# CASE REPORT

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# Cranial Arteriovenous Malformation: Suicide by Exsanguination

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**ABSTRACT:** Arteriovenous malformations of the scalp are potential sources of serious bleeding because of their location, inherent weakness, and high flow rates. A 40-year-old man used his vascular lesion as a means of suicide. Selected aspects of the historical background, diagnosis, and treatment of these vascular disorders are described.

**KEYWORDS:** pathology and biology, arteriovenous malformation (AVM), suicide, scalp, exsanguination

The rupture of abnormal, thin-walled vessels of several parts of the body surface, with rapid exsanguination leading to death, has long been recognized as a hazard accompanying certain superficial vascular disorders. One of the most dramatic accounts of sudden external bleeding was provided by the great anatomist John Baptist Morgagni in his characterization of an eroding aneurysm of the aorta involving the thoracic wall, which fulfilled a prediction of explosive rupture and immediate death. In his final moments the patient fastidiously attempted to catch the flow in a basin [*1*].

Arteriovenous malformations of the scalp may present a risk of severe and possibly fatal external hemorrhage, sometimes initiated by trauma. The intentional initiation of bleeding from a cranial arteriovenous malformation as a means of suicide has, to our knowledge, not previously been described.

## Case Report

A 40-year-old man was evaluated in an outpatient setting because of the development of an occipital vascular scalp abnormality. There was a history of several bar fights approximately 6 years earlier, but the extent of any trauma to the scalp was unknown. The patient was neurologically normal. A computer scan disclosed normal intracranial contents and a collection of very large soft tissue densities in the scalp, particularly in the occipital region (Fig. 1). Angiographic injection of the left external carotid artery disclosed the presence of a

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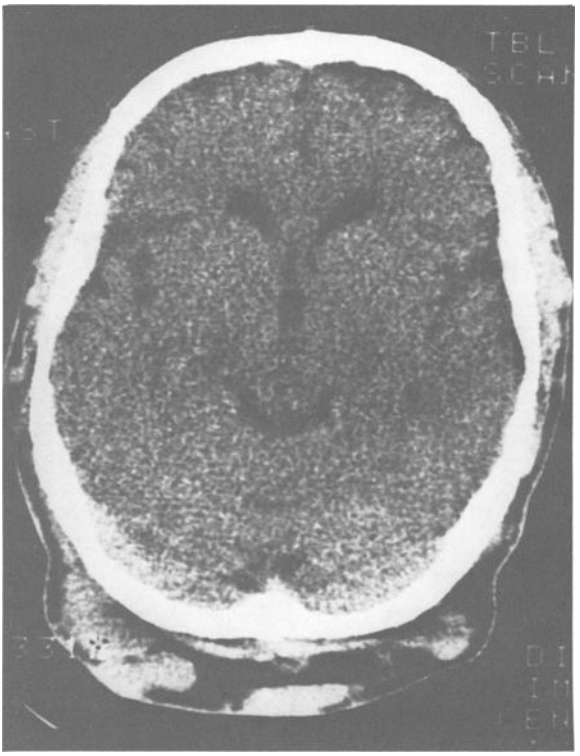

FIG. 1—*A large complex posterior extracranial soft tissue mass is seen on the computed scan.*

large arteriovenous malformation of the scalp, with major contributions by the left occipital artery and the left superficial temporal system (Fig. 2*a* and *b*). A right external carotid study demonstrated similar massive dilatation of the right occipital and superficial temporal arteries. The intracranial circulation was normal.

The patient was referred for consideration of treatment of the malformation, and reportedly was discouraged by a comment that surgical resection might be difficult. He was also despondent in relation to domestic and financial circumstances. During the evening following consultation with the surgical specialist, he drank heavily in the company of friends, and made several comments regarding suicide. Within 5 to 10 min of his departure for home he was seen sitting on a bench in a pool of blood. Police officers arrived immediately and found him slumped forward on the bench. A package of double-edge razor blades was found nearby; one blade had been used and lay beside the patient. Despite immediate attention at a local hospital, the patient could not be resuscitated. A 3-cm laceration of the occipital scalp was present. Postmortem examination was remarkable only for the presence of macronodular hepatic cirrhosis, suggesting the possible role of undocumented compromise of the coagulation process in this case.

## Discussion

Arteriovenous malformations involving the scalp may be of congenital or traumatic origin. They commonly present as a subcutaneous swelling that enlarges slowly, is some time noted to begin to pulsate, and leads to a wide variety of surface patterns, some grossly disfiguring.

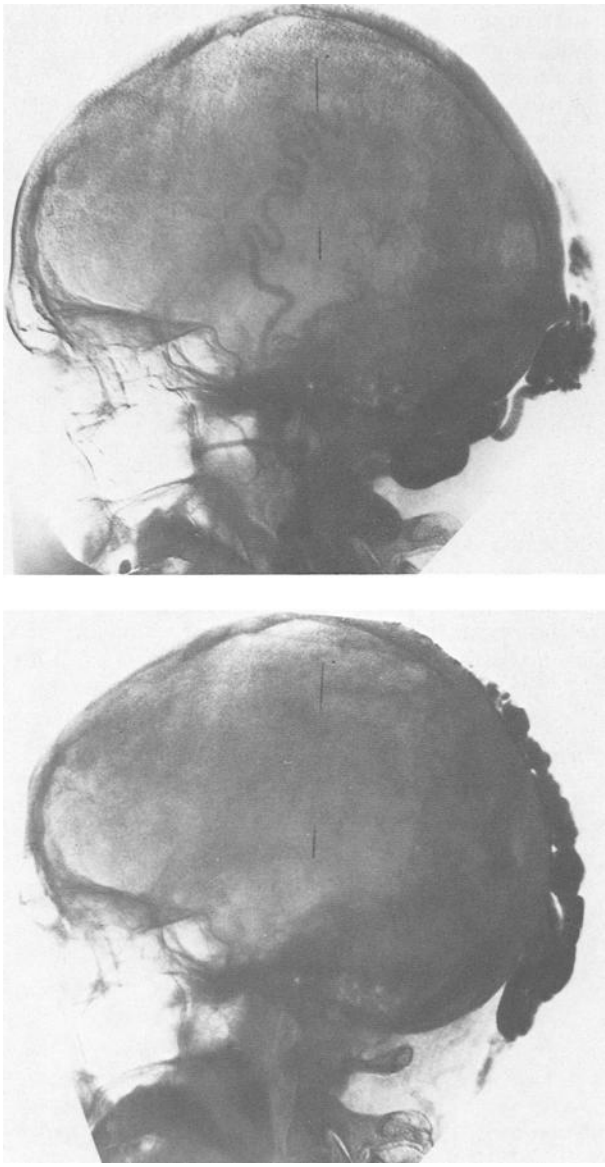

FIG. 2—*The large extracranial malformation is fed by dilated superficial temporal* (top) *and occipital* (bottom) *branches of the external carotid artery.*

These lesions produce headache and subjective bruits, and may vary in apparent size with postural changes. They represent the persistence of the arteriovenous shunts which are present in early embryonic life, with failure of development of mature arteries, veins, and capillaries [2]. The frequently used term "cirsoid aneurysm" refers to the appearance of one or more dilated superficial varices. These may become very large, with a complex system of dilated, thin-walled, incompletely supported vessels, and high-pressure, high-volume flow characteristics.

The more common congenital scalp lesions frequently receive an intracranial arterial supply by middle meningeal branches passing through the skull [3]. They may be fed by the internal carotid arterial system [4]. Asymptomatic arteriovenous malformations of the brain may accompany the visible scalp lesion, and recognition of such an association is essential in evaluation and treatment planning. Supply of the scalp lesion by greatly enlarged extracranial branches of the external carotid system, without intracranial contributions, may more frequently characterize lesions of traumatic etiology.

Although computed tomography, magnetic resonance imaging, or other diagnostic measures may suggest the presence of a cranial vascular lesion, complete angiography, including vertebral and internal and external carotid injections, remains the definitive method of demonstration of the existence and extent of both extracranial and intracranial arteriovenous malformations. In the present case, computed tomography showed engorged occipital vessels, but only angiography delineated the location and vascular supply of the lesion.

In 1829 Benjamin Brodie reported the successful treatment of an arteriovenous malformation of the scalp by circumferential ligation, based in part on surgical techniques described by Sir Charles Bell [5,6]. Dandy has summarized the early development of therapeutic approaches to these often complex lesions [2]. He recognized that only complete excision can be expected to effect their cure.

Thorek has noted that "hemorrhage is the greatest danger" of vascular malformations of the scalp [7]. The rate and volume of blood loss may be affected by the number and size of injured vessels, changes in systemic arterial pressure, head position, and integrity of coagulation mechanisms. The vessels in these lesions, like those within the arteriovenous malformations of brain, are markedly abnormal. As a result, the control of hemorrhage by vascular spasm, with the critical closing pressure mechanism of arteriolar vasomotor tone, is not available. With the possible exception of bleeding into the subgaleal space, no natural mechanism of tamponade exists, and the risk of exsanguination is present in untreated cases. Nevertheless, fatal external hemorrhage from preexisting cranial vascular lesions remains uncommon. Its deliberate provocation is apparently unique.

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