CLINICAL OBSERVATIONS

The phenomenon of intra-articular "rice bodies" in a patient with rheumatoid arthritis: a case report

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Structures resembling "rice bodies" (RB) may be found in patients with rheumatoid arthritis (RA), systemic lupus erythematosus, psoriatic arthritis, and tuberculous tenosynovitis. The joint cavity is a typical site of RB localization. Although the presence of RB may create diagnostic difficulties in patients with rheumatic diseases, they are rarely mentioned in the literature. Various etiological causes of this disorder have been proposed. Magnetic resonance imaging is the main non-invasive method for detecting RB. Pathology examination is used to verify the changes. The article presents a clinical case of RB detection in the cavity of the left shoulder joint in an elderly woman with a long history of RA and comorbidities, and describes the diagnostic methods and treatment approach.

Keywords: *rice bodies; rheumatoid arthritis; synovitis; magnetic resonance imaging; biologic disease-modifying antirheumatic drugs; arthroscopy.*
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Rice bodies (RBs) are small structures visually resembling accumulations of rice grains and localized within joints. They were first described in 1895 by H. Reise in a patient with tuberculosis [1, 2]. Subsequently, it was established that RBs can be detected in patients with rheumatoid arthritis (RA) [3, 4], systemic lupus erythematosus, psoriatic arthritis, and tuberculous tenosynovitis. Typical sites of localization include the joint cavity [5], periarticular bursae [6], less commonly the synovial sheath of tendons [7–9], and the pleural space [5]. According to the literature, RBs were detected in 72% of RA patients during joint lavage, and in 50% of cases in tuberculous tenosynovitis. Several cases of RBs associated with idiopathic tenosynovitis have been described [2, 9]. Although RBs may create diagnostic difficulties in patients with rheumatic diseases, they are rarely mentioned in literature.

We present a clinical case of RB detection in the left shoulder joint cavity of an elderly woman with a long-standing history of RA and comorbid conditions.

Clinical Observation

Patient R.*, a 72-year-old woman, was hospitalized to the day hospital of the Federal State Budgetary Research Institution "V.A. Nasonova Research Institute of Rheumatology" on 10.09.2024 with a diagnosis of: M05.8 Rheumatoid arthritis, rheumatoid factor (RF) +, anti-cyclic citrullinated peptide antibodies (anti-CCP) +, late clinical stage, erosive (radiological stage IV), high activity (DAS28 5.5), functional class 3. Comorbid conditions: osteoporosis complicated by low-energy fracture; malignant neoplasm of the upper inner quadrant of the breast; left breast cancer pT2N0M0, stage 2A, luminal type A. Status after radical mastectomy performed in 2013 with subsequent adjuvant hormonal therapy (tamoxifen until 2021), clinical group 3. Lymphadenopathy. Hypertension grade 2. Left ventricular myocardial hypertrophy. Risk of cardiovascular complications: 3. Target blood pressure level <140/90 mmHg. Mitral insufficiency grade 2. Tricuspid insufficiency grade 2. Cardiac arrhythmias: ventricular and supraventricular ectopy.*

RA was diagnosed in 2011; clinical manifestations were dominated by arthritis of hand joints and generalized arthralgias. The patient was treated with nonsteroidal anti-inflammatory drugs (NSAIDs) and methotrexate (MTX) in combination with folic acid 5 mg/week. MTX was poorly tolerated; consequently, the drug dosage was inconsistent, with brief episodes of increase to 20 mg/week during severe arthritis exacerbations and reduction to 12.5 mg/week with improvement. In 2022, MTX was discontinued and leflunomide (LEF) 20 mg/day was initiated with incomplete clinical and laboratory response; RA activity persisted. At that time, the patient noted the onset of pain and restricted motion in both shoulder joints, more pronounced on the left.

Magnetic resonance imaging (MRI) of the left shoulder joint from 25.01.2022 revealed a significant amount of encapsulated, heterogeneous fluid with admixtures of fat and fibrin within the joint cavity, and signs of chronic erosive arthritis of the shoulder and acromioclavicular joints were identified (Fig. 1a). The patient was examined by an orthopedic traumatologist, who recommended conservative treatment and intensification of anti-rheumatic therapy.

In March 2023, during routine examination, lymphadenopathy of axillary and inguinal lymph nodes was detected; consequently, LEF therapy was discontinued. The patient was examined by an oncologist, and a diagnostic search was performed for cancer recurrence and lymph node metastasis. According to fine needle biopsy of the lymph node from 06.04.2023, the changes were considered consistent with reactive lymphoid hyperplasia. In December 2023, due to high RA activity, MTX therapy was resumed at a dose of 15 mg/week.

Blood tests from 15.01.2024 showed: erythrocyte sedimentation rate (ESR) by Westergren — 31 mm/h; C-reactive protein (CRP) — 23.84 mg/L (normal: 0–5 mg/L).

In the summer of 2024, the patient noted significant deterioration manifested by pain, swelling, and restricted movement in small hand joints, and inability to make a fist. At the same time, she first noticed marked deformity and enlargement of the left shoulder

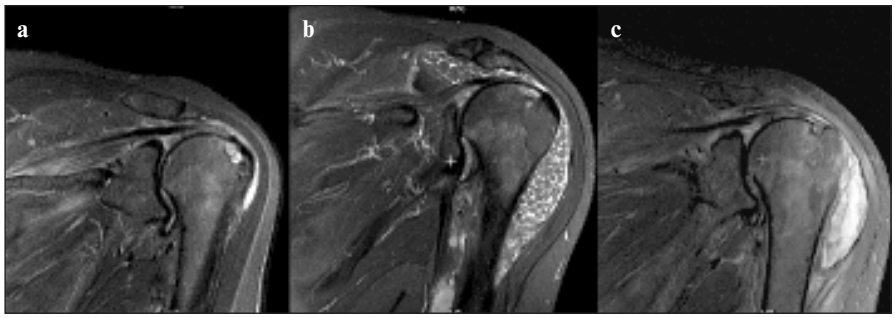

Fig. 1. *MRI of the left shoulder joint, STIR T2-weighted images: a – examination from 25.01.2022. A significant amount of encapsulated heterogeneous fluid with admixtures of fat and fibrin is visualized in the joint cavity; b – examination from 10.10.2024. Total filling of the joint cavity with chondromatous bodies is noted; c – examination from 03.12.2024. Decrease in the amount of free fluid and heterogeneous content in the joint cavity*

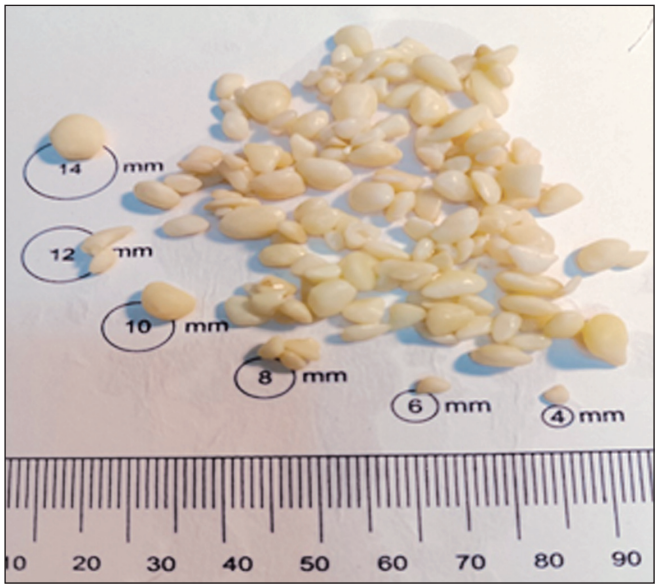

Fig. 2. *Macroscopic specimen. Rounded and oval fragments of soft-elastic tissue*

joint. Blood tests from 01.07.2024 showed: ESR by Westergren — 20 mm/h; D-dimer — 4433 ng/mL; CRP — 107.39 mg/L.

On 17.07.2024, MTX was discontinued and LEF 20 mg/day therapy was initiated. Due to intensive pain, the patient self-administered dexamethasone 8 mg intravenously three times with brief improvement.

On admission, the leading clinical manifestation was deformity of the left shoulder joint with marked functional impairment due to pain-limited movement. Hospital examination revealed increased ESR to 88 mm/h, CRP level to 166 mg/L, ferritin to 208.1 μg/L, hypochromic anemia (hemoglobin—109 g/L), thrombocytosis—578×10⁹/L as manifestations of systemic inflammatory response, as well as elevated RF concentration to 89.9 IU/mL, anti-CCP to 318.9 U/mL, leukocytosis—12.4×10⁹/L with predominance of neutrophils—8.41×10⁹/L.

MRI of the left shoulder joint on 10.10.2024: compared to the examination in 2022, there were multidirectional dynamics with partial resolution of osteitis foci and complete filling of the joint cavity with chondroid bodies exerting marked mass effect on the adjacent tendons of the shoulder rotator cuff (Fig. 1b).

Given з-х з'х the severity of comorbid pathology and medical history, during the diagnostic workup other etiological causes of pathological changes in the left shoulder joint were considered. The patient was re-examined by an oncologist, who concluded that there were no signs of active cancer and no contraindications to prescribing any RA medications. Since neutrophilic leukocytosis, significant elevation of CRP level, and persistent lymphadenopathy were detected during hospitalization, differential diagnosis with infectious joint involvement with possible dissemination was performed. Local erythema and hyperthermia were absent upon shoulder examination; destructive changes were not revealed on MRI and radiography; procalcitonin and diaskin tests were negative. Thus, compelling evidence suggesting tuberculosis or other infectious processes was not obtained, and elevated leukocyte count was interpreted as a manifestation of RA activity. The patient was examined by an orthopedic traumatologist regarding intra-articular chondroid bodies, and surgical treatment after reduction of clinical and laboratory RA activity was recommended.

The choice of therapeutic strategy was complicated by serious comorbidities, including cardiac disease. A medical council decided to add sarilumab, an interleukin (IL)-6 inhibitor, 200 mg №2 subcutaneously, according to standard regimen to prepare for surgical treatment. After the first injection, rapid reduction of laboratory markers of the disease activity was noted (ESR from 88 to 15 mm/h, CRP from 166 to 2.7 mg/L), as well as a decrease in the volume of the left shoulder joint, and pain reduction.

On 12.11.2024, arthroscopic chondroplasty of the left shoulder joint was performed. During the surgery, numerous fragments of soft-elastic consistency, rounded and oval, 0.5–1.5 cm in diameter, grayish yellow in color were found in the joint cavity. Most fragments were located separately; others formed conglomerates (Fig. 2).

Microscopically, the fragments consisted of well-demarcated fibrin masses of varying degrees of maturation, mainly of dense character (Mallory staining), with individual cellular elements embedded within them. At the periphery of fibrin masses, cellular structures in a state of degeneration and karyorrhexis were observed. In certain areas, cells were arranged in multiple rows and resembled surface synovial layer I synoviocytes (Fig. 3). Morphological examination conclusion: RBs.

Due to a 4-week treatment interruption following surgery, the patient experienced gradual increase in CRP to 51 mg/L due to increased RA activity; joint pain recurred, and some enlargement of the left shoulder joint was noted. The patient was re-hospitalized in December 2024 to resume IL-6 inhibitor therapy.

Repeat MRI was performed (03.12.2024): compared to the 10.10.2024 MRI, there was a decrease in the amount of free fluid and a marked decrease in the amount of heterogeneous content in the left shoulder joint cavity; appearance of edematous areas in the rotator cuff muscle fibers; increased intensity of osteitis areas in the left humeral head (Fig. 1c).

Levilimab was initiated according to the standard regimen. After discharge, the therapy was continued with satisfactory efficacy and tolerability. Upon examination in January 2025, there was improvement in left shoulder joint function, restoration of the range of motion, and low RA activity (CRP <2 mg/L).

Discussion

RBs represent a nonspecific reaction to chronic inflammation of the synovial membrane. These changes are frequently encountered in RA and may be its initial manifestation [10, 11]. RBs may develop regardless of the disease severity and current medication therapy [12].

The pathogenesis of RBs requires further investigation [8]. Currently, the primary mechanism considered is a nonspecific response of the synovial membrane to prolonged inflammatory process; however, there are hypotheses of non-synovial origin of RBs [2, 13]. In idiopathic tenosynovitis, RB formation is associated with synovial microinfarcts. According to other data, RBs arise in synovial fluid because of fibrin aggregation, independently of the synovial component [14]. Pathomorphologic examination of specimens reveals dense inflammatory infiltrate containing T-lymphocytes, plasma cells, and macrophages with features of proliferative synovitis with synovial cell hyperplasia and hypertrophy, as well as lymphoplasmacytic infiltrate [14, 15]. Rarely, depending on location, RBs may contain capillaries or cartilage [16]. Tenosynovitis with abundant RBs is uncommon and considered a sign of tuberculous or non-tuberculous mycobacterial infection [2, 9, 14, 15, 17].

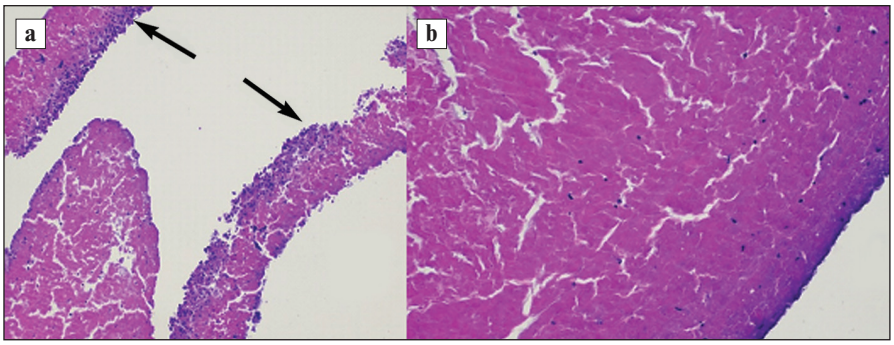

Fig. 3. *Histological specimens. Masses of fibrin. Accumulation of cellular structures at the periphery (arrows). Hematoxylin and eosin staining. ×100 (a); ×200 (b)*

Other causes of RBs may include rheumatic diseases, trauma, and fungal infections [18–21]. It is suggested that RBs in tuberculous infection are larger than those in rheumatic diseases. In our case, morphologic examination revealed fibrin masses with individual cellular elements embedded within them. Notably, in certain areas cells were arranged in multiple rows and resembled surface synovial layer I synoviocytes. Based on these findings, it can be supposed that RBs in our patient originated from the synovium because of chronic inflammatory process in the left shoulder joint.

E. Berg et al. [22] hypothesized that de novo formation of RBs is not associated with pathologic changes in the synovial membrane, and progressive growth of elements occurs as a result of post-inflammatory fibrin aggregation. H.S. Cheung et al. [23] believe that synovial microinfarcts resulting from chronic inflammatory reaction lead to rejection of synovial cells and subsequent encapsulation of these cells by fibrin in synovial fluid. The presence of cellular elements in the center of collagen fiber accumulation in RBs observed in this case suggests their synovial origin.

During patient examination, it is reasonable to include in the differential diagnostic workup pigmented villonodular synovitis, synovial chondromatosis, tuberculous and non-tuberculous tenosynovitis, and sarcoidosis [24, 25]. A distinctive feature of this case was the presence of comorbid conditions in the patient's history, which necessitated considering cancer recurrence and nonspecific infectious process in addition to standard diagnostic evaluation.

The primary instrumental diagnostic method for RBs is MRI. On MRI, RBs appear as multiple formations that are iso- or hypointense on T1-weighted images and minimally hyperintense on T2-weighted images [26], whereas in pigmented villonodular synovitis, iron ions and hemosiderin in the synovial membrane are visualized as hypointense signals on both T1- and T2-weighted images [24]. In synovial chondromatosis, T1-weighted images show iso- or hyperintense rounded formations with hypointense signal on T2-weighted images against a background of hyperintense fluid [8, 9]. Additionally, synovial chondromatosis is more frequently monoarticular. In tuberculous tenosynovitis, formations around tendons are characterized by intermediate intensity on T1-weighted images and high intensity on T2-weighted images [27]. Single publications are devoted to RBs in sarcoidosis; in this case, MRI visualizes rounded formations of high intensity on T2-weighted images [25].

MRI findings in our patient were interpreted as chondroid bodies based on unilateral involvement and typical radiologic characteristics; however, morphologic examination established that the inclusions were RBs.

Treatment of RBs is predominantly surgical, as their removal is frequently accompanied by reduction of clinical manifestations [6, 11]. In most cases, synovectomy is appropriate, especially when RBs are associated with nerve compression syndrome or to prevent complications such as spontaneous tendon rupture [2, 18, 28]. According to the literature, RBs may recur; therefore, conservative therapy and dynamic observation are also indicated [14]. In tuberculous tenosynovitis, recurrence rate exceeds 50% within the first year [9, 27, 28]. In patients with chronic inflammatory disease, RBs recur in the absence of anti-inflammatory treatment [29]. In the presented case, conservative therapy of the underlying disease using bDMARD allowed reduction of RA activity and thus improved the prognosis of the subsequent operation. Since pharmaceutical control of RA activity is necessary to reduce recurrence risk of RBs, bDMARD therapy was continued in the postoperative period with satisfactory efficacy.

Conclusion

RBs are rare pathology in rheumatic diseases that typically require not only anti-rheumatic but also surgical treatment. When corresponding changes are detected on MRI, RBs should be included in the differential diagnostic workup along with other possible pathologies.

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