

Candida Septic Arthritis with Rice Body Formation: A Case Report and Review of Literature

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Rice body formation in a joint or bursa is a rare condition, and is usually associated with rheumatoid arthritis or tuberculous arthritis. Here we describe a case of multiple rice body formation in a shoulder joint and in adjacent bursae, which was confirmed to be due to septic arthritis by *Candida* species. To the best of our knowledge, rice body formation in *Candida* septic arthritis in an immune-competent patient has not been previously reported.

Index terms: Rice bodies; *Candida*; Arthritis; MRI

INTRODUCTION

Formation of multiple rice bodies in bursae of joints is generally a rare disorder that may be a sign of underlying rheumatic condition or tuberculous infection (1-4). In addition, the condition has been reported in association with other diseases, such as sero-negative rheumatoid arthritis, atypical mycobacterial arthritis, and nonspecific arthritis (5-11). However, to the best of our knowledge, rice body formation in *Candida* septic arthritis has not been previously reported. We report a case of septic arthritis with formation of multiple rice bodies caused by *Candida* species.

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CASE REPORT

A 74-year-old male patient presented with a complaint of persistent pain and swelling of the right shoulder for six months. According to previous medical records from outside the hospital, he had a history of surgery on his right shoulder at another tertiary hospital 18 months ago, and the pathogen identified at that time was *Candida* species. In addition, he received intravenous and oral antifungal medication (fluconazole) for one month after the operation. He had no history of trauma, or any other medical problems. On physical examination, localized swelling around the shoulder and limited range of motion were observed.

Laboratory examination revealed an elevated erythrocyte sedimentation rate (106 mm/hr) and a high sensitivity C-reactive protein level (4.18 mg/dL). The results of blood examination showed increased counts of eosinophils (7.3%) and monocytes (12.1%), whereas the white blood cell count was within the normal limit (8080 mm³).

Plain radiography of the shoulder showed small bone erosion at the subchondral portion of the glenoid and humeral head (Fig. 1A, B). Subacromial spur and superior migration of the humeral head, suggestive of a rotator cuff tear, along with degenerative osteophytes and joint

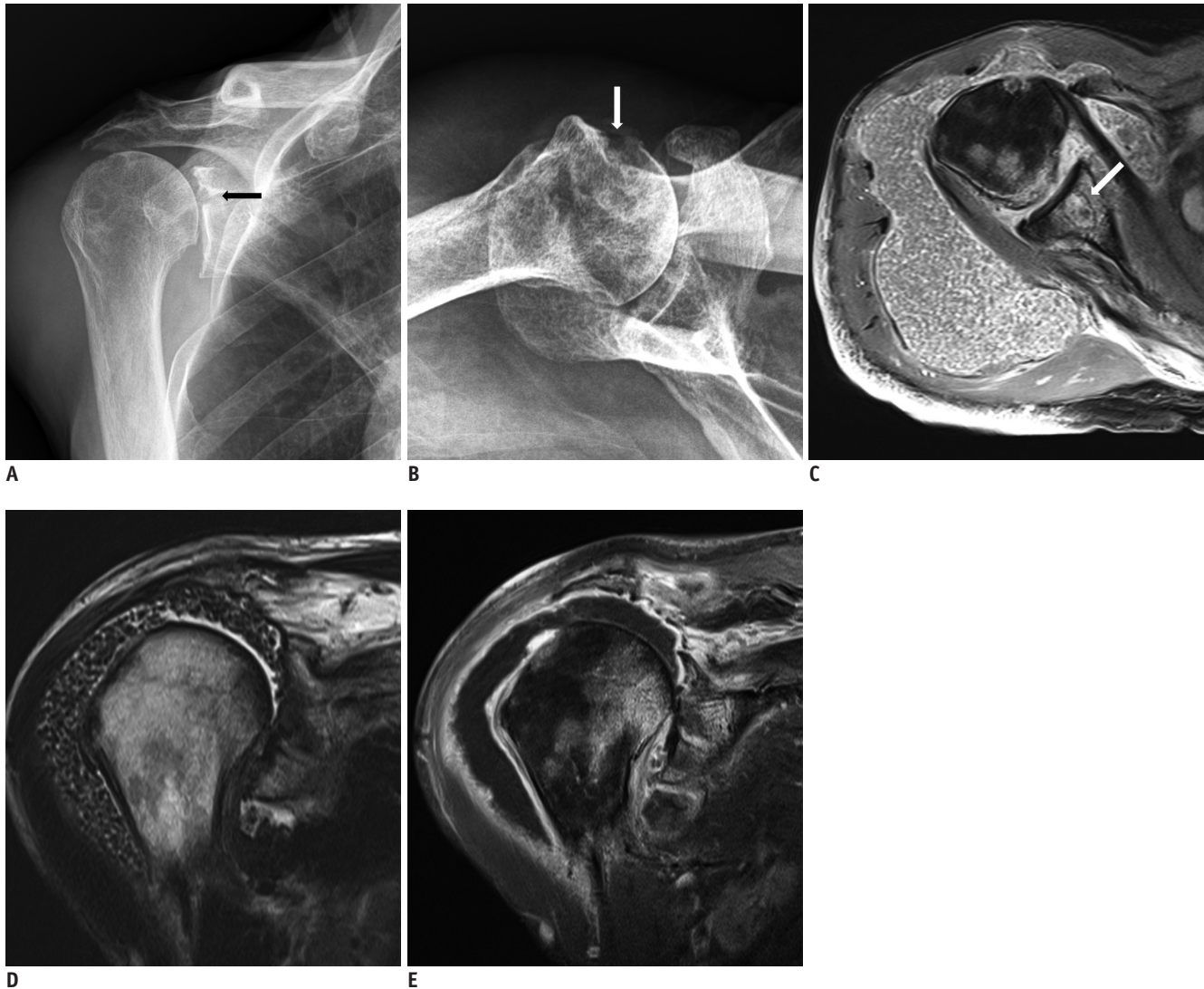


Fig. 1. Imaging and pathologic findings of *Candida* septic arthritis in right shoulder in a 74-year-old man.
A, B. Anteroposterior (A) and axillary (B) radiographs of right shoulder show small bone erosions at glenoid (black arrow) and humeral head (white arrow). Superior migration of humeral head indicates rotator cuff tear. **C-E.** Axial fat-suppressed proton-density weighted image (C) and coronal T2-weighted image (D) show innumerable rice bodies in right shoulder joint, subacromial-subdeltoid bursa, and subcoracoid bursa. Coronal fat-suppressed T1-weighted image after gadolinium enhancement (E) shows diffuse thickened synovium with enhancement suggestive of synovitis. Bone marrow edema in humeral head and glenoid (C-E) with small subchondral bone erosion at glenoid (arrow in C) suggests osseous involvement of inflammation.

space narrowing of the glenohumeral joint were noted. Subsequently, magnetic resonance imaging (MRI) of the shoulder was performed with intravenous gadolinium enhancement. 3.0T MRI (Skyra, Siemens, Erlangen, Germany) showed joint effusion and massive distension of the subacromial-subdeltoid bursa and subcoracoid bursa, with innumerable rice bodies inside, which were isointense to muscle on T1-weighted images, and slightly hyperintense on fat-suppressed proton-density weighted and T2-weighted images (Fig. 1C, D). The joint and bursal spaces communicated through a massive full-thickness tear

of the supraspinatus tendon. Diffuse and some nodular synovial thickening with enhancement was noted on post-gadolinium fat-suppressed T1-weighted images (Fig. 1E), however, the rice bodies did not show enhancement. Multifocal bone erosions were observed at the humeral head, glenoid, inferior surface of the acromion and distal clavicle. Associated bone marrow edema around the erosions of the humeral head and glenoid were visualized on fat-suppressed T2-weighted images, suggesting osseous involvement of the inflammation. In addition, multiple enlarged lymph nodes were identified in the axillary area.

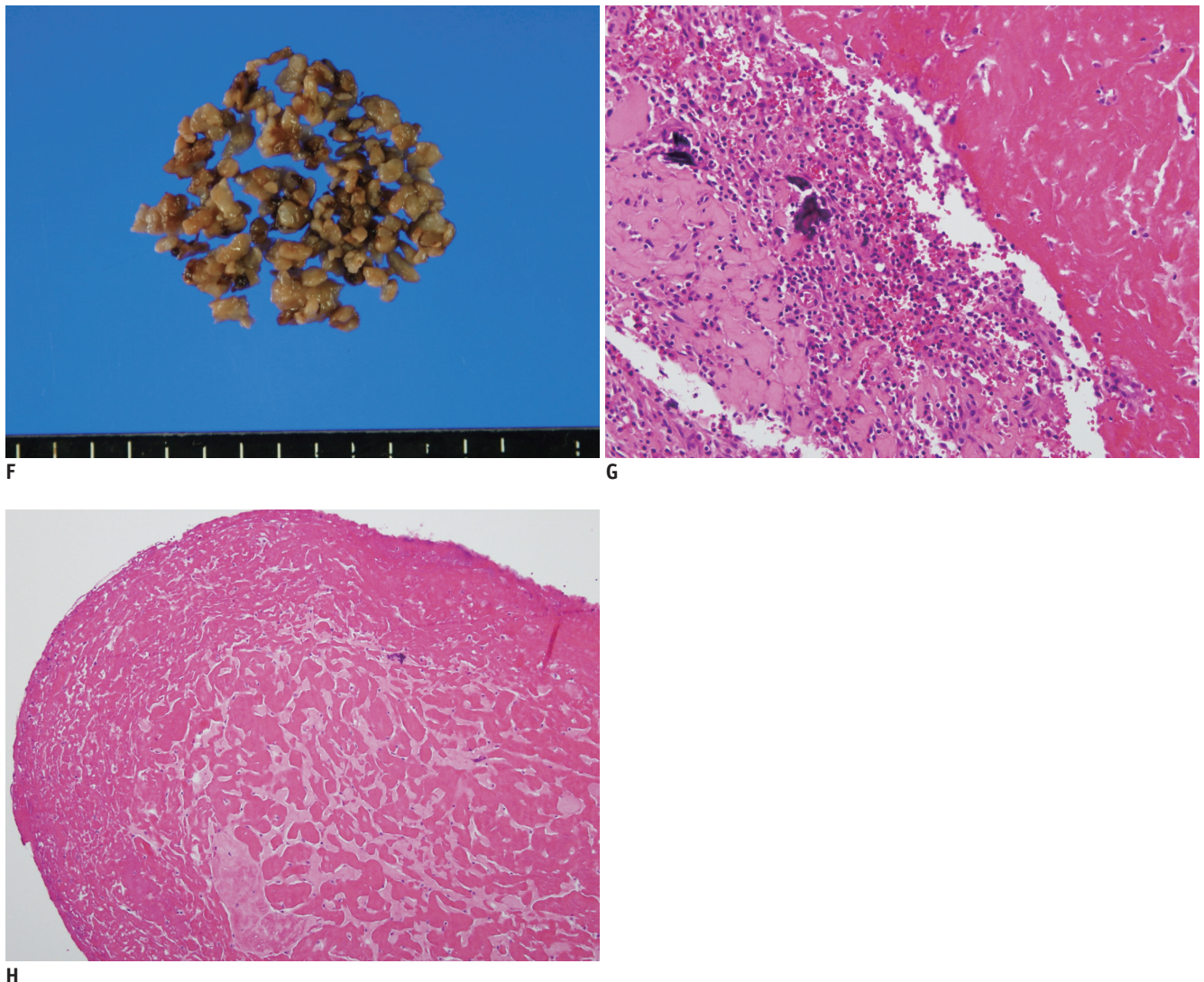


Fig. 1. Imaging and pathologic findings of *Candida* septic arthritis in right shoulder in a 74-year-old man.

F. Resected specimen includes multiple rice bodies of few millimeters in size. **G.** Resected bursa shows infiltration of chronic inflammatory cells with fibrin deposition and dystrophic calcification (H&E, x 200). **H.** Rice body is mainly composed of fibrin with some inflammatory cells (H&E, x 100).

Because he had a history of surgery due to fungal infection, the likely diagnosis was recurrent septic arthritis of fungal origin. Differential diagnoses included tuberculous arthritis and other chronic inflammatory arthritis, such as rheumatoid arthritis, because of the unusual rice body appearance on MRI. Aspiration of joint fluid was performed in the outpatient clinic before admission. Gram-staining under oil immersion detected yeasts, and culture of the fluid identified *Candida parapsilosis*. No bacteria were observed on the Gram-stain and fluid culture. Polymerase chain reaction for *Mycobacterium tuberculosis* (TB-PCR) and cultures for mycobacteria were also negative. Surgical exploration with drainage, bursectomy, and debridement

was performed. Numerous rice bodies of a few millimeters in size were found in the joint and bursae (Fig. 1F), and inflammatory changes were observed in periarticular soft tissue. Histopathologic examination of the thickened bursa and periarticular soft tissue revealed chronic active inflammation with fibrin deposition and dystrophic calcification, without evidence of pannus or granuloma formation (Fig. 1G). The rice bodies were mainly composed of fibrin with some chronic inflammatory cells, hemorrhagic infarct, and dystrophic calcification (Fig. 1H). *Candida parapsilosis* was also detected after the tissue culture. Staining of tissue sections for acid-fast bacteria and tissue cultures for bacteria and mycobacteria were all negative.

The patient did not have any other clinical or radiologic features of rheumatoid arthritis, therefore, serological testing for inflammatory arthritis was not required. He received intravenous treatment with fluconazole, and recovered from swelling and regained range of motion of his shoulder.

DISCUSSION

Rice body formation in joints or bursae is an uncommon finding. It was first described in a patient with tuberculous arthritis (1). According to the literature, the condition frequently occurs in association with rheumatoid arthritis (3, 4), however, it can also be encountered in non-tuberculous mycobacterial arthritis, sero-negative rheumatoid arthritis, juvenile arthritis, adult-onset Still's disease, non-specific arthritis (5-12), and even in osteoarthritis (13), although very rare.

The pathogenesis of rice body formation remains unclear; however, it appears to be an unusual response to synovial inflammation. Some authors have suggested that they may arise from microinfarcted synovium and release of tissue into the joint and subsequent encasement by fibrin deposition (14). Another theory regarding rice body formation is that early rice bodies are formed *de novo* in synovial fluid independently of synovial elements and show progressive enlargement with aggregation of fibrin (3). Histopathological examination of the specimen in our patient revealed fibrin deposition with inflammatory cells in the synovium, and fibrin deposition with inflammatory cells, dystrophic calcification, and hemorrhagic infarction in the rice bodies. We suggest that this pathologic finding supports the former theory on rice body formation. In addition, we propose that any chronic arthritis or bursitis, whether inflammatory or infectious, may cause formation of rice bodies.

Fungal arthritis occurs infrequently and is most commonly caused by *Candida* species. *Candida* species are currently the fourth leading cause of nosocomial bloodstream infections, and, in recent decades, the incidence of *Candida parapsilosis* has shown a dramatic increase (15). Risk factors for *Candida* infection include prolonged vascular catheterization, prior antibiotic therapy, parenteral nutrition, prior surgery, immunosuppressive therapy, malignancy, and prior colonization (15). Our patient had none of the risk factors, except old age. According to medical records from his previous hospital, *Candida* species was identified at the

time of his initial surgery. Therefore, it can be presumed that this is a recurrent infection by *Candida* species. The treatment of arthritis by *Candida parapsilosis* is difficult, showing a high incidence of recurrence (15).

Rice body formation in septic arthritis is not common, and, to the best of our knowledge, this is the first report regarding rice body formation in a patient with *Candida* septic arthritis. Clinical signs of acute infection, such as tuberculous or non-tuberculous mycobacterial infection and fungal infection, are usually not present in chronic atypical infectious arthritis; therefore, imaging studies play an important role in the diagnosis of arthritis. Rice bodies are isointense on T1-weighted MR images and slightly hyperintense on T2-weighted MR images relative to muscle (16). The main differential diagnosis by MRI is synovial osteochondromatosis, however, it rarely involves synovial-lined bursa and has slightly different imaging features. According to one previous report (5), rice bodies caused by atypical mycobacteria can be distinguished from synovial chondromatosis by MRI due to its different composition. As in our case, rice bodies were almost unperceivable or faintly detected on T1-weighted MR images, unlike synovial chondromatosis, which is slightly hyperintense on T1-weighted images. Based on MR imaging findings, we made a preoperative diagnosis of chronic arthritis and bursitis, associated with rheumatoid arthritis or tuberculous arthritis. However, for proper evaluation and treatment, fungal septic arthritis should be included in the differential diagnosis.

Conclusion

In summary, we report on a case of rice body formation in fungal septic arthritis caused by *Candida parapsilosis* in an immunocompetent patient. Given the increasing incidence and its refractory nature, fungal infection by *Candida* species is an important disease, despite its rarity. Therefore, for timely diagnosis and proper management, the differential diagnosis should include fungal septic arthritis in the case of innumerable rice body formation with arthritis or bursitis on MRI.

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