ARTERIOGRAPHY IN CEREBRAL ARTERIOVENOUS MALFORMATION

ROBERT E. WISE, M.D., and ROBERT M. GEIST, JR., M.D.
Department of Roentgenology

THE term cerebral arteriovenous aneurysm or malformation is used to designate that type of cerebral vascular lesion in which the arterial and venous circulations are connected by a nest or cluster of racemose vessels.

Such lesions have been considered relatively uncommon. Cushing and Bailey[1] in their analysis of 2023 proven brain tumors found fewer than 1 per cent, while Dandy[2] quoted a similar figure. Ray,[3] in 1941, reported 6 cases of his own and found approximately 75 in the literature. Five cases were reported by Hodes et al[4] in 1947. However, in a series of 150 arteriograms made in a period of less than 2 years we have demonstrated 8 arteriovenous malformations. It therefore appears that this is a more common entity than was believed originally.

These malformations most often involve the middle cerebral artery and are considered congenital in origin. The arteries supplying the lesions are dilated and the associated veins are large, tortuous and pulsating. Calcium deposits in and adjacent to the vessels of the malformation are common; numerous thrombi may be present. It is these degenerative changes which predispose to subarachnoid hemorrhage and cortical damage.

The patient often presents a history of epileptic seizures, migraine type headaches, and permanent or transient paralysis of varying degree. The neurologic pattern is usually bizarre, and accompanied by such changes as cranial nerve paralysis, hemiparesis, vague sensory alterations, and visual field defects. On auscultation of the calvarium a bruit may be heard. The patient frequently is not aware of this. The eye grounds may reveal dilated tortuous vessels and papilledema which is considered the result of increased venous pressure rather than increased intracranial pressure. Spinal fluid dynamics are almost always normal and fluid examination is normal except in cases of recent subarachnoid hemorrhage. This may be the first sign of the presence of a vascular malformation. Electroencephalography is normal unless there has been some secondary cortical damage.[5]

The plain skull films show varying changes depending upon the size and location of the lesion.[4,6] Frequently the diploe are enlarged. The foramen spinosum, foramen transversarium, and the groove of the middle meningeal artery may be enlarged. The presence of dilated vessels against the calvarium results in signs of local pressure atrophy. Because of the increased vascularity an overgrowth of the bones of the calvarium may result. Calcification is sometimes seen in the vessels of the malformation and in the adjacent nervous

tissue. Only rarely signs of increased intracranial pressure are demonstrated. These are observed when the malformation involves the great vein of Galen which enlarges and produces pressure against the aqueduct of Sylvius resulting in an internal hydrocephalus.[7]

The encephalogram frequently fails to demonstrate any sign of the presence of the lesion.[8] Occasionally changes indicative of a space-taking lesion may

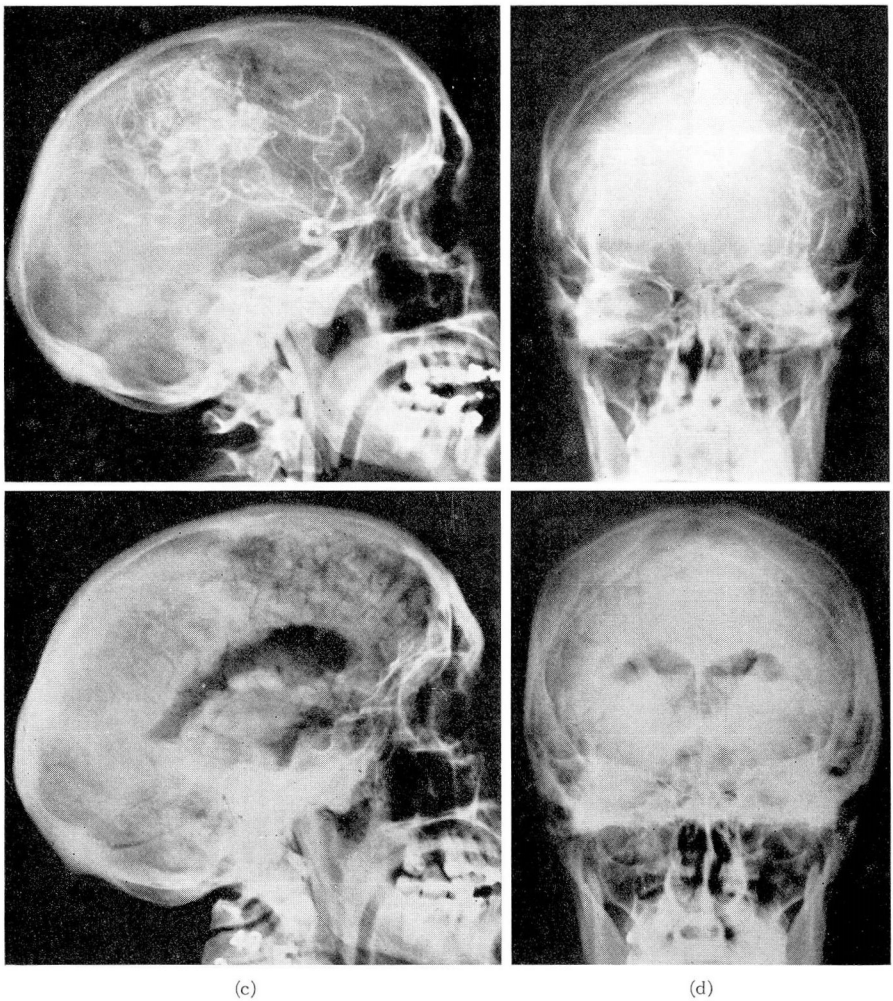

FIG. 1. (a and b) Dilated anterior cerebral artery leading to large cluster of well-developed, irregular vessels in left frontoparietal region. Associated veins dilated and tortuous. (c and d) Lateral ventricles slightly larger than usual and cortical sulci slightly exaggerated.

be present but a specific diagnosis is not usually possible. Ventriculography is considered dangerous because of the possibility of puncture of one of the involved vessels.

Cerebral arteriography offers a means of positive preoperative diagnosis of arteriovenous malformation and enables one to evaluate the lesion accurately and to decide upon the proper course of therapy. The nest of dilated dye filled vessels is easily recognized. Usually both arterial and venous components are visualized in the arterial phase of the arteriogram due to the accelerated circulation time.

The technic of arteriography is neither difficult nor dangerous. It is best performed under general anesthesia. Sodium pentothal is used routinely at the Cleveland Clinic. We prefer the use of 35 per cent diodrast as a contrast medium because of the somewhat questionable danger of thorotrast.[9] The closed or percutaneous method of injection is employed routinely. With the patient supine and the head immobilized with adhesive tape on a radiolucent rest an 18 gauge needle is inserted into the carotid artery. Ten cc. of diodrast is injected rapidly. A lateral film is made, diodrast is injected again and a posterior-anterior film is made.

Several methods of therapy are used in the treatment of arteriovenous malformation.[2] Among these are carotid artery ligation, decompression, surgical removal, and roentgen therapy.

Case Report

A 33-year-old white man was first seen at the Cleveland Clinic on October 18, 1948, complaining of weakness of his right hand and foot of 2 weeks' duration. He had first experienced difficulty 12 years previously when he awakened suddenly and noticed numbness of the right hand, and tingling of the right shoulder, right leg, and right side of the trunk. He lost consciousness for about 15 minutes and was later informed that convulsions had involved the right side during this period. At the same time the tongue was injured. Subsequent treatment consisted of anticonvulsant medication. For the next 10 years he experienced attacks of a similar nature annually. Two years prior to admission medication was interrupted for several days and he underwent a severe attack which lasted between 20 and 40 minutes; thereafter the attacks were increasingly frequent. Two weeks before admission weakness of the right extremities was first noted.

Past medical history was not significant, nor was family history.

Neurologic examination revealed an absence of deep and superficial reflexes over the left side of the body and diminished or absent reflexes over the right side. There were no pathologic reflexes. There was a lack of co-ordination on the right in response to finger to nose and heel to knee tests. The left side of the face and right side of the body showed slight impairment of sensation to pain and touch; the left temporal region was slightly tender. There was a paresis of right upper and lower extremities. The right hand and foot showed pallor and there was some unsteadiness and deviation of gait to the right.

A spinal puncture was performed. Dynamics and fluid examinations proved to be normal. Urinalysis and blood studies revealed no abnormalities. Visual field examination was slightly suggestive of bitemporal defect. An audiogram was normal.

Because of the long history of Jacksonian seizures in this case, a vascular anomaly was suspected and arteriography decided upon.

A left carotid arteriogram (figs. 1a and b) showed a dilated anterior cerebral artery leading to a large cluster of well-developed, irregular vessels in the frontoparietal region sharply demarcated at the midline. Associated veins were diluted and tortuous.

An encephalogram (figs. 1c and d) revealed lateral ventricles which appeared somewhat larger than usual with cortical sulci slightly exaggerated.

The diagnosis was arteriovenous malformation and roentgen therapy was considered the treatment of choice. Sufficient time, however, has not elapsed for evaluation.

Summary

The case described demonstrates the classic findings of intracerebral arteriovenous malformation. A large malformation is demonstrated on the arteriogram. However the lack of specific change on the encephalogram illustrates the inadequacy of air studies in lesions of this type.

We believe that cerebral arteriography is indicated in all cases which present a perplexing neurologic problem and particularly in those with a history of epilepsy or spontaneous subarachnoid hemorrhage.

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