Arteriovenous Malformations of the Scalp

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ARTERIOVENOUS MALFORMATIONS (AVMs) of the scalp, though rare, present an interesting challenge to the surgeon. Their basic etiology is not clear. They may result in complex derangements of cerebral hemodynamics, and their surgical treatment can be extremely difficult.

The purpose of this report, in addition to the presentation of six illustrative cases selected from a series of 10 patients, with 20 years' follow-up in two, is to discuss the causes of this abnormality, its incidence, and other important characteristics which are not available in the literature. It is based on review of 148 cases reported in the literature and the present series of 10 patients.

Case Reports

Group One: Patients with no history of trauma and no birth-mark.

Case 1. A 32-year-old man noticed a mass in the mid-frontal region behind the hairline 2 years prior to admission. He complained of no headaches; however, nervous excitement caused tortuosity and enlargement of the mass which was associated with throbbing and tenderness. Physical examination showed a round, irregular and pulsating mass in the midline, anterior to the coronal suture. It measured 5 cm. in diameter and 1.5 cm. in height.

The right internal carotid angiogram revealed no abnormality. The right superficial temporal angiogram showed moderate dilatation of this artery and its frontal branch with a serpiginous collection of moderately dilated vessels in the region of the bregma. There was no evidence of connection between the dilated superficial vessels and the cerebral vasculature.

The entire mass was removed following ligation of the feeding vessels. Twenty years later, the patient remains well with no sign of recurrence.

Case 2. A 44-year-old woman was admitted for the third time because of scalp bleeding. The patient's history dated back to the age of 18 years when she noted a pulsating lump in the right parieto-occipital region. A partial excision of the lesion together with multiple ligation of the feeding arteries was carried out. She had no further severe bleeding until 23 years later when uncontrollable bleeding occurred. To stop the bleeding, the right common carotid artery was ligated and a partial excision of the recurrent AVM was performed. The bleeding, however, still remained troublesome. A left carotid angiogram revealed filling of the AVM. Thus, the left external carotid was ligated. She had several skin grafts in the occipital region and on one occasion she developed osteomyelitis of the skull. The patient was finally discharged after 8 months of hospitalization with some improvement.

She was readmitted 8 months later because of bleeding, and the AVM was injected with sclerosing solution.

At the present time the patient is well except for the occasional scalp bleeding which may be severe enough to require blood transfusion. Currently, she is receiving cobalt treatment for the remaining AVM in her hometown hospital.

Case 3. A 24-year-old woman was admitted because of blurred vision. Ten years previously the patient gradually developed loss of hair in the right parietal scalp, prominent vessels, headaches, and tinnitus. The diagnosis of a right parietal scalp AVM was made by carotid angiography and the right external carotid was ligated. A few months later, the tinnitus recurred and the vessels became dilated. Surgical procedures such as partial excision and ligation of the feeding arteries were carried out with little success. Later, she noted some visual blurring and examination revealed 4 diopters of papilledema bilaterally. Skull X-rays showed a thick calvarium with markedly accentuated vascular impressions. Angiography demonstrated a diffuse AVM of the right scalp which was supplied by the branches of the left external carotid and both vertebral arteries as well as the intracavernous branches of both carotid arteries. Lumbar puncture revealed clear cerebrospinal fluid with an opening pressure of 450 mm. H_2O, 12 mg./100 ml. protein, 65 mg./100 ml. sugar, 8 crenated red blood cells and no white blood cells.

After preparation of a large pedicle graft, a complete excision of the AVM together with the involved bone was attempted. Bleeding from the scalp and bone required repeated blood transfusions and was controlled with difficulty. Following operation her condition was satisfactory for a few days; however, later she developed signs of progression of the increased intracranial pressure and elevation of the skin graft. The patient was operated on

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again on the eighth postoperative day and a large epidural clot together with a very swollen brain were found. She did not respond to the treatment and died 8 weeks after excision of the AVM.

Group Two: Patients with no history of trauma but with birth-mark.

Case 1. A 31-year-old woman was born with port-wine stains on the right side of the head, ear, and cheek, in which area she gradually developed throbbing pain and pulsation. During her first pregnancy the symptoms became more severe with occasional bleeding so that it was terminated by Caesarian section in the seventh month due to fainting and bleeding episodes.

Following pregnancy, a right carotid angiogram showed an AVM in the distribution of the right external carotid artery. At operation, this artery was ligated and partial resection of the AVM was carried out with relative improvement. A month later, the symptoms recurred and gradually increased in severity. The right carotid angiogram was repeated which showed the remaining AVM. She was operated upon and partial excision was done. After a few years, the previous symptoms recurred and an angiogram showed that the AVM was fed by branches of the thyrocervical trunk. She was operated on for the third time and excision and multiple ligations were performed with relief of symptoms.

Group Three: Patients with history of trauma but no birth-mark.

Case 1. A 35-year-old man was stabbed in the left temple 15 years previously. He was taken to a local hospital and the wound was repaired. He subsequently noticed swelling and tenderness at the site of the stab wound and as time went by throbbing headaches and buzzing in the left ear developed. The physical examination revealed dilated and tortuous vessels over the entire scalp which were more prominent on the left side. A loud bruit was present in the left temporal area.

Left internal and external carotid angiogram with subtraction films showed interruption of the superficial temporal artery at the level of the preauricular region with huge recurrent branches of the internal maxillary artery, posterior auricular artery and occipital artery draining into the preauricular region (Fig. 1). The left external jugular vein was extremely large and opacified early (Fig. 2). The arterial circulation time was 2¼ and the arteriovenous circulation time was 4¾ seconds.

The abnormal vessels in the left temporal region were removed in block following ligation of the external carotid artery. His symptoms were relieved after the operation. However, long-term follow-up was not available.

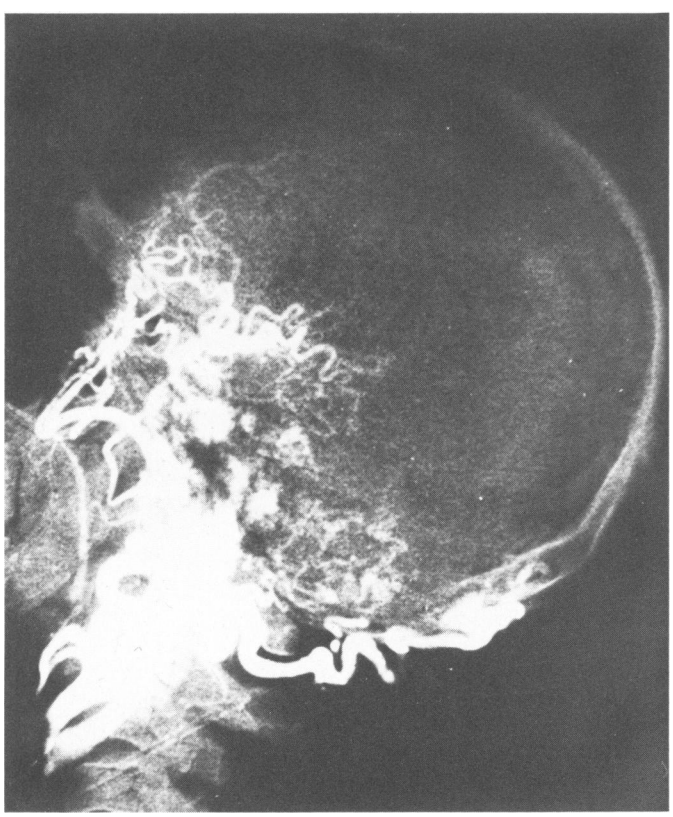

FIG. 1. An early phase of a left lateral carotid angiogram which shows dilatation of the occipital and internal axillary arteries. The superficial temporal artery which was interrupted at the time of injury is not seen in this phase.

Group Four: Patients with history of trauma and birth-mark.

A 42-year-old man had multiple birth-marks on the left side of his body involving the temple, upper neck, buttock and the calf. At the age of 16 years, he was involved in a car accident and was injured about the left forehead. Following the accident he noticed throbbing in the left side of his head and gradually the vessels in this area became enlarged and distended. The physical examination revealed an area of port-wine discoloration together with an irregular pulsating mass in the left temporal region. Bruit was present and diminished by compression of the superficial temporal artery. In an operation the left external carotid artery was ligated and a partial excision of the lesion was carried out.

The patient was relatively symptom free for about 6 years, then the throbbing and swelling recurred. He was subjected to three operative procedures consisting of multiple ligations and excisions with some relief.

Terminology and Historical Background

Arteriovenous malformations have been termed: arteriovenous aneurysm,[29,37] cirsoid aneurysm, racemose aneurysm, aneurysm by anastomosis, plexiform angioma,[33] aneurysmal varix,[28] arteriovenous fistula,[13] abnormal arteriovenous communication,[37,38] and have been known for centuries.[48]

The Greek legend of the "Gorgons" head covered by snakes has been suggested by Vollmar and associates[48] as an early description of this disease. These authors also quoted a case of "Ranken angioma" from the sixteenth century for which surgical treatment was attempted.

During the latter part of the nineteenth century the presence of free communications between an artery and a vein was recognized and reports of traumatic arteriovenous malformations appeared in the literature.[26,51] The congenital form was not described until studies of the development of the vascular system in embryos were done during the early part of this century.[39] Some years later the pathology of the arterio-

venous malformation was studied in more detail and it was shown that no capillary network exists between communicating arteries and veins.[25]

Etiology

Four major causes have been considered.

1. *Congenital AVMs*

Case reports such as that of Davies-Colley (Case 1)[10] or the Group Two patient of the present study in whom an AVM developed at the site of a red birthmark without any history of trauma may be considered congenital. Nevertheless, a congenital AVM may exist without any apparent local abnormality at birth, or a birthmark may be present without ever being manifested as an AVM.

Three explanations have been suggested for the development of congenital AVMs.

A. Persistence of the primitive arteriovenous communication and capillary agenesis.[20,28,53]

According to this theory, the development of the primitive arteriovenous communication into arteries, capillaries and veins at 4 weeks gestation (4 mm. embryo) fails to take place; instead, the embryonic plexus-like arteriovenous pattern without interposition of the capillary network is retained.

B. Hamartoma. Some authors believe that AVMs originate from vascular hamartomas.[50,52] Hamartomas are tumor-like, non-neoplastic lesions which are characterized by abnormal mixture of tissues indigenous to the part, with an excess of one or more.

C. Formation of a fistula at the site of arteriovenous crossing.[3,18] After most cranial arterial walls become thickened (40 mm. embryo) arteries and veins frequently cross and accompany each other. Since these vessels at the site of crossing are only separated by a double layer of endothelial cells, a fistula could develop and cause abnormally dilated vascular nets.

The concept of hamartoma seems to offer a better explanation for the common cause of AVMs than the persistence of primitive blood vessels or the formation of a fistula at the site of arteriovenous crossing. A hamartoma formed of primitive vascular plexus on the basis of a larger vascular bed may attract more blood than the normal primitive vascular plexus thus causing its own persistence and since there is no tissue to supply, capillaries do not develop and differentiation does not take place.

It is of note that direct arteriovenous communications are also found in blood vessel tumors such as hemangiomas[8,36] and in highly vascular neoplasms like hemangioblastomas,[35] meningiomas, gliomas and some of the metastatic tumors[12] where again a relative crowding of blood vessels and a larger vascular bed exists.

Activating factors of the congenital AVM: Symptomatic congenital AVMs are not common immediately after birth or during childhood. Most patients usually seek medical attention after the second decade. A few factors are said to be capable of establishing an active or perhaps more active blood circulation in the malformation and cause an asymptomatic AVM to become symptomatic. These include mechanical trauma, vasomotor disturbance, endocrine stimulation, and inflammation. Mechanical trauma including sneezing and parturition may cause a sudden increase in intravascular pressure and rupture the embryonic septum between the artery and the vein (the two layers of endothelial cells) and produce an arteriovenous fistula.[21] In vasomotor disturbance the increase of hemodynamic activity is considered the activating factor.[23] Whether or not inflammation and endocrine stimulation such as growth, puberty, menstruation, and pregnancy may also act by hemodynamic alteration is not clear.

2. *Traumatic AVMs*

Both penetrating and non-penetrating scalp injuries have been said to cause arteriovenous fistulae which gradually evolve into a malformation. Case reports such as that of Amyes and Courville[1] or group three patient of the present study in which an AVM developed at the site of a penetrating wound strongly suggest a traumatic origin. In cases of non-penetrating wounds, however, one cannot be certain about the trauma being the cause of the lesion, since scalp injuries may also activate a silent and unrecognized congenital AVM or be incidental. On this basis, some of the cases reported in the literature as traumatic in origin may not be so.

A non-penetrating scalp wound which causes an AVM may damage the walls of an artery and a vein in apposition, and produce a fistula which enlarges due to shunting a high pressure arterial blood into a low pressure venous system.

In addition to the scalp injuries received during childhood or later in life, trauma and pressure exerted on the scalp at childbirth have been also considered a possible origin of traumatic scalp AVM.[38] This kind of lesion may be confused with true congenital AVM.

3. *AVMs Caused by Infection and Inflammation*

Although primary arterial disease, ulceration and infection are considered rare causes of arteriovenous fistulae we have not found a reported case of this kind of AVM involving the scalp.

4. *Familial AVM*

Familial scalp AVM seems to be extremely rare. We had the opportunity to observe this condition in a Persian family.[19] To our knowledge, no similar example has been

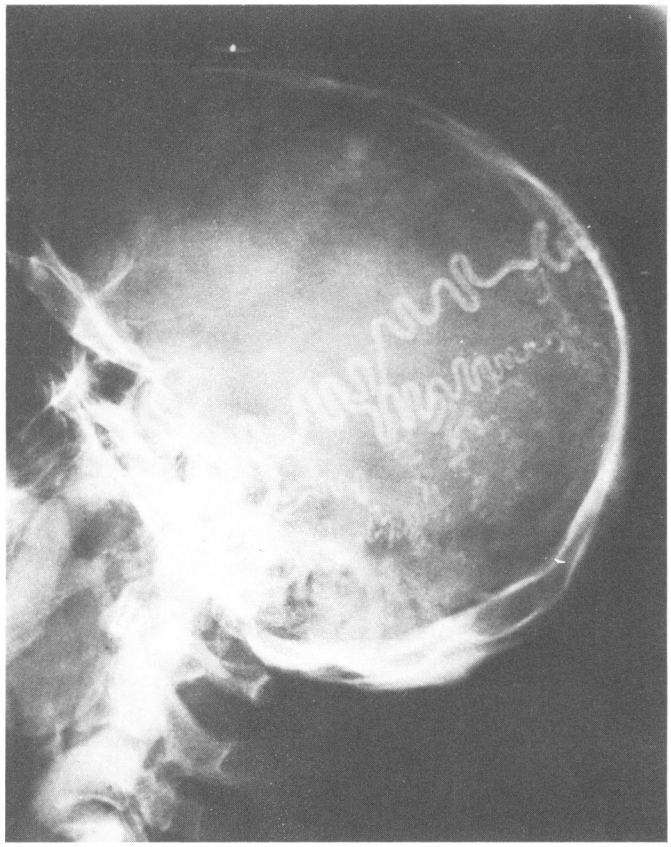

FIG. 2. A later arterial phase of the same angiogram shown in Fig. 1 which demonstrates reformation of the superficial temporal artery in a retrograde fashion by branches of the occipital artery. The greatly dilated external jugular vein shows early opacification.

reported. Tonnis and Lange-Cosack[46] have described an example of familial AVM involving the cerebral vessels.

Incidence

Many authors[1,2,7] believe that AVMs are more common in the scalp than in any other part of the body, and according to Watson and McCarty,[49] over 50% of them occur in the head region. Olivecrona and Ladenheim[28] have reported that scalp AVMs are about 20 times less common than the intracranial AVMs. Of 158 cases of scalp AVMs that were reviewed, in 85 (55%) there was no history of trauma. Of these, 15 patients had either a birthmark at the site of lesion or the lesion was present at birth. Two of these instances occurred in sibs.[19] Of the remaining 73 cases, in 66 (43%) there was a history of non-penetrating or penetrating injury. Three of these patients had port-wine stains which apparently became symptomatic following trauma. Of the remaining seven cases, five were iatrogenic AVMs, due to temporal arteriotomy[6,16,22] and two were possible false aneurysms.[4,40]

Some authors consider the congenital AVM to be more common[1,15,47] and others express an opposite view.[17,42] Owing to the difficulties in distinguishing between the true congenital and the traumatic AVMs, relative incidence of these lesions cannot be established with certainty.

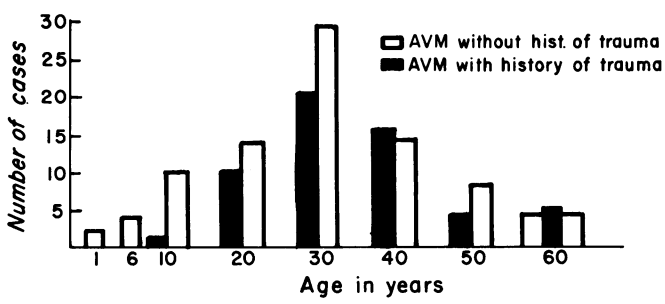

FIG. 3. Age incidence of 143 scalp AVMs. Fifteen patients in whom the age was not recorded are not included.

Age

The third decade was the period in which the highest incidence was found in both congenital and traumatic forms. The youngest patient of the non-traumatic group was 3 months and the oldest 59 years. In the traumatic group, the youngest one was 8 years and the oldest 61 years (Fig. 3).

Sex

In both groups, the condition was more common in males than in females. Of 84 patients with no history of trauma in whom sex was given, 48 (60%) were male and 36 (40%) were female. Of 68 patients with history of trauma 46 (66%) were male and 22 (34%) were female.

Location

In both groups the AVM occurred almost equally in the frontal, temporal, and occipital scalp (Table 1). The incidence of AVM in these regions was almost two times greater than the posterior auricular and parietal areas. No traumatic AVM was found in the posterior auricular region.

Combined Extracranial-Intracranial AVM

AVMs involving both intracranial and extracranial vasculature[5,9,14,34] are thought to arise from a single embryonic maldevelopment which has failed to be split completely into layers.[14,28] This concept is not in agreement with the principle of development of the head vasculature in the embryo. The brain arteries are derived from the internal carotid arteries (they are later connected with the vertebrobasilar system) during the stage that the embryo is about 30 days old and 5 to 8 mm.[30,31] The scalp arteries are derived from the external carotid arteries in a later stage when the embryo is about 6 weeks old and 18 mm. to 26 mm. Before this stage, the thin

TABLE 1. *Distribution of 148 Scalp AVMs. Five Iatrogenic Malformations and Two Possible False Aneurysms are not Included. In Three Additional Cases the Site of the Lesion was Not Recorded.*

	FRONTAL					PARIETAL				TEMPORAL					OCCIPITAL				POSTERIOR AURICULAR				HALF OF THE SCALP				ENTIRE SCALP	TOTAL
	Right	Left	Bifrontal	Side not given	Total	Right	Left	Biparietal	Total	Right	Left	Bitemporal	Side not given	Total	Right	Left	Bioccipital	Total	Right	Left	Side not given	Total	Right	Left	Side not given	Total		
AVMs without history of trauma	7	5	6	1	19	4	1	2	7	9	6	0	2	17	4	6	9	19	5	1	2	8	4	5	0	9	4	83
AVMs with history of trauma	6	7	5	0	18	3	1	1	5	11	11	1	0	23	2	5	4	11	0	0	0	0	0	3	1	4	4	65
Total					37				12					40				30				8				13	8	148

scalp covering the membranous skull is avascular and is separated from the skull by a considerable interval of avascular tissue.[30] The emissaries later develop and communicate the intra- and extracranial vasculature.

Thus, it seems that there is a separate origin for the scalp, dura, and cerebral congenital AVMs and when a combined extracranial-intercranial AVM occurs it is due to a secondary communication.

Communication of the Scalp AVM with Intracranial Venous Sinuses

No example of this abnormality was found among the reported traumatic AVMs. Congenital arteriovenous communication between the occipital artery and transverse sinus has been reported.[11,27,41,47] We have also observed communications between a scalp AVM and superior sagittal sinus.[19]

In the embryo, the dural sinuses originate from the dural plexus and develop independently from the scalp and the brain vasculature. The superior sagittal sinus is formed by an anterior subdivision of the plexus when the embryo is 6 weeks old. The transverse sinus develops from the tentorial plexus and later joins the superior sagittal sinus.[43]

It appears that the sinus communication of the scalp AVM develops in a later period when the emissary veins are formed, and depending on the location of AVM, the communication takes place either through the persistent primitive vessels or ordinary emissary veins. The size of the skull defect through which the communicating vessels pass is determined by the diameter of these communicating structures.[19]

Association with Other Abnormalities

Multiple intracerebral AVMs and association of intracerebral AVM and congenital aneurysm are occasionally reported.[32,41,45] No such multiplicity or association was noticed in the cases reviewed in the present study. Nevertheless, a patient with congenital venous malformation of the scalp associated with plexiform neurofibroma and cranial defect has been recently described.[24]

Clinical Picture and Treatment

Symptoms and signs of the scalp AVM are well described and they range from a simple disfiguration to a life threatening hemorrhage. Tinnitus and throbbing which are aggravated by physical activities may become most troublesome to the patient. Thrill and bruit are usually present. Papilledema is uncommon and its pathogenesis is not clear. Cardiac symptoms and failure are rare complications of the scalp AVM. An angiogram discloses the nature of the disease. The ideal treatment is complete excision of the AVM as is demonstrated in Case 1 in Group One. Partial excision and ligation of the feeding vessels or carotid artery do not usually bring permanent relief (Case 2, Group One). Other methods of therapy such as x-ray radiation, injection of sclerosing solution, electrocoagulation and compression are rarely successful. It is also doubtful that artificial embolization could be used as a definitive therapy, unless it is combined with total resection.

It is best to identify the feeding arteries from the angiograms and ligate them first before exposing the AVM. A skin flap larger than the main bulk of the lesion is preferable for the ease of dissection and reduction of

the bleeding. When possible, the AVM is removed in one piece, piece-meal excision increases the amount of bleeding and the operating time. Whatever surgical technic is used total excision is essential for permanent cure.

Summary

Six illustrative instances of arteriovenous malformations of the scalp, one of which proved fatal are presented. A further 152 cases are analyzed. Of 158 cases, 85 patients (55%) had no history of scalp injury. Of these, two occurred in sibs. Of the remaining 73 cases, 66 patients (43%) had a history of non-penetrating or penetrating scalp trauma, five had iatrogenic AVMs and two were thought to be false aneurysms. Sixty per cent of the instances without history of trauma and 66% of the instances with history of trauma occurred in men. The highest incidence in both groups were seen during the third decade. The common sites for both groups were frontal, temporal, and occipital regions. The parietal and posterior auricular areas were less commonly involved. On the basis of the cerebrovascular embryology, it is stated that there ought to be a separate origin for the scalp, dura, and cerebral congenital AVMs and when a combined extracranial-intracranial AVM occurs it is due to a secondary communication. Finally, it is concluded that the best therapeutic results are obtained by early diagnosis and complete surgical excision.

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Section VI. Arteriovenous malformations. J. Neurosurg., **25**:467, 1966.

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