AV Malformation affecting Maxillofacial region: A Case Report

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Introduction

Vascular anomalies are a set of changes of blood vessels that could be associated, or not, to endothelial cells proliferation that results in a vascular hamartomatous growth [1]. Arteriovenous malformations (AVMs) are the result of errors of vascular development between the 4th and 6th weeks of gestation [2]. Failure to prune unwanted primitive communications between the arterial and venous systems may result in a malformation. Most of these lesions are obvious at birth while some are obvious during adolescence or adulthood.

It is believed that enlargement is the result of changes in pressure and flow, ectasia, shunting and collateral proliferation, rather than cellular proliferation. AVMs have a tendency to grow with the child and after the individual has attained full growth, AVMs remain stable throughout life. Some enlargement may occur in response to trauma or hormonal disturbances during puberty or pregnancy [3]. Treatment of AVMs can be difficult, as frequently, following an apparently successful extirpation, there is regrowth of the tumour to a size larger than its original size, often with supply by surgically inaccessible vessels [4].

Case Report

A 45 year old lady reported to the department of oral medicine and Radiology with chief compliant of diffuse swelling of face particularly around eyes since 6 years (Figure 1). The thickness of the lesion was variable. The overlying skin appeared normal over the swelling with no change in temperature or colour and was not associated with pain. Swelling was pulsatile with visible pulsations seen near left supra orbital region (Figure 2). No significant medical history was reported by patient and parents.

Based on history provisional diagnosis of AV malformation was formulated and patient was advised one USG of the affected region.

On USG multiple bilateral dilated tortuous AV malformations were seen extending from supraorbital region to neck. Superior ophthalmic vein was dilated on left side. Bilateral proptosis was also present. Eyeballs appeared normal however anterior chamber capsule was thickened bilaterally. There was no evidence of internal hemorrhage. So finally lesion was diagnosed as AV malformation. Further patient was advised one MRI angiography but patient was not cooperative for that hence lost in follow up (Figure 1 & 2).
Discussion

AVMs are the result of a failure of regression of the arteriovenous channels in the primitive retiform plexus [5]. They are composed of a central nidus with anomalous congenital shunts between the arterial and venous systems. These abnormal vascular channels may not canalize or conduct blood flow for many years. An enlargement is the result of dilatation of the adjacent arteries and veins (collateralization and recruitment) [6] rather than endothelial proliferation. Fast-flow vascular malformations usually become evident during childhood and puberty. Kohout et al. [7] in their study found that AVMs were present at birth in 59% of cases, in childhood 10% of cases, in adolescent 10% of cases, and in adulthood 21% of cases [8].

In the present case report, AVMs were present in late adulthood. AVMs grow synchronously with the growth of the child. Puberty and trauma are found to have triggered the rapid growth of the lesion leading to the manifestation of symptoms. Holt et al. [9] reported that trauma leading to the AVMs may be penetrating, blunt, postsurgical, or inflammatory [10]. The enlargement occurs due to the change in the pressure and flow within the malformation, ectasia, shunting, and collateral proliferation rather than cellular proliferation [11]. In the present case report, symptoms were triggered insidiously.

AVMs can be diagnosed with:

a. A Duplex or Doppler ultrasound uses sound waves to image your blood vessels and measure your blood-flow speed.

b. Computed tomography angiogram (CTA) uses a contrast agent (dye), which is injected into your vessels, to look for abnormalities. CT scans that do not use dye may also be taken of your head and neck.

c. Magnetic resonance angiography (MRA) combines an injected contrast agent (dye) with magnetic and radio waves to create 3-D cross-sections of the arteries in your neck and brain. An MRI (without dye) creates images of your head and neck.

d. In a catheter angiogram, the surgeon inserts a thin catheter through your groin and threads it into your carotid arteries; a contrast agent (dye) is injected to help clinicians visualize the arteries on X-rays.

Management of AVMs is most difficult due to the replacement of normal tissue by the diseased vessels and high-flow rate of recurrence. This management mainly consists of surgery, vascular embolization, or a combination of both. Surgical treatment consists of wide resection which is difficult and potentially hazardous due to significant blood loss during surgery [12].

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