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Chronic alcohol consumption as a predisposing factor for multiple tendon ruptures in unusual sites in a patient with rheumatoid arthritis

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Abstract

Simultaneous bilateral patellar tendon ruptures are a rare complication of rheumatoid arthritis (RA). Systemic inflammatory diseases (RA, systemic lupus erythematosus (SLE), chronic renal failure, primary and secondary hyperparathyroidism, diabetes mellitus, obesity, sports activity, older age (>50) and drugs (prolonged use of high doses of steroids, local steroid injections and quinolones) are considered as potent predisposing factors for tendon rupture. We report a case of an alcoholic patient with RA and bilateral spontaneous tendon ruptures of the knees. Circumstantial evidence suggest that in this patient, chronic alcohol consumption, a very frequent cause of toxicity to striated and cardiac muscle, contributed to the injury.

Introduction

Rheumatoid arthritis (RA) is a chronic autoimmune disease that leads to substantial disability and reduced quality of life. In more than 50% of patients, tendons of the wrists are affected (1) and tendon ruptures may occur, possibly by overproduction of matrix metalloproteinases (MMPs 1, 2, 3, 8, 13) (2).

Although there are several case reports with bilateral simultaneous, non-traumatic, tendon ruptures of the knees, no large single-center case series exists due to the rarity of this injury. Recently Kellersmann *et al*. analyzed all reports and predisposing factors of bilateral simultaneous patellar tendon ruptures reported during the last 43 years (3). In 62% of 50 cases, no known predisposing factors existed. This observation suggests that in such cases investigation for additional predisposing factors is required.

Case

A 52-year-old man was referred to our clinic with a 6-year history of rheumatoid arthritis with lung involvement. He had inadequate response to methotrexate and cyclosporine that had been discontinued 3 years ago. He occasionally received 2 mg of methylprednizolone daily for small periods during the last 6 months.

The patient experienced spontaneous bilateral tendon ruptures of his knees 4 months ago, while walking peacefully on a level surface and he underwent plastic reconstruction of patellar tendons with no sequela.

Regarding his medical history, he reported consumption of more than 60 g of alcohol daily for the last 20 years and a serious car accident 12 years ago. Fifteen years ago he experienced a spontaneous rupture of the long head of the biceps brachii tendon of the right arm, but he did not seek medical attention. He was divorced and worked as a barman for the last 20 years. He had no history of quinolone intake.

Clinical examination revealed active RA (DAS28 = 7.35) and atrophy of the quadriceps muscles. Rales were present at the base of both lungs. The patient had nodulosis in the exterior surface of both elbows, while conventional radiographs of hands and feet showed soft tissue swelling and periarticular osteopenia of metacarpophalangeal and proximal interphalangeal joints of both hands, with no erosions. A muscular mass because of the rupture of the long head of the biceps brachii tendon was obvious in the middle of his right arm.

Laboratory tests revealed macrocytosis due to alcohol abuse with no anemia, elevated lactate dehydrogenase (359U/l), hyperlipidemia (cholesterol = 253mg/dl, LDL-C = 179mg/dl), elevated C- reactive protein (4.44 mg/dl) and high titer of rheumatoid factor (3260 IU/ml). Antinuclear antibodies and anti-neutophile cytoplasmic antibodies were negative. Thyroid tests and levels of parathormone were normal.

A high-resolution computed tomography (HRCT) of the chest revealed interstitial lung disease with honeycombing and no evidence of active alveolitis.

The histological examination of the surgical specimens from tendon and quadriceps, muscle revealed signs of chronic inflammatory arthritis and degenerative disorders of the striated muscle.

Magnetic resonance (MR) imaging

The MR imaging study for both knees included a sagittal T1-weighted, an axial T1-weighted and an axial short-tau inversion recovery (STIR) sequence. MR imaging revealed rupture of the

right quadriceps tendon (Figs. 1A and 1B) and rupture of the left patellar tendon (Figs. 2A and 2B). Additionally, there were hyperintense areas on STIR images within the quadriceps muscles bilaterally and within the right posterior calf muscles (Fig. 1B); these signs suggested muscle inflammation. No pathologic effusion, bone erosions or bone marrow edema were seen in both knees. The patient had no clinical evidence of tendon ruptures in other sites, thus MR imaging was not performed in other joints.

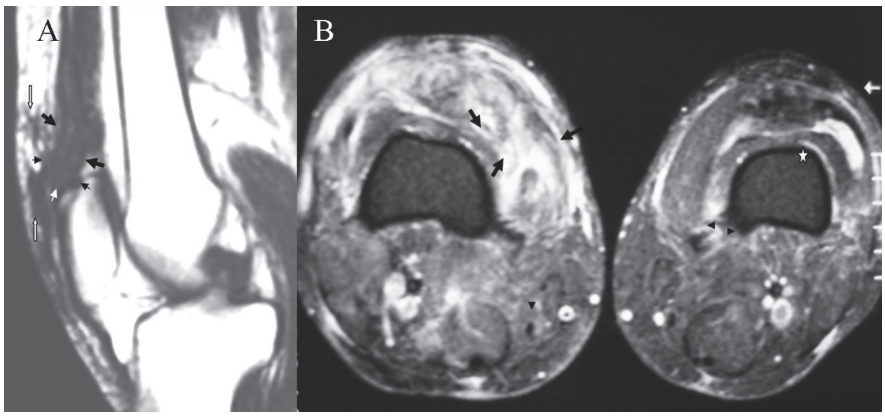

Fig. 1. A. A sagittal T1-weighted MR image of the right knee shows partial rupture of the right quadriceps tendon at its patellar insertion, seen as interruption of the trilamelated tendon (arrows) by hypointense fluid (small arrows). Hypointense areas and strations in the prepatellat fat represent edema and fluid (open arrows).
B. An axial STIR image shows a ruptured right quadriceps tendon as an ill-defined and inhomogenously hyperintense "mass like" lesion (black arrows). The rupture extends to the right vastus medialis muscle (open arrow). Diffuse hyperintense areas within the quadriceps muscles bilaterally and within the posterior muscles of the right thigh suggest underlying muscle inflammation (arrowheads). Note that the contralateral left quandriceps tendon is slightly hyperintense and enlarged implying mild tendinopathy (asterisk).

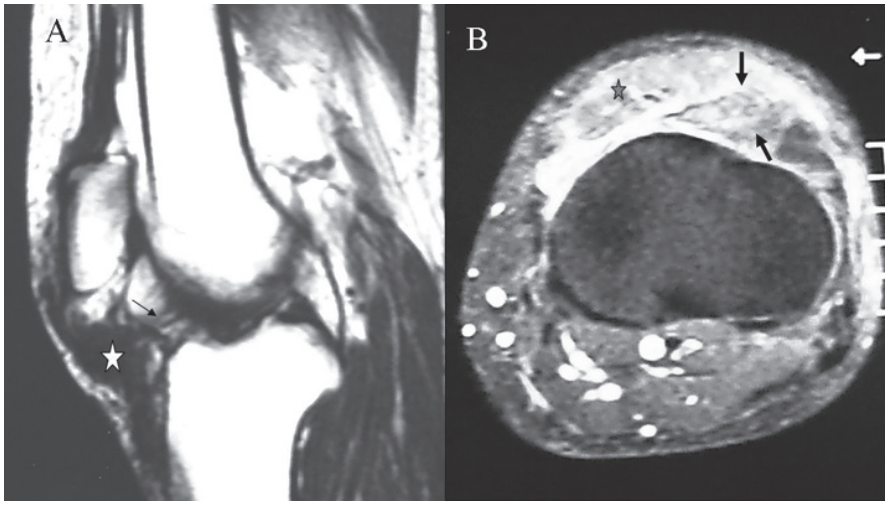

Fig. 2. A. A sagittal T1-weighted MR image of the left knee reveals amorphous thickening of the left patellar tendon in its mid portion (asterisk), implying tendon rupture, with striations in the adjacent Hoffa's fat pad (arrow).
B. An axial STIR image demonstrates an enlarged and hyperintense left patellar tendon the ruptured tendon. (black arrows). The adjacent prepatellar fat exhibits diffuse hyperintensity (asterisk).

Discussion

Simultaneous bilateral patellar tendon ruptures is a rare complication of RA. Systemic disorders (SLE, RA), chronic renal failure, primary and secondary hyperparathyroidism, diabetes mellitus, obesity, sports activity, older age (> 50) and drugs (prolonged use of high doses of steroids, local steroid injections and quinolones), have been implicated as potent predisposing factors for tendon rupture (3, 4). Tenosynovitis is a characteristic sign of RA involving the extensor tendons of the wrist in 50-64% of patients (1).

It is known that alcohol intoxication leads to skeletal muscle changes. Chronic alcohol myopathy, characterized by progressive proximal weakness and muscle atrophy (5) affects 45-70% of alcoholics (6). Because of the asymptomatic nature of the myopathy, diagnosis may be underestimated. Histochemical analysis of biopsy specimens reveals atrophy of type II with a relative sparing of the type I fibers (7).

Alcohol consumption impairs translation efficiency and protein synthesis in striated muscle and induces apoptosis (8). Insulin-like growth factor-1 (IGF-1), a crucial factor for protein synthesis and cell-proliferation and an essential factor for tendon and ligament healing (9), is decreased in alcoholic rats (10). Administration of tumor necrosis factor-α, interleukin (IL)-1 or IL-6 in rats, decreases levels of IGF-1 (11, 12 and 13) while low IGF-1 and high levels of insulin-like growth factor binding protein (IGFBP)-3 have been previously found in RA patients (14). This pathophysiology fits in with our case.

Although RA is a well-established predisposing factor for tendon rupture (3), this cannot be the only cause in our case. We suggest alcohol as an additional factor because our patient experienced rupture of the long head of the right brachii biceps 15 years ago, when no RA was present. Moreover, MR imaging a useful method for the assessment of inflammatory arthritides (15) reveals signs of myopathy as previously described in alcoholics (16), suggesting tissue injury due to alcohol.

This is the first report implicating different predisposing factors for tendon rupture in RA patient with multiple tendon ruptures. Our patient was an alcoholic, with severe RA. Probably, low IGF-1, due to alcohol abuse and RA, impaired both protein synthesis of striated muscle and inflamed tendons' healing. In RA, tendon ruptures usually occur in tendons with a sheath, such as the long head of the biceps tendon or the wrist and hand tendons. However, alcoholic myopathy in our

case enhanced the possibility of tendon rupture in weight-bearing joints, such as the knees. Corticosteroids cannot be implicated for the injury due to the low dose and the short term of intake.

In conclusion, a careful medical history for alcohol abuse should be taken down for every patient with no traumatic tendon rupture. Alcoholic RA patients represent a group with multiple comorbidities. A holistic approach and cooperation with other specialists is essential in order to face alcohol addiction and comorbitities related to alcohol consumption.

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