle or patella sleeve fracture predominate rather than tendinous rupture [2,3]. Patellar tendon rupture without bony avulsion is extremely rare in the pediatric population, with only a few cases reported in the literature [2-5].

In the absence of trauma, spontaneous patellar tendon ruptures are associated with several predisposing factors, including systemic metabolic or rheumatic diseases [6,7] and prior corticosteroid or fluoroquinolone usage [8,9]. Additionally, repetitive microtrauma and existing tendinopathy might contribute to the development of degenerative changes and subsequent patellar tendon ruptures in the pediatric population [2,10]. Osgood-Schlatter Disease (OSD) is a type of osteochondrosis

described as a traction apophysitis resulting from the repeated contraction of the quadriceps femoris muscle transferred by the patellar tendon on the tibial tuberosity [11]. The association between OSD and the development of patellar tendon rupture in the absence of any systemic disease or corticosteroid/fluoroquinolone use is a rare condition, however has been described in the literature [2,12-14]. Among these studies, only one study [2] included pediatric patients. In this report, we present a 14-year-old male patient who had been previously followed-up with the diagnosis of OSD and operated for bilateral spontaneous patellar tendon rupture without bony avulsion. This study aims to emphasize the importance of OSD as a possible reason for bilateral patellar tendon rupture and the results of treatment.

CASE REPORT

A 14-year-old male patient was referred to our outpatient clinic with complaints of pain in front of both knees that increased with activity. He described a sudden loss of power in his legs after a jump with a height not exceeding half a meter from the ground without any direct trauma or falling. Physical examination revealed swelling and excessive tenderness with palpation on both tibial tuberosities. Distal poles of both patellae were palpated well above the joint line, and the patellar tendon could not be felt clearly with palpation. The passive range of motion of both knees was within the normal range, whereas the patient could not performactive knee extension. He stated that he had been playing basketball regularly for the last year with occasionally anterior knee pain. Because of that, he had been followed up with the diagnosis of OSD in our clinic for about six months. The patient was treated with non-steroidal anti-inflammatory drug, ice application and activity modification. He did not have any complaints with all these treatment modalities. Otherwise, he denied any significant orthopedic or rheumatologic family medical history and had no recent antibiotic, steroid, or other medication usages.

Plain lateral radiographs taken at 30° of flexion showed bilateral patella alta (Figure 1). The Insall-Salvati ratio on the right and left knees were 1.7 and 1.8, respectively. Magnetic Resonance Imaging (MRI) studies revealed discontinuity in the patellar tendons at the tibial attachment site, increased T2 signal density in both patellar tendons, and medullary edema in both tibial epiphyses (Figure 2). The ruptures were in the form of the tendinous avulsion, and there was no bone fragment distal to the tendons. The Imaging modalities did not reveal any associated bone lesions or intra-articular pathologies suggesting patellar dislocation, bone bruises, meniscal tear, chondral pathology, and cruciate or collateral ligament rupture. Blood tests for all the markers of rheumatic, metabolic, and renal diseases were performed and found within normal ranges.

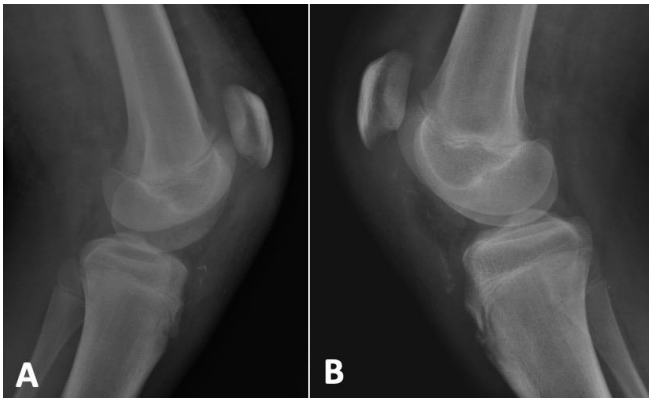

Figure 1: Plain lateral radiographs show high riding patella. The Insall-Salvati ratio on the right (A) and left (B) knees are1.7 and 1.8, respectively. Demineralized bone with segmental sclerotic deposition of the tibial tubercle on both knees is likely indicative of OSD.

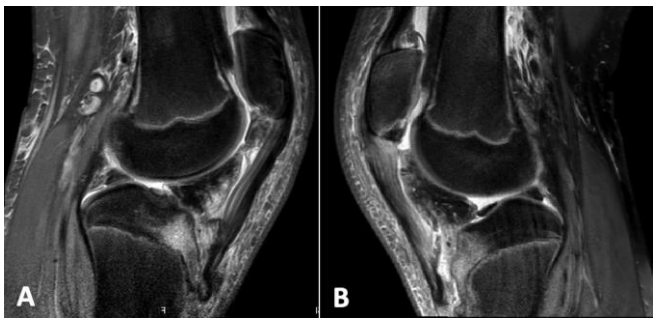

Figure 2: Sagittal T2-weighted MRI of the right (A) and left (B) knee show patella alta with disruption of the patellar tendon fibers in the form of delamination shear injury consistent with distal tendinous disruption from its bony attachment.

The surgery for both knees was proposed. The patient and his parents were informed about the surgical procedure and informed consent was obtained. The surgery was performed for both knees under general anesthesia on a supine position with tourniquet control. Straight anterior median skin incisions were preferred. Both avulsed ends of patellar tendons were explored (Figure 3A,3B). Both knees had full-thickness patellar

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tendon tears that were completely detached from the attachment points. After the release of all tissues in the anatomical plane, tendon ends were refreshed. Ruptured ends were repaired with suture anchors (TwinfixTi 5.0-mm/Ultrabraid 2-suture anchor; Smith & Nephew; Massachusetts; USA) by using Krackow tendinous repairing technique (Figure 3C) and reinforced by using U staples which put on through the distal tendon ends (Figure 3D).

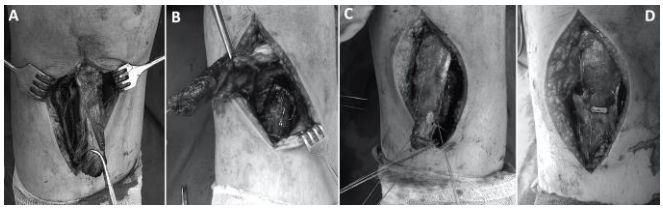

Figure 3: Total rupture of the patellar tendon on the right (A) and left (B) knees are repaired with suture anchors by using Krackow suture technique (C) and reinforced by using U staples (D).

Bilateral above knee splints were applied to the patient in full extension for three weeks postoperatively, and the wounds healed without any complications. Isometric quadriceps exercises were started on the second postoperative day. The patient was allowed partial weight-bearing with double support for six weeks. At the end of the 3rd week, the patient's knees were locked at 30° angle with an adjustable brace. Knee flexion exercises were started at 30° and increased gradually. At the end of the 6th week, both knees reached 90° of flexion, and physical therapy was initiated to regain the range of motion. At the end of the 4th month, full range of motion and adequate quadriceps power were observed for both knee joints.

In the 5th postoperative year, the patient had no symptoms and achieved full range of motion in both knees without terminal extension lag (Figure 4A,4B). The quadriceps muscle strength at the final follow-up visit was 5/5 according to the manual muscle testing grade. The patient stated that he could achieve daily activities and sports without any pain. The Insall-Salvati ratio on the right and left knees were 1.1 and 0.9, respectively (Figure 4C,4D). The clinical result was evaluated with the Tegner Lysholm activity scale [15] and calculated as 96 for both knees at the final follow-up.

Figure 4: Full range of motion without terminal extension lag on the right (A) and left (B) knee is observed at the end of the 5th year. The Insall-Salvati ratio on the right (C) and left (D) knees are 1.1 and 0.9, respectively.

DISCUSSION

Bilateral patellar tendon rupture is an extremely rare injury in children and adolescents. However, due to increased participation in sports activities during childhood, this injury's frequency has risen progressively [2]. In the immature skeleton, the muscles, ligaments, and tendons are generally stronger than the growth plates [16]. Thus, it is rare to observe the rupture of the tendon's substance in children and adolescents [16]. Patellar tendon rupture often results from strong eccentric contraction of the quadriceps muscle while the knee is partially flexed. With continuing knee flexion against a hardly contracting quadriceps muscle, disruption of the patellar tendon attachment may occur [2].

Bilateral ruptures can be classified into three categories according to the etiological factors [1]. The first category includes systemic diseases such as systemic lupus erythematosus [17], rheumatoid disease [6], diabetes mellitus [7], hyperparathyroidism [18], and chronic renal failure [1,7]. These systemic diseases cause chronic inflammation and amyloid deposition that affect tendon structure and predispose the tendons to rupture [1,16]. The second category includes corticosteroid use (both oral and injectable), which is believed to damage the tendon blood supply and alter the collagen synthesis [2,8]. The third category refers to histologic findings of inflammatory and degenerative changes in spontaneously ruptured tendons suspected of repetitive microtrauma. These histopathological changes result in the weakening of the tendon and development of degenerative tendinopathy, thus predisposing ruptures in jumping athletes [2,10]. These types of ruptures usually occur at the osteotendinous junction. In addition to these three main categories, several case reports have described fluoroquinolone-associated bilateral patellar tendon

rupture [9]. The pathological condition in our case is probably related to the third category. Repeated micro-stress was added to the tendon-bone junction, causing OSD disease and eventually avulsion of the tendon from the tibial tubercle. We did not observe any avulsion fracture by radiographic evaluation or macroscopic findings; however, it can be assumed that it was an avulsion fracture microscopically [2,10].

OSD is a type of osteochondrosis which is described as a traction apophysitis resulting from the repeated contraction of the quadriceps muscle and patellar tendon on the tibial tuberosity [11]. It is one of the most common causes of knee pain in adolescents and related to activities and performance specific to sports such as soccer, basketball, and volleyball [19]. Approximately 10% of all adolescents are affected by OSD, with a higher prevalence among those who are very active [19]. It is thought to be growth-related, occurring most commonly in boys between the ages of 12 and 15 years and in girls between the ages of 8 and 12 years19.

Yousef MAA and Rosenfeld S [2] evaluated the frequency of pediatric patellar tendon ruptures and describe the radiological findings, treatment modalities, and outcome of such injuries. They identified five male patients (7%; mean age 13,6 years) among 71 pediatric patients who sustained acute traumatic injury of the knee extensor mechanism. The injury occurred with sports activities in 4 patients. Osteogenesis imperfecta and OSD were identified in two patients. In another study reported by Moy A et al. [12], a healthy 33-year-old male with a prior history of bilateral patellar tendonitis and a diagnosis of OSD during adolescence experienced simultaneous bilateral patellar tendon rupture after playing volleyball. He underwent bilateral patellar tendon repair without complications. Another two studies [13,14] reported partial ruptures of distal patellar tendon in adult patients with a history of tendinosis and previously diagnosed OSD. They concluded that the diagnosis of OSD and the surgical intervention combined with chronic tendinopathy resulted in a predisposition to traumatic rupture.

Surprisingly, the diagnosis of bilateral patellar tendon rupture can sometimes be challenging. It can be misdiagnosed due to the lack of a normal comparative knee [20]. Careful physical examination and critical evaluation of imaging studies are essential for early and accurate diagnosis. Acute onset knee pain and swelling, loss of active knee extension, and a palpable infrapatellar gap should raise concerns about possible patellar tendon rupture. However, it is possible to retain some flexor ability if the medial and lateral retinacula remain intact. Additionally, a significant effusion of the knee joint may obstruct the palpable infrapatellar gap and obscure the patella alta [20]. Imaging studies such as plain radiographs, arthrography, ultrasound, and MRI are usually used to confirm the diagnosis. Plain radiograph has been identified as the most cost-effective method, and high riding patella is the hallmark diagnostic sign to identify patellar tendon rupture. MRI is performed to confirm the diagnosis and provide further information regarding the location of tendon disruption, which is of paramount importance for the surgical plan [2].

Early surgical repair is the treatment of choice for the management of patellar tendon rupture 3. A significantly better functional outcome score has been reported in patients diagnosed and treated in the first seven days after the injury [2]. If surgery is delayed more than six weeks, poor functional outcomes will be obtained, and the duration of rehabilitation will be prolonged because of the quadriceps atrophy [2]. Various surgical techniques have been described in the literature including suture bridge double row technique, transpatellar suturing, and transosseous suturing through the proximal tibia [2,3,12]. The repair site can be reinforced with cerclage wire, staple, bioabsorbable screw, or biocomposite suture anchor [2]. We preferred the Krackow technique to repair the patellar tendon because this technique is strong enough to compensate for the stress that may occur in major tendons [21]. Adequate healing of the avulsed tendons was achieved with strong fixation and appropriate postoperative rehabilitation. Patellar tendon rupture is considered a severe injury and necessitates early diagnosis and surgical intervention. A high index of suspicion is required for the diagnosis to avoid delays in the treatment which would lead to possible suboptimal functional outcomes [20]. In the present case, spontaneous bilateral patellar tendon rupture developed without any additional underlying pathology, other than OSD. Structural alterations resulting from inflammation and tendon degeneration due to existing OSD can be a causative factor for bilateral patellar tendon rupture in adolescents.

ETHICAL APPROVAL

Informed patient consent obtained for the case included.

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