

Received: 2012.07.06 Accepted: 2012.09.04	Shoulder joint tuberculosis
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	Summary
Background:	Despite the fact that joint tuberculosis is one of the most common forms of extrapulmonary tuberculosis, it is a disease entity that is very rare in Poland (less than 100 cases a year in the last 10 years). The symptoms are non-specific, and thus the disease is rarely taken into account in preliminary differential diagnosis.
Case Report:	A 68-year-old female patient was admitted to the Internal Diseases Clinic due to oedema and pain of the right shoulder joint. The pain has been increasing for about 8 months. Physical examination revealed increased circumference and elevated temperature of the right shoulder joint. Limb function was retained. The full range of radiological and laboratory diagnostic examinations was performed, including the biopsy of the affected tissue which revealed the presence of <i>Mycobacterium tuberculosis</i> in the bacterial culture. Clinical improvement was obtained after introduction of TB drugs.
Conclusions:	Radiological diagnostic methods (X-ray, CT scans, MRI scans) provide high precision monitoring of articular lesions. However, the decisive diagnosis requires additional laboratory tests as well as histopathological and bacteriological assays.
Key words:	tuberculosis • joint
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Background

Bones and joints are the fourth most common location of extrapulmonary tuberculosis in Poland. Mycobacteria infection of the joints lads to skeletal inflammation and cessation of necrotic lesions. The most common locations are the vertebral bodies and periarticular parts of long bones. In the initial period, the process is non-characteristic. It develops mostly in immunocompromised and paediatric patients [1,2].

Case Report

A 68-year-old female patient was admitted to the Internal Diseases Clinic due to increasing pain and oedema of the right shoulder joint. First symptoms occured 8 months before admission to the hospital. About 5 months earlier, the patient had suffered a trauma resulting in restricted mobility of limb and intense pain (no medical documentation available). For about two months, the patient has observed increasing oedema around the shoulder joint. In addition, the patient reported loss of ca. 6 kg of body weight within the last six months.

Medical history included hypertension and primary biliary cirrhosis (for 15 years). The patient was treated with ursodeoxycholic acid, propanolol and spironolactone. About 30 years back, the patient had been diagnosed with non-otherwise specified seronegative arthritis, treated with steroids. The patient discontinued the treatment without recurrence of pain (no medical documentation available).

On admission, the patient presented with pain upon the movements of the right upper limb with arm function being retained. Contracture and slight pain in the left ulnar joint. Physical examination revealed skin discolouration with several white spots typical for leukopathy, increased circumference, redness and elevated temperature of the right shoulder joint with slight crackling and both lung bases. The body temperature was 36.6°C, the heart rate was 60 bpm, and the blood pressure was 140/70 mmHg.

Case Report







Figure 2. Lytic lesions of the humeral bone head with bone fragmentation and relocation of bone fragments. Distension of the proximal part of the humeral bone trunk with cortical thinning. Destruction of joint acetabulum with cortex continuity rupture.

Laboratory tests revealed the following values: leukocytes 4.2×10^9 /L ($4.5-10 \times 10^9$ /L), haemoglobin 10.0 g/dL (11.0-15.6 g/dL), MCV 88.2 fL (81.0-94.0 fL), platelets 165×10^9 /L ($150-400 \times 10^9$ /L), AST 78 U/L (5-31 U/L), ALT 47 U/L (5-31 U/L), alkaline phosphatase 254 U/L (35-104 U/L), gamma-glutamyl transferase 56 U/L (5-36 U/L), CRP 27.0 mg/L (<5 mg/L). ESR 74 mm/h. Other chemistry panel parameters, including the levels of creatinine, urea, nitrogen and electrolytes within normal limits.

In order to visualize the pathological processes taking place in the right shoulder joint and the left ulnar joint, radiograms of both joints were taken in anteroposterior and lateral projections (Figure 1). Extensive bone destruction was demonstrated in both the shoulder (Figure 2) and the ulnar joint radiograms. The figure presents lytic lesions of the humeral bone head with bone fragmentation and relocation of bone fragments, distension of the proximal part



Figure 3. Bone destruction, synovial hypertrophy with formation of numerous fluid reservoirs.



Figure 4. Humeral bone head destruction.

of the humeral bone trunk with cortical thinning, and destruction of joint acetabulum with cortex continuity rupture. Radiogram of the left ulnar joint revealed lesions similar in nature to these observed in the right shoulder

Figure 5. Synovial hypertrophy, presence of fluid reservoirs, extensive destruction of muscles, stabilizing tendons and capsule of the shoulder joint with humeral bone marrow infiltration.

joint. The lesions included a radial bone neck fracture with dislocation of bone head in the cubitoradial joint. The $% \left({{{\bf{x}}_{i}}} \right)$

periarticular and intra-articular soft tissue was inlaid with numerous spots of high-attenuated substance.



Case Report



CT scan (Figures 3, 4) confirmed massive destruction of the bony elements of the joint. In addition, significant synovial hypertrophy was observed with formation of numerous fluid reservoirs. The largest reservoir (sized $100 \times 70 \times 60$ mm), of heterogeneous density, was located under the right deltoid muscle, penetrating into the shoulder joint and filling the bed left by the degenerated head of the humeral bone. The reservoir extended under the pectoral muscle.

In order to determine the nature of lesions in the soft tissues surrounding the right shoulder joint, MRI scans were performed with and without contrast administration in three planed using T1-wieghted FSE and PD FATSAT sequences (Figures 5, 6).



Figure 6. Enhancement of hypertrophic synovium and edge enhancement of fluid reservoirs after intravenous contrast administration. Lymph nodes in axillary fossa.

The MRI revealed destructive skeletal lesions, synovial hypertrophy, presence of fluid reservoirs as reported by previous tests, as well as extensive destruction of muscles, stabilizing tendons and capsule of the shoulder joint with humeral bone marrow infiltration. Enhancement of hypertrophic synovium and edge enhancement of fluid reservoirs were observed after intravenous contrast administration.

Lymph nodes $(21 \times 14 \text{ mm})$ of intensity similar to that of the tissue infiltrating the humeral bone trunk were visualized in the axillary fossa.

The decisive diagnosis was made on the basis of bacterial culture (bioptate was collected from one of the fluid reservoirs) revealing the presence of *Mycobacterium tuberculosis*.

Antibiogram-based treatment was initiated, including: rifampicin (RMP), isoniazid (INH), and pyrazinamide (PZA). Reduction in the circumference of the left shoulder joint was observed after 12 months. Limb mobility remained restricted.

Discussion

Based on the performed studies, the fractures of the right humeral bone and the left radial bone were found to be pathological fractures. Considering the radiological image (bone destruction, synovial hypertrophy, inlaid soft tissue) and the dynamics of lesion development (a relatively fast progression of the disease), as well as the general condition of the patient, the following differential diagnosis was proposed: chondrocalcinosis (Milwaukee shoulder) [3,4], proliferative diseases [5] (synovioma, sarcoma).

Due to the extremely rare prevalence of lesions caused by *Mycobacterium tuberculosis* characterized by isolated affection of joints only (chest X-ray revealed no lesions typical for tuberculosis), joint tuberculosis was not taken into account in the differential diagnosis [6].

Conclusions

Radiological diagnostic methods (X-ray, CT scans, MRI scans) provide high precision and sensitivity monitoring of articular lesions. A differential diagnostic approach based

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on these methods may be proposed. However, the specificity of such approach is low, and therefore, correct diagnosis requires additional laboratory tests as well as histopathological and bacteriological assays.

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