Congenital Arteriovenous Malformation of the Scalp Involving the Orbit

Alberto Feletti[1] Stavros Dimitriadis[1] Stefano Vallone[2] Giacomo Pavesi[1]

[1] Department of Neurosurgery, Azienda Ospedaliero-Universitaria di Modena, Via Giardini 1355, 41121 Modena, Italy
[2] Department of Neuroradiology, Azienda Ospedaliero-Universitaria di Modena, Modena, Italy

Address for correspondence Alberto Feletti, MD, PhD, Department of Neurosciences, Azienda Ospedaliero-Universitaria di Modena, Via Giardini 1355, 41121 Modena, Italy (e-mail: alberto.feletti@gmail.com).

J Neurol Surg A 2018;79:541–549.

Abstract

Background Arteriovenous malformations (AVMs) of the scalp are rare and infrequently encountered by the neurosurgeon.

Case Description We report a unique case of a 42-year-old patient who presented with a progressive worsening of visual acuity in the right eye (lower quadrantanopia) and palpebral ptosis. Physical examination revealed a right exophthalmos and a right frontoparietal scalp soft swelling when the patient was in the supine position. Neurologic work-up showed a scalp AVM extending into the orbit and connected to an intraorbital cavernous angioma. The patient was treated with a frontotemporal craniotomy and decompression of the orbit.

Conclusions In the rare case of intraorbital extension of a scalp AVM, neurologic symptoms may appear when the size of the vascular malformation increases with age. The aims of surgery should be decompression of the orbit and aesthetic preservation, rather than complete excision. A review of the literature is also provided.

Keywords
- arteriovenous malformation
- cavernous angioma
- cirsoid aneurysm
- intraorbital
- scalp

Introduction

Arteriovenous malformations (AVMs) of the scalp are rare vascular malformations.[1] They are also referred to as cirsoid aneurysms because their altered hemodynamics causes a tortuous and progressive dilation of the veins.[2] Although posttraumatic AVMs have been described, in most patients a clear etiology cannot be established, and the malformations are therefore defined as "congenital" or, in some cases, "idiopathic."[3–6] Presenting symptoms include pulsating compressible scalp swelling, headache, local pain, and less commonly hemorrhage and necrosis.[2,7] However, in the rare case of intraorbital extension, the AVM can cause neurologic and aesthetic impairments that justify an aggressive surgical treatment. We report on a patient with an AVM extending into the orbit and associated with an intraorbital cavernous angioma. We also provide a review of the pertinent literature and discuss the natural history and the management of these uncommon malformations.

Case Report

A 42-year-old male patient presented with a progressive worsening of visual acuity in the right eye (lower quadrantanopia) and palpebral ptosis. In addition, the physical examination revealed right exophthalmos and a right frontoparietal scalp soft swelling when the patient was in the supine position. No birthmarks were noted on the skin. No external ocular movement impairment was detected. Medical history was positive for obesity and hypertension. The right parietal skull appeared thin and discontinuous on computed tomography (CT) (►**Fig. 1**, ►**Video 1**). Magnetic resonance imaging showed a right intraorbital superolateral vascular malformation (►**Video 2**), and digital subtraction angiography revealed the presence of a frontoparietal extracranial pathologic venous malformation fed by vessels from the occipital artery (►**Fig. 2**, ►**Video 3**). Angiography confirmed the AVM, outlining arterial feeders from the right occipital and temporal arteries, extensive venous dilation on the scalp, and engorged veins in the right orbit.

received
August 17, 2017
accepted after revision
February 8, 2018
published online
June 15, 2018

DOI https://doi.org/10.1055/s-0038-1641178.
ISSN 2193-6315.

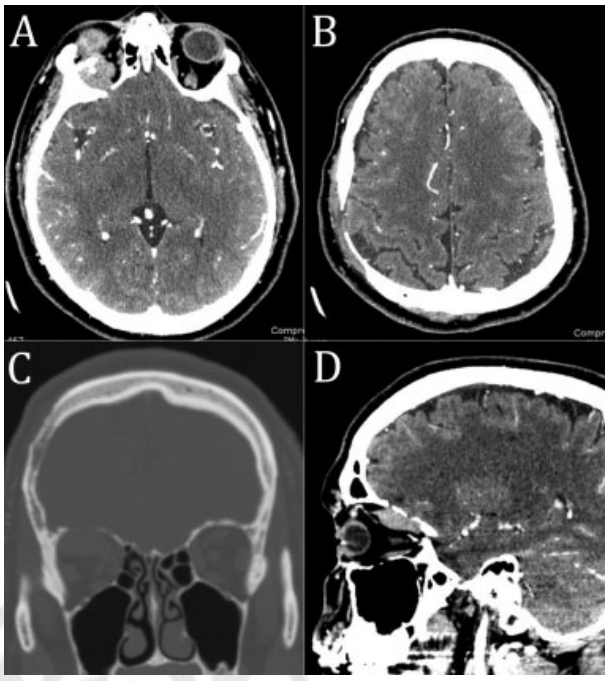

Fig. 1 Preoperative computed tomography scan showing the right intraorbital part of the arteriovenous malformation (AVM), with three small calcifications (**a**) and the right frontoparietal part of the scalp AVM with skull erosion (**b**). Coronal (**c**) and sagittal (**d**) reconstructions evidence the interruption of the right orbital roof.

Video 1

Preoperative computed tomography angiography. Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10.1055/s-0038-1641178.

Video 2

Preoperative magnetic resonance imaging. Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10.1055/s-0038-1641178.

Video 3

Preoperative digital subtraction angiography. Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10.1055/s-0038-1641178.

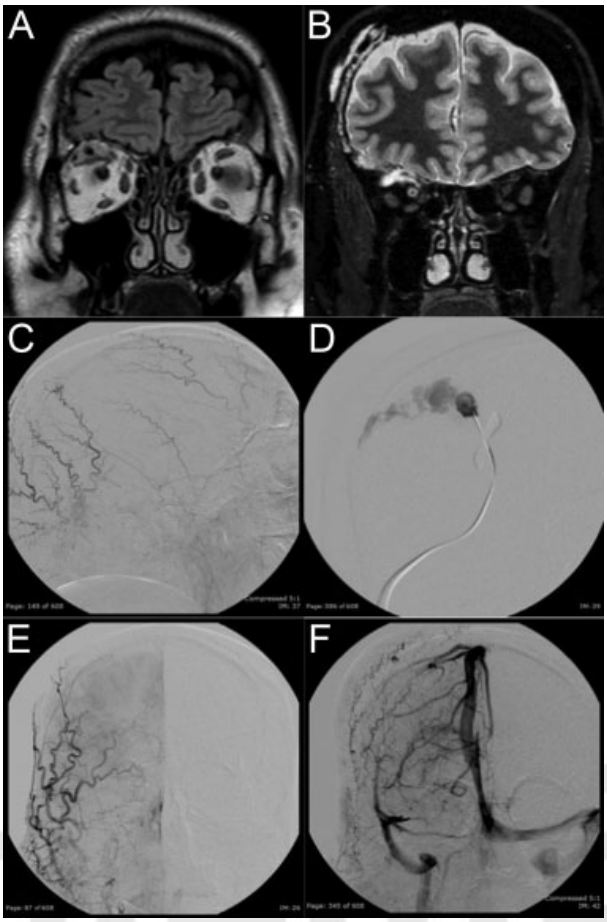

Fig. 2 (**a**) Coronal fluid attenuation inversion recovery magnetic resonance imaging (MRI) showing the intraorbital part of the arteriovenous malformation (AVM) displacing the superior rectus muscle of the right eye. (**b**) Coronal short tau inversion recovery MRI showing the connection between the scalp AVM and the intraorbital part of the malformation. (**c–f**) Angiography demonstrates the feeders of the scalp AVM arising from the occipital artery and, to a lesser extent, from the superficial temporal artery.

The patient underwent a frontotemporal craniotomy. Immediately after the scalp incision, abundant venous bleeding occurred from the AVM that was controlled by cauterization and ligation. The skull below the malformation appeared thin, irregular, discontinuous, and perforated by several thin vessels. No intracranial extension of the malformation was detected. The pathologic features of the bone were also evident on the orbital roof, which was removed. Hemostasis on the pathologic bone was obtained using a diamond drill and bone wax. The intraorbital malformation consisted of very soft venous sinusoids embedded in the orbital fat and tightly attached to the elevator palpebrae and superior rectus muscles. For this reason, we performed just a lesion biopsy, avoiding any attempt of radical removal to prevent further damage to the extrinsic eye muscles. Two spherical calcifications were removed from the intraorbital space. To reconstruct the roof of the orbit, the craniotomy bone was split and the bone piece was properly shaped and fixed to the frontal bone with microplates and microscrews.

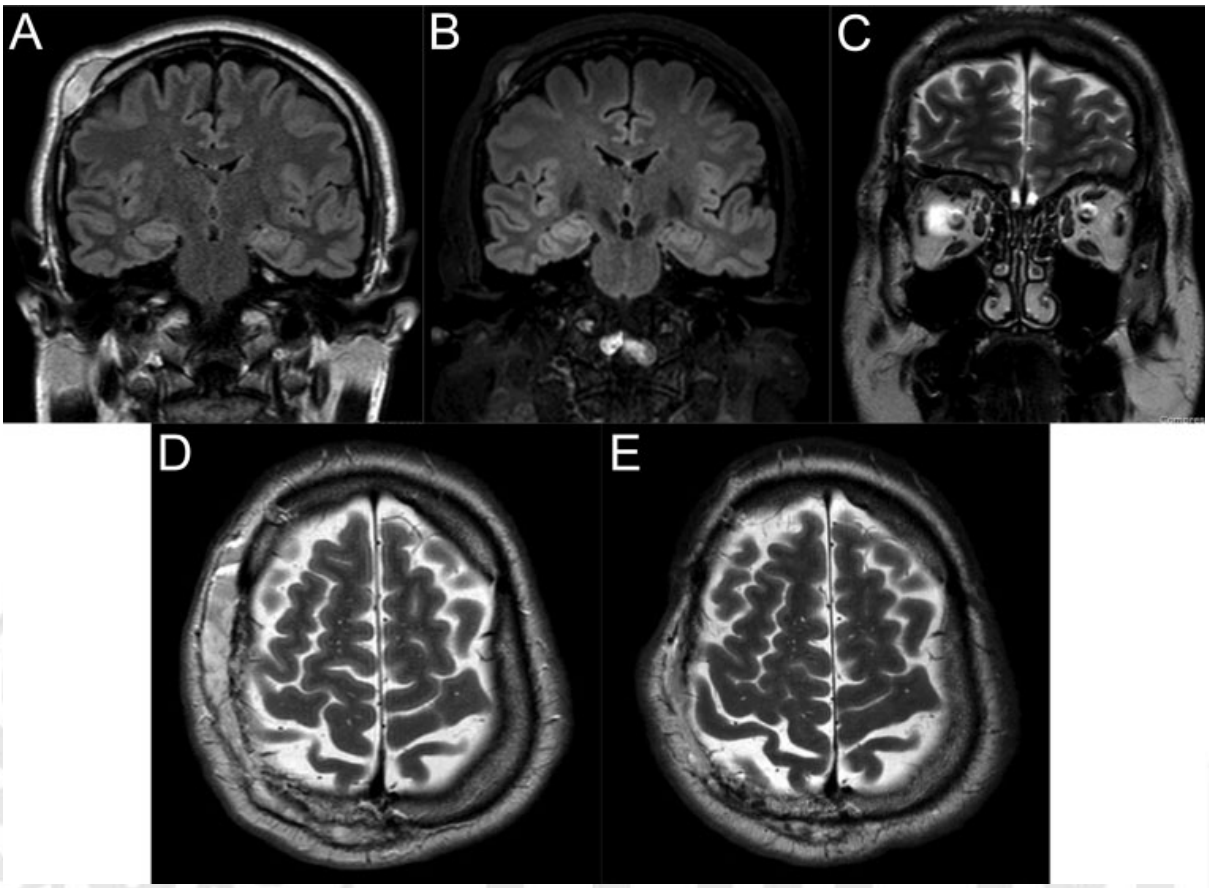

Fig. 3 (**a**) Preoperative coronal fluid attenuation inversion recovery (FLAIR) magnetic resonance imaging (MRI). (**b**) Postoperative coronal FLAIR MRI. (**c**) Postoperative coronal T2-weighted MRI. The volume and extension of the scalp arteriovenous malformation (AVM) is reduced, as well as the intraorbital compression. (**d**) Preoperative axial T2-weighted MRI. (**e**) Postoperative axial T2-weighted MRI showing the reduction of the scalp swelling due to the AVM.

The postoperative course was uneventful, and a CT ruled out any complication. Exophthalmos and the soft swelling on the scalp were not evident in the supine position immediately after surgery. The 3-month follow-up neuroradiologic work-up confirmed the satisfactory decompression of the ocular cone, and physical examination revealed a partial progressive recovery of the right palpebral ptosis and a complete resolution of exophthalmos and scalp swelling (►**Fig. 3**, ►**Video 4**).

Video 4

Postoperative magnetic resonance imaging. Online content including video sequences viewable at: www.thieme-connect.com/products/ejournals/html/10.1055/s-0038-1641178.

Discussion

AVMs of the scalp are rare vascular malformations whose origin is still not completely clear.[2,8] About 10 to 20% of scalp AVMs develop after penetrating or nonpenetrating head trauma.[2,3,9] However, according to the vast majority of the published literature, in most cases there is no history of trauma, and the scalp AVMs are therefore defined as either idiopathic or congenital. Some authors distinguish congenital and idiopathic AVMs based on the presence of the malformation since birth.[5] Nevertheless, it was claimed that congenital scalp AVMs can become evident and clinically relevant only after the second or third decade, making the distinction between congenital and idiopathic extremely difficult.[10] The failure of the embryonic vasculature to differentiate into arteries and veins results in a persistent communication between aberrant vessels, with the absence of an intervening capillary bed.[8,11] Trauma, pregnancy, and hormonal changes can cause growth of the lesion and worsening of symptoms.[12] A role of vascular endothelial growth factor was also proposed but remains to be confirmed.[13]

Scalp AVMs are also known as arteriovenous aneurysms, cirsoid aneurysms, and arteriovenous fistulas.[4] These lesions are difficult to manage because of their complex vascular anatomy and the extension of the venous dilations in the scalp. They may present with pulsatile swelling, headache, and hemorrhage; neurologic deficits are rare.[2,7] The altered hemodynamics cause the progressive dilatation of the veins

Table 1 Congenital arteriovenous malformations of the scalp: literature review

Study	Cases	Age, y/Sex	Location	Size, cm	Presentation	Feeding artery	Treatment
Beaumont[17]	1	22/F	F		Pulsating mass		Ligation and surgical electrocautery
Elkin[9]	1	20/M	T		Pulsating mass, bruit, thrill		Ligation and total excision
Oldfield and Addison[33]	3	34/M	T		Pulsating mass, swelling		Total excision
		17/F	FTP		Pulsating mass, swelling	STA	Ligation and total excision
		51/F	FPO		Pulsating mass, thrill, headache		Conservative treatment
Vasconez[40]	1	2/F	Median	10 × 12	Large dark eschar	STA, OA	Total excision
Stucker[36]	2	59/M	Lateral		Tinnitus	OA	Ligation and surgical electrocautery
		53/F	Lateral		Tinnitus	OA, PAA	Ligation and surgical electrocautery
Waga et al[41]	2	45/M	Lateral		Pulsating mass	VA	Embolization
		38/M	Lateral	10 × 10	Bleeding	STA, OA	Total excision
Mohanty and Rao[28]	1	1.5/F	PTO	10 × 6	Pulsating mass, convulsion	STA	Ligation
Takahashi et al[37]	1	29/F	T	10 × 8	Pulsating mass	IMA, STA	Total excision
Yoneda et al[44]	1	11/M	Lateral		Pulsating mass	STA, OA	Conservative electrocautery
Kaufman et al[24]	2	17/F	Lateral		Bleeding	STA, OA	Embolization and total excision
		4/M	Lateral		Bleeding	STA, OA	Embolization and total excision
Schultz And Hermosillo[11]	1	45/F	Lateral		Pulsating mass	STA, OA	Ligation and total excision
Goya et al[22]	2	52/M	Median	8 × 8	Pulsating mass, bleeding	STA, MMA, OA	Embolization and total excision
		17/F	Lateral	3 × 3	Pulsating mass, dizziness	OA	Total excision
Ohno et al[32]	1	32/M	O	4 × 7	Dizziness	OA	Embolization and total excision
Irving et al[1]	1	33/M	Lateral	Large	Pulsating mass, bleeding	STA, SO	Ligation and total excision
Konishi et al[25]	1	44/M	O	4 × 5	Pulsating mass	OA	Total excision
Yamaki et al[43]	1	31/M	T	5 × 5	Pulsating mass	DTA	Ligation, embolization and total excision
Takahasi et al 1983	1	22/M			Pulsating mass	STA, OA, PAA	Embolization and total excision
Shimoda et al[35]	2	25/M	O	15 × 15	Pulsating mass	STA, OA	Total excision
		49/M	TP	1 × 1	Pulsating mass, tinnitus	STA, OA	Embolization and total excision

Table 1 (*Continued*)

Study	Cases	Age, y/Sex	Location	Size, cm	Presentation	Feeding artery	Treatment
Komatsu et al[8]	1	43/M	F	4 × 4	Pulsating mass, bleeding	STA	Total excision
Barnwell et al[14]	7	30/M	P			STA	Embolization
		21/M	P			STA, OA	Embolization and total excision
		62/M	P		Pulsating mass	STA	Embolization
		62/M	RM		Bleeding	IMA	Embolization
		33/M	P		Headache, swelling	MMA, OA, PAA	Embolization and total excision
		40/M	Au		Pulsating mass	STA, PAA, OA	Embolization and total excision
		12/F	Au			PAA, OA, IMA	Embolization
Mourao et al[16]	1	50/F	T		Swelling	STA, OA	Ligation and embolization
Worm et al[42]	1	33/M	TO		Pulsating mass	STA, OA	Excision
Nishimura and Kubota[30]	1	47/F	TP	3 × 3	Pulsating mass	STA	Ligation and total excision
Fisher-Jeffes et al[3]	13						
Kuroki et al[26]	1	23/M	T	4 × 3	Tinnitus	STA, OA, PAA, MMA	Total excision
Nishiura et al[31]	1	27/F	O	3.5 × 3.5	Pulsating mass	OA, MMA, pial a	Embolization and total excision
Muthukumar et al[6]	5	25/M	P	3	Pulsating mass, bruit, thrill	STA, PAA	En bloc resection and repair
		18/M	O	4	Pulsating mass, bruit, thrill	OA, PAA	Total excision
		30/M	O	4	Pulsating mass, bruit, thrill	OA, PAA	Total excision
		20/M	O	4	Pulsating mass, bruit, thrill	OA, PAA	Total excision
		12/M	O	9	Pulsating mass, bruit, thrill	OA, STA	Injection of sclerosing agents twice and total excision
Matsushige et al[13]	1	21/F	TP	7 × 7	Pulsating mass, tinnitus	STA, OA, PAA, MMA, pial artery	Embolization and total excision
Shenoy and Raja[34]	5						
Gurkanlar et al[4]	16	7/F	P		Pulsating mass	STA	Subtotal excision
		21/M	F		Pulsating mass	STA	Total excision
		21/M	TP		Pulsating mass	STA	Total excision
		21/M	T		Pulsating mass	STA, OA	Embolization and total excision
		21/M	P		Pulsating mass	STA, OA	Total excision
		22/M	FP		Pulsating mass	MMA, STA, SO	Total excision

(*Continued*)

Table 1 (*Continued*)

Study	Cases	Age, y/Sex	Location	Size, cm	Presentation	Feeding artery	Treatment
		20/M	FP		Pulsating mass	STA, OA	Total excision
		22/M	P		Pulsating mass	STA	Total excision
		21/M	F		Pulsating mass	STA	Total excision and repair
		28/M	P		Pulsating mass	STA	Total excision
		21/M	F		Pulsating mass	STA	Total excision
		23/M	O		Pulsating mass	OA, PA	Total excision
		22/M	F		Pulsating mass	STA	Total excision
		24/M	F		Pulsating mass	STA	Total excision
		20/M	T		Pulsating mass	STA	Total excision
		23/M	F		Pulsating mass	STA	Total excision
Mohamed et al[27]	1	30/M	F		Pulsating mass	STA, OA	Total excision
Senoglu et al[7]	1	35/M	O		Pulsating mass, thrill	OA	Ligation
Dalyai et al[15]	1	60/M	O	4 × 3	Pulsating mass	OA, PAA	Embolization
Hasturk et al[23]	1	60/M	FT		Pulsating mass	STA	Excision
Kumar et al[2]	2	?	PO	7 × 6		OA, PAA	En bloc resection and skin grafting
		?	F	10 × 4		STA	Embolization and total excision
Tauro et al[39]	1	40/F	TO		Headache, swelling	STA, OA	Conservative treatment
El Shazly and Saoud[19]	4	30/M	P		Pulsating mass	STA, PAA	Total excision
		32/M	O		Pulsating mass, headache	OA, PAA	Total excision
		20/M	O		Pulsating mass	OA, PAA	Total excision
		25/M	F		Pulsating mass, headache	SO, STA	Total excision
Chowdhury et al[18]	6	19/M	F	10 × 5	Headache, swelling	STA, SO	Total excision
		43/M	TP	15 × 19	Headache, tinnitus, ulceration, bleeding	STA, SO, PA, OA	Total excision
		18/F	FP	7 × 18	Headache, swelling	STA, SO, PAA, ST	Total excision
		35/M	T	10 × 12	Headache, swelling	STA, SO, PA	Total excision
		39/M	F	10 × 13	Headache, swelling	STA, SO, ST	Total excision
		47/F	T	7 × 5	Headache, swelling	STA, SO, PAA	Total excision
Goel et al[21]	1	16/F	FP		Pulsating mass, swelling	STA	Ligation and total excision
Gupta and Kayal[10]	5	11/M	FT	9.4	Swelling, headache	STA	Excision

Table 1 (*Continued*)

Study	Cases	Age, y/Sex	Location	Size, cm	Presentation	Feeding artery	Treatment
		12/F	FTP	18.6	Swelling, headache, bleeding	STA	Excision
		17/M	F	4	Swelling	STA	Excision
		14/M	F	11.2	Swelling, headache	STA	Excision
		16/M	O	3	Swelling	OA	Excision
Munakomi et al[12]	1	38/F	FTP	12 × 4	Swelling	STA, OA, CMA, PCA	
Gangadharaswamy et al[20]	3	30/F	T		Pulsatile mass, headache	ECA	Complete excision
		50/F	F		Pulsatile mass, headache	STA	Complete excision
		12/M	O		Pulsatile mass	OA	Complete excision
Present case	1	42/M	FP		Swelling, lower quadrantanopia, palpebral ptosis, exophthalmos	OA	Subtotal excision

Abbreviations: Au, auricular; CMA, callosomarginal artery; DTA, deep temporal artery; ECA, external carotid artery; F, frontal; FP, frontoparietal; FPO, fronto-parieto-occipital; FTP, fronto-temporo-parietal; IMA, internal maxillary artery; MAA, middle meningeal artery; O, occipital; OA, occipital artery; P, parietal; PAA, post auricular artery; PCA, posterior cerebral artery; PO, parieto-occipital; PTO, parieto-temporo-occipital; RM, retromandibular; SO, supraorbital artery; ST, supratrochlear artery; STA, superficial temporal artery; T, temporal; TO, temporo-occipital; TP, temporo-parietal; VA, vertebral artery.

forming so-called cirsoid aneurysms.[2,4] Treatment of these lesions includes endovascular embolization, direct intralesional injection of sclerosing agents, ligation of feeders, and surgical excision.[8,13–16]

After a thorough review of the English literature, we found 108 published cases of congenital scalp AVMs (►**Table 1**).[1–4,6–44] The mean age at diagnosis is 27.8, and males are significantly more affected than females in a 2:1 ratio. The most common feeders are the temporal artery (55%) and the occipital artery (40%). Scalp AVMs may be asymptomatic or they can cause symptoms such as pulsation, bleeding, skin erosion, blood steal, and cosmetic problems.[2,7] Although scalp AVMs can occasionally drain into dural veins, they have only rarely been reported extending intracranially. In some cases, parasitizing arterial vessels from intracranial arteries as internal carotid or posterior cerebral arteries were shown to contribute to feeding of the AVM.[12]

Although ligation, embolization, and injection of sclerosing agents have been used, the most effective and widely adopted treatment is surgical excision.[7] In only five cases the scalp AVM was not treated and just observed over time. In four of these cases, the patient refused surgery. In one case, reported by Oldfield and Addison in 1962, the scalp AVM was large and connected with an intracranial arteriovenous fistula; the authors considered observation preferable due to the high risks of surgical resection.[33] Final cure after surgical excision is reported in $>$ 90% of operated cases. The observed complications include scalp necrosis (4.5%) and infection (4.5%).

Endovascular transarterial or transvenous embolization has been also performed with good results, as well as intralesional injection of sclerosing agents, with complete obliteration of the lesion in selected cases.[19] However, these techniques also carry the risk of complications such as skin necrosis, permanent patchy hair loss, pain, skin tenderness, and leakage of embolization material into the systemic circulation.[19] Moreover, embolization alone can be unsuccessful in $\sim$ 10% of cases.[35] The combination of preoperative embolization and excision of the scalp AVM has a risk of skin necrosis of $\sim$ 8%. It is worth noting that two deaths were reported after surgical excision, with or without preoperative embolization, due to hypovolemic shock or brain edema.[2,34]

Treatment of scalp AVMs is required when they cause neurologic symptoms and is generally advisable when they are so large that a direct accidental trauma might cause profuse bleeding. Our case is particularly interesting because the AVM extended through the orbital roof into the orbit, where a cavernous angioma had its venous drainage into the venous part of the AVM. Although cavernous angiomas are the most frequently encountered intraorbital mass lesions, the association between an intraorbital cavernoma and a scalp AVM is exceptional. The orbital roof was thinned and cribriform due to the crossing of several venous connections. A similar pathologic appearance was evident on the frontoparietal skull that was thin and discontinuous. The malformation was completely extradural. Such findings are in accordance with a congenital origin of the malformation.

Notably, despite their congenital origin, the surgeon usually encounters these malformations when the patient is already a

young adult.[5,6,8] This is not surprising because scalp AVMs may grow and progressively increase both their aesthetic impact and their mass effect on neural, bony, and muscular structures. The lesion growth can be particularly disfiguring and functionally dangerous when it involves the orbit, as shown by our case. The patient did not refer to the physician until the onset of palpebral ptosis and visual impairment. Because our first aim was immediate decompression of the orbit to avoid further neurologic decline, we did not consider embolization, which could be a valid treatment option in cases without neurologic deficit. We initially thought about embolization as a second step to complete the exclusion of the AVM. However, the scalp swelling and venous engorgement completely disappeared after surgery, requiring no further treatment.

This is the second reported case of a scalp AVM extending into the orbit after the one published by Beaumont in 1897,[17] and the first one reporting a scalp AVM anatomically connected to an intraorbital cavernous angioma. Management of scalp AVMs is difficult and challenging because of their high shunt flow, complex anatomy, and cosmetic issues. In general, total resection should be the goal because it offers the only way to radically cure the lesion. However, in cases when neurologic impairment is the onset symptom, and the AVM is complex and directly connected with the orbital compartment, the main aim of surgery should be to avoid further neurologic worsening. For this reason, an effective decompression of the orbit with restoration of cosmesis is indicated, with no need for a hazardous total removal of the malformation.

Conclusions

Scalp AVMs are rare extracranial vascular malformations, seldom encountered by neurosurgeons. Surgical excision is indicated when they cause functional or cosmetic problems. Although they are congenital, their size can progressively increase and bring patients to medical attention when they are already young adults. The rare cases of intraorbital extension of a scalp AVM are particularly threatening because their growth or increase of venous engorgement can produce a mass effect leading to a neurologic deficit like visual acuity impairment and weakness of the ocular muscles. The aim of surgical excision is decompression of the orbit and aesthetic preservation.

References

1 Irving AD, Thakur A, Walker WF. Cirsoid aneurysm of the scalp. J R Coll Surg Edinb 1982;27(02):115
2 Kumar R, Sharma G, Sharma BS. Management of scalp arteriovenous malformation: case series and review of literature. Br J Neurosurg 2012;26(03):371–377
3 Fisher-Jeffes ND, Domingo Z, Madden M, de Villiers JC. Arteriovenous malformations of the scalp. Neurosurgery 1995;36(04): 656–660; discussion 660
4 Gurkanlar D, Gonul M, Solmaz I, Gonul E. Cirsoid aneurysms of the scalp. Neurosurg Rev 2006;29(03):208–212
5 Khodadad G. Arteriovenous malformations of the scalp. Ann Surg 1973;177(01):79–85
6 Muthukumar N, Rajagopal V, Manoharan AV, Durairaj N. Surgical management of cirsoid aneurysms. Acta Neurochir (Wien) 2002; 144(04):349–356
7 Senoglu M, Yasim A, Gokce M, Senoglu N. Nontraumatic scalp arteriovenous fistula in an adult: technical report on an illustrative case. Surg Neurol 2008;70(02):194–197
8 Komatsu Y, Narushima K, Kobayashi E, Nose T, Maki Y. Congenital arteriovenous malformation of the scalp—case report. Neurol Med Chir (Tokyo) 1989;29(03):230–234
9 Elkin DC. Cirsoid aneurysm of the scalp; report of four cases. Trans South Surg Assoc 1946;57:122–131
10 Gupta R, Kayal A. Scalp arteriovenous malformations in young. J Pediatr Neurosci 2014;9(03):263–266
11 Schultz RC, Hermosillo CX. Congenital arteriovenous malformation of the face and scalp. Plast Reconstr Surg 1980;65(04):496–501
12 Munakomi S, Bhattarai B, Cherian I. Conquering the odds: cirsoid aneurysm with holocranial feeders—staged embolization, excision and grafting. Asian J Neurosurg 2015;10(03):259–261
13 Matsushige T, Kiya K, Satoh H, Mizoue T, Kagawa K, Araki H. Arteriovenous malformation of the scalp: case report and review of the literature. Surg Neurol 2004;62(03):253–259
14 Barnwell SL, Halbach VV, Dowd CF, Higashida RT, Hieshima GB. Endovascular treatment of scalp arteriovenous fistulas associated with a large varix. Radiology 1989;173(02):533–539
15 Dalyai RT, Schirmer CM, Malek AM. Transvenous balloon-protected embolization of a scalp arteriovenous fistula using Onyx liquid embolic. Acta Neurochir (Wien) 2011;153(06):1285–1290
16 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 cases. J Neurosurg 1991;75(04):634–637
17 Beaumont WM. Cirsoid aneurysm of the orbit, forehead, and scalp. BMJ 1897;2(1909):273–274
18 Chowdhury FH, Haque MR, Kawsar KA, Sarker MH, Momtazul Haque AF. Surgical management of scalp arterio-venous malformation and scalp venous malformation: an experience of eleven cases. Indian J Plast Surg 2013;46(01):98–107
19 El Shazly AA, Saoud KM. Results of surgical excision of cirsoid aneurysm of the scalp without preoperative interventions. Asian J Neurosurg 2012;7(04):191–196
20 Gangadharaswamy SB, Maulyavantham Nagaraj N, Pai BS. Surgical management of scalp arteriovenous malformations using a novel surgical technique. Case series. Int J Surg Case Rep 2017; 37:250–253
21 Goel V, Verma AK, Singh S, Puri SK. Cirsoid aneurysm of scalp: demonstration on CT angiography (CTA). BMJ Case Rep 2013; 2013:bcr2013202459
22 Goya T, Kitano I, Kinoshita K, Mihara K. Congenital arteriovenous malformation of the scalp–report of two cases (author's translation) [original in Japanese]. No Shinkei Geka 1981;9(09):1089–1097
23 Hasturk AE, Erten F, Ayata T. Giant non-traumatic arteriovenous malformation of the scalp. Asian J Neurosurg 2012;7(01):39–41
24 Kaufman SL, Kumar AAJ, Roland JMA, et al. Transcatheter embolization in the management of congenital arteriovenous malformations. Radiology 1980;137(1 Pt 1):21–29
25 Konishi Y, Tamagawa T, Hara M, Takeuchi K, Toyoda H. Cirsoid aneurysm of the scalp: case report [in Japanese]. Neurol Med Chir (Tokyo) 1982;22(08):673–676
26 Kuroki K, Taguchi H, Sumida M, Eguchi K, Saitoh Y. A case of hemorrhagic non traumatic arteriovenous fistula of the scalp [in Japanese]. No Shinkei Geka 1999;27(09):851–853
27 Mohamed WN, Abdullah NN, Muda AS. Scalp arteriovenous malformation: a case report. Malays J Med Sci 2008;15(03):55–57
28 Mohanty S, Rao CJ. A large cirsoid aneurysm of the scalp associated with epilepsy. J Neurol Neurosurg Psychiatry 1976;39(09): 835–836
29 Nagasaka S, Fukushima T, Goto K, Ohjimi H, Iwabuchi S, Maehara F. Treatment of scalp arteriovenous malformation. Neurosurgery 1996;38(04):671–677; discussion 677
30 Nishimura T, Kubota S. A case of congenital AVM in temporoparietal muscle [in Japanese]. No Shinkei Geka 1996;24(03):277–280

31 Nishiura T, Nishida A, Handa A, Gotoh M, Tsuno K, Ishimitsu H. Congenital arteriovenous malformation of the scalp with a drainage to the transverse sinus: a case report [in Japanese]. No Shinkei Geka 1999;27(10):895–901
32 Ohno K, Tone O, Inaba Y, Terasaki T. [Coexistent congenital arteriovenous malformation an aneurysms of the scalp (author's translation) [in Japanese]. No Shinkei Geka 1981;9(10): 1187–1191
33 Oldfield MC, Addison NV. Cirsoid aneurysms of the scalp. BMJ 1962;2(5296):23–24
34 Shenoy SN, Raja A. Scalp arteriovenous malformations. Neurol India 2004;52(04):478–481
35 Shimoda M, Matumae M, Shibuya N, Yamamoto I, Sato O. Three cases of scalp arteriovenous malformations [in Japanese]. No Shinkei Geka 1987;15(02):173–178
36 Stucker FJ. Extracranial arteriovenous fistulas. Laryngoscope 1974;84(06):970–975
37 Takahashi S, Furuhashi N, Koshu K. Congenital arteriovenous malformation of the extracranial region–report of a case with review of literatures (author's translation) [in Japanese]. No Shinkei Geka 1977;5(08):889–893
38 Tani S, Kawamoto K, Kawamura Y, Matsumura H, Tanaka M, Ikeda K. Vascular tumor of the scalp. Case of hemangioendothelioma and two cases of cirsoid aneurysm [in Japanese]. Neurol Med Chir (Tokyo) 1983;23(09):755–760
39 Tauro LF, Suhith G, Shetty P, Rao D. Cirsoid aneurysm of scalp. J Neurosci Rural Pract 2012;3(01):95–96
40 Vasconez LO. Congenital defect of the skull and scalp due to an arteriovenous malformation. Case report. Plast Reconstr Surg 1973;51(06):692–695
41 Waga S, Otsubo K, Handa J, Handa H. Extracranial congenital arterio-venous malformations. Surg Neurol 1974;2(04):241–245
42 Worm PV, Ruschel LG, Roxo MR, Camelo R. Giant scalp arteriovenous malformation. Rev Assoc Med Bras (1992) 2016;62(09): 828–830
43 Yamaki T, Yoshino E, Uchibori M, Odake G, Hirakawa K. Artificial embolization with cyanoacrylate for the treatment of the extracranial arteriovenous malformation [in Japanese]. No Shinkei Geka 1982;10(09):991–995
44 Yoneda S, Matsuda M, Shimizu Y, Goto H, Handa H. Electrothrombosis of arteriovenous malformation. Neurol Med Chir (Tokyo) 1977;17(1 Pt 1):19–28