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ODONTOGENIC MYXOMA OF THE MAXILLA MANAGED BY PARTIAL MAXILLECTOMY AND IMMEDIATE OBTURATOR REHABILITATION: A CASE REPORT AND REVIEW OF LITERATURE

Oral and Maxillofacial Surgery

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ABSTRACT

Odontogenic myxoma (OM) is a rare benign mesenchymal odontogenic neoplasm accounting for approximately 3–6% of all odontogenic tumors. Despite its benign histological appearance, the lesion demonstrates locally aggressive behavior because of its infiltrative growth pattern, absence of encapsulation, and tendency to invade cancellous bone. Although the posterior mandible is the most frequently affected site, maxillary lesions are comparatively uncommon and often exhibit greater clinical aggressiveness because of their propensity to extend into the maxillary sinus, nasal cavity, and adjacent craniofacial structures. Early diagnosis remains challenging because of the slow-growing and asymptomatic nature of the lesion. A patient presented with a gradually enlarging painless swelling involving the posterior maxilla. Clinical examination demonstrated cortical expansion with obliteration of the buccal vestibule. Cone-beam computed tomography (CBCT) revealed a multilocular expansile radiolucent lesion involving the posterior maxilla with cortical thinning and extension into the maxillary sinus. Histopathological examination demonstrated stellate and spindle-shaped cells dispersed within abundant myxoid extracellular matrix containing delicate collagen fibers and scattered odontogenic epithelial rests, confirming the diagnosis of odontogenic myxoma. Complete surgical excision with peripheral curettage was performed. The postoperative course was uneventful, and no evidence of recurrence was observed during follow-up. Odontogenic myxoma should always be considered in the differential diagnosis of multilocular radiolucent lesions of the jaws. Clinicoradiographic correlation together with histopathological confirmation is essential for definitive diagnosis. Surgical excision with adequate margins remains the treatment of choice, while long-term postoperative surveillance is mandatory because of the significant risk of recurrence.

KEYWORDS

Odontogenic Myxoma; Fibromyxoma; Weber Fergusson Approach

INTRODUCTION

Odontogenic myxoma (OM) is an uncommon benign odontogenic neoplasm originating from the ectomesenchymal component of the developing tooth apparatus. It is characterized by slow but persistent growth, local invasiveness, and a remarkable tendency to infiltrate adjacent cancellous bone despite lacking metastatic potential.[1-4] The tumour constitutes approximately 3–6% of all odontogenic tumours and approximately 0.04–3.7% of all primary bone tumours, making it one of the least frequently encountered odontogenic neoplasms in routine clinical practice.[2,5] The term myxoma was first introduced by Virchow in 1863 to describe tumours resembling the gelatinous tissue of the umbilical cord.[6] Subsequently, Thoma and Goldman recognized the odontogenic nature of jaw myxomas in 1947, proposing their origin from the mesenchymal components of the developing tooth germ.[7] Since then, considerable advances in histopathology, immunohistochemistry, and molecular biology have strengthened the concept that odontogenic myxoma arises from primitive ectomesenchymal tissues such as the dental papilla, periodontal ligament, or dental follicle.[8-10]

The 2022 World Health Organization (WHO) Classification of Head and Neck Tumours categorizes odontogenic myxoma as a benign mesenchymal odontogenic tumour with or without inactive odontogenic epithelium.[11] Histologically, the tumour is composed of stellate, spindle-shaped, and angular fibroblast-like cells suspended within an abundant myxoid extracellular matrix rich in hyaluronic acid and chondroitin sulfate. Small islands of inactive odontogenic epithelium may occasionally be identified, further supporting its odontogenic origin.[8,12] Although histologically benign, odontogenic myxoma exhibits locally aggressive biological behaviour because it lacks a true fibrous capsule. Tumour cells infiltrate the surrounding cancellous bone through microscopic extensions that frequently extend beyond the radiographically visible margins. This infiltrative growth pattern contributes significantly to postoperative recurrence following conservative surgical procedures.[13-15] Reported recurrence rates vary between 10% and 33%, depending upon tumour size, anatomical location, surgical approach, and duration of follow-up.[15-17] The lesion predominantly affects young adults during the second and third decades of life, with a slight female predilection reported in most epidemiological studies.[3,15,18] Approximately 65–70% of cases arise within the mandible, particularly the posterior body, angle, and ramus. Maxillary lesions are considerably less common but generally demonstrate more aggressive clinical behaviour owing to the porous architecture of the maxilla and the proximity of the maxillary sinus, nasal cavity, orbit, and pterygomaxillary region, allowing tumour extension before clinical detection.[19-21] Clinically, odontogenic myxoma is often asymptomatic during its early stages and typically presents as a slowly enlarging painless swelling producing facial asymmetry. Progressive enlargement may result in cortical expansion, tooth displacement, malocclusion, root resorption, delayed eruption, mobility of adjacent teeth, and, occasionally, perforation of cortical plates. Pain, paraesthesia, and ulceration are uncommon and usually indicate secondary infection or extensive local invasion.[3,14,22]

Radiographically, odontogenic myxoma demonstrates considerable variability. Small lesions frequently appear as unilocular radiolucencies, whereas larger tumours commonly exhibit multilocular radiolucent patterns with characteristic internal bony septa producing the classical "soap bubble," "honeycomb," "tennis racket," "spider web," or "step-ladder" appearance.[8,23] Conventional panoramic radiography often underestimates lesion extent, whereas cone-beam computed tomography (CBCT) provides superior evaluation of cortical perforation, internal trabecular architecture, root resorption, relationship to adjacent teeth, maxillary sinus involvement, and surgical planning.[24,25] Magnetic resonance imaging (MRI) may further assist in delineating tumour extension into adjacent soft tissues.

Typically, odontogenic myxoma demonstrates low-to-intermediate signal intensity on T1-weighted images and high signal intensity on T2-weighted sequences because of its abundant myxoid matrix (26). However, MRI is generally reserved for extensive maxillary lesions involving neighbouring anatomical spaces. The differential diagnosis of odontogenic myxoma includes ameloblastoma, odontogenic fibroma, odontogenic keratocyst, central giant cell granuloma, aneurysmal bone cyst, central haemangioma, intraosseous mucoepidermoid carcinoma, fibrous dysplasia, and desmoplastic fibroma.[8,27] Histopathological examination therefore remains indispensable for establishing a definitive diagnosis. Microscopically, odontogenic myxoma is characterized by a hypocellular myxoid stroma containing loosely arranged spindle-shaped and stellate fibroblast-like cells embedded in abundant glycosaminoglycan-rich extracellular matrix. Variable amounts of collagen fibres may be present, giving rise to the term fibromyxoma when the fibrous component predominates.[12,28] Recent molecular studies have demonstrated overexpression of matrix metalloproteinases (MMP-2 and MMP-9), tenascin, fibronectin, and various extracellular matrix proteins that are believed to contribute to the tumour's locally invasive behaviour. The optimal treatment of odontogenic myxoma remains controversial. Conservative procedures such as enucleation and curettage preserve adjacent anatomical structures but are associated with higher recurrence rates because of residual microscopic tumour infiltration. Conversely, marginal or segmental resection provides lower recurrence rates but increases functional and aesthetic morbidity. Consequently, treatment should be individualized according to tumour size, anatomical location, cortical perforation, and patient-related factors.[15,17]

The present report describes a rare case of odontogenic myxoma involving the maxilla and discusses its clinical presentation, radiological characteristics, histopathological findings, surgical management, and relevant literature, emphasizing the importance of early diagnosis, meticulous surgical excision, and prolonged postoperative surveillance.

CASE REPORT

A patient presented to our department with the chief complaint of a gradually enlarging swelling involving the posterior region of the maxilla in left side. The swelling had increased progressively over several months without any associated pain or constitutional symptoms. There was no history of trauma, infection, previous surgery, or similar lesions elsewhere in the body. The patient's medical, dental, and family histories were non-contributory, and no systemic illness or syndromic association was identified. The patient primarily sought treatment because of the progressive facial asymmetry and intraoral swelling, which had begun to interfere with mastication and oral hygiene maintenance. No history of paraesthesia, epistaxis, nasal obstruction, visual disturbances, or weight loss was elicited.

Clinical Examination

Extraoral Examination: General physical examination revealed a patient in good systemic health with stable vital signs. Facial inspection demonstrated mild-to-moderate unilateral facial asymmetry caused by diffuse swelling involving the left side of the midface (Figure 1). The overlying skin appeared normal without erythema, ulceration, or sinus formation. On palpation, the swelling was: diffuse, non-tender, firm to hard in consistency, non-fluctuant, non-compressible, non-pulsatile, fixed to the underlying bone. The margins were ill defined because of gradual blending into the surrounding facial contours. Regional lymph nodes were not palpable, and no facial nerve deficit was observed. Maximum mouth opening was within normal limits, and temporomandibular joint movements were normal.

Figure 1: Extraoral view of the patient.

Intraoral Examination: Intraoral examination revealed a diffuse bony hard swelling involving the posterior maxillary alveolar process extending from the 23 region to the 26 region (Figure 2).

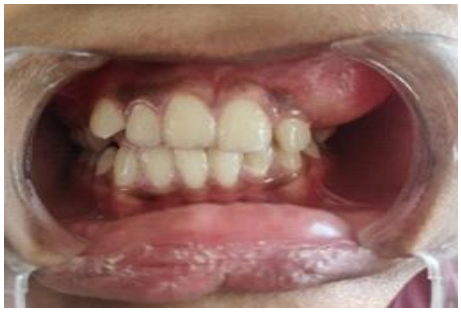

Figure 2: Intraoral view of the patient.

The lesion demonstrated: Buccal cortical expansion , Obliteration of the buccal vestibule, Intact overlying mucosa, Smooth surface, Normal mucosal colour, Palpation confirmed expansion of both cortical plates without evidence of crepitus. There was no pus discharge, ulceration, or mucosal breach. The lesion was painless throughout clinical examination.

Radiographic Assessment

Orthopantomogram (OPG): Panoramic radiography demonstrated a well-defined multilocular radiolucent lesion occupying the posterior maxilla (Figure 3).

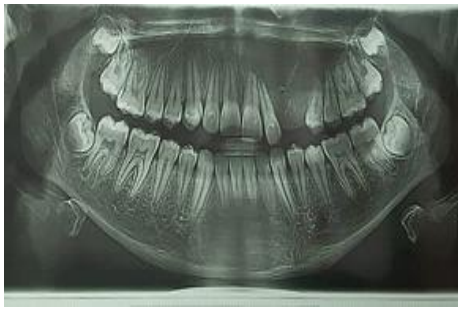

Figure 3: OPG of the patient.

The lesion exhibited: Multilocular appearance, Internal fine bony septae, Expansion of surrounding bone, Displacement of adjacent teeth, Variable root divergence. The lesion extended superiorly toward the floor of the maxillary sinus with partial loss of sinus floor continuity. The radiographic appearance was suggestive of a benign but locally aggressive odontogenic lesion such as odontogenic myxoma or ameloblastoma.[23,24]

Cone-Beam Computed Tomography (CBCT): CBCT examination provided detailed three-dimensional assessment of the lesion. The lesion appeared as a well-defined expansile osteolytic lesion demonstrating: Buccolingual cortical expansion, Cortical thinning, Focal cortical perforation (if present), Fine internal trabeculations, Multilocular architecture, Extension into the maxillary sinus, Inferior displacement of the sinus floor, Displacement of adjacent teeth, loss of cortical boundaries (Figure 4). CBCT was particularly valuable for evaluating: Exact tumour dimensions, Relationship with adjacent teeth, Cortical perforation, Maxillary sinus involvement, Surgical accessibility, Reconstruction planning. These findings are consistent with previous reports emphasizing the superiority of CBCT over conventional panoramic radiography for evaluating odontogenic myxoma.[24-26]

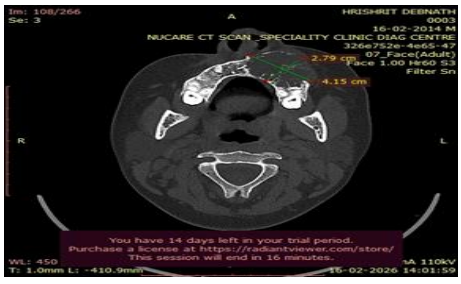

Figure 4: CBCT of the patient.

Histopathological Examination: An incisional biopsy was performed under local anaesthesia after obtaining informed consent. The biopsy specimen consisted of wedge-shaped tissue from GBS sulcus. Microscopic examination using haematoxylin and eosin staining revealed: Hypocellular loose myxoid connective tissue, Numerous spindle-shaped fibroblast-like cells, Stellate cells with long cytoplasmic processes, Oval mesenchymal cells, Abundant extracellular myxoid matrix, Delicate collagen fibres, Scattered thin-walled capillaries, islands of inactive odontogenic epithelium, Absence of cellular atypia, No abnormal mitotic figures, No necrosis. The extracellular matrix demonstrated abundant mucopolysaccharide-rich ground substance consistent with a myxomatous lesion.[8,12] Immunohistochemical evaluation, typically demonstrates: Positive Vimentin, Variable Smooth Muscle Actin (SMA), Negative Cytokeratin, Low Ki-67 proliferation index (<5%) These findings support the diagnosis of odontogenic myxoma and exclude spindle-cell malignancies.

The histopathological findings, together with the clinical and radiological features, established the definitive diagnosis of Odontogenic Myxoma.

Final Diagnosis: Odontogenic Myxoma involving the posterior maxilla with extension into the maxillary sinus.

Surgical Management

Following comprehensive clinical, radiographic, and histopathological evaluation, definitive surgical treatment was planned. Owing to the extensive involvement of the left maxilla, cortical expansion, and extension of the lesion, left partial maxillectomy was considered the most appropriate treatment to achieve complete tumour removal while minimizing the risk of recurrence. The surgical plan was explained to the patient, and informed written consent was obtained. The procedure was performed under general anaesthesia with nasotracheal intubation under strict aseptic precautions.

Surgical Procedure: The patient was positioned supine, and the surgical field was prepared and draped in the standard sterile fashion. Local infiltration of 2% lignocaine with 1:100,000 adrenaline was administered along the planned incision line to provide haemostasis and facilitate soft tissue dissection. A Weber–Fergusson incision was used to obtain adequate surgical exposure of the left maxilla. The incision extended from the upper lip, around the lateral aspect of the nose, and along the infraorbital region, providing excellent access to the maxillary lesion while allowing complete visualization of the surgical field (Figure 5).

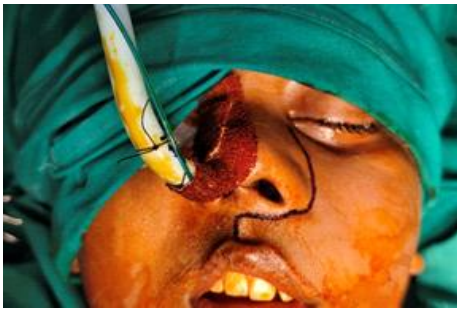

Figure 5: Weber-Fergusson Incision.

The skin and soft tissues were carefully elevated, preserving the surrounding neurovascular structures wherever possible. Following elevation of the soft tissue flap, the anterior wall of the left maxilla was exposed. The tumour had caused expansion and thinning of the cortical bone with involvement of the underlying maxillary bone. Based on the extent of tumour involvement, a left partial maxillectomy was performed. Osteotomy cuts were made using rotary instruments under copious sterile saline irrigation according to the predetermined surgical margins. The tumour-bearing segment of the left maxilla was removed en-bloc, ensuring adequate margins of uninvolved bone to reduce the possibility of residual microscopic disease. The excised specimen was carefully inspected to confirm complete tumour removal and immediately submitted for histopathological examination. The surgical cavity was thoroughly irrigated with sterile normal saline, and meticulous haemostasis was achieved. Because of the resulting maxillary defect, an immediate surgical obturator was placed intraoperatively to restore separation between the oral and nasal cavities. The obturator provided immediate functional rehabilitation by facilitating speech, swallowing, mastication, and maintenance of facial contour during the postoperative healing period. The prosthesis also acted as a surgical dressing by supporting the soft tissues and protecting the wound. The surgical wound was closed in layers using 3-0 polyglactin (Vicryl®) sutures for the deeper tissues and 3-0 nylon for skin closure. The Weber–Fergusson incision was carefully approximated to achieve optimal cosmetic healing. The patient tolerated the procedure well without intraoperative complications.

Gross Pathological Examination: The resected specimen consisted of the tumour-bearing segment of the left maxilla with attached soft tissue. Grossly, the specimen measured approximately 2.5 × 3.5 × 2cm and demonstrated a grey-white, lobulated, gelatinous cut surface with focal fibrous areas. The tumour appeared non-encapsulated and infiltrative, consistent with the known biological behaviour of odontogenic myxoma (Figure 6).

Histopathological Examination: Microscopic examination of haematoxylin and eosin-stained sections revealed abundant loose myxoid connective tissue composed of spindle-shaped, stellate, and oval mesenchymal cells embedded within an extracellular matrix rich in mucopolysaccharides. Delicate collagen fibres and scattered thin-walled blood vessels were present. Occasional inactive odontogenic epithelial rests were identified. No cellular atypia, increased mitotic activity, or areas of necrosis were observed. These histopathological features confirmed the diagnosis of odontogenic myxoma.

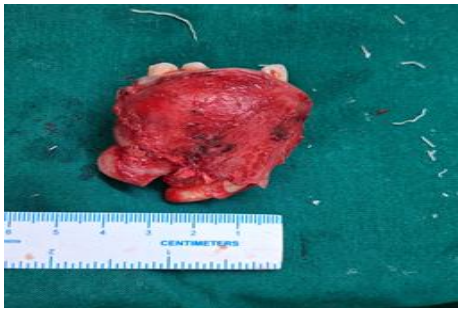

Figure 6: Lesion after excision

Postoperative Care: The postoperative period was uneventful. Intravenous antibiotics, analgesics, and anti-inflammatory medications were administered according to institutional protocol. Oral hygiene instructions were reinforced, and the patient was advised to maintain a soft diet during the initial healing period. The surgical obturator was evaluated regularly for stability, comfort, and function. The patient was instructed regarding its insertion, removal, cleaning, and maintenance. Functional rehabilitation, including speech and swallowing, improved progressively following adaptation to the prosthesis. Skin sutures were removed on the seventh postoperative day, and healing of the Weber–Fergusson incision was satisfactory, with acceptable facial aesthetics (Figure 7).

Follow-up: The patient was reviewed at 1 week, 1 month, 3 months, postoperatively. Clinical examination demonstrated satisfactory healing of the surgical site with good adaptation of the obturator and restoration of oral function. Serial radiographic evaluation revealed no evidence of residual or recurrent disease.

Figure 7: Post-operative view of the patient.

DISCUSSION

Odontogenic myxoma remains one of the least common odontogenic tumors but continues to represent a significant therapeutic challenge because of its infiltrative nature and tendency for recurrence.[7,9] Although biologically benign, the tumor lacks a true capsule, permitting microscopic extension into adjacent cancellous bone beyond the apparent radiographic margins.[10,11] The exact histogenesis of odontogenic myxoma remains controversial. Nevertheless, most investigators agree that the lesion originates from the ectomesenchymal components of the tooth-forming apparatus, including the dental papilla, dental follicle, and periodontal ligament, owing to its exclusive occurrence within tooth-bearing regions and the occasional presence of odontogenic epithelial rests.[6-8]

Clinically, odontogenic myxoma usually presents as a painless, slowly enlarging swelling causing facial asymmetry, cortical expansion, displacement of teeth, and occasionally root resorption.[9,13] Pain and paresthesia are uncommon unless secondary infection or neural compression develops.[18]

Radiographically, the lesion demonstrates variable appearances ranging from unilocular to multilocular radiolucencies with internal bony septa producing the classical "soap bubble," "honeycomb," "tennis racket," or "spider-web" appearance.[10,16] CBCT has become indispensable in evaluating cortical expansion, cortical perforation, sinus extension, and proximity to adjacent vital structures, thereby improving surgical planning.[16] The principal differential diagnoses include ameloblastoma, odontogenic fibroma, odontogenic keratocyst, central giant cell granuloma, aneurysmal bone cyst, central hemangioma, and fibrous dysplasia.[7,9] Microscopically, odontogenic myxoma is characterized by loosely arranged spindle-shaped, stellate, and angular cells suspended in abundant myxoid stroma rich in glycosaminoglycans, predominantly hyaluronic acid and chondroitin sulfate. Scattered collagen bundles and inactive odontogenic epithelial islands may also be observed, particularly in fibromyxoma variants.[7,17]

Several molecular studies have demonstrated increased expression of matrix metalloproteinases (MMP-2 and MMP-9), explaining the infiltrative behavior of the lesion.[19,20] These enzymes facilitate degradation of extracellular matrix components and may contribute to tumor invasion and recurrence. Management remains controversial because of the balance between minimizing recurrence and preserving function. Small lesions may be managed successfully by enucleation with peripheral curettage, whereas larger or recurrent lesions often require marginal or segmental resection with adequate bony margins.[11,12] Recent systematic reviews recommend individualized treatment based on tumor size, cortical perforation, anatomical location, and patient age. The reported recurrence rate varies from 10% to 30%, with most recurrences occurring within the first two postoperative years.[11,21] However, delayed recurrence has been documented several years after treatment, emphasizing the necessity for long-term clinical and radiographic follow-up extending beyond five years.[12]

Recent case reports published between 2022 and 2024 continue to support complete surgical excision with regular postoperative surveillance as the most predictable treatment protocol for maxillary odontogenic myxoma because of the difficulty in obtaining tumor-free margins in the maxilla.

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