RadioGraphics

Imaging of Complications of Acute Mastoiditis in Children[1]

CME FEATURE

See accompanying test at http://www.rsna.org/education/rg_cme.html

LEARNING OBJECTIVES

After reading this article and taking the test, the reader will be able to:

- List the main neurologic complications in children with acute mastoiditis.
- Identify the main CT features of incipient and coalescent acute mastoiditis as a basis for recommending conservative or surgical management.
- Describe how to achieve early diagnosis of the infrequent severe suppurative complications by using MR imaging and MR angiography.

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Acute mastoiditis is a serious complication of acute otitis media in children. Suppurative disease in the mastoid region occasionally spreads to the adjacent dura mater of the posterior and middle cranial fossae and the sigmoid sinus by means of thrombophlebitis, osseous erosion, or anatomic pathways, producing intracranial complications. Computed tomography (CT) should be performed early in the course of the disease to classify the mastoiditis as incipient or coalescent and to detect intracranial complications. On the basis of the clinical features and imaging findings, the disease is managed conservatively with intravenously administered antibiotics or treated with mastoidectomy and drainage plus antibiotic therapy. CT is therefore a decisive diagnostic tool in determining the type of therapy. In addition, magnetic resonance imaging is performed in patients with clinical symptoms or CT findings suggestive of intracranial complications because of its higher sensitivity for detection of extraaxial fluid collections and associated vascular problems.

Index terms: Children, infections, 214.26 • Ear, inflammation and infection, 214.26 • Temporal bone, abnormalities, 214.26

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Introduction

The middle ear or tympanic cavity is an air-containing space within the temporal bone, which communicates with the nasopharynx via the eustachian tube and with the mastoid air cells via the tympanic antrum. It constitutes an extension of the upper respiratory tract and is subject to viral and bacterial invasion by way of the eustachian tube (1,2). Acute otitis media is the most common localized infectious process occurring in the first 5 years of life due to the relatively horizontal orientation of the eustachian tube and the presence of hypertrophied nasopharyngeal adenoidal tissue. *Streptococcus* (particularly group A β-hemolytic *Streptococcus* and *Streptococcus pneumoniae*) and *Haemophilus influenzae* account for 65%–80% of bacterial cases. The clinical course of acute otitis media is usually short, and the process terminates with activation of the host immune system and the proper use of antibiotics. However, a small proportion (1%–18% in different series) of untreated or inadequately treated patients may experience complications (2,4,5).

Acute mastoiditis occurs mainly in young children and, though less frequent since the introduction of more effective oral antibiotic therapy, is still a serious complication of acute otitis media with potentially life-threatening consequences. It may be the first evidence of ear disease, especially in very young children, with the main symptoms being pain, fever, or otorrhea that persists despite appropriate treatment (5,6). The clinical diagnosis is based on the presence of retroauricular swelling, erythema, or protrusion of the auricle plus evidence of coexistent recent otitis media (7). Extension of the infectious process beyond the mastoid system can lead to a variety of intracranial and extracranial complications, including meningitis; epidural, subdural, and intraparenchymal abscesses; vascular thrombosis; osteomyelitis; and abscesses deep within the neck (Fig 1). The severity of these complications underlines the importance of clinical awareness and early imaging diagnosis to establish suitable treatment (7–10).

This article presents a review of 12 years experience in radiologic study of children with acute mastoiditis referred to our third-level pediatric hospital who had imaging features of associated intracranial complications. In addition, we describe a protocol developed over this time for early imaging classification of the local inflammatory disease and diagnosis of complications.

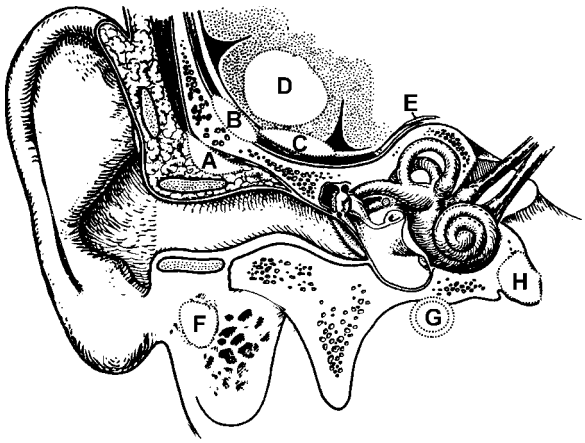


Figure 1. Complications in acute mastoiditis. Extension of the infectious process beyond the mastoid system leads to intracranial and extracranial suppurative complications, including subperiosteal abscess *(A)*, epidural abscess *(B)*, subdural empyema *(C)*, brain abscess *(D)*, meningitis *(E)*, lateral sinus thrombosis *(F)*, carotid artery involvement *(G)*, and apical petrositis *(H)*.

Complications in 75 Patients with Acute Mastoiditis

Complication	No. of Patients
Epidural abscess	8
Sigmoid sinus thrombosis	5
Subdural empyema	1
Carotid artery spasm or arteritis	1
Petrous apicitis	4

Materials and Methods

A retrospective review of the imaging findings and clinical features of 75 children aged 3 months to 15 years with acute mastoiditis was performed. All patients were imaged with temporal bone and cranial computed tomography (CT) as soon as possible, usually within 24 hours after admission, and only those children with suspected intracranial complications were also studied with magnetic resonance (MR) imaging (Table). On the basis of the CT findings alone, a distinction could be made between incipient and coalescent mastoiditis.

Our study protocol in these patients includes temporal bone CT with acquisition of contiguous 1.0-mm-thick axial and reformatted coronal sections, which are obtained slightly below the orbitomeatal line to avoid unnecessary radiation to the eye by using a high-resolution bone algorithm and a low milliampere-seconds value (30–50 mAs). This is followed by acquisition of axial pre- and

a. b.

c.

Figure 2. Acute incipient mastoiditis in a 3-year-old girl with suspected right acute mastoiditis. **(a, b)** Axial CT scans of the temporal bone (**a** obtained inferior to **b**) show increased attenuation of the entire right middle ear with no osseous defects. Note the normal aeration of the tympanum and mastoid cells on the left side. **(c)** Axial contrast material–enhanced CT scan shows no complications.

postcontrast cranial sections for cerebral parenchyma visualization. Sedation of the patient is usually unnecessary with the fast, modern helical CT equipment. MR imaging with angiographic sequences, which always includes contrast material administration, is performed only when CT findings are inconclusive or when intracranial complications are suspected. MR images are usually acquired with a head coil and T1-weighted and turbo T2-weighted sequences in the axial and coronal planes, with two-dimensional or three-dimensional time-of-flight or phase-contrast techniques used for additional MR angiographic sequences.

Discussion

Acute otitis media manifests clinically in children as otalgia, fever, and erythema or edema of the tympanic membrane at otoscopy. Inflammation involves the mucoperiosteum of the middle ear and mastoid cells with resulting fluid collections (serous, mucoid, or purulent). In most patients, diagnostic imaging is not required and the condition resolves with antibiotics (11,12). In acute otitis media, CT shows nonspecific increased attenuation of the middle ear cavity and mastoid cells, sometimes with fluid levels but with preservation of the ossicular chain, trabeculae, and mastoid cortical bone (13).

Nevertheless, when this process is not controlled, the aditus ad antrum can be blocked by the resulting edema and swollen mucosa. This results in accumulation and trapping of secretions in the antrum and air cells and development of acute mastoiditis. With effective therapy, the inflammatory process can be arrested and the mastoid cells can recover their normal appearance. The clinical signs and symptoms of acute mastoiditis mimic those of severe acute otitis media but are longer or recurrent. The presence of middle ear effusion and increased attenuation of the mastoid cells at temporal bone CT with no osseous resorption or periostitis in the proper clinical context is classified as incipient mastoiditis (Fig 2); clinical recovery is usually achieved with antibiotic therapy. The worldwide sensitivity of CT in acute mastoiditis has been reported to be 87%–100% (1,3,13).

Intratemporal and Cervical Complications

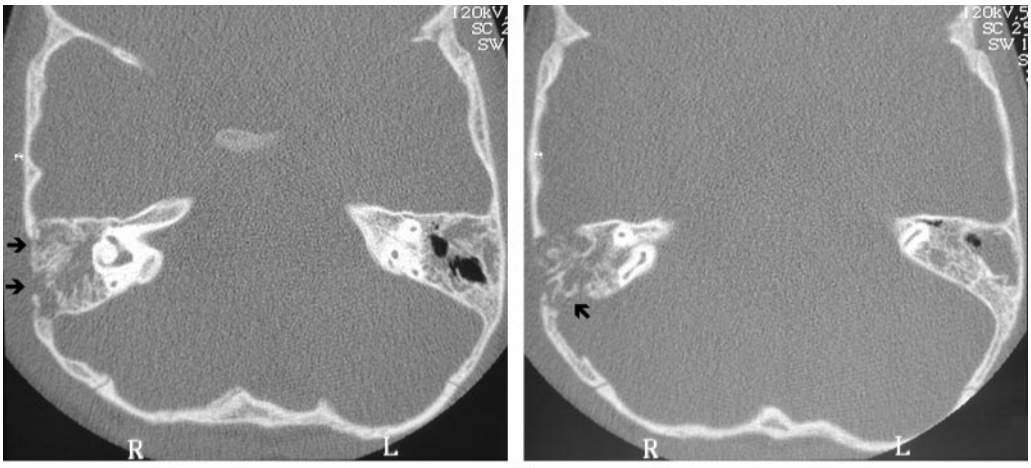
Temporal bone complications of mastoiditis (eg, coalescent mastoiditis, petrous apicitis, labyrinthitis, facial nerve paralysis, hearing loss) are rare today because of prompt antibiotic treatment; however, with the emergence of resistant organisms, the prevalence may be higher in the future. In fact, an increase in the frequency of acute mastoiditis in the pediatric population has been reported in the recent literature and is attributed to changes in microbial flora or to the natural selection of multidrug-resistant organisms (8). Both clinicians and radiologists must have a high index of suspicion so that a critical diagnosis will not be missed.

Coalescent Mastoiditis.—There are four main mechanisms of extension of the infection in acute mastoiditis: preformed pathways, osseous erosion, thrombophlebitis, and hematogenous seeding. When mastoid region inflammation cannot be arrested, the suppuration under pressure causes local acidosis and osseous decalcification, ischemia, and osteoclastic dissolution of the pneumatic cell walls. The pneumatic cells can coalesce into larger cavities filled with purulent exudates and granulations, resulting in empyema and the stage of coalescent mastoiditis. This osteoclastic osseous resorption proceeds in all directions, and intratemporal or intracranial complications threaten to occur before spontaneous resolution (6,13,14). Spread of the inflammatory debris anteriorly to the middle ear via the aditus ad antrum can result in spontaneous resolution if the tympanic membrane was previously perforated. The infection may also spread laterally and produce a subperiosteal abscess or spread medially to the petrous air cells, causing petrositis. Coalescent mastoiditis is diagnosed when temporal bone CT demonstrates erosion of the mastoid septa or mastoid walls (15). This complication can follow a more acute and aggressive course (coalescent acute mastoiditis) or a more subclinical progression (latent or "masked" mastoiditis) (14,16,17).

Latent mastoiditis is an indolent, smoldering temporal bone infection with few clinical clues, owing to the previous use of broad-spectrum antibiotics to treat middle ear disease (18). In classic latent mastoiditis, the tympanic membrane is intact and the middle ear shows no abnormalities at otoscopy, probably related to attic blockade and anaerobic infection. The disease progresses silently until an intracranial complication such as venous dural thrombosis occurs. The diagnosis is often made with CT, which demonstrates the temporal bone disease and the accompanying intracranial complication (14,15).

Management of coalescent mastoiditis is controversial. Options for therapy are antibiotics alone, antibiotics plus myringotomy, incision and drainage of the subperiosteal abscess, and mastoidectomy. Clinical indications for surgery include absence of clinical improvement after 24–48 hours of intravenous antibiotics or evidence of mastoid coalescence at temporal bone CT. Progression of symptoms in a patient in whom conservative management was initially used also warrants surgical intervention. Simple myringotomy can resolve uncomplicated or incipient mastoiditis, but surgical drainage with mastoidectomy is usually necessary to release the mastoid purulence under pressure in coalescent mastoiditis (14). The goal of surgery is therefore effective drainage that eliminates the edema and granulation in the aditus ad antrum and establishment of a good communication between the middle ear and mastoid (17).

Subperiosteal Abscess.—In suspected acute coalescent mastoiditis, radiologists should examine the external mastoid cortex for evidence of osteolysis and subperiosteal abscess (Fig 3). Decalcification of the outer mastoid cortical bone results in an abscess that can extend toward the



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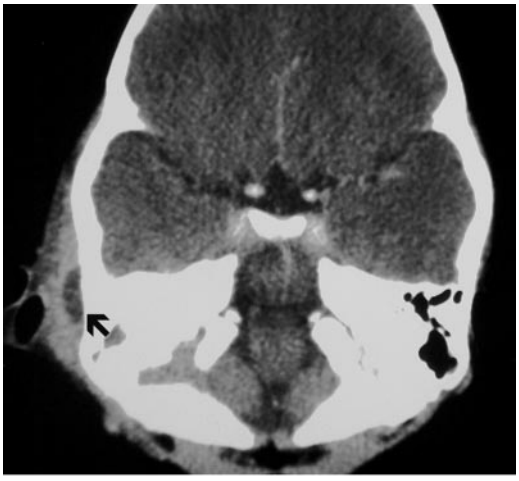

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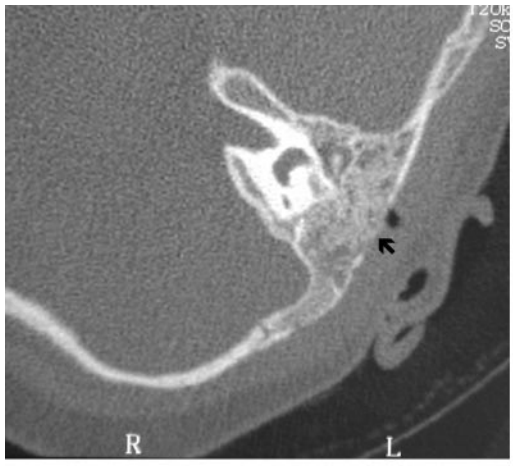
Figure 3. Acute coalescent mastoiditis with a subperiosteal abscess in a 2-year-old girl with right acute otomastoiditis. **(a, b)** Axial CT scans of the temporal bone (**a** obtained inferior to **b**) show abnormal soft-tissue attenuation in the right mastoid air cells with erosion of the external cortex and sigmoid plate (arrows). Abnormal attenuation was also seen on the left side, which was probably due to serous otitis. **(c)** Axial contrast-enhanced cranial CT scan shows a subperiosteal abscess (arrow). No intracranial complications were identified.

external auditory canal, spread along the zygomatic bone, or have a postauricular location (2,6).

Bezold Abscess.—When osteolysis occurs at the mastoid tip, the phlegmonous debris may extend inferiorly into the soft tissues of the neck and form an abscess referred to as the *Bezold abscess* (19). Aeration of the mastoid bone with resulting thinning of the osseous walls is believed to be a predisposing factor for developing this abscess. Incomplete mastoid pneumatization in infancy and early childhood is responsible for the rare appearance of a Bezold abscess, with only five cases reported in children under 5 years of age (20).

Perisinus Abscess.—When osteolysis occurs in the internal mastoid cortex, there is direct apposition of the inflammatory debris to the dura over the sigmoid sinus with subsequent formation of a perisinus and epidural abscess (Fig 4). A retrospective review of patients with coalescent mastoiditis was performed by Antonelli et al (15); they concluded that erosion of the cortical plate overlying the sigmoid sinus was the most sensitive and specific CT finding for distinguishing coalescent from incipient acute mastoiditis.

Figure 4. Perisinus abscess in an 18-month-old girl with left acute mastoiditis. **(a, b)** Axial CT scans of the temporal bone show increased attenuation of the left mastoid air cells with subtle erosion of the mastoid cortex (arrow in **a**) and superior sigmoid plate (arrow in **b**). **(c, d)** Axial unenhanced **(c)** and contrast-enhanced **(d)** cranial CT scans show an unsuspected small epidural abscess (arrow). The ipsilateral sigmoid sinus *(S)* demonstrated normal enhancement on CT scans and normal flow void on T2-weighted MR images. **(e)** Posterior coronal MR venogram shows slightly diminished venous flow with decreased size of the left sigmoid sinus.



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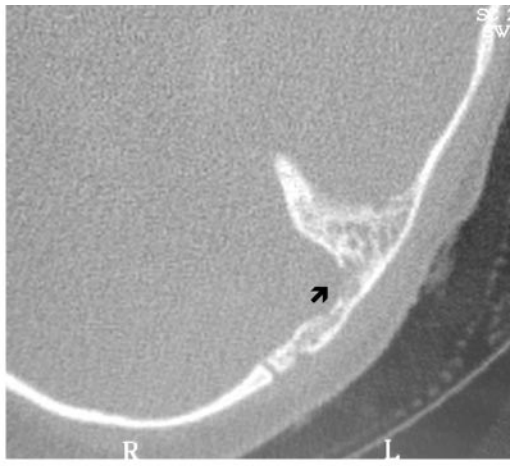

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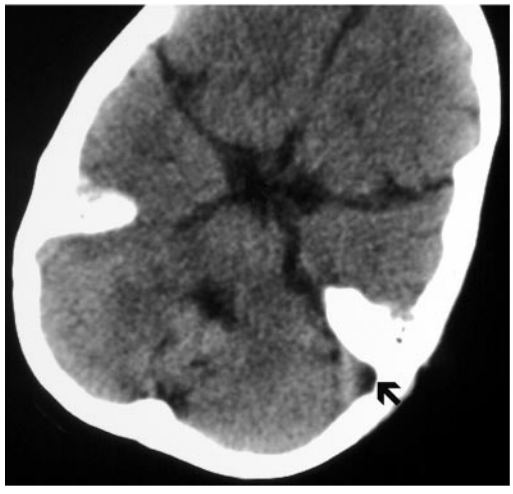

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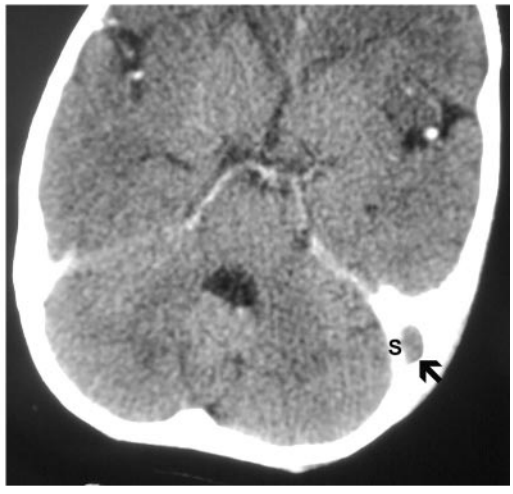

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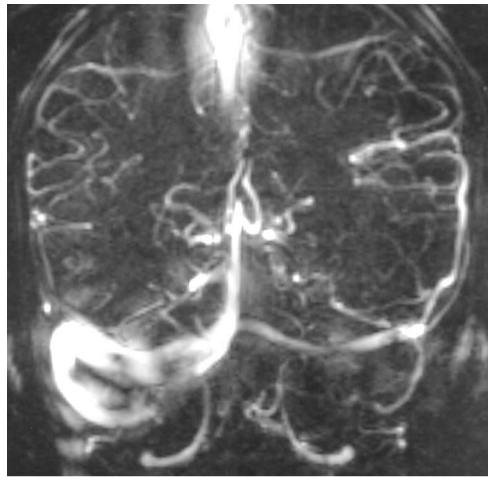

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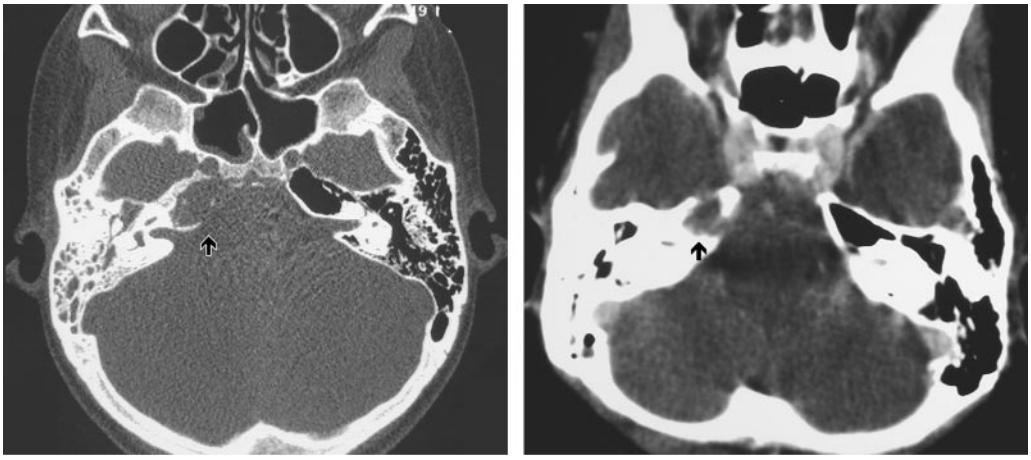
Figure 5. Petrous apicitis in a 7-year-old girl with fever, right-sided facial pain, and diplopia. **(a)** Axial CT scan of the temporal bone shows increased attenuation of the mastoid air cells and erosion of the right petrous apex (arrow) with a well-pneumatized left petrous apex. **(b)** Axial contrast-enhanced CT scan obtained with the soft-tissue algorithm shows a hypoattenuating area (arrow) without a significant enhancing soft-tissue mass. **(c, d)** Axial T1-weighted **(c)** and T2-weighted **(d)** MR images show a lesion (arrow) with low **(c)** and high **(d)** signal intensity. The patient was treated with mastoid drainage and intravenous antibiotics. **(e)** Follow-up axial CT scan shows postmastoidectomy changes and progressive reossification of the right petrous apex.



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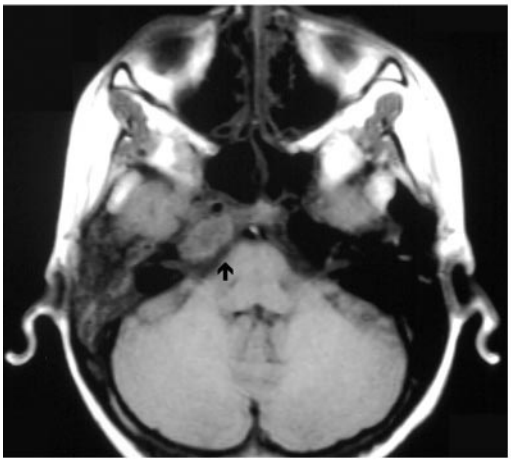

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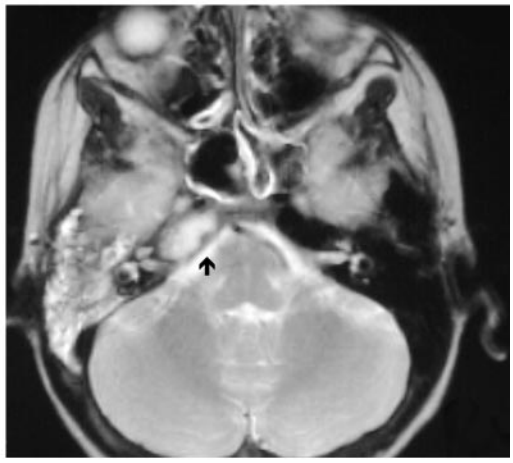

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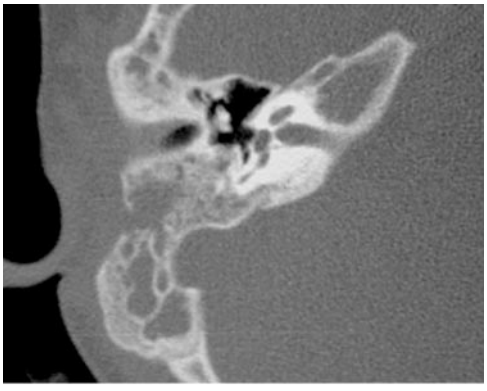

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Petrositis.—Petrositis or petrous apicitis is a rare complication of otomastoiditis that theoretically occurs only in individuals with a pneumatized petrous apex (30% of the population), although this hypothesis is controversial. There is a classic triad of sixth nerve palsy, deep facial pain, and ipsilateral otorrhea, which is referred to as *Gradenigo syndrome* (21), although it may not be present in every patient. The facial pain is due to focal meningitis over the petrous apex with irritation of the gasserian ganglion in the Meckel cavity. Abducens nerve involvement occurs at its course through the Dorello canal (22,23). Imaging studies demonstrate erosive changes of the petrous apex with abnormal enhancement of the adjacent meninges (Fig 5). Differential diagnosis

should be performed with neoplastic disease (rhabdomyosarcoma, metastasis) and epidermoid tumors.

Other Complications.—Labyrinthitis, facial nerve paralysis, and hearing loss are other complications that can be seen in acute mastoiditis, although they are more common in chronic otitis media and cholesteatoma. Infection can spread into the labyrinth via preformed pathways, such as the round or oval window, or by direct invasion of the bony labyrinth, resulting in the uncommon suppurative labyrinthitis. The clinical diagnosis should be suspected when a patient with an infected ear develops vertigo, nystagmus, and hearing loss. The final diagnosis can be made with MR imaging demonstration of abnormal labyrinthine enhancement (2,14). Facial palsy is rare and most commonly affects the tympanic and upper mastoid segments; in cases of acute otomastoiditis, the lesion is often not destructive and reversible.

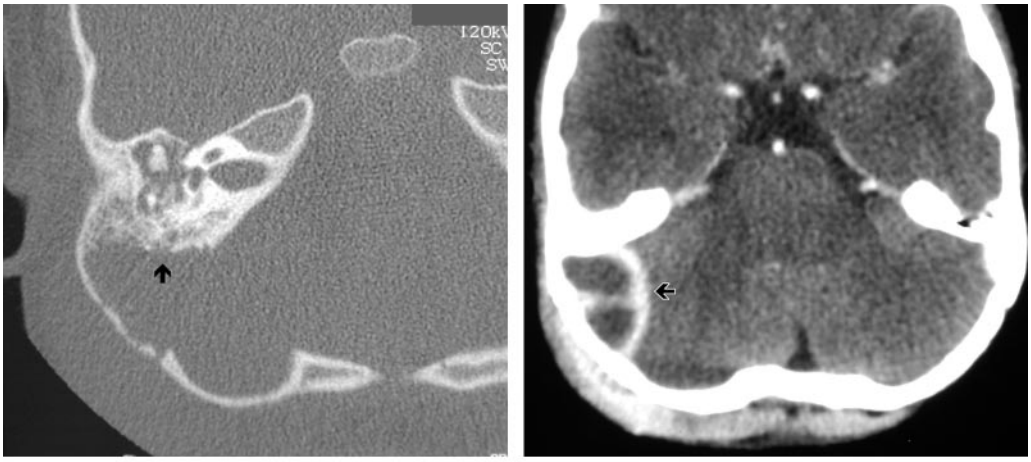
Intracranial Complications

Since the introduction of antibiotic therapy, the percentage of intracranial complications from otitic disease has decreased. However, a high index of suspicion and early diagnosis are essential because of the mortality associated with these complications. The most common early symptoms are increased otorrhea, fever, and headache, with later altered mental status, cranial nerve palsies, and nuchal rigidity (17,24).

Epidural Abscess.—The most common intracranial complication arising from middle ear infection, epidural abscess, usually results from spread by contiguity following bone destruction in coalescent mastoiditis (7). Although it can occur in the middle cranial fossa, the posterior fossa location is far more common due to osseous destruction in the Trautmann triangle over the sigmoid sinus plate or in the posterior cortex of the petrous pyramid (1,2). In keeping with previous reported cases (7), our experience has shown that these perisinus abscesses can be clinically silent and CT is mandatory for disclosing them (Fig 6). Early diagnosis and treatment prevent progression to more serious complications (9).

Dural Venous Thrombophlebitis.—Dural venous thrombophlebitis is known to result from an extradural abscess in more than half of all cases. The epidural abscess may lead to sigmoid sinus thrombosis as a protective mechanism in an attempt to localize the infection. The thrombus may cause propagation to the jugular vein, to other dural sinuses, or through emissary veins to the subcutaneous tissue. Less common mechanisms for dural thrombosis may be osteothrombophlebitis with an intact osseous plate (6,12). These complications may be difficult to diagnose on the basis of the clinical picture alone. They can be completely asymptomatic or present as signs of toxemia, intracranial hypertension, or hydrocephalus.

Indirect imaging signs of dural venous thrombosis include low attenuation of the sinus on unenhanced and contrast-enhanced CT scans, absence of normal flow void on spin-echo MR images, and absence of flow-related enhancement



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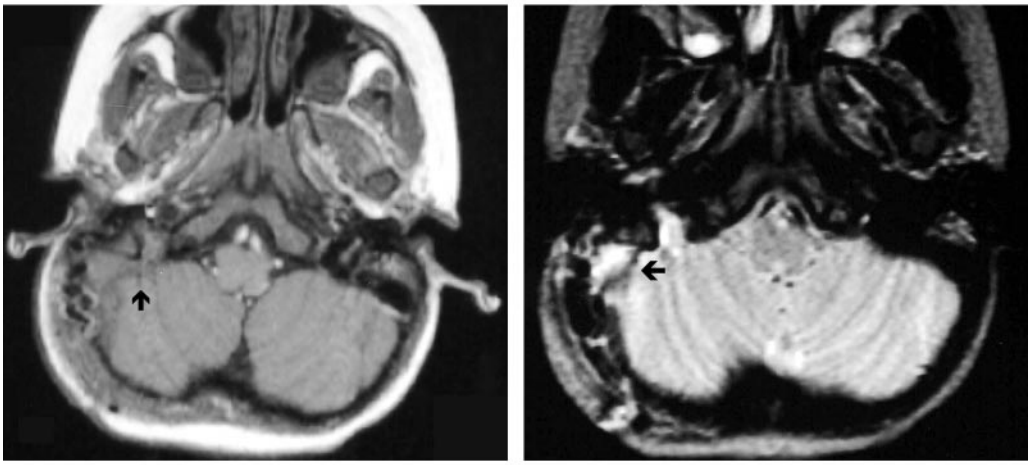

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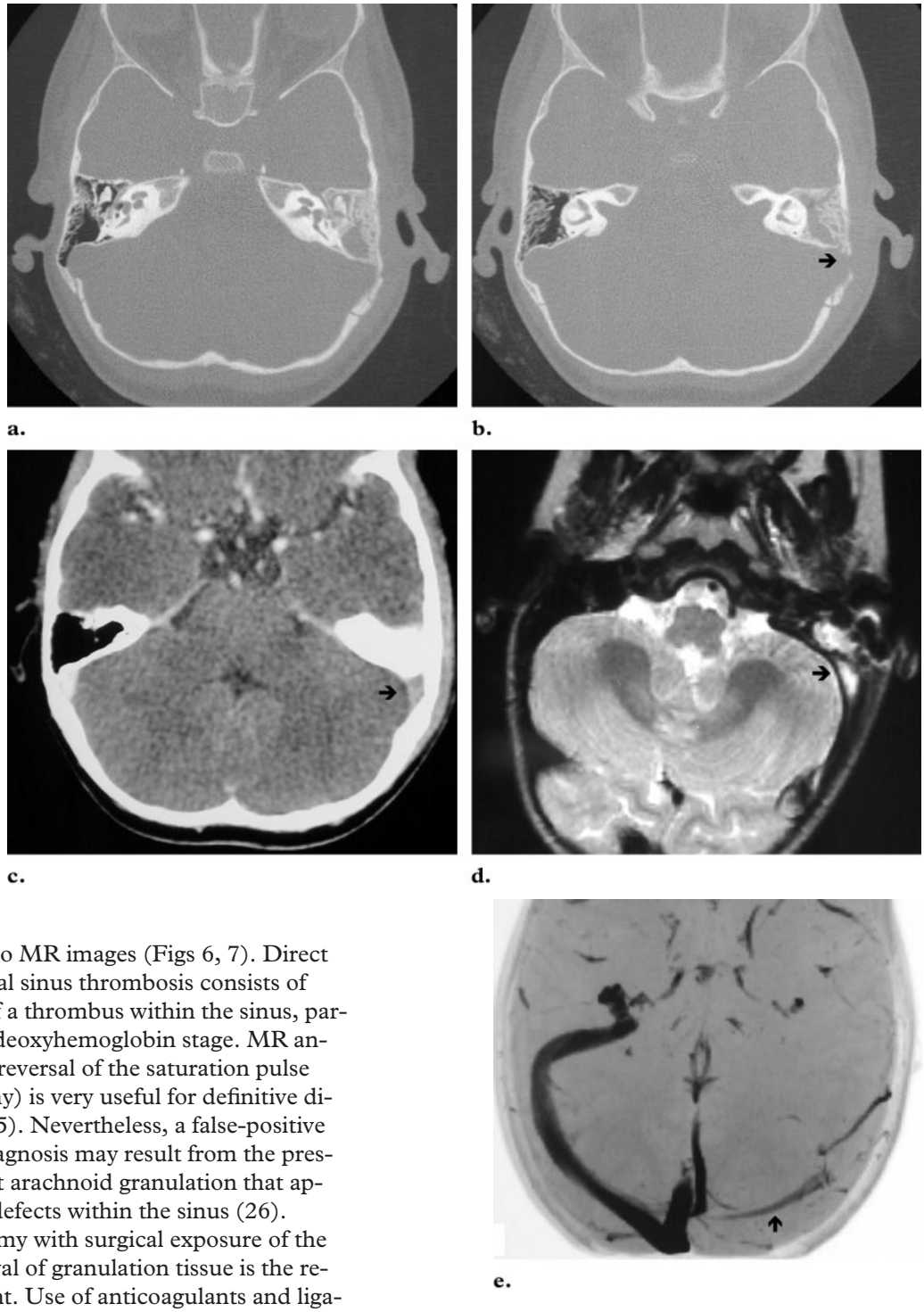
Figure 6. Epidural abscess with sigmoid sinus phlebothrombosis in a 3-year-old girl with acute right mastoiditis but no neurologic symptoms. **(a)** Axial CT scan of the temporal bone shows increased attenuation of the right middle ear with a large osseous defect over the sigmoid sinus plate (arrow). **(b)** Axial contrast-enhanced CT scan shows a large epidural fluid collection (arrow) with some septa. Mastoidectomy with surgical drainage was performed, and intravenous antibiotic therapy was started. MR imaging was subsequently performed. **(c–e)** Axial T1-weighted **(c)** and T2-weighted **(d)** MR images and coronal MR venogram **(e)** show absence of flow (arrow) in the right sigmoid sinus.

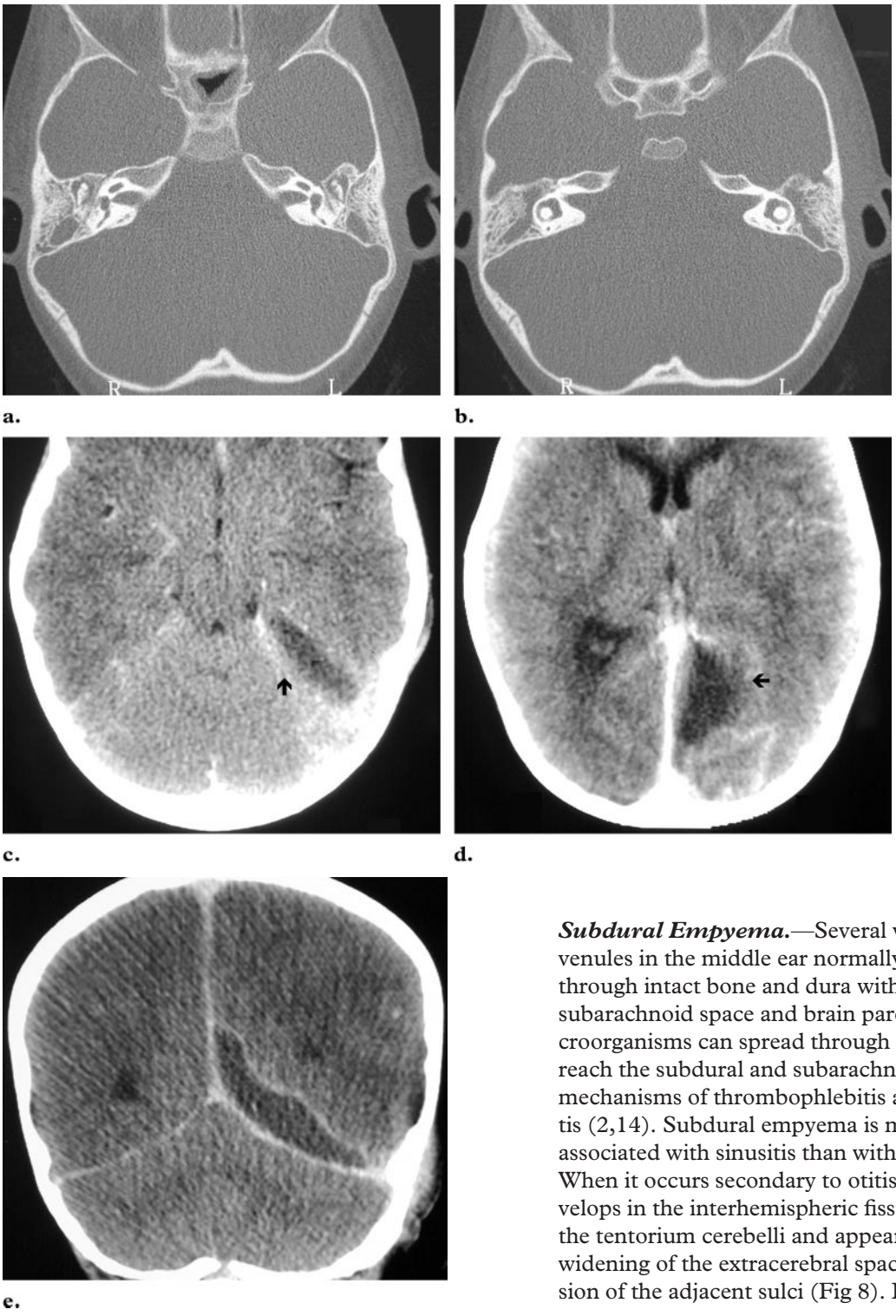
Figure 7. Dural venous phlebothrombosis in a 4-year-old girl with left acute mastoiditis. **(a, b)** Axial CT scans of the temporal bone (**a** obtained inferior to **b**) show increased attenuation of the left mastoid air cells with a clear defect in the posterior and lateral mastoid cortex (arrow). **(c)** Axial contrast-enhanced cranial CT scan shows asymmetric enhancement of the sigmoid sinus (arrow). **(d)** Axial T2-weighted MR image shows hyperintense material in the left mastoid cells with a hyperintense thrombus at the level of the sigmoid sinus (arrow). **(e)** Axial MR venogram shows absence of normal venous flow with occlusion at the level of the mid transverse sinus on the left side and some evidence of adjacent collateralization (arrow).



on gradient-echo MR images (Figs 6, 7). Direct evidence of dural sinus thrombosis consists of identification of a thrombus within the sinus, particularly in the deoxyhemoglobin stage. MR angiography with reversal of the saturation pulse (MR venography) is very useful for definitive diagnosis (2,14,25). Nevertheless, a false-positive MR imaging diagnosis may result from the presence of aberrant arachnoid granulation that appears as filling defects within the sinus (26).

Mastoidectomy with surgical exposure of the dura and removal of granulation tissue is the required treatment. Use of anticoagulants and ligation of the internal jugular vein are controversial alternatives, particularly when there are septic emboli. Early antibiotic therapy will control this

Figure 8. Subdural empyema in a 3-year-old girl with left acute otomastoiditis who developed lethargy, stiff neck, and headache. **(a, b)** Axial high-resolution CT scans (**a** obtained inferior to **b**) show bilateral middle ear disease with no evidence of osseous erosion. **(c, d)** Axial contrast-enhanced cranial CT scans show fluid collections (arrow) extending along the left tentorium cerebelli **(c)** and posterior interhemispheric fissure **(d)**. **(e)** Direct coronal contrast-enhanced CT scan shows continuity of both fluid collections as well as subperiosteal inflammatory soft tissue on the left side.



Subdural Empyema.—Several veins and venules in the middle ear normally communicate through intact bone and dura with vessels in the subarachnoid space and brain parenchyma. Microorganisms can spread through these veins and reach the subdural and subarachnoid spaces by mechanisms of thrombophlebitis and periphlebitis (2,14). Subdural empyema is more commonly associated with sinusitis than with otitis media. When it occurs secondary to otitis, it usually develops in the interhemispheric fissure and along the tentorium cerebelli and appears at imaging as widening of the extracerebral space with compression of the adjacent sulci (Fig 8). Loculation

sinus thrombophlebitis, although the impaired venous drainage may lead to hydrocephalus in severe cases.

Figure 9. Carotid artery involvement in an 18-month-old boy with acute left otomastoiditis but no neurologic symptoms. **(a)** Axial CT scan of the temporal bone obtained at admission shows mastoid disease without evidence of complications. Cranial CT also showed no evidence of complications. Owing to persistent fever and otorrhea, a second CT examination was performed 3 days later. **(b)** Axial CT scan shows abnormal enhancement of the left sigmoid sinus (arrow). MR imaging was performed the same day. **(c)** Axial MR venogram shows thrombosis of the left lateral and sigmoid sinuses. **(d)** Coronal gadolinium-enhanced T1-weighted MR image shows associated pachymeningeal enhancement. **(e, f)** Axial gadolinium-enhanced T1-weighted MR image **(e)** and coronal MR angiogram **(f)** show unsuspected markedly decreased flow in the left internal carotid artery. The patient was quickly treated with surgical mastoid drainage and broad-spectrum antibiotics. **(g, h)** Follow-up coronal MR images obtained 1 week **(g)** and 2 weeks **(h)** later show slow recovery of normal flow in the left carotid artery.

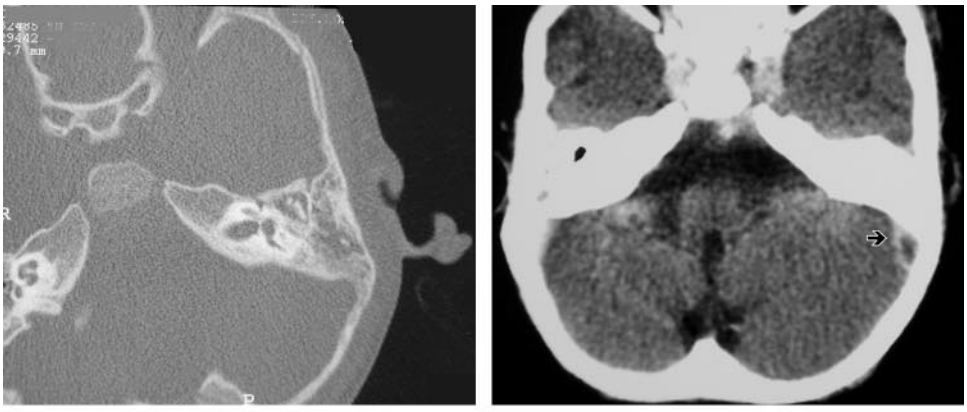
within the collection is not uncommon. Because surrounding vasogenic edema does not occur in association with empyema, the presence of abnormal attenuation or signal intensity in the adjacent parenchyma is usually the result of associated cerebritis. In contrast to epidural abscess, subdural empyema never manifests as a clinically silent complication; affected children usually have meningismus, focal neurologic findings, or focal seizures. It is always a very serious complication that requires prompt neurosurgical drainage (27).

Carotid Artery Involvement.—Infections such as cervical abscess or otomastoiditis affecting the carotid artery have become exceptional in the era of antibiotics but still constitute a potentially life-threatening complication. The internal carotid artery is more likely to be involved than the external and common carotid arteries; this selectivity might be due to the large number of lymph nodes adhering to the internal carotid artery. In our patient, spread by contiguity from the adjacent jugular vein was considered the most likely mechanism of infection (Fig 9). Several clinical findings are suggestive of arterial complications, such as a protracted clinical course; recurrent hemorrhage from the throat, nose, or ear; and Horner syndrome. Nevertheless, the diagnosis is often made at an advanced stage, when the patient develops hemorrhage, pseudoaneurysm, or acute hemiplegia. Imaging studies, particularly MR imaging combined with MR angiography, can show the earlier features of carotid spasm or arteritis, which should be treated with emergency drainage before complete carotid occlusion or rupture occurs (28).

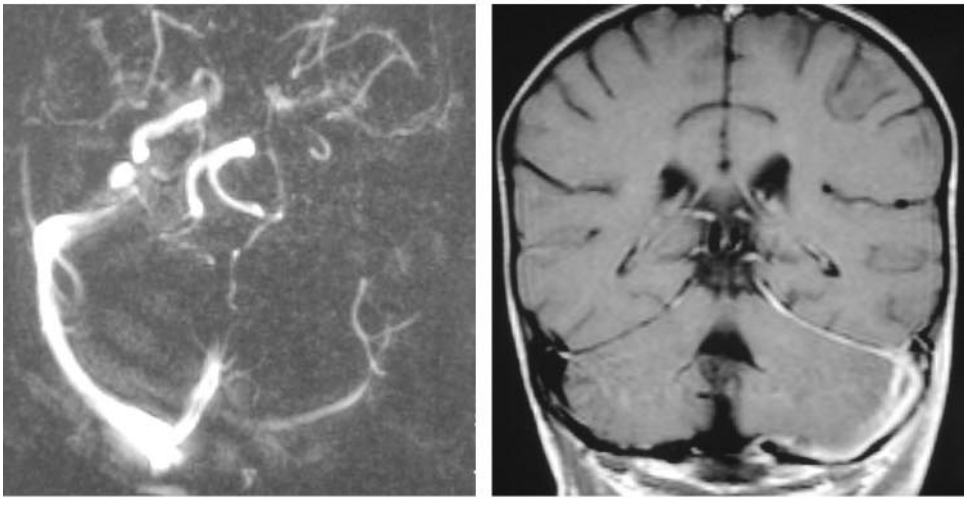
Other Intracranial Complications.—Meningitis, hydrocephalus, encephalitis, and parenchymal cerebral or cerebellar abscesses have also been reported as complications of acute otitis media and mastoiditis. Most cases of meningitis occur in young children by hematogenous dissemination. Cranial CT is usually performed prior to lumbar puncture and cerebrospinal fluid analysis to rule out signs of increased intracranial pressure; radiologists should look for middle ear effusion, particularly if there is a recent history of ear infection. Invasive microorganisms such as *H influenzae* type B are common causal agents, and neurologic sequelae with mental retardation or deafness may occur (16,17).

Conclusions

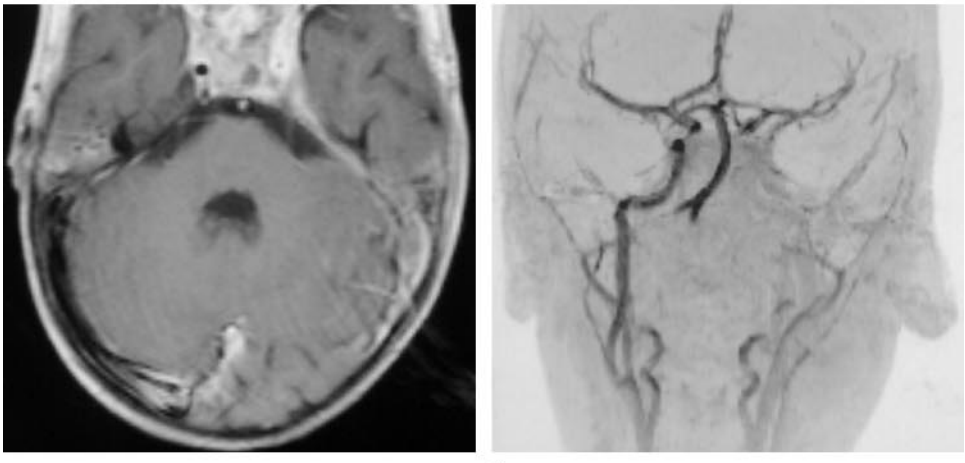
The prevalence of intracranial complications of acute mastoiditis in children has decreased significantly since the introduction of antibiotics; however, this clinical problem persists. These severe complications can be very difficult to diagnose because antibiotic use can mask important clinical symptoms. Early imaging diagnosis enables pediatric physicians to institute prompt, adequate therapy and prevent life-threatening consequences. Therefore, CT should be performed as early as possible in all children with suspected acute mastoiditis, followed by cranial MR imaging if an intracranial complication is suspected. This imaging approach is of great help in planning effective surgical treatment.



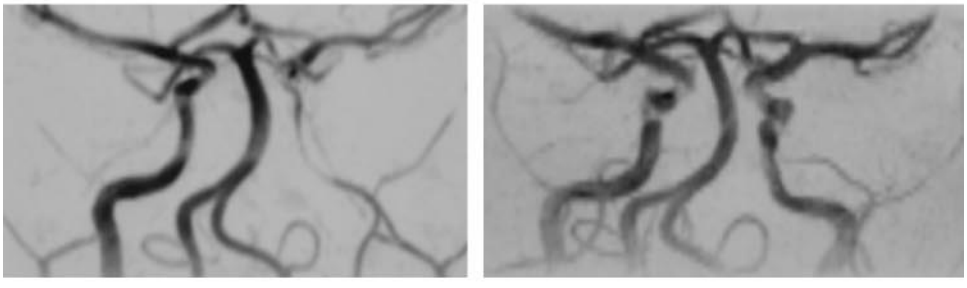
a. b.



c. d.



e. f.



g. h.

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