Case Report

Scalp Arteriovenous Malformation - A Rare Case
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Abstract: I am reporting a rare case of arteriovenous malformation [AVM] of the scalp in a 22 year old female who presented with painless right tempo-parietal scalp swellings since birth. Skull X-ray, ultrasound, color Doppler and Contrast enhanced CT performed and the findings are discussed.

Keywords: Arteriovenous Malformations [AVM], Contrast enhanced computed tomography [CECT].

INTRODUCTION

An arteriovenous malformation [AVM] of the scalp is an abnormal fistulous connection between the feeding arteries and draining veins, without an intervening capillary bed within the subcutaneous layer of the scalp [1].

AVM of the scalp is a rare lesion when compared with other subcutaneous or cervicofacial vascular anomalies such as the haemangioma or venous malformations [2].

The vascular malformation of the scalp is an abnormal arteriovenous communication situated within the subcutaneous fatty layer of the scalp with the feeding arteries derived from the vessels supplying the scalp. Various names being used to describe the vascular malformations of the scalp include aneurysm cirsoide, aneurysma serpentinum, aneurysm racemosum, plexiform angioma, arteriovenous fistula and arteriovenous malformation [3].

The etiology of this scalp AVM is still controversial. However, it is generally accepted that it may be either of congenital or traumatic origin [1, 3]. It is generally thought that AVM arises from multiple developmental defects causing the primitive capillary bed to fail to persist [4].

CASE REPORT

A 22 year old female patient came with painless swellings in right tempo-parietal scalp region. These swellings are present since birth and they are gradually increasing in size, presently the larger lesion measures 6x5cm in right high parietal region. There was no previous history of trauma or head injury (Figure 1).

Skull X-ray showed scalp soft tissue lesions, underlying skull vault is normal no periosteal erosion/sclerosis/ scalloping is seen (Figure 2).

On Ultrasound mixed echoic lesions are seen in scalp soft tissue planes which are supplied by dilated, tortuous (Serpigenous) vessels. On color Doppler high velocity low resistance arterial flow is seen (Figure 3).

On CT mixed density scalp soft tissue lesions are seen right tempo-parietal scalp region. Calcification noted in larger lesion in right high parietal region. These lesions are supplied with dilated serpigenous vessels.

On post contrast CT scan these lesion are intensely enhancing and having feeding arteries from right superficial temporal artery, right posterior auricular artery and right occipital artery. There was neither intracranial extension of the lesion nor any intracranial AVM noted. Neck vessels and circle of Willis appeared normal in caliber. Brain parenchyma and ventricles did not reveal any significant abnormality (Figure 4A and 4B).

DISCUSSION

Congenital AVM of the scalp may present at birth, but in most patients, it is asymptomatic until adulthood. Scap AVMs are most frequently confused with hemangioma and cavernomas. No arteriovenous shunt is present in such pathologies, and they are seen as well-demarcated lesions [1].

CECT is helpful to differentiate scalp AVMs from other vascular lesions and aid in the correct diagnosis as well as to distinguish whether there is intracranial extension or involvement [2].

Depending upon the origin of the feeding arteries, scalp arteriovenous malformations are classified into two groups: Group I-primary scalp vascular malformations and Group II-secondary scalp venous dilatations [3].
Management of scalp AVM is difficult for several reasons because of its high shunt flow with complex complicated vascular anatomic connection and also involves cosmetic problems. Thus there has been no consensus on the treatment of scalp AVM. There are various techniques and method of treatment for scalp AVM. Among the treatment options include surgical excision, ligation of feeding vessels, transarterial and transvenous embolization, injection of sclerosant into the nidus and electrothrombosis [4, 5].

In the past, the treatment of choice for scalp AVM was surgical excision or ligation of the feeding arteries. However, with progress in endovascular surgical technique, embolization has become an integral part of the treatment of these malformations. Cure of these lesions may be attained by embolization alone in some patients, or by embolization followed by surgical removal [4].
CONCLUSION
This case suggests that scalp AVM can become enlarged by capturing subcutaneous feeders, and that the consequent hemodynamic stress. Ultrasound, color Doppler and CECT are excellent non-invasive and cheap methods to visualize AVM.

REFERENCES