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Patellar Tendon Rupture as a Manifestation of Lyme Disease

Nirav K. Pandya, MD, Miltiadis Zgonis, MD, Jaimo Ahn, MD, PhD, and Craig Israelite, MD

Patellar tendon ruptures can result from overloading eccentric contraction injuries (as occur in athletic patients), chronic tendon weakening from repetitive microtrauma,[1] and chronic inflammation leading to degeneration and subsequent tear.[2] Ruptures that result from acute, traumatic events generally occur at the attachment near the inferior pole of the patella,[3] whereas midsubstance disruptions are more common with inflammation from systemic conditions.[4-6]

Here we present what we believe is the first reported case of a patellar tendon rupture as an extra-articular manifestation of the late phase of Lyme disease around the knee. We informed our patient that data concerning her case would be submitted for publication, and she gave us written permission to publish this report in print and electronically.

"...midsubstance disruptions are more common with inflammation from systemic conditions."

Case Report

A 50-year-old woman with a known history of Lyme disease presented to our institution with 4 days of fevers, right knee swelling, and pain. Five years earlier, she had been diagnosed with Lyme disease after presenting to an emergency department with rapid-onset chills, generalized myalgias, lethargy, and an erythema migrans rash—but no joint symptoms. She had been treated with a full course (3 weeks) of oral doxycycline antibiotic therapy and had had no posttreatment sequelae.

Approximately 13 months before the current presentation, the patient had been involved in a motor vehicle accident in which she was a restrained driver. She suffered a right inferior pole patella fracture with disruption of the extensor mechanism. At our institution then, the patient underwent an uneventful inferior pole patellectomy with reattachment of the patellar tendon (Figures 1, 2). She was kept immobilized for 1 month and then over 2 months performed gentle range-of-motion (ROM) exercises followed by strengthening exercises. She did quite well and by time of discharge from our care had regained full strength and ROM.

Approximately 4 days before the current presentation, the patient experienced low-grade fevers, myalgias, and fatigue. Then she noticed right knee swelling and pain. She denied any specific traumatic event, joint pain, or weakness leading up to the swelling and pain, as well as insect bites, tick exposure, swelling in other joints, recent increase in activity, and development of cutaneous lesions.

Physical examination revealed a moderate-sized effusion on the right knee. The scar from the prior surgery was intact. There was a significant extensor lag, to approximately 40° to 45°, but from 45° to 90° the patient was able to actively extend the knee against mild resistance. She was unable to perform a straight-leg raise. She was otherwise neurovascularly intact.

There was concern about a partial or possibly complete rerupture of the patellar tendon on examination, and radiographs (Figures 3, 4) showed patella alta. Aspiration of the right knee elicited 20 mL of a greenish fluid. Synovial fluid white blood cell (WBC) count was 22,880/mm^3 (90%

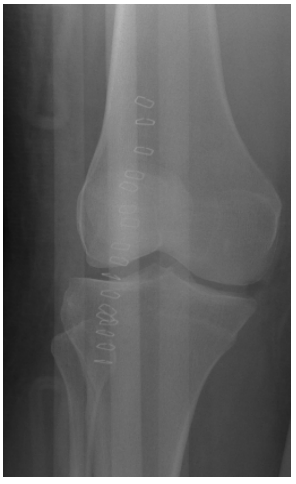

Figure 1. Anteroposterior radiograph of patient's right knee after operative fixation at time of initial injury (13 months before current presentation) shows inferior pole patellectomy.

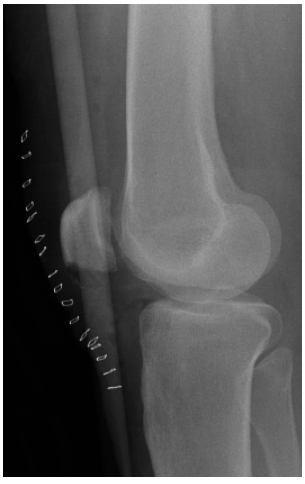

Figure 2. Lateral radiograph of patient's right knee after operative fixation at time of initial injury (13 months before current presentation) shows inferior pole patellectomy.

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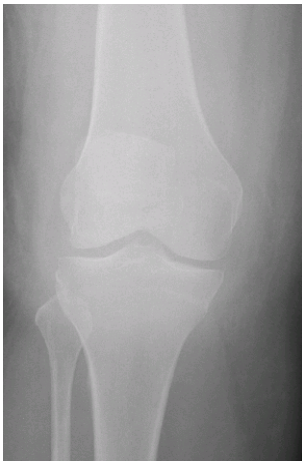

Figure 3. Anteroposterior radiograph of patient's right knee at time of current presentation shows no obvious fracture or dislocation.

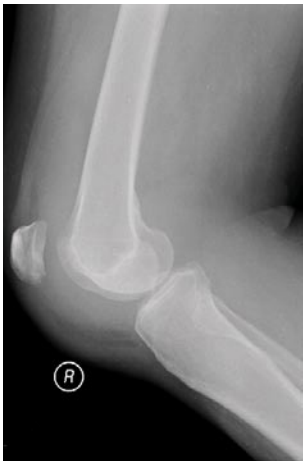

Figure 4. Lateral radiograph of patient's knee at time of current presentation shows knee effusion and patella alta.

segmented neutrophils, 8% monocytes, 2% lymphocytes), and red blood cell count was 2000/mm^3. Gram stain and fluid culture were negative. Given the history of Lyme disease, the synovial fluid was also sent for DNA polymerase chain reaction (PCR) testing for *Borrelia burgdorferi,* the causative agent of Lyme disease; the result was positive. In addition, serum blood tests were positive for *B burgdorferi* antibodies with elevated levels (normal, <10 mg/L) of immunoglobulins IgM (19 mg/L), IgG (>25.4 mg/L), and IgA (14 mg/L), results consistent with a late-phase infection. Also of note were elevated serum WBC count (11.9×10^9/L), C-reactive protein (CRP, 63.7 nmol/L), and erythrocyte sedimentation rate (ESR, 52 mm/h).

The patient was taken to the operating room. The patellar tendon was found intact at its insertion (area of prior repair), but a new midsubstance tear was found distal to the area of prior injury/repair. Tendon areas that appeared atrophic and scarred were débrided. The proximal and distal stumps of the tendon were repaired with modified Krackow stitches. After the repair, the patient was taken through 0°-to-90° ROM without undue tension on the repair. Pathology showed tendinous soft tissue with focal chronic inflammation.

Given the patient's diagnosis of recurrent Lyme disease, she was prescribed a 3-week course of oral doxycycline per the recommendation of our infectious disease team, which followed her postdischarge progress at its clinic. The patient finished the antibiotics and, 12 months out from surgery, was doing well, with full return of strength and ROM. Follow-up aspiration at the most recent clinic visit revealed straw-colored fluid with a WBC count of 333/mm^3 with negative gram stain, culture, and PCR testing of the synovial fluid for *B burgdorferi,* indicating eradication of joint infection.

Discussion

Patellar tendon ruptures are thought to result from overloading eccentric contraction injuries (as found in athletic patients), chronic tendon weakening from repetitive microtrauma,[1] and systemic inflammatory states with subsequent degeneration and failure.[4-6] In the last condition, failure may occur during minimal activity or even during activities of daily living.

The consensus is that, in healthy patients, traumatic tears of the patellar tendon result from extensor mechanism overloading. Tears preferentially occur at the attachment to the inferior pole of the patella. Biomechanical studies measuring stress and strain during progressive loading of the patellar tendon show that, before failure, tendon strain is much (3-4 times) increased at the patellar insertion site than within the midsubstance,[3] lending support to this hypothesis.

Midsubstance tears of the patellar tendon, conversely, are usually found in patients older than 40 who have a structurally degenerated tendon midsubstance, as in patients with chronic localized tendinopathy secondary to overuse microtrauma (often unilateral) or generalized systemic inflammatory conditions, such as systemic lupus erythematosus, rheumatoid arthritis (often bilateral), and chronic renal failure.[4-6]

Our patient had a nontraumatic midsubstance tear (away from the prior area of surgical repair), and therefore we can postulate that the inflammation caused by the Lyme infection led to tendon degeneration and subsequent rupture. Some would argue that this patient was already at increased risk for rerupture because of the history of prior repair and that Lyme disease (even with the patient's presenting with constitutional symptoms) was a secondary finding. Yet, the rate of rerupture after primary repair has been shown to be very low to zero in multiple series.[7-9]

"...in healthy patients, traumatic tears of the patellar tendon...preferentially occur at the attachment to the inferior pole of the patella."

Reruptures are thought to occur when patients return to running and jumping before repairs are healed.[10] This was not the case with our patient, who complied with rehabilitation, had a nontraumatic onset of pain, and presented 13 months after repair. In addition, pathology results for her ruptured tendon were consistent with focal chronic inflammation, not an acute process. Therefore, given the midsubstance location of the tear, lack of antecedent trauma, compliance with rehabilitation, and extremely low rate of rerupture after primary repair (in the literature), the rerupture likely resulted from the inflammation caused by the Lyme infection and not by a new acute traumatic event or previous surgery.

Some would still contend that this patient's prior surgical repair compromised the tendon in some way and that this presents a significant confounder. Yet, if the tendon were

weakened from the prior repair, it likely would have failed in the early months after surgery rather than nontraumatically 13 months after surgery (a surgery done proximal to the new tear). Furthermore, the patient did not have the clinical signs and symptoms of a rerupture until she experienced acute onset of constitutional symptoms (ie, fever), nontraumatic knee pain, and elevated inflammatory markers consistent with a Lyme disease relapse manifesting in the knee. Although the tendon theoretically may have been "weaker," it reruptured when Lyme disease manifested itself—suggesting that the disease can present itself in this extra-articular manner by causing a patellar tendon rupture (or rerupture in this case).

Lyme disease is caused by the spirochete *B burgdorferi*. Infection has 3 distinct phases: early, early disseminated, and late.[11] The early phase is characterized by the common erythema migrans rash or constitutional symptoms. The early disseminated phase (weeks to months after initial presentation) is characterized by multiple erythema migrans lesions, migratory musculoskeletal pain, and cardiac or neurologic findings. The late phase is characterized by musculoskeletal complaints, including acute or chronic arthritis, months to years after initial symptoms. Patients whose symptoms continue even after initial adequate treatment are thought to have persistent infection, which afflicts up to 10% of all patients who are treated appropriately.[12-15] In addition, patients with chronic or delayed musculoskeletal symptoms may have developed synovial autoimmunity, in which the original Lyme infection triggers a synovial autoimmune response, leading to continued inflammation.[16] As a result, a combination of persistent infection and synovial autoimmunity may have created an environment in which our patient's patellar tendon rupture was possible.

Patients with joint manifestations of Lyme disease usually present with low-grade fever, painful oligoarthritis (the knee being most commonly affected), and joint effusion.[11,17] All these symptoms were present in our patient. In addition, her laboratory studies—negative synovial fluid bacterial culture (fluid WBC count, 22,800) in the context of elevated serum WBC count, CRP, and ESR—pointed to a nonbacterial, inflammatory etiology. Further, presence of elevated IgG antibodies in the blood confirmed prior exposure/infection with *B burgdorferi*.[18]

Yet, confirming the diagnosis of Lyme arthritis depends on examining synovial fluid, particularly on performing synovial fluid PCR, which can detect spirochete DNA.[19-21] PCR has been shown to have specificities and sensitivities as high as 96% and 80%, respectively, in detecting the DNA of *B burgdorferi* in synovial fluid.[22] In addition, PCR can track response to treatment, as PCR becomes negative after disease eradication.[19] Our patient had a positive synovial fluid PCR analysis—which was negative after treatment—further confirming that the etiology of knee inflammation (and subsequent tendon rupture) was likely Lyme disease.

Consistent with the late phase of Lyme disease[23] is our patient's combination of a 5-year delay from initial disease exposure, infection, and oral treatment to onset of knee effusion and pain; markedly elevated IgG; nonspecific elevation in IgM; and positive PCR synovial fluid analysis for *B burgdorferi*. Yet, we believe that the present report is the first of a patient with late-phase Lyme disease (ie, oligoarthritis) presenting with an extra-articular manifestation of the disease around the knee joint, or a nontraumatic midsubstance patellar tendon rupture as a sequela of the inflammation caused by the disease.

"Lyme disease should be in the clinician's differential for patients presenting nontraumatically with patellar tendon disruptions."

This case report in fact exemplifies another possible manifestation of late-phase Lyme disease. It is essential that clinicians realize this possible connection between Lyme disease and patellar tendon rupture so that they can adequately treat the underlying disease process with antibiotics (rather than simply correct the rupture itself) when faced with a patient presenting to clinic with a history of Lyme disease, constitutional symptoms, knee effusion, and an examination suggestive of patellar tendon disruption. In addition, if the clinician encounters a midsubstance tear in the operating room (away from the area of most biomechanical stress[3]), suspicion for an inflammatory condition such as Lyme disease leading to the rupture should also be heightened. This is particularly important, as failure to treat Lyme arthritis can lead to prolonged synovitis, cartilage loss, bone cysts, and chronic pain.[24,25] Lyme disease should be in the clinician's differential for patients presenting nontraumatically with patellar tendon disruptions, particularly when a history of exposure has been documented.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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