

Mycobacterium avium intracellulare complex causing olecranon bursitis and prosthetic joint infection in an immunocompromised host



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ARTICLE INFO

Article history:

Received 30 June 2015

Revised 10 November 2015

Accepted 25 November 2015

ABSTRACT

Case: A 73-year-old immunocompromised male presented with recurrent left elbow swelling due to *Mycobacterium avium intracellulare* complex (MAC) olecranon bursitis. 3 years after completing MAC treatment, he underwent right total knee arthroplasty (TKA). 1 year later, he developed TKA pain and swelling and was diagnosed with MAC prosthetic joint infection (PJI). He underwent TKA resection, reimplantation, and 12 months of anti-MAC therapy. This patient is the seventh case report of MAC olecranon bursitis and the third case report of MAC PJI. He is the only report of both MAC olecranon bursitis and PJI occurring in the same patient.

Informed consent: This patient was informed and agreed to the publication of this material.

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Introduction

Nontuberculous mycobacteria (NTM) comprise over 125 species and are ubiquitous in soil, water, and animals. *Mycobacterium avium intracellulare* complex (MAC) is the most common pathogenic NTM species and consists of *M. avium* and *M. intracellulare*, which are indistinguishable based on traditional laboratory testing. MAC usually causes pulmonary disease but may also cause lymphatic, skin/soft tissue, skeletal, or disseminated disease. Water sources, such as recirculating hot-water systems, are the reservoir for most MAC infections.[1] We illustrate a rare case of MAC causing both olecranon bursitis and prosthetic joint infection (PJI) in an immunocompromised host.

Case report

A 73-year-old male with history of multiple myeloma (in remission for 3 years after thalidomide and dexamethasone treatment) and chronic cough, presented to the Emergency Department with 3 days of left elbow swelling, which was diagnosed as olecranon bursitis

based on clinical presentation and X-ray (Fig. 1). There was no history of elbow trauma. His bursitis improved with a steroid injection but recurred 2 months later and was treated with a repeat steroid injection. Unfortunately, his bursitis recurred again 2 months later, and bursa aspirate yielded a white blood cell (WBC) count of 45,708 with 98% neutrophils, suspicious for septic bursitis. Bacterial cultures were negative, and he had not been on antibiotics previously. He received a 14-day course of cephalexin.

Because the elbow was still edematous after 8 days of cephalexin, he underwent elbow debridement, which revealed purulent fluid with erythematous grayish-brown tissue. Histology showed acutely inflamed synovium consistent with infection. 2 out of 3 samples were smear-positive for acid-fast bacilli. All 3 operative mycobacterial cultures and the initial aspiration grew MAC.

On review of systems, he mentioned a chronic productive cough. The patient smoked a pipe for 10 years but quit 65 years ago. He had no formal diagnosis of COPD. Sputum cultures grew MAC. A chest X-ray showed basal atelectasis and multiple bilateral calcified pleural plaques, consistent with prior asbestos exposure (Fig. 2). There was no significant change compared to a chest X-ray done 3 years prior to presentation, and tuberculosis skin testing was negative. Other exposures included gardening, hot-tub use, and a pet dog.

The MAC isolate was susceptible to rifabutin, ethambutol, and clarithromycin; intermediate to rifampin, streptomycin, and moxifloxacin; and resistant to ciprofloxacin, kanamycin, cycloserine, ethionamide, and amikacin. His initial treatment regimen included clarithromycin 500 mg PO BID, ethambutol 1600 mg PO daily, and

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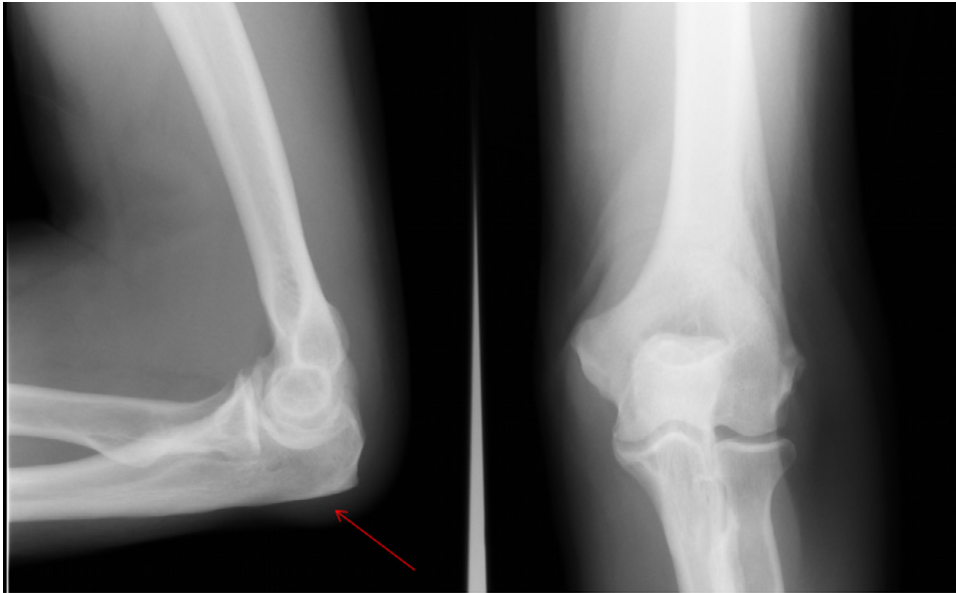


Fig. 1. Left elbow X-ray (left panel: lateral view, right panel: anteroposterior view) shows soft tissue swelling (red arrow) overlying the extensor surface of the olecranon, consistent with olecranon bursitis. There is also a tiny olecranon spur and minimal bone spurring at both humeral epicondyles. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).



Fig. 2. Anteroposterior view of chest X-ray shows basal atelectasis and multiple bilateral calcified pleural plaques (red arrow) consistent with prior asbestos exposure. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

rifabutin 300 mg once daily. The rifabutin was discontinued 3 weeks into therapy due to neutropenia and transaminitis. His bursitis resolved after a 12-month course of ethambutol and clarithromycin. Repeat sputum cultures were not obtained after completion of therapy.

One year later, his multiple myeloma recurred, and he was started on lenalidomide and dexamethasone. Around this time, he was also diagnosed with seronegative rheumatoid arthritis (RA) and initiated methotrexate. 3 years after completing his MAC treatment, he un-

derwent an elective right total knee arthroplasty (TKA) for degenerative joint disease. This procedure was performed at an outside facility with presumed peri-operative prophylaxis. One year later, he developed right TKA pain, instability, and swelling. X-ray showed a well-seated right TKA with a large effusion (Fig. 3). C-reactive protein (CRP) was elevated at 13.5 mg/L (reference range < 8 mg/L), and erythrocyte sedimentation rate (ESR) was elevated at 65 mm/h. Synovial fluid aspiration yielded 4524 total nucleated cells; 57% neutrophils,

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