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High-Grade Partial Tear of the Biceps Femoris Tendon in a Patient with Type 2 Diabetes and Calcium Pyrophosphate Deposition Disease: A Case Report

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Abstract: Calcium pyrophosphate deposition (CPPD) disease is a common condition that results in intra-articular and periarticular deposition of calcium pyrophosphate crystals. Tendon tear, however, is infrequently reported in these cases. The factors contributing to tendon tear in patients with CPPD disease may include crystal-induced prolonged inflammation that could cause chronic tendinous attrition, anatomical and biomechanical factors that could compromise tendon structural integrity, and/or degenerative tendon changes that might be caused by co-occurring diabetic tendinopathy. We report a case of a high-grade partial tear of the biceps femoris tendon in a patient with type 2 diabetes and CPPD disease.

Keywords: *tendon rupture, calcium pyrophosphate dihydrate crystals, CPPD, biceps femoris tendon, calcium pyrophosphate dihydrate deposition disease, chondrocalcinosis*

Introduction

Calcium pyrophosphate deposition (CPPD) disease is a common arthropathy that results in intra-articular and periarticular deposition of calcium pyrophosphate (CPP) crystals.[1,2] The disease manifests itself as intermittent acute monoarthralgia or polyarthralgia and swelling simulating a gout attack. For this reason, the disease was formerly called pseudogout. In fact, CPPD disease is commonly associated with gout and other metabolic conditions, such as hemochromatosis, hypomagnesemia, hypophosphatasia, and hyperparathyroidism.[1] The radiographic hallmark of CPPD disease includes chondrocalcinosis, most commonly involving the triangular fibrocartilage complex of the wrist, the menisci of the knee, and the acetabular labrum.[1] The features of a more advanced disease — subchondral sclerosis, subchondral cysts, and osteophytes, resemble those of osteoarthritis, but have a different anatomic distribution.[3] Tendon tear, however, is infrequently reported.[4] We present a case of a high-grade partial tear of the biceps femoris tendon in a patient with type 2 diabetes and CPPD disease.

Key Points

- Tendon tear can be associated with calcium pyrophosphate deposition disease.
- Factors contributing to tendon tear in patients with calcium pyrophosphate deposition disease might include chronic inflammation, mechanical forces, anatomical abnormalities, and/or co-occurring diabetic tendinopathy.

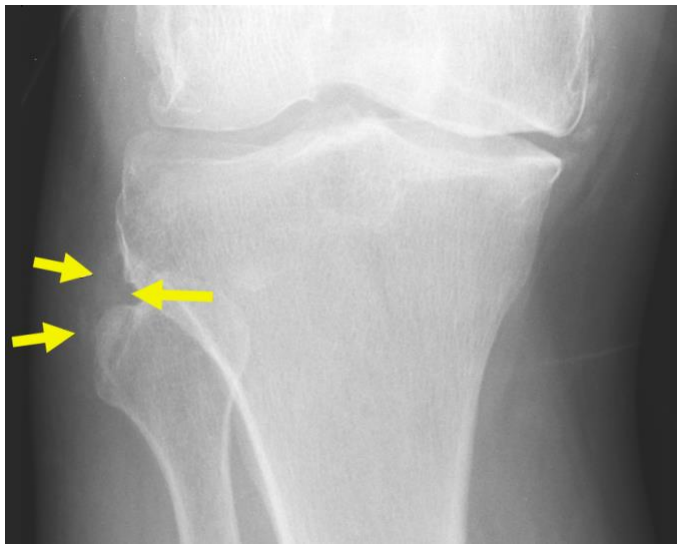
Case Presentation

A 69-year-old man with a history of type 2 diabetes (Hemoglobin A_{1c} – 9.8%; reference range, <5.7%), hyperlipidemia, hypertension, and osteoarthritis presented to a primary care physician after three days of pain in the lower part of the right leg. There was no recent history of trauma or deep vein thrombosis. On physical examination, the patient was afebrile, but the region of the right fibular head was warm, swollen, and tender, although with no surrounding cutaneous wound or calf tenderness. The results of the initial laboratory analysis showed white blood cell count within the reference range and mildly elevated C-reactive protein. The radiograph of the patient's right tibia and fibula showed some soft tissue swelling around the fibular head, and faint chondrocalcinosis in the region of the distal fibular insertion of the biceps femoris and the fibular collateral ligament insertion (Figure 1A), which was similar to that seen 5 years earlier (Figure 1B) on one of the several occasions when the patient was evaluated for knee pain due to osteoarthritis. The patient was subsequently referred to an orthopedic and sports physician and underwent magnetic resonance imaging (MRI) of the right lower extremity that showed fibular head edema and cortical irregularity as well as edema and enhancement of the distal fibular insertion of the biceps femoris (Figure 2). The fibular head edema was thought to indicate a nondisplaced fibular head fracture, so the patient's leg was placed in a short-leg cast. The patient returned two weeks later with new tenderness in the calf of the right leg. The lower extremity ultrasound showed thrombus of the anterior tibial vein (Figure 3). The deep vein thrombosis was likely caused by immobilization, and the patient was subsequently given rivaroxaban.

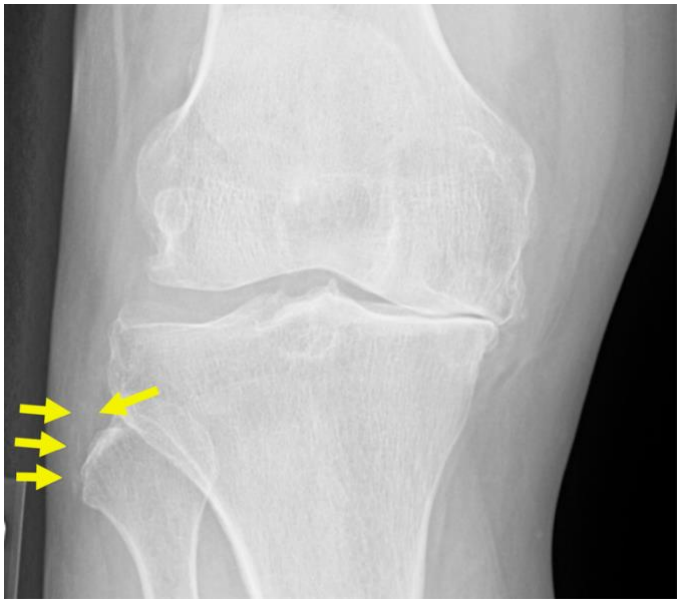
Tenderness of the fibular head, however, did not subside after immobilization. A repeated radiography redemonstrated meniscal chondrocalcinosis. A repeated MRI of the right lower extremity and dedicated MRI of the right knee showed persistent edema of the fibular head, a high-grade tear of the biceps femoris tendon, and cystic change of the lateral tibial plateau (Figure 4). Postcontrast sequences revealed enhancement of the fibular head and the distal fibular insertion of the biceps femoris as well as mild enhancement of the lateral tibial plateau (Figure 4). At this time, the patient declined further workup. However, after presenting

Figure 1. Radiography of the Right Lower Extremity of a 69-Year-Old Man with Calcium Pyrophosphate Deposition (CPPD) Disease

A Anteroposterior view



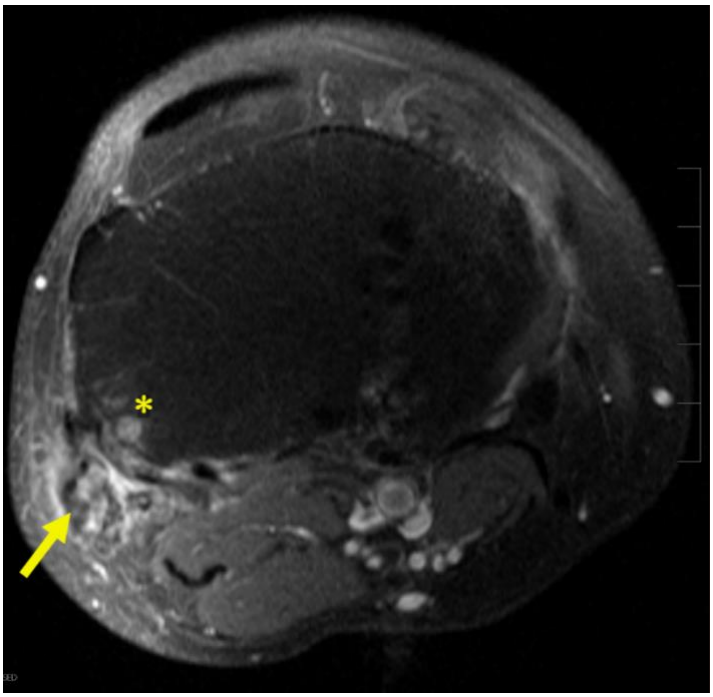
B Anteroposterior view (comparison)



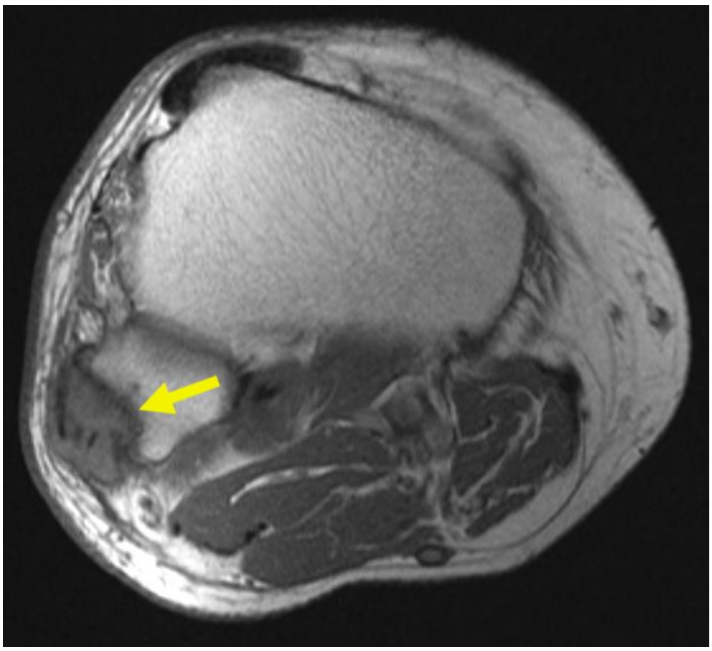
(A) Radiograph of the right lower extremity shows faint chondrocalcinosis and soft tissue swelling (A, arrows) in the region of the distal fibular insertion of the biceps femoris and the fibular collateral ligament insertion. (B) Radiograph obtained 5 years earlier shows that the faint chondrocalcinosis (B, arrows) was present then.

Figure 2. Initial Magnetic Resonance Imaging (MRI) of the Right Lower Extremity of a 69-Year-Old Man with Calcium Pyrophosphate Deposition (CPPD) Disease.

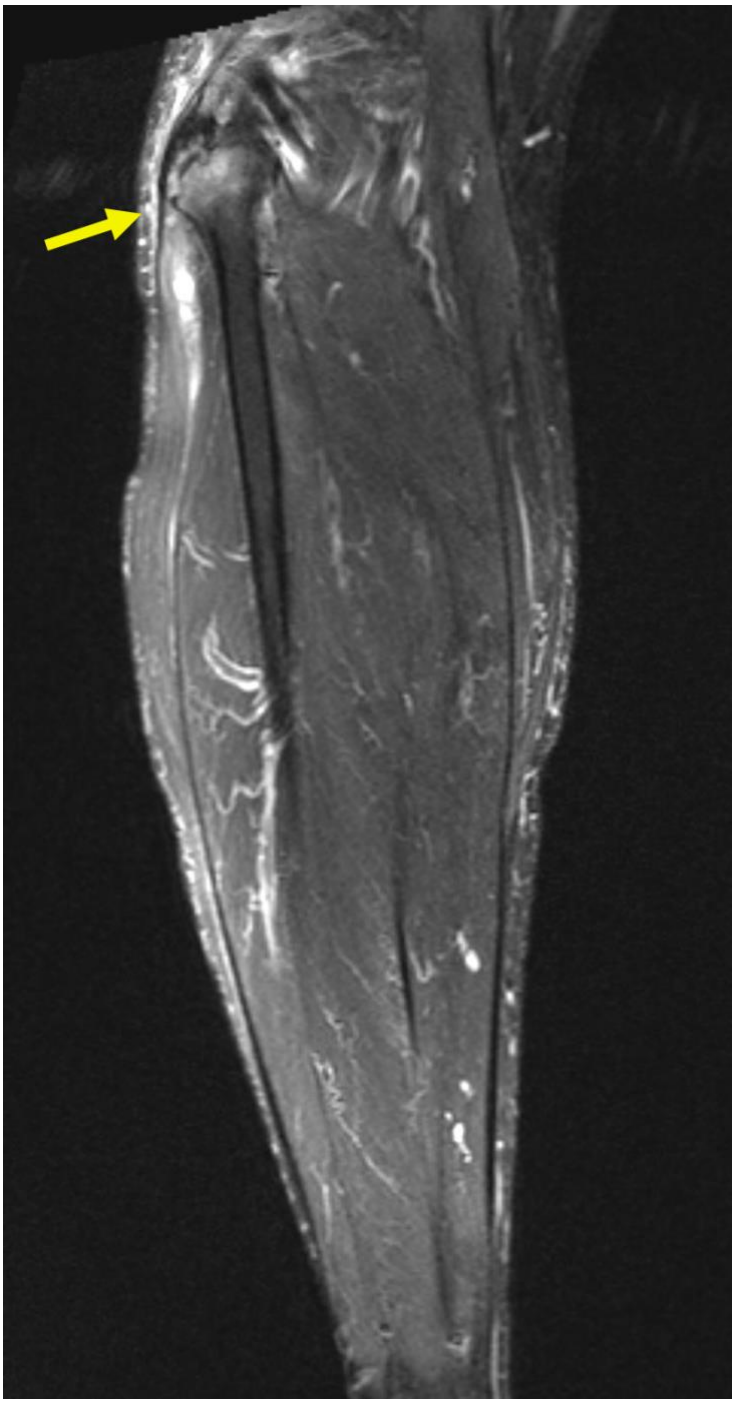
A Fat-saturated postcontrast T1-weighted turbo-spin echo MRI, axial view



B T1-weighted MRI, axial view

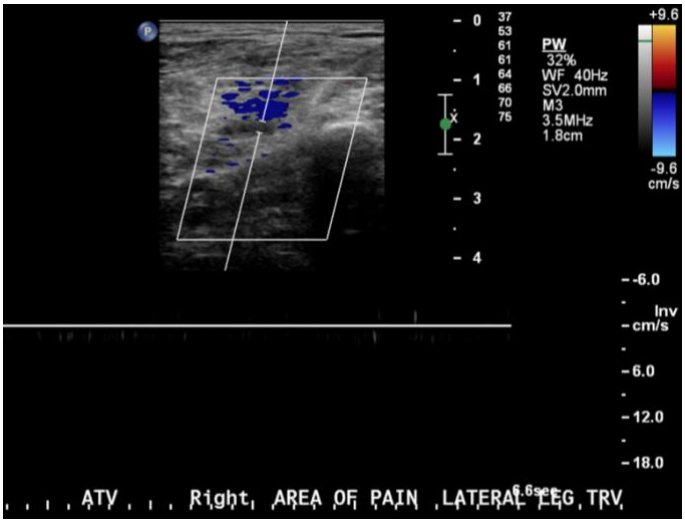


C Short TI inversion recovery MRI, coronal view



(A) The image shows contrast enhancement, thickening, and surface irregularity of the biceps femoris tendon (A, arrow). The adjacent lateral tibial plateau has focal round enhancement (A, asterisk) suggestive of a focal erosion.(B) There is focal contour irregularity at the lateral fibular head (B, arrow) suggestive of a focal remodeling or erosion. (C) The image shows a high-intensity signal at the lateral fibular head and around the distal biceps femoris tendon (C, arrow) suggestive of a nondisplaced fibular head fracture and tendinopathy.

Figure 3. Ultrasonography of the Right Lower Extremity Veins of a 69-Year-Old Man with Calcium Pyrophosphate Deposition (CPPD) Disease



Transverse ultrasonogram of the right lower extremity veins shows an occlusive thrombus of the right anterior tibial vein, as marked by spectral Doppler gate.

to the emergency department one week later with acute pain and swelling in the left elbow, the patient consented to arthrocentesis. The analysis of elbow fluid revealed the presence of intra-articular CPP crystals, elevated white blood cells count (19730/μL; reference range, 4160/μL – 9950//μL), and no bacteria. The patient was prescribed 0.6mg of colchicine twice daily as needed for pain and weekly physical therapy for 12 weeks, after which pain in both the left elbow and the right lateral knee nearly resolved.

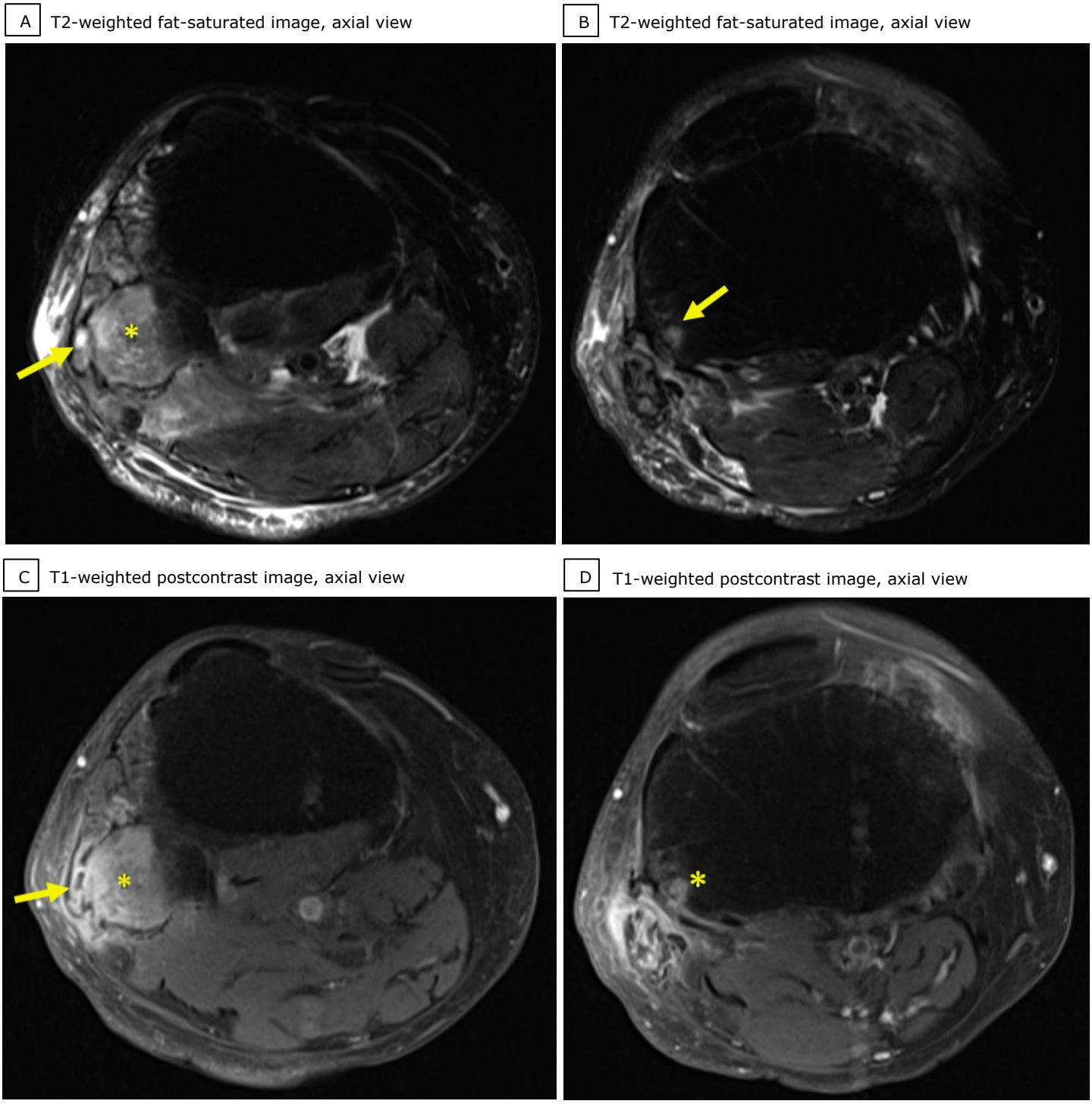
Discussion

Although the pathogenesis of CPPD disease remains unknown, it has been linked to several predisposing conditions, such as hyperparathyroidism, hemochromatosis, hypophosphatasia, hypomagnesemia, gout, and rheumatoid arthritis, as well as to mutations in the progressive ankylosis protein homolog (*ANKH*) (OMIM *605145) and the osteoprotegerin (*TNFRSF11B*) (OMIM *602643) genes.[3,5] High concentration of extracellular pyrophosphate in synovial fluid in joints of patients with gout, osteoarthritis, or rheumatoid arthritis increases the risk of CPPD disease.[6] Furthermore, damage of the cartilage matrix, the presence of type I collagen and articular cartilage vesicles as well as the composition of the latter, and, possibly, excessive osteoclastogenesis in the subchondral bone can also contribute to CPP crystal formation and increase the risk of CPPD disease.[3,5] Acute flares of the disease may be the result of shedding of CPP crystals into the joint space, crystal phagocytosis, and activation of inflammasome,[3,5] triggering release of a potent proinflammatory cytokine inetrleukyn-1-beta and formation of neutrophil extracellular traps.[3,5,7] Chronic CPPD disease-associated arthritis can result in a prolonged inflammatory response that contributes to cartilage degeneration.[3,5]

While being formed extracellularly, CPP crystals may deposit in all anatomical aspects of the joint and in the soft tissue.[5] In reports of cases with CPPD disease, calcifications in the gastrocnemius tendon were found to be in the range of 21%-41% and always associated with chondrocalcinosis.[8] Tendon rupture in the setting of CPPD disease has been infrequently reported.[4,9-11] As the etiopathology of tendon attrition and rupture in cases of CPPD disease remains unknown, it is attributed to multiple aspects of CPP crystal-induced inflammation – high-grade synovitis,[9] synovial hyperplasia,[9] calcium deposits in the tendon's sheath,[4] the presence of osteophytes,[11] and/or to local anatomical[9] and biomechanical abnormalities[4,9] as well as type 2 diabetes[12] when it co-occurs with CPPD disease.

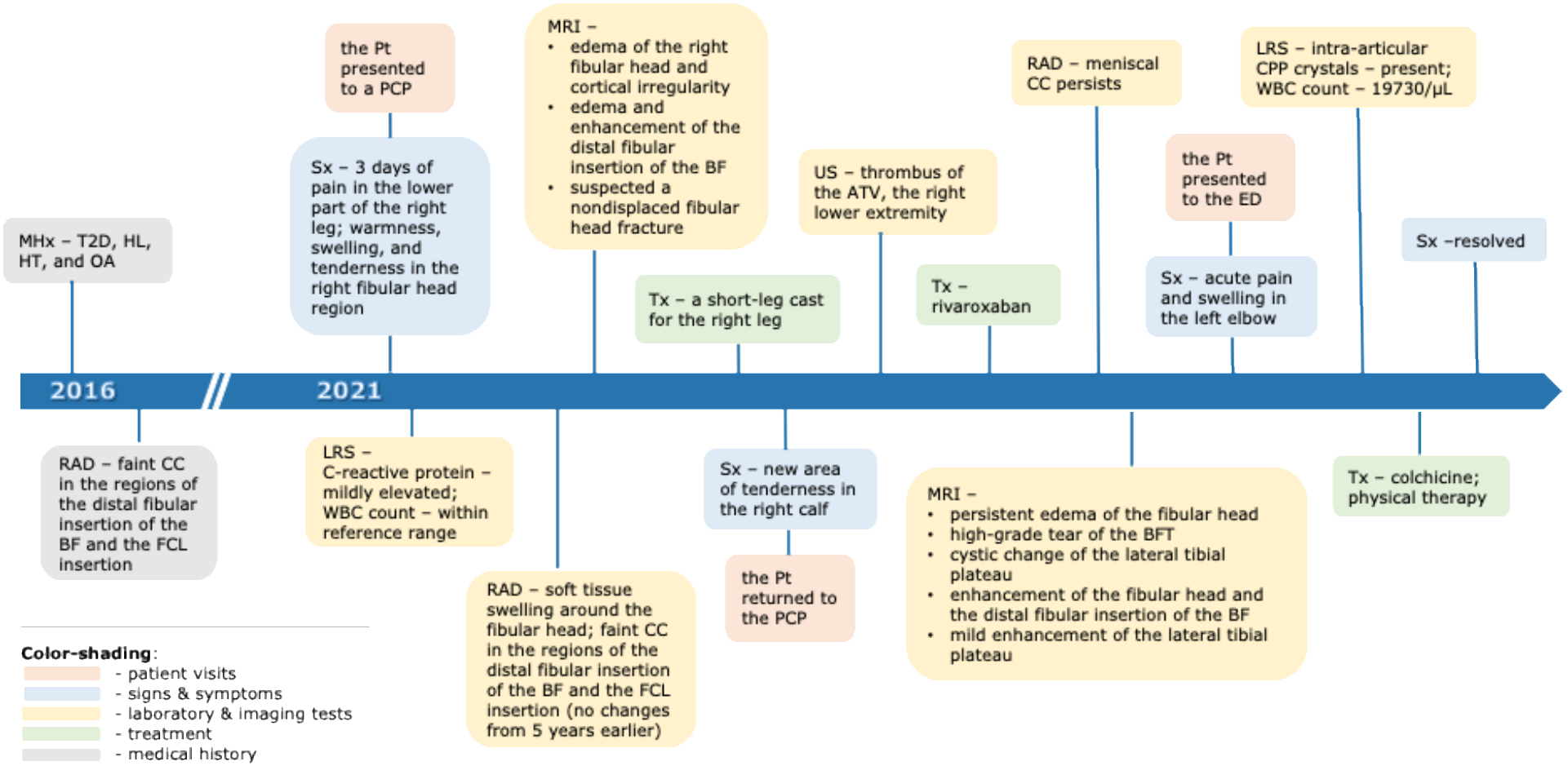
Although the presence of CPP crystals in our patient's knee was not verified because of the patient's refusal to undergo arthrocentesis, the procedure was used to confirm a subsequent acute CPPD disease flare in the patient's elbow. This made us believe that the acute partial tear of the biceps femoris tendon occurred likely secondary to CPPD disease-induced inflammation, especially considering that chondrocalcinosis was noted at the fibular head in the expected location of the biceps femoris tendon insertion. In addition, the radiograph of the patient's right lower extremity showed meniscal chondrocalcinosis that was also the sign of the presence of CPPD disease, the finding that suggested that arthropathy caused by CPPD and not just primary osteoarthritis was most likely the cause of pain experienced by the patient in the past. The MRI findings showed that this

Figure 4. Repeated Magnetic Resonance Imaging (MRI) of the Right Lower Extremity and a Dedicated MRI of the Right Knee of a 69-Year-Old Man with Calcium Pyrophosphate Deposition (CPPD) Disease.



(A) The image shows persistent edema of the fibular head (A, asterisk) and a high-grade tear of the biceps femoris tendon (A, arrow). (B) An area of cystic change is seen at the lateral tibial plateau (B, arrow). (C) At the level of the tibial plateau, there is a persistent enhancement of the bicpes femoris tendon at its attachment to the fibular head (C, arrow) and of the fibular head itself (C, asterisk). (D) A focus of enhancement is seen at the lateral tibial plateau (D, asterisk).

Case report timeline



Abbreviations: ATV, anterior tibial vein; BF, biceps femoris; BFT, biceps femoris tendon; CC, chondrocalcinosis; CPP, calcium pyrophosphate; ED, emergency department; FCL, fibular collateral ligament; HL, hyperlipidemia; HT, hypertension; LRS, laboratory results; MHx, medical history; MRI, magnetic resonance imaging; OA, osteoarthritis; PCP, primary care physician; Pt, patient; RAD, radiography; Sx, symptoms; T2D, type 2 diabetes; Tx, treatment; US, ultrasonography; WBC, white blood cells

partial tear of the biceps femoris tendon occurred on the background of a large-volume synovial effusion, synovitis, and chondromalacia, all of which indicated the presence of chronic inflammation with sudden exacerbations of the disease and cartilage degeneration. It is possible, however, that a high-grade partial tear of the biceps femoris tendon in our patient was a result of a synergistic effect of co-occurring type 2 diabetes that caused diabetic tendinopathy and CPP crystal-induced inflammation that aggravated the tendon's structural and functional deterioration.

Author Contributions

Conceptualization, V.G. and P.A.; Acquisition, analysis, and interpretation of data, V.G. and P.A.; Writing – original draft preparation, P.A.; Review and revisions, K.N. and R.L.; Supervision, V.G. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All authors had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Disclosures

None to report.

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