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Case Report

The paradox of benignity and clinical aggression: An extreme odontogenic myxoma

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Abstract

Odontogenic myxoma is a rare, benign but locally aggressive odontogenic tumor of ectomesenchymal origin, accounting for approximately 3–6% of all odontogenic tumors. It most commonly affects individuals in the second to fourth decades of life, with a mean age of presentation around 30–40 years and shows a slight female predilection. The mandible is more frequently involved (60–70%), particularly the posterior body and ramus, while maxillary lesions (30–40%) tend to demonstrate more aggressive behaviour due to early sinus involvement. Clinically, odontogenic myxoma presents as a slow-growing, painless jaw swelling, often reaching a considerable size before diagnosis. Radiographically, it appears as a uni or multilocular radiolucency with fine internal septations producing a characteristic honeycomb or soap-bubble pattern, along with cortical expansion and thinning. Owing to its infiltrative nature and high recurrence potential, accurate imaging is essential for management. This report describes a case of odontogenic myxoma in a 45-year-old male presenting with a large swelling of jaw with pus discharge.

Keywords: Odontogenic myxoma, Benign odontogenic tumour, Multiloculated radiolucency, Myxoid stroma, Fine needle aspiration cytology

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1. Introduction

Odontogenic myxoma is an uncommon benign neoplasm of odontogenic ectomesenchymal origin that arises almost exclusively within the maxillofacial region, reflecting its close association with tooth-forming tissues such as the dental papilla, dental follicle, and periodontal ligament.[1,2] Although histologically benign, odontogenic myxoma demonstrates locally aggressive and infiltrative behavior within cancellous bone, accounting for approximately 3–6% of all odontogenic tumors.[1,3] The lesion most commonly affects individuals in the second to fourth decades of life, with a slight female predominance reported in several series.[2,4] Anatomically, the mandible is more frequently involved than the maxilla; however, maxillary lesions are often considered more aggressive because of early extension into adjacent anatomical spaces such as the maxillary sinus and nasal cavity.[3,5]

Clinically, odontogenic myxoma usually presents as a slow-growing, painless swelling, which often results in delayed diagnosis and extensive bone involvement at presentation.[2,4] Radiographically, the lesion exhibits variable appearances ranging from well-defined unilocular radiolucencies to multilocular lesions with fine internal septations, producing characteristic honeycomb, soap-bubble, or tennis-racket patterns, along with cortical expansion, tooth displacement, and occasional root resorption.[5,6] Histopathologically, it is composed of stellate and spindle-shaped cells dispersed in an abundant myxoid stroma with minimal collagen content.[1] Due to its infiltrative growth pattern and notable recurrence rate following conservative treatment, accurate diagnosis supported by thorough radiologic and histopathologic evaluation is essential for appropriate surgical planning and long-term follow-up.[3,6]

2. A Case Report

A 45-year-old male patient came to the department of oral medicine and radiology with chief complaint of large extensive swelling in the jaw region nearly since birth. The swelling was painless and associated with continuous pus discharge from 3-4 months. He was not having any systemic illness or drug history. On extraoral examination (In Figure 1) there was a large well-defined swelling extending from left infraorbital region till the hyoid bone and mediolaterally from left ear till right angle of mandible. The size of jaw swelling approx. 14 x 12 cm (SI x TR). A single punctate extraoral draining sinus noted with inferior aspect of swelling with active pus discharge noted. Facial asymmetry present due to large swelling. Reduced mouth opening till 9 mm. On palpation swelling was soft to firm to bony hard in consistency with local rise in temperature. On intraoral examination (In Figure 2) poor oral hygiene with teeth present were 18, 17, 16,15,14,13,12,11, 21, 22, 23, 31, 32, 33, 34, 41,42,43,44,45,46,47,48. Missing teeth were 24,25,26,27,28. Root piece were 41,42,43. Entire mandibular jaw got deviated to right side with buccolingual expansion and severe teeth displacement. Detailed examination was not

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possible due to reduced mouth opening.

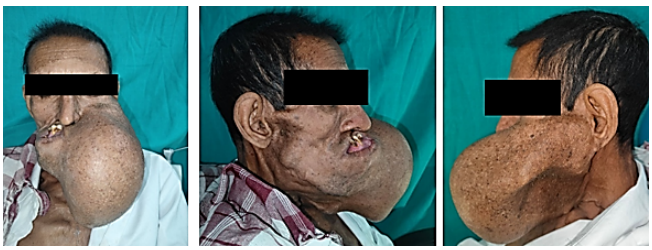

Figure 1: *Extraoral photographs*

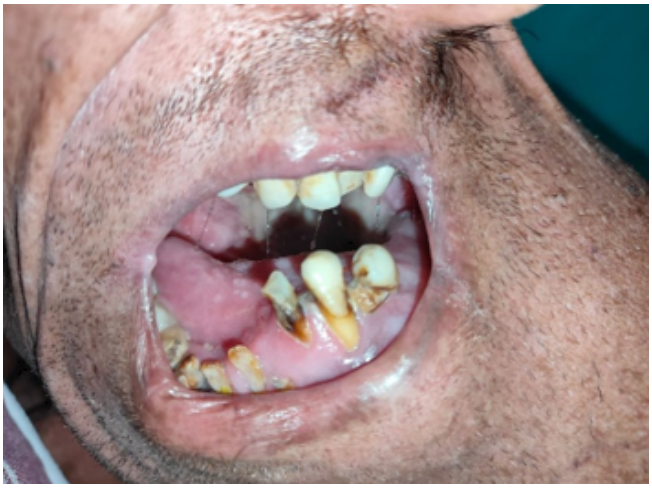

Figure 2: *Intraoral examination*

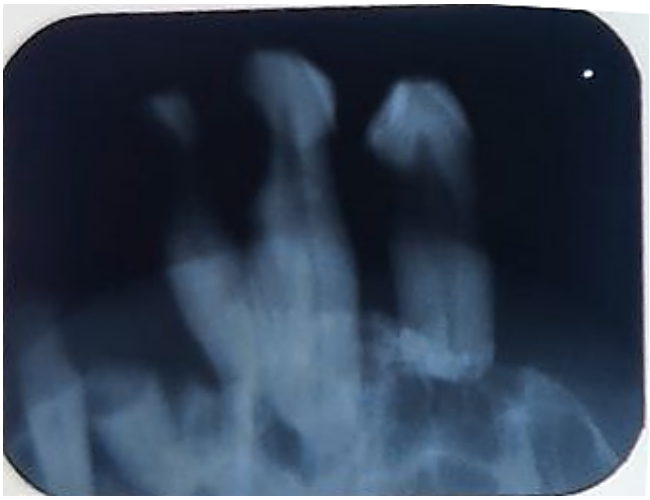

Figure 3: *IOPA 34,33,32,31,41*

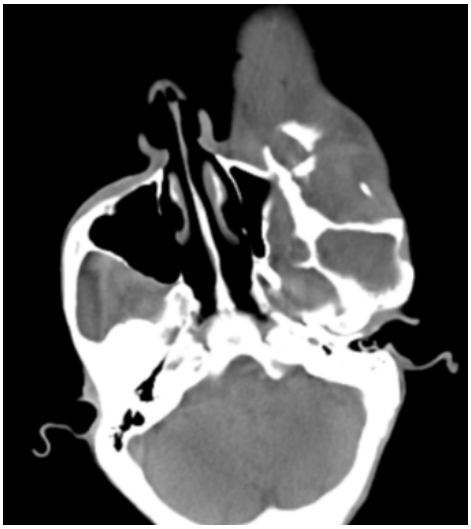

Figure 4: *Axial section*

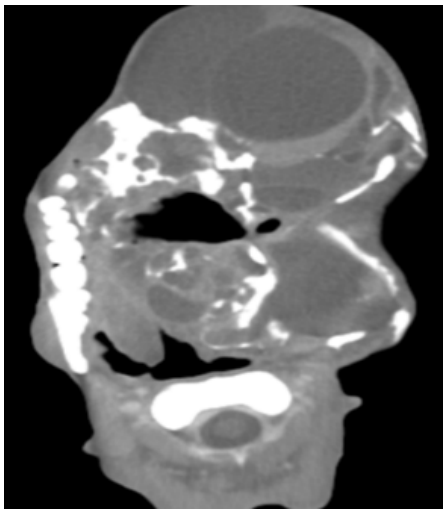

Figure 5: *Axial section*

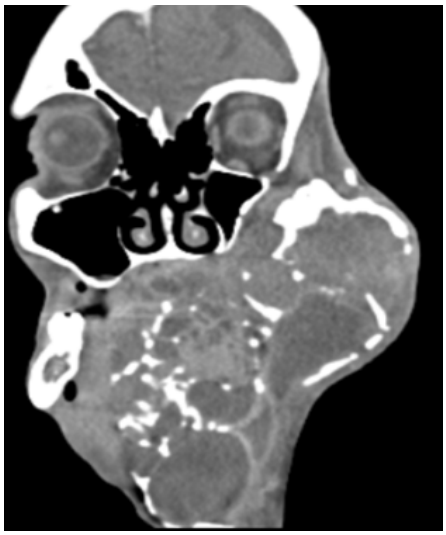

Figure 6: *Coronal section*

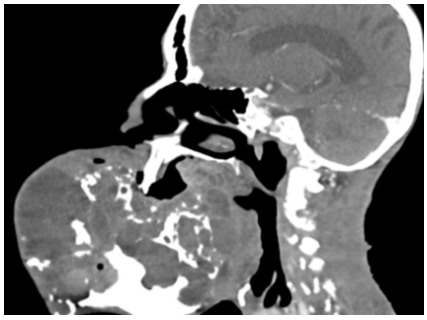

Figure 7: *Sagittal section*

Figure 8: *Fine needle aspiration cytology*

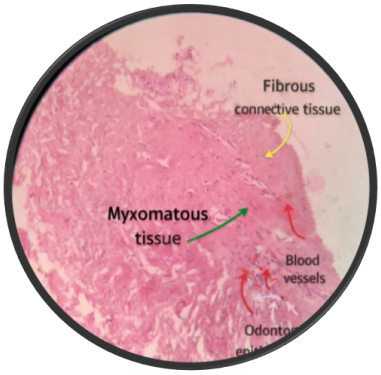

Figure 9: *Histopathology slide*

Radiological investigation – Initially Intraoral periapical radiograph (In Figure 3) was taken. Teeth seen were 31,32,33,34,41, they were destructed due to caries. Periapical bone shows multilocular radiolucency. Then he was sent for a Computed Tomography scan.

On CT examination (In Figure 4,5,6,7,) - A large multiloculated expansile lytic lesion measuring 16.5x12.5x14.5 cm (AP x TR x CC) is noted involving the left mandible crossing midline to involve body of right mandible. The lesion exhibits multiple internal septations within (giving soap bubble appearance), predominantly hypodense fluid content with peripheral and septal enhancement, marked cortical thinning and gross expansion of buccal and lingual cortex with extraosseous soft tissue noted. It has caused mass effect on nearby structures such as mild tracheal narrowing, superiorly it is, extending to left maxilla with resultant remodelling, displacement of left masticator space, medially causing thinning and scalloping of lateral wall of left maxillary sinus. The lesion shows communication with oral cavity with resultant air foci within. Tongue was not seen separately from the lesion. No involvement of hard palate, left pterygoid plates, left styloid process. Features s/o benign odontogenic tumour, considering differential diagnosis as Ameloblastoma.

Patient was sent for further investigations like fine needle aspiration cytology (FNAC) and incisional biopsy for diagnosis.

FNAC (Figure 8) – On aspiration syringe with frank blood shows haemorrhagic aspirate, straw-coloured fluid (clear yellow) shows myxoid aspirate, composed of mucin-rich extracellular matrix, Hyaluronic acid, dark yellow / slightly turbid fluid represents concentrated myxoid material with old altered blood pigments.

On histopathological investigations of biopsy specimen – (Figure 9)

Microscopic examination reveals a non-encapsulated, locally invasive tumour composed of a hypocellular and abundant loose, myxoid (mucoid) stroma. Within this matrix, there is a proliferation of randomly oriented stellate, spindle-shaped, and occasionally round mesenchymal cells that possess long, delicate, and often anastomosing cytoplasmic processes. The nuclei are typically small and hyperchromatic, with low mitotic activity and a lack of cellular atypia. While the stroma is primarily myxomatous, it may contain varying amounts of delicate collagen fibres and thin-walled blood vessels. Small, inactive islands or nests of odontogenic epithelium are frequently interspersed throughout the lesion but are not a mandatory feature for diagnosis. Residual bony trabeculae may also be present, reflecting the tumour's infiltrative nature as it permeates through the surrounding marrow spaces of the jaw. Final diagnosis were made as “Odontogenic Myxoma”.

Patient was admitted and prepped for surgery but got died due to systemic condition.

3. Discussion

The present case demonstrates an exceptionally rare and advanced presentation of odontogenic myxoma, primarily distinguished by its enormous size, long-standing duration since early life, midline crossing, extraosseous extension, and secondary infection. While odontogenic myxoma is known for slow but infiltrative growth, lesions of such magnitude are seldom reported in contemporary literature, largely due to earlier detection facilitated by improved access to diagnostic imaging and dental care.[7]

One of the most striking aspects of this case is the extraordinary size of the lesion, which far exceeds the average dimensions reported in most clinicopathological series. Simon et al. noted that the majority of odontogenic myxomas are diagnosed before reaching extensive proportions, as facial asymmetry and jaw expansion usually prompt patients to seek treatment earlier.[8] The prolonged neglect in this case allowed uninterrupted tumor growth, resulting in extensive mandibular destruction, deviation of the jaw, and involvement of adjacent maxillofacial structures.

Radiologically, the lesion exhibited a multiloculated "soap-bubble" appearance with fine internal septations, marked cortical thinning, and gross expansion of both buccal and lingual cortices. Although such imaging characteristics are well documented for odontogenic myxoma, the presence of extraosseous soft-tissue extension, airway compromise, maxillary remodelling, and communication with the oral cavity is unusual and indicative of aggressive local behaviour.[9] Kaffe et al. emphasized that cortical perforation and soft-tissue extension are relatively uncommon findings and are typically associated with long-standing or recurrent lesions.[9]

The crossing of the mandibular midline and extension into the contralateral body of the mandible further illustrates the infiltrative nature of odontogenic myxoma. This behaviour can be attributed to the tumour's lack of encapsulation and its propensity to permeate through cancellous bone marrow spaces rather than displacing surrounding structures.[10] Additionally, superior extension with scalloping of the maxillary sinus wall and remodelling of the maxilla is particularly significant, as maxillary involvement is often associated with more aggressive clinical behaviour due to anatomical proximity to vital spaces.[11]

Secondary infection, manifested clinically as continuous pus discharge through an extraoral draining sinus, is rarely reported in odontogenic myxoma. This complication likely resulted from cortical perforation and oral cavity communication, compounded by poor oral hygiene and extensive tumour necrosis. Such features further complicated the clinical picture and contributed to functional impairment, including severe trismus and difficulty in detailed intraoral examination.

Histopathological findings in the present case were consistent with classic odontogenic myxoma, showing a hypocellular myxoid stroma containing stellate and spindle-shaped cells with long cytoplasmic processes, minimal collagen, and absence of atypia. Despite its alarming clinical and radiologic presentation, the microscopic appearance reaffirmed the benign nature of the lesion, highlighting the well-recognized discrepancy between histologic blandness and clinical aggressiveness in odontogenic myxoma.[7,12]

From a management perspective, large odontogenic myxomas typically warrant radical surgical resection with adequate margins, as conservative approaches such as curettage are associated with high recurrence rates.[8,11] Unfortunately, in the present case, definitive surgical intervention could not be carried out due to the patient's deteriorating systemic condition, culminating in death. This outcome underscores the potentially life-threatening consequences of delayed diagnosis and treatment, even in histologically benign odontogenic tumors.

Overall, the sheer size, extent, secondary infection, and functional compromise observed in this case place it among the rarest and most severe presentations of odontogenic myxoma reported, reinforcing the need for early recognition and timely intervention.

4. Conclusion

This case highlights an exceptionally rare presentation of a giant odontogenic myxoma with extensive mandibular involvement, midline crossing, extraosseous extension, and secondary infection. Despite its benign histopathological nature, the lesion exhibited aggressive local behaviour resulting in significant functional and anatomical compromise. Delayed diagnosis played a crucial role in the progression to this advanced stage. Early detection, comprehensive imaging evaluation, and timely surgical management are essential to prevent severe morbidity and mortality associated with such lesions. Long-term follow-up remains critical due to the infiltrative growth pattern and recurrence potential of odontogenic myxoma.

5. Source of Funding

None.

6. Conflict of Interest

None.

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