Cerebellar infarction resulting from traumatic occlusion of a vertebral artery

Case report

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A case is reported of a patient who suffered a gunshot wound of the neck which resulted in occlusion of a vertebral artery. Within a few hours he deteriorated neurologically to a comatose state with a gaze paresis and a facial paresis ipsilateral to the occluded vertebral artery. The diagnosis of cerebellar infarction with brain-stem compression was made clinically, and a posterior fossa decompression was carried out promptly. The patient has made an excellent recovery. Cerebellar infarction, like cerebellar hemorrhage, may act as a posterior fossa mass requiring neurosurgical decompression. This report emphasizes that such a pathological process may occur after certain injuries that are likely to result in occlusion of a vertebral artery.

KEY WORDS cerebellar infarct □9 cerebellum □9 vertebral artery □9 vertebral occlusion

TRAUMATIC occlusion of a vertebral artery is not a rare event. Chiropractic manipulation of the neck has resulted in several instances of occlusion or symptomatic "spasm" of the vertebral artery. Calisthenics, football injuries, anterior cervical discectomy, stab-wound of the neck, and atlanto-axial and lower cervical dislocations. Occasionally spontaneous forced rotation and hyperextension of the neck have resulted in vertebral occlusion. Many of these patients have suffered severe strokes in the vertebrobasilar system, and several have died as a result of this problem. In some of these cases the primary pathological process found at postmortem examination was cerebellar infarction. This is a report of a patient with traumatic occlusion of a vertebral artery resulting from a gunshot wound of the neck. A clinical diagnosis of cerebellar infarction led to surgical decompression of the posterior fossa with consequent prompt recovery.

Case Report

This 22-year-old man suffered a gunshot wound of the left supraclavicular fossa. Upon arrival at the emergency room he was agitated and in a state of hypovolemic shock. No focal neurological abnormalities were noted. He promptly underwent exploration by the vascular surgical team, and a tear of the subclavian artery was repaired. The left vertebral artery had been torn and required ligation. There was a significant period of hypotension during the operation. Immediately after surgery an arch aortogram showed a normal right vertebral artery, normal common carotid arteries, and a patent left subclavian artery. The left vertebral artery was occluded at its origin. He awoke slowly from anesthesia and gradually became agitated; within about 6 hours, he became progressively more lethargic. A computerized tomography (CT) scan at this point showed mild to moderate ventricular dilatation and no other specific abnormality; there was some motion artifact. Initially it was thought that probably the hypotensive period had resulted in a diffuse encephalopathy.

Examination. Neurosurgical examination 14 hours after his initial injury showed the patient to be comatose with alternating decerebrate and decorticate posturing. There was a left abducens paralysis and a gaze palsy to the left, which did not correct with caloric stimulation. In addition, the left side of the face seemed weaker than the right.

Operation. Based on these findings, the diagnosis of left cerebellar infarction was made and the patient was taken immediately to the operating room for a ventriculostomy followed by a suboccipital craniectomy. At surgery, a large infarct of the posterior inferior portion of the left cerebellar hemisphere was found and resected. The necrotic cerebellar tissue exuded upon the dura being opened. The posterior arches of
C-1 and C-2 were removed to decompress the herniated and swollen left cerebellar tonsil.

Postoperative Course. The patient improved immediately after surgery and the ventricular drain was removed 3 days later. He was discharged home in 2 weeks, and returned to work in 2 months. His only neurological residua are a minimal subjective diplopia on looking to the left, and minimal left-sided appendicular ataxia.

Discussion

It is known that spontaneous cerebellar infarction, like cerebellar hemorrhage, may act as a posterior fossa mass and require neurosurgical decompression. Surgical decompression for cerebellar infarct was first reported in 1956 by Fairburn and Oliver and Lindgren. Subsequently, Wood and Murphey reported five cases with good results in three. The pathological and clinical features of this syndrome have been well defined. About 40% of the patients have a history of hypertension. Occlusion of the intracranial portion of a vertebral artery is responsible for about 50% of the cases, and occlusion of the posterior inferior cerebellar artery (PICA) accounts for another 30%. Embolism is implicated in approximately one-third to one-half of the cases, and the infarct becomes hemorrhagic in about 25% of the patients. The area of infarction is located in the posterior inferior portion of the cerebellar hemisphere (PICA territory) in almost 85% of the cases that become symptomatic. Associated signs and symptoms of lateral medullary infarction are present in nearly one-third of the patients. The onset is usually sudden, with headache, vomiting, vertigo, gait ataxia, and dysarthria. Early findings include confusion and disorientation, ipsilateral gaze palsy, truncal and appendicular ataxia, and ipsilateral abducens and facial palsies. With progressive brain-stem compression, stupor and coma supervene with spastic quadriparesis and later decerebration with pinpoint, sluggishly reacting pupils. Once signs of brain-stem compression develop, the mortality rate without surgery approaches 80%. Death usually occurs between 6 and 30 hours after the appearance of obtundation and stupor in untreated cases.

In the series reported by Momose and Lehrich, five patients in deep coma from cerebellar infarction underwent suboccipital craniectomy; three of these made a good recovery. Our own personal experience with five patients who underwent suboccipital decompression for cerebellar infarction includes four patients who were in coma before surgery. Of these, one is nearly normal (the present case), and the other three are functioning independently at home with mild to moderate neurological disability. In most of these cases the comatose state results not from intrinsic parenchymal damage but rather from extrinsic compression of the brain stem by the swollen cerebellar hemisphere.

Richardson has emphasized the danger of ventricular drainage performed without suboccipital decompression in patients with cerebellar hemorrhage. Several of his patients seemed to deteriorate acutely after this maneuver, probably as a result of upward herniation of the brain stem. For this reason we join other authors in recommending posterior fossa decompression by craniectomy in all cases of cerebellar infarct (or hemorrhage) that develop signs and symptoms of brain-stem compression. A ventriculostomy may be carried out to gain precious time when the patient is rapidly failing, but it should be followed rapidly by a suboccipital decompression. Since in most cases the infarct involves the posterior inferior cerebellar hemisphere, a low midline craniectomy through the foramen magnum, extended more toward the side of suspected involvement, is recommended. It is frequently necessary to resect the posterior arch of C-1 and sometimes C-2 to adequately decompress a grossly herniated tonsil. The pulped, necrotic cerebellar tissue frequently herniates through the initial dural opening and is readily removed by suction. The area of infarct is well demarcated from surrounding normal cerebellum.

In most cases of cerebellar infarction due to vertebral occlusion the occlusive process has involved the intracranial portion of the vertebral artery. Fisher has emphasized that extracranial vertebral occlusion is well tolerated except in cases of occlusion or hypoplasia of the contralateral vertebral artery, or in cases in which the vertebral artery ends as a PICA without joining the basilar artery. In the present case a normal contralateral vertebral artery was demonstrated. To explain the occurrence of infarction in this case, it could be postulated that thrombosis extended distally to the intracranial portion of the vertebral artery or that the occluded artery ended as a PICA. It is more likely, however, that the significant period of hypotension suffered by this patient made vertebral occlusion less tolerable than it may have been under normal circumstances.

The diagnosis of cerebellar infarction (or cerebellar hemorrhage) should be made clinically. The only neuroradiological investigation of practical value in these cases may be CT scanning. However, it should be emphasized that the CT scan may not be diagnostic in cases of cerebellar infarction, as demonstrated by the present case. Depending on the stage of development of the infarct and on the presence of some degree of hemorrhagic changes, the involved tissue may be of a density similar to surrounding cerebellum. Hydrocephalus and displacement or obliteration of the fourth ventricle may be the only clues to the presence of a cerebellar infarct. In the present patient the fourth ventricle was not well visualized, which may be of significance but is not of conclusive diagnostic value. Neither angiography nor ventriculography is recommended in a deteriorating patient with signs of brain-stem compression if the clinical picture is suggestive of...
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cerebellar infarction or hemorrhage. Instead, prompt surgical intervention should be carried out.

Conclusions

Spontaneous cerebellar infarction, like cerebellar hemorrhage, can act as a posterior fossa mass and may require neurosurgical decompression. The literature has not emphasized that the same situation may develop after certain traumatic events that may lead to occlusion of a vertebral artery. Recognition of this syndrome and prompt neurosurgical attention may prevent a fatal outcome and result in gratifying recovery, even in comatose patients, as emphasized by the present case.

References

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