

Case report

Persistent petrosquamosal sinus: Underlying cause of otitic hydrocephalus with lateral sinus thrombosis



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ABSTRACT

Lateral sinus thrombosis (LST) occurs when a middle ear infection disseminates directly via bone erosion or disseminates indirectly through the venous networks. The petrosquamosal sinus (PSS) is the residual accessory dural sinus connecting intracranial to extracranial drainage. This report describes a case of a patient with persistent PSS running through the mastoid in context of otitic hydrocephalus with LST. To identify PSS, enhanced CT and reconstructed image from CT venography were more useful than MRI. The possibility of persistent PSS running through the mastoid should be considered if LST without marked inflammation and bone erosion is noted.

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1. Introduction

Lateral sinus thrombosis (LST) is a rare intracranial complication of otitis media and mastoiditis. While LST is a still serious complication, the advent of antibiotics has reduced the incidence of this complication. Most cases of LST occur secondary to middle ear infections, except in patients with increased coagulopathy [1]. Middle ear infections spread directly to the sigmoid sinus or to spread indirectly via retrograde dissemination through the venous networks or small emissary veins [2]. When the infection causes thrombophlebitis of dural sinuses, stagnant blood flow results, which can promote formation of thrombus. If a thrombus obstructs the cerebral venous outlet to the internal jugular vein, it may lead to the elevations in intracranial pressure (i.e., otitic hydrocephalus).

The petrosquamosal sinus (PSS) is the one of the anomalous accessory sinuses running along the angle between the petrous and the squamous part of the temporal bone [3]. Though the existence of PSS has been known in anatomical fields, the CT and MRI features of PSS were first described only in 2001 [4]. Some

studies have reported that PSS may be involved in dissemination of middle ear infections to the intracranial space. However, there is no previous literature that investigates the relationship between PSS and LST. Therefore, the purpose of this report was to determine whether LST might be attributable to persistent PSS running through the mastoid.

2. Case reports

A 5-year-old male was brought to the Emergency Department with acute right otalgia. His past medical and family histories were unremarkable. His right tympanic membrane was reddish and slightly bulging. Then he was prescribed oral ampicillin and analgesics under the diagnosis of acute otitis media. On the next day, he revisited the Pediatric Emergency Department with nausea and vomiting. He was given fluids and went home, because he had no neurological deficit and did not appear seriously ill. However, he was later admitted to the pediatric ward for management of dehydration after 4 days of intermittent nausea and vomiting. On interview and examination, he complained of nausea, abdominal pain and intermittent headache, but he had no fever or otalgia. The laboratory tests showed a slight elevation of the C-reactive protein (1.9 mg/dl) but the other tests were almost within the normal range. He was treated under a tentative diagnosis of cyclic vomiting syndrome because his complaints and symptoms were ambiguous. On the seventh day of hospitalization, his intermittent headache and nausea had not changed in quality or severity. Brain

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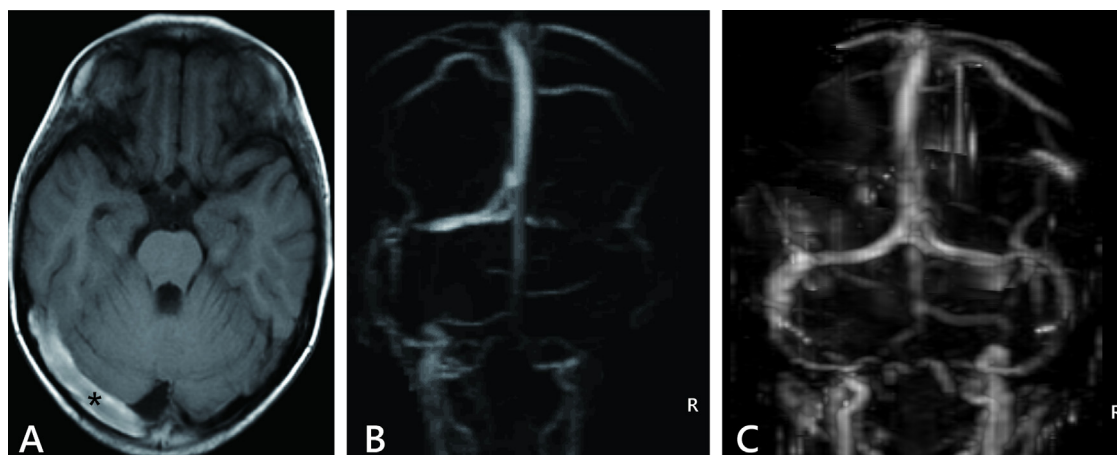


Fig. 1. (A) MRI T1-weighted axial image. The high intensity signal in the right lateral sinus indicates thrombus (*). (B) Posterior view on MR venography reveals the absence of flow in the lateral sinus. R indicates the right side of the patient. (C) MR venography performed at 2 months after treatment shows the recovery of flow in the right lateral and sigmoid sinuses.

MRI with MR venography showed that his right lateral sinus, sigmoid sinus, and the upper part of the internal jugular vein were nearly completely obstructed (Fig. 1). CT showed the soft tissue density in the tympanic cavity and mastoid cells without destruction of the surrounding bone. Reconstructed images from CT venography revealed abnormal connections between his right lateral sinus and external jugular vein (Fig. 2). His right tympanic membrane was seemed to be dull, which suggested the otitis media with effusion. Ophthalmologic examination revealed mild papilledema. The cerebrospinal fluid was clear without signs of meningitis, and the opening pressure was elevated (290 mm H₂O).

Osmotic diuretics were transiently effective in alleviating the patient's headache and nausea, but his symptoms recurred. Therefore, a ventricle-peritoneal shunt and ventilation tube insertion to the right ear was performed on hospital day 23. The culture of the middle ear effusion was resulted to be negative. Laboratory results regarding coagulation indices, including PT, APTT, AT III, and protein C and S were all within normal limits. Therefore, anticoagulation therapy was not performed. Follow-up CT and MRI at 2 months after surgery showed recanalization of the lateral and sigmoid sinuses. Enhanced CT revealed a tubular enhanced structure running through the right mastoid cavity along

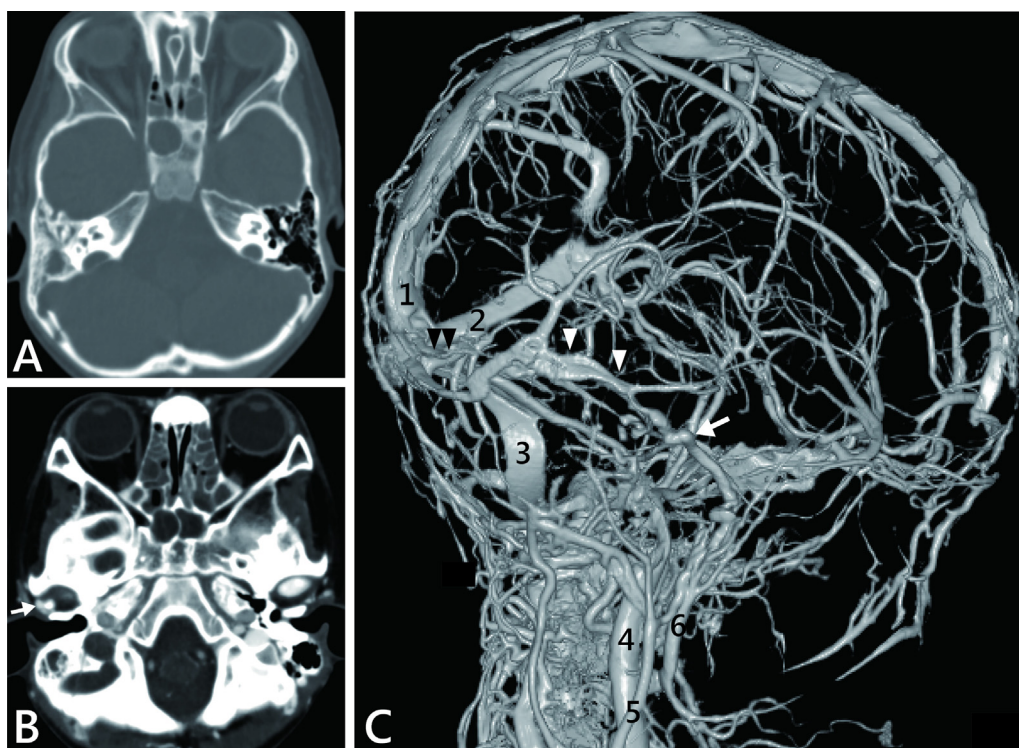


Fig. 2. (A) Bone window of the axial CT image. Soft tissue density in the mastoid without surrounding bone destruction is seen. (B) Enhancement defect of right sigmoid sinus is seen in the axial image of the enhanced CT. Right postglenoid foramen is strongly enhanced in the glenoid fossa (white arrow). (C) Three-dimensional CT images of the cranial venous systems on right lateral views. The flow of right transverse sinus is defected (black double arrowhead), and the contour of sigmoid sinus is lost. The petrosquamosal sinus (PSS) drains through the postglenoid foramen (white arrow) into the external jugular vein via connecting branches with the retromandibular vein. (1) Superior sagittal sinus; (2) Left transverse sinus; (3) Left sigmoid sinus; (4) Right internal jugular vein; (5) Right external jugular vein; (6) Right retromandibular vein.

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