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Acute otomastoiditis and its complications: Role of imaging

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The use of modern antibiotics in the 1940s to treat acute otitis media has significantly reduced the incidence of acute coalescent mastoiditis and otogenic intracranial complications. However, a small proportion (1%-5%) of untreated or inadequately treated patients may experience complications. Computed tomography and magnetic resonance scanning have proven exceedingly valuable in determining the extension of the middle ear and mastoid infections into the neighboring structures, and in particular for the evaluation of otogenic intracranial complications. The objective of this article is to demonstrate the important role of magnetic resonance imaging and computed tomography in the evaluation of the otogenic intracranial complications.

Introduction

The clinical course of acute otitis media is usually short, and the process terminates because of the host's immune system, the infection-resistant properties of the mucosal linings, and the susceptibility of the major organisms (β-hemolytic streptococcus or pneumococcus) to penicillin and other modern antibiotics.[1,2] However, a small proportion (1%-5%) of untreated or inadequately treated patients may experience complications.[1,32] Acute mastoiditis (AM) is a suppurative condition that remains a serious complication of acute suppurative otitis media, despite its dramatic decreased incidence since the introduction of effective antibiotic therapies.[1-32] The use of antibiotics in the 1940s to treat otitis media has significantly reduced the incidence of petrous apicitis.[30] AM is subdivided according to its pathologic stage: (1) incipient mastoiditis, that is, inflammation of the mucosal lining of the air cells and purulent material in the air cells and (2) coalescent mastoiditis, that is, when the inflammatory process destroys the tiny bony walls (bony trabeculae) of the mastoid air cells, resulting in an organized abscess.[1-7]

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AM may spread to its neighboring structures. The most common intracranial complication of AM is meningitis. Additional intracranial complications include subdural empyema, epidural empyema, intraparenchymal brain abscesses, sigmoid or transverse sinus thrombosis, brain infarcts, petrous apicitis (and associated Gradenigo syndrome), and otitic hydrocephalus.[1-32] Extracranially, the list of complications include peripheral facial paralysis, labyrinthitis, labyrinthine fistula, Horner syndrome, subperiosteal abscess, Bezold abscess, osteomyelitis, and cervical or cranial fasciitis.[1-19,21,22,30-32]

Many of these complications can develop quickly, resulting in a significant mortality and morbidity. Imaging plays an important role in the diagnosis and management of these complications. The purpose of this article is to review the state-of-the-art imaging techniques in the identification and follow-up of acute otomastoiditis and its complications[1-3,23-28] and to demonstrate with illustrative examples the important role of magnetic resonance imaging (MRI) and computed tomography (CT) in diagnosing various stages of acute coalescent mastoiditis and its complications.

Imaging techniques

In this section, we briefly review various imaging modalities or techniques that are typically included in the workup of

AM and its complications. A routine temporal bone and head CT is usually pursued in the evaluation of a severely ill patient. Specific signs of AM include high-density material within the middle ear and mastoid air cells with or without air bubbles and air-fluid level. CT can be diagnostic of complications such as meningitis (high-density material in the subarachnoid space, basilar cistern effacement, and leptomeningeal enhancement), herniation, hydrocephalus, subdural effusions (often seen in infants) and empyema, otogenic pneumocephalus, infarction (venous or arterial), and abscess formation.[1,3-27,29]

High-resolution temporal bone CT is crucial for better characterization of the intratemporal findings. This study is typically performed using a state-of-the-art high-resolution multidetector row CT scanner and employing helical, narrow collimation (0.625 mm or less) imaging to yield thin-section images, which can be reconstructed in axial, coronal, and sagittal planes (see article by Chen et al in this issue). Involvement of vital structures, such as the facial nerve canal, ossicles, tegmen tympani, and carotid artery, is best demonstrated on this study.

MRI is superior to CT in visualizing otogenic labyrinthitis and retrocochlear or intracranial abnormalities. Meningitis is best seen on unenhanced or enhanced fluid-attenuated inversion recovery MR pulse sequence, reflecting altered protein content of the cerebrospinal fluid or inflammation of the leptomeninges or both. Contrast-enhanced T1-weighted images are more specific in confirming meningitis and detecting associated parenchymal abnormalities.[6,19] Diffusion-weighted imaging (DWI) is extremely sensitive in the early detection of both arterial infarctions from meningitis-induced vasospasm and sinus venous thrombosis, brain abscesses, and epidural and subdural empyema. MR angiography and MR venography (MRV) are used when indicated to assess for arterial or venous complications, such as vasospasm, Horner syndrome, mycotic aneurysm, and dural sinus thrombosis. Most state-of-the-art MRI techniques for vessel imaging employ noncontrast 3-dimensional (3D) and 2D time-of-flight techniques and multiplanar reconstructions for MR angiography and MRV, respectively.

Illustrative cases

Case 1

A 42-year-old man without otologic history presented with hearing loss of the right ear, purulent otorrhea, otalgia, and dizziness. A CT scan revealed acute coalescent mastoiditis with subperiosteal abscess and erosion into posterior external auditory canal (EAC) wall (Figure 1). There was a dehiscence present in the lateral cortex of the lateral semicircular canal (Figure 1A). There was a subperiosteal abscess over the outer cortex of the mastoid (Figure 1B and C) that was drained at the bedside. An audiogram revealed severe to profound sensorineural hearing loss, and given this and the coalescent mastoiditis, he was considered a candidate for right canal wall down tympanomastoidectomy. At surgery, a 0.5-cm polyp was noted in the posterior EAC wall just lateral to the tympanic membrane. A 1-cm defect through the mastoid cortex was encountered just posterosuperior to the spine of Henle. The mastoid cavity was filled with edematous, inflamed granulation tissue that bled copiously. No cholesteatoma or squamous material was encountered. Minimal frank purulence was noted corresponding to the findings on MRI and DWI (Figure 1C). The ossicles were intact and were not eroded. The incus was removed and the head of the malleus was cut and removed. The manubrium of the malleus and the stapes remained. The anterior drum remnant was mildly edematous but appeared intact. A fascial graft was placed medial to the drum remnant in an underlaying fashion and draped onto the facial recess and the lateral semicircular canal bony defect. No ossicular chain reconstruction was performed given the severity of his sensorineural hearing loss. The facial nerve was identified through the thin bone; the nerve was stimulated at high current levels (0.5 mA) through the bone and the nerve was kept intact. No tegmen dehiscence was noted. Multiple small dehiscences through the posterior EAC wall were noted. Meatoplasty was performed to ensure a wide, open EAC meatus posterior and superior osseous EAC. The patient responded well and was discharged.

Case 2

A 56-year-old woman with a history of active polysubstance drug use and weeks of antecedent left ear pain and fever for 3 weeks presented to a nearby hospital with headache, vomiting, and inability to walk. She was found to have been hypertensive (systolic blood pressure = 192) and tachycardic (heart rate = 126), had a fixed, dilated right pupil, and demonstrated decorticate posture to painful stimulus on physical examination. Initial head CT images showed inflammatory changes in the left middle ear and mastoid air cells, and intracranial findings suggested meningitis and hydrocephalus (Figure 2A). She was transferred to our institution for further management; high-resolution temporal bone CT was pursued and the left otomastoiditis finding was better characterized, which revealed a subtle focal dehiscence of the tegmen tympani (Figure 2B and C). MRI, including MRV, revealed and better characterized several findings (Figure 2D-I).

- Diffuse intracranial leptomeningitis—evident by high signal within the subarachnoid space or leptomeninges on fluid-attenuated inversion recovery images (Figure 2D) and corresponding abnormal enhancement on postcontrast T1-weighted images (Figure 2D).
- Left distal transverse and sigmoid sinus thrombosis—evident on noncontrast MRV and best shown on the 3D reformats (Figure 2G.)
- Small posterior cerebellar venous infarcts—evident only on the diffusion-weighted images, highlighting the