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【 CASE REPORT 】

Calcium Pyrophosphate Deposition Disease Involving a Lumbar Facet Joint Following Urinary Tract Infection

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Abstract:
A 75-year-old woman was admitted with urosepsis due to *Escherichia coli* infection. After improvement with a ureteral stent and antimicrobial agent, she complained of back pain and showed elevated inflammation marker levels. Arthralgia and arthritis of multiple peripheral joints were noted, and radiography indicated cartilage calcification. Magnetic resonance imaging revealed lumbar facet joint effusion. Her symptoms improved with nonsteroidal anti-inflammatory drug administration. Thus, she was diagnosed with calcium pyrophosphate deposition (CPPD)-related facet joint arthritis (FJA) rather than infectious FJA. CPPD-related FJA is an important differential diagnosis in elderly individuals with a risk of CPPD disease who complain of back pain.

Key words: calcium pyrophosphate deposition, facet joint arthritis, pseudo gout

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Introduction

Calcium pyrophosphate deposition (CPPD) disease involves an inflammatory response to calcium pyrophosphate crystals in the articular cartilage of joints. Generally, plain radiography of joints shows cartilage calcification. The common site of CPPD is the knee and rarely the spine, which is involved in crown dens syndrome. Facet joint arthritis (FJA) has been shown to be commonly caused by bacteria spreading hematogenously (1). Although infectious facet joints are well-recognized, CPPD-related FJA (CPPD-FJA) is not, as it is difficult to prove the existence of CPPD in the facet joint (2).

We herein report a highly suspicious case of CPPD-FJA after improvement of bacteremia involving *Escherichia coli.* CPPD-FJA is a major disease affecting elderly patients complaining of back pain after a febrile illness.

Case Report

A 75-year-old woman was admitted to our hospital with an altered mental status and a fever. She had a history of kidney stones that had been treated with ureterolithotomy 25 years previously. Although her Glasgow coma scale score was 12 (E4V2M6) initially, her mental status immediately became normal after hydration. She had a high fever (temperature, 40.5℃) and tachycardia (heart rate, 102 beats per minute) without hypotension. Additionally, she had tenderness at the right costovertebral angle. Laboratory findings indicated severe inflammation [C-reactive protein (CRP) level, 18.1 mg/dL; white blood cell (WBC) count, 1.24×10^4/μL], renal dysfunction (blood urea nitrogen level, 32.1 mg/dL; serum creatinine level, 1.66 mg/dL), and mild elevation of hepatobiliary enzymes (aspartate aminotransferase level, 81 IU/L; alanine transaminase level, 63 IU/L; lactate dehydrogenase level, 455 IU/L). A urinalysis and Gram staining of urine revealed pyuria with many Gram-negative rods. Ab-

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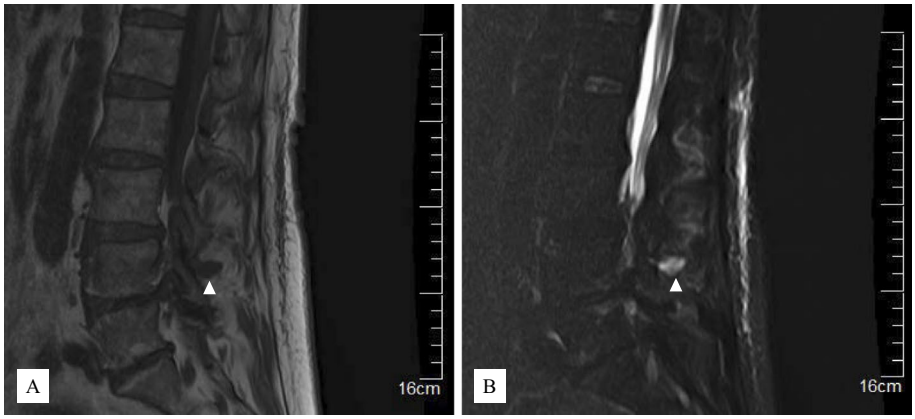

Figure 1. **T1-weighted (A) and short-tau inversion recovery (B) sagittal images showing liquid retention at the facet joint of L4/L5 (white arrowhead).**

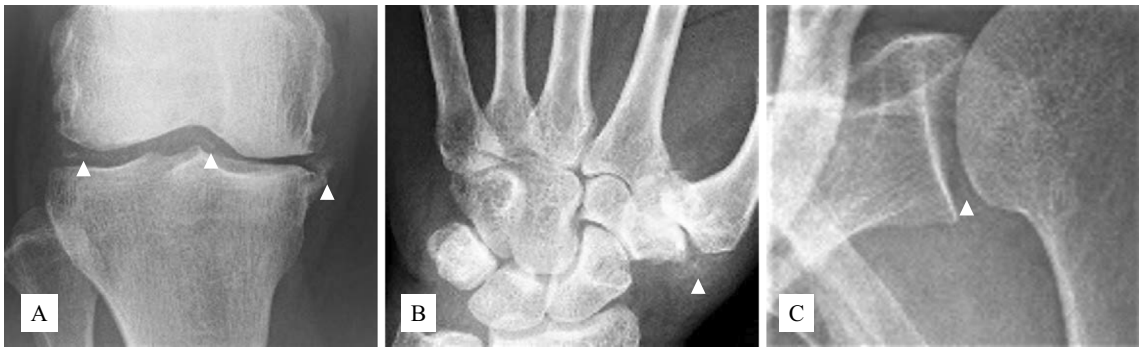

Figure 2. **Plain radiographs showing cartilage calcification (white arrows) in the right knee joint (A), left wrist joint (B), and left shoulder joint (C).**

dominal computed tomography revealed hydronephrosis on both sides associated with a right ureteral stone and left kidney stone. A double-J ureteral stent was placed in the right urinary tract, and doripenem administration (0.5 g every 8 hours) was initiated empirically. *E. coli* was isolated from both blood and urine. It showed resistance to ampicillin and intermediate resistance to cefazolin but was susceptible to other antibiotics. Based on the results of bacterial culture, doripenem was changed to ceftriaxone (2 g once daily) 3 days after admission. Her CRP level and WBC count improved to within normal limits. She was bedridden for several days after being hospitalized, but she showed an improvement in the performance of activities of daily living. We therefore changed ceftriaxone to oral levofloxacin (500 mg once daily).

However, 8 days after starting levofloxacin, she complained of a fever and low back pain, which worsened on knocking or rotating her lumbar spine. On an assessment, high inflammation (WBC count, 12×10^4/μL; CRP level, 20.1 mg/dL) was detected. Although we suspected recurrent pyelonephritis associated with occlusion of the urethral stent, there was neither pyuria nor bacteria on a urinalysis nor any worsening of hydronephrosis. We performed blood culture tests again, but the results were negative. She had transient joint pain, and tenderness of the right knee, left wrist, and left shoulder was noted 9, 10, and 11 days after hospitalization, respectively. Magnetic resonance imaging (MRI) of the lumbar region was performed to assess her persistent back pain, and it revealed liquid retention at the lumbar facet joint (L4-L5) without findings compatible with discitis or vertebral osteomyelitis (Fig. 1). Radiography showed cartilage calcification at her wrist, knee, and shoulder (Fig. 2). Although we continued to administer levofloxacin because bacterial FJA could not be excluded, her symptoms did not improve despite receiving antibiotics effective against *E. coli* isolated initially. CPPD-FJA was suspected considering her imaging findings and clinical course; thus, nonsteroidal anti-inflammatory drugs (NSAIDs) (loxoprofen sodium, 180 mg/day) were administered. After starting NSAID administration, her joint pain and fever improved dramatically. NSAID administration was discontinued on the fifth day, and she was discharged from our hospital 19 days after admission. She did not show recurrence after discontinuation of NSAID administration.

Discussion

We encountered a case suspected of being CPPD-FJA following treatment for urosepsis due to *E. coli.* Although it was difficult to distinguish the cause of FJA between CPPD and bacterial infection, we eventually made a diagnosis of CPPD-FJA because of the patient's poor response to suscep-

tible antibiotics and the presence of arthritis in multiple joints with cartilage calcification that was controlled by NSAID administration.

FJA is a well-known rare clinical feature of spinal infection that was first reported in the literature in 1911 (3). However, spinal MRI has facilitated the diagnosis of FJA regardless of a septic or non-septic condition. The most common cause of FJA is bacterial hematogenous dissemination from a distant focus of infection, such as acute pyelonephritis (4). Non-septic FJA has been reported to occur due to cartilage damage associated with osteoarthritis and crystal-induced arthritis as CPPD disease.

Acute calcium pyrophosphate crystal arthritis is the most widely recognized form of CPPD disease and is referred to as pseudogout. The inflammatory response to calcium pyrophosphate crystals manifests as redness, heat, swelling, pain, and loss of function in the joint and resembles septic arthritis. CPPD disease tends to occur in elderly women and is rare in patients younger than 60 years of age (5). Although CPPD disease typically manifests as acute monoarthritis, migratory or additive arthritis and polyarthritis have also been noted as rare clinical presentations (6). The common sites of CPPD are the knees, pelvis, wrists, and other joints (7). While CPPD disease sometimes involves the cervical spine, as in the case of crown dens syndrome (8), that involving the lumbar spine is rare (2, 8-11), although all segments of the spine can be affected. CPPD disease affecting a spinal joint may be attributed to a previous injury, tissue necrosis, or degenerative disease of the spine (2). Although our patient did not have a history of spinal injury, spondylolisthesis, which is common in postmenopausal women, may have played a role. CPPD disease is diagnosed by the detection of calcium pyrophosphate crystals in the synovial fluid. However, it is difficult to collect fluid from the facet joint, as the articular cavity is very small. The imaging findings for cartilage calcification are commonly identified in patients with CPPD disease. Even if imaging findings are not obtained for patients with severe cartilage loss, the possibility of CPPD disease cannot be disregarded (7).

In the present case, the patient was considered to have CPPD-FJA because she responded well to NSAIDs, and radiography findings of many joints indicated cartilage calcification. Furthermore, FJA in this case was confirmed not to have been caused by another blood stream infection incurred during treatment of urinary tract infection because the blood culture results after the second febrile episode were negative. CPPD-FJA is not generally recognized due to its difficult diagnosis. Thus, its prevalence might be underestimated in elderly patients with back pain. With regard to the management of FJA, septic FJA should be ruled out because spinal infection often results in severe complications, such as neurological deficits, and it is necessary to consider the possibility of CPPD-FJA in elderly individuals.

In conclusion, we encountered a case suspected of being CPPD-FJA following improvement of bacteremia involving *E. coli*.

The authors state that they have no Conflict of Interest (COI).

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