Incarceration of a Vertebral Artery in the Cleft of a Longitudinal Fracture of the Skull

Case Report*

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In linear fractures of the base of the skull, the dura is often torn. Occasionally, the arachnoid may be caught in the fracture but rarely any other intracranial structures. Recently, a case of traumatic occlusion of the basilar artery within a clivus fracture was published. The present report describes another case of incarceration of an artery in a fracture, this time one of the vertebral arteries.

Case Report

A 42-year-old man fell off a truck and landed on the vertex of the head. This caused a wound of the scalp measuring 4 inches in length, extending from the vertex to the occiput. It was not known whether he had temporary loss of consciousness, but he was able to walk and to state his name, date of birth, etc., when he was admitted to the hospital, 45 minutes after the accident. There was no outward evidence of a skull fracture at the time of surgical repair of the scalp wound.

Examination. After this procedure, the patient became drowsy and vomited; his pulse was 46. A few hours after hospitalization, he became unconscious and incontinent. The head was freely movable. There was conjugate deviation of the eyes to the left. The pupils and optic fundi were normal. The corneal reflexes were absent. It was observed that the right arm and leg could not be moved spontaneously. The hemiplegia did not involve the facial muscles. There were pseudospontaneous movements with fumbling and grasping of the left hand. Abdominal and cremasteric reflexes could not be elicited. Reflexes of the extremities were normal except for bilaterally positive Babinski and Oppenheim signs.

The pulse increased to 112. The temperature was normal. No fractures were seen on x-ray. The condition of the patient remained unchanged for the first 4 days. No new symptoms of intracranial pressure developed during this time.

The patient regained consciousness on the 5th day after the accident. He was able to comprehend questions but his voice was low and his speech hard to understand. On the 6th day, his general condition improved further; however, he had bulbar speech, dysphagia and flaccid paralysis of the right arm and leg. There was no facial weakness. Sensory testing was unreliable. Lumbar puncture yielded slightly bloody fluid.

During the daytime, the patient was usually responsive to questions but towards evening he became con-
13th day when aspiration pneumonia developed. He died on the 14th day after the accident.

Postmortem Examination. The galea over the vertex showed some hemorrhage in the region of the healed scalp wound. The calvarium was not fractured. There was a thin layer of recent subdural hemorrhage over the cerebral convexity extending from frontal to occipital lobes. Some subarachnoid hemorrhage was present near the interhemispheric fissure.

Upon removal of the brain, it was apparent that the left vertebral artery was caught in the fissure of a skull fracture which extended from the body of the sphenoid bone through the clivus and continued at the dorsal side of the foramen magnum through occipital bone to the torcular (Fig. 1). The left vertebral artery, which was twice as wide as the right one, was incarcerated near its junction with the basilar artery for a length of 7–8 mm. Rostrally from the point of incarceration, a short stretch of the vertebral artery and a small artery, branching off in this area to supply the left pyramid, were thrombosed (Fig. 2). The left pyramid was softer than the right. Cross sections through this region showed a recent softening involving the left pyramid and extending like a wedge parallel to the midline into the region of the left hypoglossal nucleus.

Coronal sections through the cerebrum showed only small cortical contusions in both gyri recti and in the right temporal pole. Midbrain, pons, cerebellum and ventricles were normal.

Histologic examination was confined to the medulla oblongata which was examined in serial sections.

The focus showed the typical picture of a softening in the state of resorption and organization. Compound granular cells were abundant, all traces of nerve cells had disappeared, and myelin sheaths and axon cylinders had disintegrated.

The area of softening was sharply demarcated from the surrounding tissues. Its upper border was near the transition from medulla oblongata to pons. The nuclei and fiber tracts of the 6th and 7th nerves were not affected. Caudally, the area of softening enlarged, reaching its greatest extent in sections 2 mm. from its upper pole. At this level the entire pyramid was involved. The softening extended along the midline without crossing it and reached the medial bundle of the central tegmental tract (Fig. 3a). About 1 mm. below this level, a small ventro-medial part of the inferior olive was included in the softening (Fig. 3b). In lower sections the lesion became smaller and finally involved only the upper part of the hypoglossal nucleus where it reached the wall of the 4th ventricle (Fig. 3c). The caudal part of this nucleus remained intact.

Fig. 3. Extent of softening demonstrated in sections of upper medulla oblongata, taken at 3 consecutive levels. X 34. (a) 2 mm. below the upper border of the area of softening. The wedge-shaped focus involves the entire left pyramid and reaches the medial bundle of the central tegmental tract. Van Gieson stain. (b) 1 mm. below level of (a). A small ventro-medial part of the left inferior olive is included in the softening. Myelin stain. (c) Near the lower limit of focus. Softening is confined to left hypoglossal nucleus and vicinity. Myelin stain.