Arteriovenous malformation of maxilla: A rare and life threatening condition

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Abstract

Vascular lesions are congenital malformations of capillaries, veins, lymphatic vessels, arteries or a combination of different types of vessels. The gold standard treatment is to eliminate the lesion surgically after endovascular embolization. Preoperative endovascular embolization is widely accepted, but there are limitations to the treatment. We report a case where we employed a technique in which the lesion is devoided of blood supply after external carotid artery ligation and complete excision of the lesion and curettage is effective and could be used instead of the standard treatment especially in hospitals with limited facilities for endovascular embolization.

Keywords: Arteriovenous malformations, embolization, external carotid artery ligation, curettage, extraction

1. Introduction

Vascular malformations can be categorized into low-flow lesions (capillary malformations, lymphatic malformations and venous malformations) and high-flow lesions (arteriovenous malformations (AVMs) and arteriovenous fistulae), according to blood flow characteristics [1]. Arteriovenous malformation occur due to disturbance in vascular development between 4th and 6th week of gestation [2]. They are the most common high-flow lesions, frequently identified in the limbs, head, neck and lungs. However, lesions of the oral and maxillofacial regions are very rare, AVMs have been reported in the maxilla, but at half the frequency of AVMs in the mandible [3]. AVMs usually appear in adolescence but has an age range of 3 months to 74 years, predominance in females (female-to-male ratio, 2:1) [4]

2. Materials and Methods

2.1 Case Report

A female patient aged 14yrs reported to our unit with chief complaint of pain and swelling over left back tooth region since 6 months. History of moderate pain which is intermittent in nature, aggregates on chewing food and radiating to forehead, History of mobile tooth in that region since 1 year, severely tender, palpable left submandibular lymph nodes, the swelling was initially pea sized and gradually increased to current size over the span of one year. On intraoral examination inspectory findings included diffuse swelling noted over left maxillary alveolus measuring about 3x3cm extending from attached gingiva of 24 till distal of 27. Erythematous gingival overgrowth noted with respective to 26, 27 with mild vestibular obliteration and on palpation the swelling was tender, soft in consistency palataly and firm buccally, bleeding on probing noted and compressible socket with respect to 26, provisional diagnosis of vascular malformation was given based on compressibility and mobility and differential diagnosis of venous malformation, lymphatic malformation and AVM was suspected, opg, cross-sectional occlusal was done, fine needle aspiration biopsy yielded arterial blood, CT angiogram revealed well defined expansile cystic lesion with prominent feeding vessels from maxillary artery and few feeders from facial artery, as maxillary artery was involved, external carotid artery ligation was employed under general anesthesia followed by excision of the entire lesion. A horizontal incision was placed at the level of thyroid prominence 1cm from the midline parallel to the skin crease, after blunt dissection carotid sheath was identified and external carotid artery was identified and ligated using 3-0 mersilk, superior thyroid artery was identified and found arising from common carotid artery, closure...
achieved using 3-0 mersilk for deeper layers and 4-0 prolene for skin, complete enucleation and curettage of the lesion done through intraoral left vestibular approach and the defect was subjected to chemical cautery using carney's solution to prevent recurrence rate and extraction of 25, 26, 27 done, hemostasis achieved using ab gel which was packed in the defect, primary closure achieved using 3-0 vicryl intraorally.

Fig 1: Pre-operative intra oral view

Fig 2: Intra oral periapical radiograph of left posterior maxilla

Fig 3: Occlusal radiograph showing radiolucency at the apex of 26,27 region

Fig 4: CT angiography showing maxillary feeding artery

Fig 5: Pre operative orthopantomogram

Fig 6: CT scan showing lesion involving left maxillary sinus

Fig 7: External carotid artery ligation

Fig 8: Curetted cavity

Fig 9: Curetted lesion and extracted tooth
3. Discussion

The head and neck areas are the locations which are associated with 50% of all vascular lesions [9]. AVMs usually appear in adolescence but have an age range of 3 months to 74 years. Some authors noted predominance in females (female-to-male ratio, 2:1) while others have reported equal prevalence among males and females [8]. AVMs of the jaws are seen as painless, slow-growing soft-tissue enlargement with an audible bruit or palpable thrill, mucous membrane or cutaneous pigment changes, gingival bleeding and mobile tooth. Other patients are asymptomatic or may just have a loose tooth [7]. However, when it involves the jaws, it is potentially life threatening because of uncontrollable massive haemorrhage during tooth extraction. The most persistent objective sign of an intra-osseous AVM is recurrent spontaneous bleeding from the gingival region. Other symptoms suggestive of AVM include gingival discoloration, hyperthermia over the lesion and a subjective feeling of pulsation in the presence of bruits [8]. Radiographically, the most specific examination has traditionally been the arteriogram. CT scan has proven to be useful because it can delineate the lesion without the invasive nature of angiography. MRI may become a more valuable study in the future, since most these lesions involve almost entirely soft tissue [9]. Recently, digitalized equipment for Doppler sonography allows non-invasive depiction of small vessels concomitantly with the surrounding morphological features of anatomic structures. It also enables measurement of velocity of blood flow and resistance of the vessels. Thus, ultrasonography using colour Doppler flow analysis can confirm fast-flow anomalies [10]. Hormonal variations definitely influence these masses, and pregnancy often produces progression of these lesions, conservative methods of treating these lesions includes Sclerosis of the lesion with 95% ethanol injected via a percutaneous catheter has been used. This causes hemoglobin coagulation and internal disruption. Control of distribution of the ethanol is the major challenge here [11]. The use of KTP-532 and YAG lasers for photocoagulation of sinonasal vascular lesions using endoscopic delivery systems is another promising method of treatment of these lesion [12]. Angiographically controlled vascular embolization is especially useful with these lesions, but the complete ablation of these without recurrence using this technique has been disappointing to date [13]. Surgery continues to represent the most effective and controllable mode of treatment, especially when combined with preoperative embolization or preoperative ethanol injection sclerosis. The goal of surgery is control of symptoms, improvement in cosmesis, and total resection of the lesion.

4. Conclusion

Vascular anomalies embody a myriad of blood vessels abnormalities that are thought to occur peri-natally, correct diagnosis is imperative for appropriate treatment as they have high propensity to bleed and cause catastrophic complications during routine dental procedures. treatment of vascular anomalies is complex and requires multi-disciplinary approach with long term follow-up to curtail recurrence rates.

5. References