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Authors

Kalantar-Zadeh, K
Singh, K
Kleiner, M
et al.

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Case Report

Nontraumatic bilateral rupture of patellar tendons in a diabetic dialysis patient with secondary hyperparathyroidism

K. Kalantar-Zadeh, K. Singh, M. Kleiner, M. P. Jarrett and F. C. Luft

Staten Island University Hospital, Department of Internal Medicine, Staten Island, New York, USA, and Franz Volhard Clinic, Humboldt University of Berlin, Germany

Key words: patellar tendon; bilateral tendon rupture; chronic renal failure; secondary hyperparathyroidism; haemarthrosis

Introduction

Acute, spontaneous, simultaneous, bilateral rupture of the patellar tendons in patients with chronic renal failure is unusual [1,2]. We report a diabetic patient with end-stage renal disease who developed acute bilateral patellar tendon rupture without evidence of previous trauma or major musculoskeletal disease. Based on previous cases of spontaneous tendon rupture, we believe that secondary hyperparathyroidism is the primary causative factor in her case.

Case report

A 43-year-old black woman came to our emergency room with acute onset of pain, massive effusions, and ecchymoses of both knee joints. While going upstairs several hours earlier, she experienced a sudden, moderately severe pain and a popping sound in her right knee, followed by a similar pain and sound in her left knee a few seconds later. She was then no longer able to bear weight in the standing position, observed the development of swelling and discoloration, and sought medical attention. Diabetes mellitus type II and arterial hypertension had been diagnosed 10 years earlier. Progressive diabetic nephropathy necessitated chronic haemodialysis treatments 2 years earlier. Her last haemodialysis session was performed successfully for 4 h 1 day prior to admission. The patient had anaemia which responded poorly to erythropoietin and iron supplementation, as well as worsening secondary hyperparathyroidism. She had been hospitalized several times earlier for haemodialysis vascular access repair. No previous history of fractures or joint prob-

lems were reported. Her current medications included an angiotensin converting enzyme inhibitor, a long-acting calcium channel blocker, a centrally acting imidazoline derivative, and famotidine. Ferrous sulfate and subcutaneous recombinant human erythropoietin were given for her anaemia. Other medications included calcium carbonate 2 g thrice daily. She had been given calcium citrate until 1 week prior to admission, but had received no vitamin D preparations recently because of elevated phosphate values.

On physical examination her blood pressure was 140/85 mmHg, the pulse was 86/min, the respiratory rate was 16/min, and her temperature was 37°C. The eyes showed changes consistent with laser coagulation treatments. She had a systolic ejection murmur, but no clinical evidence of cardiac enlargement or failure. Her left upper arm dialysis fistula functioned normally. Both knee joints were moderately swollen, ecchymotic and very tender with severely restricted movement capabilities. She was unable to extend her knees even after adequate analgesic administration.

Her serum electrolytes were: sodium 144 mmol/l, potassium 5.3 mmol/l, chloride 106 mmol/l, and bicarbonate 20 mmol/l, respectively. The creatinine was 908 µmol/l, and glucose was 5.5 mmol/l. Other pertinent values were: calcium 2.5 mmol/l, phosphorus 2.45 mmol/l, magnesium 2.3 mmol/l, albumin 34 g/l, total protein 70 g/l, uric acid 506 µmol/l and alkaline phosphatase 3.93 µkat/l, respectively. Other liver function tests were normal. The complete blood cell count showed: haemoglobin 4.5 mmol/l, hematocrit 0.23, mean corpuscular volume 84.2 fl. The white blood cell count was $8.1 \times 10^9/l$ and the platelet count was $264 \times 10^9/l$. The prothrombin time was 13.0 s (control 11.6 s) and the activated partial thromboplastin time 29 s (control 28 s). Increasing serum concentrations of intact parathyroid hormone (PTH), 1410 ng/l and 1686 ng/l (normal, 10–55 ng/l), with serum phosphate concentrations of 3.2 mmol/l and 2.9 mmol/l had been reported 6 weeks and 2 weeks prior to admission. The serum beta-2-microglobulin was within the normal range.

Roentgenogram of the knees revealed complete dis-

Correspondence and offprint requests to: Friedrich C. Luft, MD, Franz Volhard Clinic, Wiltberg Strasse 50, 13122 Berlin, Germany.



Fig. 1. Lateral roentgenogram of the right knee joint shows significant proximal dislocation of patella or high riding patella (patella alta). Significant sclerotic changes of the major vessels secondary to long-standing diabetes mellitus is apparent.

location of both patellae medially and upwards, but no signs of fracture. Mild diffuse osteopenia and mild degenerative changes of the bony structures were also noted (Figure 1). Magnetic resonance imaging (MRI) of the right knee revealed complete rupture of the proximal mid aspect of the patellar tendon. There was increased signal representing haemarthrosis and oedema within the severed ends of the patella tendon. The patella was proximally retracted and the distal quadriceps tendon was up-buckled and up-lifted. There was increased laxity and a redundant contour to the tibial end of the patellar tendon. There was no evidence for a popliteal cyst and the menisci and ligaments were intact (Figure 2). The left knee was not examined with MRI because of increasing pain and inability of the patient to be able to cooperate. Bilateral arthrocentesis was performed after the imaging studies were completed; 20–30 ml bloody fluid were removed from each joint.

At operation, a complete and symmetrical patellar tendon rupture at the inferior poles of both patellar bones was found bilaterally. There were no small bone fractures nor any other sign of avulsion fracture. Both patellar tendons appeared normal, other than being



Fig. 2. Magnetic resonance imaging of the right knee joint showing disruption of patella tendon with up buckling of the distal end of the quadriceps muscle.

ruptured at their osseo-tendinous junctions. A primary repair was performed using heavy absorbable sutures. Post-operatively, long-leg plaster cylinders were applied bilaterally. The patient performed active knee motion exercises and ambulated with a walker. She resumed a mobile lifestyle 2 months later. Pathological examination of the tendinous tissue revealed evidence of acute rupture. Histologically, the collagen fibers showed no evidence of degeneration, no calcifications were present, and the small vessels showed no evidence of sclerosis.

Discussion

Our patient's simultaneous, spontaneous, bilaterally symmetrical, patellar tendon ruptures were unusual in that she had no antecedent trauma other than climbing stairs and that the ruptures involved solely the tendinous tissue at the inferior aspects of both patellae. To our knowledge, two previous cases of bilateral, symmetrical, and simultaneous patellar tendon ruptures in patients with chronic renal failure have been reported; however, both involved significant trauma and none occurred in a diabetic patient [2].

Recently, Jones *et al.* [2] reviewed all published cases of tendon ruptures in uraemic patients in the last two decades and concluded that tendon ruptures in such patients occur at a younger age than in nonuraemic patients. Affected patients were more anaemic than is generally seen in dialysis patients, which was also the case in our patient. Jones *et al.* [2] also stressed the association of tendon ruptures with progressive secondary hyperparathyroidism, rapidly increasing levels of serum PTH, inorganic phosphorus, alkaline phosphatase, and a higher concentration of serum

calcium, compared to that of the general dialysis population. This situation was also present in our patient. Other possible predisposing conditions include metabolic acidosis with concomitant catabolism, dialysis-associated amyloidosis, fluoroquinolone administration, abnormalities in collagen metabolism, lax tendons, and corticosteroid treatment.

With few exceptions, spontaneous tendon rupture in chronic renal failure occurs mostly in weight bearing tendons. The quadriceps, Achilles tendon, and patellar tendon in that order, are most frequently affected. In the upper extremity, the triceps brachii is most commonly involved. Bilateral ruptures may occur sequentially or simultaneously; however, the latter is very rare indeed [2]. Patellar tendon ruptures may occur in otherwise healthy individuals (so-called idiopathic ruptures [3]). Patellar tendon rupture in monozygotic twins has been reported suggesting the influence of genetic variance [4]. Most cases of bilateral tendon rupture occur during a sudden quadriceps contraction when the patient stumbles, jumps, runs, or ascends stairs [5]. Patients usually notice a popping sound from the joint area and acute pain. They are then unable to extend the affected leg and a palpable gap and ecchymoses develop above the tendon. In elderly patients, bilateral tendon ruptures occur in the tendinous region of the patellar ligament, where the blood supply is meagre. In younger patients, tears occur in the quadriceps muscle. In general, the patellar tendon is the least common site and tears most frequently in

muscular young athletes during competition or intensive training [3].

MRI is a valuable tool in diagnosing acute ruptures of patellar tendon [5]. Although complete tendon rupture is a clinical diagnosis, MRI can provide additional information such as location of the tear, the condition of the tendon, and the appearance of the surrounding soft tissues. These findings not only aid in pre-operative planning, but also reduce overall operative time.

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