



Otitic Hydrocephalus or Obstructive Hydrocephalus?

Dear Editor:

I came across the article entitled "Fatal Otitic Hydrocephalus Due to Sinus Thrombosis : A Case Report", and I would like to thank the authors for sharing their experience with this case.

In their discussion, the authors state that otitic hydrocephalus "is characterized by increased intracranial pressure with **clear CSF**, transient sixth nerve palsy, headache, vomiting, papilledema with no other detectable CNS signs and **no actual dilation of ventricles.**" They go on to state that "otitic hydrocephalus is a misnomer according to some because it may occur in the absence of otitis and because **patients do not show the ventricular dilatation seen in true hydrocephalus.**" Lastly, they state that "the diagnosis of OH is made by exclusion and **a brain abscess should be ruled out** by CT scan."

However, in the description of their case, the authors state the following:

- 1) "Lumbar tap showed elevated cerebrospinal fluid (CSF) opening pressure of 270 mm H₂O, **decreased glucose and increased protein content**"
- 2) "Repeat CT scan on the second post-op day showed **marked dilatation of the third and lateral ventricles due to compression of the fourth ventricle...**"
- 3) "He underwent ventriculostomy and **evacuation of abscess** on the fourth post-operative day."

These statements contradict the main diagnostic features of otitic hydrocephalus, and thus call into question the diagnosis of otitic hydrocephalus in this particular case. In fact, the clinical data points to the presence of an obstructive hydrocephalus. There is no doubt that the patient has evidence of lateral sinus thrombophlebitis. However, not all cases of lateral sinus thrombophlebitis are associated with otitic hydrocephalus.

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Response from the Authors

Dear Editor,

Herewith is our reply to the letter sender.

Thank you for your interest in our article "Fatal Otitic Hydrocephalus Due to Sinus Thrombosis: A Case Report" published in the Philippine Journal of Otolaryngology – Head and Neck Surgery Vol. 37 No. 1 Jan – Jun 2022. We analyzed the case, consulted specialists, reviewed the CT scans, and did further research to answer the issues raised in the inquiry. Most of the references and content in this reply were not included in the article.

First, we would like to correct our discussion statement that the fixed lateral gaze to the right was a conjugate gaze deviation that was due to abducens palsy; we now know that it may have been caused by compression of either the frontal or pontine gaze center or its interconnection which may have been affected by the enlarged ventricle.¹ Conjugate gaze deviation was also observed in an 18-year-old young man diagnosed with otitic hydrocephalus reported in the British Medical Journal in 1932.²

In the same article,² Symonds said that the average normal cerebrospinal fluid (CSF) pressure is 150 mmH₂O, in brain abscess the pressure is normal up to 250 mmH₂O while in hydrocephalus it is around 300 mmH₂O. By the time symptoms of increased intracranial pressure (ICP) are evident, cerebellar abscess will manifest with "localizing signs" of which the most important is nystagmus and limb incoordination ipsilateral to the lesion. In left temporal lobe abscess, "word forgetfulness," weakness of the opposite face, and homonymous defect in the opposite visual field may be manifested.² In our case, the CSF pressure was 270 mmH₂O prior to mastoidectomy which doubled to 543 mmH₂O four days later when ventriculostomy was done. There were no localizing signs except for meningeal irritation and slight weakness on the right upper (4/5) and lower extremities (3/5) which improved with antibiotics. After VP shunt, conjugate gaze was relieved while ICP and CSF normalized. However, the GCS continued to decline and central herniation due to hydrocephalus and bacterial meningitis were considered for his demise.

In 1939, Gardner reported a case of otitic sinus thrombosis in a 9-year-old girl where postmortem examination showed a

fibrous cord on the left lateral sinus that caused increased ICP, acute thrombophlebitis of the left superior petrosal and sagittal sinuses and fibrinopurulent leptomeningitis.³ He said these findings led Symonds to consider that otitic hydrocephalus may be due to thrombosis of the intracranial sinuses which by partially obstructing the outflow of venous blood, causes cerebral venous engorgement and consequent rise in ICP.³ The thrombus is a protective measure and is not infected from the beginning. It is nature's way of blocking the blood channel in an effort to prevent further spread of infection, but it is the secondary infection of the clot and not the thrombus that constitutes the danger.³

A 7-year-old girl diagnosed with otitic hydrocephalus presented with subacute mastoiditis in the right ear and initially showed increased ICP with no CSF protein elevation, but after 2 months of confinement, died of pneumococcus type III meningitis.³ Gardner explained that this may be due to thrombosis of the right jugular bulb which occurred with the onset of otitis; sterile subdural effusion occurred as a result of either thrombosis or external pachymeningitis.³ He further opined that the subdural effusion plus the venous congestion occasioned by the thrombosis of the right jugular bulb was responsible for the high ICP and that the thrombophlebitis gradually spread from the right jugular bulb to most of the major intracranial sinuses and cerebral veins, with meningitis terminating the picture.³ Similarly, a 12-year-old girl with 6-year history of otorrhea was diagnosed with otitic hydrocephalus and cerebellar abscess secondary to atticofurrow ear disease,⁴ and the CT scan of another case of otitic hydrocephalus showed dilatation of the left lateral ventricle body.⁵ Although rare, otitic hydrocephalus, meningitis, cerebellar abscess and ventricular dilatation may develop at the same time.

Our patient had CSOM, headache, papilledema, lateral sinus thrombosis (LST) on CT and perisinus abscess and cholesteatoma on mastoidectomy. Secondary infection of the clot or thrombus, subdural effusion, and meningitis may have developed requiring drainage and ventriculostomy four days later when the patient's condition deteriorated. Serial CT scans showed no ring enhancement although fluid accumulation or possible abscess formation was noted 10 days after ventriculostomy. Culture of the ear discharge grew *Proteus mirabilis*





while samples of the brain abscess drained during ventriculostomy had no growth after 3 days.

Communicating hydrocephalus may develop at the outset and may become obstructive later especially when the condition is associated with meningitis or brain abscess. Lateral sinus thrombosis brought about by chronic ear disease may have altered the hemodynamics of the central venous circulation which in turn compromised the CSF environment. This was documented in the imaging studies that showed progression of dilatation of the right sigmoid, transverse and superior sagittal sinuses. Weeks prior to admission when the patient was experiencing headache, alterations in ICP and CSF protein may have occurred and subsequently elevated when the condition worsened upon admission. Since the veins and venous sinuses are in the subarachnoid space, inflammation may reach the CSF and pleocytosis may develop.⁶ This hypothetical sequence of events should have been documented with serial CSF determinations, angiography and autopsy.

We postulate that otitic hydrocephalus may be considered as a clinical spectrum and a dynamic disease process evolving in a continuum. In the early phase, sigmoid sinus thrombophlebitis, increased ICP, clear CSF, and absent ventricular dilatation may be manifested which usually improve with antibiotics and VP shunt and are therefore reversible. These were the early observations^{2,3} which may be attributed to the short duration of ear infection, early detection and management. These may represent the initial clinical picture of the patient. However, if ear infection continues with no intervention, the late phase may unfold characterized by propagation of sigmoid sinus thrombosis, subdural effusion, meningitis, increase ICP and CSF protein, and ventricular dilatation with which our patient may be

classified. These may represent the dire outcome of persistent chronic ear infection and LST; the latter fraught with the risk of complete sagittal sinus occlusion which may result in severe neurologic deficits and death.⁷

We took note of the contradictory issues in our case and if we are going to follow definitions to the letter, concede that the case may not qualify. But if we are going to consider the parameters as preludes or antecedent events and the late phase manifestations as possible consequences which may have transpired, then our diagnosis may not be that farfetched or remote.

We hope that this reply explains our side. We agree with and fully respect the opinion given by the good doctor.

Thank you.

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