Spontaneous disruption of the bilateral knee extensor mechanism: a report of two cases

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INTRODUCTION

Spontaneous disruption of the knee extensor mechanism is associated with systemic disease such as chronic renal disease, hyperparathyroidism, rheumatoid arthritis, systemic lupus erythematosus, and connective tissue diseases.1-7 Repeated microtrauma to the tendon and local or systemic steroid administration are risk factors for tendon rupture.1,2,8 The mechanism of patellar and quadriceps tendon injury is usually a sudden contraction of the quadriceps tendon with the knee in flexion during a fall; symptoms include knee pain, effusion, and inability to extend the knee.9

Patellar or quadriceps tendon rupture disrupts the extensor mechanism of the knee. Early diagnosis and treatment is imperative for optimal outcome.10 This study reports 2 cases of spontaneous disruption of the bilateral knee extensor mechanism.

CASE REPORTS

Patient 1
In October 2013, a 29-year-old woman presented...
with sudden onset of pain and swelling of both knees, and increasing difficulty in walking after a feeling of her knees ‘giving way’ while walking. The patient had no previous trauma. She had a history of undifferentiated connective tissue disease and pyoderma gangrenosum 4 years earlier, and was being followed up by a rheumatologist and a dermatologist. She was on prednisolone (60 mg/day) and azathioprine (100 mg/day) for a chronic ulcer on the left leg. Clinical examination revealed bilateral knee bruising and effusion, with limited range of motion owing to severe pain. Radiographs showed patella alta in both knees, but no evidence of fracture (Fig. 1a). Magnetic resonance imaging confirmed the diagnosis of bilateral patellar tendon rupture: a mid-substance tear in the right knee and a tear near to the insertion at the tibial tubercle in the left knee (Fig. 1b).

Emergency bilateral patellar tendon repair was planned. Intra-operatively, the tendons were found to be attenuated and not suitable for primary repair (Fig. 1c). In view of her history of connective tissue disease and long-term steroid usage, reconstruction with allograft and defunctioning wire was decided. An autograft was not used, as it may be affected by the underlying disease. A transtibial bony tunnel was made and the allograft was passed through

Figure 1 Patient 1: (a) radiographs showing patella alta of both knees; (b) magnetic resonance imaging showing bilateral patellar tendon rupture (arrows): a mid-substance rupture in the right knee and a rupture near the insertion at the tibial tubercle in the left knee; (c) the tendons are attenuated and not suitable for primary repair; (d) reconstruction with an allograft and defunctioning wire; and (e) radiographs showing correction of patella alta.
the tunnel. Two parallel trans-osseous tunnels were made and the allograft ends passed through the tunnels in a figure-of-8 configuration. The allograft was then tensed through the knee range of motion, and the isometric point was achieved at about 30° of knee flexion. The tendon ends were then tied down at the superior patella pole, and the whole construct was protected by a defunctioning wire (Fig. 1d).

Postoperatively, the patient remained non-weightbearing for 6 weeks, with a knee brace locked in full extension for 2 weeks. Passive range of motion of the knee was allowed after 2 weeks. Partial weightbearing with crutches was allowed after 6 weeks. This strict regimen aimed to regulate quadriceps contraction to prevent compromising the reconstruction.

At 6 months, the patient was able to bear full weight without pain. Her bilateral knee range of motion was 0° to 60°. At 7 months, the defunctioning wire was removed from both knees. At one-year follow-up, the patient achieved range of motion of 0°–100° with no extension lag in both knees.

**Patient 2**

In January 2014, a 27-year-old man presented with pain and swelling of the right knee after a fall with the knee in flexion. Clinical examination revealed moderate effusion and limited range of motion of the right knee owing to pain. Radiographs revealed a high-riding patella but no fracture (Fig. 2a). The diagnosis of knee contusion and possible patellar tendon rupture was made. A long backslab was used to immobilise the knee. Four days later, the patient fell again, this time on his left knee. Clinical examination revealed a left knee effusion and limited range of motion with tenderness at the suprapatellar region. Radiographs revealed no fracture. The patient had end-stage renal failure necessitating haemodialysis for the past 12 years. He was not on any long-term...
steroids. Magnetic resonance imaging showed a complete acute rupture of the right infrapatellar tendon and the left quadriceps tendon (Fig. 2b).

Emergency primary repair of the tendons was planned. Intra-operatively, the diagnosis of complete tendon rupture was confirmed (Fig. 2c). The remnant tendons showed adequate integrity, and primary repair was decided. For the right knee, a double whip stitch was applied to the patellar tendon and passed through 2 parallel trans-osseous drill holes in the patella and secured at the superior patellar pole. A defunctioning wire was then added to protect the construct (Fig. 2d). For the left knee, a Krakow suture was applied to the quadriceps tendon and passed through 2 parallel trans-osseous drill holes in the patella and was secured (Fig. 2d). No defunctioning wire was used.

Postoperatively, the patient remained non-weightbearing for 6 weeks. The right knee was locked in full extension with a brace, and the left knee was allowed 30º of knee flexion. After 6 weeks, partial weightbearing and passive range of motion exercises were allowed. At 3 months, the patient was able to bear full weight without pain. The range of motion was 0º to 45º in the right knee and 0º to 90º in the left knee. At 6 months, the defunctioning wire was removed from the right knee. At one-year follow-up, the patients achieved range of motion of 0º–90º in the right knee and 0º–100º in the left knee, with no extension lag.

**DISCUSSION**

Disruption of the knee extensor mechanism should be treated promptly to achieve optimal functional outcome. Delayed treatment for more than 2 weeks may result in poorer functional outcome, as primary repair may not be feasible owing to tendon scarring and retraction. Extensive soft tissue releases and the use of an allograft or autograft may be necessary. Delayed treatment may lead to tendon atrophy and a longer rehabilitation period.

Disruption of the knee extensor mechanism is difficult to diagnose. A high index of suspicion is needed for patients with a history of steroid usage and systemic disease (end-stage renal failure and connective tissue disease). The extensor mechanism of the knee is difficult to assess in the acute setting when the patient has severe pain. In some cases, the patient can still extend the knee owing to incomplete patellar tendon rupture or intact medial and lateral knee retinaculum (despite a complete patellar tendon rupture).

A normal healthy patellar tendon is able to withstand a large load. A force 17.5 times of the body weight is required to rupture the tendon. If the tendon has been affected by a degenerative or inflammatory process, its strength is significantly reduced and the risk of rupture increases. The mechanism of injury is usually a sudden contraction of the quadriceps tendon with the knee in flexion. The unopposed pull of the quadriceps tendon after patellar tendon rupture leads to patella alta. This can be seen on a lateral radiograph of the knee in 30º flexion. Patella alta is determined by the Insall-Salvati ratio, which is the ratio of the longest patellar diagonal length to tendon length with the knee in 30º of flexion. A value of ≤0.8 is indicative of patella alta, and the diagnosis of patellar tendon rupture should be considered. Magnetic resonance imaging should be used to confirm the diagnosis when in doubt.

Patellar tendon ruptures are classified according to the site of the tear. Type 1 refers to a tear originating at the inferior patellar pole; a type 2 tear is at the mid-substance of the tendon; a type 3 tear is at the tibial tubercle insertion. The causes of tendon rupture include oral and injectable steroids that affect collagen synthesis and compromise blood supply, systemic diseases (systemic lupus erythematosus and rheumatoid arthritis) that alter tendon structure secondary to inflammatory changes, and repetitive microtrauma of the tendon with degenerative and inflammatory changes.

Spontaneous quadriceps tendon rupture in patients with renal failure requiring haemodialysis has been reported. Renal osteodystrophy secondary to elevated parathyroid hormone levels leads to generalised osteoporosis with subperiosteal bone resorption and secondary weakness of the bone-tendon junction. In addition, vitamin D deficiency leads to osteomalacia and impaired collagen metabolism and thus weakening of the tendon structure. Amyloidosis can lead to abnormal production and retention of β2-microglobulin, which tends to accumulate in the tendon and reduce its elasticity and predispose it to rupture following minor trauma. Most patients report a sudden pain in the suprapatellar region and instability while trying to prevent a fall with the knee extended against resistance. This places immense stress on the quadriceps and patellar tendons. The final fall is usually a sequela of the tendon rupture.

**DISCLOSURE**

No conflicts of interest were declared by the authors.
REFERENCES