Intratemporal Complications From Acute Otitis Media in Children: 17 Cases in two Years

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Abstract

Background: To describe the clinical features of intratemporal complications of acute otitis media (AOM) in children, a retrospective study was carried out for 2 years.

Methods: We considered acute mastoiditis, presence of postauricular swelling, erythema and tenderness, and anteroinferior displacement of the auricle; labyrinthitis, presence of vestibular symptoms (spontaneous nystagmus and ataxia with or without vomiting; and facial nerve palsy, absence or decreased motility in hemiface.

All children underwent otomicroscopy for evidence of coexistent or recent AOM. Cultures were obtained by tympanocentesis and myringotomy, drainage of subperiosteal abscess, or from granulation tissue during tympanomastoidectomy.

Results: From January 2008 to December 2009, 17 patients fulfilled the entry criteria. Median age: 54.2 months. Of the 17 cases, 30% were infants younger than 12 months and most were boys (70.6%).

8/17 were receiving antimicrobial treatment. Diagnoses included 9/17 (52.9%) acute mastoiditis, 7/17 (41.2%) labyrinthitis, and 1/17 (5.9%) facial nerve palsy. Intracranial complications were present in 17.6%. All required intravenous antimicrobial treatment. Myringotomy was performed in 16/17, drainage of subperiosteal abscess in 3/17, and tympanomastoidectomy in 2/17. Bacteriology was positive in 9/17 cases, isolating Streptococcus pyogenes (S. pyogenes) in 44% of patients. Secuelas: One unilateral sensorineural hearing loss.

Conclusions: Acute mastoiditis is the most common complication. Labyrinthitis was diagnosed in 41.2% of cases. S. pyogenes was prevalent in these serious invasive infections in our area. Associated intracranial complications were present in 17.6% cases. Resolution of AOM complications required surgical procedures in all cases (myringotomy, drainage of subperiosteal abscess, or tympanomastoidectomy).

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Introduction

Acute otitis media (AOM) can be divided into 5 clinical stages directly correlated with their clinical symptoms and overlapping with one another:

**Tubotympanicitis phase**; causes discomfort, aural fullness, retracted tympanic membrane, and loss of luminous reflex. An initial serous effusion can be observed.

**Hyperaemic phase**; appears with otalgia, general discomfort, and fever up to 39 °C, as well as congestive membrane and opacity of the eardrum.

**Exudative phase**; presents intense otalgia, which may disrupt sleep. There is nausea, vomiting, and anorexia; myalgia, arthralgia, and diarrhoea at times. Fever over 39 °C. Marked hyperaemia of the membrane with loss of anatomical landmarks.

**Suppurative phase**; accompanied by fever higher than or equal to 40 °C, as well as intense and throbbing otalgia. Bulging tense membrane, with pronounced peripheral hyperaemia and sometimes yellow areas denoting necrosis. There may be spontaneous perforation of the membrane and otorrhoea of more than 2 weeks’ duration.

**Acute mastoiditis phase**; this represents a complication of AOM by infectious, inflammatory extension to mastoid cells.¹ The complications of AOM remain a cause of hospitalisation in children, despite the fact that their incidence went from frequent and usually fatal to rare and with low morbidity and mortality rates since the advent of antibiotics. In Argentina there is a high number of unreported cases of complications due to otitis media.² These complications are traditionally divided into intratemporal and intracraniel, with the former being the most frequent and the main focus of this work.

Intratemporal complications of AOM include labyrinthitis, peripheral facial paralysis (PFP), acute mastoiditis (AM), and petrositis and Gradeningo’s syndrome.

Labyrinthitis is due to the spread of the infectious process or its toxins to the inner ear. It is divided according to the levels of severity into perilabyrinthitis, serous labyrinthitis, and suppurative labyrinthitis. The third type presents cochlear symptoms with rapidly progressive sensorineural hearing loss, tinnitus of intense tones, and aural fullness. It also causes severe vertigo, neurovegetative symptoms, and nystagmus that is initially irritative (towards the affected side) and subsequently becomes ablative (contralateral). The severe vertigo is usually an alert sign. Cases of suppurative labyrinthitis require conducting extensive cortical mastoidectomy and intravenous antibiotic therapy.¹

The PFP appears when the tympanic portion of the facial nerve is dehiscent (50% of the general population) and comes into contact with the infectious material of the tympanic cavity, producing neuritis.¹ In such cases, tympanocentesis and myringotomy with or without placement of ventilation tubes are also required in addition to intravenous antibiotic treatment. Steroids are employed as well. If not treated promptly within the space of 3 days, cortical
mastoidectomy with debridement around the fallopian canal may be required.\(^3\)

Acute mastoiditis is characterised by infectious, inflammatory extension towards mastoid air cells, with accumulation of purulent discharge, osteitis, and destruction of bone trabeculae forming a common cavity. Anatomically, it may extend in 6 different directions: lateral (towards the soft tissues of the outer ear); anterior (towards the external auditory canal); posterior (towards the sigmoid sinus or the posterior cranial fossa causing lateral sinus thrombosis); medial (towards the labyrinth or the petrous apex causing labyrinthitis, PFP or petrositis); superior (towards the middle cranial fossa causing an epidural abscess); and inferomedial (towards the mastoid tip causing a Bezold abscess).\(^4\) Subperiosteal and Bezold abscesses are secondary to subperiosteal extension of infection from mastoiditis. Subperiosteal abscesses present anterior displacement of the auricular pavilion, oedema, and hyperaemia with pain in the mastoid region and in the posterior quadrants of the external auditory canal. Bezold abscesses consist of the extension of the infection to the medial subperiosteal space of the mastoid process, presenting spread to the neck.

Petrositis and Gradenigo’s syndrome are the extension of the infectious process into the cells of the petrous portion of the temporal bone, leading to symptoms known as Gradenigo’s triad: otitis media, paralysis of the sixth cranial nerve, and neuralgia of the fifth cranial nerve, characterised by intense, hemifacial, retro-orbital pain. These can be complicated by intracranial extension.\(^1\)

Lateral sinus thrombosis may be an intracranial complication of AOM or mastoiditis. The proposed pathogenesis is a spread of infection from the small veins of the mastoid bone to the sigmoid sinus, with subsequent spread of inflammation through the eroded or coalescing bone, causing a perisinusal abscess. A thrombus occurs from adhesion of fibrin, blood cells, and platelets with subsequent obliteration of the lateral sinus. Clinically, the most common manifestation is the presence of fever, although otorrhoea, retroauricular oedema, otalgia, headache, nausea, vomiting, and meningeal signs are also possible. It may be associated with paralysis of the sixth and seventh cranial nerves, and may spread into the jugular bulb or occasionally to the dural sinus, giving rise to neurological symptoms.\(^5\)

**Material and Methods**

We conducted a review of the literature and clinical histories of 17 patients with intratemporal complications of AOM seen at the ENT service of a paediatric hospital for 2 years.

All patients underwent diagnostic otomicroscopy for the diagnosis of acute coexisting otological disease. Cases with presumption of cholesterolotomatus chronic otitis media (C-COM) were differentiated according to background and tympanic images, and were also excluded. Material was taken for culture by tympanocentesis or culture of mastoid subperiosteal abscess by puncture prior to surgical drainage. Purulent and granulomatous material from the mastoid cavity was obtained intraoperatively when antromastoidectomy was indicated.

The diagnostic criteria for patients with intratemporal complications of AOM, for each type of complication, were as follows:

- **Mastoiditis**: presence of erythema and retroauricular oedema, protrusion of the auricular pavilion, drop of the posterior wall of the external auditory canal. In imaging studies by computed tomography (CT): occupation of mastoid cells with osteitis and destruction of bone trabeculae.
- **Labyrinthitis**: presence of vertigo with or without nausea and vomiting, nystagmus, refusal to walk due to gait instability.
- **Peripheral facial palsy**: presence of immobility or decreased movement in 1 side of the face.

Suspicion of intracranial complications led to the request for studies by the usual imaging techniques, including CT with axial and coronal sections, with bone window of both temporal bones, and magnetic resonance imaging (MRI) or MRI angiography (MRT-A).

**Results**

A total of 25 patients were attended due to complications of otitis media between January 2008 and December 2009. We excluded 8/25 cases because they were children suffering C-COM with intratemporal and intracranial complications.

We selected 17 patients with intratemporal AOM complications.

The mean age of patients was 54.23 months (range between 1 and 152 months). Of these, 30% (5/17) were younger than 12 months. The gender distribution showed a predominance of males (12/17 patients) over females (5/17).

A total of 47% (8/17) were receiving antibiotic therapy for AOM at the time of diagnosis of the complication, 3/8 of which were parenteral.

ENT diagnoses included 9/17 (52.9%) AM, 7/17 (41.2%) labyrinthitis, and 1/17 (5.9%) PFP. Up to 17.6% (3/17) presented intracranial complications (2 sigmoid sinus thromboses and 1 meningitis).

The diagnosis of AOM with retention of purulent exudate was performed otomicroscopically in 13/17 patients, of whom 3 were bilateral, 7 on the left side, and 4 on the right. Moreover, 4/17 presented suppurative AOM and spontaneous perforation of the insufficient tympanic membrane.

All cases required surgical procedures and antibiotic therapy. Myringotomy was performed in 16/17 (94.1%), drainage of subperiosteal abscess in 3/17 (17.6%), and antromastoidectomy in 2/17 (11.8%).

Samples were taken for microbiological study in all procedures. Bacteriology was positive in 9/17 (54%), with S. pyogenes being isolated in 44% of cases. Two cases developed Haemophilus influenzae (H. influenzae). A further 47% (8/17) tested negative, 5 of which were under antibiotic treatment (2 with amoxicillin, 1 with amoxicillin and clavulanic acid, 1 with cefadizime, and 1 with ceftriaxone). Anaerobe germs were found in 11.8% (2/17) of cases. In total, 3 different germs were cultured, 1 of them being Fusobacterium necrophorum (F. necrophorum).
Clinically, only 47% (8/17) consulted due to fever. Of the 9 patients with AM, 3 presented fever, 1 had headache, 3 suffered otalgia, and 2 had lateral sinus thrombosis as intracranial complications.

One patient presented otalgia and suppuration of 2 weeks’ evolution, which progressed with mastoid phlogosis, fever, and stiff neck. The CT scan of both temporal bones showed veiling of the left cavity and mastoid with coalescence, bone erosion at the level of the sigmoid sinus, and thickening of retroauricular soft tissues. Sigmoid sinus thrombosis was suspected, leading to the performance of an MRI-A, which revealed sinus hypoplasia without thrombosis.

The 6 patients with labyrinthitis presented vertigo, 4/6 had nausea, 4/6 febrile syndrome, and 2/6 headache. One 7-month-old patient presented labyrinthitis as a complication of otogenic meningitis, which was manifested by horizontal nystagmus and retropulsion when attempting to sit him down. The symptoms improved after 24 h of myringotomy being performed along with intravenous treatment with ceftriaxone.

One patient consulted due to left PFP. Interconsultation with the ENT service revealed that the patient was receiving intravenous ceftriaxone. The diagnosis was AOM with bilateral retention of purulent exudate and fever. A bilateral myringotomy was performed, which resolved the PFP in 24 h.

All patients presented a good clinical evolution after undergoing myringotomy or retroaural drainage together with antibiotic treatment, which was intravenous in 14/17 (with third-generation cephalosporins in 13 cases).

The initial evolution was torpid in only 3 patients, in 2 patients with labyrinthitis with development of S. pyogenes, and 1 patient with AM who developed H. influenzae, S. viridans/prevotella, and F. necrophorum.

There were no complications due to the procedures performed. The only sequel was 1 case of unilateral sensorineural hearing loss by labyrinthitis.

Discussion

It is important to remember that an entrapment of purulent secretions occurs in the middle ear in cases of anatomical or functional obstruction of the Eustachian tube and aditus ad antrum from mucoperiosteal oedema. Antibiotic therapy is not sufficient and the suppurative collection must be drained by myringotomy or tympanocentesis to avoid complications, especially in infants and young children.

In our study, the prevalence of AOM complications differed significantly in relation to the literature. Our series showed: 9/17 (52.9%) AM, 7/17 (41.2%) labyrinthitis, and 1/17 (5.9%) PFP. In addition, 17.6% (3/17) also presented endocranial complications (2 cases of sigmoid sinus thrombosis and 1 case of meningitis).

Labyrinthitis was diagnosed in 41.2% of cases. Consequently, it occupied a prominent position not described in the literature reviewed. Luntz et al. described labyrinthitis in 0.45% of their population; Suarez Castaño et al. in 3.6% (1/28), and Tarantino et al. described vomiting in 15% (6/40) children with mastoiditis.

In our study, 57.1% (4/7) of children with labyrinthitis presented nausea and/or vomiting. In febrile cases, the labyrinthine signs and symptoms simulate a meningeal syndrome; in afebrile cases, it may go unnoticed, especially in children who are still too young to walk. The prognosis with antibiotic therapy and myringotomy is generally favourable, with the possibility that many become cured by the treatment for AOM before a diagnosis of the complications is completed.

Up to 70.6% (12/17) of children with AOM complications improved and became cured after tympanocentesis and myringotomy (minor surgical procedure), as described by other authors (Ghaffar et al. and Spratley et al. described improvement in 60% of their cases; Bahadori et al. only in 40%).

Retroaural subperiosteal mastoid abscess drainage was performed in only 17.6% (3/17) of cases, and 2/17 (11.8%) required antromastoidectomy for the surgical toilett of granulomas in mastoid cells, antrum, and attic.

S. pyogenes was isolated in 44% of cases and thus emerged as the most common pathogen within our environment, unlike in other series (Harley et al. found it in 2%; Segal et al. in 3.1%; Spratley et al. in 7%; Ghaffar et al. in 8%; Luntz et al. in 9.2%; Butbul-Aviel et al. in 15%; Bahadori et al. in 17.6%, and Niv et al. in 23%). Segal et al. reported an increased risk of AM in patients with AOM due to S. pyogenes.

S. pneumoniae is the main aetiological agent described in AOM and invasive infections in paediatrics. However, it was only isolated in 11.8% of cases in our series.

In no case was P. aeruginosa isolated as a pathogen in AM as a complication of AOM, as was the case in the series published by Butbul-Aviel et al. (25%) and Nussinovitch et al. (34%). On the other hand, we agree with the study by Van Den Aardweg et al. In their review of 65 articles on AM, these researchers concluded that some authors mistakenly included children with AM from cholesteatoma in their casuistry, despite this entity having very different clinical and bacteriological characteristics from those of AOM complications.

A special mention should be made about the presence of anaerobes, which were found in 11.8% (2/17) of cases. Up to 3 different germs were cultured, 1 of them being F. necrophorum, a microorganism that causes necrotic diseases such as Lemierre’s syndrome, described as a septic thrombophlebitis of the internal jugular vein secondary to mastoiditis. It is important to note that the results from the microbiology laboratory are usually positive for anaerobic bacteria 7–15 days after processing the samples.

Intracranial complications appeared in 17.6% of patients with AM. This percentage was much higher than those reported in other series, such as those by Luntz et al. (8.5%), Harley et al. (1.7%), Ghaffar et al. (3.5%) and Spratley et al. (11.6%).

Regarding the frequency of AOM complications in our environment, these amounted to 8.5 cases per year, an incidence similar to that described in schools in other countries (Israel and Portugal). In developed countries, the prevalence in different studies is between 2 and 5 cases per year (the U.S.A., Australia, Italy and Spain). Undoubtedly, socioeconomic-cultural variables and limited access to primary health centres play a fundamental role in the high prevalence in our population.
Conclusions

Acute mastoiditis is the most common complication of AOM. The predominant signs and symptoms are: presence of erythema and retroauricular oedema, bulging of the auricular pavilion, and drop of the posterior wall of the external auditory canal.

Labyrinthitis was diagnosed in 41.2% of cases in our series, thus occupying an important place. Its diagnosis depends on a high degree of suspicion.

The absence of fever should not rule out otological disease. In our study, only 47% of cases presented fever when the AOM complication was diagnosed.

*S. pyogenes* was isolated in 44% of cases, emerging as the most common pathogen in our environment. Until a specific bacterial aetiology is obtained, the initial antibiotic treatment should cover both aerobic and anaerobic microorganisms. The latter, isolated in 11.8% of cases, could hinder the evolution of complications unless specific antibiotic treatment is carried out from the onset.

Intracranial complications should be suspected, given that 17.6% of the cases in our series presented them. Sigmoid sinus thrombosis and meningitis were the most frequent.

The resolution of AOM complications requires surgical procedures (minor or major) and parenteral antibiotic therapy in all cases.

Conflict of Interest

The authors have no conflicts of interest to declare.

References