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Case Report

Spontaneous patellar tendon rupture in a case followed up for diagnosis of systemic lupus erythematosus

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Abstract

Spontaneous patellar tendon rupture is a rare condition that usually occurs secondary to conditions, such as rheumatoid arthritis, systemic lupus erythematosus (SLE), and use of steroids and fluoroquinolones. This paper presents a full-thickness patellar tendon rupture detected with magnetic resonance imaging, which was performed due to pain and swelling that started spontaneously on the front side of the left knee without a history of any trauma, of a 35-year-old male patient who had been followed up for a diagnosis of SLE for approximately 4 months and who had started taking methylprednisolone 4 mg/day 4 months prior, used it for 1 month, and then stopped using it. In patients who are followed up for a diagnosis of SLE, it should be kept in mind that there is a risk of developing a spontaneous tendon rupture secondary to chronic inflammation and use of corticosteroids.

Key words: Spontaneous patellar tendon rupture, systemic lupus erythematosus, corticosteroid, inflammation

Introduction

Patellar tendon rupture is a relatively rare condition in people apart from professional athletes (1). Spontaneous patellar tendon rupture is even more uncommon. It usually occurs secondary to conditions, such as rheumatoid arthritis, systemic lupus erythematosus (SLE), primary and secondary hyperparathyroidism, chronic renal failure, renal transplantation, gout, and use of steroids and fluoroquinolones (2).

Although SLE is a multi-systemic disease that can involve the skin, joints, kidneys, nervous system, serous membranes, blood cells, and other organs of the body, spontaneous tendon rupture in SLE is a rare condition. However, some publications have reported patellar and Achilles tendon ruptures in SLE patients (3-5).

This paper will present a case diagnosed with spontaneous patellar tendon rupture, which is a rare condition during the course of SLE.

Case Presentation

A 35-year-old male patient, who had been followed up by the rheumatology polyclinic for 4 months for a diagnosis of SLE, applied with the complaint of pain and swelling on the front side of the left knee, which had started spontaneously 2-3 days prior while he was walking. The patient recalled no history of trauma; the pain in his left knee increased with movement and decreased with rest, and he was having difficulty walking. The patient had been using hydroxychloroquine 400 mg/day for 4 months. Additionally, it was learned that he had started taking methylprednisolone 4 mg/day 4 months prior, used it for 1 month, but then discontinued its use, because his complaints subsided. The patient had no history of any other drug use or systemic disease. When asked about his familial medical history, he stated that his aunt and his brother had SLE.

The patient's physical examination did not reveal active arthritis but revealed arthralgia on the 2nd and 3rd metacarpophalangeal joints of his left hand and on the 3rd proximal interphalangeal joint of his right hand. The examination of his left knee revealed that he was unable to do active knee extension, there was sensitivity and a slight increase in temperature at the patellar tendon adhesion location, and his patellar shock test was positive. His other examination findings were normal.

His laboratory findings were as follows: creatine 0.8 mg/dL, ALT 26 u/L, leukocyte 5.52, hemoglobin 15.5 g/dL, and platelet 292.103/uL. C-reactive protein was 7 mg/L; erythrocyte sedimentation rate was 20 mm/hour; hepatitis markers were negative; and electrolytes, lipid panel, thyroid function test, and whole urine test were normal. Antinuclear antibody was at 1/320 titer, and anti-dsDNA was positive.

Magnetic resonance imaging (MRI) of the left knee of the patient showed that his patellar cartilage was normal, and there was minimal increase of liquid inside the knee joint, an increase in intra-tendon signal of

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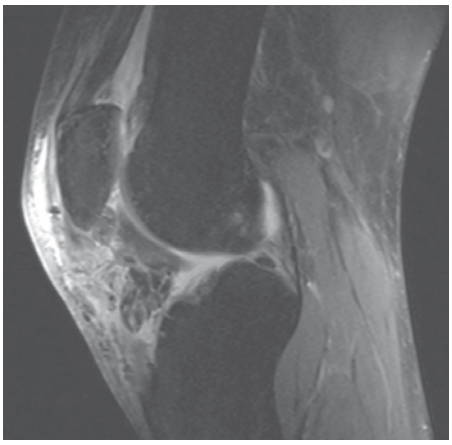
Figure 1. MRI of left knee shows increase in intra-tendon signal, peritendinous liquid, inflammation, and a full-thickness patellar tendon rupture



the patellar tendon, peritendinous liquid and inflammation, and a full-thickness patellar tendon rupture (Figure 1).

The patient, who had been followed up for a diagnosis of SLE and found to have a full-thickness patellar tendon rupture in his left knee, was evaluated by the orthopedics department, and his patellar tendon was fixed surgically.

Discussion

This case report presents a patient diagnosed with spontaneous patellar tendon rupture, which is a rare condition during the course of SLE.

The extensor mechanism of the knee consists of the quadriceps muscle, quadriceps tendon, medial and lateral retinacula, patella, patellar tendon, and tibial tubercle. This structure and especially the patellar tendon are quite resistant to tension and trauma. Normally, it takes a force greater than 15 times the body weight to cause a rupture in the quadriceps and patellar tendon. However, some systemic and metabolic diseases reduce the intrinsic strength of tendons and cause them to become weak against force. These include rheumatoid arthritis, SLE, chronic renal failure, renal implantation, gout, diabetes, hyperthyroidism, obesity, use of fluoroquinolones, use of steroids, and previous knee surgeries (2). In diseases, like SLE, that progress especially with systemic inflammation, the tendon structure is affected and can become prone to ruptures (1).

One of the causes of spontaneous tendon rupture in SLE is the use of corticosteroids, one of the main treatment agents. Corticosteroids can be administered as injections into the tendon or can be used systemically by oral administration. Although the role of corticosteroids in patellar tendon rupture is not entirely known, it is thought that in addition to the anti-inflammatory effect, they reduce collagen synthesis and blood flow and weaken the tendon (1). Corticosteroids affect collagen in 3 ways: by reducing the replication of fibroblasts, by reducing the synthesis of fibroblasts, and by increasing the collagenase enzymes (6). There is no clear information as to the dosage and duration of steroid treatment leading to spontaneous tendon rupture. Strejbek et al. (7) reported a bilateral patellar tendon rupture that occurred in a 63-year-old patient when getting off a bed, who had been followed up for a diagnosis of SLE and who had been using prednisone 5 mg/day. Rascher et al. (8) reported a bilateral patellar tendon rupture that occurred in a 45-year-old patient when jumping rope, who had been followed up for a diagnosis of SLE and who had been using prednisone 5 mg/day. Spontaneous Achilles tendon rupture was seen in a patient who was newly diagnosed with SLE following pulse methylprednisolone treatment (9). In a study that followed up on 180 SLE patients for 10 years, only 4 patients had a patellar tendon rupture. According to that study, tendon rupture was associated with duration of illness (7-20 years) and duration of chronic use of corticosteroids (7-15 years) (10). Despite these findings, as the number of patients is low, it is difficult to say that these are risk factors for patellar tendon rupture, and the existence of patellar tendon rupture due to the use of corticosteroids in SLE patients has not been definitely shown (1).

In addition to the use of corticosteroids for spontaneous tendon rupture in SLE, chronic inflammation and chronic degenerative changes also play a role. Even in cases of slight disease activity in SLE, subclinical inflammation still persists. Histological findings in the ruptured tendon show inflammatory changes that progress with perivascular mononuclear infiltration due to SLE. This degenerative and inflammatory process weakens the tendon and increases the risk of rupture (1, 6).

Our case was diagnosed with SLE 4 months ago, and he had a history of 1-month use of low-dose corticosteroid. However, the fact that the patient's family had a history of SLE suggested that the patient might have had SLE prior to the diagnosis, meaning that he might have had subclinical inflammation for quite some time. Although the dose of corticosteroid used by our patient was low, it has been shown in some cases that spontaneous tendon rupture may occur, even with the use of low-dose steroids (7-9). For this reason, we wanted to draw attention to this issue by presenting the patient, who we think developed a spontaneous patellar tendon rupture secondary to the use of corticosteroid, in addition to chronic inflammation and degeneration, and who had been followed up for a diagnosis of SLE. As patients who are followed up for a diagnosis of SLE have the risk of chronic inflammation and the risk of developing spontaneous tendon rupture due to the use of corticosteroid, the patient should be informed when he/she develops a spontaneous rupture to reduce the risk of rupture, and follow-up of the patient should be done with special attention.

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