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# Gingival Vascular Hamartoma In Relation To Maxillary Primary First Molar Region Of Pediatric Patient: A Case Report

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***Abstract:***

*Oral hamartoma is a benign developmental malformation of tissue native to the oral cavity, most often detected in childhood, and managed effectively with surgery if needed. Gingival hamartomas are rare benign proliferations of disorganized local tissue. Few cases are reported in pediatric patients, particularly associated with primary teeth.*

*A 7 years-old male patient reported with a swelling which was well defined, reddish in colour and 8mm x 5mm when measured over the buccal mucosa extending from the mesial aspect of tooth number 53 up to mesial aspect of tooth number 55. Surgical excision was carried out and sent for histopathological examination. It revealed hamartomatous lesion predominantly vascular with pronounced secondary changes including ulceration, haemorrhage and infection. Head and neck hamartomas have shown that these benign entities may expand and recur, and chronic inflammation may heighten the risk of malignant transformation. Early diagnosis and management are needed.*

***Keywords:*** *Gingival Hamartoma, Pediatric Oral Lesions, Vascular Lesion.*

**Case report:**

A 7-year-old male patient presented to the Department of Pediatric and Preventive Dentistry with a chief complain by the parents as they noted a gingival growth over the upper right primary first molar, causing difficulty during brushing and mild bleeding.

The gingival hamartoma was characterized by a slow-growing, non-painful mass in the anterior maxillary gingiva. The lesion was firm, non-ulcerated, and exhibited a normal mucosal surface. Clinically, the patient appeared healthy, with no signs of systemic involvement. There was no significant family history of similar conditions or genetic disorders. The child's developmental milestones were age-appropriate, and there were no reported delays in speech or motor skills.

The patient's oral hygiene practices were adequate, with regular brushing and no history of trauma or previous dental procedures. Dietary habits included a balanced intake of food items, with occasional consumption of sugary snacks.

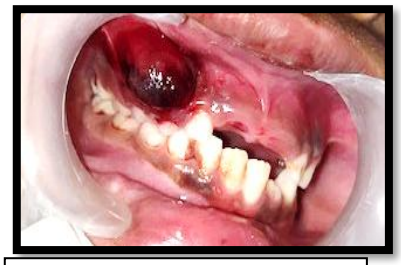
Clinically, the lesion appeared as a firm, non-tender, non-pulsatile mass with well-defined margins, measuring 8mm × 5mm on the buccal mucosa, extending from the mesial aspect of tooth number 53 to the mesial aspect of tooth number 55 (Table/Figure 1). The surface appeared to be smooth and coral pink, consistent with the surrounding gingival tissue. The lesion was non-compressible and did not exhibit mobility. There was no evidence of ulceration or bleeding upon probing. The involved tooth number 54, was grossly decayed and exhibited Grade II mobility. Radiographic examination revealed no associated bony involvement or resorption. Histopathological analysis confirmed the diagnosis of a gingival hamartoma, characterized by an overgrowth of normal gingival tissue components.

Radiovisiography (RVG) revealed dental caries invading the enamel and dentin, approaching the pulp of tooth number 54. Additionally, more than two-thirds of the distal root was resorbed. Based on the patient's clinical examination, a differential diagnosis of *Hamartoma, Pygogenic granuloma* and *Peripheral Giant Cell Granuloma* were considered. Surgical excision was performed, and the specimen was sent for histopathological examination (Table/Figure 2).

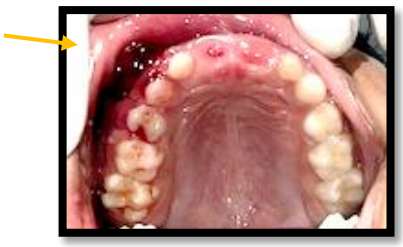
The histopathological report (Table/Figure 4) revealed ulcerated and inflamed keratinized stratified squamous mucosa, showing hyperplasia at the edge of the ulcer along with vascular structures in the epithelial and subepithelial tissue. Some bacterial colonies were also observed in the histological section. The lesion was identified as a predominantly vascular hamartomatous lesion with pronounced secondary changes, including ulceration, haemorrhage, and infection.

## INTRAORAL PICTURES:

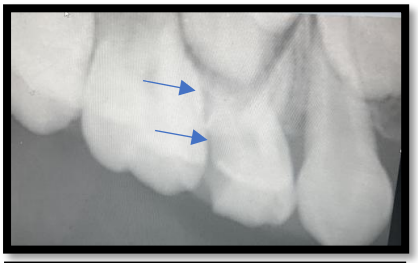
**Table/Figure 1 (Showing pre-operative intraoral pictures)**



**1 a. Maxillary Occlusal**

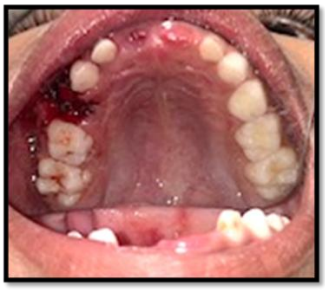


**1 b. Lesion with 8mm x 5mm**

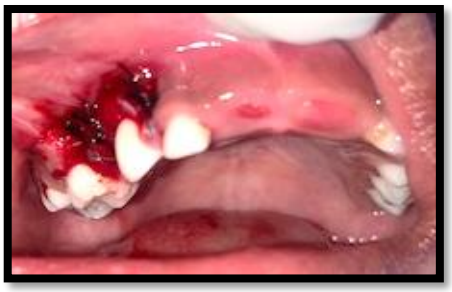


**1 c. RVG i.r.t. 54**

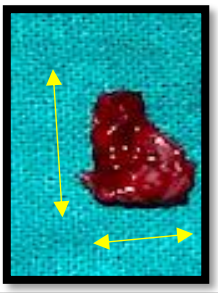
**Table/Figure 2- Immediate post-operative pictures**



**2 a. Maxillary Occlusal**

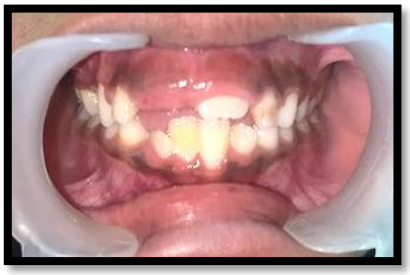


**2 b. Sutures Placed**

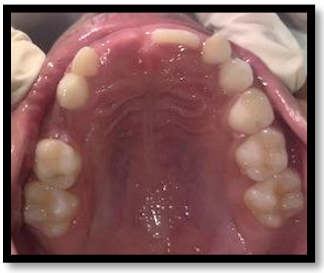


**2 c. 8 x 5 x 5 mm Excision Grayish brown soft tissue**

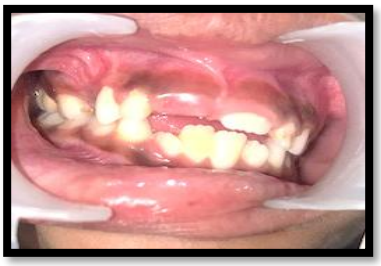
**Table/Figure 3 – 3 months post-operative pictures**



**3 a. Maxillary Occlusal View**

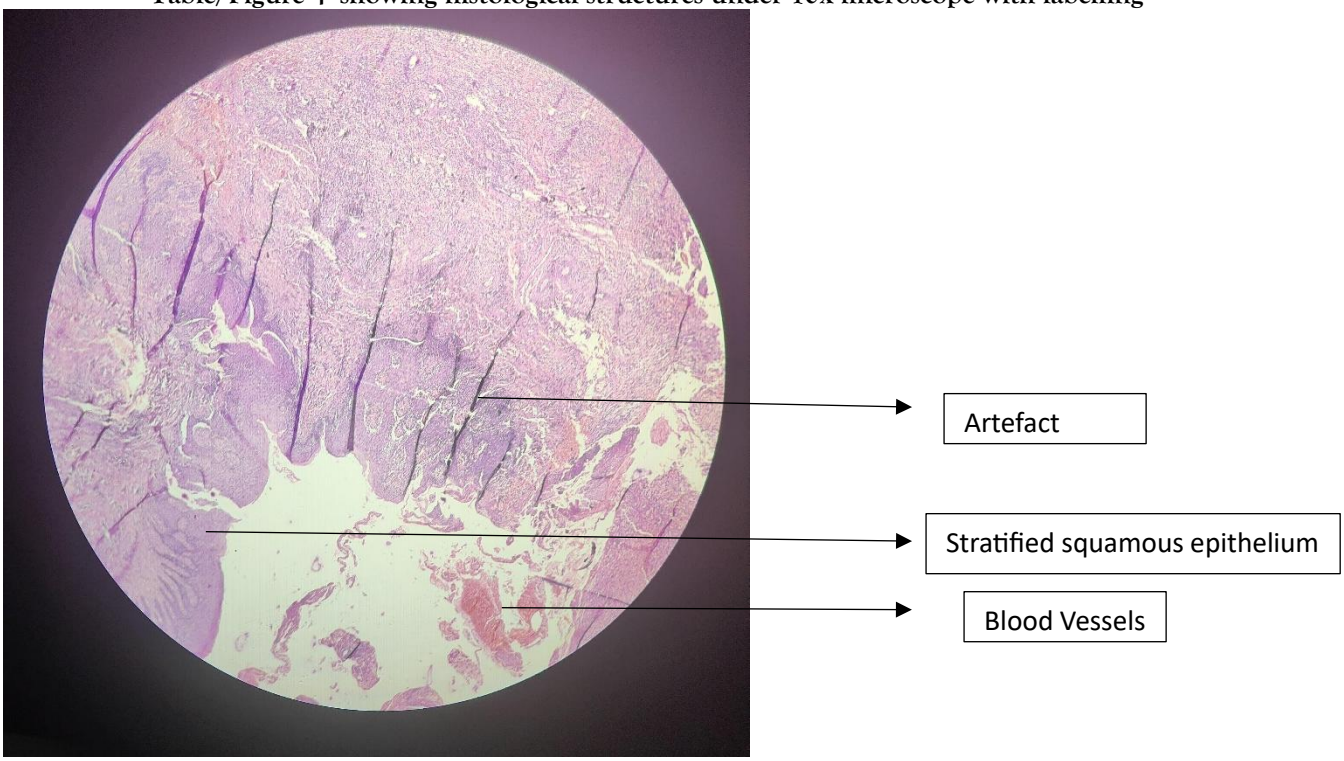


**3 b. Right Lateral View**



**3 c. Frontal view**

**Table/Figure 4- showing histological structures under 10x microscope with labelling**




**DISCUSSION:**

The word "hamartoma" originates from the Greek term "hamartia," meaning a flaw or mistake. It was first coined by Albrecht in 1904 to refer to tumor-like developmental abnormalities. Hamartomas are non-neoplastic and can be either unifocal or multifocal developmental anomalies composed of a combination of cytologically normal mature cells and tissues native to the anatomical site.[1] These structures exhibit a disorganized architectural arrangement, with one component typically being more predominant than the others.[2]

Tissues found naturally in the oral cavity that can play a role in the development of hamartomatous growths encompass both odontogenic and non-odontogenic epithelial derivatives, along with smooth and skeletal muscle, bone, blood vessels, nerves, and adipose tissue. According to existing literature, several features of a hamartoma include developmental anomalies that may be evident at birth but typically become apparent later, as well as growth that is self-limiting and synchronized with the surrounding tissues. [2,3] It may present as either a single mass or multiple masses and can resolve on its own. Generally, it lacks encapsulation and features indistinct borders. Although it is not classified as a true neoplasm, a neoplasm can develop within a hamartoma. On a microscopic level, it is composed of cytologically normal mature cells that are characteristic of the specific anatomical site and is linked to chromosomal abnormalities and various syndromes.[4]

The primary features of hamartomas include limited growth, possible progression after puberty, and microscopic examination revealing an unencapsulated aggregation of mature cells characteristic of the specific anatomical location, along with an association with chromosomal irregularities. [4,5] The precise mechanisms behind hamartoma formation remain largely speculative. These lesions arise from multiple embryonic origins, with the mesoderm being the most implicated. This contrasts sharply with neoplasms, which originate from a single cell clone.[5]

Hamartomas are predominantly located in the tongue and palate, with the maxillary gingival area being the next most common site, and they occur more frequently in females. These lesions are usually identified in individuals aged between two months and 19 years. Within the maxilla, the maxillary incisive papilla is the most affected area, and the lesions typically measure less than 1 cm. The literature indicates follow-up durations varying from six months to five years. Roy CF et al. (2023) [7] reported a case of a 20-day-old male patient with an ulcerated lesion on the hard palate, which gradually increased in size. Histopathological examination confirmed it to be a neurovascular hamartoma of the hard palate. Berberi A (2023) [8] described a gingival overgrowth on the maxillary gingiva, which exhibited an unusual endovascular papillary aggregate of endothelial cells, with a final histological diagnosis of gingival vascular hamartoma. These lesions are considered developmental anomalies—**hamartomas—**

rather than true neoplasms, driven by localized overgrowth of capillary or vascular channels. Proposed mechanisms include: Placental endothelial cell emboli lodging in foetal tissues acting as progenitor sources, Genetic mutations (e.g. loss-of-function in chromosome 5q) causing constitutive angiogenesis, Upregulation of angiogenic factors like VEGF, bFGF, and GLUT-1 in response to local hypoxia.[8] These lesions typically progress through three phases:

1. Proliferative phase: rapid endothelial cell proliferation driven by VEGF, bFGF, and possibly oestrogen receptors.[9]
2. Rapid growth phase: endothelial cell enlargement and lesion expansion.[10]
3. Involution phase: endothelial cells regress and are replaced by fibro-fatty tissue. [10]

Surgical excision was considered the appropriate treatment of choice for our patient.[11] The patient is currently under follow-up, is in good health, and has no complaints of pain (Table/Figure 3a,3b,3c). He has been advised to return for regular check-ups every three months to monitor for any recurrence. However, due to financial constraints, the patient's parents could not afford further treatment and declined the recommendation for a band and loop space maintainer for the 55-region following extraction.[12]

## CONCLUSION

Gingival hamartoma was successfully excised under local anaesthesia without complications. Histopathology confirmed a benign vascular (capillary) hamartoma—consistent with previous case reports in children. The lesion was completely removed with clear margins, and post-operative healing was uneventful—no excessive bleeding or recurrence at follow-up.

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