Spasm of the extracranial internal carotid artery resulting from blunt trauma demonstrated by angiography

Case report

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Two cases of spasm of the extracranial portion of the internal carotid artery following trauma are described. In one case, the spasm did not cause lasting clinical symptoms, while in the other the spasm caused clinical symptoms with probable infarction.

Key Words • internal carotid artery • trauma • vasospasm

Spasm of the extracranial portion of the internal carotid artery may be seen and authenticated by angiography. Whether the spasm may cause clinical symptoms depends on the severity of the spasm and the adequacy of the collateral blood supply if the affected artery is almost completely occluded during the spasm.

We will report two cases of spasm of the extracranial portion of the internal carotid artery. In Case 1 the spasm did not cause permanent neurological deficit, and in Case 2 the spasm was associated with a neurological deficit which improved during a period of over 6 months.

Case Reports

Case 1

This case was briefly reported in 1963. A 39-year-old man was admitted on August 5, 1962, 2 weeks following an injury to the head and left side of the neck by a car door. He had been asymptomatic until the day of admission when he developed a transient attack of numbness of the right arm and hand with dysphasia. The patient did not lose consciousness, and the attack lasted about 30 minutes.

Examination. Neurological examination revealed an alert and cooperative patient. The speech and understanding were normal. There was no external evidence of injury, and the neck was supple. The cranial nerves were intact. The fundi were normal, and the visual fields were full on confrontation. The deep tendon reflexes were normal, the plantar responses were downgoing. Carotid compression on the right side caused dizziness. The electroencephalogram was normal. Bilateral carotid angiography revealed a complete occlusion of the left internal carotid artery. The arterial lumen was narrow distally (Fig. 1).

Operation. The left carotid artery was explored 19 days after injury. On exposure, the arterial wall was hemorrhagic, suggesting a bruise of the artery at the carotid bifurcation.
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FIG. 1. Case 1. Carotid angiogram showing complete occlusion of the left internal carotid 15 days after injury. Note the tapering of the arterial lumen distally (arrows).

The vessel was incised between clamps. It was filled with jelly-like clots, and after removal by suction and saline irrigation, the intima was found torn in several places with intimal tags helping to occlude the artery. Initially there was no bleeding from the distal end, but after irrigation and suction with an appropriate catheter, good retrograde flow was re-established. The intimal tags were excised, and the free edge of the intima was sutured to the wall of the vessel distally. The arteriotomy opening was closed with continuous No. 08 suture. An angiogram on the operating table revealed carotid patency but the vessel lumen was very narrow above the operative site (Fig. 2). This was due to a spasm of the internal carotid artery, since an angiogram 3 months later showed the vessel to be of normal size with complete disappearance of the vascular constriction (Fig. 3).

Comments. This patient manifested no symptoms of carotid involvement after the initial transient numbness of the right arm and dysphasia which brought him to the hospital. The complete occlusion of the left internal carotid artery was asymptomatic after the transient ischemic attack. The preopera-

FIG. 2. Case 1. Left carotid angiogram after re-establishment of flow. Note the marked narrowing of the arterial lumen above the operative site and all the way to the region of foramen lacerum (arrows). This constriction or spasm of the left internal carotid was asymptomatic.
Case 2

This 41-year-old man fell on July 28, 1969, while water skiing. He remembered the fall, and there was no initial blurring of the conscious state. After he was on shore helping to put away his gear, he developed aphasia and right hemiparesis with severe involvement of the right arm. Over a period of several minutes he became drowsy and eventually comatose. At the local hospital he was awake and lumbar puncture revealed clear fluid. He was transferred to Grace Hospital the same day. There was no history of previous attacks of focal neurological abnormalities or unconsciousness.

Examination. There was motor and sensory aphasia. Understanding was somewhat better than the ability to verbalize. Fundi revealed no hemorrhage or choking of the discs. Visual fields appeared full on confrontation. There was right hemiparesis with lower facial weakness and marked weakness of the right hand. The patient was able to move his right arm but there was an upgoing toe on plantar stimulation of the right side. Sensory examination was normal except for hypesthesia of the right hand and astereognosis. X-ray films of the skull and neck were negative. The electroencephalogram (EEG) showed a rhythmic slowing in the left temporal area. Bilateral carotid angiography 5 days later revealed a constriction of the left internal carotid artery about 10 cm above the bifurcation, near the foramen lacerum and involving about 4 cm of the arterial length (Figs. 4 left, 5 left, and 6 left). In a left carotid angiogram 11 days after the accident the stenosis had apparently improved. The dysphasia disappeared in 3 days. The weakness in the right half of the body improved slowly. Both the clumsiness of the right hand and the astereognosis persisted for many weeks. By 6 months the right hemiparesis had completely disappeared. An angiogram obtained 5 months after the initial study showed complete resolution of the constriction of the left internal carotid artery (Figs. 4 right, 5 right, and 6 right).

Comments. The narrowing of the left internal carotid artery was thought to be due to spasm resulting from the patient's fall into the water with hyperextension of the neck and stretching of the left internal carotid artery. One wonders whether the spasm may have been more severe initially with complete or almost complete obliteration of the blood flow in the vessel. The rapid appearance of the focal abnormalities suggests severe vascular insufficiency with infarction. An infarction in the distribution of the ascending Rolandic and anterior parietal branches of the left middle cerebral is concluded because of the long-lasting disability. Embolism seemed to us to be unlikely.
Fig. 4. Case 2. Left: Left carotid angiogram, anteroposterior view, obtained 5 days after water skiing accident. Note the narrowing of the internal carotid near the foramen lacerum and extending toward the heart (arrows). Right: Left carotid angiogram obtained 5 months later. The carotid spasm has disappeared.

Fig. 5. Case 2. Left: Left carotid angiogram, oblique view, obtained 5 days after the water skiing accident. Note constriction of the internal carotid high up at the base of the skull (arrows). Right: Left carotid angiogram, oblique view, 5 months later. The carotid spasm has completely disappeared.
Discussion

In the systemic circulation, vasodilation and vasoconstriction primarily involve the arteriolar-capillary-venular portion of the vascular tree, the larger vessels are not usually constricted or dilated, but operative exposure of these vessels may be associated with constriction and dilation. Many vascular surgeons have seen constriction and dilation of the aorta, iliac, and femoral vessels during operative exposural manipulations. Occasionally we have observed that the exposed carotid artery appeared constricted during dissection. The brachial artery frequently showed spasm when exposed for open retrograde brachial angiography.

Vascular spasms of the larger vessels of the general circulation causing clinical symptoms is not well authenticated. We think that our Case 2 is an example, with spasm causing focal abnormalities. This must be a rare occurrence, however, for in a rather large cerebrovascular experience we have recognized undisputed spasm of the extracranial portion of the internal carotid artery in only two cases.

Subintimal injection of contrast medium may cause a juxtapositional appearance of what may be mistakenly taken for vascular spasm. We have seen this pattern a few times. In one case, exploration revealed a normal-appearing vascular contour and angiography on the table was normal with no evidence of the previously seen narrowing of the lumen just above the subintimal injection of the dye. Atherosclerotic disease of the larger vessels should not pose any difficulty in diagnosis and recognition. The narrowing is localized, and usually there is a dilatation of the artery above the stenosis (poststenotic dilatation. Vascular spasm does not have the appearance of atheromatous luminal narrow-
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Atheromatous dilatation may at times simulate an aneurysmal lesion. In fact, atheromatous aneurysmal dilatation of the vessels may be seen by angiography or by dissection in autopsy cerebrovascular material.

The duration of the vascular constriction in our two cases is similar to the duration of spasm of the vessels constituting the circle of Willis seen after subarachnoid hemorrhage. The spasm may last for several days to several weeks. In our Case 1, the angiogram showing spasm was obtained 19 days after injury. In Case 2, angiography showing constriction was obtained 5 days after injury, but the spasm was not demonstrated in the angiograms obtained 6 days after the first angiogram.

References


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