CASE REPORT
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Recurrent Facial Nerve Palsy After Acute Otitis Media in a 20-Month-Old: A Case Report

Karlien Van den Bossche[1], Gerd Claes[2]

[1]University of Antwerp, Translational neurosciences, Antwerp, Belgium
[2]AZ Sint Maarten, Mechelen, Belgium

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ABSTRACT

Facial nerve palsy in children is a distressing condition with potential long-term functional and psychosocial consequences, making early identification of the underlying cause and timely intervention crucial. Acute otitis media (AOM)-associated facial palsy is the second most common cause after idiopathic origins. The precise pathophysiology of AOM-associated facial palsy remains unclear, and no universally accepted treatment guidelines currently exist for managing such cases. Repeated episodes of facial nerve palsy following AOM are seldom documented. We present a rare case of a 20-month-old girl with no prior medical history with recurrent episodes of facial nerve palsy associated with AOM. This case contributes to the limited literature on recurrent facial palsy linked to AOM in a pediatric cohort, emphasizing the need for universally accepted treatment guidelines and vigilance for recurrence with importance of imaging to identify anatomical alterations and surgical drainage in refractory cases.

Keywords: Acute otitis media, case report, facial nerve palsy, pediatrics

Introduction

Facial nerve palsy in children is a distressing condition characterized by a sudden onset of unilateral facial muscle weakness. It can present with impaired expressions, altered taste perception, and reduced corneal sensitivity.[1] Evaluation of facial nerve function is commonly standardized using the House–Brackmann grading system, ranging from grade I (normal function) to grade VI (complete paralysis) (Figure 1).[2] Though relatively rare, facial nerve palsy is unexpectedly frequent among pediatric patients with an incidence between 5 and 21 per 100 000 children annually.[1,3] Given its potential for functional and psychosocial consequences, early identification of the underlying cause and timely intervention are crucial.

Acute otitis media (AOM) affects nearly 80% of children before they enter school.[4] While complications such as mastoiditis, meningitis, intracranial abscesses, and facial nerve palsy have become rare due to widespread antibiotic use, these adverse events still present diagnostic and therapeutic challenges when they do occur.[5] The precise pathophysiology of AOM-associated facial palsy remains unclear, and no universally accepted treatment guidelines currently exist for managing such cases.

Repeated episodes of facial nerve palsy following AOM are seldom documented. Here, we present a rare case of a 20-month-old girl with no prior medical history with recurrent episodes of AOM-associated facial nerve palsy.

Case Presentation

A 20-month-old girl presented in our hospital with acute left-sided facial nerve paralysis and left-sided AOM, including high fever and severe otalgia (Figure 2 A). Otoscopic examination demonstrated a diffusely erythematous, opaque, and bulging tympanic membrane, consistent with AOM. Facial nerve dysfunction was graded IV in the upper facial branches and grade V in the lower branches using the House–Brackmann scale.

One day prior, her general practitioner had diagnosed bilateral AOM and initiated treatment with oral amoxicillin, according to current AOM guidelines.[6] Initial investigations ruled out meningitis. Blood tests revealed elevated C-reactive protein levels and leukocytosis (25 000/μL). Molecular testing on a

Corresponding author: Karlien Van den Bossche, e-mail: Karlien.vandenbossche@student.uantwerpen.be
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Grade	Description	Characteristics
I	Normal	Normal facial function
II	Mild dysfunction	Slight facial weakness with complete eye closure with minimal effort and slight asymmetry of mouth movement
III	Moderate dysfunction	Noticeable weakness/asymmetry with possible eye closure with effort
IV	Moderately severe dysfunction	Obvious weakness/asymmetry also at rest and incomplete eye closure
V	Severe dysfunction	Barely perceptible motion, mouth movement, and eyelid movement
VI	Total paralysis	No movement

Figure 1. House–Brackmann scale.

nasopharyngeal sample revealed co-infection of adenovirus and rhinovirus. Serological tests for Epstein–Barr virus and Borrelia were negative. The child had received the recommended pneumococcal conjugate vaccination (PCV13) according to the Belgian vaccination schedule, consisting of three primary doses at 8, 16, and 24 weeks of age, followed by a booster dose at 12 months.

Due to the severity of the condition, intravenous ceftriaxone and glucocorticoids were initiated.

After 24 hours, insertion of an intermediate-lasting grommet was performed in the left ear.

Despite intervention, there was no notable improvement after 2 days. A CT scan of the mastoid with a slice thickness of 0.6 mm showed no signs of bony erosions, no apparent dehiscence of the tympanal segment of the bony facial canal (within limits of the image resolution), and no evidence of abscess formation (Figure 3). Given persistent symptoms, a mastoidectomy was performed to decompress the mastoid and middle ear. No decompression of the facial nerve itself was performed, since the paralysis was not complete. Histological examination of granulation tissue confirmed necrotizing inflammation. Postoperatively, the patients' facial function began to improve slowly. By day 2, upper branches had improved to grade III and lower branches followed with improvement to grade III by day 6.

Glucocorticoid treatment was discontinued on day 6, and she was discharged with oral antibiotics.

However, 2 weeks after surgery, her case became unusual.

Main Points

- Acute otitis media–associated facial palsy is the second most common cause after idiopathic origins.
- Long-term follow-up is essential due to the risk of recurrence.
- More research is needed to identify predictors of recurrence.

Facial weakness (grade IV—upper branches and grade V—lower branches) recurred during a new episode of AOM. Otoscopic examination revealed a blocked grommet and serous otitis media. At 1 month postoperatively, a new grommet was inserted, and facial function improved to grade II within a week.

Twenty-eight months after the initial event, the child remains under ENT follow-up every 4 months.

Over this period, she has experienced more than 10 recurrences of facial palsy, documented by her mother. All have a similar pattern, occurring during an upper airway infection with a severity of grade III to IV and resolving spontaneously within 3-7 days (Figure 2 B). Fortunately, the grommet stayed functional for 2 years to maintain middle ear ventilation, protecting the patient from more severe palsy recurrences.

Recent examinations showed extrusion of the grommet and a millimetric tympanic membrane perforation. Although episodes of facial palsy still occur, their frequency and severity have become less.

An informed written consent was obtained from the parents of the patient prior to writing this paper, with an emphasis on keeping all personal information anonymous.

Discussion

This case illustrates several important issues regarding pediatric facial nerve palsy and its association with AOM. Facial palsy in children, while often idiopathic (Bell's palsy), may also be linked to infectious, traumatic, neoplastic, or congenital etiologies.[7-9] Infections, especially otitis media, constitute the second most common cause after idiopathic origins, occurring in approximately 12% of pediatric cases.[7-9]

Several pathogenic mechanisms for the occurrence of facial palsy associated with AOM were described:

First, through direct nerve involvement by a bony dehiscence or neurovascular channels connecting to the middle ear. Second, inflammatory edema causing compression and secondary thrombosis of the microvasculature of the facial nerve, leading

Figure 2. Left-sided facial nerve palsy. (A) First admission: grade IV upper facial branches and grade V lower branches. (B) After recurrence: grade III upper facial branches and grade IV lower branches.

to ischemia and infarction. Last, demyelination of the facial nerve induced by bacterial toxins or reactivation of dormant neurotropic viruses.[10,11] However, in this case, no signs of bony erosions or dehiscence of the bony facial canal were observed.

While most cases are isolated, recurrence is not unheard of, with documentation in up to 10% of cases in children with idiopathic Bell's palsy.[8,12] In a retrospective study of Karalok et al,[7] recurrence time between attacks ranged from 6 months to 6 years, emphasizing the necessity of long-term follow-up of these pediatric patients. A notable differential diagnosis is Melkersson–Rosenthal syndrome, a rare genetic disorder characterized by a triad of recurrent facial palsy, orofacial edema, and lingua plicata.[13] Throughout clinical follow-up, the patient demonstrated no evidence of this triad or family history suggestive of Melkersson–Rosenthal syndrome. Furthermore, each facial palsy episode occurred in close temporal association with clinically documented AOM,

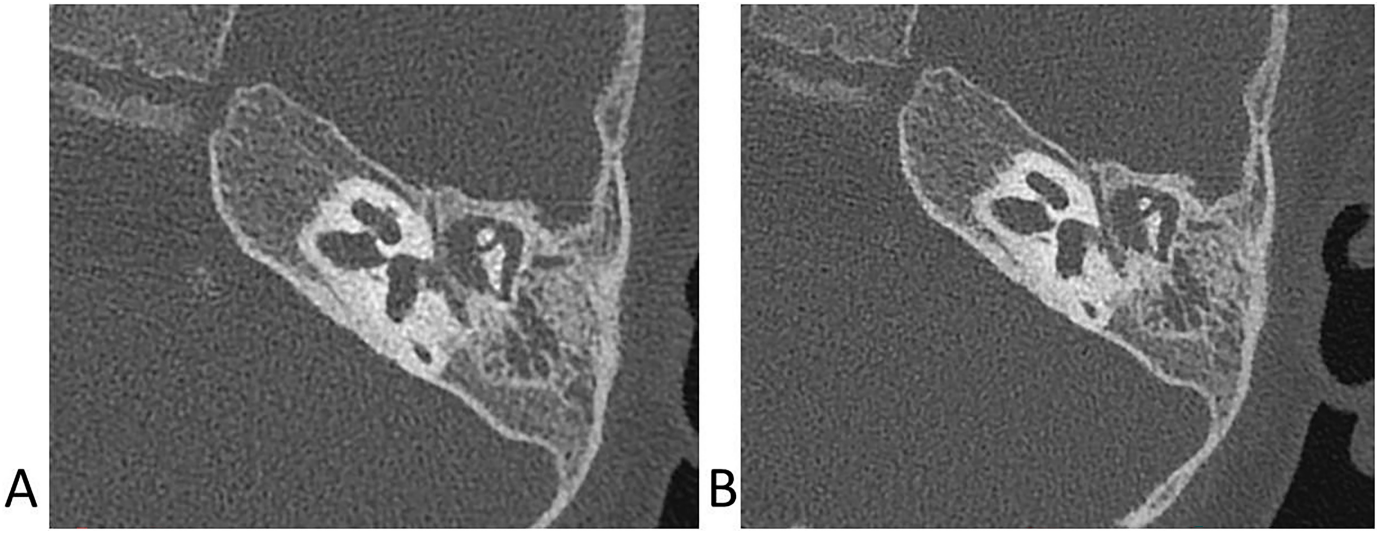

Figure 3. Computed tompgraphy scan of left-sided mastoid bone showing no signs of bony erosions, no evidence of abscess formation, and no dehiscence of the bony facial canal.

supporting an infectious trigger rather than idiopathic recurrent Bell's palsy. Although Melkersson–Rosenthal syndrome cannot be excluded with absolute certainty in a young child before the full clinical triad develops, the available clinical findings make this diagnosis unlikely. Another rare association includes fibrous dysplasia, which can distort the bony facial canal.[14] Özkale et al[9] observed that younger patients generally exhibited less favorable outcomes compared to older patients irrespective of disease etiology. However, recurrent facial nerve palsy triggered by upper airway infections, as seen in this case, has not yet been documented in the literature to our knowledge. As a limitation, no additional CT or MRI examinations were performed following the recurrent episodes. Given the close temporal association of each recurrence with a clinically confirmed episode of AOM, the rapid and complete recovery with conservative treatment, and the absence of atypical neurological features or clinical progression, additional imaging was not considered clinically indicated at the time. Consequently, the absence of follow-up imaging limits our ability to draw definitive conclusions regarding the underlying anatomical or structural mechanisms.

The causative mechanism in our case remains speculative and is based on previously published literature rather than direct evidence from our patient. We hypothesize that it may involve a combination of anatomical vulnerability, micro-dehiscences of the bony canal, viral triggers, and bacterial superinfection affecting a facial nerve still recovering from the initial palsy.

Another important issue we encountered is that there is so far no established consensus on the treatment of AOM-related facial palsy. Despite their widespread use, the role of corticosteroids remains controversial; however, their anti-inflammatory and neuroprotective properties may justify consideration.[7,15] Multiple studies indicate that conservative management with intravenous broad-spectrum antibiotics, typically third-generation cephalosporins, is often sufficient to achieve complete recovery in most patients and is generally recommended as the first-line treatment.[11,16] If no clinical improvement is observed within the first 48 hours, myringotomy with grommet placement should be considered according to a recent systematic review, as it may play a role in relieving the facial nerve from inflammation and pressure.[11] Moreover, this review stated that performance of a mastoidectomy is indicated only for cases unresponsive to medical therapy or those complicated by acute mastoiditis.[11] Some authors oppose surgical decompression of the facial nerve due to the absence of large-scale clinical trials and the potential risk of sensorineural hearing loss.[3,16] Conversely, others advocate for electrophysiological assessment and recommend decompression when axonal function loss exceeds 95%.[11] In this case, surgical decompression of the facial nerve was not indicated as there was an incomplete palsy. Even without electrophysiological assessment, we could assume that axonal function loss would not exceed 95%, so the risks of a decompression outweighed the possible rewards.

Electrophysiological testing was not performed, which could have provided additional objective information on axonal function loss and recovery; however, it was not considered clinically indicated given the complete recovery after each episode.

Conclusion

This case contributes to the limited literature on recurrent facial palsy linked to AOM in a pediatric cohort, emphasizing the need for universally accepted treatment guidelines and vigilance for recurrence with importance of imaging to identify anatomical alterations and surgical drainage in refractory cases.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no artificial intelligence tool was used in the preparation of the manuscript.

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

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