Arteriovenous fistula around the ventriculoperitoneal shunt system in a patient with a dural arteriovenous malformation of the posterior fossa

Case report

SHIN-ICHI YOSHIMURA, M.D., NOBUO HASHIMOTO, M.D., KIYOSHI KAZEKAWA, M.D., ATSUSHI OBCATA, M.D., CHIKAO YUTANI, M.D., AND JUN OGATA, M.D.

Departments of Neurosurgery and Pathology, and Research Institute, National Cardiovascular Center, Suita, Osaka, Japan

A dural arteriovenous malformation (AVM) of the posterior fossa can produce persistent tinnitus, convulsions, and dementia. Successful therapeutic embolization may result in a complete cure, but in some cases, patients do not respond to the treatment. The authors report a patient with a dural AVM of the posterior fossa that did not respond to repeated intravascular treatments, but resulted in an arteriovenous shunt in the scalp around the ventriculoperitoneal shunt system. Although several hypotheses have been proposed on the pathogenesis of dural AVMs, the underlying mechanisms remain unknown. The rare complication we encountered may shed some light on the pathogenesis of dural AVMs.

KEY WORDS • arteriovenous fistula • arteriovenous malformation • embolization • ventriculoperitoneal shunt

THE case of a patient with a dural arteriovenous malformation (AVM) of the posterior fossa, which did not respond to repeated intravascular treatments is reported. The dural AVM had many feeding vessels and was accompanied by occlusion of the bilateral sigmoid sinuses and stenosis of the superior sagittal sinus. We report an unusual complication in our patient which we believe may help to elucidate the pathogenesis of dural AVMs.

Case Report

This 56-year-old man was transferred to our clinic on March 11, 1992 with status epilepticus. He had experienced generalized convulsions, gait disturbance, finger tremor, and memory disturbance since 1991 and also had a 13-year history of chronic renal failure and systemic hypertension after suffering acute glomerulonephritis.

Examination. On admission, he was in status epilepticus, which could not be controlled by an intravenous injection of diazepam. He was intubated under controlled respiration and received a continuous infusion of barbiturate.

Cerebral angiography obtained on March 12, 1992 revealed a dural AVM of the right transverse and sigmoid sinuses, which was supplied by the right occipital, ascending pharyngeal, and middle meningeal arteries and muscle branches of the vertebral artery. The sigmoid sinus was not visualized on either side. The posterior third of the superior sagittal sinus appeared to be stenotic (Fig. 1). Retrograde filling from the lesion to cortical veins could be seen through the stenotic superior sagittal sinus; these veins drained mainly into the sphenoparietal sinuses.

Operations. The patient underwent two transarterial embolizations with platinum coils and ethylene vinyl al-
cohol copolymer mixtures on May 3, and May 11, 1992. All feeding vessels except the tentorial artery were successfully embolized. On July 14, a craniotomy and embolization of the right transverse sinus with coils were performed. This treatment resulted in control of the seizure and the patient gradually regained consciousness.

Postoperative Course. Despite the administration of anticonvulsant medications, generalized convulsions recurred in October. Cerebral angiograms obtained at that time revealed occlusion of the superior sagittal sinus and appearance of a dural AVM around the superior sagittal sinus. It was observed on the angiograms that retrograde venous drainage into the supratentorial cortical veins had increased, and the circulation time was apparently prolonged. Direct infusion of urokinase into the superior sagittal sinus during the craniotomy did not recanalize the sinus.

Ventriculoperitoneal Shunt Installation. On November 18, 1992 a left ventriculoperitoneal (VP) shunt (the Rudertz shunt system) was placed to treat the patient’s hydrocephalus. No abnormal findings were seen on his scalp and the operation was uneventful; however, 10 days later, the scalp around the shunt system swelled pulsatile. This swelling regressed after compression of the left superficial temporal artery. Left external carotid angiograms obtained at that time showed an arteriovenous shunt around the VP shunt system, supplied by the left superficial temporal artery and drained into the left subclavian vein (Fig. 2). This satellite fistula and the other vascular anomalies were not seen on the selective left external carotid angiograms obtained on March 12, May 3, and May 11.

Third Operation and Outcome. Embolization with coils and ethylene vinyl alcohol copolymer mixture was performed on December 4, 1992. The pulsatile swelling of the scalp regressed and angiography demonstrated disappearance of the arteriovenous shunt; however, the patient progressively deteriorated and died on June 23, 1993.

Autopsy. The walls of the transverse-sigmoid and superior sagittal sinuses were thickened and there were many small vessels in the wall of the affected sinuses (Fig. 3 left). An old thrombus was observed in the narrow lumen of the sinuses. The scalp around the VP shunt system also contained irregularly dilated vessels (Fig. 3 right).

Discussion

In this patient, the dural AVM had many feeding vessels and was accompanied by occlusion of the bilateral sigmoid sinuses and stenosis of the superior sagittal sinus. Although the convulsive seizures and consciousness disturbance abated somewhat after endovascular surgery, the patient deteriorated due to occlusion of the superior sagittal sinus. Thrombolytic therapy with urokinase did not prove effective.

There are different hypotheses on the etiology of dural AVM associated with sinus occlusion. It has been suggested that dural AVMs may lead to sinus occlusion, because sinus occlusion has appeared after spontaneous regression of this lesion. Other researchers believe that sinus occlusion may produce dural arteriovenous fistulae (AVFs) by opening an arteriovenous shunt in the wall of the sinus. It is not clear whether the AVF is restricted to one or two sinuses. In our patient, the sinus occlusion extended to the superior sagittal sinus, which looked normal early in the course of his disease. It is interesting to
note that the dural AVM appeared around the superior sagittal sinus simultaneously with the occlusion of the sinus. Yamashita, et al.,10 reported the development of a dural AVM in the posterior fossa after intravascular treatments of the carotid cavernous fistula. Although their observations do not confirm that this lesion resulted from the occlusion of the sinus, or vice versa, they indicate that a dural AVM with sinus occlusion can extend progressively.

A very unusual finding in our case was a scalp AVF that appeared after a shunt operation; however, there was no evidence of accidental penetration of the superficial temporal artery during this surgery. Although iatrogenic AVFs have been reported after trauma or surgery, there are few reports of such distant scalp AVFs around a VP shunt system.4,5,9,10 It is possible that our patient was predisposed to the formation of an arteriovenous shunt after minute arterial trauma. If more of these cases can be documented, they may shed light on the pathogenesis of this disorder.

References


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Address reprint requests to: Nobuo Hashimoto, M.D., Department of Neurosurgery, National Cardiovascular Center, Fujishirodai, Suita, Osaka, Japan 565.