Traumatic occlusion of the middle cerebral artery

Case report

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A case is reported of a patient rendered unconscious in a motorcycle accident. After a 6-hour lucid interval, he became unconscious again, and a left hemiplegia was noted. Arteriography demonstrated complete occlusion of the right middle cerebral artery. Comparable reported cases and theories of pathogenesis are discussed.

KEY WORDS  middle cerebral artery  occlusion  trauma  embolism  thrombosis  dissecting aneurysm

TRAUMATIC middle cerebral artery occlusion has rarely been discussed in the neurosurgical literature; in fact, only 20 cases have been adequately detailed and documented.1-4,7,8,10-13 We believe the entity deserves reemphasis because of its clinical similarity to other traumatic intracranial lesions, and are therefore reporting our experience with one case.

Case Report

A 40-year-old man was admitted unconscious to the West Virginia University Hospital 6 hours after a motorcycle accident. No description of the accident was available, but it was known that he had been unconscious for about 15 minutes following the accident. On awakening, he had been taken to his home where after a few hours he became confused, hostile, and finally unconscious.

Examination. Examination on admission showed purposeful responses to painful stimuli on the right, but a decerebrate response on the left. The right pupil was dilated. Deep tendon reflexes were hyperactive and the plantar response extensor on the left. There was no external evidence of head or neck trauma, but a fracture of the distal right clavicle was palpable. Immediately after admission a right retrobrachial arteriogram demonstrated complete occlusion of the right middle cerebral artery at its origin (Fig. 1). There was no evidence of thrombosis elsewhere in the carotid system. A left carotid angiogram performed at the same time failed to demonstrate collateral circulation to the right hemisphere.

Course. Following these studies suppor-
Fro. 1. Right retrobrachial arteriogram performed on the day of admission demonstrates complete occlusion of the right middle cerebral artery.

tive care and dexamethasone administration were initiated. The patient improved and regained purposeful movements of his left arm and leg. However, about 24 hours after the injury his condition worsened. He showed evidence of brain stem involvement with bilateral decerebrate posturing and fixed, dilated pupils. A second right retrobrachial arteriogram showed partial clearing of the previously occluded middle cerebral artery; a frontal branch containing a small embolus was now visible (Fig. 2 left). There was also a new small filling defect, most likely a mural thrombus, in the internal carotid artery just distal to the carotid bifurcation (Fig. 2 right). The patient died soon after the second arteriogram was completed. Autopsy revealed emboli in all major branches of the right middle cerebral artery.

Discussion

In 1942, DeVeer and Browder reported a case of hemiparesis and coma resulting from craniocerebral trauma and following a lucid interval of 24 hours. Autopsy revealed a dissecting aneurysm involving the middle cerebral artery, resulting in its complete occlusion. Including ours, 20 cases of posttraumatic middle cerebral artery occlusion have been reported since then. These patients range in age from 6 to 67 years with an average age of 38 years; 15 were male. The injury proved fatal in 33% of the cases and resulted in significant permanent neurological deficit in most of the survivors. Fourteen patients presented in coma following lucid intervals ranging from minutes to 2 weeks. As in our case, several patients had signs of increased intracranial pressure with a unilateral dilated pupil or decerebrate posturing. Arteriography was performed during the initial investigation in 20 cases and repeated in 10 at intervals ranging from 24 hours to 9 months. Significant clearing of the obstruction had occurred in all but one of the latter group, but this could not be correlated with the severity of the residual neurological deficit.

Although the clinical characteristics of traumatic middle cerebral artery occlusion can be fairly well defined, the pathogenesis remains obscure. None of the patients was found to have any disease predisposing to vascular occlusion, and the age and sex distributions are those predictable in a group of trauma victims.

Postmortem findings were available in six cases. In two of these the diagnosis of dissecting aneurysm of the middle cerebral artery was made at autopsy, and in a third, the author's diagnosis of thrombosis of the middle cerebral was changed to dissecting aneurysm by Hollin, et al., after review of the photomicrographs.

Thrombosis of the middle cerebral artery was found in only one case at autopsy, and was attributed to transient occlusion of the cervical portion of the carotid artery. This mechanism of intracranial arterial thrombosis was originally proposed by Boldrey, et al., who stated that thrombus formation could occur at any point distal to a transient carotid occlusion.
Traumatic middle cerebral artery occlusion

Postmortem examination of our case revealed emboli in all of the major branches of the middle cerebral artery. Unfortunately the cervical portion of the carotid system was not examined, but angiography had demonstrated a mural thrombus in the proximal internal carotid artery which was thought to be the source of the emboli. This mechanism for middle cerebral artery embolism is well known in atherosclerotic vascular disease and has been previously proposed in cases of trauma on the basis of clinical and angiographic findings. In our case the carotid thrombosis probably occurred at the site of an intimal injury caused by cervical trauma. Although there was no physical or historical evidence of trauma to that region, others have suggested that even forced hyperextension and rotation of the neck can result in carotid thrombosis. Therefore, it is not surprising that evidence of severe cervical injury was lacking.

The remaining case in which postmortem examination was made was reported by Hollin, et al. Despite the angiographic demonstration of a complete middle cerebral occlusion 19 days prior to death, the artery was found to be completely patent at autopsy. Death was attributed to cerebral edema and increased intracranial pressure.
No cause of the previous arterial occlusion was found, but perhaps either dissolution of an embolus or segmental spasm\(^5\) may be implicated in this case.

In four of the surviving patients, angiographically demonstrable filling defects compatible with mural thrombi were found in the ipsilateral internal carotid artery.\(^4,^7,^10\) In these cases embolization from these thrombi seems the most likely cause of the middle cerebral artery occlusion.

In two other cases, subdural hematomas were found on the side of the middle cerebral occlusion.\(^11,^18\) Mechanical compression of the vessels by these mass lesions probably accounted for the obstruction of blood flow, although the presence of concomitant vascular lesions must also be considered.

In the remaining 10 cases the pathogenesis of the arterial occlusion is not evident; it seems likely that the previously discussed processes of embolization, thrombosis, and intimal dissection played a role.

References

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