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Diagnostic and Imaging Findings in Inflammatory Opacifications of the Middle Ear: A Review of the Literature

Ali Mahdavi*

ABSTRACT

Opacification in the middle ear and mastoid region can stem from a wide range of factors. In terms of diagnostic imaging, CT is the primary tool due to its exceptional spatial resolution, particularly for examining the temporal bone and ossicles. MRI complements this by offering detailed soft tissue lesion characterization and assessing involvement in the inner ear and cranial nerves. This study focuses on inflammatory causes of opacification in the middle ear and mastoid, with an emphasis on the utility of CT and MRI. This comprehensive review aimed to provide a practical framework for considering potential differential diagnoses.

Keywords: Acute otomastoiditis, Otitis, Opacification, Imaging, Radiographic findings.

Department of Radiology, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

***Send correspondence to**
Ali Mahdavi
Department of Radiology, Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran, E-mail: ali-mahdavi@sbmu.ac.ir

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INTRODUCTION

Opacification of the middle ear and mastoid region can result from a diverse range of causes, including inflammatory, neoplastic, vascular, fibro-osseous, and traumatic factors. These changes may manifest locally, but more extensive involvement can extend into the inner ear or even intracranially. The diagnostic implications of these conditions are significant, as they can lead to issues like hearing loss, pain, tinnitus, vertigo, and cranial nerve problems. Careful evaluation, combined with otoscopic examination through the tympanic membrane, is crucial as it can provide valuable hints regarding the underlying cause[1-5]. For instance, the observation of a “blue” hue might suggest a cholesterol granuloma or a dehiscent jugular bulb, a “white” lesion could point to cholesteatoma or schwannoma, and a “red” lesion might indicate a glomus tumor or an aberrant internal carotid artery[6-9].

When it comes to assessment, CT takes the lead as the primary imaging tool. Its outstanding spatial resolution and the ability to create thin sections make it ideal for a thorough examination of opacification in the temporal bone and the ossicles. MRI supplements this by offering better characterization of soft tissue lesions and an excellent assessment of involvement in the inner ear and cranial nerves[10-12]. This study delves into the various inflammatory causes of opacification in the middle ear and mastoid region, with a particular emphasis on the applications of CT and MRI. It aims to provide a practical framework for determining potential differential diagnoses. It’s important to note that throughout this article, the term “opacification” will also encompass abnormal MRI signal intensities.

Acute Otomastoiditis (AOM)

AOM is the most prevalent inflammatory condition affecting the temporal bone. It is characterized by the invasion of bacteria from the nasal and nasopharyngeal regions into the temporal bone, typically following damage to the mucosal lining caused by a viral upper respiratory infection[13]. This condition is particularly common among young children due to factors like enlarged adenoids and the relatively horizontal orientation of the Eustachian tube, which can lead to blockages, air absorption issues, and the leakage of fluid into the middle ear and mastoid. Symptoms of AOM may include fever, ear pain (otalgia), ear discharge (otorrhea), or swelling behind the ear (retro-auricular swelling). In most cases of AOM, imaging is not required. However, when complications are suspected, such as coalescent AOM, imaging becomes necessary[14, 15].

In histological examination, AOM typically reveals the presence of pus and granulation tissue. Culture tests may indicate the growth of bacteria such as Streptococcus pneumoniae or Haemophilus influenzae. When using CT scans, opacification of the tympanic cavity and/or mastoid region is observed, often accompanied by the presence of fluid levels. In more complex cases, AOM can progress to coalescent AOM, which is characterized by structural changes in the mastoid, including mastoid trabeculae and cortical damage. In rare instances, a subperiosteal abscess may develop, leading to a condition known as Bezold abscess, where the infection spreads between the digastric and sternocleidomastoid muscles[16, 17].

MRI scans of AOM typically display high T2-weighted signal material in the tympanic and mastoid areas. Contrast-Enhanced MRI (CE-MRI) is a valuable tool for evaluating complications. It can reveal labyrinthitis through inner ear contrast enhancement, dural venous sinus thrombosis with venous filling defects, extracranial or intracranial abscess formation characterized by rim-enhancing collections, and meningitis with leptomeningeal enhancement. Additionally, the spread to the petrous apex can result in petrous apicitis, a condition that may cause deep facial pain and abducens nerve palsy, a syndrome referred to as Gradenigo’s syndrome. It’s important not to confuse this with the asymptomatic finding of trapped residual fluid within the petrous apex air cells[18-20].

The potential diagnoses to consider in this case comprise cholesteatoma, cholesterol granuloma, and, in pediatric cases, Langerhans’ Cell Histiocytosis (LCH). Notably, cholesteatoma and cholesterol granuloma are typically linked to chronic otitis. In instances of temporal bone LCH, one can expect to observe both bone damage and the presence of soft tissue masses. It is worth mentioning that although osseous destruction may occur in rare cases of sarcoidosis and Wegener’s granulomatosis, these conditions are typically characterized as multi-organ diseases. Another condition to contemplate, albeit infrequent, is pediatric rhabdomyosarcoma, which typically presents as an extracranial soft tissue mass with potential extension into the External Auditory Canal (EAC)[13, 14, 16].

Necrotizing Otitis Externa (NOE)

Necrotizing Otitis Externa, also recognized as malignant otitis externa, is an infrequent yet highly aggressive infection that initiates in the External Auditory Canal (EAC) and extends deeply into the soft tissues, middle ear, mastoid, and skull base. The most common causative agent for this condition is Pseudomonas aeruginosa. Typically, patients affected by NOE are elderly, diabetic, or individuals with compromised immune systems. They usually present with symptoms such as fever, otorrhea[21-23], and severe, relentless otalgia[24]. Notably, Pseudomonas infection can extend from the EAC to the skull base through cartilaginous clefts, potentially resulting in osteomyelitis along with lower cranial nerve impairments, which often signifies a more challenging prognosis[25].

Histological examination of tissues affected by this condition reveals inflammation and granulation tissue, typically found at the junction of the bone and cartilage in the External Auditory Canal (EAC). When cultured, Pseudomonas, a Gram-negative aerobic bacillus, is often identified. This bacterium induces necrotizing vasculitis, which involves the inflammation of blood vessels, leading to thrombosis and necrosis of the surrounding tissue. Computed Tomography (CT) imaging displays signs of inflammation in the auricle and EAC soft tissues, as well as potential erosion of the EAC bone. Additionally, it may reveal opacification and destruction of the tympanic and mastoid regions. Magnetic Resonance Imaging (MRI) demonstrates specific signal characteristics, with T1-hypointense and T2/STIR-hyperintense signals. These MRI findings reflect edematous changes within the bone, indicating the presence of osteomyelitis in the skull base and inflammation in the adjacent soft tissues[25-27].

Osteomyelitis can involve various critical structures such as the stylomastoid foramen, leading to potential facial paralysis. Additionally, it may affect the jugular foramen, leading to lower cranial neuropathies impacting the IX, X, XI cranial nerves. The hypoglossal canal can also be involved, resulting in neuropathy of the XII cranial nerve. Furthermore, the petrous apex may be affected, influencing cranial nerve VI, often with observable cranial nerve MRI contrast enhancement. The condition can also lead to other complications, including the formation of extra- or intracranial abscesses, dural venous sinus thrombosis, or meningitis, which can be detected through Contrast-Enhanced MRI (CE-MRI). In the case of skull-base osteomyelitis, bone scintigraphy and gallium nuclear medicine studies may reveal increased tracer uptake. These imaging methods can also be employed to monitor the response to treatment, depending on factors such as accessibility and cost[25-30].

The potential differential diagnoses to consider in such cases include other medical conditions like squamous cell carcinoma of the External Auditory Canal (EAC), osteoradionecrosis, and more rarely, an inflammatory pseudo-tumor of the temporal bone. EAC squam ous cell carcinoma typically presents with either focal or diffuse soft tissue that enhances on contrast imaging. This condition is often associated with bone destruction and is characterized by the presence of skin ulceration, which may also be accompanied by regional lymph node metastases. Osteoradionecrosis is another condition to consider, which can result in thickening of the soft tissues and bone destruction[31-33]. This may occur months to years following radiotherapy treatment. In rare instances, one might come across an inflammatory pseudo-tumor, which is typically diagnosed through the exclusion of other possibilities. This condition is exceedingly uncommon and is characterized by an osteo-destructive fibroblastic soft tissue mass. On Magnetic Resonance Imaging (MRI), it exhibits low MR T2-weighted signal and demonstrates avid enhancement with contrast[25-30].

Chronic Otomastoiditis (COM)

Chronic Otitis Media is a frequently occurring condition characterized by inflammatory changes in the middle ear, often extending into the mastoid region. This condition typically arises due to problems with the Eustachian tube or as a consequence of Acute Otitis Media (AOM). Patients with COM may present symptoms like conductive hearing loss, otorrhea (ear discharge), tympanic membrane perforation, retraction of the eardrum, and may exhibit scarring when examined using otoscopy[34, 35].

Histological examinations reveal the presence of granulation tissue and mucosal hyperplasia. When viewed through a CT scan, the accumulation of granulation tissue and the long-term effects of inflammation result in opacity of the tympanic membrane. This process may also lead to potential disruption of the ossicular chain, thickening of mastoid trabeculae, increased sclerosis, and obliteration of mastoid cells. In a normal ossicular chain, there is a characteristic structure often referred to as an “ice-cream cone” when seen on axial CT images. This structure comprises the malleus head as the “ice cream” and the short process of the incus as the “cone.” In cases of COM, it’s common to observe thickening and retraction of the tympanic membrane, with potential erosions affecting the “cone,” the long process of the incus, and involvement of fibrous tissues in the incudostapedial joint, which can result in apparent joint widening[34-37].

COM can lead to various complications, such as post-inflammatory ossicular fixation, where fibrous tissue causes fixation, acquired cholesteatoma characterized by additional soft tissue leading to ossicular and/or tympanic wall destruction, and tympanosclerosis, which manifests as ossification within the tympanic membrane. Contrast-Enhanced MRI (CE-MRI) is particularly valuable for evaluating fewer common complications, including labyrinthitis, dural venous sinus thrombosis, the formation of intracranial abscesses, and cases of meningitis[34-39].

When considering differential diagnoses, it’s important to take into account conditions like cholesteatoma or cholesterol granuloma, as they can exhibit similar opacity in imaging. However, there are distinctive features that can help differentiate them. Cholesteatoma, for instance, tends to present a high signal on DWI (diffusion-weighted imaging) MRI. On the other hand, a cholesterol granuloma typically displays a high T1-weighted signal. It’s worth noting that the presence of middle ear and mastoid effusion could also be linked to Eustachian tube blockage, which might be caused by a nasopharyngeal neoplasm[35-37].

Tympanosclerosis

Tympanosclerosis is a long-lasting reactive process characterized by the accumulation of calcified plaques within the tympanic cavity. It is observed in about 30% of adults who have chronic otitis media. Some patients may not experience any symptoms, while others might have conductive hearing loss due to the encasement and fixation of the ossicular chain[2, 40, 41].

This condition is marked by an abnormal deposition of collagen, which leads to the formation of plaques made up of calcium and phosphate crystals. When using imaging techniques, CT scans are the preferred method. They reveal the presence of punctate or web-like calcified densities that do not enhance with contrast. Interestingly, these calcifications may resemble "extra ossicles" and can even extend to involve the tympanic membrane. When the crura of the stapes are affected, they appear unusually prominent, and in cases involving chronic otitis media, there may also be residual debris in the middle ear cavity. If the condition is confined solely to the tympanic membrane, it is referred to as myringosclerosis[2, 42-44].

The possible alternative diagnoses encompass otitis media, cholesteatoma, and, in rare instances, a venous malformation of the facial nerve. Otitis media typically lacks calcification but may exhibit fluid levels. Cholesteatoma, while not characterized by calcification, can lead to erosions in the tympanic region and otic capsule fistulas. On the other hand, a facial nerve venous malformation appears as a contrast-enhancing lesion, typically with a calcified "honeycomb" matrix. It's important to note that the term "tympanosclerosis" should not be confused with "otosclerosis" (otospongiosis), which occurs in the region of the oval window and/or cochlea and does not result in soft-tissue opacification in the tympanic area[2, 45, 46].

Cholesterol granuloma of the middle ear

Cholesterol granuloma is a relatively rare condition characterized by the buildup of blood products and cholesterol, often occurring against the backdrop of Chronic Otitis Media (COM). It is believed to result from persistent inflammation causing a negative pressure build-up, leading to damage to the mucosa and recurrent small-scale bleeding. Typically, patients do not experience pain but may show symptoms such as conductive hearing loss and hemotympanum, which is identifiable by a bluish appearance of the tympanic membrane[47-51].

Histopathological examination typically reveals the presence of cholesterol crystals along with macrophages loaded with hemosiderin and significant granulation tissue. On CT scans, a lesion in the tympanic soft tissue is evident. Larger lesions may cause smooth and expansive scalloping of the adjacent bone and, in some cases, displacement or erosion of ossicles. When examined using MRI, this lesion appears as a non-contrast-enhancing structure with a high signal on T1-weighted and T2-weighted images. Areas of heterogeneity or low T2-weighted signal can occur due to hemosiderin deposition. These MRI characteristics are akin to those observed in cholesterol granulomas located in the petrous apex, although the latter usually display more extensive bone expansion on CT scans. The differential diagnosis should consider conditions like chronic otitis media, cholesteatoma, glomus tympanicum tumor, and post-traumatic changes. Notably, chronic otitis media leads to non-hemorrhagic alterations, cholesteatoma exhibits a hypointense signal on T1-weighted images, and a glomus tympanicum tumor typically demonstrates strong contrast enhancement. In cases of post-traumatic changes, the presence of recent trauma is a clear indicator[47-51].

Cholesteatoma

Cholesteatoma in the middle ear is a non-neoplastic, benign but proliferating lesion characterized by the buildup of keratin debris within a sac formed by stratified squamous epithelium. These cholesteatomas are predominantly acquired (about 98% of cases) and are typically associated with chronic inflammation. In rare instances, they can be congenital, known as "epidermoid" cholesteatomas, originating from abnormal epithelial remnants within the middle ear (making up the remaining 2% of cases). Congenital cholesteatomas are typically found in children or young adults who have an intact tympanic membrane and no history of ear infections. These patients may show no symptoms or may present with hearing loss, ear drainage, or a white, pearly lesion in the anterior superior quadrant of the middle ear when examined with an otoscope[52-54]. Acquired cholesteatomas, on the other hand, are more common in older individuals, often adolescents or adults, who have a history of recurrent ear problems. When observed through otoscopy, these cholesteatomas appear as pearly lesions that may be discolored due to inflammatory changes. Patients with acquired cholesteatoma can experience conductive hearing loss, chronic ear discharge, or episodes of vertigo. The condition can progress to affect the ossicular chain, scutum, lateral semicircular canal, and tegmen tympani. Despite surgical treatment, cholesteatoma recurrence is reported in approximately 20% of cases, and some individuals may experience residual issues, with recurrence rates going as high as 40%[52-54].

Examination of tissue samples reveals the presence of exfoliated keratin within stratified squamous epithelium, accompanied by cholesterol crystals and signs of chronic inflammation such as granulation tissue. The most prevalent form of acquired cholesteatoma is the Pars Flaccida cholesteatoma, which originates in the superior part of the tympanic membrane, specifically in the Pars Flaccida region, and extends into the lateral epitympanic recess or Prussak space. When visualized through CT scans, these cholesteatomas are characterized by the displacement of the malleolar head and body of the incus towards the medial direction and the erosion of neighboring bony structures like the scutum and ossicles[55-57]. Further extension into the epitympanum may result in the erosion of the tegmen tympani, which could potentially pose a risk of intracranial involvement. In cases of posterolateral expansion into the mastoid antrum or the facial canal, these cholesteatomas can cause destruction of the mastoid bone. In contrast, the less common Pars Tensa cholesteatomas occur in the posterior mesotympanum, specifically near the sinus tympani and the facial nerve recess. On CT scans, these cholesteatomas exhibit expansion into the medial epitympanic space, which results in the displacement of ossicles toward the lateral aspect and their initial erosion along the medial edges. This inward extension brings them into contact with the lateral semicircular canal, potentially causing erosion and the formation of a fistula. Mural and External Auditory

Canal (EAC) cholesteatomas represent other infrequent variants of acquired cholesteatoma. Mural cholesteatoma leads to extensive destruction of the middle ear and mastoid, accompanied by EAC wall dehiscence and the extrusion of central matrix. The resulting cavity, lined by a residual layer of cholesteatoma, closely resembles the appearance of a surgical mastoidectomy defect, often referred to as an "auto mastoidectomy"[55-57]. In contrast, EAC cholesteatoma is characterized by a soft-tissue mass in the EAC with accompanying bone erosion of the osseous EAC. In cases of congenital cholesteatoma, CT imaging reveals a relatively smooth and well-defined soft tissue density lesion situated in the anterosuperior quadrant of the middle ear cavity, all while the tympanic membrane remains intact. Although bone erosion can occur, especially affecting structures like the lateral semicircular canal, tegmen tympani, and ossicles, it is notably less frequent than in acquired cholesteatoma[58, 59].

When examined through MRI, cholesteatomas typically appear as lesions with low T1-Weighted (T1W) and intermediate to high T2-Weighted (T2W) signals. In some cases, adjacent trapped secretions may exhibit a higher T2 signal. Contrast-Enhanced MRI (CE-MRI) can reveal rim enhancement, often attributable to granulation or scar tissue[58, 59]. Importantly, cholesteatomas stand out on Diffusion-Weighted Imaging (DWI) due to their high signal resulting from restricted diffusion. As a result, after surgery, DWI can effectively differentiate between residual or recurrent cholesteatomas, which exhibit restricted diffusion, and granulation or scar tissue, which do not. This capability allows for the detection of cholesteatomas smaller than 5 mm using modern MRI DWI techniques, potentially eliminating the need for a second-look surgery in patients with a history of cholesteatoma resection. CE-MRI also proves valuable in assessing rare complications, including labyrinthitis, characterized by inner ear contrast-enhancement, dural venous sinus infiltration, intracranial invasion, meningitis, or intracranial abscesses[52-54].

Distinguishing cholesteatomas from other conditions like Chronic Otitis Media (COM), cholesterol granulomas, or glomus tympanicum tumors in MRI imaging is crucial. Cholesteatomas present as lesions with restricted diffusion, setting them apart from these conditions. Additionally, glomus tympanicum tumors typically have a distinct location around the cochlear promontory and often exhibit strong contrast enhancement, making them distinguishable from cholesteatomas in imaging[52-59].

Temporal bone trauma with fracture

Temporal bone fractures typically result from high-impact incidents like car accidents (accounting for 45% of cases), falls (32%), and assaults (12%). These traumatic events can lead to hemorrhage in the middle ear and mastoid, resulting in various symptoms in predominantly male patients. Common clinical signs include blood in the ear canal (hemotympanum), swelling around the ear (peri-auricular swelling), bruising (ecchymosis), known as the "battle sign," dizziness (vertigo), hearing loss (either conductive or sensorineural), facial nerve dysfunction, leakage of Cerebrospinal Fluid (CSF), and pain. Interestingly, about 20% of individuals with a skull base fracture also experience a concurrent temporal bone fracture[60-62].

CT scans reveal distinct characteristics in cases of temporal bone fractures, including high-density opacification of the tympanic and mastoid regions due to hemorrhage. Other possible findings comprise temporal bone fractures, potentially involving the ossicular chain with fractures or dislocations, air within the labyrinth (known as pneumo-labyrinth), peri-lymphatic fistulas, and collections of gas either within the cranial or extracranial regions. These fractures are traditionally classified concerning the long axis of the petrous bone. Longitudinal fractures, accounting for 90% of cases, are frequently linked with disruption of the ossicular chain, while transverse fractures (30%) often result in pneumo-labyrinth and/or facial nerve damage[63-67]. A newer classification distinguishes otic capsule-violating fractures (20%) from those sparing the otic capsule. In otic capsule-violating fractures, injuries occur to the cochlea, semicircular canals, or vestibule, which increases the likelihood of facial nerve injury, sensorineural hearing loss, and cerebrospinal fluid leakage. More frequently than ossicular fractures, dislocation of the ossicular chain occurs, particularly at the incudostapedial joint. CT angiography is employed to assess vascular complications, including dissection, occlusion, or the formation of pseudoaneurysms in the internal carotid artery (ICA). MRI imaging may depict high T1W and T2W signal due to blood products within the tympanic cavity, hemolabyrinth, or pneumo-labyrinth. These findings can cause a loss of the expected high T2W labyrinthine signal as a result of otic capsule fractures. MRI serves as a means to evaluate acute complications, such as facial nerve injury with contrast-enhancement of the nerve or arterial dissection, often accompanied by high T1W fat-suppressed intramural hematomas[68-71].

A differential diagnosis is essential to distinguish these findings from other conditions that result in hemorrhagic opacification. Although conditions like cholesterol granuloma or otitis can cause similar opacification, the absence of trauma is a clear distinguishing factor. Additionally, it's worth noting that normal skull sutures and fissures, such as the tympano-squamous, petrotympanic, and occipitomastoid sutures, might resemble fractures. However, these anatomical features are typically symmetrical, exhibit sclerosis, and have well-defined cortication, setting them apart from traumatic fractures[60-63].

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