

Tuberculosis: a rare cause of foot and ankle pain

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ABSTRACT

Skeletal tuberculosis is an uncommon infectious disease that occurs in 1-3 % of all tuberculosis cases. Apart from spine it can emerge in weight bearing joints. Tubercular involvement of the ankle is the most uncommon and infrequent amongst those of all joints. If imaging techniques and laboratory results are not sufficient while setting a precise diagnosis, this rare problem should be always taken into consideration just like other infections of the musculoskeletal system. In this paper we present a case of 83-year-old female patient who underwent conservative treatment for ankle tuberculosis.

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Introduction

Tuberculosis is an infectious disease caused by the bacteria mycobacterium tuberculosis. It commonly occurs in lungs but can occasionally infect any part of the body. Bones and joints are involved in 1-3% of all tuberculosis cases and in 15% of extra-pulmonary tuberculosis cases [1]. According to dispersion and frequency range, bone and joints tuberculosis mainly infects the vertebra (50%), whereas occurrences in hips, knees, foot-ankle area (10%) are seldom observed [2-4]. The present paper describes a case of ankle and ankle area osteoarticular tuberculosis.

Case Presentation

An 83 years old woman presented with pain in her left foot and ankle. The pain in the foot area had been lasting for about 1 year and along with sensitiveness

it had been gradually intensifying for last months. The patient had not sustained any traumas. Apart from hypertension, she had not suffered from any systemic disease.

Examination findings included; short distance antalgic gait (as weight was not placed to the left lower extremity) and swelling in foot and ankle. By palpation, pain in areas near the ankle (especially in the distal crural region) was detected. Neurovascular examination seemed to be normal. Ankle movements seemed to be limited in all directions. (flexion-dorsiflexion 20-0-10, supination pronation 5-0-5).

The patient's inflammatory markers were normal as WBC = 6.03 mm³/ml (normal = 3.5-10.5), lymphocyte ratio = 22.2% (normal = 20-51.1), neutrophil ratio = 4.14 (normal = 1.7 -7), C-reactive protein (CRP) = 3.3 mg/l (normal = 0 - 5), erythrocyte sedimentation rate (ESR) = 47 mm/h (normal = 0- 20).

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Conducted Brucella Rose-Bengal test showed negative result.

Radiographs show severe osteopenia in anterior-posterior foot and ankle, significant sclerosis in subtalar joint, extensive osteoporosis and a bone cyst in talus and calcaneus (Figure 1).

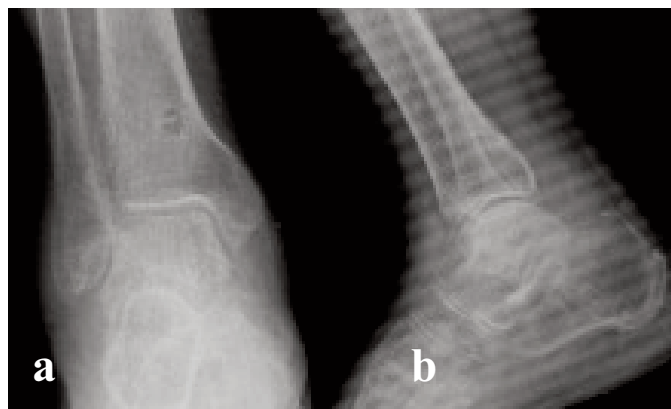


Figure 1. The graphy of anterior, posterior (a) and lateral ankle. Extensive osteopenia in bones, trabecular coarsening, narrowing in tibiotalar and talocalcaneal joint spaces, along with sclerosis (a, b). Radiolucent lesion, concordant with calcaneus sclerotic contoured cyst (b)

CT scan of left foot and ankle showed osteoporosis in the left foot and extensive radiolucency along with trabecular coarsening associated with fatty bone marrow degeneration. Moreover, in the central part of calcaneus a cystic cavity approximately 21 mm in diameter was detected. Inside of the cavity a 15 mm-sized hyperdense area was seen (Figure 2).

The magnetic resonance imaging show that minimal increasing signal intensity in the distal tibia,

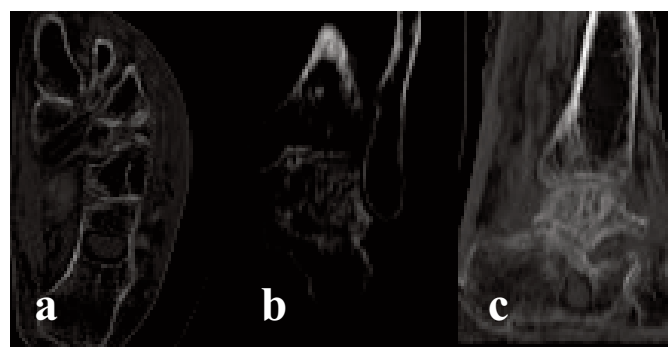


Figure 2. Ankle ct scan axial (a), coronal (b) and saggital (c) incisions: axial ct incision (a), coronal and sagittal ct reformat- ted images (b, c): generalized increased bone density, trabecular coarsening, along with bone thinning and disorders without peri- ost reaction in cortex. Sclerotic contoured cyst in 2 cm in dia- meter in calcaneus.

talus and calcaneus surfaces; sporadically developing cortical erosions; narrowing in talocalcaneal joint spaces which emerged along of existing inflammation; and cortical destruction-associated collapse (Figure 3).

Due to manifested symptoms the patient was thought to have developed osteomyelitis as a pre- diagnosis. Therefore an open biopsy had been planned. During the open biopsy samples were taken from talus, calcaneus and subtalar joints for culture and pathology examination. The culture examination revealed no growth. The histology showed granulomatous inflammation with epitheloid granuloma including multinucleate gaint cell and peripheral rim of lymphoid cells (Figure 4).

After histopathologic examination the diagnosis of ankle osteoarticular tuberculosis was given and anti-tuberculosis multi-modal therapy administered. For the first 2 months the patient had been prescribed rifampicin 300 mg/day oral, isoniazid

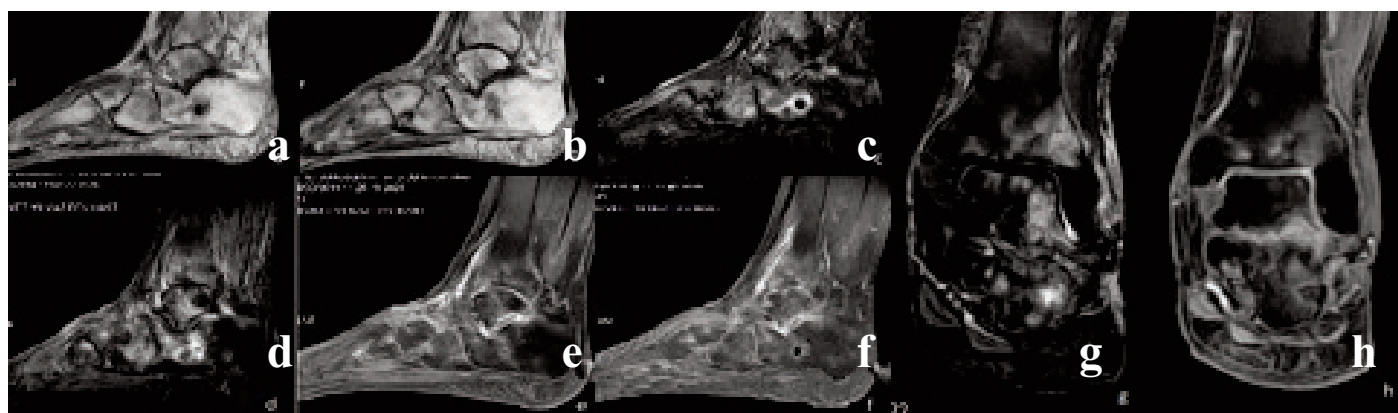


Figure 3. Magnetic resonance imaging of the ankle: axial – coronal – sagittal incisions. Sagittal t1 (a), stir (b), contrasted fs t1 (c), coronal stir (g) and contrasted fs t1 (h): hypointense in the distal tibia, the talus, the calcaneus and in the tarsal bones t1; hyperintense in stir; contrast retaining heterogeneous signal records in contrasted fs t1; bone disorders and erosions (osteomyelitis) in cortical bone. Contrast retention and thickening (synovitis, arthritis) in the ankle area and soft tissue. Hypotense in calcaneus t1, hypertense in stir, wall contrast retaining hypotense cystic lesion (necrotic cyst?) In contrasted fs t1.

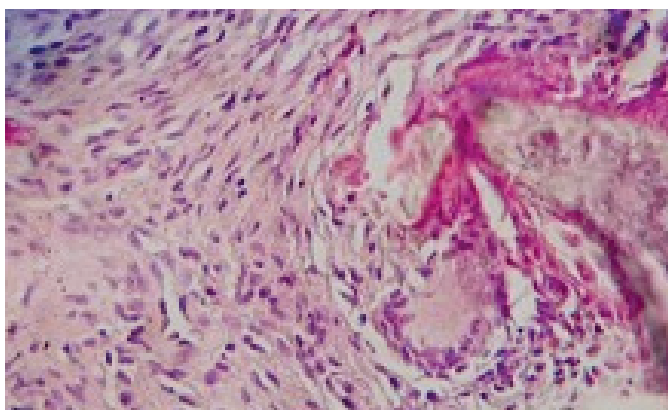


Figure 4. Granulomatous inflammation with epithelioid granuloma including multinucleated giant cell and peripheral rim of lymphoid cells

300 mg/day oral, pyrazinamide 500 mg/day oral, ethambutol 500 mg/day oral (ripe). Two months later, the treatment with ethambutol and pyrazinamide was discontinued; only isoniazid and rifampicin were administered for the following next 10 months. Thus, medication lasted 12 months. Three months after the anti-tuberculosis treatment started a regression of symptoms had been noticed. The patient's follow-up period was performed 6 months after completion of treatment. The patient reported that sensitiveness and ankle swelling had disappeared, nor did weight bearing still cause difficulties, and therefore she had been able to walk painlessly ever since.

Discussion

As skeletal tuberculosis occurs on rare occasions only, it is seldom considered for pre-diagnoses given by orthopaedic surgeons. Consequently, sometimes it can be difficult to make a precise diagnosis. The missing diagnosis and consequently inadequate treatment can cause increase of inflammation and progressing bone and joint degeneration.

Skeletal tuberculosis generally disseminates via haematogenous spread from a primary focus to bone areas where more blood components are formed. After that it can leap to joints by nearby ways [5]. Less frequently the disease can spread through lymphatic system [6]. Besides these, direct post-traumatic infections are reported to emerge in any bone or joint [7-8]. It is related to increasing vascularization or decreasing local resistance and connected to forming new foci which will cause reactivation of infection in post-traumatic area [9-10].

Generally, the progression of skeletal tuberculosis is slow and insidious. Classical symptoms of pulmonary tuberculosis such as fever, night sweats and weight loss might not occur in the case of developing skeletal tuberculosis. Typically, symptoms such as swelling, sensitiveness, decreased range of joint motions, muscle spasms, malformations can be seen here.

In laboratory tests high levels of such parameters as WBC, CRP and sedimentation, which seem to become normal, prevent from giving the precise diagnosis. Normal levels of WBC, CRP and sedimentation complicate diagnosing as it draws orthopaedists away from infection diagnoses. However, in cases where radiological findings along with clinical findings substantiate chronic infections the possibility of emergence of bone and joint tuberculosis should be taken into consideration.

Informed Consent

Written informed consent was obtained from the patient for the publication of this case report.

Conflict of interest

The authors declared that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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