Streptococcus pyogenes otitis media with ipsilateral hearing loss facial paralysis complicated by acute mastoiditis and petrous apicitis

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Abstract

Streptococcus pyogenes *is a common Gram-positive coccus frequently encountered in clinical medicine as the causative agent of skin and soft tissue infections and pharyngotonsillitis. It is also a rare causative agent of acute otitis media, where it is more frequently associated with a complicated disease course when compared to typical causes of acute otitis media. We present a novel case of acute otitis media caused by* S. pyogenes *that was complicated by ipsilateral hearing loss, ipsilateral facial paralysis, acute mastoiditis, and petrous apicitis in a young adult woman with no significant past medical history.*

Keywords: Streptococcus pyogenes, acute otitis media

Introduction

Streptococcus pyogenes, also referred to as Lancefield Group A Streptococcus (GAS), is a Gram-positive coccus characterized by beta hemolysis when plated on blood agar. It is a common cause of a variety of human infections, including, but not limited to, pharyngitis and skin and soft tissue infections ranging from erysipelas and cellulitis to necrotizing fasciitis.[1] Despite being a ubiquitous cause of the infections mentioned above, it is rarely isolated as the causative agent of acute otitis media (AOM), accounting for only 2–3% of cases of AOM in children.[2] However, when *S. pyogenes* is the causative agent of AOM, it is more frequently associated with complications than more common pathogens, such as *Streptococcus pneumoniae* and *Haemophilus influenzae*.[3] For example, the risk of acute mastoiditis (AM), the most commonly encountered life-threatening complication of AOM, has been found to be highest when *S. pyogenes* is isolated as the causative agent.[4] Despite a decrease in the incidence of complications of AOM since the advent of antibiotics, many are still encountered in the clinical setting. These complications can range from those more commonly observed conditions, such as hearing loss and tympanic membrane perforation, to rarely encountered complications, including petrous apicitis, facial paralysis, meningitis, brain abscesses, and lateral sinus thrombosis.[5] We present a novel case of AOM caused by *S. pyogenes* that was complicated by ipsilateral hearing loss, ipsilateral facial paralysis, acute mastoiditis, and petrous apicitis in a young adult woman with no significant past medical history who was treated with intravenous vancomycin, myringotomy and tympanostomy tube placement, left cortical mastoidectomy, and left endoscopic tympanoplasty and atticotomy.

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Case Description

A 23-year-old woman with no significant past medical history and allergies to penicillin, with a reported reaction of generalized swelling and pruritis, and to trimethoprim-sulfamethoxazole, with a reported reaction of skin pruritus, presented to the emergency department (ED) in January of 2023 with two-week history of left ear pain, left purulent otorrhea, left-sided hearing loss, left-sided facial drooping, and fever.

Physical examination was significant for the absence of proptosis of the left ear, moderate to severe left ear pain, purulent drainage of the left external auditory canal, inability to visualize the left tympanic membrane due to fluid collection, and House-Brackmann (HB) grade IV left-sided facial paralysis, with near complete eye closure and diminished eyebrow raise and smile. The otoscopic examination of the right ear was within normal limits. The patient was afebrile and hemodynamically stable on presentation. In the ED, laboratory findings were notable only for mild anemia (hemoglobin, 11.0 g/dl). While in the ED, she received a single dose of 2 grams of ceftriaxone IV. Upon receiving IV ceftriaxone, the patient reported a sensation of numbness, tingling, and swelling in the back of her head, generalized pruritus, and a sensation of a "thick tongue". She denied any symptoms of airway compromise, however, was reluctant to receive any further beta-lactam antibiotics. In addition, a computed tomography (CT) scan obtained in the ED revealed left otomastoiditis with partial opacification of the left external auditory canal without dehiscence or erosion of the ossicles or canals, for which an otolaryngology (ENT) consultation was obtained. The patient was then admitted to the internal medicine service for intravenous (IV) antibiotic treatment with levofloxacin 750 mg q24 hours and vancomycin 1.25 g q8 hours for 48 hours, as well as treatment with ofloxacin ear drops, five drops TID in her left ear, with close monitoring. If she did not respond appropriately to conservative management, cortical mastoidectomy would be considered. A culture of her left ear was also obtained, which the day following her admission grew *Streptococcus pyogenes* that was reported to be sensitive to all tested antibiotics.

Despite 48 hours of IV antibiotic therapy with vancomycin, levofloxacin, and ofloxacin ear drops, the patient's symptoms were largely unchanged, with only a mild improvement in facial paralysis, from House-Brackmann (HB) grade IV to grade III. A repeat CT scan demonstrated continued opacification of the left mastoid air cells, external auditory canal, and middle ear space without evidence of a drainable coalescence. As a result, ENT performed an examination under anesthesia (EUA) of the patient's left ear with myringotomy and placement of a tympanostomy tube. Intravenous (IV) levofloxacin was also discontinued, while IV vancomycin was continued. The day following the procedure, the patient reported significant improvement in her left otalgia; however, her facial paralysis remained stable compared to pre-procedure. At this time, she was also started on 60 mg of oral prednisone for seven days, with a planned taper. Two days after the EUA and left tympanostomy tube placement, the patient's pain level increased significantly, despite initial improvement as stated above. Her facial paralysis also worsened to HB grade IV from HB grade III. A decision was made to continue monitoring the patient for clinical improvement for 96 hours, after which mastoidectomy would be considered if clinical improvement was not significant. IV vancomycin was discontinued, and IV levofloxacin 750 mg q24 hours was restarted. During the time of clinical monitoring, the patient's pain was unchanged, however, her facial paralysis improved slightly back to HB grade III; ENT also ordered magnetic resonance imaging (MRI) of the head with and without contrast to rule out any intracranial involvement. A repeat CT scan demonstrated continued evidence of mastoiditis. The MRI showed abnormal enhancement in the mastoid air cells in the left temporal bone extending to the petrous apex, concerning for petrous apicitis, as well as abnormal enhancement of the internal auditory canal secondary to dural enhancement, therefore, suspicious for meningitis. The patient was subsequently taken to the operating room for left cortical mastoidectomy with facial nerve monitoring, left temporalis fascia graft to canal wall defect, and left endoscopic tympanoplasty and atticotomy. During this procedure, intraoperative cultures of mastoid granulation tissue were taken.

After the procedure, her facial paresis improved to HB grade II. ENT began ciprofloxacin/dexamethasone ear drops, 4 drops BID to the left ear, for seven days. An Infectious Disease consultation was obtained for antibiotic management due to concern for meningitis, and recommended restarting IV vancomycin with dosing per pharmacy AUC/MIC ratio more than 400, as well as continuing IV levofloxacin 750 mg every 24 hours. On the following day, infectious disease made the recommendation to discontinue IV levofloxacin, and continue IV vancomycin for 6 weeks as an outpatient

through a peripherally inserted central catheter (PICC), with recommended vancomycin trough level between 15–20 mg/L. The patient was instructed to follow up in the infectious disease clinic in 4–5 weeks. She was discharged from the hospital two days later with significant improvement in her left-sided otalgia and HB grade II left facial paralysis.

At her hospital follow-up with ENT six days after discharge, the patient reported continued left otalgia, rated 6/10 in severity, with continued left otorrhea. She reported that her left-sided facial paralysis had significantly improved after her operation, with objective physical examination findings demonstrating HB grade I–II facial paralysis. Four weeks after discharge, she presented for her hospital follow-up with the infectious disease team, where she stated that she had still not regained her hearing in her left ear, despite continued improvement in her facial paralysis and pain. In addition, despite receiving approximately five weeks of IV vancomycin through her PICC, her vancomycin levels had been largely non-therapeutic. For this reason, it was decided to discontinue IV vancomycin and the PIC, and start clindamycin 600 mg PO q8 hours for at least four weeks.

Finally, at her ENT follow-up seven weeks after discharge, she was noted to have only minimal, if any, residual facial paralysis and an audiogram was scheduled to be done in three months. However, at this time, she was lost to follow-up in both the ENT and infectious disease clinics.

Discussion

Acute otitis media refers to the acute onset of middle ear inflammation and is one of the most commonly encountered diagnoses in the pediatric population, with approximately 70% of children experiencing at least one episode by the age of two.[6] However, the incidence of AOM is negatively correlated with age, with ages 35 to 44 representing only 1% of the incidence of global cases.[3] Before the use of antibiotics in the treatment of AOM, it is estimated that complications occurred in up to 6% of cases of AOM, but with antibiotics, this has declined to up to 1%, with one retrospective analysis observing complications in only 0.26% of cases. Of this 0.26%, AM was responsible for 0.16% of complications, facial paresis was responsible for 0.03% of complications, and petrous apicitis represented only 0.002% of complications. Interestingly, in this small group of patients that had complications, 75.9% were adults, leading the authors to hypothesize that increased rates of comorbidities in the adult population may have a role in the increased incidence of complications.[5] This is the first case to our knowledge of AOM that was simultaneously complicated by AM, facial paralysis, and petrous apicitis.

Acute mastoiditis is a potentially life-threatening infection of the mastoid air cells of the temporal bone and is the most common complication of AOM.[7] From the mastoid air cells, infection can spread both hematogenously and contiguously, resulting in multiple possible complications, including cerebral venous sinus thrombosis, skull base osteomyelitis, and petrous apicitis.[8,9] Petrous apicitis, also known simply as petrositis, rare, but severe, complication of AOM and AM that refers to the infection of the petrous apex of the temporal bone. In the case of associated AM, it occurs due to the contiguous spread of infection from the mastoid air cells into the petrous apex.[9] Petrous apicitis was originally described by Gradenigo in 1904, as a clinical triad of otorrhea, retro-orbital or deep-rooted facial pain, and abducens nerve palsy. When all three components of this triad are present, the presentation is referred to as Gradenigo syndrome.[9,11] However, since the advent of antibiotic therapy and advances in cranial imaging techniques, this clinical triad is rarely seen in its entirety.[10] Our patient, for example, presented with only one component of the triad, otorrhea, and notably lacked retro-orbital pain and abducens nerve palsy.

Facial paralysis results from damage to cranial nerve seven (VII), also known as the facial nerve. As the facial nerve innervates the muscles of facial expression, injury results in an inability to use these muscles, with the exact distribution depending on whether the lesion is an upper motor neuron (UMN) or lower motor neuron (LMN) injury. Differentiating between UMN and LMN facial nerve palsies is critical, as their etiologies differ significantly. Facial nerve palsy can result from a variety of etiologies, including

idiopathic, neoplastic, trauma, ischemic, and infection. When evaluating facial paralysis, the degree of severity is determined using the House-Brackmann grading system.[12]

As mentioned above, facial paralysis is a rare complication of AOM, especially in the antibiotic era.[5] Although the exact mechanism of facial paralysis in AOM has yet to be elucidated, there are several hypotheses; these include retrograde infection of the facial nerve, bacterial toxin-induced peripheral demyelination of the facial nerve, and inflammatory edema-mediated compression of the facial nerve.[13,14] As facial paralysis is a rare complication of AOM, there is currently no consensus regarding its management. Current treatment is focused on eliminating infection of the middle ear and other associated structures with antibiotic therapy and surgical intervention when indicated. Despite this, typically, AOM-associated facial nerve palsy has a good prognosis, as observed in our patient.[14]

Hearing loss is a known complication of AOM in both children and adults, especially in repetitive cases. Initially, hearing loss tends to be conductive or mixed in nature, secondary to inflammation and effusion in the middle ear; however, the conductive portion of this hearing loss has been shown to resolve within weeks of presentation. Persistent sensorineural hearing loss (SNHL) has been shown to occur after even a single case of AOM, with the predominant frequencies affected being the extended high frequencies of 8 to 16 kHz.[15] Although our patient showed significant improvement in pain and facial paralysis, before she was lost to follow-up, she stated that there was no improvement in her left-sided hearing loss. Although it is not possible to tell if her hearing loss has improved since then, it is possible that the hearing loss could be permanent.

Conclusion

Acute otitis media is a very common infection in the pediatric population, with the most common causative agents being *S. pneumoniae* and *H. influenzae*, *whereas S. pyogenes* being a significantly less common cause of AOM. In addition, the incidence of AOM declines with age, with the overwhelming majority of cases occurring in children. In the modern era of antibiotics, complications of AOM are much less common than they were before widespread antibiotic use, however, they still occur, with AM representing the most common complication. This case report represents, to our knowledge, the first description of a previously healthy adult woman who presented with AOM caused by *S. pyogenes*, complicated by ipsilateral hearing loss, AM, petrous apicitis, and facial nerve paralysis. It serves to add to the extensive body of literature about AOM and its complications.

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