Vertebrobasilar spasm: A significant cause of neurological deficit in head injury

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Cerebral vasospasm in the anterior circulation has been recognized as a significant factor in the sequelae of head injury; however, vertebrobasilar spasm resulting from trauma has received much less attention. In the past year we have observed six patients where spasm in the major vessels of the posterior circulation was primarily or in part responsible for the neurological deficit. In such cases, the neurological examination may suggest a supratentorial mass with herniation and, in three of our cases, burr holes or carotid angiography were performed first. However, in every instance neurological signs present on admission indicated primary brain-stem dysfunction. In each of the six cases vertebral angiography demonstrated significant spasm in either the vertebral or basilar arteries. Intracranial pressure was monitored in each of the six patients and did not exceed 25 mm Hg in any. In cases of head injury where the neurological examination indicates brain-stem dysfunction inconsistent with or after a supratentorial mass has been excluded, vertebral angiography may aid in the diagnosis and subsequent management of such patients.

KEY WORDS • spasm • posterior circulation • angiography • head injury • vertebrobasilar artery

Cerebral vasospasm in the anterior circulation has been generally recognized as a significant factor in the sequelae of head injury since publication of the observations of Wilkins and Odom, although vasospasm in head trauma had been described previously by others. In a more recent publication, Suwanwela and Suwanwela described the angiographic findings in 350 patients with moderate-to-severe head injury, and reported that significant spasm was seen in 18.6%. None of these previous reports, however, has recognized the occurrence of spasm in the posterior circulation in head injury. In the period from 1973 through 1974, we observed six patients where spasm in the vertebrobasilar circulation was primarily or partially responsible for the neurological deficit. In such cases the neurological examination tended to suggest a supratentorial lesion with tentorial herniation, and in three of our cases burr holes or carotid angiography were performed before vertebral angiography. In retrospect in each of these patients, neurological signs were present that strongly suggested primary brain-stem dysfunction. Because the clinical signs in each of our cases were strikingly similar, suggesting a common etiology, and because they may mimic a supratentorial
Vertebrobasilar spasm in head injury

FIG. 1. Case 1. Vertebral angiograms. Left: On admission there was spasm of the basilar artery (arrow) with post-stenotic dilatation. Right: Thirteen days after admission the basilar artery was normal in configuration.

mass or intracerebellar hemorrhage, we are describing the findings in our patients to alert neurosurgeons to this entity.

Case Reports

Case 1

This 47-year-old man was admitted in a deeply stuporous state. There was a 6-cm transverse laceration of the scalp in the occipital region. The cranial nerves were intact, but there was a flaccid quadriplegia. Within 40 minutes of admission the left pupil became dilated, mannitol was administered, and the patient intubated. He was brought to the operating room for emergency trephination, and while he was being transferred to the operating table it was noted that he had developed disconjugate gaze. The eyes were open and the patient appeared to follow loud sounds despite being unable to move any extremities in response to deep pain. Bilateral frontoparietal burr holes were placed and no mass or brain swelling was found. A cannula was inserted in the lateral ventricle and clear cerebrospinal fluid (CSF) under normal pressure was obtained. Vertebral angiography was then performed and demonstrated severe spasm of the distal basilar artery and no evidence of a mass lesion (Fig. 1 left). He remained able to move his eyes to auditory stimuli. Over the next 36 hours he became alert and oriented, but marked weakness of the proximal musculature in all extremities and ataxia of the limbs was noted. By the seventh day following admission, the patient was able to walk with only minimal difficulty. The intracranial pressure (ICP) was normal during the entire course. Vertebral angiography was repeated 13 days following admission and was normal (Fig. 1 right). At the time of discharge the only residue was subjective numbness of the hands.

Case 2

This 22-year-old man was thrown from a moving car. At the time of admission he was alert and had no neurological deficit, but he did complain of a stiff neck. Within 24 hours he could not be aroused. The pupils were equal and reactive to light, but the doll's eye and caloric responses could just be elicited. There was bilateral spasticity in the lower extremities and the Babinski responses were extensor. Pan-cerebral angiography demonstrated a normal anterior circulation but spasm with severe stenosis of the basilar artery was seen on vertebral injection (Fig. 2). Fourteen hours after angiography the patient suffered a respiratory arrest. The ICP was normal initially but rose at the time of respiratory arrest and remained elevated until his death.

Case 3

This 23-year-old man was shot in the back of the head during a holdup. At the time of
admission he was alert and neurologically intact. However, within 20 minutes of admission he became unresponsive and apneic. The pupils were noted to be small and the gaze was disconjugate. Caloric responses were absent. Emergency suboccipital decompression revealed small epidural and subdural hematomas and intracerebellar hemorrhage.

Following evacuation of the hematomas a ventriculostomy was placed for drainage and monitoring of the ICP. During the first postoperative week the patient gradually improved and began to obey commands. He would track objects but was quadriplegic. The ICP remained normal. On the seventh postsurgical day he became quadriplegic and would no longer follow commands. Caloric responses, which had recovered, were now absent. Computerized tomographic (CT) scanning demonstrated a small area of infarction in the left cerebellum, but no evidence of shift or mass. Vertebral angiography performed via transfemoral catheterization demonstrated occlusion of the left vertebral artery (Fig. 3 left) and severe spasm of the right vertebral and basilar arteries (Fig. 3). Carotid angiography was normal. He died 7 days after angiography.

Case 4

This 14-year-old boy was struck by an automobile and rendered immediately unconscious. He was admitted in coma and at that time brain-stem function was intact. Skull x-ray films demonstrated diastasis of the clivus and vertebral angiography (Fig. 4) showed moderate spasm of the mid-basilar artery and a small clival hematoma. Two hours following angiography caloric testing

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Vertebrobasilar spasm in head injury was repeated and response was absent, despite preservation of the pupillary response to light. The ICP was monitored and never exceeded 25 mm Hg. Despite hyperventilation and pharmacological support of the blood pressure the patient died.

Case 5

This 17-year-old boy was beaten over the head with a pipe. On admission he was awake, but markedly dysarthric. There were palsies of the right facial and left hypoglossal nerves and ataxia of the left arm. Over 48 hours the patient became progressively stuporous. Although the pupils remained equal and reactive to light, the caloric responses were absent. The ICP was monitored and did not exceed 10 mm Hg at any time. Pan-cerebral angiography demonstrated a small right parietal contusion and marked spasm of the proximal vertebral artery. Over the next 12 hours a left hemiparesis developed which evolved into a flaccid quadriplegia. Fulminant pulmonary edema then supervened, and the patient died.

Case 6

This 29-year-old man was beaten over the head with a club. On admission he was alert but disoriented. The pupils were equal and reactive to light. There was a left facial and a right hypoglossal nerve palsy. Over the next 4 hours severe dysmetria of the arms developed and the patient became stuporous. The pupillary response to light remained brisk, but the caloric responses were absent. The ICP was monitored and was normal. Vertebral angiography demonstrated severe spasm of the proximal basilar artery and slowing of the circulation. Immediately following angiography the patient suffered an irreversible cardiac arrest. Permission for autopsy was denied.

Discussion

In establishing the diagnosis of traumatic vertebrobasilar spasm, supratentorial mass must be ruled out, by 1) clinical signs, 2) pan-cerebral angiography, and 3) intracranial pressure monitoring. A brisk pupillary response when caloric responses are absent or greatly diminished is essential to diagnosis. It is important to remember that clear ventricular CSF does not rule out spasm. Lack of careful examination for brain-stem and cerebellar dysfunction, or failure to recognize the significance of the finding can mean that the correct diagnosis is missed.

In each of these six cases certain features of the clinical presentation were suggestive of primary brain-stem or cerebellar dysfunction (Table 1). The pupillary response to light

![Fig. 4. Case 4. Plain skull film showing moderate spasm of the middle portion of the basilar artery.](image)

TABLE 1

<table>
<thead>
<tr>
<th>Signs</th>
<th>No. of Cases</th>
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<tr>
<td>level of consciousness</td>
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<tr>
<td>alert</td>
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</tr>
<tr>
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</tr>
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<td>2</td>
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<tr>
<td>focal deficit</td>
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<tr>
<td>cerebellar signs</td>
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<tr>
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</tr>
<tr>
<td>hemiparesis</td>
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<tr>
<td>pupillary response to light</td>
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</tr>
<tr>
<td>absent or diminished</td>
<td>5</td>
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remained brisk in five of six patients at a time when the caloric responses were absent or greatly diminished, indicating a ponto-medullary disturbance with an intact midbrain. In three cases, limb ataxia of the cerebellar type was found at some point in the patient's course. In the first patient, pupillary dilatation with absent motor response to pain suggested tentorial herniation, but the response to sound in the absence of any response to pain was strong evidence for brain-stem dysfunction producing a "locked-in state."

Monitoring of ICP aided in establishing the diagnosis and led us to perform early vertebral angiography. The ICP was normal in four patients and mildly elevated in two, indicating that the clinical symptomatology could not be explained on the basis of secondary brain-stem compression, but rather that the disturbance was of a primary type.

Case 4 was the only patient with blood in the CSF at the time of posterior circulation vasospasm. In one patient, diastasis of the clivus was seen on x-ray film, indicating severe mechanical trauma at the base of the brain. In Case 3 there was a fracture of the occiput secondary to the gunshot wound. In the other four patients, skull radiographs were normal and we have no definite explanation for the occurrence of spasm in these patients.

The availability of CT scanning has significantly reduced the number of emergency angiograms in patients with head injury. It is likely, therefore, that in many patients with vertebrobasilar spasm, the diagnosis will not be definitely established. However, in patients who do not have a posterior fossa mass, and in whom the pupillary light response is present but the caloric response is totally diminished or absent, brain-stem ischemia secondary to vertebrobasilar spasm should be considered.

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References

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