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When Joint Swelling Hides A Secret: The Unexpected Discovery Of Rice Bodies And Literature Review

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Received Date: 17 Jul 2025

Accepted Date: 26 Jul 2025

Published Date: 31 Jul 2025

Citation:

Madalina-Pusa Rosu. When Joint Swelling Hides A Secret: The Unexpected Discovery Of Rice Bodies And Literature Review Annals of Clinical and Medical Case Reports 2025; 14: 1-5

1. Abstract

Rheumatoid arthritis (RA) is a chronic, autoimmune disease characterized by inflammation of the joints and potentially other body systems. It is a long-lasting condition with no cure, but signs and symptoms and disease progression can be managed with treatment. An inflaming rheumatoid arthritis joint contributes to joint tissue damage. This condition may cause chronic or long term discomfort, instability and deformation with functional involvement. In long standing RA, chronic inflammation of the synovial membrane leads to synovial hyperplasia, necrotic or damaged synovial tissue and deposition of fibrin. Over time, this material coalesces into rice bodies.

2. Keywords: rheumatoid arthritis, synovitis, rice bodies

3. Introduction

Rice bodies are small, loose bodies composed of fibrin and other materials, commonly found in joints, bursae, and tendon sheaths. They are often associated with chronic inflammation caused by conditions like rheumatoid arthritis (RA), tuberculous arthritis, or other inflammatory arthropathies [1]. These bodies can cause symptoms such as pain, swelling, and limited range of motion.

In RA, their presence may suggest a more aggressive or long-standing disease, appearing especially in seropositive cases. MRI is the most sensitive imaging method. Definitive diagnosis is often made after aspiration or synovectomy, with histopathological confirmation.

Arthroscopic surgery may be necessary to remove the rice bodies, particularly when they cause significant symptoms or when conservative treatments fail [2, 3, 4].

4. Case Report

A 59-year-old woman with RA since 2003, actually on combined treatment of leflunomide and etanercept, is presenting with arthritis in the hands and left shoulder.

Over time, she received sulfasalazine and methotrexate, but she stopped the treatment due to digestive side effects. From 2013 to 2023 the patient took variable doses of prednisone adjusted according to the severity of the disease. In December 2023 she is presenting to our clinic for the first time with polyarthralgias of the small joints of the hands, right pain in the hip area with marked functional impotence. Corroborating all the data, the side effects of long-term corticosteroid therapy were noted: skin infections, osteoporosis with multiple bone fractures, venous leg ulcer (Figure 1) and, of course, irreversible joint destructions (Figure 2) caused by RA over the course of 20 years. Staphylococcus aureus was isolated from ulcer leg and antibiotic treatment according to the antibiogram was initiated. Also, for RA, the treatment with leflunomide started and corticosteroids were tapered off and discontinued over time.



Figure 1. Venous leg ulcer

Figure 2. Joint destructions

In may 2024, taking into account the active disease (DAS28=5.1), despite multiple Disease-Modifying Anti-Rheumatic Drugs (DMARDs) treatment strategies and after healing venous ulcer (Figure 3) with several courses of antibiotics, we consider it suitable to initiate biologic therapy with etanercept with appropriate screening (hepatitis B and C, latent tuberculosis infection).

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Figure 3. Clinical aspect of venous leg ulcer after antibiotic treatment (remarkable recovery at 4 months)

The evolution was good, with a decrease in inflammation and joint pain for 1 year, until April 2025, when her symptoms relapsed. She was readmitted for arthritis in the hands and left shoulder, with stiffness, and swelling in these joints, impacting daily activities and causing discomfort at rest. Physical examination confirmed a mildly painful soft-tissue mass, mobile, 10 by 10 cm, overlying the left shoulder region (Figure 4).



Figure 4. Swelling of the left shoulder

The mass was first imaged with ultrasound, which revealed an effusion in the subacromial bursa, multiple hyperechoic, intra-articular loose bodies, associated with signs of arthritis including hypervascular synovial, left bicipital tendinitis and fine muscle tears in the shoulder (Figure 5 a,b).

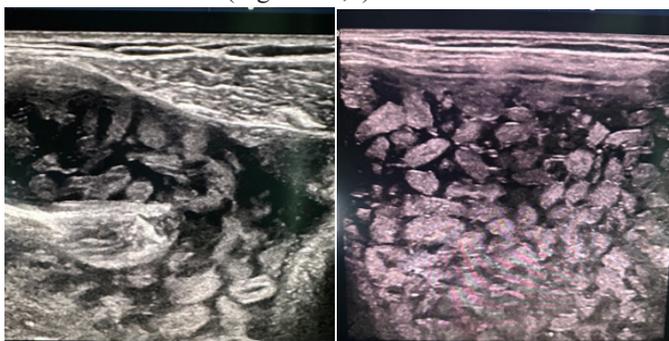


Figure 5 a, b. Effusion in the subacromial bursa, intra-articular loose bodies

Laboratory tests were abnormal suggesting important inflammation. The erythrocyte sedimentation rate was 96 mm/h, and the C-reactive protein was 254 mg/L. Other laboratory findings included leukocytosis 18.820 μ L, elevated procalcitonin 36 ng/ml (negative <0.5), high levels of rheumatoid factor 447 IU/mL and anti-citrullinated protein antibodies (ACPA) 296.8 IU/mL. Kidney and liver function tests were normal. Nasal exudate and pharyngeal exudate were negative too and lung X-ray showed no obvious abnormalities. Plain radiograph of left shoulder revealed lytic and sclerotic lesions in the head of the humerus and scapulohumeral osteoarthritis changes (Figure 6). Hand X-ray showed demineralization with an inhomogeneous appearance at the level of the bilateral carpus, microgeodes, narrowing to the point of disappearance of the joint space and transformation into a bone block, metacarpophalangeal and interphalangeal subluxations (Figure 7).



Figure 6. Lytic and sclerotic lesions in the head of the humerus, scapulohumeral osteoarthritis changes (left shoulder)



Figure 7. Joint-space narrowing, deformities and erosive changes of the hands

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Empiric parenteral antibiotic therapy was initiated (ceftriaxone and vancomycin) together with combination of analgesics for short-term management of pain.

Following the normalization of inflammatory markers and after procalcitonin became negative, patient was referred to the orthopedics department for further evaluation and management. T2-weighted magnetic resonance imaging showed inflammatory changes in the acromioclavicular joint, bone erosions, voluminous intraarticular fluid effusion, free-floating loose bodies, partial rupture of the supraspinatus tendon and hypersignal of the biceps brachial tendon. The bursal lumen was filled with multiple free-floating loose bodies ranging from 1-9 mm in size (Figure 8 a,b).



Figure 8 a, b. MRI of left shoulder axial, coronal and sagittal T1 weighted images showing multiple iso-intense rice bodies and hypo-intense rice bodies.

She underwent an arthroscopy, synovectomy and removal of the suspicious mass of the shoulder. Multiple shiny white rice bodies of different sizes were observed occupying the sub-acromial space and extending into the anterior, lateral and posterior sub-deltoid space. Sub-acromial bursa was excised and loose body excision was performed (Figure 9 a,b,c,d).

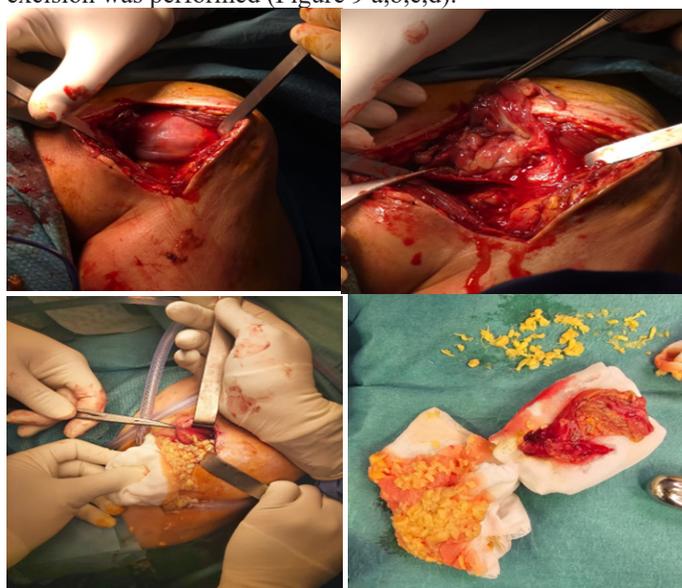


Figure 9 a, b, c. Intraoperative picture showing numerous white-colored loose bodies resemble rice bodies

Synovial fluid was submitted for fluid evaluation, Gram stain,

Ziehl-Neelson stain, Tuberculosis Polymerase Chain Reaction (TB-PCR) and bacterial culture.

Histopathological examination showed hyperplastic synovial lining with marked lymphoplasmocytic inflammatory infiltrates represented by lymphocytes, plasma cells and rare neutrophils. Villous structures were represented by fibrinoid necrosis and rice bodies were composed of dense fibrin deposition with fibroblast and mononuclear cells (Figure 10 a,b,c).

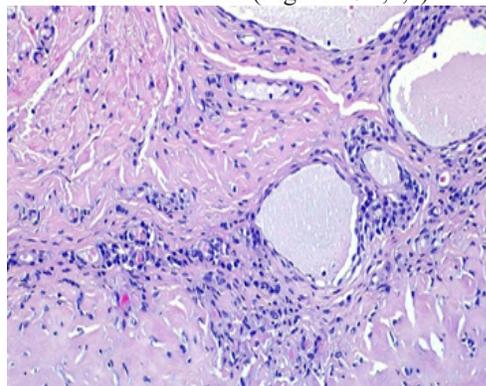


Figure 10 (a). Microscopic detail of the subsynovial area showing marked fibrosis, chronic inflammatory infiltrate and dilated vascular structures. HE stain 20x

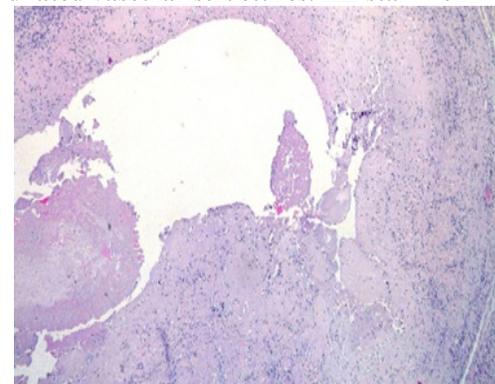


Figure 10 (b). Cystic wall with marked fibrosis, almost completely deprived of synovial epithelium, presenting villous structures inside represented by fibrinoid necrosis. HE stain 10x

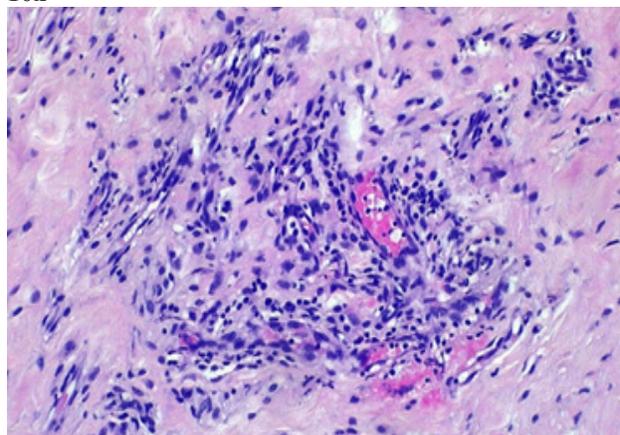


Figure 10 (c). Perivascular inflammatory infiltrate represented by lymphocytes, plasma cells and rare neutrophils. HE stain 40x

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The final diagnosis was rice bodies bursitis secondary to RA. Two weeks after surgery, the wound healed, and the stitches were removed. During the two-month follow-up period, a spectacular improvement in symptoms was seen and the patient restarted leflunomide and etanercept.

5. Discussion

We describe a rare case of a patient with a longstanding RA, suboptimal treated, and chronic low-grade synovitis, insufficiently controlled by corticosteroid therapy for over 10 years, that may have favored the development of rice bodies over time. Although the patient had been undergoing treatment with etanercept for 1 year, it is likely that the rice bodies developed in subacromial bursa due to the persistent localized inflammation through the years. One possible explanation is that biologics may not fully penetrate vascularized bursal tissue or it happens in cases with persistent local inflammation.

Till now, the current medical literature does not describe documented cases of rice body formation in the shoulder specifically in RA patients under biologic treatment.

Considering the risk of developing active TB disease in patients on biologic medications, first of all you must exclude synovial masses with rice bodies associated with tuberculosis infection.

Rice bodies were first described in tuberculosis joints by Riese in 1961 [5, 6]. Rice body formation is usually associated with rheumatoid arthritis, seronegative inflammatory arthritis and tuberculous joints. Rheumatoid involvement of shoulder joint is very rare, especially with association of rice bodies; as seen in our case.

The exact incidence of rice bodies in RA is not well established, as most data come from case reports and small series. However, available studies suggest that up to 20-30% of patients with long-standing or severe RA may develop rice bodies, particularly in bursae or tendon sheaths. In RA-related tenosynovitis, rice bodies have been found in 30-50% of surgical specimens in some orthopedic series. They are more common in seropositive RA and in patients with active, chronic synovitis [7].

The pathogenesis of rice body formation is unclear. Rice bodies usually form secondary to nonspecific response to synovial inflammation. Some authors suggest that they arise from micro-infarcted synovium leading to synovial shedding and subsequent encasement by fibrin derived from synovial fluid. Popert et al [4] propose that early rice bodies are formed in synovial fluid independently of synovial elements and progressively enlarge with aggregation of fibrin. On histological examination, they consist of an inner amorphous core of acidophilic material surrounded by collagen and fibrin. Some contain a core of collagen with a mantle of fibrin while others contain only fibrin. The collagen generally consists of types I, III, and V in a proportion of 40-40-20, very similar to that found in synovial membranes.

The main differential diagnoses of multiple rice bodies in sub-deltoid and sub-acromial space are synovial chondromatosis, tuberculosis, synovial osteochondromatosis and pigmented villonodular synovitis. The MRI characteristics of rice bodies have been well documented, with well-defined nodules of intermediate signal on T1-weighted images and relatively low signal on T2-weighted sequences. In contrast, the nodules

in synovial chondromatosis will appear high in signal on T2-weighted sequences because of the presence of cartilage component. Tubercular rice bodies are easily differentiated from other condition because of involvement of gleno-humoral joint with significant destruction of joint apart from classical clinical symptoms of tuberculosis. Characteristic histopathological picture of caseating granuloma confirms the diagnosis. While synovial osteochondromatosis of the subacromial bursa is rare, the typical presence of soft tissue ossification on radiography should allow differentiation from multiple rice body formation. Pigmented villonodular synovitis can be differentiated from rice bodies by foci of signal voids reflecting hemosiderosis deposition and lack of susceptibility artifact on gradient echo sequences, which is not seen with rice bodies [8, 9].

This case underscore the importance of integrating imaging and histopathology in managing RA patients with persistent joint symptoms, long-standing disease and poorly controlled RA, as well as the necessity for a tailored therapeutic approach to address both systemic inflammation and localized pathology. There is no large-scale epidemiological data quantifying how many RA patients overall develop rice bodies, or how frequent is in the general RA population.

To our knowledge, there are no previously reported cases describing rice body formation in the shoulder of RA patients receiving biologic DMARDs [10, 11, 12]. We presented what we believe is the first documented case of rice body formation in the shoulder of a RA patient treated with etanercept, discovering during surgical exploration.

This apparent gap in the literature may represent underreporting asymptomatic or subclinical rice body formation. Further case series could help clarify whether biological therapy prevent or simply mask rice body formation in RA.

Author Contributions

MR has written the manuscript, DP has carefully corrected and critiqued the manuscript, and all authors have approved the final version of the manuscript.

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