REVIEW ARTICLE

Indications and Radiological Findings of Acute Otitis Media and Its Complications

Elena Pont, a * Miguel Mazón b

a Servicio de Otorrinolaringología, Hospital General de Onentiente, Valencia, Spain
b Sección de Neurorradiología y Radiología de cabeza-cuello, Área Clínica de Imagen Médica, Hospital Universitario y Politécnico La Fe, Valencia, Spain

Received 19 January 2016; accepted 24 February 2016

KEYWORDS
Complications; Intratemporal; Intracranial; Acute otitis media; Radiology

Abstract Most cases of acute otitis media resolve with antibiotics and imaging is not required. When treatment fails or a complication is suspected, imaging plays a crucial role. Since the introduction of antibiotic treatment, the complication rate has decreased dramatically. Nevertheless, given the critical clinical relevance of complications, the importance of early diagnosis is vital.

Our objective was to review the clinical and radiological features of acute otitis media and its complications. They were classified based on their location, as intratemporal or intracranial. Imaging makes it possible to diagnose the complications of acute otitis media and to institute appropriate treatment. Computed tomography is the initial technique of choice and, in most cases, the ultimate. Magnetic resonance is useful for evaluating the inner ear and when accurate evaluation of disease extent or better characterisation of intracranial complications is required.

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PALABRAS CLAVE
Complicaciones; Intratemporales; Intracraniales; Otitis media aguda; Radiología

Indicaciones y hallazgos radiológicos de la otitis media aguda y sus complicaciones

Resumen La mayor parte de casos de otitis media aguda se resuelven con el tratamiento farmacológico, y los estudios de imagen no están indicados. En los casos en los que el tratamiento fracasa o se sospecha complicación, las pruebas de imagen tendrán un papel fundamental. Desde la introducción del tratamiento antibiótico la frecuencia de complicaciones ha disminuido drásticamente. No obstante, dada su relevancia clínica crítica, es de vital importancia su diagnóstico precoz.

Abreviaturas: AOM, acute otitis media; CT, computed tomography; ME, middle ear; MR, magnetic resonance.

* Please cite this article as: Pont E, Mazón M. Indicaciones y hallazgos radiológicos de la otitis media aguda y sus complicaciones. Acta Otorrinolaringol Esp. 2017;68:29–37.

* Corresponding author.
E-mail address: elenapont@hotmail.com (E. Pont).

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Introduction

Acute otitis media (AOM) is a bacterial infection of the middle ear (ME). It is more common in winter and at a paediatric age due to the Eustachian tube’s horizontal direction and hypertrophied nasopharyngeal adenoid tissue. It is usually unilateral and generally a consequence of a cold in the upper aerodigestive tract or other nasopharyngeal infection. It is usually a monomicrobial infection, most commonly with Streptococcus pneumoniae and Haemophilus influenza which reach the ME via the Eustachian tube. They produce inflammation of the mucoperiosteum of the ME, seromucous hypersecretion and cellular infiltration of elements on the route causing purulent detritus to appear among abundant discharge. The fibrous layer of the tympanic membrane is also affected and it ruptures when it cannot withstand the pressure of the ME contents, discharging towards the external auditory canal.

Clinically it is characterised by the speedy and sudden onset of signs and symptoms in the ME. The patients initially present earache and fever. Other possible symptoms include irritability, anorexia, vomiting and diarrhoea. As the contents of the eardrum increase and the tympanic membrane thickens, conductive hearing loss develops. When this happens, otoscopy shows a reddened, bulging and opaque tympanic membrane due to the liquid content of the eardrum and its mobility is reduced. This clinical status can progress to tympanic perforation, when the discharge starts and the patient’s earache and general condition improve.

It should be distinguished from other types of acute otitis media, such as serous or seromucinous otitis media, which result in an accumulation of serous and/or mucoid fluid with no infection, and from 2 other specific subtypes such as influenza otitis media and necrosing otitis media; the former secondary to flu and characterised by the presence of haemorrhagic phlyctenules in the tympanic membrane and the latter associated with scarlet fever and mumps, which is more aggressive and can cause necrosis of the tympanic membrane and disruption of the ossicular chain.

In the majority of cases of AOM the infectious process resolves with antibiotic treatment, and imaging studies are not indicated. Computerised tomography (CT can identify opacification of the ME and mastoid with possible hydro levels, with no bone erosion of the ossicular chain, walls of the mastoid or cortical cells of the temporal bone. However, in cases where antibiotic treatment fails and the infection persists, symptoms worsen or new symptoms appear, a complication must be suspected. In this scenario, imaging tests play a crucial role, since they enable early diagnosis of complications and immediate appropriate treatment. Computed tomography is therefore a decisive diagnostic tool in the choice of treatment necessary in these cases.

In this study we show our experience in recent years in the process of diagnosing the complications of AOM. We describe the clinical symptoms of each complication and the recommended imaging technique, and review and illustrate the characteristic radiological findings.

Materials and Methods

A retrospective review was undertaken of patients diagnosed with AOM and an AOM complication in our centre over the past 8 years. The clinical picture and imaging studies performed were reviewed. The process of diagnosis was re-examined and the imaging studies most representative of each complication were selected. An urgent CT with intravenous contrast was performed in all cases with a suspected complication, the study was completed with MRI in selected cases where it was considered necessary in order to corroborate the findings or, more precisely, characterise/establish the extent of intracranial involvement. CT equipment and the study protocol have changed in recent years, with a view to optimising spatial resolution and reducing radiation. We currently use multi-slice CT equipment (CT Multidetector Brilliance 64; Philips Healthcare, Best, Basque Country), if a complication is suspected, intravenous contrast is administered and volumetric data acquired which reconstruct in a bone window on transversal and coronal planes with a thickness of 0.3–0.6 mm, and a soft tissue window on transversal and coronal planes with a thickness of 1.2 mm. We use Signa HDxt 1.5 and 3.0T MR equipment (GE Healthcare, Waukesha, WI, U.S.A.); intravenous contrast is also administered in the study protocol in the event of a suspected complication.
Results

Since the introduction of antibiotics in the treatment of AOM, the frequency of complications has reduced considerably. Only a small proportion of untreated or inappropriately treated patients develop complications (1%-18% depending on the series). However, there are already publications that have demonstrated a progressive increase in bacterial resistance and a change in microbial flora, which have produced an increase in the incidence of acute mastoiditis in the paediatric population who are receiving the appropriate treatment.

We classify the complications into intratemporal or intracranial according to their anatomic location. CT is the initial imaging technique of choice and usually the definitive technique. This is a rapid procedure that is comfortable for the patient and widely available, it enables very accurate evaluation of bone involvement and extracranial involvement, and of most intracranial complications with acceptable sensitivity and precision. Magnetic resonance (MR) is reserved for cases where more precise demarcation and/or characterisation of intracranial complications is required. In cases where it is available it can be used as a first choice if there is high suspicion of an intracranial complication, although we prefer to use CT beforehand in our clinical practice, because in most cases it will be sufficient and in any case will facilitate interpretation of the MR findings. The role of MR in assessing the inner ear is undeniable, and is indicated if a labyrinthine complication is suspected.

Intratemporal complications include: coalescent otomastoiditis, subperiastic abscess, labyrinthitis, facial palsy and petrositis. Intratemporal complications include: meningitis, dural venous sinus thrombosis, epidural abscess, subdural empyema and encephalic abscess. We describe the clinical and radiological features of these complications below.

Intratemporal Complications

Coalescent Otomastoiditis and Subperiastic Abscess

Coalescent otomastoiditis is a consequence of persistent infection of the ME. It is characterised by the osteoclastic resorption of the mastoid cell walls, due to the formation of microabscesses below the mucosa that cause bone demineralisation as a consequence of an increase in local pressure and osteolytic enzymes. It is the most common complication, although it only occurs in 0.02%-0.2% of AOM, presenting most frequently between 6 months and 3 years of age. It presents clinically with swelling and pain in the retroauricular region. Occasionally there can be effacement of the retroauricular crease, known as signo de Jacques. The mastoid cells coalesce in wider cavities full of purulent exudate and granulation tissue, which results in the formation of empyema and coalescent mastoiditis.

The bone resorption osteoclastic process as it develops can extend to the external or internal cortex. If there is cortical extension through the mastoid cortex a subperiastic abscess can form in the overlying soft tissues, with surrounding cellulitis, which can even spontaneously fistulate to the skin. When located in the retroauricular region, the pinna displaces anteriorly becoming detached from the skull. Bezold’s abscess is a specific type of subperiastic that occurs when osteolysis affects the tip of the mastoid. It forms in the digastric cleft and extends inferiorly through the internal face of the insertion of the sternoclavomastoid muscle. In small children the tip of the mastoid is not usually pneumatized, and therefore this complication is rare.

CT is the test of choice to confirm diagnosis and assess the need for urgent surgical treatment. This technique enables evaluation of mastoid septum and cortex erosion – the characteristic finding in coalescent mastoiditis (Fig. 1). It should be borne in mind that due to the great variability in pneumatisation of the mastoid, comparison with the contralateral side is very helpful in detecting lysis of the bony trabeculae. Likewise, CT provides information on the existence of encapsulated liquid collections liquid with an enhanced wall on the surface of the temporal bone corresponding to abscesses, usually in a retroauricular location, throughout the zygomatic process, or through the external auditory canal (Fig. 2). MR also enables diagnosis of subperiastic abscesses, but it is less precise in evaluating bone.

Labyrinthitis

This is infection/inflammation of the membranous and sensorineural portions of the inner ear. The use of antibiotics in the management of AOM has reduced its incidence very significantly. Clinically it should be suspected when a patient with AOM develops vertigo, nystagmus or sensorineural hearing loss. There are two distinct clinical forms: diffuse, usually in the context of AOM, and circumscribed, specific to cholesteatoma.

In cases of complicated AOM, labyrinthitis is termed tympanogenic, since it is the consequence of the direct spread of inflammation or bacterial infection from the ME through the oval or round window. There are 2 types of diffuse labyrinthitis: serous, also termed toxic acute, usually reversible, caused by an inflammatory reaction of the labyrinth due to the translocation of toxins and inflammation factors from the ME; and supplicative, also termed destructive or necrosing, due to the invasion of the labyrinthine spaces by bacteria and inflammatory cells with pus and inflammatory tissue, which usually results in the irreversible destruction of the labyrinth and presents with intense vertiginous syndrome and ablative or paralytic nystagmus.

Diagnosis is essentially clinical, with the abovementioned symptoms, although it must be taken into account that a final diagnosis can be made using imaging techniques. It is classified into 3 evolutionary stages: acute, fibrous and ossifying. In the acute phase there is enhancement on MR of the membranous portion of the inner ear after administration of contrast, whereas its fluid signal intensity is usually normal (Fig. 3A). If the infection is not resolved, labyrinthine fibrosis can occur, in which intensity of fluid signal is lost and pathological enhancement can persist. It can eventually progress to ossification; a process termed ossifying labyrinthitis. In this case, in addition to the loss of fluid signal intensity on MR, CT can identify high density deposits in the membranous labyrinth, corresponding to ossification (Fig. 3B). The most
Figure 1  *Cochlear otomastoiditis.* Transversal bone-window CT images (A and B, lower and upper) which show occupation of the middle ear and the mastoid cells, destruction of the bone septa of the cells (arrow tips) and the internal cortex (arrows).

Figure 2  *Subperiosteal abscess.* Transversal bone-window and soft tissue CT images (A and B). (A) Otomastoid occupation is identified with erosion of the bone septa of the cells and external cortex (arrow tips). (B) A collection can be observed in the soft tissues on the surface of the temporal bone corresponding to a subperiosteal abscess (arrows). (C) On the ultrasound image the abscess can be identified as an encapsulated liquid collection with a thick wall and anechoic content (arrows). Cortical lysis can also be observed (arrow tips).

frequently ossified area is the scala tympani of the basal turn.  

Quantifying enhancement in the acute stage of labyrinthitis is the first objective of some studies in order to differentiate inflammation/infection from enhancement of tumour aetiology.

Facial Palsy
This is a very rare complication of AOM. The segments of the intratemporal facial nerve most commonly affected are the tympanic and mastoid. The lesion is not usually destructive and is reversible, although it can be a consequence of osteoclastic lysis of the intratemporal facial nerve bone

Figure 3  *Acute and ossifying labyrinthitis.* (A) Transversal MR image with intravenous contrast showing pathological increased uptake of the entire labyrinth (arrows) corresponding to a diffuse labyrinthitis. (B) The follow-up CT study of the patient shows high-density foci in the vestibule due to ossification (arrow) indicative of ossifying labyrinthitis.
Other more common causes of otogenic facial palsy, from which it should be distinguished, are cholesteatoma and malignant external otitis. In these disorders, in addition to facial palsy, there is usually lysis of the otic capsule with dehiscence and fistula of the horizontal semicircular canal, which causes symptoms of dizziness.

Lysis of the bone canal of the intratemporal facial nerve can be assessed using CT. MR enables evaluation of the pathological uptake of contrast by the nerve itself. It should be borne in mind that enhancement of the geniculate ganglion and the tympanic segments and mastoid is considered normal, whereas enhancement of the cisternal, intracanalicular, labyrinthine and extracranial mastoid segments is pathological. In routine practice, comparison with the contralateral facial nerve is helpful. Uniform contrast of increased uptake is frequently identified along the length of the facial nerve, which is of normal thickness. Nodular enhancement or focal thickening are atypical findings that suggest neoplastic disease, such as Schwannoma or perineural dissemination of carcinoma.

Petrositis
This is a very rare complication. It is the spread of infection to the petrous apex. It requires pneumatisation of the tip of the petrous bone (30% of the population), and in this case there is a connection with the mastoid cells via several supra and infralabyrinthine canals that enable the spread of infection from the mastoid and the ME. It is more common in children under 7 and in immunosuppressive states.

The most common germs involved are Streptococcus pneumoniae, Haemophilus influenzae and Staphylococcus aureus. Clinically it presents with worsening earache, fever and orbital pain; the characteristic clinical sign is the onset of strabismus with diplopia which affects the same side as the AOM. Severe facial pain and diplopia are a consequence of inflammatory neuralgia of the 5th and 6th cranial nerves respectively, known as Gradenigo’s syndrome. It can also affect the vidian nerve presenting with vasomotor rhinitis – symptoms known as Vail’s syndrome.

Urgent CT is indicated. It shows opacification of the air cells of the petrous apex, lysis of the bone trabeculae with coalescence and possible destruction of the cortex. MR enables occupation of the petromastoid complex to be assessed. MR enables assessment of the petromastoid complex - if it contains pus, spread will restricted - after administration of contrast the petrous apex enhances and is usually accompanied by pachymeningeal thickening (Fig. 4). MR might also demonstrate enhancement of the affected cranial nerves and help assess the existence of other intracranial complications.

Intracranial Complications
Advances in antimicrobial therapy have substantially reduced the incidence of these complications varying between 0.04% and 0.36%. Greater therapeutic effectiveness has been demonstrated with long-term treatment.

There are 3 possible routes of spread of infection to the intracranial space: progressive ascending osteothrombophlebitis, direct spread due to bone erosion of the internal cortex and through preformed access routes such as the round window, iatrogenic bone dehiscence or as a consequence of other disease processes such as cholesteatoma.

Clinically, they should be suspected in a patient with a history of AOM presenting with fever, headache, lethargy and nausea. An altered state of consciousness, cranial nerve palsy, diplopia, ataxia, aphasia, hemiplegia and other neurological signs can present in the most advanced cases.

Meningitis
Most cases of meningitis as a complication of AOM occur due to haematogenous dissemination, although they can also be secondary to direct spread. Other more specific signs and symptoms present in general to additional symptoms of fever and headache, such as neck stiffness, altered mental state and Brudzinski’s sign (involuntary flexion of the hips and knees when the neck is flexed) and Kernig’s sign (pain
and resistance on flexing hips and knees and attempting to extend the knees). Although diagnosis is confirmed by laboratory tests and analysis of cerebrospinal fluid, and imaging tests are usually not necessary, prior to performing lumbar puncture a CT scan is generally requested to rule out intracranial hypertension. If CT or MR are performed due to a suspected additional complication, enhancement of the brain surface (pia mater) and exudate in sulci and cisterns, as well as possible hydrocephaly can be identified (Fig. 5).

**Epidural Abscess**

This is one of the most common intracranial complications. It is due to the direct spread of infection that erodes the internal cortex and spreads to the epidural space of the posterior fossa or middle cerebral fossa. An epidural abscess forms when pus collects in this space. Clinically these abscesses can be silent, and therefore imaging studies are essential for their diagnosis. Both CT and MR are useful and enable diagnosis. CT enables evaluation of erosion of the cortex and MR enables more detailed evaluation of the remaining intracranial components. Both techniques enable identification of extra-axial collection in an epidural location, of purulent content and with a thick wall of increased uptake. The presence of gas is possible in its interior (Fig. 6).
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Trombophlebitis of Dural Venous Sinus
In more than half the cases this is a consequence of an epidural abscess. Thrombosis acts as a protective mechanism to prevent the spread of infection.\(^5\) Another possible pathogenic mechanism, although less common, is osteothrombophlebitis with neither erosion of the sigmoid plate nor formation of an abscess. Usually the transverse and sigmoid dural venous sinuses are affected. As a consequence of thrombosis, cerebral venous drainage can be made difficult, intracranial pressure can increase and there is the possibility of cerebral venous infarction, frequently located in the area of the vein of Labbé. Clinically it is difficult to diagnose, patients can present with signs of intracranial hypertension or hydrocephaly such as intense headache, photophobia, dizziness and nausea.

Imaging techniques, CT and MR, are necessary for diagnosis of the condition. Otomastoid occupation and sigmoid plate lysis can be observed with bone-window CT scan. Morphological MR sequences show loss of venous sinus flow gap. CT and MR venography sequences enable direct diagnosis, by using both techniques the thrombus will be identified as a repletion defect in the affected dural venous sinus (Fig. 7).

Subdural Empyema
This is a purulent collection in the potential space between the dura mater and the arachnoid membrane. Because this is a preformed space the correct term is empyema and not abscess. The majority of cases are a consequence of the spread of microorganisms from the middle ear to the subdural space, through veins and venules by means of a mechanism of trombophlebitis and periphebitis; less frequently it is the result of direct spread.\(^9\) The clinical picture is highly symptomatic, patients can present meningism, neurological focality or seizures. CT and MR show a purulent collection in the subdural space, frequently located on the tentorium.

Figure 7 Dural venous sinus trombophlebitis. (A) Transverse CT image with intravenous contrast showing the repletion defect of the transverse dural venous sinus due to trombophlebitis (white arrows, black arrow for comparison with the normal contralateral sinus). (B) MR with intravenous contrast of the same patient identifying the thrombus in the transverse and sigmoid venous sinuses in the same way.

Figure 8 Coalescent otomastoiditis and brain abscess. Bone-window CT images (A and B, lower and upper) showing the occupation of the middle ear and mastoid cells with destruction of the bone septa (arrows) and the inner cortex of the posterior edge of the petrous bone (arrow tip). On the transversal CT image of the head with IV contrast of the same patient (C), a rounded collection with a thick wall of increased uptake can be seen in the right cerebellar hemisphere corresponding to a brain abscess.
Brain Abscess
This is a rare complication of AOM. The most usual routes of spread are thrombophlebitis and bone erosion with meningeal rupture and direct invasion of the brain parenchyma. The abscess is usually located close to the source of infection, and therefore the cerebellar hemisphere and the temporal lobe are the most frequently affected anatomic regions. Clinically, neurological symptoms have to be added to the symptoms of AOM due to the central involvement of the brain, such as dizziness, nystagmus, visual field defects. As with the remaining intracranial complications, imaging tests enable diagnosis to be confirmed and help in planning surgery for cases where it is necessary. CT and MR with intravenous contrast will show rounded intra-axial collection, with a thick wall of increased uptake, whose purulent content restricts the diffusion sequence (Fig. 8).

Other Intracranial Complications
Encephalitis, or cerebritis, is another possible complication characterised by an oedematous and inflamed focal area of the brain. It is usually the consequence of another complication such as an extra-axial abscess or dural venous thrombophlebitis. And finally, another possible complication, although very rare, is involvement of the internal carotid artery. This should be suspected if there are symptoms such as Horner’s syndrome. MR is recommended for its diagnosis with specific angiographic sequences.

Conclusions
A diagnosis of AOM is based on clinical findings, and the majority of cases resolve with antibiotic treatment. Therefore imaging tests are usually not indicated. However, when treatment fails or there is a suspected complication, CT and MR will play an essential role in diagnosis, enabling early administration of suitable treatment. CT because it is rapid and available, is the initial technique of choice and is generally the definitive scan. MR is reserved for assessing the inner ear and for cases where more precise evaluation or radiological control of intracranial complications is necessary. We have carried out a detailed review in this article of the radiological features of AOM and its complications, to provide a guideline for the diagnostic process.

Conflict of Interest
The authors have no conflict of interest to declare.

References
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