Cerebellar Venous Infarction in Chronic Suppurative Otitis Media

A Case Report With Review of Four Other Cases

Atasu K. Nayak, MD; Dilip Karnad, MD; Madhuri V. Mahajan, MD; Asha Shah, MD; Yogini V. Meisher, MD

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 rt-PA and 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

Intracranial complications of middle ear infection include extradural and subdural abscess formation, pyogenic meningitis, brain abscess, lateral sinus thrombosis, and otitic hydrocephalus.1 The ear pathology generally consists of cholesteatoma and associated mastoiditis. We recently encountered a patient who was suspected to have a cerebellar abscess but subsequently proved to have cerebellar venous infarction. The features of cerebellar venous infarction, of which only four cases have been previously reported, are reviewed.

Case Report

A 20-year-old man presented with high fever, headache, vomiting, and altered consciousness for 3 days before admission. He also gave a history of purulent discharge from the left ear on and off for the last year. On examination he was febrile (40.6°C) and had a pulse rate of 120 beats per minute and normal blood pressure. On central nervous system examination he was drowsy and disoriented with normal doll's eye movement and normal pupillary reaction to light. No abnormality of cranial nerve function was detected. Power in all four limbs was normal with mild spasticity, exaggerated tendon jerks, and bilateral extensor plantar responses. Ear examination showed the presence of purulent discharge with a perforation of the ear drum, mastoiditis, and cholesteatoma of the left ear. He was suspected to have otitic hydrocephalus following chronic suppurative otitis media, 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 plantar 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 the cholesteatoma. The patient started vomiting the next day, for which cerebral dehydration measures (oral glycercol 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...
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

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

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.
Clinical Features in Five Reported Cases of Cerebellar Venous Infarction

<table>
<thead>
<tr>
<th>Case</th>
<th>Age, y/Sex</th>
<th>Symptoms</th>
<th>Signs</th>
<th>Hydrocephalus</th>
<th>Imaging Modality</th>
<th>Etiology</th>
<th>Ventricular Shunt</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22/F</td>
<td>Headache, vomiting, gait disturbance</td>
<td>Right cerebellar ataxia, left nerve VI weakness, right plantar extensor</td>
<td>+</td>
<td>Ventriculography</td>
<td>Pregnancy</td>
<td>Yes</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>2</td>
<td>46/M</td>
<td>Headache</td>
<td>Not reported</td>
<td>+</td>
<td>Angiography, CT scan</td>
<td>Antithrombin III deficiency</td>
<td>No</td>
<td>Complete recovery</td>
</tr>
<tr>
<td>3</td>
<td>42/F</td>
<td>Multiple cranial nerve palsies, incoordination</td>
<td>Left cerebellar ataxia; papilledema; left nerves V, VI, VII, VIII, IX, X, XI involved</td>
<td>+</td>
<td>Angiography, CT scan</td>
<td>Idiopathic</td>
<td>Yes</td>
<td>Mild facial weakness and ataxia persisted</td>
</tr>
<tr>
<td>4</td>
<td>57/M</td>
<td>Diabetic hyperosmolar coma with dehydration</td>
<td>Deep coma, horizontal and rotary nystagmus, extensor posturing</td>
<td>+</td>
<td>CT scan</td>
<td>Dehydration</td>
<td>No</td>
<td>Died (autopsy: diffuse cerebellar infarction)</td>
</tr>
<tr>
<td>5 (present case)</td>
<td>20/M</td>
<td>Otitis media, headache, vomiting</td>
<td>Left cerebellar ataxia, dysarthria, nystagmus, titubation</td>
<td>+</td>
<td>CT scan, angiography, MRI</td>
<td>Chronic supportive otitis media</td>
<td>No</td>
<td>Complete recovery</td>
</tr>
</tbody>
</table>

CT indicates computed tomographic; MRI, magnetic resonance imaging.

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 drains 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 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.8,9 Angiography was available in 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.8,9

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

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