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Case Report

Ophthalmic and Neurological Manifestations of Otitic Hydrocephalus: A Pediatric Case Report

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Abstract

Pediatric Otitis Media (OM) can result in rare intracranial complications such as otitic hydrocephalus. This case report describes a 6-year-old girl with acute OM, right esotropia, and bilateral papilledema. Imaging revealed mastoiditis and intracranial complications, which required mastoidectomy, ventilation tube insertion, tympanomastoidectomy, and lumbar puncture. Early recognition of ophthalmic signs is crucial for prompt diagnosis and intervention.

Keywords: Esotropia, Hydrocephalus, Mastoidectomy, Mastoiditis, Papilledema, Spinal puncture

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Introduction

Pediatric Otitis Media (OM) is the most common clinical condition that can lead to rare complications, such as otitic hydrocephalus and Intracranial Hypertension (IH). *Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus*, and *Streptococcus pigeons* are the main pathogens responsible for mastoiditis. In a study by Favre *et al*, only 1.2% of 2061 patients (mostly <7 years old) had otitic hydrocephalus, which was characterized by elevated Cerebrospinal Fluid (CSF) pressure and normal CSF biochemical parameters (1). In addition, Penido *et al* did not report any cases of otitic hydrocephalus in a 15-year study focusing on the intracranial complications of OM (2).

IH is characterized by papilledema, elevated Intracranial Pressure (ICP), and the absence of intracranial and extracranial space-occupying lesions. ICP is associated with mastoid effusion (3) and can drive spontaneous Tympanic Membrane Displacement (TMD) in humans (4). In some patients, the degree of TMD has been correlated with ICP levels, measured through Lumbar Puncture (LP) manometry (5). However, there is a significant correlation between visual field impairment and high CSF pressure (6). Radiological imaging in the form of Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) has been instrumental in localizing the source and consequences of infectious complications of untreated OM (7). Early diagnosis and treatment planning are required to preserve visual function. Antibiotic therapy is the main treatment, and some surgical interventions, such as incision of abscesses, mastoidectomy, and neurosurgical procedures, are performed in combination with medical therapy for severe complications (8).

In the present case, IH was caused by untreated or incompletely treated mastoiditis. This report describes a case of IH associated with otitic hydrocephalus based on the CARE (Case Report) guidelines (https://www.equator-network.org/reporting-guidelines/care/).

Case Report

A 6-year-old girl presented to the Ophthalmology Department of Bu-Ali Sina Hospital in 2024 with esotropia of the right eye (Figure 1), which was first noticed three weeks before the consultation. She had a history of acute OM one month prior, and no other significant medical or developmental abnormalities were reported in her history. Fundus examination revealed bilateral papilledema, while other neurological examinations were unremarkable. In accordance with institutional policy and the regulations of the Ethics Committee at Mazandaran University of Medical Sciences, written informed consent was signed from the parents, and the child's assent was also obtained.

Figure 1. The presence of esotropia in the right eye with outward restriction suggests a significant 6th nerve palsy (abducens nerve palsy).

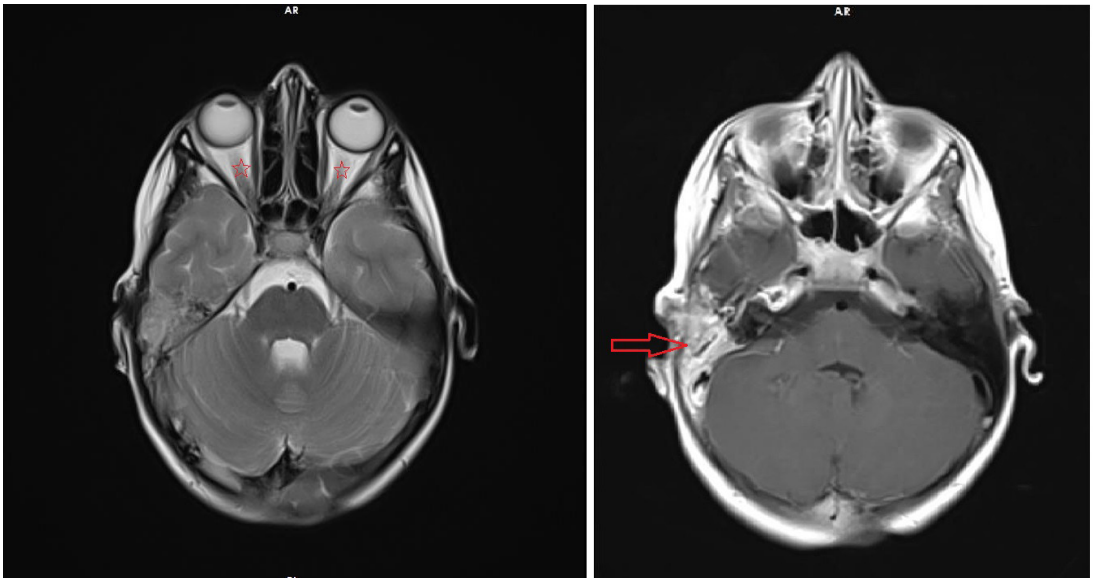

Figure 2. The brain MRI showed bilateral papilledema (marked by stars), thickening of the sphenoid mucosa, and fluid collection in the right mastoid air cell (highlighted by a red arrow), confirming a diagnosis of right-sided otomastoiditis.

Clinical findings

The patient was diagnosed with mastoiditis following an MRI scan that showed a significant fluid signal intensity in the right mastoid air cells, indicative of inflammatory effusion (Figure 2). Normal cerebral MRI findings ruled out parenchymal lesions and venous sinus thrombosis in this patient. The high T2 signal intensity in the right transverse and sigmoid sinuses was probably due to sluggish flow, as evidenced by normal contrast enhancement after gadolinium-based MRI contrast agent

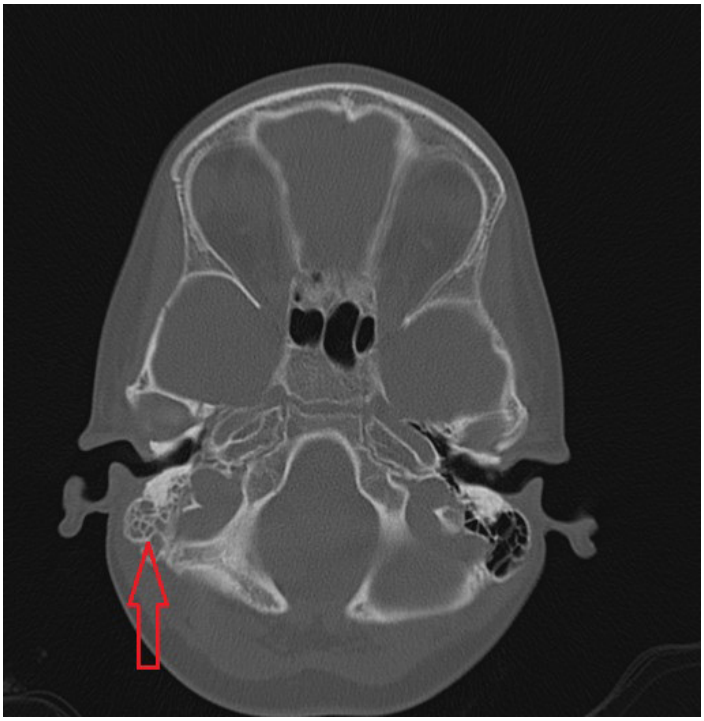

Figure 3. A CT scan without intravenous contrast identified acute otitis media on the right side, accompanied by sclerosis, opacity of mastoid cells, and thickening of the tympanic membrane (highlighted by a red arrow).

Table 1. Chronological summary of clinical events, investigations, and interventions

Day onset	Clinical symptoms	Investigations	Key findings	Interventions	Outcome
Day 0	Onset of acute otitis media	-	Ear pain, fever	Initial oral antibiotics prescribed locally	Partial symptom relief
Day 5	Worsening headache, vomiting	ENT exam, otoscopy	Mastoid tenderness, bulging TM	Referred for imaging	-
Day 6	First hospital evaluation: acute otitis media with progression to mastoiditis	CT, MRI, MRV	Mastoid opacification; papilledema; patent venous sinuses	Mastoidectomy, ventilation tube insertion; started IV vancomycin 250 *mg* q 6 *hr*	Close neuro/ ophthalmic monitoring
Day 7	Persistent headache, Ophthalmic symptoms (papilledema, abducens nerve palsy)	Ophthalmology exam, fundoscopy, LP attempted	Bilateral papilledema; parental refusal of repeat LP	Initiated acetazolamide 250 *mg* q 8 *hr*	Papilledema persisted but controlled
Day 8	Continued papilledema	Ophthalmology review, fundoscopy	Disc swelling stable	Continued vancomycin+ acetazolamide; ENT follow-up	Multidisciplinary monitoring
Day 9	During hospitalization	Ophthalmology follow-up	Disc swelling decreased, abducens nerve palsy improved	Continued vancomycin+ acetazolamide	Progressive improvement
Day 10	Discharge	Fundoscopy, clinical exam	Papilledema decreased, stable vision	Transitioned to oral co-amoxiclav +continued acetazolamide	Outpatient follow-up planned
Week 4	Follow-up visit	Fundoscopy, MRI, visual acuity	Papilledema regressing; VA stable	Continued tapering acetazolamide	Good clinical recovery
Month 3	Long-term follow-up	Fundoscopy, visual acuity	Papilledema resolved; VA Snellen 20/20	Off acetazolamide and antibiotics	Full recovery

Computed Tomography (CT); Magnetic Resonance Imaging (MRI); Magnetic Resonance Venography (MRV); Tympanic Membrane (TM); Lumbar Puncture (LP).

(GAD) injection and normal Magnetic Resonance Venography (MRV) findings. A CT scan of the right temporal bone without intravenous contrast showed OM, mastoid cell opacity (loss of pneumatization of the mastoid air cells), and thickening of the temporal membrane (Figure 3).

In addition, LP showed a high opening pressure of 450 *mm* H_2O with normal CSF parameters. The clear CSF with a protein level of 20 *mg/dl* and glucose level of 78 *mmol/l*, as well as the absence of white and red blood cells in the CSF culture, were all within the normal range for CSF composition. In terms of ophthalmic complications of OM, esotropia secondary to abducens nerve palsy and papilledema developed.

Therapeutic intervention

The patient underwent mastoidectomy and Ventilation Tube (VT) placement based on the CT scan results. Tympanomastoidectomy of the right ear was successful revealing excessive granular tissue and dural corrosion. The granular tissue was removed, and myringotomy was performed. After consultation with an ENT specialist, the patient was started on vancomycin (250 *mg* every 6 *hr*) as part of her treatment plan.

Follow-up and outcomes

Based on the ophthalmological consultation and the presence of papilledema, it was recommended to continue medical treatment and perform a follow-up LP. After one week of antibiotics and a primary LP, the ophthalmological examination showed normal results for abducens nerve palsy with the presence of bilateral papilledema, which had decreased since the previous examinations. The ophthalmologist recommended treatment with acetazolamide and repeated LP. Acetazolamide was selected to reduce CSF production and thereby lower intracranial pressure, which is the standard practice in the management of IH. The initial LP confirmed markedly elevated opening pressure (450 *mm* H_2O), and repeat LPs were advised for both therapeutic relief and monitoring response. However, the parents declined further LPs after the initial procedure. Given this limitation, acetazolamide was used as a pharmacological alternative to serial LPs. The starting dose of 250 *mg* every 8 *hr* (half a tablet) was chosen based on pediatric dosing guidelines (approximately 15–25 *mg/kg*/day, divided into 2-4 doses), while also considering the patient's age and tolerance. This regimen aims to provide effective ICP control while minimizing potential side effects, such as paresthesia, metabolic acidosis, or gastrointestinal upset. Ultimately, the combination of mastoidectomy, antibiotics, and acetazolamide led to the resolution of papilledema and sixth nerve palsy, with full visual recovery at the one-month follow-up. Thus, acetazolamide plays a critical role in controlling IH.

The patient was discharged after 10 days in good condition. The prescribed treatment included oral antibiotics (co-amoxiclav) and 250 *mg* of acetazolamide (half a tablet every 8 *hr*) with weekly follow-up. Acetazolamide was continued for three months until the complete resolution of papilledema. Ophthalmic and neurological complications were resolved, with normal visual acuity (Snellen 20/20) and normal fundus examination at a mean follow-up of one month (Table 1).

Discussion

Otitic hydrocephalus in children is primarily caused by mastoiditis, leading to cerebral venous sinus thrombosis and resulting in elevated intracranial pressure without ventricular dilation. Although OM in children has been managed with the prescription of antibiotics, signs and symptoms may be masked during the treatment process, and the onset of symptoms may be delayed by several weeks (9-11). Thus, some unmanaged cases of acute OM may enter the acute phase and lead to otitic hydrocephalus, IH, and even death (12). The exact mechanism of otitic hydrocephalus remains unclear; however, increased intracranial pressure may be caused by the direct transmission of increased venous pressure to the CSF or by the impairment of arachnoid villus function (13). If the dominant venous sinus is obstructed, venous drainage may be sufficiently impaired to cause increased intracranial pressure in the presence of inadequate cross-communication at the trochlea. Probably because of this anatomical difference, otitic hydrocephalus is expected to be more common in right ear disease (14) and consequently may cause sixth nerve palsy in the right eye.

The main physiological mechanisms of otitic hydrocephalus can be identified using radiological imaging, particularly MRI (7). Recent studies have highlighted the importance of comprehensive imaging approaches for the accurate diagnosis of this condition. Subtle imaging signs, such as loss of flow void and intrinsic T1 shortening in the sigmoid sinus, can be indicative of thrombosis, underscoring the need for detailed imaging evaluation (15). In the current case, multimodal imaging was essential in determining the cause of the elevated intracranial pressure. MRI revealed subtle T2 hyperintensity in the transverse and sigmoid sinuses, which initially raised concerns regarding venous thrombosis. However, MR venography confirmed preserved venous flow, and contrast-enhanced MRI revealed patent sinuses, thus ruling out cerebral venous sinus thrombosis. CT scans revealed mastoid opacification and bony changes consistent with mastoiditis. These findings support the diagnosis of otitic hydrocephalus, in which impaired venous drainage and reduced CSF absorption, rather than true sinus occlusion, underlie the development of papilledema and IH.

The use of acetazolamide and repeated LP has been advocated in these cases (16), and mastoidectomy, drainage of any abscess, and clot removal are the most common treatments. (17) Similar to the strategy described by Maiz *et al* (15), our approach combined

acetazolamide with targeted antibiotic therapy to address both raised intracranial pressure and the underlying infectious cause, thereby offering a more comprehensive management. In contrast, Chen *et al* (18) studied 15 patients aged 2-15 years and found that, although most developed papilledema, only one-fifth presented with sixth cranial nerve palsy. They emphasized the importance of systemic corticosteroid therapy in preventing the need for invasive neurological interventions and minimizing the risk of vision loss associated with otitic hydrocephalus.

Early identification and treatment of patients with ocular complications of OM can help prevent permanent neurological and visual complications. The patient of this study had a history of OM that developed due to neurological and ophthalmic complications. These patients are not routinely referred to ophthalmology unless they experience blurred vision, double vision, strabismus, or papilledema. With prompt intervention, most children with mild disc edema and visual field defects completely recover. In this case, one month after treatment, the patient had a visual acuity of 20/20 bilaterally and complete recovery from disc and macular edema. Papilledema is a reliable indicator of elevated intracranial pressure in children, and careful fundoscopic examination can avoid the need for invasive procedures in these children. Similarly, Mitchell *et al* (19) confirmed that papilledema identified by fundoscopy demonstrated a Positive Predictive Value (PPV) of 1.00 and a Negative Predictive Value (NPV) of 0.64, with a sensitivity of 48% and specificity of 100% for detecting raised intracranial pressure ($p<0.0001$).

Clinicians should maintain a high level of suspicion in patients presenting with headache, blurred vision, or diplopia. They should be particularly alert to the development of neurological symptoms, which, in our case, were associated with a high incidence of IH.

Conclusion

Complications associated with acute mastoiditis as a unique clinical entity remain to be discussed. Patients with intracranial complications were more likely to require extensive treatment, with longer hospital stays and higher total costs, which is consistent with the current understanding of this disease process. However, there are still considerable variations in the management of these intracranial complications, and the ability to establish a standard of care for children with complicated acute mastoiditis requires further investigation.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Source of support

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Informed consent

The clinical evaluations were explained to the patient and the patient signed the informed consent form. Written informed consent was obtained from the patient.

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Conflict of Interest

There was no conflict of interest in this manuscript.

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