The management of the intracranial complications of acute otitis media in children

Abstract
Acute Otitis Media (AOM) is a very common condition in children. Complications of acute otitis media have reduced with the introduction of antibiotics. However, intratemporal and intracranial complications still arise, particularly if there is acute mastoiditis. We discuss the intracranial complications of acute otitis media in children together with evidence-based management.

Keywords
Otitis media, intracranial, complications, abscess, thrombosis.

Introduction
Acute otitis media (AOM) can be defined as inflammation of the middle ear cleft associated with a middle ear effusion which is of rapid onset and infective in origin. It can occur with an intact tympanic membrane or in the presence of a tympanic membrane perforation or ventilation tube. It is a common condition in children, with about 30% of children aged under 3 years presenting to general practitioners with acute otitis media each year. Indeed, up to 10% of children will have an episode of acute otitis media by 3 months of age.1 Although acute otitis media is usually a benign self-limiting disease, it can progress to a life-threatening condition. The advent of immunisations and antibiotics in the 20th century led to a reduction of complications of acute otitis media.2 However, serious complications continue to occur and in some cases are increasing, with increasing antibiotic resistance being implicated.3 Acute otitis media can result in acute mastoiditis. The age at which mastoiditis occurs has been relatively constant over the last decade and is most common in children. The risk reduces with age according to HES data (Figure 1). Further evaluation of HES data indicates that the mortality within 30 days of admission to hospital is 1-3 deaths per year.
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_Acute Mastoiditis rate_

Pneumococcal-7-valent conjugate vaccine (PCV-7) was introduced in September 2007 and probably resulted in a change in the principal causative organism in recurrent AOM (from S pneumoniae to H influenza). The Health Protection Agency has reported that the number of invasive pneumococcal infection caused by types covered by PCV-7 has reduced. However, using Hospital Episode Statistics (HES) data it is possible to identify that there has not been any apparent change in the rate of hospital admissions for patients admitted with acute mastoiditis in the UK over the last decade (Figure 2). This may be explained since many different serotypes cause AOM.

_Figure 2:_ Finished Consultant Episodes per year for Acute Mastoiditis from 2000-2010.

Figure 2 demonstrates that the rate of acute mastoiditis has been relatively stable for the last decade in England.

**Complications**

The complications of acute otitis media can be divided into the intratemporal and intracranial. Although we are focusing on the intracranial complications of acute otitis media in this article, some mention must be made of the intratemporal complications.

Intratemporal complications include:

- **Acute mastoiditis**
  - Infection and inflammation from the middle ear cleft can easily spread to the mastoid cavity and be seen as opacification on CT or MR scans. The infection can then spread to the mastoid periosteum causing periostitis. This can manifest as loss of the post-auricular sulcus and protrusion of the pinna. Soft tissue inflammation and oedema occurs and results in sagging of the postero-superior wall of the external auditory meatus. Additionally, cellulitis can occur around the pinna before an abscess forms (Figure 3a & 3b). Osteitis and destruction of the mastoid air cells can occur, leading to a subperiosteal abscess, Bezold’s abscess or Citelli abscess. The infection spreads from the middle ear cleft either directly by bone erosion through the cortex or indirectly via the emissary vein of the mastoid. Drainage of the abscess and a cortical mastoidectomy reduces the duration of the condition and identifies occult cholesteatoma which may be otherwise difficult to identify in the severely inflamed and distorted tympanic membrane.

_Figure 3a:_ Significant oedema over the left temporal bone of a child with AOM. Repeated ultrasound was used to monitor any abscess formation. The condition responded to grommet insertion and intravenous antibiotics.
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A lumbar puncture must not delay the administration of parenteral antibiotics, which is the mainstay of treatment. The preferred antibiotic regime in the United Kingdom is intravenous ceftriaxone for at least 10 days, depending on the course of the illness.8

Myringotomy is not initially required unless a specimen is needed if a lumbar puncture cannot be performed. Consideration for mastoid surgery in the presence of other intracranial or intratemporal complications should usually be made once the patient’s medical condition has stabilised.9

Intracranial complications – Meningitis

Meningitis has previously been reported to be the most common intracranial complication of acute otitis media in children.4, 5, 6 However, more recent series have reported that intracranial abscess is more common and indicates a changing trend. Streptococcus pneumonia is the causal organism in an increasing proportion of cases since the haemophilus influenza type B vaccination was introduced.7

Prompt input from a paediatrician should be obtained if meningitis is suspected. A lumbar puncture should be performed as a primary investigation unless this is contraindicated. The contraindications to performing a lumbar puncture include:

- signs suggesting raised intracranial pressure
- reduced or fluctuating level of consciousness (Glasgow Coma Scale score less than 9 or a drop of 3 or more)
- focal neurological signs
- papilloedema
- coagulation abnormalities
- platelet count below 100 x 109/litre

In the preantibiotic era, lateral sinus thrombosis was the second most frequent fatal complication of acute otitis media, with mortality approaching 100%. It is now more often seen in the adult patient associated with chronic otitis media and cholesteatoma10 and mortality in some cases series has been reported as approaching 0%.11

The clinical features of patients with otogenic lateral sinus thrombosis include headache, otorrhoea and fever. The signs of raised intracranial pressure or otitic hydrocephalus are present only in few cases.10, 12 There is also the risk of distant septic emboli from an infected thrombus (Lemierre’s), although this is rare (Figure 4 demonstrates lung septic emboli from a lateral sinus thrombosis).

Intracranial complications – Lateral Sinus Thrombosis

The right lateral sinus is usually a direct continuation of the superior sagittal sinus and is larger than the left. The left is usually a continuation of the straight sinus and is smaller. The portion of the lateral sinus which curves inferiorly, occupying a groove in the mastoid part of the temporal bone and leads to the internal jugular vein is termed the sigmoid sinus. The proximity of the middle ear cleft and the mastoid air cells to the lateral sinus puts the sinus at risk with acute mastoiditis.

Lateral sinus thrombosis can develop as a complication of acute mastoiditis by the direct dissemination of the infection through neighbouring eroded bone. However, it can also occur in acute otitis media with an intact sigmoid plate, indicating propagation by thrombophlebitic spread through the mastoid emissary vein.

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Patients with a suspicion of lateral sinus thrombosis must have radiological evaluation to confirm the diagnosis. CT scanning is readily available and quicker to perform. Bony erosion and coalescence within the mastoid are well demonstrated by CT. This can be crucial in distinguishing otogenic lateral sinus thrombosis from non-septic lateral sinus thrombosis if the otological signs are not clear. Contrast enhanced CT in the venous phase shows a filling defect in the lateral sinus, sometimes with an enhancing rim from contrast in collateral veins (the empty delta sign). More recently MRI and MR venography have been used to more accurately differentiate slow venous flow from occlusion by thrombus and is more sensitive than CT. MR can be used in follow up scans to monitor recanalisation. However, MRI scanning requires more time hence some children may need a general anaesthetic to ensure less movement artefact. Some authors now advocate both CT and MR modalities in the workup and management of lateral sinus thrombosis. Figures 5a & 5b demonstrate venous thrombosis on CT and MRI scanning.

The management of otogenic lateral sinus thrombosis has been evolving. Certainly intravenous antibiotics have been a mainstay of treatment and a six week course is appropriate. Traditional teaching also mandated a mastoidectomy with attempted needle aspiration of the lateral sinus to confirm the presence of a thrombus. If a thrombus was confirmed, the sinus was to be incised and the clots removed until free bleeding occurred. The bleeding was controlled by iodoform packs. However, retrospective review suggests there is no difference in clinical outcome between simple needle aspiration and thrombectomy. The concept of simple needle aspiration is unlikely to effect any real change to the clinical outcome and therefore one may extrapolate to state there is no benefit with or without thrombectomy. If the thrombus extended to the IJV, some advocate ligation to prevent septic emboli. However, septic emboli are relatively rare when antibiotics are used. Indeed, IJV ligation may be associated with an increase in septic emboli.

With regards to otogenic non-cholesteotoma lateral sinus thrombosis, some authors recommend a cortical mastoidectomy with removal of the overlying sinus plate in order to allow drainage of any perisinus disease but without incising the sinus. A ventilation tube can also be placed at the time of mastoidectomy. The sensitivity of MR venogram makes needle aspiration or opening of the sinus in order to diagnose a thrombus unnecessary. Indeed, in non-septic lateral sinus thrombosis, surgical thrombectomy is not required. Previous studies recommended IJV ligation at the time of mastoidectomy if IJV thrombosis was present. However, more recently there has been a move to not ligate even if the thrombus extends to the IJV.

The routine use of anticoagulation in lateral sinus thrombosis remains a contentious issue. In a case series of 62 adults with isolated lateral sinus thrombosis, all were treated with anticoagulation and not with surgery. However, only one of these patients had otitis media. In a case series by Holzmann et al, all 15 patients with lateral sinus thrombosis were anticoagulated with IV heparin. No patients died or had deterioration in their neurological status. However, only 2 out of 15 patients reached a therapeutic level of anticoagulation. Other studies suggest only anticoagulating if the patient is found to be in a hypercoagulable state, or if neurological symptoms deteriorate despite antibiotic and surgical treatment. A case series identified five children to be heterozygote for C677T MTHFR mutation which led to a prothrombic state. All were treated successfully with anticoagulation. Future management of LST may indicate evaluation of such prothrombotic disorders. If anticoagulation is to be considered, the use of low molecular weight heparin should be used with caution due to reports of post-operative bleeding and haematoma. Some options for treating symptoms of raised intracranial pressure are discussed in the section below.

Children form a significant proportion of the group with lateral sinus thrombosis. We propose that it is not justifiable...
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to incise and remove a thrombus from a venous sinus if the intention is to then anticoagulate as there is a risk of intracranial haemorrhage. Paediatric neurologists should be consulted if anticoagulation is considered for children. Young children fall more readily and anticoagulation is generally not desirable. If the thrombosis is significant and affects bilateral venous sinuses, hydrocephalus and raised intracranial pressure requiring lumbar puncture drainage is contra-indicated in an anticoagulated child. Additionally, septic emboli are a significant problem during the extraction of the venous sinus thrombosis.

No difference in venous sinus recanalisation rates has been demonstrated between anticoagulated and non-anticoagulated groups. However, the role of repeated follow-up scanning as a means of monitoring thrombus is debateable as the neurological complications that occur as a consequence of LST seem to be independent of recanalisation.

Intracranial complications – Otitic hydrocephalus

The term “otitic hydrocephalus” was coined by Symonds in the 1930s. Symonds described patients with symptoms of raised ICP and swelling of the optic discs with haemorrhage that became symptomatically better following lumbar puncture. With the introduction of CT scanning, we now know that the term is an unfortunate misnomer as there is often no dilatation of the ventricles in otitic hydrocephalus and the raised intracranial pressure is probably the result of venous congestion (not even complete obstruction). It is now the accepted term for increased intracranial pressure in the absence of focal neurologic signs, together with normal CSF cytology and biochemistry in association with middle ear disease. Care must be taken when performing a lumbar puncture in the presence of papilloedema due to the risk of cerebellar coning.

The clinical features of raised ICP in otitic hydrocephalus include:

- Headache
- Nausea and vomiting
- Drowsiness or irritability
- Papilloedema
- Diplopia (due to abducens nerve palsy, presumably caused by stretching of the nerve)

The treatment of otitic hydrocephalus very much depends on the other clinical findings. Lateral sinus thrombosis is often, but not always found. This in turn can be caused by acute mastoiditis. In addition, not all patients with lateral sinus thrombosis will have otitic hydrocephalus. Anatomical variation of the intracranial venous sinuses is one possible explanation. The right lateral sinus is more often dominant and is the main drainage for the sagittal sinus.

If the symptoms of raised intracranial pressure do not resolve, then the following management strategies could be followed:

- IV Mannitol (an intravascular osmotic agent)
- This can be used to treat raised ICP prior to surgery
- Acetazolamide reduces CSF formation hence pressure
- Drainage of CSF via repeated lumbar puncture or an intraventricular catheter
- Antiepileptic medication for seizures
- The use of anticoagulation for lateral sinus thrombosis has been discussed earlier.

Intracranial complications – Extradural, subdural and brain abscesses

Abscesses of otologic origin are epidural, subdural or intraparenchymal. Commonest symptoms include: pyrexia, otalgia and headache but a fluctuating mental state may also be apparent. Neurosurgical opinion is essential though the primary management requires treatment of the ear infection; mastoidectomy with or without craniotomy. However, reports of conservative management are published. Contemporary reports suggest very low mortality in developed regions. A small series of five brain abscesses due to otogenic origin were managed successfully with mastoidectomy and no neurosurgical intervention.

Empyema is an uncommon complication. When the empyema is small it can be managed conservatively with mastoidectomy, antibiotics and serial MRI scans to check progress (Figure 6 demonstrates a relatively small middle fossa empyema managed in this way). Larger collections in the posterior fossa may be drained relatively easily during mastoid exploration by additionally taking down bone in the immediately posterior to the lateral sinus (Figure 7 demonstrates a posterior fossa empyema that was managed as described).

Conclusion

Acute Otitis Media is still a very common condition yet the complication rate is low. Acute mastoiditis is relatively uncommon however the complications are proportionately more likely to result in intracranial complications. The mainstay of treatment is antibiotics and mastoid exploration. The management of LST is more controversial. We present evidence that questions the traditional management of the thrombosed lateral sinus. We propose a more conservative approach with the benefit of other specialists such as neurologists, neurosurgeons and perhaps increasingly haematologists.

Conflict of Interest

All authors have no conflict of interest to declare. No extraneous funding was obtained.
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**Figures 6 and 7: Examples of empyema.**

### References


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