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Short communication

Case of tuberculosis of the temporomandibular joint

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Abstract

Tuberculosis (TB) of the temporomandibular joint (TMJ) is rare and misdiagnosis is common. We describe an unusual case of the disease in a 27-year-old Zimbabwean woman.

Keywords: Tuberculosis; Temporomandibular joint

Introduction

Tuberculosis (TB) is an infectious bacterial disease usually caused by *Mycobacterium tuberculosis* and less commonly by *Mycobacterium bovis*.[1] Roughly a third of the world's population has been infected with *M. tuberculosis*, and new infections occur at a rate of one/second,[2] but not all of them cause symptomatic disease. The primary infection is usually pulmonary although it may also occur in bones, joints, and the central nervous system. The increased incidence of extrapulmonary TB in the last few years is thought to be related to the AIDS epidemic.[3] TB of the head and neck forms nearly 10% of all extrapulmonary manifestations of the disease. Involvement of the facial bones has been described and we know of only six case reports of primary TB infections of the temporomandibular joint (TMJ).[4]

The incidence of tuberculosis in 2009 was reported by the World Health Organization as 14.9/100 000 of the UK population, a 5.5% increase from 2008. Seventy-three percent of cases were in people born outside the UK, most were from South Asia (55%) and sub-Saharan Africa (30%).[5]

Case report

A 27-year-old Zimbabwean woman presented to the accident and emergency department with a two-week history of increasingly severe trismus and a constant dull pain over the left TMJ. Her medical history included mild asthma, long-standing left talar arthropathy, and an allergy to penicillin.

Clinical examination showed left-sided facial asymmetry with trismus of 10 mm, and left masseteric tenderness. Blood tests showed raised C-reactive protein (CRP) of 27 mg/L and a normal full blood count.

Dental panoramic tomography showed periapical radiolucency associated with the mesial root of the lower left wisdom tooth (Fig. 1).

Differential diagnosis included acute pericoronitis associated with the lower left wisdom tooth with dysfunction of the TMJ and associated trismus. The left wisdom tooth was removed and she was treated with a five-day course of erythromycin and metronidazole.

Two weeks after the extraction she presented again with worsening pain, pyrexia, and increased swelling. Serum blood tests showed an increased CRP of 38 mg/L. Mumps serology was negative. Magnetic resonance imaging (Fig. 2) showed abnormal soft tissue extending from the left TMJ anteroinferiorly, and inflammation of the masseter with apparent fluid collection caudal to the left

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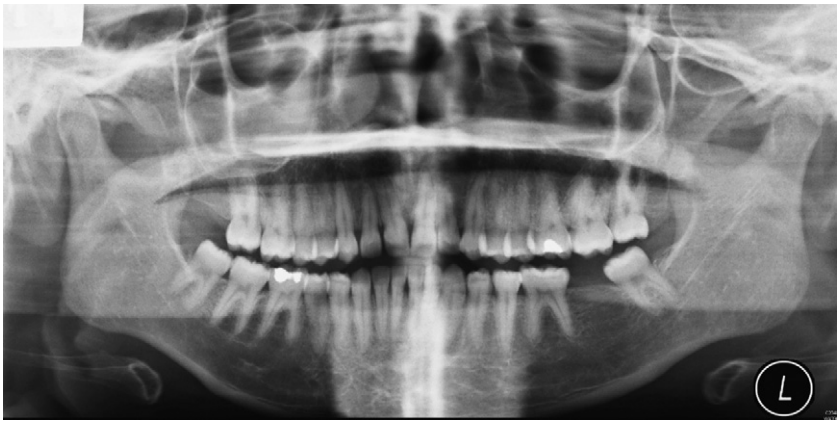

Fig. 1. Dental panoramic tomogram on initial presentation.

TMJ. There was loss of cortex of condylar bone extending into the left mandibular ramus. She was admitted for intravenous antibiotics for a presumed odontogenic infection.

Diagnostic ultrasound-guided aspiration of the fluid collection was done, and cytological examination showed scattered polymorphonuclear leucocytes and histiocytes within a blood clot; no malignant cells were seen and culture showed no growth. A revised differential diagnosis included inflammatory or infective arthropathy, myositis or muscle disease with secondary involvement of the bone, unusual lymphoma, TB, or human immunodeficiency virus (HIV). A subsequent HIV test was negative and bone scans showed increased bony metabolism in the left mandibular region and the T12 region of the spine, which suggested metastatic or other focal bony disease (Fig. 3).

Open biopsy of clinically abnormal precapsular tissue by a minimal preauricular approach was done in March 2009. Further microbiological samples grew fully sensitive *M. tuberculosis* after eight weeks.

She started antituberculous therapy, which included rifampicin, isoniazid, pyrazinamide, and ethambutol for eight weeks. She improved, and mouth opening increased to 28 mm three months after treatment began.

One year after diagnosis and treatment she started to experience severe pain and limitation in translatory movement of the TMJ. Repeated imaging showed features in keeping with sequelae of the previous tuberculosis (Fig. 4), which included destruction of the mandibular condyle with complete loss of the convexity of the condyle and dystrophic calcification with ankylosis. There was no evidence of reactivation or residual collections.

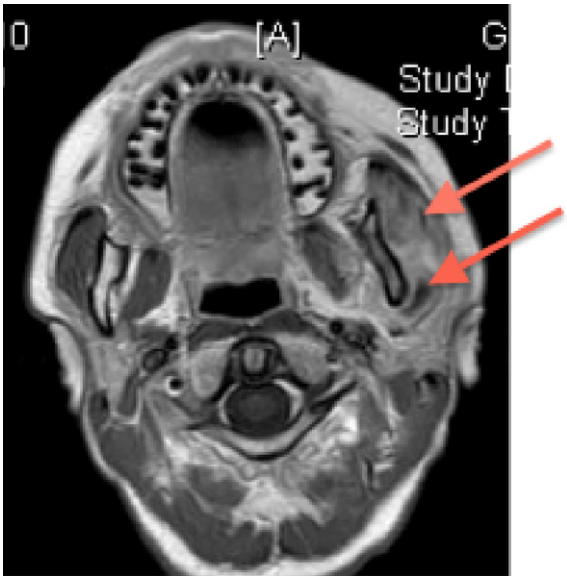

Fig. 2. Magnetic resonance image showing inflammation of the masseter (top arrow) and fluid collection caudal to the left TMJ (bottom arrow).

Fig. 3. Bone scan showing increased uptake of the left TMJ extending to the left ramus of the mandible.

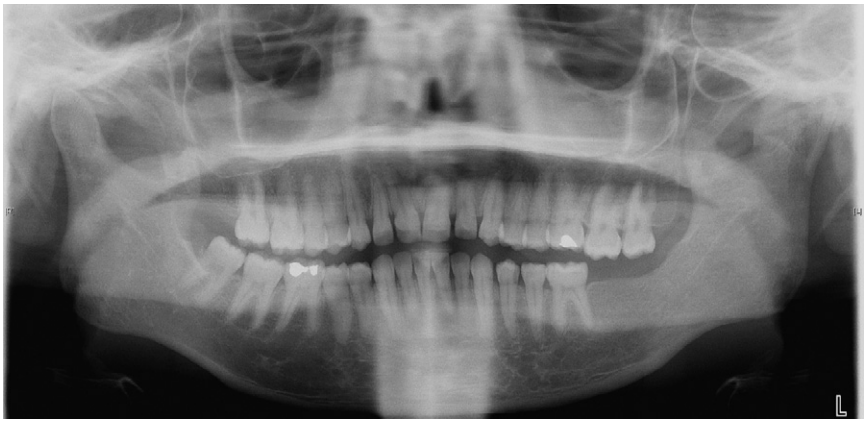

Fig. 4. Dental panoramic tomogram showing enlargement of the left condyle and flattening of the condylar head.

A successful prosthetic joint replacement improved the function of the left TMJ.

Discussion

TB is a multiorgan caseating granulomatous disease caused by the acid-fast bacilli *M. tuberculosis* and rarely *M. bovis*.[1] TB of the bone has been reported commonly in the long bones and the spine. Most of the previously described cases in the TMJ have been in women (mean age 33.7 years, median 30, range 22–59). All the cases affected the left side with similar presenting features of swelling, pain, trismus, pyrexia, and preauricular swelling, and imaging showed bony destruction of the TMJ and necrotic bone. To our knowledge this is the only documented case where another focus (in the spine) was identified.[4]

TB of the TMJ is easily misdiagnosed. In patients from endemic areas, or those at risk of HIV, it should be considered as a differential diagnosis in those with acute swelling of the TMJ and bony destruction. Long term combination antituberculous therapy must be started swiftly to reduce bony destruction. We hope this report highlights the importance of considering TB of the TMJ as a possible diagnosis in unusual presentations of preauricular swelling and trismus.

Conflict of interest

No conflict of interest.

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