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CASE REPORT

Traumatic Bone Cyst of the Jaws: A Report of Two Cases and Review

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ABSTRACT

Traumatic bone cyst (TBC) is an uncommon disorder of the jaw bones, as well as other skeletal bones, particularly the long bones. Traumatic bone cyst is an asymptomatic, slow growing lesion commonly diagnosed incidentally during routine radiographic examination of the jaw bones. Of the jaw bones, mandible is affected much more than the maxilla. The etiology is unclear and trauma cannot be definitely determined to be the cause. Surgical exploration of the cavity is curative in most cases and do not require any other treatment. We present two well documented, radiographically and histopathologically confirmed cases of TBC involving the jaw bones. Based on the literature available, the etiopathogenesis, treatment, associations and possible complications of this disorder have been discussed.

Keywords: Traumatic bone cyst, Idiopathic bone cyst, Mandible, Maxilla.

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INTRODUCTION

Traumatic bone cyst is a benign pseudo cystic cavity in bone that is either empty or contains fluid. In 1929, Lucas and Blum for the first time described traumatic bone cyst (TBC) as a separate disease entity.[1] TBC may be identified by a great variety of names in the literature and different causative factors or a combination of such factors have been proposed for its pathogenesis.[2,3] Most of the idiopathic bone cysts (IBC) are diagnosed incidentally in orthopantomogram (OPG) and most of the individuals affected are in teens or early 20s.[3] The body of the mandible (75%) is usually affected with a repeated predilection in the premolar and molar regions.[4]

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Pain is the presenting symptom in 10 to 30% of the patients.[3-5] Other, more unusual symptoms include tooth sensitivity, paresthesia, fistulas, delayed eruption of permanent teeth, displacement of the inferior dental canal and pathologic fracture of the mandible.[6-10] The WHO classification of Head and Neck Tumours, 2005 describes TBC as a non-neoplastic osseous lesion because it shows no epithelial lining, which differentiates this lesion from the true cysts of age.[11] Radiographically, it manifests as a well defined, unilocular, radiolucent area which occasionally presents a typical festooned pattern around the apices of the adjacent teeth.[12,13]

The definite diagnosis of traumatic cyst is invariably achieved at surgery when an empty bone cavity without epithelial lining is observed. Sometimes, the cavity contains a straw-colored fluid or bright blood.[3,4,14,15] Surgery is the management of choice, particularly as it also leads to confirmation of the diagnosis. Simple curettage of the bone walls is performed and it typically heals in 6 to 12 months. Clinical and radiological follow-up after surgery is thus indicated.[16] The present case reports describe the clinical and radiographic characteristics as well as the surgical and histopathological findings of TBC.

CASE REPORTS

Case 1

A 25-year-old male patient reported to the department of oral medicine and radiology with a chief complaint of pain in the right and left sides of lower jaw since 3 months, the pain was reported to be intermittent in nature. Medical history revealed an alleged history of domestic violence to the lower jaw 10 years back. Clinical examination revealed no evidence of lymphadenopathy, swelling or facial asymmetry. Intraoral examination did not show any soft tissue abnormality or bony expansion. Periodontium was noted to be healthy with no evidence of gingivitis, periodontal pockets or tooth mobility. There were no carious lesions or tooth displacement. There was no derangement of occlusion.

Vitality tests were done in relation to right mandibular premolars and molars, which were positive for vital teeth. Routine periapical radiograph revealed a well-defined radiolucency with scalloping in between the roots of mandibular second premolar and first molar. Panoramic

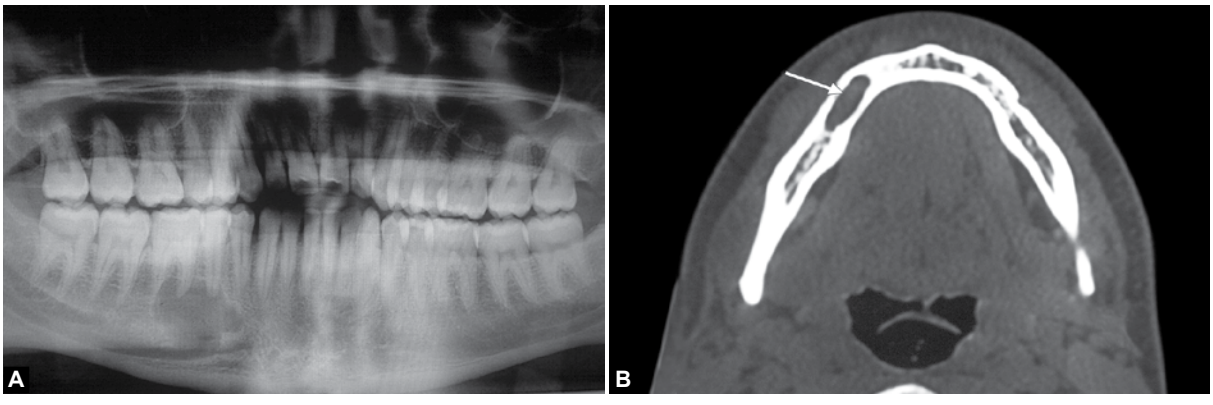
radiography confirmed the extension and size of the lesion which revealed a well-defined unilocular radiolucent area of size 3 × 2.5 cm extending from the distal aspect of the root of 44 to the distal aspect of the root of 46 and inferiorly extending till the mandibular canal without crossing it. Computerized tomography scan revealed unilateral involvement of right posterior mandible. Aspiration from the lesion was negative for any fluid. Based on these findings, the clinical and radiographic differential diagnoses of TBC, odontogenic keratocyst, odontogenic myxoma, central giant cell granuloma were considered (Figs 1A and B).

On surgical exploration, the lesion was found to be a bone cavity which was completely devoid of any type of fluid or visible soft tissue lining. Curettage of the cavity was performed. The removed pieces of bone with minimal amount of soft tissue were sent for histopathological examination which revealed dense connective tissue stroma with extravasated RBC's along with necrotic changes and dense chronic inflammatory infiltrate. There was no evidence of any cystic epithelium (Figs 2A and B). Periapical and panoramic follow-up radiographs of affected region at intervals of 6 months and 1 year from the surgical intervention showed signs of local bone repair (Fig. 3).

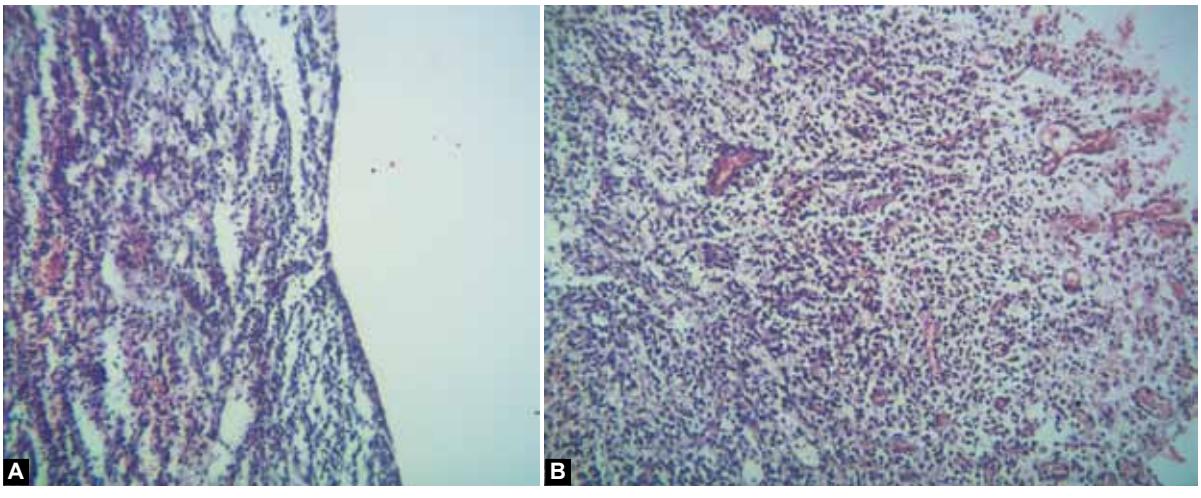
Case 2

A 50-year-old female patient reported to the department of oral medicine and radiology with a chief complaint of pain in the right and left lower jaw that began 1 week back. Dental history revealed a history of extraction 5 years back and root canal treatment 2 years back. Medical history was not contributory.

On intraoral examination, 36, 46, 48, 18, 15 and 16 were found to be missing. On inspection of alveolar mucosa a swelling of size 2 × 2 cm was seen on the right lingual aspect in relation to 47 and 48. On palpation, the swelling was found to be tender, soft and fluctuant with bilateral expansion of buccal and lingual cortical plates (Figs 4A and B). Vitality tests were positive for 44, 45, 47, 33, 34, 35, 37 and 38. On aspiration, blood tinged straw colored fluid was obtained from the lesion. Clinical

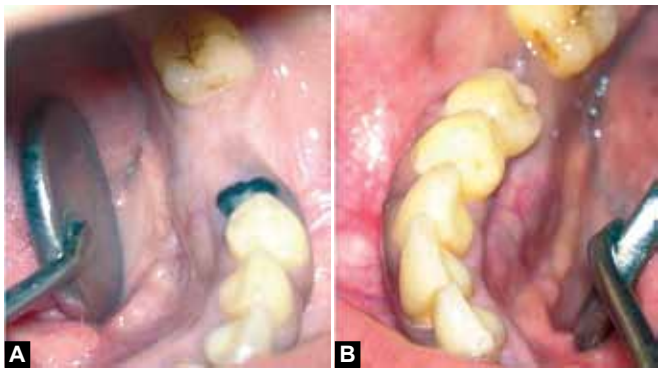


Figs 1A and B: (A) Orthopantomograph showing radiolucent area extending from the distal aspect of the root of 44 to the distal aspect of the root of 46 and (B) computerized tomography scan showing unilateral involvement of right posterior mandible



Figs 2A and B: (A) Photomicrograph showing dense connective tissue stroma with extravasated RBC's along with necrotic changes and dense chronic inflammatory infiltrate. There was no evidence of any cystic epithelium. 40×, magnification and (B) photomicrograph showing dense connective tissue stroma with extravasated RBC's along with necrotic changes and dense chronic inflammatory infiltrate. There was no evidence of any cystic epithelium (100×, magnification)

Fig. 3: Orthopantomograph showing process of bone repair



Figs 4A and B: (A) Clinical intraoral photograph showing alveolar mucosa with a swelling was seen on the right lingual aspect in relation to 47 and 48 and (B) clinical intraoral photograph showing normal alveolar mucosa

Fig. 5: Orthopantomograph showing three separate radiolucent lesions with scalloping borders that were in between the roots of 44 to 47 region, 33 to 38 region and 11 to 13 region respectively

differential diagnosis included cystic ameloblastoma, central giant cell granuloma, odontogenic myxoma, odontogenic keratocyst.

Panoramic radiography revealed three separate radiolucent lesions with scalloping borders that were in between the roots of 44 to 47 region, 33 to 38 region and 11 to 13 region respectively (Fig. 5). Computerized tomography scan revealed bilateral involvement of posterior aspect of mandible and anterior aspect of maxilla showing bicortical expansion in relation to right and left posterior mandible. Radiographic differential diagnosis of ameloblastoma, central giant cell granuloma, odontogenic myxoma, odontogenic keratocyst were put forth.

On surgical exploration under general anesthesia, a bony cavity was identified. It was devoid of tissue, fluid or epithelial lining.

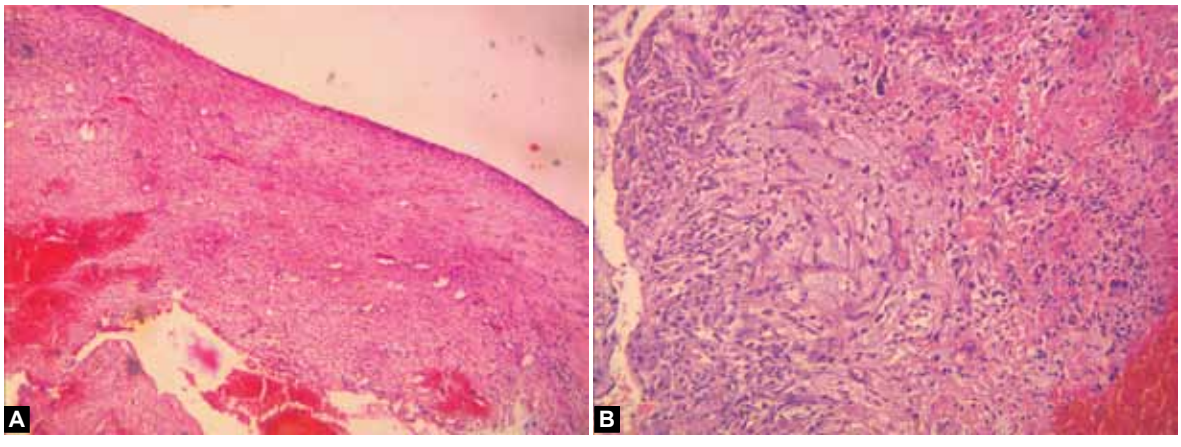
Histopathological examination revealed densely inflammed connective tissue stroma with extravasated RBCs. Necrotic changes and chronic inflammatory cells were also seen in the connective tissue. There was no evidence of any type of cystic epithelium (Figs 6A and B).

Panoramic follow-up radiographs of previously affected regions at the intervals of 6 months and 1 year after surgical intervention showed evidence that process of bone repair had occurred locally (Fig. 7).

DISCUSSION

TBC has been identified by a great variety of names in the literature, such as hemorrhagic bone cyst, simple bone cyst, solitary bone cyst, extravasation cyst, IBC, primary bone cyst.[2]

Trauma is the most frequently discussed etiologic factor in the formation of TBC. Of the pathogenic hypothesis of TBCs evaluated by Howe (1965)[3], the most widely accepted mechanism involves microtrauma an event sufficient to precipitate intramedullary hemorrhage with osteoclastic



Figs 6A and B: (A) Photomicrograph showing dense connective tissue stroma with extravasated RBC's along with necrotic changes and dense chronic inflammatory infiltrate. There was no evidence of any cystic epithelium (40×, magnification) and (B) photomicrograph showing dense connective tissue stroma with extravasated RBC's along with necrotic changes and dense chronic inflammatory infiltrate. There was no evidence of any cystic epithelium (100×, magnification)

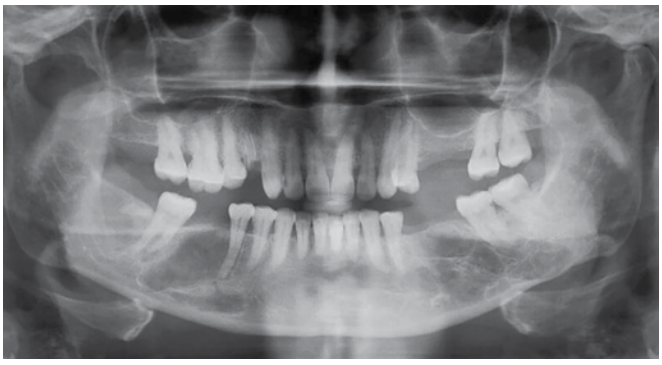


Fig. 7: Orthopantomograph showing process of bone repair

activity and elimination of trabeculae within cancellous bone compartment, yet a failure of the hematoma to undergo organization and tissue replacement. Degeneration of the hematoma results in cavitation, proposed by Olech et al.[17] Other theories that have been put forward to explain the pathogenesis of the condition include:[18]

- Infection of bone marrow
- Loss of blood supply to hemangioma or lymphoma
- Cystic degeneration of existing bone tumor
- Changes and reduction in the osteogenic activity
- Faulty calcium metabolism as a result of systemic disease (parathyroid diseases)
- Ischemic necrosis of the fatty bone marrow
- Low grade chronic infection
- Imbalance between the osteoclastic and osteoblastic activity due to trauma
- Developmental defect
- Failure of mesenchymal tissue to form bone and cartilage, and instead becomes immature as multiple bursa-like synovial cavities.

Idiopathic bone cavities are most frequently asymptomatic. Expansion of the mandible rarely occurs. A few authors have reported the occurrence of multiple TBC and their association with fibrous/bony lesions, such as florid cemento-osseous dysplasia especially in older patients. Wakasa et al (2002) have suggested that the florid cement-osseous dysplasia may precede TBC when these two conditions are associated, which suggests that disordered production of trabeculae in the former may obstruct lymphatic drainage and induce TBC formation.[19-21]

Panoramic radiographs and CT scans are essential for the diagnosis and evaluation of lesions and in many cases are the primary indicators of the pathology. Radiographic features of the lesion have generally been described as unilocular with scalloping between the roots of associated teeth.[3,4] Although, generally described as being empty, there have been reports that TBCs may rarely contain straw colored fluids, blood, connective tissue or blood clot. One school of thought postulates that the reason aspiration of idiopathic bone cavities return blood in some cases is not because the cavity is filled with blood, but the disturbance of the tip of the needle to capillaries and negative pressure in the marrow space consequently.

Typically, histopathological findings in TBC are nonspecific and reveal fibrous connective tissue and normal bone with no evidence of any epithelial lining. The lesion may show areas of vascularity, fibrin, erythrocytes and occasional giant cells adjacent to bone surface.

Central giant cell lesions, odontogenic keratocyst, ameloblastoma, lateral periodontal cyst, and other destructive bone cysts and tumors should be taken into consideration in differential diagnosis.

The most commonly recommended treatment for TBC is surgical exploration of the lesion followed by curettage of the bony walls. This surgical exploration serves as both diagnostic maneuver and definitive therapy by producing bleeding in the cavity. This bleeding helps to form a clot which is eventually replaced by bone.[22] Literature mentions about the filling of cavity with blood, bovine lyophilized bone, autologous blood with bone from patient or synthetic bone materials or single bone grafting without blood as possible treatment options, if the surgical exploration done is ineffective. Although, these materials have been sparingly used, its efficacy and efficiency are yet to be fully studied.[23]

Careful curettage of the lesion favors progressive bone regeneration, offering a good prognosis and an almost negligible relapse rate. Osseous regeneration can be verified after some months by radiographic evaluation.

CONCLUSION

Generally all maxillofacial lesions present with a standard set of clinical or radiographical presentations. Proper recording of case history, knowledge of symptoms and correct radiographic interpretation will be helpful for proper diagnosis and management. Being open to all probabilities and possibilities would be an added advantage. Traumatic bone cyst should be a differential diagnosis in any case with an asymptomatic occult, cystic space especially with a history of trauma. Although devoid of complications, if left untreated, it may result in pathological fracture as a result of the hollowing out of the affected bone.

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