Spontaneous and Simultaneous Quadriceps and Patella Tendon Rupture in a Patient on Chronic Hemodialysis

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Bilateral spontaneous ruptures of the extensor mechanism of the knee are uncommon, especially if they occur simultaneously.\(^1,2\) Bilateral quadriceps tendon rupture has been described as a complication of numerous disease states, such as systemic lupus erythematosus,\(^1\) chronic renal failure,\(^3,3\) gout,\(^1\) secondary hyperparathyroidism,\(^4,5\) and rheumatoid arthritis.\(^1\) The first case of spontaneous bilateral quadriceps rupture was described by Steiner and Palmer,\(^6\) in 1949; since then only 66 additional cases have been reported, including 16 cases in secondary hyperparathyroidism.\(^1\)

Bilateral rupture of the patella tendon also was described as a rare condition; 48 cases were reported in the literature and of those, 16 in the absence of trauma.\(^1\) Only one case of spontaneous quadriceps tendon rupture of one knee with spontaneous simultaneous patella tendon rupture of the contralateral knee in a patient on chronic hemodialysis has been reported in the literature.\(^4\)

This article presents a case of spontaneous quadriceps tendon rupture with simultaneous spontaneous patella tendon rupture in a 46-year-old man on chronic hemodialysis with documented secondary hyperparathyroidism.

CASE REPORT

A 47-year-old man presented with an inability to support his weight after a sudden fall while walking. On initial evaluation both knees were swollen and tender with markedly reduced range of motion.

His past medical history was significant for chronic anemia and hypertension. He also was on maintenance hemodialysis for the past 6 years, twice a week for end-stage renal disease secondary to polycystic kidney disease.

The patient was taking the following medications at the time of admission: Nifedipine 20 mg twice a day, Atenolol 100 mg once a day.

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CACO3 1250 mg 2 tbl 3 times a day. Laboratory data included a white blood cell count 9400/ mm³, hemoglobin 8.5 g/dL, hematocrit 28.2%, platelet count 330,000/mm³, sodium 137 mEq/L, potassium 5.1 mEq/L, chloride 102 mEq/L, calcium 2.2 mg/dL, phosphorous 4.8 mg/dL, blood urea nitrogen 20.5 mg/dL, creatinine 814 μmol/L (normal: 53–97 μmol/L), alkaline phosphatase 270 IU/L (normal: 42–98 IU/L), and serum intact parathyroid hormone 1317 pg/mL (normal: 9–55 pg/mL).

The patient initially was treated in the emergency room. Aspiration of both knees revealed hemorrhrosis. Ice packs were applied to both knees. Three days after admission the patient was referred to the orthopedic department.

Physical examination revealed swelling and tenderness of both knees. The patient could not extend his knees actively. A palpable defect of the quadriceps tendon was noted on the right side in the suprapatellar region and the patella was displaced inferiorly; on the left side the patella was displaced superiorly (Figure 1).

Figure 2A shows patella baja in the right knee and Figure 2B shows patella alta in the left knee, consistent with quadriceps tendon rupture of the right knee and patella tendon rupture of the left knee. Ten days after injury, repair of the ruptured tendons was performed.

An anterior median longitudinal approach was used for the quadriceps tendon reconstruction. Avulsion of the quadriceps tendon from the patella was found. Operative repair was performed using Ethibond pull-out sutures (Ethibond 5 Excel; Johnson & Johnson, Somerville, NJ) starting as a Krakow suture in the substance of the tendon, then through drill holes in the patella, reinforced with a triangular flap from the quadriceps extension.

The same approach was used for the patellar tendon reconstruction. Avulsion of the tendon from bone at the inferior pole of the patella was revealed. Operative repair was performed using Ethibond pull-out sutures (Ethibond 5 Excel) through drill holes in the patella. The repair was protected with a temporary wire loop from the patella to the tibial tuberosity.

Tendon biopsy specimens obtained from the ruptured ends of the tendons were examined histologically. There was evidence of granulation tissue, bleeding, slight chronic inflammation, and reactive fibrosis with entrapped conventional bone fragments in the avulsed tendon. No amyloid deposits in the ruptured tendons were identified by staining with Congo-red.

Cast immobilization was maintained for 30 days, after which continuous passive motion and active range of motion exercises were started. Partial weight bearing was allowed 5 weeks after surgery, and ambulation without support 7 weeks after surgery.

Four months after surgery the patient had 120° of flexion of both knees without an extension lag and could walk without support.

**DISCUSSION**

Spontaneous ruptures of major tendons (quadriceps, patella, Achilles, and triceps) have been described as a complication of several disease states. The first case of bilateral simultaneous rupture of the quadriceps tendons was reported by Steiner and Palmer in 1949. The first case of simultaneous avulsion of the quadriceps tendons in a patient with secondary hyperparathyroidism on chronic hemodialysis was reported by Preston and Adicoff in 1962. Since then, 66 cases of bilateral quadriceps tendon rupture have been reported. Of the 66 cases, only 16 were patients with chronic renal failure. The pathogenesis of spontaneous tendon rupture remains controversial.

Although a number of possible pathogenetic mechanisms have been proposed, none has been proven. In some chronic disease states such as systemic lupus erythematosus and rheumatoid arthritis, spontaneous tendon rupture has been attributed to the side effects of the glucocorticoids. Also fluorocinolone-induced tendinopathy with spontaneous rupture has been reported.

In patients with end-stage renal disease, three etiological theories for spontaneous tendon ruptures have been proposed: degeneration due to chronic acidosis leading to elastin deposition in the tendons, predisposing them to rupture; β-2 microglobulin amyloidosis; and recently weakness of the bone-tendon junction due to increased osteoclastic cortical bone resorption in secondary hyperparathyroidism.

There seems to be increasing evidence...
that secondary hyperparathyroidism, osteitis fibrosa, and increased bone resorption may result in repeated minor avulsion fractures ultimately leading to weakening of the tendon attachment site and finally its complete rupture.8,9,13-15

In one report of three patients with known secondary hyperparathyroidism, a gradual increase in the serum alkaline phosphatase was documented prior to the tendon rupture.6 An elevated serum alkaline phosphatase level correlates well with parathyroid hormone level and also with subendinal bone erosion in patients with end-stage renal disease and the occurrence of spontaneous tendon ruptures.15

De Franco et al16 reported two patients with end-stage renal disease, secondary hyperparathyroidism and spontaneous quadriceps rupture; the highest serum parathyroid hormone and alkaline phosphatase levels were found in the month preceding tendon rupture.

Shiota et al13 reported seven spontaneous ruptures of major tendons in five patients on chronic hemodialysis. These patients had radiologic signs of hyperparathyroidism, increased alkaline phosphatase and parathyroid hormone serum levels. None of the patients had amyloid deposits in the ruptured tendons.

In our patient, there was no history of corticoid or fluroquinolone medication. His serum parathyroid hormone 1317 pg/mL is considered significant as levels higher than 500 pg/mL are known to be associated with histologic evidence of severe osteitis fibrosa due to secondary hyperparathyroidism.8 Staining for amyloid of tendon biopsy specimens was negative, but there were fragments of bone providing clear evidence of an avulsion of the tendon attachment to the bone.

Although in our case the delay in accurate diagnosis was only three days, reportedly up to 50% of spontaneous bilateral quadriceps ruptures are initially misdiagnosed.10 A high degree of suspicion is needed, although the clinical features usually are readily evident in patients initially presenting; diffuse swelling around the knee, inability to lift the straight leg, and a visible or palpable suprapatellar defect.6

REFERENCES