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Oral Myxofibroid Lesions-Report of Two Cases Showing Two Ends of The Spectrum with Cytohistological Features

Dr Neelam Sood,
Senior consultant, India

Dr Neetu Yadav
Senior resident, Department of Pathology, Deen Dayal Upadhyay hospital, India

Dr Ruhi
Senior resident, Department of Pathology, Deen Dayal Upadhyay hospital, India

CORRESPONDING AUTHOR: Dr Neelam Sood, Senior Consultant, Department of Pathology, Deen Dayal Upadhyay hospital

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Abstract

Introduction: Oral myxofibroid lesions represent a histopathological spectrum of soft tissue tumors characterized by varying degrees of myxoid stroma and fibroblastic/myofibroblastic proliferation. Although rare in the oral cavity, understanding their spectrum is crucial for accurate diagnosis.

Case Summary

Case 1- A 37-year-old male presented with a growth in the anterior alveolus with a prior history of dental extraction. FNAC yielded few spindle shaped cells and scant fibrillary stroma. No epithelial cells were identified. Possibility of myxoid lesion was suggested. X-ray findings revealed a lytic lesion.The lesion was excised and histopathology was suggestive of odontogenic myxofibroma. Ki67 index was low.

Case 2- A 50-year-old male presented with ulcerated growth on left alveolus. FNAC yielded pink fibrillary stroma, plump spindle cells with eccentric nucleus, marked anisonucleosis,hyperchromasia and multinucleation.The possibility of a sarcomatoid lesion was rendered. X-ray findings revealed a lytic lesion in the left molar region extending into maxillary sinus.The lesion excised was suggestive of a Myxofibrosarcoma. IHC was negative for pancytokeratins and S100 with high Ki67 index.

Discussion: Myxofibroid lesions are prone to misinterpretation with ameloblastic tumors,neurogenic tumors and some sarcomatoid carcinomas. IHC markers such as pancytokeratins, S100, CD56, CD99, Ki67 aid in differentiation and grading.

Conclusion: The cytological features in myxofibroid lesions need to be carefully evaluated with clinicoradiological findings. Histopathological examination and ancillary studies are essential in reaching the final diagnosis.

Keywords: Myxofibroma, myxofibrosarcoma, odontogenic, mandible, IHC (immunohistochemistry), spindle cells

Introduction

Oral myxofibroid lesions represent a histopathological spectrum of soft tissue tumors characterized by varying degrees of myxoid stroma and fibroblastic/myofibroblastic proliferation. They originate from embryonic mesenchymal tissue.

Incidence of myxofibroma is estimated to be approximately 0.05 new cases per million individuals annually [1]. Whereas the incidence of myxofibrosarcoma is extremely rare and accounts for 1-3% of all the sarcomas [2].

Limited evidence is present in literature regarding the cytological diagnosis of oral myxofibroid lesions. We hereby report the cytomorphological features of two cases of oral myxofibroid lesions along with their clinical, radiological and histopathological features. The cases are being reported here because of their rarity and the diagnostic challenges due to their overlapping clinical and microscopic features with other soft tissue tumors.

Case Summary

Case 1 is a 37-year-old male who presented with a growth in the anterior lower alveolus measuring 4x3 cm with a prior history of dental extraction. X-ray findings revealed a lytic lesion in the mandible. FNAC yielded few spindle shaped cells and scant fibrillary stroma. A few plump cells? squamoid were seen. Possibility of low grade myxoid lesion was suggested. The lesion was excised and sections showed hyperplastic stratified squamous epithelium with underlying fibromyxoid background consisting of stellate cells, spindle cells admixed with fair number of lymphoplasmacytic infiltrates and mast cells. Bands of fibrosis were seen toward the deeper edge of lesion. Mitosis was negligible. No atypia was seen. On IHC, tiny epithelial islands showed positivity for CD56 suggestive of odontogenic epithelium. S100 was negative and Ki67 index was low. Final diagnosis of low grade odontogenic myxofibroma was made. Patient is on a regular followup and is doing well.

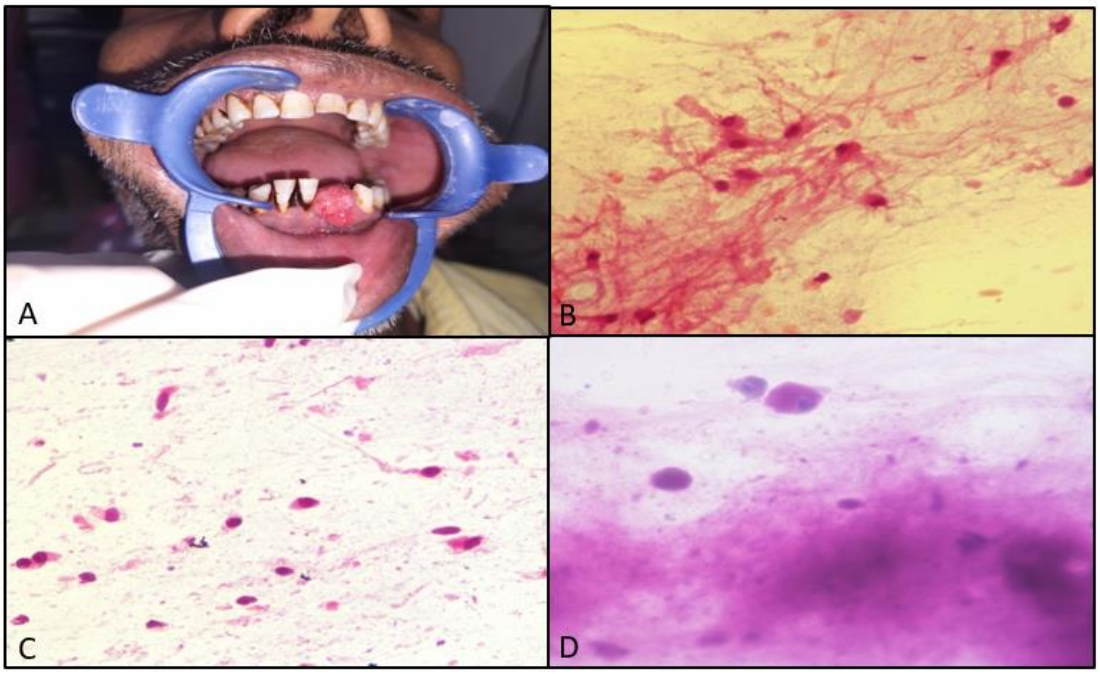

FIG 1:

A- growth in anterior lower alveolus;

B and C – scant fibrillary stroma with few spindle cells (10x, Giemsa stained);

D- Few plump cells? squamoid (40x, Giemsa stained)

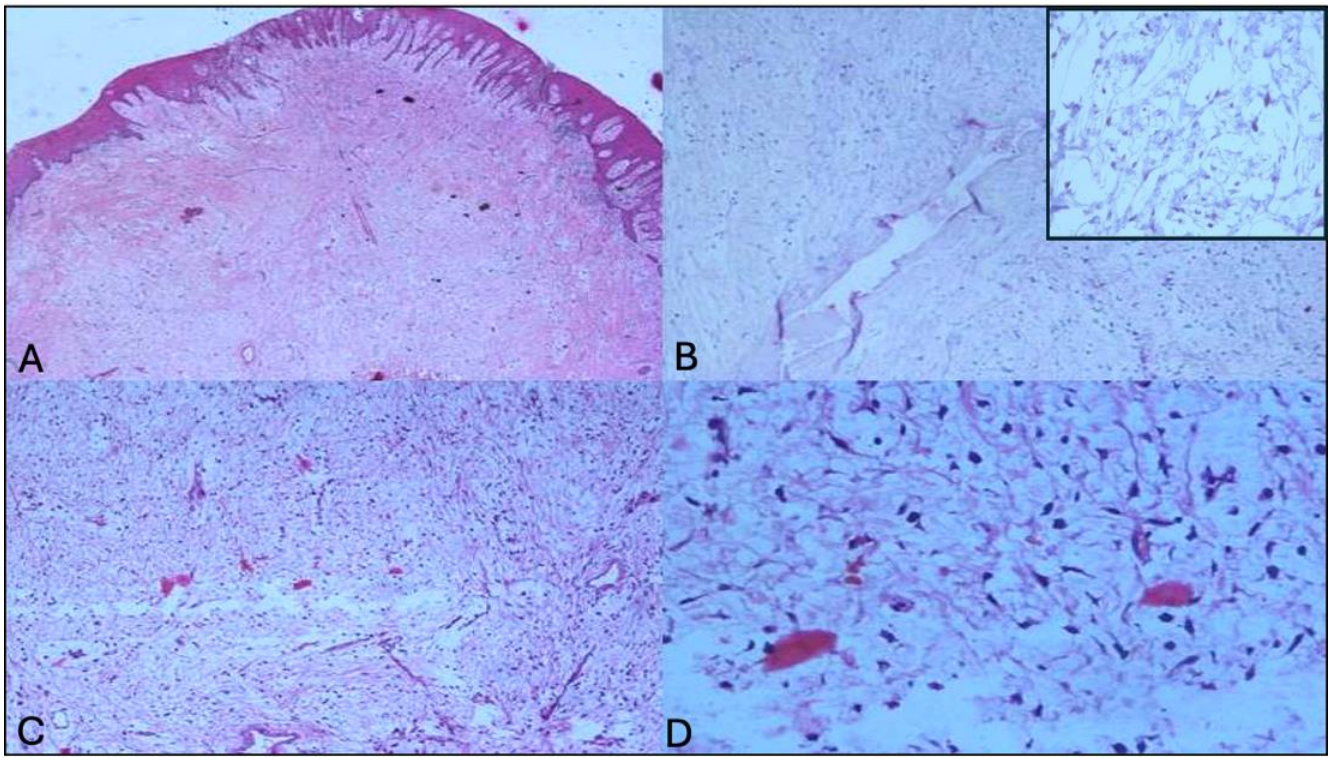

FIG 2:

A- hyperplastic stratified squamous epithelium with underlying fibromyxoid background (4x, H&E stained)

B- Spindle and stellate cells (10x, H&E stained) with inset showing low Ki67 index;

C- Bands of fibrosis towards deeper edge of lesion (10x,H&E stained);

D- Lymphoplasmacytic infiltrate and mast cells (40x, H&E stained).

Case 2 is a 50-year-old male who presented with a growth on left lower alveolus with soft tissue extension in the cheek. X-ray findings revealed a lytic lesion in the left mandible extending into TMJ. FNAC yielded moderately cellular smears showing fibromyxoid stroma, scattered and clustered plump spindle cells with eccentric nucleus, marked anisonucleosis, hyperchromasia and multinucleation. A few interspersed squamous cells and mixed inflammatory cells seen. Stretched out capillaries also seen. The possibility of a sarcomatoid lesion was rendered. The lesion was excised and revealed ulcerated overlying epithelium with underlying inflammatory infiltrates. Interlacing bundles and fascicles of spindle shaped cells showing moderate anisonucleosis and hyperchromasia were seen. Interspersed myxoid areas were seen. IHC was negative for pancytokeratins, SMA and S100 thus ruling out sarcomatoid carcinoma and other mesenchymal tumors. Ki67 index was high. It was suggestive of a Myxofibrosarcoma. Patient was referred to higher centre for further management.

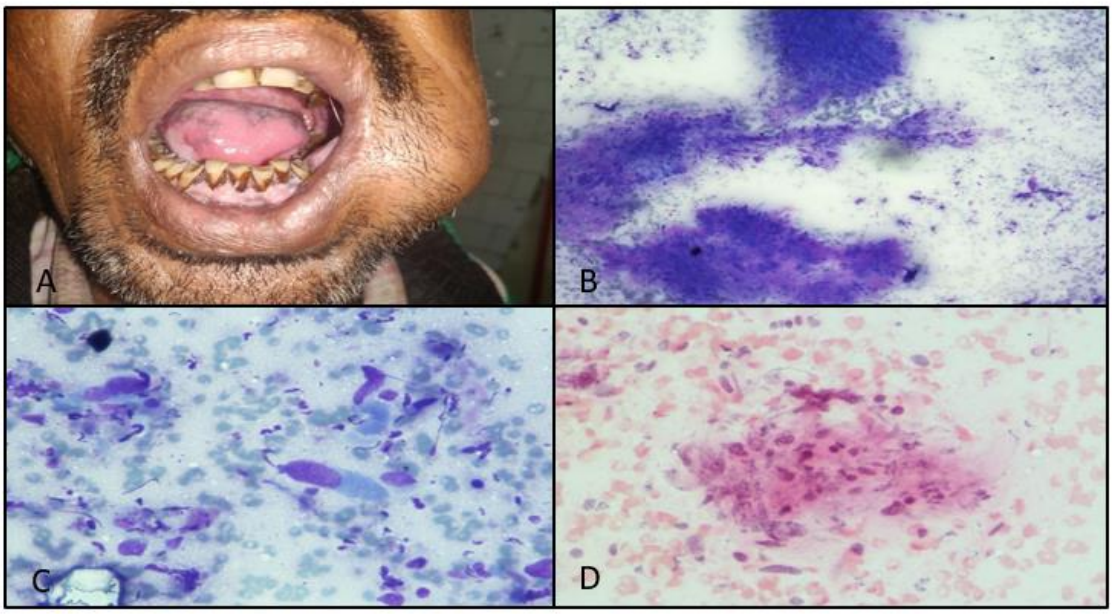

FIG 3:

A- growth on left lower alveolus extending into cheek

B-Cellular smears showing fibromyxoid stroma,clustered and scattered spindle cells (4x, Giemsa stained);

C- Spindle shaped cells with eccentric nucleus,hyperchromasia and multinucleation (40x, Giemsa stained);

D- Clustered and scattered spindle shaped cells (10x, H&E stained)

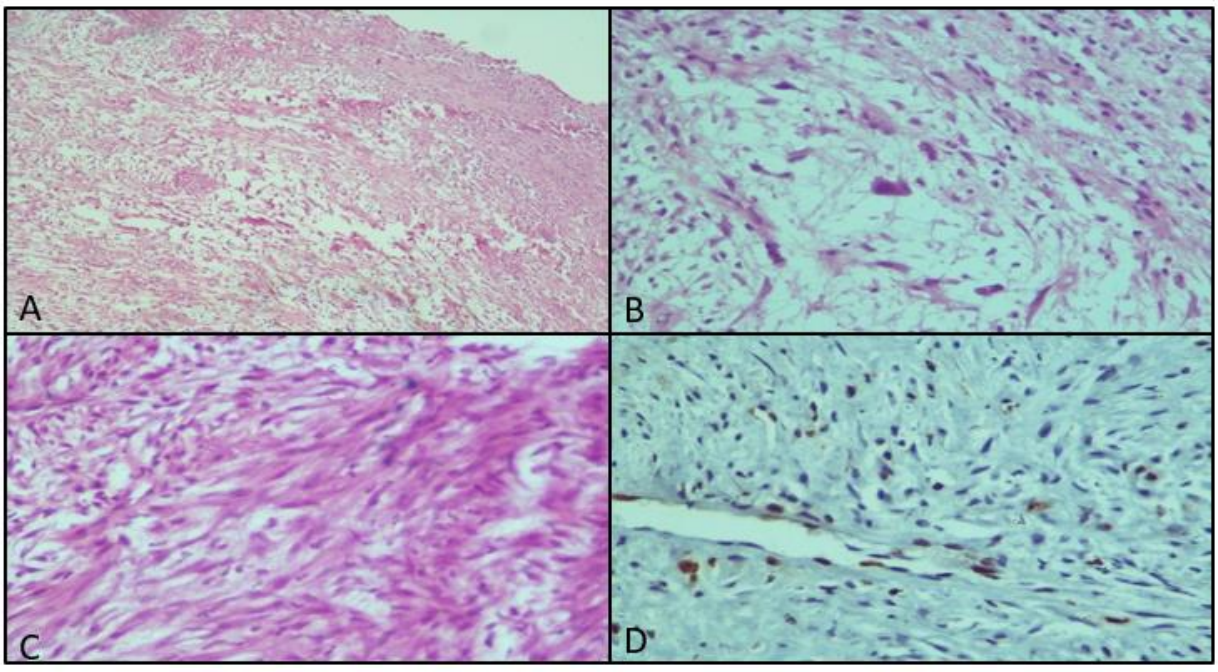

FIG 4:

A-Ulcerated overlying epithelium with underlying inflammatory cells (4x, H&E stained)

B-Interspersed myxoid areas (40x, H&E stained);

C- Fascicles and bundles of spindle cells showing anisonucleosis and hyperchromasia (40x, H&E stained);

D- IHC- high ki67 index

Discussion

Oral myxofibroid lesions represent a rare and heterogenous group of soft tissue neoplasms. In the present study, we included two cases- one benign and one malignant entity. According to literature, majority of the cases of myxofibroma are identified during the age range spanning from the second to fourth decade. The youngest patient to be recorded was 8 years old at the time of diagnosis [3]. Myxofibrosarcoma is more common in fifth to sixth decade. However, an occasional case has been reported in pediatric age group too[4]. Consistent with previous studies, the cases of myxofibroma and myxofibrosarcoma in our study were diagnosed in the third and the fifth decade of life respectively. Myxofibromas have female preponderance whereas myxofibrosarcoma show equal sex predilection [4].

Myxofibroid lesions rarely affects head and neck region. The mandible exhibits a higher incidence of involvement compared to maxilla. While mandibular involvement in our cases aligns with previous studies, the lesions were uniquely situated in the anterior region, differing from the more commonly reported posterior location.The majority of myxofibroma exhibit no symptoms, although a small number of patients have reported experiencing escalating pain due to the invasion of adjacent structures[5]. The clinical courses of myxofibrosarcoma vary significantly. Low grade myxofibrosarcoma often demonstrates expansive growth while high grade shows local invasion/compression of surrounding anatomical structures.

Histogenesis of myxofibroma is still a topic of debate to date. Although Thoma et al theorize that because the myxofibroma is derived from degeneration of a connective tissue tumor, myxofibroma is, in reality, an odontogenic fibroma that has undergone myxomatous degeneration. [6] Bruce and Royer, however postulated that it is possible for embryonic mesenchymal rests located in the area to proliferate and form a myxofibroma. [6] The appearance of synchronous or metachronous odontogenic myxofibromas, in patients with rare clinical entities which may be characterized by genetic predisposition, supports the notion of a possible genetic background underlying the manifestation of myxofibroma. In particular, MF with angiosarcoma, MF with tuberous sclerosis and MF with myasthenia gravis are reported in the literature. No such pathology was found in our study.

SM Williems et al hypothesized that myxofibrosarcoma has a highly complex karyotype and shows clonal and nonclonal aberrations, ring chromosome, translocations etc. [6]

Radiologically, MF presents as well-defined osteolytic lesion which shows multilocular radiolucency. Various other tumors/lesions (ameloblastoma. central giant cell granuloma, central hemangioma, odontogenic myxoma) also show multilocular radiolucency and thus should be differentiated clinically and microscopically.

MFS presents as an osteolytic lesion with ill-defined margins, lacking any internal structures as observed in our case.[7] Thus in absence of any other characteristic feature, it is difficult to distinguish it from other malignant osteolytic lesion including carcinoma and myeloma.

Preoperative diagnosis of jaw lesions is not always possible on the basis of clinicoradiological findings alone. FNAC being minimally invasive with rapid turnaround time and ability to provide preliminary cytological impressions makes it useful in guiding further management. In cases of oral myxofibroid lesions, FNAC may reveal characteristic features such as spindle shaped cells embedded in a myxoid background. However, due to absence of architectural details and potential for overlap with other myxoid and spindle cell tumors, FNAC has inherent limitations in providing a definitive diagnosis.Thus histopathological confirmation remains essential.

Since smears of our first case showed spindle cells, scant fibrillary stroma and only a few plump cells? squamoid, the possibility of myxoid lesion was suggested.

The microscopic differentials considered were hyperplastic myxoid dental follicle, ameloblastoma, myxoid nerve sheath tumors, cherubism and epulis. Histopathological features such as spindle and stellate cells in fibromyxoid background, with negligible mitosis and absence of cytological atypia, were instrumental in establishing the diagnosis, particularly when supported by immunohistochemical findings.

Hyperplastic dental follicle was ruled out by clinicoradiological findings and absence of peripheral bordering by odontoblasts. Ameloblastoma was a close

d/d however the characteristic ameloblastic epithelium depicts reversely polarized nuclei in the peripheral layer of tall columnar cells which was not observed in our case. Myxoid nerve sheath tumors were ruled out by absence of long wavy nuclei. As the lesion was unilateral, cherubism was ruled out. While epulis was a clinical differential, it was ruled out due to the presence of a lytic lesion on imaging, as epulis generally does not exhibit bony destruction. IHC analysis revealed CD56 positivity in tiny epithelial islands, negative staining for S100 and a low Ki67 proliferation index. Final diagnosis of low grade odontogenic myxofibroma was rendered.

In the second case,the smears showed fibromyxoid stroma and clustered plump cells showing atypia. Possibility of sarcomatoid lesion was given. Microscopic differential diagnosis included leiomyosarcoma, spindle cell carcinoma, malignant nerve sheath tumors and ameloblastic fibrosarcoma. [8] On histopathology, sections showed interlacing bundles and fascicles of spindle shaped cells showing anisonucleosis and hyperchromasia with interspersed myxoid areas. Spindle cell carcinoma and ameloblastic fibrosarcoma were ruled out by virtue of absence of any epithelial differentiation. IHC analysis showing vimentin positivity and negativity for S100, SMA and pancytokeratins helped to reach a conclusive diagnosis. Ki67 index was high and final diagnosis of myxofibrosarcoma was made.

Conclusion

The cytological features in oral myxofibroid lesions need to be carefully evaluated with clinicoradiological findings. Our study underscores the importance of a comprehensive diagnostic approach incorporating cytopathology, histopathology, and, when necessary, immunohistochemistry for establishing accurate diagnosis.

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