Traumatic bone cyst of an anterior mandible with previous symphyseal fracture in a pediatric patient: a rare finding and etiopathologic correlation

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Traumatic bone cysts (TBCs) are uncommon intraosseous lesions, classified as pseudocysts because they lack an epithelial membrane lining. The etiology of a pseudocyst has not been determined. Various hypotheses have been put forward to explain its pathogenesis, of which the traumatic-hemorrhagic theory is the most commonly accepted. Minor trauma, insufficient to cause fracture or iatrogenic injury, is commonly implicated as the stimulus initiating cyst formation. A TBC presenting after jaw fracture has been rarely reported in the literature. This article presents a case of a TBC of the anterior mandible in a child with a previous history of trauma and fracture of the symphysis. The article also reviews the literature to corroborate the possible role of major trauma in the pathogenesis of TBC.

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Case report

A 12-year-old boy was referred to an oral surgery clinic for evaluation of an asymptomatic periapical lesion that was discovered incidentally during routine radiographic screening. The lesion predominantly involves the posterior mandible, located between the inferior alveolar canal and root apices of the mandibular premolar and molar teeth. This case report describes a TBC of the anterior mandible in a boy with a history of trauma and symphyseal fracture. A literature review of the association between TBC and major trauma or jaw fracture is also presented.
border of the mandible was noted at the midline, suggestive of previous symphyseal fracture (Fig 1). The lamina dura of the anterior teeth was intact.

The radiographic findings were suggestive of a benign intraosseous lesion, with evidence of previous symphyseal fracture. A provisional diagnosis of TBC was made; differential diagnoses included central giant cell granuloma, aneurysmal bone cyst, and ameloblastoma.

Surgical exploration and biopsy of the lesion were planned and executed under local anesthesia. A vestibular incision was placed, and the mucoperiosteal flap was raised over the labial cortex. There was no expansion or thinning of the cortical bone. A bony window was created to access the lesion, which was empty and had no soft tissue lining. The intraoperative findings, along with the clinical and radiographic features, confirmed the diagnosis of TBC. The bone cavity was explored, and surrounding bone was curetted to induce fresh bleeding. Primary closure was attained using resorbable suture (Fig 2).

**Discussion**

The diagnostic criteria for a TBC include an intraosseous lesion without an epithelial lining, surrounded by bony walls. The lesion is either empty or contains liquid or connective tissue.

The fact that TBCs have been known by a variety of names over the years reflects the lack of understanding of the true etiopathogenesis of the disease. Of the various theories proposed to explain the development of a TBC, the traumatic-hemorrhagic theory is most widely accepted. This theory states that when a hematoma from intramedullary hemorrhage subsequent to trauma fails to consolidate, it gives rise to a TBC. The hematoma then liquefies and induces transudation of fluid into the cyst. This causes an increase in internal pressure, leading to venous stasis, bone marrow necrosis, and osteoclastic resorption, contributing to the formation and growth of the TBC.

One type of trauma implicated in the formation of TBCs is iatrogenic damage (such as tooth extraction, orthodontic traction, or any external injury) that is insufficient to cause jaw fracture. A TBC arising in a patient subsequent to mandibular fracture rarely has been reported. A search of the English-language literature in the PubMed database—using the terms traumatic, solitary, idiopathic, cyst, mandible, and fracture—revealed 102 articles, of which only 4 cases were reported to be associated with mandibular fracture (Table). Fractures in 3 of the cases were secondary to trauma to a mandible weakened by a cystic lesion (pathologic fracture). In 1 case, multiple cysts were diagnosed in a patient with osteogenesis imperfecta who...
had a previous history of condylar fracture. In the present case, the lesion was found in an anterior mandible with a history of trauma and radiographic evidence of previous symphyseal fracture. Despite earlier theories that only minor external injury initiates TBCs, these findings suggest that major trauma leading to jaw fracture can also trigger pathogenesis of the lesion. This further emphasizes the need to closely monitor pediatric patients with a history of mandibular fracture to rule out development of a TBC.

Although trauma is believed to be the single most important cause of TBCs, not all cases are associated with a history of trauma. Studies have reported a clear history of trauma in less than 29% of cases. This may be attributed to lack of careful medical history recording or the fact that minor trauma may not be remembered by the patient. The higher incidence of trauma in the anterior part of the mandible is also at odds with the higher incidence of TBCs in the posterior region of the mandible. Other hypotheses proposed to explain pathogenesis of TBCs include degeneration of bone tumors, disorders of calcium metabolism, various mild infections, local disorders of bone growth, venous obstruction, intensified osteolysis, degeneration of a bone cyst, and ischemia with necrosis of the bone caused by primary vascular or neurogenic disorders.

The majority of maxillofacial TBCs are located in the mandibular region between the apices of premolar or molar teeth and the inferior alveolar canal. Although uncommon, cases have also been reported in the symphysis, ramus, condyle, and maxilla. In a review of 21 cases of TBC, 16 (76%) were seen in the mandibular region, 3 (14%) cases were located in the symphysis, and 2 (10%) were in the ramus. In a similar retrospective review of 26 patients with TBC, 8 (30%) were seen in the anterior mandible, 1 (4%) in the ramus region, and 17 (66%) in the mandibular body. The traumatic-hemorrhagic theory seems to be the explanation for the more common involvement of the mandible rather than the maxilla. The mandible, which has more cortical bone, repairs itself more slowly than the maxilla.

Although they may be detected at any age, TBCs are more commonly seen in the first 2 decades of life. The greater incidence of TBCs in young patients can be possibly explained by the higher likelihood of trauma in this age group, while the reduced prevalence in older patients suggests that this lesion may be self-repairing. There is no consensus on sex predilection, with some studies noting a male predominance while others report equal involvement or a female predisposition.

The TBC usually presents as an asymptomatic lesion incidentally detected during a radiographic survey. In symptomatic cases, pain or tooth sensitivity is the most common presentation. Less frequently reported features include cortical expansion, paresthesia, and fracture. Radiographically, TBCs typically present as well-defined, unilocular radiolucencies with wavy borders. In its most common location, the radiolucency lies between the apices of mandibular posterior teeth and the inferior alveolar canal. The structure of the mandibular canal may or may not be preserved. The superior margin of the radiolucency is indented between the teeth, creating a scalloped appearance. There is no resorption or displacement of teeth. The cortical bone or lamina dura is usually not involved.

In the case described in this report, the lesion presented as a well-defined, unilocular, irregular radiolucency between the apices of the incisor teeth and the lower border of the anterior mandible. A disruption of the lower border at the symphyseal region, in combination with a history of trauma, was suggestive of previous symphyseal fracture. This could have been the trigger for the initiation of the TBC, supporting the traumatic-hemorrhagic theory.

Surgical exploration serves for both confirmation of the diagnosis and management of the TBC. Curettage induces fresh bleeding within the bone cavity, which consolidates and ultimately ossifies with complete healing. Specimens for histopathologic evaluation are either scanty or completely absent, limiting the role of pathologic evaluation for diagnosis. The clinical presentation, radiographic features, and intraoperative finding of an empty bone cavity with fluid, blood, or scanty connective tissue are sufficient for diagnosis of TBCs. Some TBCs heal spontaneously without any intervention. Recurrence is extremely low.

**Conclusion**

Traumatic bone cysts are mostly asymptomatic and are usually discovered during routine radiographic examination. They are predominantly seen in pediatric patients and commonly involve the posterior mandible with or without a history of minor trauma or iatrogenic injury. This article highlights a case of an anterior mandibular TBC in a child with previous symphyseal fracture, which suggests that major trauma causing jaw fracture may possibly initiate pathogenesis of this lesion. This finding highlights the importance of closely monitoring pediatric patients after trauma to rule out development of a TBC.
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References