Cortical arteriovenous fistula associated with skull fracture

Case report

PAUL A. LAHAYE, M.D., AND PABLO M. LAWNER, M.D.

Division of Neurosurgery, Harbor/UCLA Medical Center, Torrance, and University of California, Los Angeles, California

The case is reported of a patient who presented with a skull fracture and delayed neurological deterioration due to a cortical arteriovenous fistula at the fracture site. The clinical course and surgical therapy are described. Theories as to the pathogenesis of this lesion as well as a discussion of other intracranial vascular injuries are presented.

KEY WORDS • arteriovenous fistula • cerebral artery • head injury • skull fracture

Intracranial vascular lesions as a consequence of skull fracture are not unusual and can take many forms. Traumatic aneurysms of cortical cerebral arteries in association with skull fractures are well documented. A variety of arteriovenous (AV) fistulas have been described in this setting, most commonly involving the meningeal vessels and occasionally the diploic veins, dural sinuses, or cortical veins. A case of a traumatic dural AV fistula with a cortical arterial feeder was reported from this institution in 1980.

This present report describes an unusual case in which an AV fistula involved only cortical arteries and veins, without contribution from the external cerebral circulation or dural sinuses. Of further interest is the reversal of the patient’s sudden neurological deficit by surgical obliteration of the fistula.

Case Report

This 60-year-old right-handed man was brought to the emergency room by paramedics. He had been found unresponsive some 2 to 3 hours following an unobserved fall down several concrete steps. The patient had no history of seizures or of transient cerebral ischemia or stroke. He had suffered a myocardial infarct 6 months earlier, and his medications consisted solely of oral nitrates. A penetrating injury to the left eye had necessitated enucleation 30 years before, but he had no other history of head trauma.

Examination. The patient was normotensive and, except for moderate obesity, had an unremarkable general physical examination. On admission, his Glasgow Coma Scale score was 8. The patient was arousable only in response to painful stimuli and could only verbalize incoherently. The scalp was intact except for confluent contusions about the right frontal and periorbital regions. Cranial nerve survey disclosed no detectable deficits. Motor examination revealed increased tone on the left side and a marked left hemiparesis, with the leg weaker than the arm. The patient was symmetrically areflexic, with bilateral plantar flexor responses. No sensory deficits were detectable.

Baseline laboratory data, including clotting parameters, were normal. Plain skull films (Fig. 1) revealed an irregular linear non-depressed skull fracture beginning in the left frontal region, crossing the midline in the region of the coronal suture, and extending parasagittally to the right parietal region 2 to 3 cm lateral to the sagittal suture. No other fractures were noted. Computerized tomography (CT) was interpreted as normal and the patient was admitted to the neurosurgical service.

The patient’s mental state improved over the ensuing 24 to 36 hours. He became alert, but remained episodically confused. His left hemiparesis persisted, with development of left-sided hyperreflexia and an extensor plantar response. He was also noted to have a fairly pronounced nondominant parietal lobe syndrome with
Cortical AV fistula after skull fracture

left-sided neglect and extinction of simultaneous stimuli. The patient's clinical course and neurological findings implicated predominantly right frontoparietal dysfunction, presumably due to cortical contusion. A second CT scan 6 days after admission revealed a low-density area in the right frontoparietal parasagittal region, seeming to corroborate our clinical impression.

This slow but steady clinical improvement was sustained until the 10th hospital day when he was found to be arousable only to painful stimuli. His previously improving left hemiparesis had suddenly markedly worsened, and for the first time he demonstrated contralateral findings in the form of marked paresis of the right lower extremity. A CT scan at this time revealed no change since the previous study. Concern over the possibility of superior sagittal sinus thrombosis or another intravascular process prompted emergency cerebral angiography, which revealed an AV fistula at the fracture site approximately 6 cm posterior to the coronal suture (Fig. 2). The sagittal sinus was patent. The arterial component of the fistula was a cortical branch of the right callosomarginal artery, with venous outflow via superficial cortical veins overlying the right parietal convexity. Neither the sagittal sinus nor any external carotid branches were involved.

Operation. Through an "inverted U" incision over the right temporoparietal area, a non-depressed diastatic fracture line was visualized. The site of the AV fistula within the fracture was isolated with a rongeur, leaving a 2 x 2-cm island of bone overlying the fistula. The dura along the fracture line was noted to be torn and the underlying cortex was hyperemic and congested. Two arterialized veins were visualized on the adjacent cortical surface. The island of bone was gently removed and the arterial feeder visualized and coagulated. This resulted in prompt darkening of the blood in the arterialized veins. A pseudo-endothelialized hematoma was excised after coagulation of the vein draining it. An acrylic cranioplasty completed the procedure.

Postoperative Course. The patient's neurological recovery was quite remarkable. Within 48 hours of surgery his mental status had normalized and the right-sided paresis completely resolved. The left hemiparesis

FIG. 1. Anteroposterior radiograph of the skull showing the linear non-depressed fracture.

FIG. 2. Left: Anteroposterior angiogram showing the fistula site (arrowhead). Right: Lateral angiogram showing the callosomarginal artery feeder (small arrowhead) and cortical venous drainage (large arrowhead).
exhibited a slower pace of steady improvement, the leg remaining weaker than the arm. Follow-up angiography revealed no evidence of the AV fistula. The patient was discharged for rehabilitation within 10 days of operation.

Discussion

Traumatic aneurysms of intracerebral vessels are well documented in the literature. Peripheral cerebral aneurysms as sequelae of head injury are associated with closed trauma in 62% of cases, penetrating injury in 27%, and iatrogenic injury during surgery in 11%. Fifty-eight percent lie in the middle cerebral artery distribution and 37% in the anterior cerebral artery circulation. Posttraumatic aneurysm formation in cortical arteries is associated with a skull fracture in some 50% to 98% of reported series. In the absence of fracture or dural laceration, rotatory forces acting on cortical vascular structures at the time of impact are thought to produce arterial wall injury.

Damage to the cerebral vasculature through contact with adjacent intracranial structures can also occur in the absence of skull fracture. Examples include anterior cerebral artery injury due to impact against the adjacent falk and middle cerebral artery injury by the sphenoid wing. A more obvious mechanism for vascular injury in head trauma could be an outbursting type of skull fracture with herniation of brain tissue and entrapment or laceration of vessels at the fracture site. This mechanism has been described in association with vertebral artery injury and basilar artery injury, and is the mechanism most likely responsible for our patient's lesion.

Traumatic peripheral cerebral aneurysms are commonly associated with cortical lacerations, intracerebral hematoma, or subdural hematoma. In the absence of acute sequelae, a syndrome of delayed hemorrhage can be seen in some 40% to 50% of cases, typically within 15 days of injury. These lesions represent a formidable complication of head trauma, with mortality figures ranging from 54% in the early series to approximately 20% in more recent series.

While posttraumatic cerebral aneurysms are not unusual, AV fistula formation as a consequence of head injury is much less common. The formation of these lesions in association with the external carotid circulation in the posttraumatic period is well described. Middle meningeal AV fistulas can manifest as epidural hemorrhage or may undergo spontaneous thrombosis. Other variations of this type of injury include venous efflux via diploic veins, the sphenoparietal sinus, the greater petrosal sinus, or the cortical veins. A posttraumatic dural AV fistula with a cortical arterial feeder and sagittal sinus involvement represented a formidable surgical challenge to our colleagues at this institution in 1980.

The lesion in our present report is unusual in that only cortical vascular structures were involved. Rum-bough, et al., alluded to a posttraumatic pseudoaneurysm with a connecting cortical vein, without elaborating on the clinical correlates. Parkinson and West described 11 cases of traumatic intracranial aneurysms, five being arteriovenous in nature. Two of these involved the intracerebral vessels alone: one was an AV fistula of the posterior inferior cerebellar artery beneath an occipital skull fracture. The other case described was of an AV fistula between the ascending frontal branch of the middle cerebral artery and a cortical vein in a patient without a skull fracture but with recurrent subdural hemorrhage.

In postulating the mechanism of formation of our patient's lesion, one would first implicate cortical arterial damage due to distraction of the fracture margins and cortical herniation through this site at the moment of impact. Arterial damage in this setting could lead to any number of sequelae. A "true" aneurysm would result from partial disruption of the arterial wall, leaving intact the outer layer of the parent vessel to serve as a limiting structure to aneurysmal dilation at the site of injury. Full-thickness laceration of a cortical vessel can lead to frank subdural hemorrhage or the formation of a "false" or "pseudo" aneurysm, in which the arterial laceration is contained by a surrounding hematoma. Organization of the hematoma with attendant hemodynamic excavation results in aneurysm formation and expansion. A "mixed" aneurysm would result from formation of a "true" aneurysm with subsequent rupture and formation of a secondary pseudoaneurysm.

A cortical pseudoaneurysm can pursue a benign course via spontaneous thrombosis or slow enlargement. It can also manifest as delayed hemorrhage resulting in subarachnoid, subdural, or intracerebral hemorrhage.

The pathogenesis of an AV fistula in this setting would require the anatomic disruption of contiguous arterial and venous structures with containment by a common hematoma. The organization and hemodynamic excavation that produces pseudoaneurysm formation and enlargement could lead to AV fistula formation in this situation, and this mechanism is well described in the peripheral vascular tree. The rarity of a similar phenomenon in the peripheral cerebral vasculature is probably due to the concomitant cortical venous thrombosis that very likely occurs in association with an injury of this nature. This early venous thrombosis would avert development of an AV connection during canalization of the common hematoma from the arterial side of the lesion. An alternative explanation and one that we favor is the early development of an AV fistula due to concomitant arterial and venous injury, with limitation of initial high flow through the system by proximal traumatic arterial vascular spasm. Some time after the injury, as the surrounding hematoma organizes and the spasm abates, the flow through the fistula increases, producing venous congestion and ischemia which leads to delayed neurologic deficit.

Our patient's clinical deterioration on the 10th day...
Cortical AV fistula after skull fracture

after injury probably corresponds with the resolution of proximal arterial spasm and establishment of a high-flow AV shunt with resultant venous hypertension and ischemia. Neurological correlates included a declining mental status, worsening of an existing left hemiparesis, and severe paresis of the contralateral lower extremity. Any pathophysiological explanation would have to account for the observed decline in sensorium as well as the new bilaterality of the neurological findings. Clinical suspicion at this time included superior sagittal sinus thrombosis or rupture of an intracranial aneurysm with concomitant vasospasm. In the absence of overlying scalp changes or an audible bruit, the diagnosis of AV fistula at the fracture site was not entertained prior to angiography. However, this lesion could account for the observed findings, since there was increased venous pressure in the ipsilateral cortex as well as in the opposite parasagittal region. Prompt surgical intervention resulted in reversal of the acute manifestations of high-flow AV shunting, although the observed contusion and hemorrhagic venous infarction of the ipsilateral parietal region make the prognosis for complete neurological recovery less likely.

The case is instructive as an unusual manifestation of intracranial vascular injury following skull fracture. Early clinical suspicion as well as appropriate use of cerebral angiography revealed a surgically correctable abnormality before irreversible neurological deterioration occurred. The response to surgical treatment was gratifying.

References


Manuscript received May 19, 1983.
Address reprint requests to: Pablo M. Lawner, M.D., 12626 Riverside Drive, Suite 408, North Hollywood, California 91607.