A CHILD WITH OTITIC HYDROCEPHALUS

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Abstract

Acute otitis media (AOM) is a widespread disease which may lead to extra- and intracranial complications. Intracranial complications of AOM are currently still potentially health and life threatening even with proper treatment. Otitic hydrocephalus is a rare intracranial complication of otitis media. It is characterized by elevated cerebrospinal fluid (CSF) pressure with normal CSF biochemistry and without any focal neurologic abnormality other than those related to the increased intracranial pressure. The precise mechanism underlying the development of otitic hydrocephalus is unknown. Eradication of ear disease and lowering of the elevated intracranial pressure are the goals of the therapy. The authors of this essay will introduce a 7 years old boy with otitic hydrocephalus and review the literature. It can be concluded that, in patients with acute or chronic ear infections, complaints of headache, blurred vision, diplopia, or photophobia may be a heralding sign of an intracranial complication. On the other hand, MRI is very important for detection of the complications of otitis media. Contrast-enhanced MRI and magnetic resonance venography must be immediately performed in patients with neurological symptoms such as nerve palsy, neck stiffness, or confusion.

Keywords: acute otitis media, increased intracranial pressure, otitic hydrocephalus, neurological effects

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Background

Otitic hydrocephalus manifests as signs and symptoms indicative of increased intracranial pressure. This condition can arise as a rare complication of acute otitis media, chronic otitis media, or otologic surgery (1,2). In a series of 100 cases of intracranial complications secondary to infectious ear disease, Gower and McGuire found that only 5 patients had otitic hydrocephalus (3). In a 15-year study of intracranial complications of otitis media, de Oliveira Penido et al did not find any case of otitic hydrocephalus (4).

Case presentation

A 7 years old Iranian boy with no obvious previous medical history, no significant past otological history or other neurological problems, was admitted with 10 days of headache, unilateral eye gaze, photophobia and fever. His symptoms initiated with headache and then, after 5 days, fever, photophobia and diplopia added. He had no complains of nausea, rhinorrhea, cough or otalgia. No changes in his level of consciousness was reported. He didn't have any previous recurrent otitis media or sinusitis. And also, he had no history of head trauma and or recent upper respiratory tract infection.

On physical examination at the time of admission, blood pressure was 100/75 mmHg, Pulse rate 100/ minutes and temperature was 38.8. There was no conjunctival injection. The funduscopic examination was normal. his right eye had inward deviation and outward movement restriction with normal pupil size and normal light response of pupil was significant, which was remarkable as the 6th cranial nerve palsy (abducens nerve palsy (CN VI palsy)) (fig.1). He had decreased range of motion in right eye scored as -4 and other eye examinations were in normal range. He examined for Brown syndrome, which was negative. No uveitis was seen with slit lamp examination. tympanic membrane was intact and normal. All other physical examinations were normal.

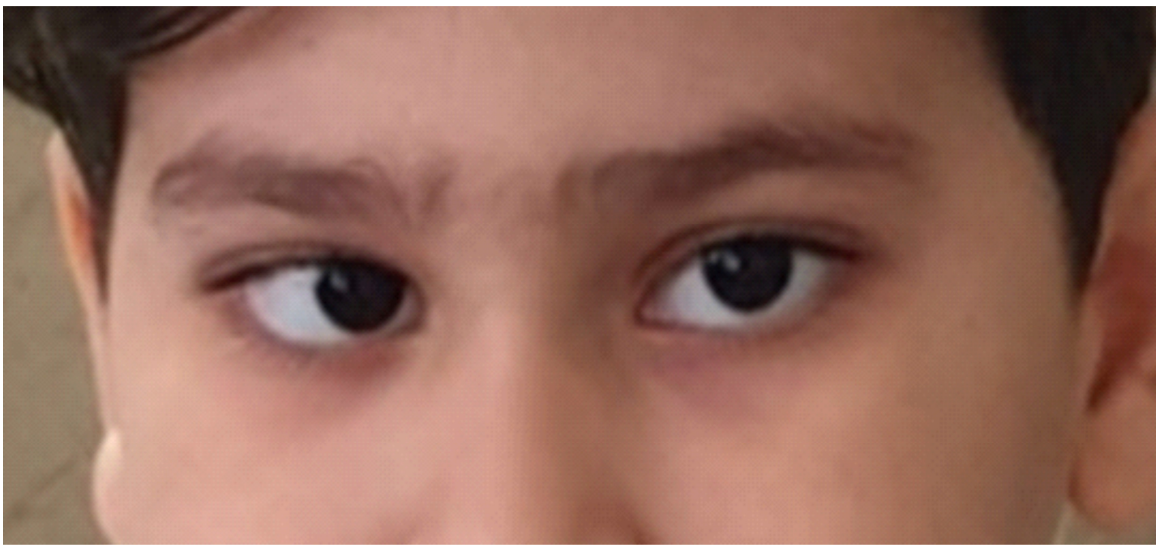

Fig. 1

His audiogram demonstrated normal hearing in both ears on pure tone averages.

In his laboratory tests, he had Normal complete blood count (CBC), C-reactive protein(CRP) 46, erythrocyte sedimentation rate(ESR) 127. Blood culture was positive with strep viridans. A lumbar puncture was done. So, CSF analysis: WBC: 7 PMN, RBC:547, Glu:62, Pro:24, Smear: normal, Culture: negative. Cerebrospinal opening pressure was normal.

In his rheumatologic workups, a high lupus anticoagulant antibody was reported, while an-

tiphospholipid Ab (IgM, IgG), HLA-B5 and B51, Anti b2 globulin, FANA, Anticardiolipin Ab (IgM, IgG), Anti b2 glycoprotein Ab (IgM, IgG), PANCA, CANCA, ACE, Protein C & S, Factor V Leiden, Ferritin, IL-6 and Procalcitonin were all within normal laboratory range. A chest X-ray was done, which was normal. Normal echocardiogram with normal anatomy of valves with no vegetation.

A tentative differential diagnosis of acute bacterial meningitis complicated by cerebral edema, acute hydrocephalus or cerebral abscess, cerebrovascular events and intracranial hemorrhages was made.

To resolve the diagnostic dilemma, Magnetic Resonance Image (MRI) of brain with and without intravenous contrast ordered. Intravenous Ceftriaxone (50mg/kg/dose 4 times a day), vancomycin (20 mg/kg/dose 4 times a day) and acyclovir (10mg/kg/dose three times a day) started with the first impression of meningitis.

Brain MRI confirmed Mild mucosal thickening of sphenoid. Fluid retention at right mastoid air cell with apparent intra septa as well as abnormal signal intensity at petrosal apex which is noted for ruling out of petrosal apititis. Right side otomastoiditis was notable. Fig.2-6

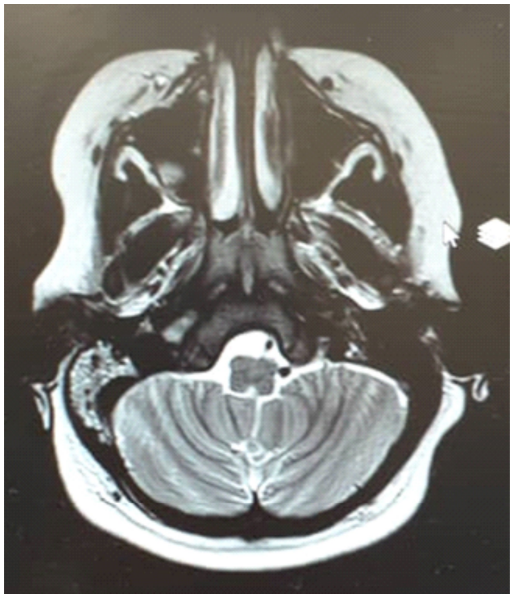

Fig. 2

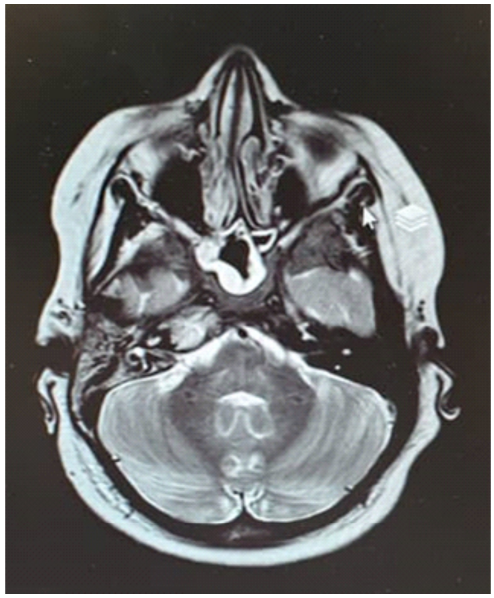

Fig. 3

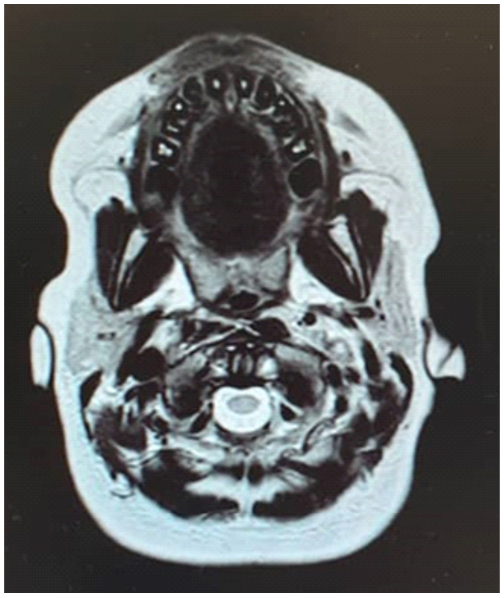

Fig. 4

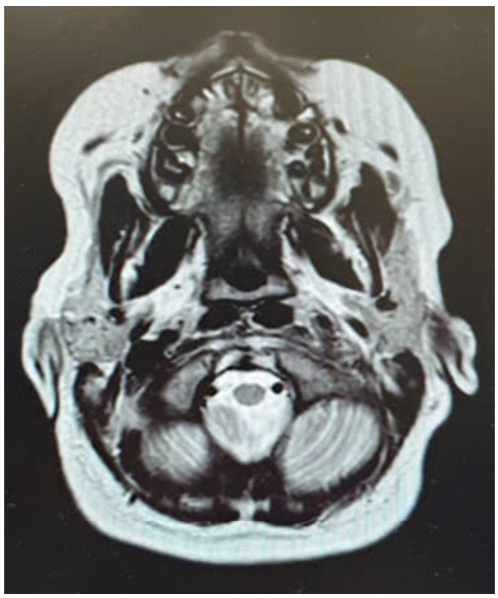

Fig. 5

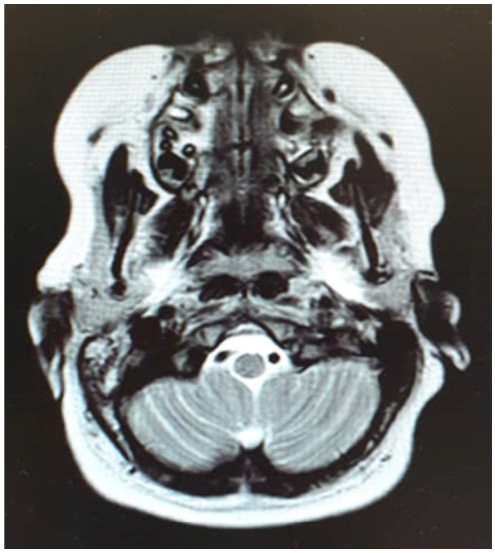

Fig. 6

Brain MRV showed Central filling defect in left sigmoid sinus. Left jugular bulb and proximal part of left internal jugular vein were compatible with CVT (central venous thrombosis).

Right temporal bone CT scan with and without intravenous contrast was done, in which right side chronic otitis media with sclerosis and mastoidial cells turbidity (loss of pneumatisation of the mastoid air cells) and thickening of temporal membrane reported.

In order to prevent life-threatening complications such as thrombosis of the sinous, the patient was given Enoxaparin 1mg/kg/dose (max 40mg) twice a day, subcutaneous injection, started. His antibiotics changed to Linezolid and Meropenem and continued for 14 days. An intravenous Methylprednisolone with dose of 1mg/kg/day and oral Acetazolamide prescribed.

After 5 days of IV antibiotics, a complete recovery of pyrexia was obtained, and 10 days after the initiation of treatment the neurological examination showed a partial recovery of the sixth nerve palsy. After 3 weeks of admission His inflammatory markers returned to normal.

Discussion and Conclusion

Acute otitis media (AOM) is a widespread disease which may lead to extra- and intracranial complications. Intracranial complications of AOM are currently still potentially health and life threatening even with proper treatment. The use of antibiotics has led to reduction in the incidence of intra- and extracranial complications from approximately 17%to 1%(5). Because these complications have declined markedly since the advent of antibiotics, many contemporary otolaryngologists have been unexposed to these complications. Furthermore, patients' symptoms are now often masked, so patients appear remarkably well despite the presence of potentially fatal complications (10). Severe complications of AOM such as subperiostal abscess, Bezold's abscess, facial nerve paralysis, osteomyelitis, meningitis, lateral sinus thrombosis (LST), extradural abscess, subdural empyema, brain abscess and otitic hydrocephalus(OH) have become very rare today (6-8).

The classical syndrome of otitic hydrocephalus consists of the signs and symptoms of elevated CSF pressure. It is more common among children and adolescents. The onset may delay several weeks after the acute ear disease or after years of chronic ear disease. (9). There are no focal neurological findings other than papilledema and occasionally 6th nerve palsy. In affected patients, the results of cerebrospinal fluid (CSF) bio-chemistry studies on lumbar puncture are normal.

The precise mechanism underlying the development of otitic hydrocephalus is unknown. Because superior sagittal sinus thrombosis should be associated with more neurologic deficits than are found in otitic hydrocephalus, it is postulated that a mural non-obstructing thrombus extending to the superior sagittal sinus impedes CSF resorption by pacchionian bodies and results intracranial hypertension (11). An alternative mechanism proposes that the presence of thrombus in the lateral sinus leads to impeded venous drainage into the neck, especially if the thrombus occurs in a dominant lateral sinus

(12). An increase in the intra cranial pressure may then be produced either by direct transmission of the raised venous pressure to the CSF or by impending the function of the arachnoid villi (13). However, ligation of the internal jugular vein in the neck does not cause hydrocephalus; thus, that mechanism must be the suspect. If a dominant venous sinus becomes obstructed, in the presence of inadequate cross communication at the trochlea, venous drainage may be sufficiently impaired to cause raised intracranial pressure. Probably because of this anatomical variation, otitic hydrocephalus is expected to be seen more commonly in right-sided ear disease (14).

Radiologic imaging may help us to identify the possible main physiologic mechanisms of otitic hydrocephalus. In this regard, as Magnetic resonance imaging allows for superior evaluation of the venous sinuses, it is the imaging modality of choice (2). for the determination of intra-sinus thrombosis, Magnetic resonance venography (MRV) can be used, which could not be diagnosed by classic venography (15).

Eradication of ear disease and lowering of the elevated intracranial pressure are the goals of the therapy. Acute cases of ear disease may be resolved spontaneously. Due to some vital merits persisting middle ear infection has to be treated. Surgical procedures must consist of cleaning the disease completely from middle ear and mastoid and draining the perisinus abscess, if present. Nowadays, to control suppurative thrombophlebitis complete mastoidectomy with evacuation of all middle ear and mastoid disease, drainage of the perisinus abscess, and clot removal, along with high-dose appropriate antimicrobial medications administered intravenously, is considered to be adequate. Those cases, in which sepsis continues, despite an adequate surgical procedure and appropriate intravenous antibiotic therapy, Ligation of the internal jugular vein is reserved (2). Raised intracranial pressure may be treated by the use of steroids, diuretics such as acetazolamide, and hyperosmolar dehydrating agents (mannitol). Repeated lumbar puncture has been advocated, but this is not risk free in the presence of raised intracranial pressure. Furthermore, in some articles, repeated LPs are referred to have only historic importance (10,15,16). Because otitic hydrocephalus occurs regularly in conjunction with lateral sinus thrombosis, anticoagulation therapy may be considered as a therapeutic option.

All in all, necessitating a high index of suspicion, Otitic hydrocephalus is an uncommon complication of otitis media, with potentially significant morbidities. In patients with acute or chronic ear infections, complaints of headache, blurred vision, diplopia, or photophobia may be a heralding sign of an intracranial complication.

And also, MRI is very important for detection of the complications of otitis media. Contrast-enhanced MRI and magnetic resonance venography must be immediately performed in patients with neurological symptoms such as nerve palsy, neck stiffness, or confusion.

Declaration

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Resumo

Akuta meza otito (AMO) estas ofta malsano, kiu povas konduki al ekster- kaj intrakraniaj

komplikaĵoj. Intrakraniaj komplikaĵoj de AMO ankaŭ nuntempe estas ebla minacoj por sano kaj vivo eĉ dum adekvata kuracado. Otita hidrocefalo estas malofta intrakrania komplikaĵo de AMO-meza. Ĝi estas karakterizita per altigita premo de cerebrospina likvaĵo (CSL) kun normala CSL-biokemio kaj sen iu fokusa neŭrologia anomalio krom tiuj en rilato al la pliigita intrakrania premo. La preciza mekanismo de la evoluo de otita hidrocefalo estas nekonata. Elradikigo de orelmalsano kaj malpliigo de la altigita intrakrania premo estas la celoj de la terapio. La aŭtoroj de ĉi tiu eseo prezentos 7-jaran knabon kun otita hidrocefalo kaj recenzos la literaturon. Oni povas konkludi, ke ĉe pacientoj kun akraj aŭ kronikaj orelinfektoj, plendoj de kapdoloro, neklara vizio, diplopio aŭ fotofobio povas esti anonca signo de intrakrania komplikaĵo. Aliflanke, magneta resonanca bildigo (MRB) estas tre grava por detekto de la komplikaĵoj de AMO. Kontrast-plifortigita MRB kaj magneta resonanca venografio devas esti tuj faritaj en pacientoj kun neŭrologiaj simptomoj kiel ekzemple nerva paralizo, kolo-rigideco aŭ konfuzo.

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