Total excision of an arteriovenous malformation of the corpus callosum

Case report

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Cerebral arteriovenous malformations involving the corpus callosum are usually regarded as inoperable because of their location. The first publication of a surgical attempt on such a lesion seems to be that of Basset in 1951. Recently, Seljeskog, et al., described the complete removal of a similar lesion which involved the inferior sagittal sinus in an infant. Dany, et al., reported in the French literature three cases operated on with good results. In only one, however, was the angiomatous malformation removed completely; in the other two, control postoperative angiography disclosed fistulous vessels remaining. We are reporting the total excision, under operative angiographic control, of an arteriovenous malformation of the corpus callosum.

Case Report

A 7½-year-old boy was referred for surgery on March 3, 1968, with the diagnosis of pericallosal arteriovenous malformation. One year earlier he had been admitted to this hospital in deep coma. Large subhyaloid hemorrhages were then observed in the ocular fundi; the pupils were maximally dilated and non-reacting; there was decerebrate rigidity, which later on resolved into left spastic hemiplegia in extension. The cerebrospinal fluid (CSF) was grossly hemorrhagic. Right cerebral angiography demonstrated a shift of the anterior cerebral artery to the left and a cluster of vessels at the anterior part of the pericallosal artery (Fig. 1). Left carotid and vertebral angiography excluded alternative blood supply to the malformation. The boy was in coma for 2 days and subsequently remained lethargic for 3 weeks. He then gradually recovered consciousness. He was discharged on April 3, 1967, with markedly spastic left hemiparesis. Since that time he had complained occasionally of severe headaches and blurred vision, sometimes diplopia, but not further episodes of loss of consciousness.

Examination. On admission to our depart-

Fig. 1. Right cerebral angiogram, lateral view, showing shift of the anterior cerebral artery to the left and a cluster of vessels at the anterior part of the pericallosal artery (March 2, 1967).
ment the child was frail and pallid; he responded to questioning with lucid and intelligent answers. He dragged the left leg heavily and complained of dizziness and headaches, particularly when walking. There was a severe left spastic hemiparesis with sustained ankle clonus and a left Babinski response. There was papilledema but no hemorrhages or exudates on the right; the left ocular fundus was normal. The visual fields were full and the visual acuity corrected was 6/6 for each eye. The general physical examination was noncontributory. Blood pressure was 110/70 mm Hg. The electrocardiogram was normal. Skull and chest x-ray films revealed no abnormality. The electroencephalogram was noticeably disturbed, showing bursts of sharp waves over a background of slow wave abnormal activity over the right hemisphere. Combined air and angiographic studies were performed on March 12. Cerebral angiography now showed a noticeable growth of the arteriovenous malformation (Fig. 2). In addition, a large paramedian porencephalic cavity communicating with the lateral ventricle was demonstrated on the pneumoencephalogram (Fig. 3).

Operation. On March 12, a right fronto-temporal flap was turned down under endotracheal anesthesia. A Polyethylene P.E. 160 catheter was advanced into the internal carotid artery for angiographic control curing operation. Controlled respiration and Mannitol were used but no hypothermia or hypo-
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tension. The pericallosal artery was identified and followed distally until a cluster of pulsating vessels extending into the corpus callosum and gyrus cinguli was encountered. The pericallosal artery was not directly involved in the malformation, which was fed by a number of vessels branching off the main trunk. Dissection with the magnifying loupe, and occlusion of the feeding vessels as they were encountered, was carried out with care not to injure the pericallosal artery. Isolation of the malformation was facilitated after the porencephalic parasagittal cavity was penetrated. A large fistulous pericallosal vein draining into the inferior sagittal sinus was identified and occluded. Subsequently, the corpus callosum was incised and additional vessels of the malformation were occluded. Finally, the fistulous connection of the aneurysm with the internal cerebral vein within the cavity of the lateral ventricle was exposed and occluded. The charred vessels of the collapsed vascular malformation were extirpated. The bone and skin flaps were temporarily replaced and an angiogram done. Additional dissection was necessary to remove the clusters of abnormal vessels. A second angiographic control during operation confirmed total excision of the malformation (Fig. 4). The bone flap and the wound were then closed as usual.

Complete angiographic series were carried out in the x-ray department at the end of the operation. Those showed the total removal of the lesion with preservation of the pericallosal artery, which now was filling normally along its length (Fig. 5). The histological examination of the lesion was typical for an arteriovenous malformation.

Postoperative Course. Recovery was uneventful; the child made a quick recovery and left the hospital a month after operation. The hemiparesis improved, there was better control of movements, and walking was less laborious. When last seen in August, 1969, the child was doing well and even participating in games at school.

Discussion

In their cooperative aneurysm study Per-
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ret and Nishioka\textsuperscript{a} found that 81 of 453 intracranial angiomatosus malformations, or 18\%, were intraventricular or paraventricular lesions. The latter included angiomatosus malformations of the basal ganglia, corpus callosum, septum pellucidum,\textsuperscript{b} and the interhemispheric surfaces. Such a relatively high incidence of paraventricular arteriovenous malformations is also suggested in our unselected series of 21 patients treated during a 5-year period for arteriovenous malformations, four of which were paraventricular. This relatively high incidence seems to justify more attention to arteriovenous malformations in this location, since a radical excision of the lesion, when feasible, may result in a cure.\textsuperscript{1-4,10,11} Furthermore, "in patients over 50 years of age the need for excision is less urgent than in younger ones, since progressive enlargement of the malformation is less likely."\textsuperscript{79}

The angiomatosus malformations of the corpus callosum are embedded mainly in its substance\textsuperscript{4} but may also penetrate into the gyrus cinguli,\textsuperscript{4} as in our case. In the interhemispheric cistern, angiomatosus vessels may expand considerably to form aneurysmal ectasias.\textsuperscript{12}

The malformation derives its blood supply from branches of the pericallosal artery. It is possible, therefore, to preserve the continuity of the main arterial trunk. Thus, in our case, following excision of the lesion the blood supply was restored to important cerebral areas along the distal pericallosal artery.

The major venous drainage of a corpus callosum arteriovenous malformation is formed by fistulous channels connecting to the inferior sagittal sinus. The alternative venous drainage through the internal cerebral vein is often found with malformations of the septum pellucidum,\textsuperscript{a} head of the caudate nucleus,\textsuperscript{1} and thalamus.\textsuperscript{10,11} The vein of Galen and the straight sinus are the common final outlets of venous blood from the malformation.

In our case, the main body of the arteriovenous malformation was embedded in the corpus callosum. There were pulsating vessels in the interhemispheric cistern and also in the gyrus cinguli. The gyrus cinguli was thin and represented the medial wall of a large porencephalic cyst. This cavity seems to have been formed by the spontaneous evacuation into the ventricular cavity of the massive intracerebral hemorrhage which occurred at the first bleeding of the malformation 1 year previously. Incidentally, spontaneous evacuation of massive intracerebral hemorrhage seems to explain the survival of the child in our case.

As in a case previously reported, angiographic control during operation was essential for the successful resection of the lesion. Repeated injections\textsuperscript{3} were necessary in our previous case whereas in this one total extirpation was achieved after only one control injection.

The striking growth of the malformation within 1 year, or at least the angiographic evidence for such a growth, is of importance because of the scant information contained in the literature in this regard. Olivecrona and Riives\textsuperscript{5} reported an arteriovenous malformation located in the right frontal region which appeared to have increased in size. Tönnis and Schiefer\textsuperscript{14} also had a case of an arteriovenous aneurysm lying immediately superior to the fissure of Sylvius which increased in size despite ligation of afferent blood vessels 16 years earlier. More recently, Höök and Johanson,\textsuperscript{6} in a follow-up study of 12 patients with cerebral arteriovenous malformations, found increase in size in eight patients whose malformations were mostly situated adjacent to the Sylvian fissure and in a ninth whose malformation was near the cisterns above the entrance to the sella. In our case, it may be possible that the porencephalic cavity was responsible for the striking growth of the malformation since the mass of pulsating vessels in the interhemispheric cistern, which protruded into the gyrus cinguli, was not buttressed by surrounding healthy brain parenchyma. This evidence contradicts the argument that cerebral arteriovenous malformations do not grow and hence do not need surgery.

Summary

The successful total excision of an arteriovenous malformation of the corpus callosum has been described. The remarkable growth of the lesion during the year of observation has been cited as an argument for surgery. It is suggested that paraventricularly-situated arteriovenous malformations, such as those of the septum pellucidum, corpus callosum,

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interhemispheric fissure, striatum, and the thalamus, justify particular attention because of their high incidence and the feasibility of total excision in certain cases.

References


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