INTRODUCTION

Acute otitis media is one of the most common infections in children. Usually a benign self-limited disease, it may however progress to potentially life-threatening conditions. Clinicians must be aware of the natural course and complications of this frequent disease.

The complications of otitis media can be divided into intratemporal and extratemporal (intracranial). Among the intratemporal complications, acute mastoiditis accounts for the majority of cases. Palva et al. reported an annual acute mastoiditis incidence of 0.3/100000 in a study of a million population in Finland [1].

Mastoiditis is characterized by a granulomatous and inflammatory process of the mastoid bone with disruption of its cellular system and demineralization by osteoclastic activity. One should suspect mastoiditis if, after a period of quiescence of about two weeks following an acute otitis media, the child has recurrent ear discharge and fever. If the underlying bony partitions within the mastoid are eroded and resorption of the bony septa occurs, then coalescent mastoiditis ensues. This is thought to be an uncommon progression of acute otitis media.

When the infection surpasses the mastoid cortex, a subperiosteal abscess can result. It may present, most commonly, as a postauricular fluctuance or abscess, or as a zygomatic abscess, or as a Bezold’s abscess, formed by erosion of the tip of the mastoid with extension, down to the posterior triangle of the neck. The treatment of these abscesses is cortical mastoidectomy and drainage, with the latter probably necessitating a neck exploration [5-6].

Mastoiditis can extend medially to the petrous apex of the temporal bone, resulting in petrositis. Petrositis is an uncommon and late complication of purulent otitis media. When the inflammation extends into Dorello canal, which transmits CN VI and the Gasserian ganglion (CN V), then Gradenigo syndrome develops. It is characterized by a triad of symptoms: lateral rectus (CN VI) palsy, retro-orbital pain (in the distribution of CN V), and otorrhea. The treatment of choice includes intensive antimicrobial therapy and mastoidectomy, with opening of the involved cell tracts leading to the petrous apex [11, 16-17].

Other intratemporal complications include labyrinthitis and facial nerve paralysis [3]. Intracranial complications include abscesses (temporal lobe, cerebellum, extradural, subdural), meningitis, sigmoid sinus thrombosis, and otitic hydrocephalus.

The purpose of this article is to report three cases of the complications of otitis media in children. The clinical presentation, diagnostic modality employed and management options used are discussed with relevance to the literature.

Case Reports

Case Study 1

A ten-month-old baby girl presented to the outpatient clinic after a three-week history of right otorrhea, and progressive postauricular swelling and erythema. The otologic examination revealed a right ear discharge and a polyp filling the medial part of the external auditory canal, associated with erythema, swelling and tenderness over the right mastoid region with anterior displacement of...
the auricle (Figure 1a). The patient was previously treated elsewhere with antibiotics: Cefuroxime (125 mg BID for five days) then Azithromycin (10 mg/kg on day 1 followed by 5 mg/kg daily for five days) then Lincocin (15 mg/kg daily for seven days) with no improvement. Ear cultures taken from a swab from the external auditory canal showed a heavy growth of *Pseudomonas aeruginosa* sensitive to common large spectrum antibiotics given for this germ. The patient was admitted to the pediatric ward with intravenous broad spectrum antibiotics (Ceftriaxone 70 mg/kg/day). The CT scan was out of order at that time. Accordingly, a brain MRI with contrast was done and it showed a right subperiosteal abscess (Figure 1b). The patient was planned for mastoidectomy.

Intraoperative findings revealed the presence of a subperiosteal abscess of the mastoid with gross cortical bone erosion. The whole cavity was filled with a coalescent mass of osteitic bone and calcareous pus. Cultures were sent for microbiological studies (bacteriological Gram staining and acid fast bacillus stain).

A canal wall-up mastoidectomy was done. Granulations were thoroughly removed from the aditus, attic, and around middle ear structures, leaving minor granulations over the stapes; the remaining necrotic portion of the tympanic membrane was also removed.

Intraoperative cultures did not yield any microorganisms and Acid Fast Bacillus test to rule out the possibility of a tuberculous infection, in a setting of florid mastoid granulations, was negative. The patient received a one week course of IV Ceftriaxone (70 mg/kg/day) followed by a total of two weeks of IV Cefepime (50 mg/kg BID) for better coverage of *Pseudomonas*. A computed tomography of the temporal bones was done seven days following surgery to attain a better delineation of the mastoid bone and to follow up the underlying infection. It showed a complete resolution of the subperiosteal abscess. The patient was then discharged home and was followed on outpatient basis for a period of six months postoperatively (Table I).

**Case Study 2**

This is the case of a five-year-old previously healthy girl with a history of recurrent left otitis media (six episodes per year), the last episode occurring a month prior to admission and treated by Ceftriaxone IM (50 mg/kg/day) for three days followed by Amoxicillin and Clavulanic acid (30 mg/ kg/day) for 10 days with poor response. She was admitted to our institution for management of left otalgia, otorrhea, headaches and strabismus. On physical evaluation, the patient had nasal obstruction. Her otologic exam revealed a left ear yellowish discharge, which after cleaning, uncovered a central tympanic membrane perforation. Her right ear showed a bulging drum with a middle ear effusion. Her eye examination revealed a left strabismus and the funduscopic exam showed normal optic discs and no papilledema. Nasofibroscopy confirmed the presence of adenoids. An MRI and MRA of the brain with gadolinium contrast revealed opacification of the mastoid air cells, with the left side being more involved, associated with an absent flow into the left sigmoid sinus. This was confirmed by post injection views that reported findings compatible with thrombophlebitis of the left sigmoid sinus. No intracranial extension was seen. Blood, ear and cerebrospinal fluid cultures were taken. The patient was planned for left mastoidectomy, adenoidectomy and bilateral PE tubes placement.

Intraoperative findings showed a well pneumatized left mastoid cavity with granulation tissue filling the attic.
Osteitic, eroded bone was seen over the sigmoid sinus which was also covered by granulations. Slight pulsations were visible in the sigmoid sinus and needle aspiration of the sinus revealed half a milliliter of blood and no pus. Mucopus filled the middle ear. Her adenoids were moderately enlarged.

The patient underwent adenoidectomy and a Shepard PE tube was inserted in the right ear. A left mastoidectomy was performed. Despite florid granulations in the attic, a mucosa lined passage was found between the aditus, attic, and middle ear. After needle aspiration of the sigmoid sinus, it was elected not to open it, allowing the sinus to recanalize. Cultures and biopsies were taken from the mastoid. A left anteroinferior myringotomy was done with the insertion of a long Armstrong PE tube. Polymerase chain reaction screening of mastoid fluid sent intraoperatively was compatible with streptococcus species.

The patient’s headache became worse and she started vomiting. Postoperatively, a funduscopic exam was repeated and it showed tortuosity of vessels with optic nerve swelling bilaterally, indicative of early papilledema. Several lumbar punctures were done that resulted in mild improvement of symptoms. Cerebrospinal fluid analysis was normal and cultures (CSF, blood, surgical wound) were negative for soluble bacterial antigens. With the presence of headaches, vomiting and papilledema compatible with increased intracranial pressure and a normal CSF analysis, the diagnosis of otitic hydrocephalus was established. So the patient was put on Acetazolamide for ten days. A lumbo-peritoneal shunt was installed due to the inefficacy of the lumbar punctures in relieving otitic hydrocephalus. The patient was not put on antiocoagulation since a CT venogram of the brain done ten days postoperatively showed partial repermeabilization of the left sigmoid sinus and the origin of the left internal jugular vein estimated to be around 30% patent. The CT scan of temporal bones showed however a possible periosteal reaction at the anterior aspect of the left petrous apex with poor aeration compared to the normally aerated right petrous apex. The diagnosis of petrous apicitis was confirmed by MRI of the temporal bones that showed opacification and enhancement of the left mastoid air cells which has increased and extending towards the petrous apex.

With the clinical triad of headaches involving the ophthalmic and maxillary distribution of CNV, left strabismus secondary to lateral rectus paralysis and acute petrositis, the patient was diagnosed with Gradenigo syndrome.

The patient was given a total of 3 weeks IV Ceftriaxone (75 mg/kg/day in two divided doses) and two weeks of steroids. The ophthalmology consult reported marked improvement in the patient’s strabismus with greatly decreased edema and loss of tortuous blood vessels on funduscopic exam. A repeat MRI of the temporal bones showed a decrease in the enhancement of the left petrous bone and mastoid air cells. No definite thrombosis was seen in the left sigmoid sinus or jugular vein. The lumbo-peritoneal shunt was removed after one year (See Table I).

### Table I

<table>
<thead>
<tr>
<th>Clinical Presentation, Time to Diagnosis, Imaging Modality, Treatment, and Outcome of Cases 1 and 2</th>
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<tbody>
<tr>
<td><strong>Age</strong></td>
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<tr>
<td>10 months</td>
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<td>5 years</td>
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Osteitic, eroded bone was seen over the sigmoid sinus which was also covered by granulations. Slight pulsations were visible in the sigmoid sinus and needle aspiration of the sinus revealed half a milliliter of blood and no pus. Mucopus filled the middle ear. Her adenoids were moderately enlarged.
Case Study 3

A three-year-and-a-half-old boy presented to our institution with a four-day history of frontal headaches, following an episode of left acute otitis media diagnosed by his pediatrician 10 days earlier. There was no associated fever, vomiting, neck stiffness or visual disturbances. The ENT exam revealed a persistent left otitis media and a normal neurological exam. Due to persistence of headache, a CT scan and an MRI of the brain were done. The CT scan showed full opacification of the left mastoid and an abscess in the location of the left sigmoid sinus which is either thrombosed or compressed. On MRI, a 2.5 cm x 1 cm x 2.5 cm possible abscess formation is seen at the site of the left sigmoid sinus. It shows a fluid level with rim enhancement. It is associated with thrombosis of the left sigmoid sinus and decreased flow in the transverse sinus. There is also complete filling of the mastoid air cells (Figure 2). The patient was started on IV Ceftriaxone, Vancomycin, and Metronidazole and was scheduled for left mastoidectomy and for evacuation of the extradural collection. Preoperative ear, blood and CSF cultures were taken, and were also sent to PCR.

Intraoperative findings showed a left mastoid cavity that was filled with granulation tissue and a magma of granulations surrounding the ossicles. Intracranially, florid granulation tissue was found adherent to the dura from the level of the middle mastoid cavity to a distance of 3 cm posteriorly, which is the site of the cerebellar dura. A complete canal wall up mastoidectomy was done. Granulation tissue was removed from the aditus, antrum and off the dura. It was elected to keep the middle ear open to improve drainage. The tympanic membrane was reclined anteriorly. It was also elected not to open the sigmoid sinus since needle aspiration of the sinus did not yield pus. Intraoperative specimens were sent to culture and pathology.

The neurosurgery team performed a left occipital craniotomy for evacuation of the extradural posterior fossa collection. Intravenous antibiotic therapy was continued for a period of three weeks, with the exception of Metronidazole that was given for five days postoperatively. All cultures were negative. However, the PCR for screening of common pathogenic bacteria was compatible with Streptococcus pneumoniae and Enterococcus faecium.

Postoperatively, an MRI of the brain revealed that the left sigmoid sinus and the left internal jugular vein became patent. No brain parenchymal lesion was seen.

The patient’s symptoms completely resolved and the follow-up was done monthly for six months, after which the patient underwent exploration of the left middle ear and a left myringoplasty to repair the surgically created posterior tympanic membrane defect. A fat graft obtained from the left lower abdominal quadrant was used to repair the defect. The patient was then followed monthly for four months then twice yearly for two years, showing good healing and no complications (See Table II).

### TABLE II
CLINICAL PRESENTATION, TIME TO DIAGNOSIS, IMAGING MODALITY, TREATMENT AND OUTCOME OF CASE 3

<table>
<thead>
<tr>
<th>Age</th>
<th>3 years and a half</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time to Diagnosis</td>
<td>After a 4-day history of frontal headache following left acute otitis media</td>
</tr>
<tr>
<td>Imaging Results</td>
<td>CT scan showed left mastoiditis; evidence of extradural abscess and left sigmoid sinus thrombophlebitis</td>
</tr>
<tr>
<td>Treatment</td>
<td>IV Ceftriaxone, Vancomycin, and Metronidazole</td>
</tr>
<tr>
<td></td>
<td>Mastoidectomy, evacuation of extradural abscess through a left occipital craniotomy</td>
</tr>
<tr>
<td>Outcome</td>
<td>Symptoms resolved completely</td>
</tr>
<tr>
<td></td>
<td>Repeat MRI showed patent sigmoid sinus and no abscess</td>
</tr>
<tr>
<td></td>
<td>Exploration of left middle ear + reconstruction 6 months after</td>
</tr>
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DISCUSSION

Acute coalescent mastoiditis (ACM) is a rare complication of acute otitis media. It is, however, more common in the pediatric population. Spremo et al. reported in 2007 an 84% incidence of ACM in a series of 13 patients developing acute mastoiditis [2].

Case 1 presented with swelling, erythema and tenderness over the mastoid bone with sagging of the posterosuperior canal wall. These symptoms are consistent findings in all series describing ACM [4, 7]. The first had more than a 3-week history of right otorrhea and progressive postauricular swelling and erythema not responsive to multiple antimicrobial therapies. According to Quinn et al., masked mastoiditis should be suspected when there is persistent pain for two weeks after antibiotic treatment in a poorly aerated ear [5, 7].

The diagnosis was made based on the clinical presentation and MRI of the brain with contrast, that showed a right mastoid filled with a soft tissue enhancing lesion and evidence of a subperiosteal abscess. It is important to note that an MRI was done in this case because the CT scan was out of order. CT scan of the temporal bones is the standard in the evaluation of acute mastoiditis. In order to classify mastoiditis as incipient or coalescent, a CT scan should be performed early. CT scan is considered the diagnostic tool that will guide therapy. If intracranial complications are suspected, then MRI would be more useful in identifying soft tissue fluid, edema and vascular problems [10].

Mastoiditis can be associated with a subperiosteal abscess [5, 9]. In a series of 70 cases of mastoiditis, studied by Kuczkowski et al., a subperiosteal abscess was considered the commonest extracranial complication, occurring in 90.2% of the cases [8]. Traditionally, subperiosteal abscesses were treated by mastoidectomy. In our case of ACM concomitant with a subperiosteal abscess, a complete mastoidectomy was done.

However, according to Bauer et al., treatment of a subperiosteal abscess with intravenous antibiotics, tympanostomy tube insertion and postauricular incision and drainage was considered adequate but required lengthy follow-up. This way of management was advocated to avoid the complications of mastoidectomy [9].

Ear cultures from case 1 showed a heavy growth of *P. aeruginosa*. ACM is usually caused by the same organisms as acute otitis media [4, 11]. The most frequently involved organisms are *Streptococcus pneumoniae* and *Haemophilus influenzae*. In subacute and chronic disease, *Staphylococcus aureus*, Gram negative bacteria such as *Escherichia coli* and *Pseudomonas aeruginosa* may be present. Kuczkowski et al. (2007) reported their series of 70 children with acute mastoiditis complicated by extra- or intra-cranial disease, among which *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Pseudomonas aeruginosa* were the most common isolates in ear cultures [8]. According to Quinn et al. (1998), among anaerobes, the most common pathogen is *Bacteroides* sp. [8, 16].

Concerning the management of ACM, it is divided into medical care, consisting of appropriate antimicrobial therapy, and surgical care. The timing is of great clinical significance. Surgical therapy should be directed promptly and it involves myringotomy and/or tympanocentesis for obtaining specimens with tympanostomy tube placement to provide access to the middle ear and mastoid for antibiotics and/or steroids. A simple or complete mastoidectomy is necessary to remove the areas of coalescence in the temporal bone [7, 11]. In our case, a simple mastoidectomy with tympanostomy tube insertion was done and middle ear structures were preserved.

The second case was that of sigmoid sinus thrombosis, otitic hydrocephalus (OH) and petrositis associated with Gradenigo syndrome. Otitic hydrocephalus (OH) is one of the rarest intracranial complications of acute otitis media. The clinical characteristics include signs of increased intracranial pressure (headaches, papilledema and vomiting) with the absence of focal neurological signs and no effect of hydrocephalus (no ventricular dilation). According to Kuczkowski et al. (2006), in a patient with a history of ear disease, a normal cerebrospinal (CSF) cytology and biochemistry with an opening pressure greater than 240 mm of water is essential for the diagnosis of OH and to differentiate it from meningitis [13].

The pathophysiology of OH is still unknown. Symonds in the 1930’s suggested that mural non-obstructing thrombus, extending from the lateral to the sagittal sinus leading to malabsorption of CSF, is required to produce increased intracranial pressure in OH [12]. According to Mete et al., patients with otitic hydrocephalus require ventriculoperitoneal shunt placement [15].

Since an intracranial complication was suspected from the clinical features found in Case 2, an MRI of the brain with contrast was used for diagnosis. It showed an opacified left mastoid cavity and thrombosis of the sigmoid sinus with no evidence of ventricular dilation. All cultures including CSF, ear and blood were negative and CSF cytology and biochemistry were normal. However, the PCR of the mastoid fluid that was sent intra-operatively was compatible with streptococcus species, one of the most common microbiologic agents seen in acute otitis media and mastoiditis [8, 11].

Sigmoid sinus thrombophlebitis is an uncommon but serious complication of acute otitis media. Secondary to venous obstruction, patients present with spiking fevers and headaches. The diagnosis is made when there is a high index of suspicion because clinical signs might not be present. MRI and CT are necessary to show lack of enhancement of the thrombus filling the dural venous sinus [14].

Agarwal et al. reported that venous obstruction usually resolves after 4-6 weeks without the need for surgical exploration of the sinus or anticoagulation, thus preferring a more conservative therapy [18]. The generally accepted approach, however, is simple mastoidectomy with decompression of the sigmoid sinus without opening it [15]. Some authors, on the other hand, suggest needle aspiration of the sigmoid sinus. If pus is present, the sinus is incised and drained into the mastoid [15]. For more
advanced cases, ligation of the internal jugular vein and resection of the thrombosed sinus can be attempted.

In our case, the needle aspiration of the left sigmoid sinus yielded half a milliliter of blood and no pus, so it was elected not to open the sinus.

Although rare since the advent of antimicrobial therapy, a serious complication such as Gradenigo syndrome can arise, as an extension of a middle ear or mastoid infection [16]. First described by Gradenigo in 1904, the syndrome is characterized by a triad of otorrhea, pain in the orbital area of the trigeminal nerve distribution, and ipsilateral CNVI palsy. The tendency for mastoiditis to lead to petrositis is partly due to the fact that the petrous apex is rich in pneumatized cells which are susceptible to infection and inflammation. In addition, the close proximity of venous sinuses to the petrous apex explains the high incidence of associated venous thrombosis [5, 16].

The most common pathogens involved in this disease are Group A Streptococcus, Pneumococcus, Staphylococcus, P. aeruginosa and Mycobacterium tuberculosis. However, these organisms are difficult to recover in petrous apicitis [17]. On the other hand, Yeung et al. (2006) reported that Pseudomonas was considered the most common pathogen and that tuberculosis was identified as a potential cause in patients younger than 20 years [16].

In the evaluation of petrous apicitis, a high resolution CT scan is the diagnostic method of choice that allows for detailed visualization of the petrous bone. Cuts should include the central nervous system and the temporal bones with the assessment of bone window settings [17]. In our case, petrous apicitis was confirmed by a CT scan and an MRI that showed a periosteal reaction at the anterior aspect of the left petrous apex and opacification of the left mastoid bone extending towards the petrous apex.

The treatment of this disease may involve radical mastoidectomy [5]. Treatment should be prompt in order to avoid intracranial extension and permanent abducens nerve injury. According to Lutter et al. (2005), conservative therapy with high dose broad spectrum antibiotics and myringotomy with tympanostomy tube placement is advocated [17]. If the infection persists or even progresses, then mastoidectomy, ranging from simple to radical, is warranted. To access the petrous apex for adequate drainage, a complete petrous apicectomy is required for patients whose anterior petrous apex could not be approached by familiar routes [16].

One of the most challenging complications to deal with is an intracranial abscess, because of its associated mortality and morbidity. However, this has changed dramatically after the antibiotic era [5, 18]. In a study involving 13 children with acute mastoiditis by Spremo et al. (2007), intracranial complications (IC) occurred in three patients; two had meningitis and one had a peridural intracranial abscess [2]. The most common intracranial complication is meningitis, followed by brain abscess and sigmoid sinus thrombosis [5, 18]. Early symptoms include otorrhea, fever, and headache while late findings involve altered mental status, cranial nerve palsy, nuchal rigidity, seizures, loss of consciousness, focal neurologic findings [5,19].

Most IC abscesses are polymicrobial. Streptococcus species are the commonest pathogens, combined with Hemophilus influenzae, Pseudomonas aeruginosa and anaerobes [19]. IC abscesses are difficult to diagnose, especially in the setting of early clinical features. CT scan and more preferably MRI of the brain are essential and should precede a lumbar puncture when required [10, 19]. An abscess appears as a ring enhancing lesion or as a space occupying process. Imaging can also point the clinician to the source of infection [19]. CRP, ESR and elevated white cell counts are also useful, particularly when monitoring response to treatment.

Surgery is the mainstay of therapy after initiation of high dose broad spectrum antibiotics and urgent neurosurgical referral. Surgical options range from aspiration to craniotomy. Image-guided stereotactic aspiration has recently become the most utilized technique for abscess drainage [19]. This procedure is particularly effective in small (< 2 cm) and deep abscesses when localization is more accurate. Craniotomy can be done as a primary procedure or secondarily if the abscess recurs after initial aspiration [19]. Steroids are generally not used in the treatment of brain abscesses due to their immunosuppressive effects. However, marked edema may surround the abscess and contribute to the signs of increased intracranial pressure. Accordingly, if the clinical situation deteriorates, steroids are used for their anti-inflammatory effect [19].

**CONCLUSION**

Intracranial and extracranial complications of acute otitis media should be suspected in the setting of unrelenting otalgia and otorrhea, high fever and headaches. Prompt diagnosis with CT scan, MRI and panceultures is essential. Early antimicrobial therapy directed against the most common pathogens may avert complications of acute otitis media. However, surgical therapy remains the mainstay of treatment in most cases.

**REFERENCES**