



Intraoral Vascular Malformation of Right Cheek: A Case Report

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ABSTRACT

This is a case report of an adult untreated vascular malformation of the cheek, involving the masseter muscle, which underwent recent enlargement. After consideration of different treatment options, intraoral excision of the malformation was done including intramuscular dissection. There was no recurrence on short term follow up.

Introduction

Vascular malformations (VM) are characterized by the abnormal growth of blood vessels that can in turn lead to abnormally enlarged arteries, veins, or abnormal communication between arteries and veins. They are populated by stable mature vascular endothelium. Both males and females are equally affected. VMs are usually present at birth and, unlike hemangiomas, never involute. Instead, they increase in size during the growth of the individual. This growth can be accelerated by trauma, puberty and pregnancy.¹ Mullikin and Glowacki in 1982 reclassified vascular lesions based on the endothelial characteristics into haemangiomas and VM. The classification was later modified in 1996 by International Society for the Study of Vascular Anomalies as vascular tumors or VM. VMs are further classified based on the blood flow into low-flow lesions and high-flow lesions, arteriovenous malformations (AVMs) and arteriovenous fistulae.

Case Report

A 25-year-old female patient presented with swelling of the right cheek for one year and six months. The swelling was initially the size of a small pebble, growing to its present size (**Fig 1**). The growth was asymptomatic but the patient was concerned about the appearance and nature of the “tumor”. The patient did not give any relevant past medical or dental history. Extraoral soft tissue examination revealed a solitary swelling seen in the right cheek region measuring approximately 5x4cm, extending anterior-posteriorly 2cm from the ala of the nose and 3cm in front of the tragus of the ear and superior-inferiorly from the malar region to the 3cm above the inferior border of the mandible (**Fig 2**). The mucosa overlying the intraoral swelling was normal. On palpation the swelling was soft, fluctuant, non-tender and not adherent to the skin. Based on clinical signs and symptoms a provisional diagnosis of vascular malformation of the right cheek region was made. Hemangioma, lymphatic malformation and

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lipoma were considered as differential diagnosis. An ultrasonographic examination reported it as a heterogenous hypoechoic lesion medial to the parotid gland with minimal vascularity (**Fig 3**). Considering the size of lesion and age of patient we planned for excisional biopsy under general anaesthesia. Through an intraoral vertical oblique incision on the right cheek, avoiding the opening of the parotid duct, the malformation was carefully dissected out under loupe magnification, using bipolar electrocautery. The malformation was dissected off the subcutaneous tissues of the cheek, carefully preserving the terminal facial nerve branches and splitting the masseter muscle to excise the base of the VM (**Fig 4**). The excised specimen measured approximately 4.5x4cm in size, as shown in **Fig 5**. It was sent to the department of pathology for histopathological analysis. The result indicated multiple blood vessels with thrombus-like structures interspersed in the stroma with endothelial proliferation (**Fig 6,7**). The patient was recalled for routine follow up examination and did not show any sign of recurrence at three and six-months after surgery (**Fig 8**).

Discussion

Vascular malformations are more often seen in the skin, most commonly occur in the head and neck region (skull and the maxillo-facial region)³. The clinical manifestations of the VM in the mid-cheek region include a solitary lesion, which is moderate to hard in consistency, mobile with clear boundaries, exhibits no tenderness, and has a long course of disease exceeding a mean time period of 2 years⁴. Exact etiology and pathogenesis of AVM is not known. Arteriovenous malformations (AVMs) are rare congenital vascular lesions occurring anywhere in the body. They have normal rate of endothelial cell turnover, which are present at birth but usually become noticeable in the later age. Rapid enlargement of the malformations is usually triggered by trauma or hormonal changes during puberty or pregnancy. The enlargement of these lesions is due to the change in pressure and flow, dilatation of vascular channels, shunting and collateral proliferation rather than cellular proliferation⁵. Based on blood flow characteristics, vascular malformations can be divided into (i) low-flow lesions and (ii) high-flow lesions. Low-flow lesions include capillary, lymphatic, and venous malformations whereas high flow includes arterial and AVMs⁶. AVMs are rarely seen, only accounting for 1.5% of all vascular anomalies, and 50% of the lesions are located in the oral and maxillofacial region⁷. These lesions can be diagnosed by plain radiography, computed tomography scans, magnetic resonance imaging, or angiography. Magnetic resonance imaging (MRI) has become the investigation of choice since it depicts the extent and lack of invasion of these lesions. Angiography is useful in poorly defined cases and

for embolization before surgery. It demonstrates the flow characteristics, feeding vessels, and dangerous anastomoses. These lesions present as a pulsatile mass⁸ with a thrill, bruit, and occasionally local hyperthermia, ulceration or bleeding, functional impairment due to arterial steal, and ischaemia. Shunting of blood diminishes nutritive flow, which may result in skin necrosis, ulceration, and bleeding. Many lesions have either a warm erythematous blush or a true port-wine stain in the overlying skin. Vascular malformations may be associated with underlying disease or systemic anomalies in select situations. Most commonly occur on anterior two-thirds of the tongue, palate, and gingival and buccal mucosa. These can be life threatening due to potential massive hemorrhage. Based on endothelial characteristics, Mulliken and Glowacki¹ (1982) classified vascular lesions into hemangioma –vascular tumor and vascular malformations. According to the imaging features of the draining veins, venous malformation is divided into four types, Type I, isolated malformation without venous drainage; Type II, malformation with drainage into normal veins; Type III, malformation with drainage into dilated veins; and Type IV, dysplastic venous ectasia. This proposed classification scheme is helpful for selection of sclerosing agents. For type I and type II lesions, mild sclerosants such as pingyangmycin should be considered first; for type III and type IV lesions, strong and aggressive sclerosants such as ethanol is more suitable due to the fast vein drainage⁸. Localized or limited venous malformations can be removed surgically. For large lesions, partial excision can be considered after sclerotherapy to improve appearance. Some large venous malformations may be resectable if the MRI reveals a definitive border with only low draining veins. Before operating a large VM, MRI should be performed to define the extent of the lesion and venous drainage. Blood transfusion should be prepared because blood loss can be significant. In cases with extensive tissue defects caused by surgical removal, a skin graft or flap should be transplanted for reconstruction. For patients with large tongue lesions, surgery can first be done to reduce the size of the tongue, followed by a shortened course of sclerotherapy. After treatment of large venous malformation with absolute ethanol sclerotherapy, patients may have fibro-fatty tissue remnants, which may necessitate surgical correction². During surgery, laser treatment can also be used to remove the residual lesions.

Sclerosing agents are substances that cause a marked tissue irritation or thrombosis with subsequent local inflammation and tissue necrosis resulting in fibrosis and tissue contraction⁵. It is important for the dental practitioner to be aware of VM which may be present in the head and neck region that can produce fatal bleeding episodes during various dental procedures⁴. Preoperative sclerotherapy leads to occlusion of malformed veins and reduces blood loss during surgery¹⁰. However, fibrosis and scarring can give rise to more difficulties in identifying important nerve and blood

vessels, which should be fully considered and estimated by the surgeons before operation.

Conclusion

Proper diagnosis of VMs through complete history, thorough clinical examination, and advanced imaging modalities can help in preventing serious life-threatening complications. The primary treatment of this lesion is excision. In this case report the prognosis of such treatment was good with no recurrence reported. It is important to remember though that the treatment is helpful in the long term to control the growth and symptoms with regular follow up.

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Fig 1: Pre-operative view



Fig 2: Pre-operative worm's eye view



Fig 3: Ultrasonography of Left Cheek.



Fig 4: Intra-operative view during excision of the lesion

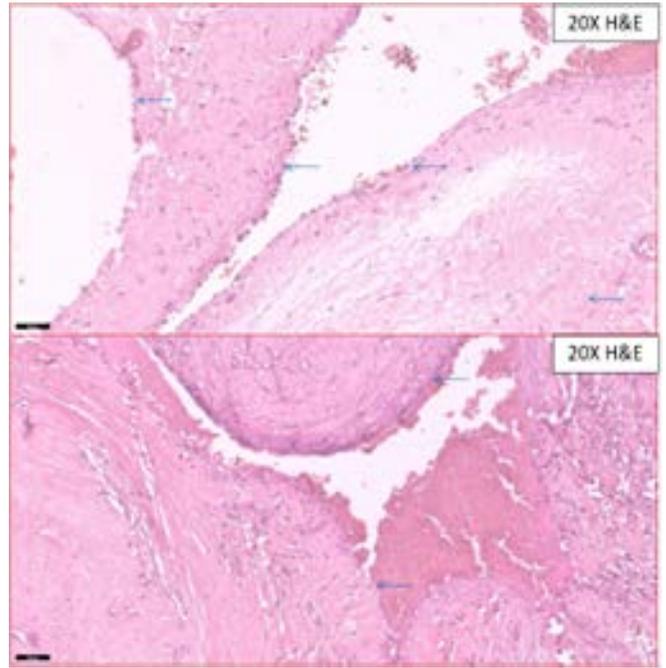


Fig.7: Histology of the excised specimen- High power magnification

1.Blue arrow - Lining endothelium



Fig 5: Excised specimen

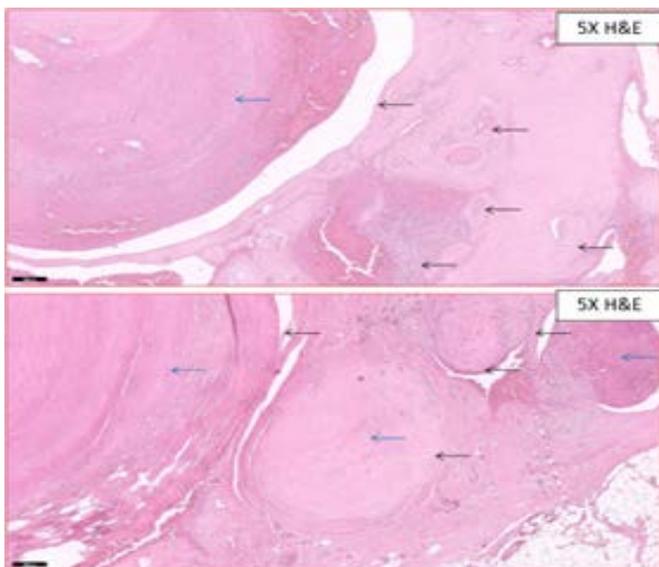


Fig.6: Histology of the excised specimen-Low power magnification

1.Black arrow - Blood vessels 2. Blue arrow - Thrombosis



Fig 8: 3 months follow up intraoral clinical photograph