CASE REPORT

SCALP ARTERIOVENOUS MALFORMATION: A CASE REPORT

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We report a rare case of Arteriovenous malformation (AVM) of the scalp in a 30 year-old Malay gentleman who presented with painless forehead swelling since birth. Magnetic Resonance Imaging (MRI) and cerebral angiogram performed and the findings are discussed.

Key words: Scalp Arteriovenous Malformations, Subcutaneous Arteriovenous malformations, Radiological features

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Introduction

Arteriovenous malformation (AVM) in the scalp is rare when compared with other subcutaneous or cervicofacial vascular anomalies such as the haemangioma or venous malformations. It is an abnormal fistula communication between feeding arteries and draining veins without intervening

Figure 1: The AVM has a bluish discolouration at the midline of the forehead could be due to dilated draining veins. The overlying skin is coarse and dry; however there was neither ulceration nor active hemorrhage present.
capillary bed. The draining veins can dilate and causes esthetic problem (1-3). It usually presents in late childhood, adolescent or early adulthood. It can also cause massive hemorrhages due to dryness of the overlying skin and injuries (1). In this report we describe the clinical and radiological features of a patient with a scalp arteriovenous malformation.

Case Report

The patient was a 30 year-old Malay gentleman who complained of a painless forehead swelling since birth, increasing in size for the past 3 – 4 years. Initially, it was a reddish small growth. The lesion bled once following trauma about a year prior to admission. The hemorrhage was secured at that time without resorted to any definitive treatment. Insidiously the forehead lesion grew in size and causing esthetic problem to him. There was no similar history in the family.

On examination, there was a soft, bluish and non-tender lesion in the midline of the forehead. The overlying skin is coarse and dry; however there was neither ulceration nor active hemorrhage present (fig 1). Bruit was heard on auscultation of the lesion. The vital signs are stable and all other systems were normal. The blood investigations were unremarkable.

Magnetic Resonance Imaging (MRI) of the brain revealed a predominantly hyperintense subcutaneous mass in the midline of the forehead on both T1 and T2 weighted images which extends superiorly beyond forehead and inferiorly down to the root of nose. There are multiple flow void serpinginous structures within the lesion which enhanced with contrast, in keeping with dilated

Figure 2: MRI revealed subcutaneous mass at midline of the forehead has inhomogenous signal intensities with flow void serpinginous structures within in keeping with enlarged draining veins. No intracranial extension.

Figure 3: Cerebral angiography revealed multiple feeding arteries with tortuous course arising from ophthalmic arteries and also from superficial temporal arteries.
vessels (fig 2). However, no MR angiography was
done at that time. There was no intracranial extension.

About 2 months after MRI, he underwent
cerebral angiogram, which showed presence of
abnormal early draining veins, in keeping with
vascular malformations in the midline frontal region.
Feeding arteries with tortuous course appeared arising
from the right ophthalmic artery with several other
feeders from the left ophthalmic artery. There are
also feeders also from right and left superficial
temporal arteries (fig 3). No supply was noted from
both anterior cerebral arteries. Enlarged early
draining veins were seen draining into the scalp
veins. There was no intracranial venous drainage.
He then underwent surgery for complete removal
of his scalp AVM in the forehead.

Discussion

AVMs in scalp are relatively rare. They are
20 times more common in the brain involving or
supplied by intracranial vasculature than in that from
external carotid arteries (1). Cervicofacial
involvement is most common in the cheeks, ears,
nose, and less commonly forehead (2). Scalp AVMs
are normally noticed in late childhood, adolescent
or early adulthood, when substantial esthetic and
social disturbance entailed, or due to various stimuli
such as trauma, pregnancy or puberty (2).

MRI is helpful to differentiate cervicofacial
AVMs from other vascular lesions and aid in the
correct diagnosis as well as to distinguish whether
there is intracranial extension or involvement (3).
MRI can also help to distinguish scalp AVMs which
are high flow lesions from other low flow lesions
such as venous or lymphatic malformations, and this
will help with the treatment planning. However,
catheter angiography is still the gold standard
modality to understand the angioarchitecture of the
lesion and to exclude any intracranial component
(4).

Management of scalp AVMs is difficult
because of its high flow, complex vascular anatomy
and cosmetic problems. There are various techniques
and method of treatment for scalp AVMs. Among
the treatment options include surgical excision,
ligation of feeding vessels, transarterial and
transvenous embolization, injection of sclerosant
into the nidus and electrothrombosis (1, 5-7).
Shenoy et al divided scalp AVMs into group 1 and II, to
help decide the treatment of choice. Group 1 will
represent primary scalp vascular malformations and
Group II will represent secondary scalp venous
dilations. This patient belongs to group I
representing primary scalp vascular malformations.

Surgical excision is the most common and
successful method of treating scalp AVMs (1).
Endovascular approaches are an option as a
definitive therapy or as an adjunct to surgical therapy
to reduce blood loss during excision (5). The most
common cause of treatment failure, even with
combined embolization and surgery is incomplete
resection. Recurrence has been reported as late as 18
years after complete surgical resection (4).

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References
1. Weinzweig N, Chin G, Polley J, Chabrel F, Shownkeen
H, Debrun G. Arteriovenous malformation of the
forehead, anterior scalp and nasal dorsum. Plast
2. Kohout MP, Hansen M, Pribaz JJ, Mulliken JB.
Arteriovenous malformations of the head and neck :
natural history and management. Plast Reconstr Surg
3. Enjolras O, Mulliken JB. Vascular malformations. In:
Harper J, Oranje A, Prose N, eds. Textbook of Pediatric
Dermatology. Oxford: Blackwell Science,2000:975-
96.
4. Wilkinson HA. Recurrence of vascular malformation
of the scalp 18 years following excision. Case report.
5. Nagasaka S, Fukushima T, Goto K, Ohjimi H,
Iwabuchi S, Maehara F. Treatment of scalp
arteriovenous malformation. Neurosurgery
6. Mourao GS, Hodes JE, Gobin YP, Casasco A, Aymard
A, Merland JJ. Curative treatment of scalp
arteriovenous fistulas by direct puncture and
embolization with absolute alcohol. Report of three
7. Gardner AMN, Stewart IA. Treatment of arteriovenous
malformation by endarterial electrocoagulation. Br J
8. Shenoy SN, Raja A. Scalp arteriovenous