Cerebellar Venous Infarction in Chronic Suppurative Otitis Media
A Case Report With Review of Four Other Cases

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Background Cerebellar venous infarction is a rare condition. Thus far only four cases have been reported in the literature. We recently encountered a patient with chronic suppurative otitis media complicated by cerebellar venous infarction. The features of cerebellar venous infarction in the other four cases are also reviewed.

Case Description A 20-year-old man presented with clinical features suggestive of chronic suppurative otitis media. Computed tomographic scan of the brain revealed left mastoiditis with cholesteatoma and moderate communicating hydrocephalus. The patient was subjected to left radical mastoidectomy, and an attico-antral cholesteatoma was removed. Subsequently the patient developed clinical features suggestive of cerebellar abscess. A repeat computed tomographic scan revealed normal posterior fossa. Four-vessel angiography revealed left sigmoid and lateral sinus thrombosis and nonopacification of the left-sided cerebellar veins. Magnetic resonance imaging showed a venous infarct in the left cerebellar hemisphere. The patient was treated with cerebral dehydration measures. The patient subsequently improved and had no neurological deficit 3 months after surgery.

Conclusions Although cerebellar venous infarction is rare, it can occur in chronic suppurative otitis media, pregnancy, antithrombin III deficiency, and diabetic osmolar coma. Sometimes no cause is found. Treatment includes admission of the underlying cause. The presence of a hemorrhagic lesion on computed tomographic scan and deep coma at presentation indicate poor prognosis. (Stroke. 1994;25:1058-1060.)

Key Words • antithrombin III • coma • cerebellar infarction • infection • sinus thrombosis
a superficial infarct in the left cerebellar hemisphere (Fig 1) with a hyperintense signal in the left sigmoid sinus compatible with sigmoid sinus thrombosis (Fig 2). There was moderate ventricular dilatation. The patient improved steadily and had no neurological deficit 3 months after surgery.

**Discussion**

Our patient had a left middle ear infection with otitic hydrocephalus and lateral sinus thrombosis as complications. Therefore, when he acutely developed left cerebellar ataxia, a cerebellar abscess was suspected, but the CT scan showed no abscess. Because supratentorial dural venous sinus thrombosis is frequently associated with occlusion of cerebral cortical veins that drain into these sinuses, we suspected that such an event might have occurred in our patient. This was confirmed by angiography and MRI.

A search of the literature yielded four previously reported cases of venous infarction of the cerebellum. The rarity of this event is presumably due to abundant venous collaterals in the cerebellum. Here we review the features of the five patients, including our patient. All five patients had symptoms of increased intracranial pressure, probably as a result of associated dural sinus thrombosis. Three patients had cerebellar ataxia. Only one patient had loss of consciousness (case 4), which could have been due to cerebral edema in diabetic ketoacidosis or increased intracranial tension in cerebellar infarction in this patient. As with cerebral venous

**Fig 1.** Magnetic resonance image shows wedge-shaped hyperintense area in superior cerebellum (arrow) suggestive of cerebellar venous infarct.

**Fig 2.** Magnetic resonance image in the region of the skull base shows hypointense signal in left sigmoid sinus (arrowhead) compatible with sigmoid sinus thrombosis.
thrombosis, the etiology included pregnancy, dehydration, thrombotic states (anti-thrombin III deficiency), idiopathic origin, and extension of infection from extracranial structures via emissary veins (middle ear infection). Veins from the superior part of the cerebellum drain into the straight sinus, which was thrombosed in one patient (case 4). A small anterior portion of the cerebellum drained into the petrosal sinus. The larger posterior part drains into the lateral sinus, including the transverse and sigmoid sinuses, which were involved in three cases (cases 1 through 3). In our patient the thrombus had further extended into the upper part of the internal jugular vein, which is a continuation of the normal empty appearance, and small superficial cerebellar infarct.

Only one patient died, and one had mild residual ataxia. Treatment included correcting the underlying cause, measures to reduce intracranial pressure, and supportive care. Insertion of a shunt for hydrocephalus and surgical removal of the swollen cerebellar infarct may be necessary in some. The presence of hemorrhagic lesions on CT and deep coma at presentation seem to indicate poor prognosis.

References

Cerebellar venous infarction in chronic suppurative otitis media. A case report with review of four other cases.
A K Nayak, D Karnad, M V Mahajan, A Shah and Y V Meisheri

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