Chapter 120 – Intracranial Complications of Otitis Media

William A. Wood,
Yael Raz

Complications from acute otitis media or chronic otitis media can be divided into extracranial complications (i.e., facial nerve paralysis, labyrinthitis, subperiosteal abscess) and intracranial complications. Intracranial complications of otitis media include meningitis, epidural abscess, subdural abscess (empyema), brain abscess, lateral sinus thrombophlebitis/sigmoid sinus thrombosis, otitic hydrocephalus, and Gradenigo's syndrome (Fig. 120-1). On occasion, an intracranial complication may be the initial symptom in a patient with a history of untreated otitis media. Multiple complications may coexist. Despite broad-spectrum antibiotics, complications of otitis media can result in high morbidity and even mortality, and surgical management is often necessary.

Spread of otitis media to intracranial structures may occur either directly or hematogenously. Direct extension can occur via multiple mechanisms:[1] preexisting congenital abnormalities such as a Mondini deformity, post–traumatic bony defects from temporal bone fracture, erosion of bony barriers as a result of cholesteatoma, normal communicating vein pathways from the mastoid to the intracranial space, and spontaneous or iatrogenic dehiscence of bone.[2] Meningitis, historically the most common intracranial complication of otitis media, frequently spreads in children via a hematogenous route or by extension from a local abscess.[3] With widespread *Haemophilus influenzae* type B and antipneumococcal vaccination of children, the incidence of meningitis as a complication of these two organisms has become very low.[1] Some recent series report a greater frequency of lateral sinus thrombophlebitis/sigmoid sinus thrombosis or brain abscess than meningitis. The frequency of various intracranial complications in a number of case series over the last decade is presented in Table 120-1.[4–17]

Patients may initially have the classic signs of otogenic infection, such as foul–smelling otorrhea, otalgia, headache, fever, vertigo, and sudden hearing loss. As the disease progresses, later signs include mental status changes such as confusion, obtundation or seizures, and cranial nerve palsies. Many patients with intracranial complications will have multiple complications, such as meningitis in addition to an abscess, and the otolaryngologist should seek to diagnose and treat all of these complications. Patients may have concurrent intratemporal complications such as subperiosteal abscess, labyrinthitis, and facial nerve paresis.

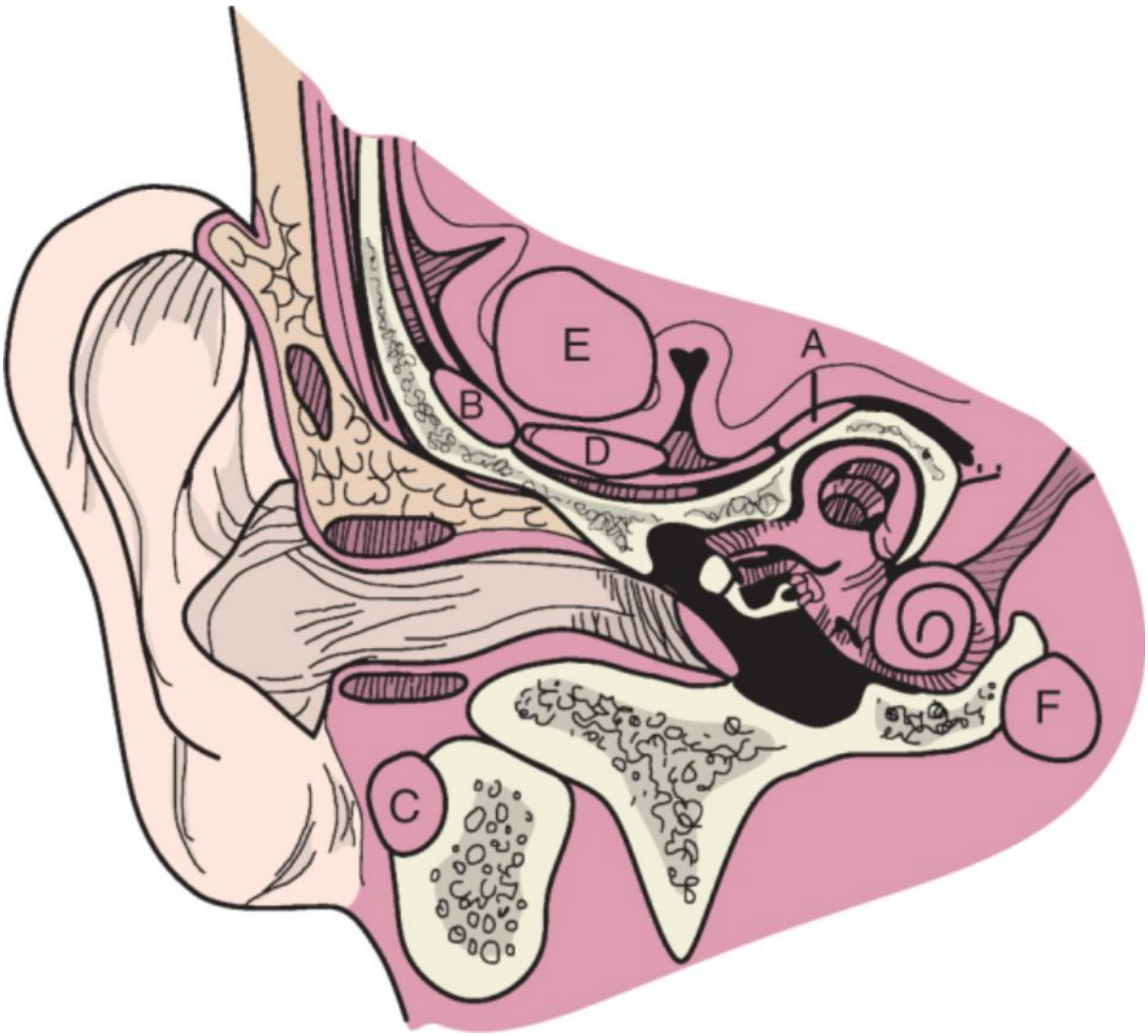

Figure 120-1 Intracranial complications of otitis media include meningitis (A), epidural abscess (B), lateral sinus thrombosis (C), subdural empyema (D), brain abscess (E), and petrous apicitis (F).
(Adapted from Vazquez E, Castellote A, Piqueras J, et al: Imaging of complications of acute mastoiditis in children. Radiographics 23:359-372, 2003.)

Table 120-1 -- RECENT REPORTED SERIES OF INTRACRANIAL COMPLICATIONS OF OTITIS MEDIA IN CHILDREN, ADULTS, AND COMBINED GROUPS OF CHILDREN AND ADULTS

Reference and Total Number of Patients	Patients with ICCOM	Meningitis	Epidural Abscess	Subdural Abscess/Empyema	Brain Abscess	Lateral Sinus Thrombophlebitis or Sigmoid Sinus Thrombosis	Otitic Hydrocephalus	Other[*]
Pediatric Series								
Zanetti,[4] 2006, *N* = 45	13	6	3	0	1	7	0	2
Migirov,[5] 2005, *N* = 28 (11 children)	11	5	3	0	0	1	0	2
Leskinen,[6] 2004, *N* = 33	1	1	1	0	0	0	0	0
Luntz,[7] 2001, *N* = 223 (214 children)	19	7		1	1	6	0	5
Go,[8] 2000, *N* = 118	8	1	4	6	0	0	0	0

Reference and Total Number of Patients	Patients with ICCOM	Meningitis	Epidural Abscess	Subdural Abscess/Empyema	Brain Abscess	Lateral Sinus Thrombophlebitis or Sigmoid Sinus Thrombosis	Otitic Hydrocephalus	Other[*]
Kaftan,[9] 2000, *N* = 22 (3 of 22 <18 yr old)	3	3	0	0	0	0	0	0
Schwager,[10] 1997, *N* = 124	11 (4 patients had >1 ICCOM)	5	5	0	2	5	0	0
Adult Series								
Leskinen,[11] 2005, *N* = 50	9	4 (1 death)	0	0	4	1	0	0
Migirov,[5] 2005, *N* = 28 (17 adults)	17	8	2	2	5	2 (1 transverse, 1 sigmoid)	0	0
Kaftan,[9] 2000, *N* = 22 (19 of 22 > 18 yr old)	19	12	0	1	5	0	0	1 (Gradenigo's syndrome)
Barry,[12] 1999, *N* = 79 with otogenic meningitis	79	79	4	2	4	2	2	8
Series with Combined Children and Adults; Results Not Separated								
Seven,[13] 2005, *N* = 32 with 59 ICCOMs	32	7	7	2	14	10	1	11
Penido,[14] 2005, *N* = 33	33	21	1	2	26 (3 deaths)	5	0	1
Matanda,[15] 2005, *N* = 343 (215 patients <20 yr old)	24	12	0	0	9	2	1	0
Goldstein,[16] 2000, *N* = 100 intratemporal complications of otitis media	16	2	7		1	5	7	2
Osma,[17] 2000, *N* = 93, 57 with ICCOMs (58% <20 yr old)	57	41	4	0	10	1	0	

* Other includes cavernous sinus thrombosis, perisinus abscess, meningocele, petrous apex suppuration, internal jugular thrombosis, cerebritis, presuppurative encephalitis, and ventriculitis. Some patients had more than one intracranial complication. ICCOM, intracranial complication of otitis media.

MENINGITIS

INDICATIONS AND PATIENT SELECTION

Patients with meningitis may have the classic signs and symptoms, such as headache, photophobia, spiking fevers, mental status changes, nausea, and vomiting. Physical signs that have been classically described for evaluating

meningitis, such as Kernig's sign, Brudzinski's sign, and nuchal rigidity, were recently shown in a prospective study of patients suspected of having meningitis to have sensitivities of only 5% to 30%,[18] and hence clinical suspicion should remain high even when these signs are negative on examination. An early lumbar puncture is essential in suspected meningitis, followed immediately by empirical antibiotic treatment, pending culture results. In addition to opening pressure, cell counts, Gram stain, and culture, a cerebrospinal fluid (CSF)–serum glucose ratio of less than 0.5 is abnormal and greatly raises suspicion of meningitis.[19] Current practice guidelines for suspected bacterial meningitis recommend computed tomography (CT) before lumbar puncture for patients in the following categories: immunocompromised state, history of central nervous system disease (e.g., stroke), new–onset seizure, papilledema, abnormal level of consciousness, or focal neurologic deficit.[20]

Fine-cut CT of the temporal bone, with and without contrast enhancement, along with imaging of the brain allows evaluation of related intracranial complications such as a brain abscess, as well as detection of bone defects in the middle fossa or posterior fossa plate and other structural abnormalities. When otitis media is diagnosed or suspected, the otolaryngologist should perform a myringotomy and send the aspirate for immediate Gram stain, in addition to culture and sensitivity testing; a ventilation tube is placed. Antibiotics should then be adjusted to the results of culture of the middle ear aspirate and CSF. If the patient can tolerate magnetic resonance imaging (MRI), it should be performed because it has greater sensitivity for evaluating the extent and type of intracranial infection.[21]

SURGICAL TECHNIQUE

Aside from myringotomy and tube insertion, meningitis alone does not require additional surgical treatment. However, if the patient fails to respond adequately to systemic therapy, mastoidectomy should be considered when the patient is neurologically stable enough to tolerate the procedure, which may reveal a chronic underlying cause (Fig. 120-2). Hearing should be monitored closely. Possible sequelae include labyrinthitis ossificans with resultant sensorineural hearing loss, in which case early cochlear implantation should be considered.

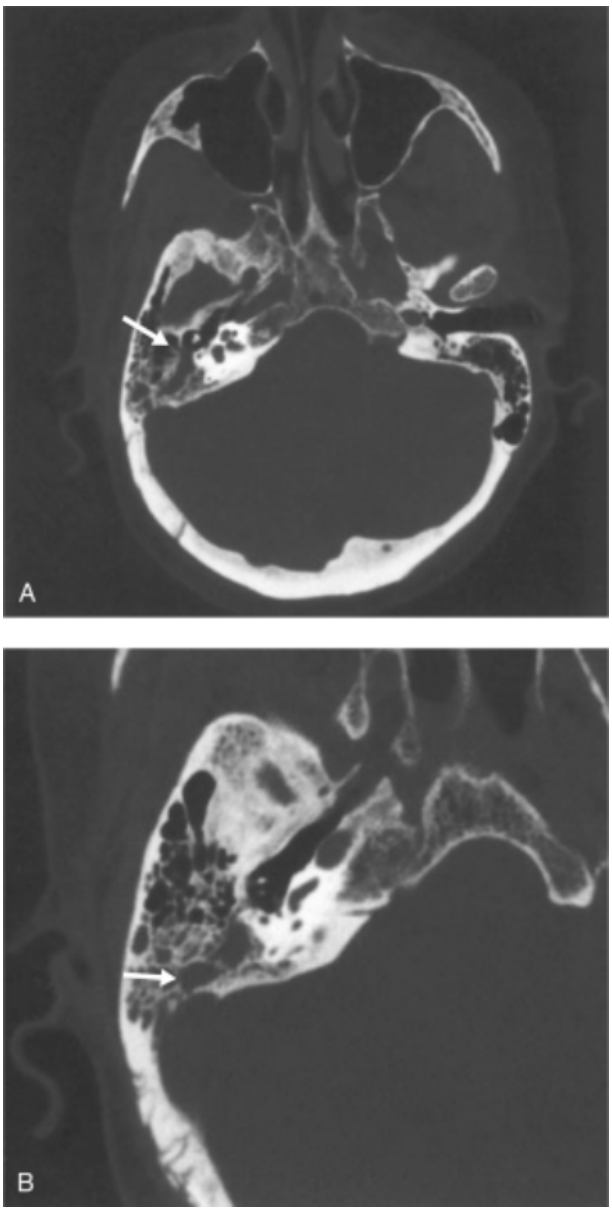

Figure 120-2 Chronic mastoiditis in a patient with meningitis. **A,** Antral block *(arrow),* chronic mastoiditis. **B,** Erosion of the posterior fossa plate overlying the sigmoid sinus *(arrow)*. At the time of the scan, the patient was asymptomatic. She had severe cardiac risk and was observed without surgical intervention. However, 6 months later the patient was evaluated for otalgia, fever, and mental status changes. Workup revealed meningitis. A mastoidectomy was performed because of inadequate response to antibiotics. The antral block was found to be secondary to a cholesterol granuloma.