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THE VASCULAR MYSTERY: PEDUNCULATED CAPILLARY HEMANGIOMA IN AN ADULT MAN

Oral Pathology

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ABSTRACT

Vascular malformations are rare anomalies arising from abnormal development of vascular structures, often presenting significant diagnostic and therapeutic challenges. This case report highlights the case of a forty seven year old male patient presented with a pedunculated swelling in the lower lip, which on histopathologically evaluation turned out to be a capillary hemangioma.

KEYWORDS

Hemangioma, Pedunculated

INTRODUCTION

Hemangioma refers to a diverse collection of benign vascular tumors with comparable histologic characteristics. It is often a proliferating mass of blood vessels that does not exhibit a malignant change. While some cases are congenital, the majority occur during childhood. Older people are occasionally affected[1].They can range in size from a few millimetres to several centimetres, are sessile or pedunculated, and manifest clinically as a smooth or lobulated soft mass[2]. To the best of our knowledge, pedunculated hemangioma of lower lip has never been documented.

Case Report

A 47-year-old male was presented to the outpatient department with a swelling in the lower lip, since six months.

The patient was a known case of coronary artery disease and had a cerebrovascular accident, 2 years priorly and is currently under medication for the same.

Oral examination revealed a solitary nodular lesion which is pedunculated, and about 1x1.5cm in size. The overlying mucosa had a reddish hue. On palpation, there was no thrill or tenderness or fluctuancy

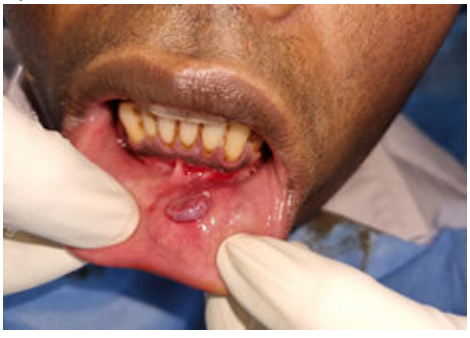

Figure 1: Intraoral photograph of patient showing solitary nodular swelling of the lower lip which was pedunculated.

The clinical appearance, led to a provisional differential diagnosis of an irritational fibroma, fibrosed mucocele or a pyogenic granuloma. The lesion was excised and sent for histopathological examination

Microscopy of the H&E-stained soft tissue bit revealed a connective tissue stroma with intense diffuse proliferation of endothelial cells, covered by a parakeratinised stratified squamous epithelium, which was hyperplastic at one end and atrophic towards the other end. Vascularity was intense with numerous formed, engorged and budding capillaries. (Figure 2). The immunohistochemical staining with CD34 revealed positivity for endothelial cells (Figure 3).

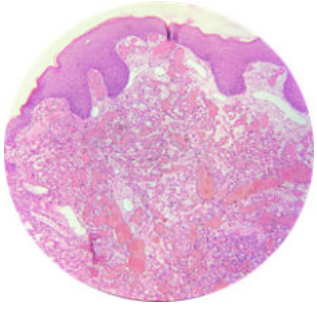

Figure 2; Photomicrograph shows stroma with numerous budding, formed and engorged capillaries(H & E Stain, x 100)

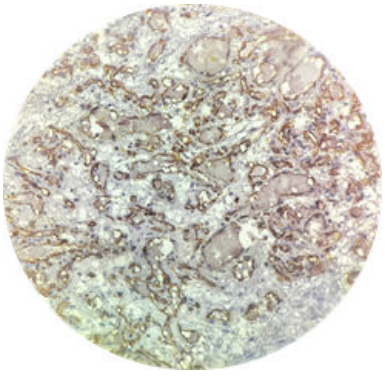

Figure 3: Photomicrograph shows immunohistochemical positivity of endothelial cells for CD34(IHC CD34, x 400)

Depending upon microscopic findings, we came to the diagnosis of Capillary hemangioma. The patient was put under follow up since then.

DISCUSSION

Hemangiomas are tumours that arise from the proliferation of endothelial cells and nearly always affect the head and neck area, in 70% of cases.[3] Clinically, haemangiomas can range in size from a few millimetres to several centimetres, present as a smooth or lobulated

soft mass, and be sessile or pedunculated. Oral cavity pedunculated haemangiomas are incredibly uncommon.[2] Clinically, there are two types of haemangiomas: congenital and infantile. While congenital haemangiomas are fully developed at birth and come in different varieties like—rapidly involuting congenital angioma (RICH), noninvoluting congenital haemangioma (NICH) and partially involuting congenital hemangioma. Whereas, infantile haemangiomas usually develop in the first two months of life, grow quickly until six months of age, and then experience a period of regression. [3,4]

There is a lack of clarity regarding the pathophysiology and origin of hemangiomas[2], the pathogenesis is influenced by abnormal amounts of proangiogenic factors (VEGF, b-FGF, and TGF-beta 1) and matrix metalloproteinases (MMP-9). Hemangioma development is also impacted by genetic abnormalities in growth factor receptors[4].

The pathogenesis of adult capillary hemangioma could be, as proposed by Boye et al., which highlighted the endothelial cells as the primary abnormal component. There was one unexpected finding that, in the presence of the angiogenic inhibitors, endothelial cell migration is promoted rather than prevented, which indicated a drastically changed cellular phenotype in individuals with exposure to such medications. According to their theory, the endothelial cells in these lesions exhibit increased migratory and proliferative capacities, which distinguish them from normal endothelial behavior. This suggests an intrinsic abnormality within the endothelial compartment, driving the growth and persistence of the hemangioma [6]

Berg and colleagues provided evidence for somatic mutations in hemangiomas by demonstrating that chromosome 5q (17) has a high prevalence of loss of heterozygosity (LOH) in hemangioma tissue, indicating that loss-of-function mutations also play a role in the development of haemangiomas[6]

Hematopoietic progenitor cells (from the placenta or stem cells) in the right environment of genetic changes and cytokines are most likely the source of infantile hemangiomas.[4]

Histologically, there are three forms of head and neck hemangiomas: capillary, cavernous, and mixed. Compared to other forms, capillary hemangiomas are more prevalent[3].

The proliferative and involutive phases of hemangiomas vary according to the tumor's stage of life. Endothelial cells are proliferating in the proliferative stage. The proliferative endothelial cells are active showing hypertrophy and a pale stained nucleus, occasionally displaying mitotic figures. There are far more mast cells than in normal tissue. Whereas, flattening of the lining endothelial cells and reduced cellularity are characteristics of the involuting phase haemangioma. As in the presenting case, the vessels supplying the tumor shows dilatation and progressive deposition of perivascular, intralobular, and interlobular fibrous tissue. The number of mast cells returns to normal. The structure of a complete involuted haemangioma is "sponge-like," with flat endothelial cells lining sporadic blood vessels with thin walls. The number of mast cells recovers to normal[7].

Immunohistochemically, GLUT1 expression discriminates infantile hemangioma from other benign vascular lesions. Vascular markers CD31, CD34, ERG and SMA, indicate endothelial cells and pericytes, respectively[8].

Diascopy, ultrasonography are commonly preferred for evaluating intraoral capillary hemangiomas. In cases where deeper tissue involvement is suspected, contrast-enhanced MRI is recommended.[9,10]

Numerous therapeutic approaches have been employed to reduce hemangioma growth and hasten its remission. Proliferative hemangiomas are treated with systemic drugs like oral propranolol, prednisone, topical imiquimod, and subcutaneous interferon α-2a or 2b.[7] Sodium tetradecyl sulphate, hypertonic glucose solution, and sodium sclerotherapy are among the treatment choices for minor and peripheral lesions, depending on their size and location. Radiation, corticosteroid therapy, electrocoagulation, laser therapy, cryotherapy, and surgical excision are further choices [3,10].

CONCLUSION

Vascular lesions like hemangiomas can be considered in the differential diagnosis of lip masses, even in adults, as they can occasionally persist or present de novo. This case highlight the importance of detailed clinical evaluation and imaging for atypical presentations of vascular lesions. Histopathological confirmation is necessary for accurate diagnosis and tailored management.

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