Case Report

Bacterial Labyrinthitis in an Adult with Acute Otitis Media

Jacob H. Reisner1 and Gregory J. Basura2

1 College of Osteopathic Medicine, Des Moines University, Des Moines, IA 50312, USA
2 Department of Otolaryngology—Head and Neck Surgery, University of Michigan, Ann Arbor, MI 48109, USA

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Abstract We report a rare case of fulminate suppurative bacterial labyrinthitis in an otherwise healthy female who presented with bilateral acute otitis media. The presentation of purulent otorrhea with concurrent vertigo, vigorous spontaneous left-beating nystagmus, and hearing loss was compelling for the diagnosis. This report discusses the clinical and radiographic presentation and findings consistent with bacterial labyrinthitis and stresses the importance of expedited diagnosis and medical and surgical intervention to avoid further complication as well as minimize permanent inner ear damage. We also discuss posttreatment inner ear evaluation and rehabilitative options based on permanent insults.

Keywords labyrinthitis; acute otitis media; nystagmus; sensorineural hearing loss; peripheral vestibulopathy

1. Introduction

In the preantibiotic era, acute otitis media (AOM) often led to serious complications that at times were even fatal [1,3,6,10]. Complications were thought to be bacterial in nature although viral causes of AOM have been documented [2]. Labyrinthitis is a rare potential complication of AOM. Labyrinthitis associated with AOM results from either serous irritation or from direct bacterial infection of the inner ear. In acute suppurative labyrinthitis, bacterial toxins penetrate into the inner ear and perilymph resulting in cochlear and vestibular irritation and inflammation. Vertigo and potentially permanent vestibular and sensorineural hearing loss (SNHL) often result [3]. While inflammation of the inner ear structures can be seen on MRI, acute suppurative labyrinthitis is typically a clinical diagnosis [4]. The differentiation of serous labyrinthitis from acute suppurative labyrinthitis can often be made from cultures from middle ear exudate following therapeutic myringotomy [3]. We present a case of unilateral bacterial labyrinthitis in an otherwise healthy adult female who presented with bilateral AOM.

2. Case presentation

A 35-year-old white female presented to the emergency department (ED) with a one-week history of progressive bilateral otalgia, purulent otorrhea, hearing loss, vertigo, nausea, and vomiting. The initial dull pain and “fullness” in the right ear was diagnosed as otitis externa and topical antibiotics were prescribed. Bilateral otalgia culminating in purulent otorrhea led to the diagnosis of AOM and oral Cefediniir was later started. She returned to the ED with worsening otottrhea, hearing loss, and new-onset spontaneous vertigo with an inability to walk independently and was admitted to the hospital. Otolaryngology was consulted and otoscopic examination revealed profuse purulent otorrhea and bulging erythematous tympanic membranes. A CT scan of the brain showed bilateral mastoid and middle ear opacification with no acute intra-cranial process (Figure 1).
On examination, she was lying in bed with eyes closed to prevent frank vertigo and upon opening was immediately found to have a vigorous spontaneous left-beating nystagmus that increased in amplitude on left gaze that did partially suppress in room light with visual fixation. Her facial nerve function was intact and symmetric in detail, yet her hearing was subjectively worse in the right than in the left ear.

The above clinical findings demonstrating clear violation/irritation of the right inner ear suggested bacterial labyrinthitis secondary to AOM. The patient was immediately given IV dexamethasone to prevent inner ear inflammatory damage and promethazine to assuage the acute central vestibular response. She was shortly thereafter taken to the operating theater for an examination of her ears under anesthesia with bilateral myringotomies and pressure equalization tube placement. Erythematous bulged tympanic membranes were incised, which liberated copious gross purulence from the middle ear space. The middle ear exudate was sent for aerobic and anaerobic culture, fungus, acid-fast bacillus, gram stain, culture, and sensitivities. Pressure equalization tubes were placed and the ears were copiously irrigated with normal saline followed by Ciprodex drops. The patient was readmitted and continued on promethazine for vestibular-based nausea and vomiting, and Unasyn as a broad-spectrum antibiotic. Several hours after the procedure the patient noticed a considerable improvement in otalgia and vertigo with remarkable reduction in the left-beating spontaneous nystagmus to the point where she could keep her eyes open without being violently vertiginous. During the next two days, the nausea and vertigo subsided and were only present with rapid head movements to the right. The ear canal and middle ear fluid cultures were positive for *Streptococcus pneumoniae* that was sensitive to Augmentin. Subjectively, she regained her hearing in the left ear with only mild auditory perception in the right ear and only complained of persistent bilateral tinnitus. She was discharged on postoperative day four with an oral prednisone taper, Augmentin, and Ciprodex drops. She was scheduled to follow-up for dedicated audiometric and videonystagmography (VNG) testing to determine the health of the inner ears. Subsequent (1 month later) audiometric testing revealed normal hearing on the left with a profound SNHL beyond the detections of the audiometer in the right ear. Vestibular testing revealed a 69% right weakness with bilateral caloric irrigations as compared to the left. Rotary chair testing was not available.

3. Discussion

Bacterial labyrinthitis following AOM results from extension of middle ear infection to the labyrinth (otogenic suppurative labyrinthitis). The infection typically penetrates the labyrinth and perilymphatic space through the bony fistula of the otic capsule or the oval or round windows. If it arises following meningitis, it typically involves bilateral inner ear structures (meningococcal bacterial labyrinthitis). In this report our patient with bilateral AOM presented with clinical signs of unilateral inner ear involvement suggesting an otogenic suppurative labyrinthitis.

The diagnosis of labyrinthitis is heralded by acute onset vertigo with associated nausea, vomiting, nystagmus, and either sudden or progressive hearing loss. Labyrinthitis may be classified as suppurative, serous/viral or chronic. In suppurative bacterial labyrinthitis, patients often present with otalgia, vertigo, nausea, erythematous or opaque tympanic membranes, and potentially purulent otorrhea if the tympanic membrane has ruptured. Retrospective reports have documented the prevalence of suppurative bacterial labyrinthitis following AOM to be between 10% and 28% [2,6,10]. Suppurative bacterial labyrinthitis can be differentiated from serous/viral labyrinthitis based on the presence of positive bacterial cultures from middle ear exudate [1,3]. Serous/viral labyrinthitis leaves mild inner ear dysfunction, while suppurative disease often results in severe irreversible auditory and vestibular hypofunction [3]. As a result, patients suffering from suppurative labyrinthitis often display spontaneous nystagmus consistent with unilateral vestibulopathy. Our patient demonstrated robust left-beating spontaneous nystagmus consistent with a right peripheral vestibulopathy. Her nystagmus was classified as third degree as it intensified in frequency with left lateral gaze in the direction of the fast phase of the nystagmus consistent with Alexander’s law but was also present on neutral and right lateral gaze [8].

In addition to vertigo, our patient noted right greater than left profound subjective hearing loss that is also consistent with suppurative labyrinthitis. This finding is typical as these patients present with profound SNHL seen with bedside Weber and Rinne testing and confirmed on formal laboratory audiologic assessment [1,5]. In the present report, our patient suffered a profound right SNHL and a 69% weakness on caloric irrigations in the right ear in the setting of *S. pneumoniae* positive cultures from middle ear exudate.

While suppurative labyrinthitis is a clinical diagnosis, many have advocated for the use of imaging. CT scans of the temporal bones often reveal middle ear and mastoid opacification with or without bone erosion or coalescence [1]. In our patient, temporal bone CT images showed similar opacification in the mastoid and middle ears bilaterally, yet she only incurred inner ear damage on the right (Figure 1). This supports the notion that bacterial labyrinthitis is a clinical diagnosis and may not be predicted or diagnosed based on CT imaging alone. In the acute phase, gadolinium-enhanced MRI images may reveal labyrinthine enhancement as consistent with labyrinthitis [4].
Treatment of bacterial labyrinthitis is both surgical and medical and timing is critical in order to minimize inner ear trauma or further complications. Surgical treatment includes myringotomy and pressure equalization tube placement that are both therapeutic and diagnostic [6]. Early myringotomy is essential for the evacuation of middle ear disease as well as to obtain culture and sensitivities to tailor antibiotic therapy to prevent irreversible damage and salvage remaining vestibular and auditory function [1,3,7]. The placement of pressure equalization tubes allows for continued liberation of middle ear exudate and provides a conduit for entry of topical antibiotics.

Medical treatment consists initially of broad-spectrum antibiotic treatment with coverage for typical bacterial pathogens [2]. Intravenous antibiotic therapy that is culture-specific is most ideal and may be more beneficial than oral therapy to obtain adequate therapeutic concentration in the inner ear, especially in the patient experiencing nausea and vomiting. Topical antibiotic drops may be administered through the tubes postoperatively for additional coverage. Intravenous corticosteroid treatment, such as dexamethasone, has been shown to decrease long-term SNHL [3,4,7]. Early administration of IV dexamethasone may also salvage auditory and vestibular function and prevent further irreversible damage [4,7]. During the acute phase of disease, patients with severe nausea and vomiting can be given IV fluids and vestibular suppressive medication such as meclizine, benzodiazepines, promethazine, or (in refractory cases) droperidol [9].

Follow-up visits should include an audiogram and formal vestibular testing to determine the extent of residual inner ear function. Cases of acute bacterial suppurative labyrinthitis frequently include persistent SNHL, especially in the higher frequencies [1,3,6]. To improve long-term auditory function, patients who have incurred permanent hearing loss may benefit from amplification; in our case of a profound unilateral hearing loss a contralateral routing of offside signals (CROS) hearing aid system or potentially a bone-anchored hearing aid (BAHA).

Following the acute phase of bacterial infection and labyrinthine inflammation, the symptoms of vertigo do decrease but disequilibrium and vertigo with sudden head movements to the effected side often persist. For patients with persistent vertigo following unilateral vestibulopathy, long-term vestibular suppressive medications are contraindicated as they delay central compensation mechanisms [9]. Rather these patients benefit from vestibular physical therapy/rehabilitation [9].

4. Conclusion

We report a case of acute suppurative bacterial labyrinthitis in a healthy 35-year-old female. Acute bacterial labyrinthitis is a rare but potentially devastating complication of AOM. Early diagnosis and treatment with corticosteroids and surgical decompression of the middle ear can potentially preserve vestibular and auditory function [3,4,7]. Close follow-up is crucial to evaluate residual inner ear deficits after the acute inflammatory response has resolved. Auditory rehabilitation and vestibular physical therapy are important to promote compensation for losses incurred [9].

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References