Delayed Arterial Spasm and Thrombosis as a Cause of Post-Traumatic Hemiplegia (Spate Thrombosis)

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Abstract: Delayed Arterial Spasm and Thrombosis as a Cause of Post-Traumatic Hemiplegia (Spate Thrombosis)

The case of a Marine in whom a vertex epidural hematoma developed, depressing the superior sagittal sinus as a result of a fracture of the vertex of the skull, is presented. The hematoma was removed, and the patient did well until signs of progressive right hemiparesis suddenly developed two days after the hematoma removal. Serial carotid angiography demonstrated the interval development of spasm of the left internal carotid, anterior cerebral and middle cerebral arteries, and occlusion of the posterior parietal branch of the left middle cerebral artery. Prior to the development of the arterial spasm an earlier angiogram had shown these vessels to be of normal diameter.

This delayed or “spate” spasm and thrombosis of major intracranial vessels is a rare but serious complication of head injuries.

In addition to the usual causes of post-traumatic hemiplegias, this vascular spasm or thrombosis should be suspected in patients in whom progressive neurological signs develop after head injuries.

ADDITIONAL KEY WORDS

cerebral infarction  epidural hematoma  skull fracture
middle cerebral distribution

Introduction

When a patient, who has been satisfactorily recovering from a head injury, then has a dilated pupil and a hemiparesis, the neurosurgeon has been trained to think of several possible intracranial complications: If the patient has had operative evacuation of an epidural, subdural or intracerebral hematoma, the postoperative development or redevelopment of a hemiparesis alerts the clinician to the possibility of recurrent hematoma formation. If the patient has not had operative removal of an intracranial hematoma, and several days after injury a hemiparesis subsequently develops, then the neurosurgeon's thinking is oriented to the formation of a delayed subdural or epidural hematoma, or cerebral embolism, or of the remote possibility of “spate apoplexy,” i.e., hemorrhage into a softened area of encephalomalacia in the brain. There is, however, another cause of delayed hemiparesis following head injury, namely the development of delayed thrombosis of a major intracranial vessel following trauma.

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It is the purpose of this article to call attention to this unusual complication of brain injury, to review prior case reports and to present carotid angiograms that document sequential spasm and thrombosis of intracranial vessels, a sequence of angiograms not presented in prior cases.

The opinions or assertions contained in this paper are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

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POST-TRAUMATIC HEMIPLEGIA

FIGURE 1
Linear fracture of the vertex of the skull as a result of a blackjack blow. No evidence of depression of the fracture. This fracture extended from left parietal bone across the midline of the right parietal bone.

FIGURE 2
Left carotid angiogram, arterial phase: All major arteries fill out well; there is no evidence of spasm or thrombosis.

CASE REPORT
A retired 37-year-old staff sergeant, U. S. Marine Corps, was admitted to the neurosurgery service of the Naval Hospital, Camp Pendleton, California, on November 12, 1968, after having been attacked by unknown assailants and hit in the head by a blackjack four days previously. The patient stated that he had been struck from behind by a blackjack in an alley after leaving a bar. He had been rendered unconscious and was taken to a civilian hospital where he was observed overnight. A scalp laceration above and behind the left ear was debrided and sutured. He was discharged from the local hospital the next day,
but over the next two days an excruciating headache developed and he was brought to the Naval Hospital in Camp Pendleton. He had had a large alcohol intake for many years. Positive findings on physical examination revealed a well-developed, somnolent male complaining bitterly of severe headache. There was blurring of the optic disks bilaterally in their temporal margins. There were a left hemotympanum and a sutured laceration of the left temporoparietal scalp, with tenderness over the left mastoid area and ecchymosis and tenderness over the parietal areas bilaterally at the vertex of the skull. The patient was able to move all of his extremities; there were no sensory deficits. Extraocular motility was normal. Deep tendon reflexes were symmetrical; Hoffman's signs were bilaterally present, but Babinski signs were not present. There was a peripheral left facial weakness as a result of the left temporal contusion. Laboratory examinations were normal. Skull X-rays revealed a bilateral fracture of the parietal bone at the vertex (fig. 1). This fracture extended from the left to the right parietal bone crossing the superior sagittal sinus. There was no depression of the fracture. Lumbar puncture revealed grossly bloody spinal fluid with a pressure of 230. Carotid angiography revealed a slow intracranial vascular circulation rate. The anterior cerebral artery was shifted 3 mm from left to right. Other than the slight shift of the anterior cerebral artery, the arterial portion of the angiogram (figs. 2 and 3) was normal without evidence of spasm or thrombosis. The late venous phase of the initial carotid angiogram (fig. 4) revealed a 6 mm depression of the superior sagittal sinus underlying the parietal fracture, indicative of an epidural hematoma depressing the superior sagittal sinus.

On the day of admission the patient was taken to surgery, where bilateral parietal bone flaps were turned utilizing a pneumatic craniotome. A 100 cc epidural hematoma depressing the dura and superior sagittal sinus was evacuated. In addition, on the left side there was a 200 cc subdural hygroma which was removed. Following surgery the patient immediately became more alert and his headaches rapidly subsided. He did extremely well, being both alert and ambulatory for two days, but then a right hemiparesis and dilated left pupil developed. A repeat carotid angiogram at that time revealed again a very slow intracranial circulation rate. There was now an area of avascularity in the left posterior parietal area with nonfilling of the posterior parietal branch of the left middle cerebral artery (fig. 5). In addition, there was marked spasm of the left internal carotid artery just prior to its bifurcation. There was also spasm of the proximal anterior cerebral artery and also of the proximal left middle cerebral artery (fig. 6). The entire area of distribution of the posterior parietal branch of the left middle cerebral artery was not opacified. To rule out the possibility of a recurrent hygroma or hematoma in the area, a left osteoplastic craniotomy was performed. There was no evidence of epidural or subdural hematoma. A venous sinogram of the superior sagittal sinus (fig. 7) was performed at the operating table which revealed patency of the superior sagittal sinus under the parietal fracture site. In the absence of hematoma and with angiographical evidence of marked post-traumatic spasm of the left internal cerebral, left middle cerebral and to a lesser extent the left anterior cerebral arteries, the patient was treated with vasodilators (carbogen [7% CO2]). His blood pressure was kept to normal levels to insure adequate cerebral arterial perfusion, but clinical evidence of progressive cerebral infarction continued. He died on November 21, 1968, 13 days after the cerebral trauma and 11 days after signs of progressive post-traumatic cerebral arterio-

**FIGURE 3**

Left carotid angiogram, arterial phase, AP view showing shift of the anterior cerebral artery from left to right of 3 mm. All major arterial vessels fill well.
al spasm with infarction of the left parietal lobe had developed.

Autopsy findings revealed that the scalp wounds were healing well. The superior sagittal sinus was patent throughout its course. The left parietal lobe of the brain in the distribution of the posterior parietal branch of the middle cerebral artery revealed encephalomalacia as a result of infarction. Detailed examination of the left internal carotid artery revealed the vessel to be anatomically normal. There were no arteriosclerotic changes in the vessel at the level of antemortem thrombosis. A recent thrombus was present in the posterior parietal branch of the left middle cerebral artery.

**Discussion**

We are indebted to the British neurosurgeons, Sir Hugh Cairns and Mr. Walpole Lewin, for calling attention to delayed thrombosis of a major intracranial artery following blunt head injury. In Cairns' review of vascular aspects of head injury in 1942 he was able to find only one prior case, that of a 23-year-old male who fell from his bike; the next day right hemiplegia and aphasia developed. A left osteoplastic skull flap revealed, as in our case, no extradural or subdural hematoma, and the patient died three days after the accident. Autopsy revealed the left Sylvian artery (the middle cerebral artery) to be obstructed by a dark, firm, slightly adherent blood clot which extended retrograde into and almost completely blocked the intracranial portion of the left internal carotid artery. At that time Cairns reported a case (case 2) of a 27-year-old male who was thrown from his motorcycle; a right hemiplegia developed nine days after the accident. Arteriography revealed obstruction of the left internal carotid artery intracranially at its bifurcation. The motorcyclist died nine months after his injury following a prolonged course characterized by coma and right hemiparesis. At autopsy the most distal portion of the left internal carotid artery was occluded by gelatinous grayish-white tissue, the remaining cerebral arteries being normal.

In 1968 Lewin, in a review of vascular lesions following head injuries, presented a 47-year-old male who had been knocked off his bicycle by a car. The patient, when seen shortly
After the accident, was unconscious with a right hemiplegia. Within two days full consciousness returned, but it was recognized that the patient was aphasic. A left carotid angiogram revealed an occlusion of the left middle cerebral artery near its origin. Two months after the accident repeat angiography showed little change except for more obvious collateral circulation. This patient survived with improving right hemiparesis but with a continued severe expressive aphasia. Hockaday, reporting from the military hospital in Wheatly, England, presented a 20-year-old soldier who, after being knocked out in a dance hall brawl, had right facial palsy and weakness of the right hand. Six weeks after the accident, arteriography revealed proximal obstruction of the left internal carotid artery. A second case of Hockaday's was that of a 21-year-old professional boxer who lost a fight on points. Forty-five minutes after the decision, the pugilist had motor aphasia and right hemiparesis. Arteriography revealed obstruction of the left internal carotid at the base of the skull. Three years after injury he continued to manifest weakness of the right upper extremity and almost complete lack of movement of the right forearm and hand. Fortunately, he was left-handed and motor aphasia was only transient (figs. 8-11).

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References


FIGURE 6
Left carotid angiogram, AP view, postoperative, showing spasm of the left internal carotid artery, of the proximal portion of the left middle cerebral artery and of the left anterior cerebral artery.

FIGURE 7
Superior sagittal sinus venogram taken at the operative table following left osteoplastic craniotomy with negative exploration for recurrent hematoma. The superior sagittal sinus is patent throughout its course.
FIGURE 8
Enlarged view of left internal carotid arteries on initial angiogram showing normal appearance.

FIGURE 9
Enlarged AP view, initial left carotid angiogram showing normal diameter of vessels prior to spasm.
FIGURE 10
Enlarged view of arterial phase of left carotid angiogram, lateral view, showing spasm in left internal carotid artery.

FIGURE 11
Enlarged view of left carotid angiogram, AP view, showing development of spasm of left internal carotid artery proximal to the bifurcation and also of proximal left middle cerebral and anterior cerebral arteries. This is in a position distant from the area of prior surgery and indicates post-traumatic spasm of the major vessels. This spasm progressed to occlusion of the posterior parietal branch of the middle cerebral artery with subsequent infarction and encephalomalacia of the left parietal lobe.
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