



CASE REPORT

LOW FLOW VENOUS MALFORMATION OF THE BUCCAL MUCOSA TREATED BY SURGICAL EXCISION - A RARE CASE REPORT

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ABSTRACT

Introduction: Low flow venous malformations are characteristic lesions with unique presenting features. The most common sites of occurrence are the cheek and the ear in the midface region. Most centres recommend sclerotherapy as the first line of treatment for low flow vascular malformations. We report a case of low flow vascular malformation in the buccal mucosa treated successfully by wide excision and reconstructed with buccal pad of fat.

Case report: A 26 year old patient reported to our hospital with a painless swelling in the buccal mucosa. Under IV sedation patient underwent angiography via the femoral artery which showed no abnormal feeding vessels or draining veins. The lesion was treated by wide excision including the surrounding nidi, which was followed by reconstruction with buccal fat pad under general anaesthesia. The patient was followed up regularly and there was good take at recipient site and also there was no evidence of recurrence.

Conclusion: Low flow venous malformations can be managed by surgical excision alone if there is no feeder vessel or very minimal uptake of the dye by a feeder vessel with the combined effects of a surgeon and vascular radiologist.

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INTRODUCTION

Low flow venous malformations falling under slow flow vascular malformations occur due to defective vascular development and they do not regress spontaneously and often cause symptoms. Intramucosal injection with sclerosing agents is now considered to be the first line of invasive therapy. Clinical evaluation includes a medical history, physical examination and imaging and angiography to locate the feeder vessel (Burrowset *al.*, 1988; McCaffery and Jones, 2011). Fast flow vascular malformations can be differentiated from slow flow vascular malformations by the presence of increased ipsilateral pulses, palpable pulsations or thrill, and increased skin or mucosal temperatures which are absent in slow low flow vascular malformations. The size of this malformation is increased as blood is pooled from the normal circulation through a low resistance system created by the nidus, the aberrant arteriovenous network at the centre (Richter *et al.*, 2007). We report an uncommon case of one in the buccal mucosa with atypical features and negligible intraoperative bleeding during excision followed by reconstruction with buccal fat pad.

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The patient underwent femoral angiography for locating the feeder vessel. All the vessels in the head and neck region were checked (facial, internal, external carotid and vertebral arteries)

CASE REPORT

A 26 year old patient reported to our hospital with a painless swelling in the buccal mucosa which was present for time duration of one year. The colour of the mucosa was reddish extending anteriorly 1cm from the corner of the mouth and posteriorly extending 1cm from the pterygomandibular raphe. Superiorly the extent was for about 1 cm from the buccal vestibule (Figure 1). The swelling was soft but the contour was not smooth showing multiple demarcations. No thrill was palpated over the swelling. The swelling showed no changes in size during mouth opening. Under IV sedation patient underwent angiography via the femoral artery. The angiogram revealed no abnormal feeding vessels or draining veins (Figure 2). Faint opacification was seen in the venous phase in the region of the swelling. In view of no prominent feeder vessels, embolization was deferred. The lesion was treated by wide excision including the surrounding nidi (Figure 3), which was followed by reconstruction with buccal fat pad under general anaesthesia (Figure 4). Very minimal bleeding was note during the excision.

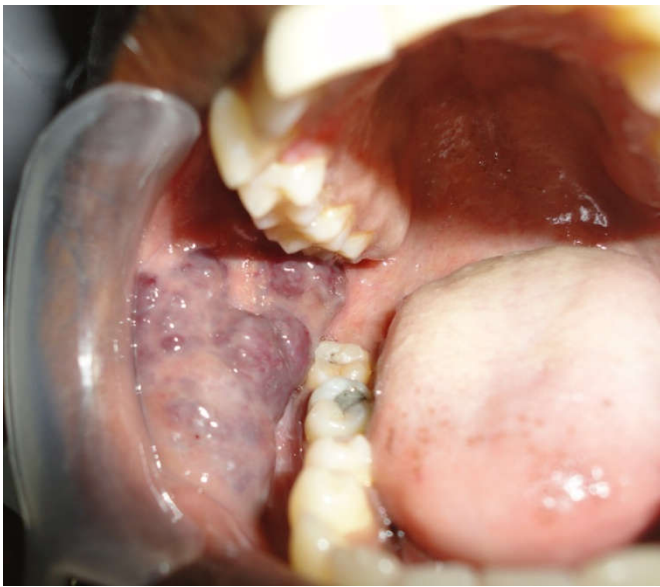


Figure 1. Preoperative intraoral lesion



Figure 2. Angiography showing no feeder vessels

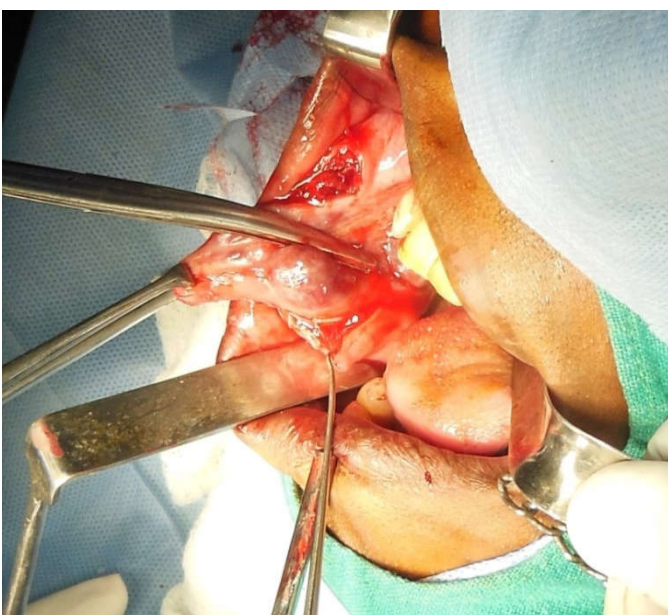


Figure 3. Surgical Excision

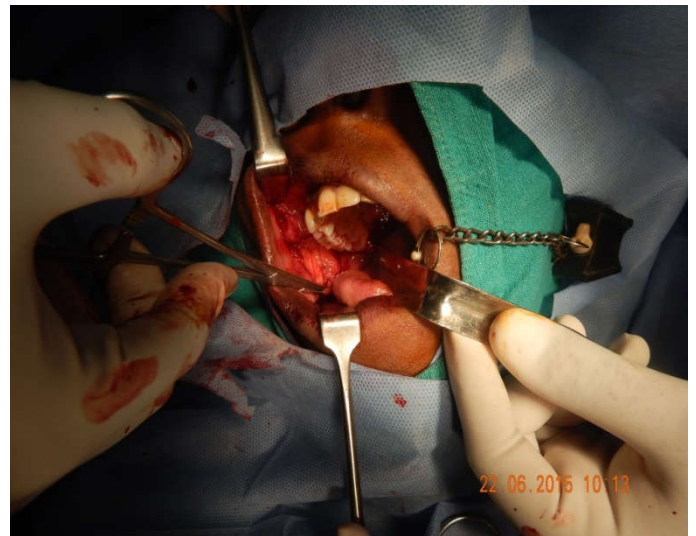


Figure 4. Buccal fat pad mobilized closure

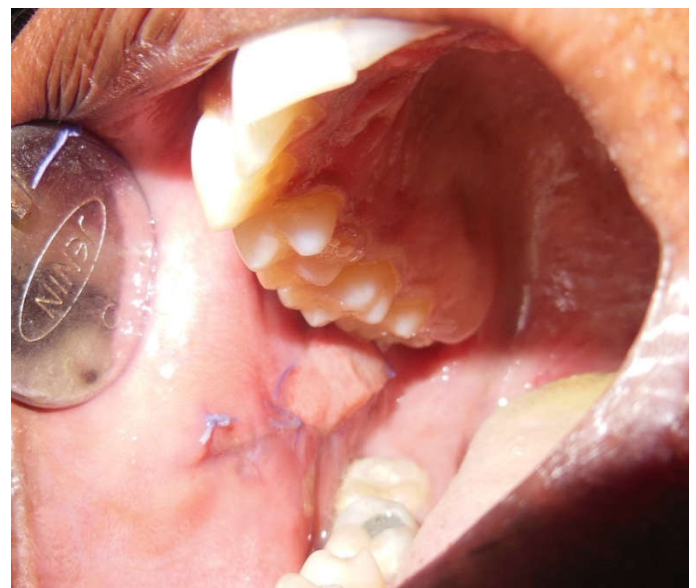


Figure 5. 1 week Postoperative picture showing satisfactory healing

On histopathological examination, the photomicrograph showed atrophic stratified parakeratinized squamous epithelium and collagenisedstroma with numerous vascular channels of cavernous pattern. Abnormally dilated irregular channels with less well- formed vascular walls are also evident. Vascular channels insinuating muscle fibre bundles causing muscular atrophy and degeneration were also evident. The patient was followed up regularly and there was good take at recipient site and also there was no evidence of recurrence (Figure 5).

DISCUSSION

As reported in the literature venous malformations originate during embryonic development and hence should be present at birth. However consistent with the study done by Kohout MP (Kohout *et al.*, 1998), in our case, the lesion was not evident at birth, but, rather noticed in adulthood. Patients with venous malformations first noticed during adulthood are often preceded by precipitating factors like trauma, hormonal changes like puberty. Necessary investigations to be done to assess the extent of venous malformation and its feeding vessels are very important. Preoperative embolization

facilitates surgical excision which will be necessary to control intraoperative bleeding (Kohout *et al.*, 1998; Kim *et al.*, 2006). As there was no uptake of the dye by any of the feeder vessel we did not plan for embolization. Reconstruction of the defect followed by excision of the lesion should be done to restore form and function (Hartzell *et al.*, 2009).

In our case the defect was reconstructed with buccal fat pad. Many sclerosing agents (sodium morrhuate, boiling water, nitrogen mustard, etc) are being used to treat high flow vascular lesions but they are not so effective as the agent is moved from the site of action due to blood flow (Kula *et al.*, 1996). Ligation of the external carotid artery is a promising adjunct but many authors don't accept it since many anastomoses (internal carotid, ophthalmic, vertebral, cervical and contralateral external carotid) create a rapid appearance of a collateral circulation (Mulliken and Glowacki, 1984). Embolization combined with surgical excision is the treatment of choice (Mohammadi *et al.*, 1997). This combined 98 approach controls the acute haemorrhagic phase, but doesn't reduce the recurrence rate because of emergence of collateral circulation (Abouzia and Symington, 1992).

Conclusion

The treatment of vascular head and neck lesions continues to evolve and with newer vascular radiologic techniques, embolic materials and surgical excision, patients get benefitted by the combined efforts of a surgeon and vascular radiologist. We conclude that Low flow venous malformations can be managed by surgical excision alone if there is no feeder vessel or very minimal uptake of the dye by a feeder vessel.

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