Rice bodies at bilateral subacromial bursa
Bilateral subakromial bursada pirinç tanesi cisimcikleri
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Abstract
Rice bodies with synovial origin look like cartilage and they are usually composed of fibrin. Tuberculosis, rheumatoid arthritis or other seronegative arthropathies may be the etiology. In this report, a 48-year-old women patient was presented with mild pain and swelling soft tissue masses in both shoulder measuring 7x7 cm. There was no history of trauma. Laboratory tests were normal. Exhaustive investigations for rheumatic disease were negative. Soft tissue masses was regarded as subacromial origin. Both masses were surgically removed and were found to include numerous rice bodies. The patient regained a full and painless range of motion in about 15 days. In the 2-year follow-up, she had neither complaints nor signs of rheumatological disease.

Keywords: Rice bodies, shoulder, subacromial bursa.

Öz

Anahtar Sözcükler: Pirinç tanesi cisimcikleri, omuz, subakromiyal bursa.

Introduction
Rice bodies (RBs), microscopically of fibrin origin and macroscopically brilliant white cartilaginous bodies are generally found in synovial liquid in large numbers. Although the etiology is not clear enough, orthopedic implants, rheumatologic, vascular and infectious pathologies have been blamed to cause synovial hypertrophy. Histological studies reveal that they are composed of an inner amorphous core surrounded by collagen and fibrin (1).

In early reports it was related with tuberculosis (Tb) arthritis (2), but later it was reported that it could also develop in rheumatoid arthritis (RA), septic arthritis, juvenile rheumatoid arthritis (JRA), osteoarthritis, hypocomplementemic arthritis and subacromial bursitis (3).

Majorly knee and shoulder joints are affected, frequently synovial originating and are found less frequently in periarticular bursa, tendon and ligament adherence points (4).

Case Report
Herein we report a female patient of 48 years old. Her complaints started about 8 months ago with pain at her both shoulders. Pains increased progressively. In the last 2 months she realized a swelling. Physical examination findings were swelling at both shoulders just under the coracoacromial line and right at the anterior of humerus head, about 7x7 cm size, soft, immobile and with unapparent margins. Shoulder movements were complete. She only described minimal pain at internal and external shoulder rotation movements. There was no pain, local erythema or temperature rise. Shoulder movements were completely open to all directions. At the direct radiographic evaluation, shoulder joint and peripheral soft tissues were normal. There was no finding related to Tb at the lungs. All biochemical and serologic examinations (for crystal arthropathy and rheumatologic examinations) were normal. MRI of the both shoulders was reported as “findings compatible with synovial chondromatosis at subacromial and subdeltoid bursae” (Figure-1a). Needle biopsy was applied that revealed non-specific inflammatory findings and afterwards the patient was operated.

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Written informed consent was obtained from the patient for publishing the individual medical records.

**Surgical Technique:** She was put into sunbed position under general anesthesia. Both of the shoulders were entered with deltoideo-pectoral incision. Mass has been reached passing through the layer sand were released by circumferential dissections. In the macroscopic evaluation, the masses were 7x7 cm in size, soft encapsulated and giving the impression of being full of liquid and reaching to the subacromial region in both of the shoulders. Both masses were removed totally. It was observed that the masses were deepen and reached to the joint capsule without adhesion. There was a lot of brilliant, pearly, rhiziform bodies with the size varying 2 to 8 mm inside the masses (Figure-1b). After homeostasis the layers were closed anatomically with 2/0 vicryl. On the postoperative 2nd day, active movements were allowed. On the 15th day sutures were removed and daily life activities were allowed. There was no relapse or recurrence in the MRI control after 24 months.

**Discussion**

RBs develop as a response to chronic synovial inflammation. Although it was identified with Tb arthritis at the beginning, later many articles were published indicating that they could be detected in many inflammatory cases such as, rheumatoid arthritis, septic arthritis, juvenile rheumatoid arthritis, osteoarthritis, hypocomplementemic arthritis and subacromial bursitis (1,3-5). In 1993 Stein et al. (6) defined MRI findings of RBs at subacromial bursa. Size of RBs can be changing, in the literature it has been stated that 55% of them is 2-7 mm, 35% of them is smaller than 2 mm and about 10% of them is bigger than 7 mm (7). In our case, they were between 2-8 mm (mostly 4-5 mm) which varied in form and sizes. Discussion about the etiopathology still continues. According one of the theories, in the later stages of RA number of increase and hypertrophy in synovial villi occur. In these villi fibrins pile up then they stretch and break away (5). According to Popert et al. (7), the reason of the RBs formation is the fibronectin and fibrin sediments that formed with the effect of glycosaminoglycan which is secreted by the abraded cartilage. Changes in the histological structure of RBs are in relation with their maturation degrees. Finally McCarty DJ and Cheung (8) argued that as a result of microcirculation failure at synovium and as a result of hypoxia related to these synovial micro infarctions the infarcted fragments fall into joint cavity and were covered with fibrin rings. In pathological examinations it was observed that some of these bodies are of completely fibrin structure and some have a collagen nucleus and covered with a fibrin coat. It was understood that this nucleus is composed of collagen type I (40%) and type V (20%). This composition is the same of the rheumatoid synovial membrane configuration. From this viewpoint, it was assumed that these bodies are formed from infarcted synovial tissue or synovial membrane origin collagen being covered with fibrin (1,4,8). Although RBs is almost always of synovial origin, its clinical meaning has almost always remained ambiguous (3). It can sometimes be seen as a reason for subacromial bursitis (1), sometimes as a reason for synovial cyst at intrapelvic region (9) and sometimes as reason for synovial hypertrophy at knee and painless effusion (4). No underlying pathology was reported with these patients. On the other hand, it was related with Tb, RA, JRA, septic arthritis, hypocomplementemic arthritis and atypical mycobacterial tenosynovitis and bursitis (2,3,10). RBs are not special to any one single disease and as in the present case it can be seen in the cases where there is no underlying pathology. Mostly RBs are confused with synovial chondromatosis at shoulder joint. Synovial chondromatosis frequently affects one joint, more often at 3-5th decants and ratio between male and female is 2 to 1. In the conventional graphic classifications are examined. It generally affects big joints such as hip and knee (5). RBs are distinguished from chondromatosis with no calcifications in the preoperative conventional graphs, a great number of free bodies in the joint cavity and bladder at MRI and histologically having cartilaginous tissue (3,4). In many centers radiologists may have difficulty in making diagnosis since they are not used to RBs. As a matter of fact, RBs at both of the shoulders of our case was diagnosed as synovial chondromatosis. Another disease which is confused with RBs is pigmented villonodular synovitis (PVNS). PVNS is also often monoarticular and affects big joints like synovial chondromatosis. At MRI T2 based imaging low signal areas with viewing hemosiderin deposits and intraoperative findings are distinguished from PVNS (5). Conventional graphs are not useful enough in diagnosing the disease, it can only give soft tissue swelling views (4,8). Especially, USG also has the property of diagnosing at bigger RB (8). However, the
most effective diagnosing device in the imaging methods is MRI (3-6). At T1 and T2 weighted images hypo- and isointense nodules seen in muscular tissues lead to RB diagnosis (5,6,10). In addition to this MRI is important in the aspect of diagnosing free bodies, and distinguishing from tumoral structures and showing the size of captured joint (1). When this pathology is ascertained, the patient should be examined in terms of rheumatology, Tb and hypocomplementemic arthritis. Sedimentation rate, rheumatoid factor, complement series and antinuclear antibody should be detected. Resection of bursa or synovia, bodies being taken out reduces the complaints but never ends the real pathology. If etiology was unveiled, one or two years after the operation orthopedic examinations together with rheumatologic follow-up should be done (1,4,8). In 2-year follow-up, our patient had no complaints and had no signs of rheumatological symptoms.

References