

Tubercular osteomyelitis of the mandible— A case report

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Abstract

Tuberculosis of jaw is an extremely rare condition, particularly in comparison to pulmonary tuberculosis. It is a diagnostic enigma, as the characteristic signs and symptoms of this disease may be absent, or sometimes radiographically can be mistaken for more common jaw lesion such as odontogenic cyst and tumors leading to therapeutic delay. Hereby, we report a case of mandibular swelling in 17 years old female which on incisional biopsy came out to be tuberculous osteomyelitis. Patient was referred to higher centre where anti-tubercular treatment (ATT) was started.

Keywords: Tuberculosis; Mandible; Osteomyelitis.

Introduction

Tuberculosis is a chronic granulomatous infectious disease caused, in humans, by *Mycobacterium tuberculosis* and less frequently by *Mycobacterium bovis*.⁽¹⁾ Tuberculosis of bone forms about 10% of extra pulmonary tuberculosis, of which 50% occur in the spine.⁽²⁾ Tuberculous osteomyelitis of mandible is quite rare and constitutes less than 2% of skeletal tuberculosis.^(3,4) Clinically it may be confused with acute inflammatory conditions of jaw, non-specific tuberculosis, odontogenic cyst, odontogenic tumor and actinomycosis. Herewith, we report a case of a 17-year-old female who presented with a swelling over the left mandible since two months which was diagnosed as to be tuberculous osteomyelitis and responded well to anti-tubercular treatment (ATT).

Case Report

A 17 year old, female patient reported to the Department of Oral Pathology & Microbiology at our institute with the chief complaint of swelling on lower left side of face since last 2 months. Extraorally solitary, diffuse, tender swelling seen on left angle region of the mandible with no draining sinus. (Fig. 1)



Fig. 1: Solitary, diffuse, tender swelling on left angle region of the mandible with no draining sinus.

The patient was prescribed conventional antibiotics but no improvement was noticed. Past dental history revealed extraction of grossly carious 36, nine months back. Past medical history revealed history of tuberculosis and to anti-tubercular treatment (ATT) for the same. Medical history also revealed hypocalcaemia, vitamin-D deficiency and under calcium medication. Family history & habit history were non-contributory. Her blood investigation revealed haemoglobin level of 11.7 gm%, calcium level of 6.4 mg/dl & vitamin D level of 9 ng/ml.

The digital panoramic radiograph showed irregular radiolucency with loss of normal trabecular pattern at the angle of the mandibular region. For better evaluation of the lesion, CBCT was done. CBCT showed irregular hypodense area at the angle of the left mandible. Thinning and perforation of the both lingual and buccal cortical plates were also noted. Some areas of diffuse bony sclerosis were also present apical to the extraction socket of mandibular first molar. A thin irregular hyperdense line was also seen lingual to lingual cortical plate suggestive of periosteal reaction. (Fig. 2a & 2b)

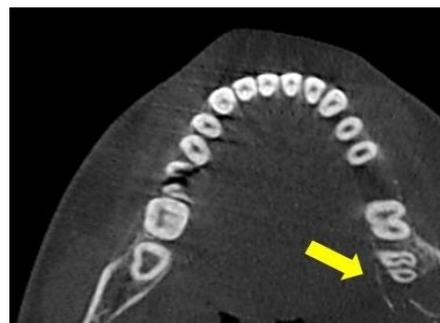


Fig. 2: a. Hypodense area at the angle of the left mandible with lingual cortical plate showing periosteal reaction (yellow arrow).



Fig. 2: b. Thinning and perforation of the both lingual and buccal cortical plate.

Incisional biopsy was undertaken, which on haematoxylin and eosin stained section showed chronic granulomatous lesion consisting of epithelioid cells, lymphocytes, necrotic areas and sequestrum (inset picture). Areas of caseous necrosis and langhans giant cells were also seen. (Fig. 3 a & b)

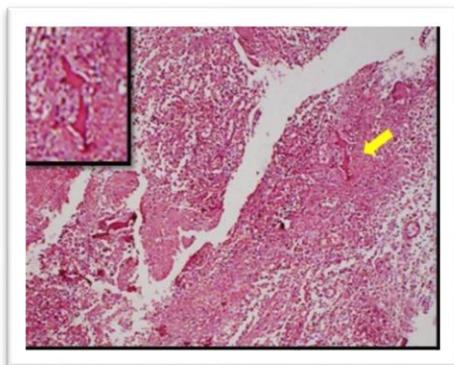


Fig. 3: a. Chronic granulomatous lesion consisting of epithelioid cells, lymphocytes and necrotic areas sequestrum also noted (inset picture) H & E X40.

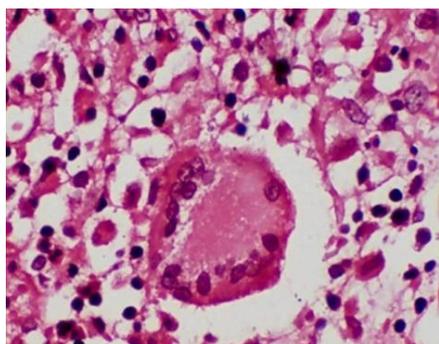


Fig. 3: b. Langhan's giant cell, epithelioid cells & chronic inflammatory cells H & E X400.

Decalcified section of bone showed irregular osseous surface, howship lacunae and chronic inflammatory cell infiltrates suggestive of resorbing bone. (Fig. 3c) Overall impression was consistent with tubercular osteomyelitis. Patient was referred to higher center, where she was put on antitubercular therapy and the swelling exhibited a marked reduction in size. Patient did not come for further follow up.

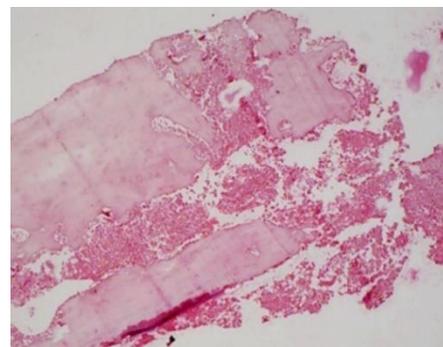


Fig. 3: c. Resorbing osseous tissue with chronic inflammatory cell infiltrates H & E X40.

Discussion

Primary tuberculosis of oral cavity is rare as compared to secondary. Most lesions are secondary to pulmonary tuberculosis. Primary lesions develop when tuberculosis bacilli are directly inoculated into the oral tissues of a person who has not acquired immunity to the disease. These frequently involve gingiva, tooth extraction sockets and buccal folds. Secondary tuberculosis of oral cavity most commonly involve tongue, followed by palate, lip, buccal mucosa. Tuberculous osteomyelitis of jaws is very rare. Most of the reported cases of mandibular tuberculosis are secondary and primary tuberculosis of the mandible is a rare occurrence.^(2,5,6) The involvement of the mandible by tuberculous infection is extremely rare as it contains less cancellous bone⁽⁷⁾ and the alveolar and angle regions have greater affinity.⁽⁸⁾ The infection may extend to the mandible by:

1. Direct transfer from infected sputum¹ or infected raw milk of cow⁶ through a) an open pulp in carious tooth b) an extraction wound or c) gingival margin or perforation of an erupting tooth
2. From direct extension from a tuberculous gingival lesion.
3. From infection within a periapical granuloma containing tuberculous granuloma
4. from haematogenous emboli^(8,9,10)

Significant number of cases of tuberculosis of jaw-bones occurred in children under the age of 16 years.⁽¹⁰⁾

Mandibular tuberculosis is often insidious in onset and may present weeks or months before symptoms arise.^(2,9) In a few cases, it appears as an acute inflammatory swelling which fails to resolve by the use of conventional antibiotics. Clinically it may confuse this with a pyogenic abscess and actinomycosis if sinus is present.⁽²⁾ In our case the swelling resemble clinically to acute inflammatory lesion and does not responded to conventional antibiotics.

Radiologically, tubercular osteomyelitis resembles non-specific osteomyelitis. Initially it begins as an area of rarefaction with trabecular blurring. As the disease progresses, the bone is destroyed, erosion of cortical bone occurs which is then replaced by soft granulation tissue and subsequently a sub-periosteal abscess formation takes place. Subsequently caseation necrosis

occur leading to liquefaction which may burst either intra-orally or outside either through single or multiple sinuses.^(2,8,9,10) it may also show a mixed radiolucent-radiopaque picture.⁽⁴⁾

In our case radiologically on OPG loss of normal trabecular pattern was seen while on CBCT lesion was osteolytic with irregular margins.

Histopathologically features of oral (jaws) tuberculosis is similar to those elsewhere in the body. Histopathologic picture shows chronic granulomatous lesion consisting of foci of caseous necrosis surrounded by epithelioid cells, lymphocytes and multinucleated langhans giant cells.^(2,5,6,9,10) Clinically tuberculosis of jaw bones can mimic dentoalveolar abscess, odontogenic cyst, tumor and actinomycosis (if sinus is present). Biopsy is indicated to rule out odontogenic cyst, tumor and actinomycosis of jaws.

Treatment of mandibular tuberculosis is with antitubercular drug therapy. The initial therapy include four conventional drugs in the form of rifampicin, isoniazid, pyrazinamide and ethambutol as an intensive regimen followed by rifampicin and isoniazid for a period of 9–12 months; however, WHO recommends a short course therapy of 6 months because of the paucibacillary nature of the disease.⁽²⁾

Conclusion

So tuberculous osteomyelitis must be consider in differential diagnosis of an inflammatory mandibular

swelling and osteomyelitis of the jaw which does not respond to conventional antibiotics.

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