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 administration of antithrombin III and parenteral antibiotics were started. Laboratory investigations showed a hematocrit of 44% and leukocytosis of 25 000/m3, with 91% polymorphonuclear cells. Escherichia coli was isolated from the pus from the left ear. Plain x-ray film of the skull showed opacification of the left mastoid sinus, and a computed tomographic (CT) scan showed left mastoiditis and cholesteatoma with a moderate communicating hydrocephalus.

The patient was subjected to radical left mastoidectomy, and an attico-antral cholesteatoma was removed. At surgery, the dural plate was found to be intact. The patient’s sensorium improved progressively, but 2 days after surgery he developed titubation and unsteadiness. Neurological examination now revealed normal optic fundi, horizontal nystagmus, cerebellar ataxia in the left arm and leg, symmetrical mild spasticity with exaggerated tendon jerks, and extensor planter responses. At this time the possibility of cerebellar abscess was considered. The CT scan was repeated and showed moderate communicating hydrocephalus, normal posterior fossa, and the operated mastoid region to be cleared of cholesteatoma. The patient started vomiting the next day, for which cerebral dehydration measures (oral glycerol and intravenous mannitol) were administered.

Three weeks later, fever and the features of increased intracranial tension had subsided, but left cerebellar ataxia and titubation persisted. Four-vessel angiography at this stage revealed left sigmoid and lateral sinus thrombosis and nonopacification of the left-sided cerebellar veins. Magnetic resonance imaging (MRI) showed
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,\(^2\) 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\(^5\)\(^-\)\(^6\) of venous infarction of the cerebellum. The rarity of this event is presumably due to abundant venous collaterals in the cerebellum.\(^6\) 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
thrombosis, the etiology included pregnancy, dehydration, thrombotic states (antithrombin 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 showed hemorrhagic infarction (presumably venous) in all three (Table). In our patient the thrombus had further extended into the upper part of the internal jugular vein, which is a continuation of the sigmoid sinus and carries a significant risk of pulmonary thromboembolism.²

Computed tomographic scans were available in four cases and showed dilatation of the lateral ventricles in all and cerebellar infarction (presumably venous) in three. The cerebellum showed hemorrhagic infarction in only one patient (who died) and appeared normal in the other three, emphasizing the limitation of this imaging modality for posterior fossa pathology, particularly when situated close to the bone.⁶⁹ Angiography was available in the three cases that showed poor opacification of the cerebellar veins on the involved side in all three (Table). MRI seems to be the imaging modality of choice for this condition because it can demonstrate ventricular dilatation, thrombosis in venous sinuses (intense signal on a T₂-weighted image instead of the normal empty appearance), and small superficial cerebellar infarct.⁸⁹

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.
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Stroke. 1994;25:1058-1060
doi: 10.1161/01.STR.25.5.1058

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