

# Etiology and Treatment of Oral Haemangiomas (OHs)

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## Description

Haemangiomas are tumours that can be recognised by their early-infancy fast endothelial cell proliferation followed by involution; all other defects are deformities brought on by the aberrant development of vascular plexuses. The abnormalities do not involute and impact the veins, capillaries, or lymphatics with a normal endothelial cell development cycle. According to one theory, Oral Haemangiomas (OHs) are caused by embolic placental endothelium cells, which act as stem cells and enter the lethal circulation after trauma or another cellular stressor. This rationale is in line with research showing an elevated risk of haemangioma following chorionic villus sampling. Another hypothesis is that haemangiomas develop as a result of constitutively activated angiogenesis caused by spontaneous or inherited loss-of-function mutations on chromosome 5q. According to a third theory, the synthesis of VEGF and GLUT1 by adjacent cells in response to hypoxic stressors is upregulated, which leads to the development of haemangiomas.

Hemangiomas, a general term for the more frequent vascular malformations of the oral cavity, are proliferating capillaries or venules that do not involute but rather remain and expand gradually over time. They typically develop on the labial mucosa, buccal mucosa, and tongue. The best description of the intrabony lesions is “vascular malformations.” A unique type of vascular malformation called an arteriovenous haemangioma, with direct blood flow from the venous to the arterial system and bypassing the capillary beds, is most likely the cause of a lesion that thrills, bruits, or has a clearly heated surface. Osler-Weber-Rendu syndrome, Sturge-Weber syndrome, and blue rubber bleb nevus syndrome are a few of the disorders connected to vascular malformation. This disorder can have a differential diagnosis of granuloma fasciale, insect bite, pyogenic granuloma, and angiosarcoma. On the mucosa of the tongue, capillary hemangiomas are a rare occurrence and are easily mistaken for other lesions like malignant tumours. Early diagnosis and biopsy are required to ascertain the clinical behaviour of the tumour and probable dentoalveolar consequences.

Despite being a relatively uncommon tumour of the oral cavity, capillary haemangiomas are significant to periodontists due to complications such as impaired nutrition and oral hygiene, increased plaque and microorganism accumulation, and increased susceptibility to oral infections that can harm

the affected person’s overall health. It is uncommon for cavernous haemangiomas to develop on the tongue. Early diagnosis and biopsy are essential in order to ascertain the clinical behaviour of the tumour and any potential consequences. The mode of treatment should be chosen in accordance with the diagnostic and outlook for the specific vascular abnormality. Hemangiomas can appear clinically, radiographically, and histopathologically similar to other lesions. Pyogenic granuloma, chronic inflammatory gingival hyperplasia (epulis), epulis granulomatosa, varicocell, talengetastia, and even squamous cell cancer are all included in the differential diagnosis of hemangiomas.

The pyogenic granuloma is the most typical vascular growth of the oral mucosa. It is a reactive lesion that grows quickly, bleeds easily, and frequently has ulceration and inflammation present. It may be hormone sensitive and is frequently lobulated, pedunculated, and red to purple clinically. Propranolol is one of the most commonly prescribed beta-blockers for treating OHs. Although the exact process is unknown, it has been proposed that it involves endothelial cell death and local vasoconstriction. Patients normally start out on a regimen of propranolol of 2 milligram-3 milligram per kilogram per day divided into three doses, and after 1-2 days of starting therapy, the appearance of the OH usually improves. Patients should be watched for side effects such as bradycardia, hypotension, hypoglycaemia, and bronchospasm during the course of beta-blocker therapy, which could last up to 6 months.

## Conclusion

Complications associated with OHs depend on the size and location of the lesion may include: Ulceration (the most common complication), hemorrhage, dysphagia and failure to thrive, speech impairment, airway compromise. Children are often the ones who are diagnosed with OHs, and most of these lesions will involute with time. In addition to scheduling routine follow-up consultations for monitoring, primary doctors play a crucial role in reassuring patients and their parents about the benign nature of OHs. Pharmacists will be useful in educating about drug side effects if medical therapy is started. Interventional radiologists or oral and maxillofacial surgeons, as appropriate, will be involved to evaluate patients as surgical candidates, present information on the risks and advantages of these procedures, and provide post-procedural monitoring and education if sclerotherapy or surgical resection is necessary.