Middle Cerebral Artery Embolectomy and Prolonged Widespread Vasospasm

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Abstract:
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Cerebral vasospasm following subarachnoid hemorrhage due to ruptured aneurysms has been discussed extensively during the past decade. Spasm involving the cerebral arteries also has been described in severe head injuries, infections, hypertensive encephalopathy and migraine. However, it has not been reported in cerebral embolism or following reconstructive cerebrovascular operations.

Widespread and prolonged cerebral vasospasm was recently observed angiographically in a 40-year-old patient two weeks after a middle cerebral artery embolectomy. Although the repaired artery was patent, the patient's neurological recovery was slow and incomplete.

With the increasing interest in cerebral microvascular procedures, more examples of vasospasm may be seen in the future. Its frequency and possible ill effects on neurological recovery and patency of the repaired artery, nevertheless, cannot be evaluated with certainty unless early postoperative angiograms are carried out.

Additional Key Words
- cerebral embolism
- subarachnoid hemorrhage
- long-standing widespread cerebral vasoconstriction
- aneurysm

Case Report
A 40-year-old right-handed woman was in good health until 12 noon, January 10, 1972, when she tried to bend down from a chair to pick up her sandwich bag. She fell, struck the right forehead against a radiator, and simultaneously developed a left-sided hemiplegia. On arrival at the hospital, she was alert and at times somewhat restless, and complained of frontal headaches. She denied any history of heart disease. Her blood pressure was 110/80 mm Hg, pulse 68 and regular. External evaluation showed no injuries, and physical examination was within normal limits except for the central nervous system, which revealed a complete left hemiplegia with a central facial palsy and Babinski sign. The arm and the leg were equally paralyzed, and only a slight degree of motion was present in the shoulder and the hip joints. Pin-prick sensation was diminished on the left, but other sensations seemed to be intact. ECG and lumbar puncture were normal. Right carotid arteriograms demonstrated occlusion of the middle cerebral artery at its distal trunk. The anterior cerebral artery was in midline, and the cervical carotid arteries appeared normal (Figs. 1 and 2). Since the patient's hemiplegia had developed simultaneously with the fall, it was felt that the trauma was not the cause of hemiplegia. This was confirmed at operation, when no sign of injury was found in the brain or in the middle cerebral artery. The diagnosis of middle cerebral artery embolism was made, but the origin of the embolus could not be determined.
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FIGURE 1
A right anteroposterior carotid arteriogram performed four hours after the onset of stroke. There is occlusion of the middle cerebral artery with retrograde filling of its terminal branches through the anterior cerebral artery. The anterior cerebral artery is in the midline.

OPERATION
About ten hours after the onset of hemiplegia, under general anesthesia and through a right frontotemporal flap, the right middle cerebral artery was exposed under the Zeiss operating microscope. The distal 10 mm of the trunk appeared dark blue, fat and fusiform. The blue color extended about 2 mm into two small side branches. Microclips were applied, and a 5-mm longitudinal incision was made in the artery. Upon releasing the proximal clip, the proximal portion of the embolus was squeezed out of the artery, and then the other clips were alternately released until the entire embolus was removed. There was an excellent proximal flow. The retrograde flow, however, was not very active. A small and soft irrigating catheter was inserted into the distal middle cerebral artery and then into the side branches, but this did not change the rate of the blood flow. The wall of the middle cerebral artery was free of atherosclerosis and appeared normal under the operating microscope. The arteriotomy was repaired with interrupted 9-0 monofilament nylon sutures. The clips were removed and circulation re-established. Although some spasm was present in the middle cerebral artery, it did not appear to be very severe.

SURGICAL PATHOLOGY
The specimen consisted of a firm blood clot $8 \times 4 \times 3$ mm in greatest dimensions. Microscopic sections revealed layers of red blood cells and fibrin with leukocytes which were of recent formation. No fibroblastic or capillary ingrowth was seen. Gram stain demonstrated no bacteria.

POSTOPERATIVE COURSE
The patient recovered from the anesthesia and neurologically remained the same during the first few postoperative days. About one week after the operation, there was some improvement in pin-prick sensation, and she was able to bend the left knee. A right carotid arteriogram was carried out on the fourteenth postoperative day, which demonstrated severe spasm of the supraclinoid carotid, middle cerebral, and anterior cerebral arteries. The trunk of the middle cerebral artery and some of its branches were opacified. The anterior cerebral artery was slightly shifted to the opposite side, indicating brain swelling (figs. 3 and 4).

Within two months after the operation, the patient was able to walk without assistance but had no voluntary movements in the left hand. At the present time, 11 months following the operation, she is able to hold or pick up objects with the left hand and is very anxious to start driving her car.

Stroke, Vol. 4, May-June 1973

FIGURE 2
A right lateral carotid arteriogram carried out four hours after the onset of stroke. It shows occlusion of the middle cerebral artery.
Blood in the subarachnoid space without mechanical trauma to the arterial wall also could cause cerebral vasospasm. Echlin has shown this in laboratory animals, and Taveras and Wood have seen it in an iatrogenic subarachnoid hemorrhage following ventricular puncture.

Short-lasting spasm following a nonhemorrhagic mechanical trauma has been observed by various investigators. Whether or not such trauma could cause a long-lasting cerebrovascular spasm is not clear. Leeds, Reid and Rosen, however, noticed that 10% of head injury patients who did not have subarachnoid hemorrhage demonstrated cerebral vasospasm.

Aside from the subarachnoid hemorrhage and trauma, meningitis, acute brain abscess, hydrocephalus, hypertensive encephalopathy, hypothermia, and migraine also may cause narrowing of the cerebral arteries. Buckle, Du Boulay and Smith have reported a 16-year-old girl who died of widespread cerebral vasospasm. She had no history of trauma, no subarachnoid hemorrhage, and no aneurysm. Migraine was discussed as a possible cause of spasm.

Cerebral vasospasm has not been described in patients with middle cerebral artery embolism. Dalal, Shah and Aiyar studied nine patients with middle cerebral artery embolism and performed follow-up angiograms within 4 to 21 days from the onset of the stroke. The authors made no mention of cerebral vasospasm in either the first or the second discussion.

Obviously, there are various causes for cerebral vasospasm. Blood in the subarachnoid space together with mechanical trauma to the wall of the artery is probably the commonest cause of all. Two-thirds of the patients with ruptured aneurysms in Du Boulay's study showed vasospasm. In Wilkins' study of ruptured aneurysms, spasm was demonstrated in one-third of the patients. Head injury patients with subarachnoid hemorrhage also may show cerebral vasospasm; however, its incidence appears to be lower. Leeds found spasm in 33% of the head injury patients and Wilkins in 6%. The higher incidence of spasm in ruptured aneurysms when compared with that in traumatic subarachnoid hemorrhage is probably due to the fact that in the former there is always a direct mechanical trauma (rupturing) which injures the wall of the aneurysm. Secondly, it may be related to the close contact of the blood with the parent artery or arteries. In the head injury, direct mechanical trauma to the major cerebral arteries is uncommon, and the arteries come in contact with the blood mostly through the subarachnoid space.
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angiograms. Zatz and Iannone described two cases of middle cerebral and one case of anterior cerebral artery embolism following cerebral angiography, also with no mention of vasospasm.

Of 14 middle cerebral artery embolectomies that we reviewed from the literature, ten had postoperative angiograms. Of these, five showed patent cerebral arteries, and one was patent but stenosed. Cerebral vasospasm was not discussed in preoperative or postoperative angiograms in any of these patients.

In experimental cerebral embolisms, Ferris et al. noticed spasm in two out of 14 monkeys studied. Mani and Newton and Rumbaugh, Davis and Gilson did not mention spasm in their studies of experimental cerebral embolism.

In our patient, no spasm was seen in the first angiogram performed four hours after the onset of stroke, but it was visualized two weeks after the embolectomy. Although none of the surgically treated patients with middle cerebral artery embolism collected from the literature showed vasospasm, on the basis of possible causes of spasm it seems reasonable to consider that the vasospasm noted in our patient was due to the surgical trauma to the middle cerebral artery and also to blood in the subarachnoid space following the craniotomy.

Suggestion has been made that partial ischemia of large segments of brain tissue might release a vasoconstricting substance which could diffuse along the fine perivascular spaces to the basal subarachnoid cisterns, giving rise to a self-perpetuating cycle of both local and distant spasm. The ground of this speculation, nonhemorrhagic stroke patients with relatively large areas of partial cerebral ischemia who do not have advanced cerebral atherosclerosis should show vasospasm if they are studied by angiography a few days after their stroke. However, this has not been demonstrated, and it was not described in the study of Dalal, Shah and Aiyar.

Of nine patients with middle cerebral artery embolism studied by Dalal, Shah and Sheth, eight showed a patent artery in the follow-up angiograms, but this spontaneous resolution of the embolis was not associated with significant neurological improvement. On the other hand, almost complete recovery has been reported within several hours following the middle cerebral artery embolectomy. Nevertheless, due to the small number of patients studied in both nonsurgical and surgical groups, no conclusion can be made as to the value of middle cerebral artery embolectomy.

How much vasospasm has caused damage or retarded the neurological improvement in our patient cannot be determined. In general, however, it appears that spasm following embolectomy or other reconstructive cerebrovascular procedures presents a more serious problem than the spasm after aneurysm surgery, because, in addition to causing cerebral ischemia, it may endanger the patency of the repaired artery, particularly when the vessel repaired is smaller than the middle cerebral artery. The incidence and real significance of this type of spasm will be better appreciated when both early and late postoperative angiograms are carried out in a large group of patients who have undergone cerebrovascular repairs.

References

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*Stroke*. 1973;4:446-450
doi: 10.1161/01.STR.4.3.446

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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