Intracranial Complications of Otitis Media

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Introduction

Complications of suppurative otitis media occur when the natural defense barriers of the middle ear cleft are overcome. Complications should be distinguished from sequelae of otitis media, which occurs as a direct result of the disease process. Sequelae do not present as an acute medical or surgical emergency. Sequelae are limited to the mucoperiosteum lining of the middle ear. An example of a sequelae is tympanosclerosis. Complications occur when the disease process extends beyond the mucoperiosteum of the middle ear. An example is meningitis, sinus thrombosis, etc. These need to be death with swiftly, effectively, and appropriately.

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The incidence of complications of otitis media has declined dramatically with the introduction of antibiotics. There is a clear divide between the era prior to the administration of antibiotics and that after it. The era after it is now known as the era of antibiotics [1–7]. The availability and administration of antibiotics have been responsible for the sharp decline in complications of otitis media. Prior to the antibiotic era, intracranial complications (ICs) occurred in 2.3–4% of cases. With the advent of antibiotics, improved understanding of diseases, advances in radiological imaging, improvements in the standard of living, and improved hygiene, the incidence of complications of otitis media has been greatly reduced to 0.15–0.04%. Mortality decreased from 25% to 8%. These are significant reductions.

From 1928 to 1910 [8–14], 3 years preceding introduction of antibiotics approximately 1 in every 40 days in a General Hospital was spent in treating an intracranial complication of otitis media. Meningitis was found to be the most common health disease followed by sinus thrombosis and brain abscess. Following the introduction of antibiotics, the incidence of complications has declined significantly [15, 16].

Mortality following lateral sinus thrombophlebitis has virtually disappeared, as has the incidence of brain abscess. The factor responsible for this decrease has been attributed to the introduction of antibiotics [17–21]. Purulent meningitis remains the most frequent intracranial complication of otitis media. Though the incidence has substantially reduced, it has not disappeared. In the past, cases of purulent meningitis had 100% mortality. In present times, if this condition is diagnosed early and treated appropriately, the recovery is nearly complete [22–29].

Inappropriate use of antibiotics result in alterations of symptoms and signs leading to patients to present differently as compared to the pre-antibiotic era. Host resistance, early identification of the symptoms, and signs lead to a quick diagnosis, followed by prompt therapy. This can prevent morbidity and mortality. Cardinal symptoms that indicate the

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possibility of impending intracranial complications [30–33] are the following:

1. Unrelenting pain. This indicates the possibility of pus under pressure in middle ear cleft leading to likely intracranial complications.
2. Headaches, drowsiness, and visual field defect points toward the diagnosis of brain abscess.
3. Unresolving, persistent, high-grade fever is indicative of the possibility of meningitis or sinus thrombophlebitis being present.

Factors That Influence the Development of Complications [34–38]

The spread of infection from middle ear to adjacent spaces is influenced by a number of factors. These are as follows:

1. The status of the hosts' immunity.
2. Virulence of the infecting organism/organisms.
3. Sensitivity to antibiotics (the adequacy of antibiotic therapy).
4. Integrity of anatomic barriers and the drainage of pneumatic spaces.

The organisms involved in intracranial complications have not changed significantly. *Streptococcus pneumoniae* haemophilus influenzae and moraxella catarrhalis cause most of the acute infections. However, the emergence of antibiotic resistance has changed the way patients now present with complications of otitis media.

Organisms involved in chronic infections are different from those involved in acute infection. Bacteria like pseudomonas aeruginosa are much more commonly isolated in chronic infections.

In immunocompromised persons, microbial infections are more likely to be atypical pathogens.

Anatomical bony barriers such a cellular septae and mucosal folds help retard and delay the spread of middle ear infections into the intracranial cavity. Intracranial extension of acute otitis media (AOM) is seen to occur more frequently when the temporal bone is poorly pneumatized. These anatomical barriers may be eroded by antecedent infections, granulation tissue, and cholesteatoma, thus allowing contamination to spread into the cranial cavity from the middle ear cleft. Trauma to the temporal bone which results in fractures can lead to the creation of passages that allow infection to bypass these natural defenses. Obstruction in drainage in middle ear cleft leads to accumulation of infected secretions which under pressure leads to erosion of natural anatomic barriers, thus opening the way for complications to occur.

Pathway of Spread of Infections in the Middle Ear Cleft [39–46]

Infections can likely spread beyond the middle ear cleft if the infection is uncontrolled or poorly controlled.

Infections can enter the intracranial cavity through one of three mechanisms listed below.

Bone Erosion

Bone erosion is the most frequent cause of spread in cases of otitis media (either acute or chronic OM) in well-pneumatized bones.

In AOM, bone erosion of the cellular septae within the mastoid bone results in coalescent mastoiditis. This causes a buildup of pus which under pressure leads to erosion and thinning of the mastoid bone which then results in either extracranial or intracranial spread of infection. In chronic cases, it is usually caused by cholesteatoma and less commonly caused by chronic osteomyelitis.

Granulations are formed on the bone and function as the last line of defense.

A dehiscent bone barrier and a layer of granulations over neighboring structure are always observed during surgeries for complications of otitis media.

Treatment of such complications implies surgical removal of suppurative, bone eroding focus. Which if not done completely may lead to recurrent or possibly more complications.

Preformed Pathways [47–52]

Direct extension by preformed pathways is seen in acute exacerbations of COM (chronic otitis media) or acute otitis media (AOM).

These pathways are normal anatomic openings in bony walls, such as the oval or round window, internal auditory canal, cochlear aqueduct, or endolymphatic duct and sac. Congenital dehiscence such as a patent suture or a dehiscent floor of the hypotympanum over the jugular bulb can also act as a conduit that serves to spread infections.

A preformed pathway can be created by a fracture, or an inadvertent defect created by previous surgery. A labyrinthine fistula can also serve as a pathway for infection.

The possibility of a preformed pathway should be considered when there is a history of a skull fracture, repeated attack of meningitis, previous surgery on the temporal bone, or previous healed otitis media.

Treatment of complications involves

(a) Treatment of the complication, i.e., treatment of the brain abscess, thrombophlebitis, etc. Intracranial complications involve prioritization of which problem needs to be addressed first.
(b) Surgical closure and sealing of the preformed pathway and.
(c) Surgical drainage of the pus and removal of disease in the temporal bone.

The complications listed below can either occur singly or can occur simultaneously. The common source from all they occur is located in the ear. Thus, if a patient presents with meningitis and a brain abscess, the physician is obliged to examine the ear carefully to determine if the source is located in the ear. It should be further cautioned that if a patient presents with acute otitis media and meningitis the physician needs to identify the source from where the ear infection was initiated. This could have been from what was thought to be a simple upper respiratory tract infection. Thus the entire ears, nose, and throat along with the other body systems will need to be evaluated equally and carefully.

(d) Aggressive and appropriate antibiotic therapy cannot be overemphasized. Surgical eradication of disease alone will not control the infection that has resulted in the complication.
(e) A multidisciplinary approach. Different specialities need to be involved in order to bring about a successful resolution.

Thrombophlebitis

Infections travel from the lining mucosa of the middle ear and mastoid through intact bone by means of thrombophlebitis. Thrombophlebitis occurs in small venules and then advances to involve larger ones. Brain abscesses have been found to be the consequence of retrograde thrombophlebitis.

A rich network of veins within the temporal bone are in direct communication with extracranial and intracranial veins and cranial diploic veins which can further the spread of the infection. Extracranial and intracranial venous systems are linked via mastoid emissary veins which enter the sigmoid sinus which in turn drain into the superior and inferior petrosal sinuses. This interconnection of dural venous sinuses can lead to sigmoid sinus thrombophlebitis which in turn could result in thrombophlebitis of other venous sinuses.

Complications resulting from acute infections usually start within a week of the infection being initiated. On occasion, it can sometimes occur within a day or two of the infection being contracted. In the presence of thrombophlebitis, surgical findings reveal that the bone of the middle ear cleft is intact. The common complication of coalescent mastoiditis is found to be absent. Thrombophlebitis in addition to causing cerebellar abscesses can also cause meningitis.

Cardinal Symptoms of Impending/Ongoing Intracranial Complications

Complications usually occur within the temporal bone first before shifting outward to cause intracranial complications.

The cardinal symptoms of imminent intracranial complications are as follows:

1. **Persistence of otorrhea**: the pus is foul smelling and thicker and on occasion is blood-stained. Decrease in discharge points toward obstruction in drainage and likely subsequent onset of intracranial complications.
2. **Pain**: patient usually complains of deep boring pain, may be generalized which is usually "the worst headache" they had in their life.
3. **Fever**: High-grade fever that never returns to base line is typical of an intracranial complication.
4. **Altered sensorium, toxemia, photophobia, and irritability**.
5. **Neck stiffness** and generalized malaise are signs indicative of infections advancing toward the cerebrospinal fluid (CSF) space.

The goals of the surgery in the presence of complications of otitis media are as follows:

Correct prioritization of treatment. Commonly both areas, the brain and the ear, will need to be treated simultaneously.

1. The total eradication of disease and the prevention of recurrence of ear disease.
2. Establishment of adequate drainage and ventilation in the temporal bone.
3. Swift, prompt, and adequate treatment of the intracranial complication.

Antibiotics must always accompany surgical intervention as well, for surgery alone will be insufficient in bringing about a cure.

A complete cortical mastoidectomy for the purpose of comprehensive eradication of the disease and the evacuation of pus is carried out. The disease removed is sent for histopathological evaluation, and the pus retrieved is sent for microbiological evaluation.

In AOM, usually high dose of appropriate antibiotics and a cortical mastoidectomy accompanied by myringotomy with ventilation tube insertion tube insertion is sufficient to provide adequate drainage and ventilation.

In chronic disease with cholesteatoma, canal wall down mastoidectomy with a wide meatoplasty is recommended.

Wide meatoplasty provides adequate drainage and allows subsequent examination and cleansing of the mastoid cavity.

Meningitis

Generalized meningitis involves infection and inflammation of the pia arachnoid and the CSF of the subarachnoid space which is continuous for the entire cerebrospinal axis.

In the pre-antibiotic era, meningitis was the most common intracranial complications of otitis media. Recovery from meningitis has improved significantly. Meningitis occurs more commonly in the presence of AOM than COM.

Bacteriology

H. influenzae and *S. pneumoniae* are most commonly identified microorganisms isolated in instances of meningitis caused by otological infections. Streptococcus pneumoniae infections of the meninges are often associated with acute otitis media. Anaerobic organisms are seen in cases of intraventricular rupture of a brain abscess. In cases secondary to otogenic infections, less than 1% of cases demonstrate evidence of polymicrobial infections.

Pathophysiology

Meningitis occurs when infections spread from the ear to the CSF via retrograde thrombophlebitis, bone erosion, and preformed pathways. Infections can also reach the CSF via the labyrinth through the round and oval windows. Infections can also travel through perineural spaces to the internal auditory canal and less frequently via the endolymphatic ducts. Trauma to the ear with resultant fractures, accidental dural tears following otologic ear surgery with resulting CSF leaks can also result in meningitis.

Clinical Presentation

Otogenic causes of meningitis should always be ruled out in all cases of meningitis. Meningitis can occur with or without otorrhoea.

In cases of AOM, the presentation is far swifter. The earliest symptoms are headache, fever, vomiting, photophobia, irritability, and restlessness. Infants may have seizures. As the infection progresses, the headaches increase in intensity, and vomiting becomes more pronounced. Neck stiffness is present and is the signature sign of meningitis.

Brudzinski's sign is the inability to flex the leg without moving the opposite leg (or flexion of the neck resulting in flexion of the hip and knee) and is a sign of meningitis. Kernig's sign, an inability to extend the leg when lying supine with the thigh flexed toward the abdomen, is a specific sign for meningitis.

Management

High-resolution CT scanning is the imaging modality of choice. Bony details are best visualized with CT scanning. High-resolution CT scans of the temporal bone help rule out the presence of congenital car malformations while demonstrating the entire anatomy of the temporal bone.

Magnetic resonance imaging (MRI) (Fig. 34.1) gives better resolution of the brain substance and shows inflammatory changes in the brain and meninges and middle ear fluid. MRI of the brain and surrounding structures and CT scanning of the temporal bone complement each other and are very valuable when assessing a patient who has meningitis.

Lumbar puncture is imperative to identify the causative organism and the source of infection. Cloudy CSF, elevated white blood cell count, low glucose, and high protein point toward meningitis.

Fundoscopic of the examination of the eye has been found to show indistinct disk margins and choking of vessels in meningitis.

Gram-staining of the fluid, confirmation by culture, and sensitivity testing help in the selection of a suitable appropriate antimicrobial drug. A swab should be taken from the ear as well.

Treatment

Antimicrobial drugs are at the core of treatment of meningitis. Meningitis resulting from acute otitis media is usually directed at *H. influenzae* type B with second- or third-generation cephalosporins which cross the blood–brain barrier.

Bactericidal drugs should be preferred over bacteriostatic drugs.

Large amounts of fragments of the inflammatory processes are released that have severe neurologic and auditory sequelae because of rapid bacteriolysis. Glucocorticoids like dexamethasone if given concurrently or before antibiotics have shown to decrease the sequelae of this catastrophic event.

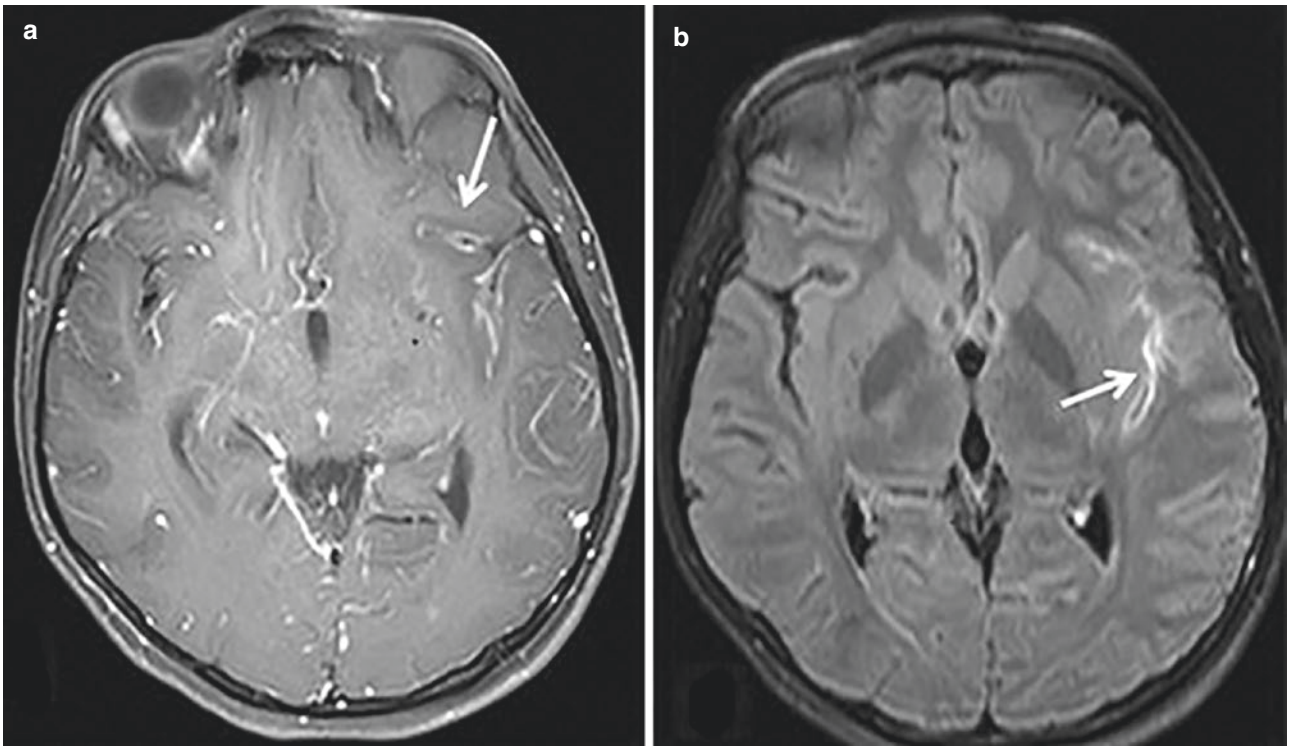

Fig. 34.1 MRI scan of a patient suffering from meningitis as a complication of otits media. (**a**) Axial postcontrast T1 and (**b**) postcontrast FLAIR shows leptomeningeal enhancement in left sylvian fissure (arrow)

These measures have been found to be useful only for meningitis caused by acute otitis media and only if there are no other intracranial complications presents.

Role of Surgery

For the patient of acute otitis media with an intact tympanic membrane who presents with no clinical or radiological evidence of mastoiditis, myringotomy with evacuation of the fluid from the middle ear is sufficient enough treatment for otogenic meningitis.

In situations where mastoiditis is clinically and radiologically evident, cortical mastoidectomy with middle ear exploration needs to be performed. Should the patient present with a history of trauma involving the head and temporal bone accompanied by rapid onset of meningitis, the surgeon should look for a likely fracture in the temporal bone. Once that site has been correctly identified, that communication which was responsible for the causation of meningitis must be repaired to prevent recurrence of meningitis. Radiological evidence in the form of CT scans of the temporal bones will help in accurately demonstrating the site of the fracture. This area should be repaired, and the passage should be sealed off.

Should a cholesteatoma be present then radical mastoidectomy with wide meatoplasty is the surgical modality of choice.

Brain Abscess

A brain abscess is a focal suppurative process within the brain parenchyma and is surrounded by a region of encephalitis.

Brain abscesses secondary to otitis media are seen mostly in children and in the fourth decade [53–60]. The male-to-female ratio has been approximately 3 to 1.

Most of the brain abscesses are associated with infections resulting from cholesteatomas.

Otogenic brain abscesses are more likely to be located in the cerebrum (predominantly in the temporal lobe) than in the cerebellum. But the majority of cerebellar abscesses are associated with middle ear infections, which are more likely to be fatal.

The most important factor in the treatment of brain abscesses depends on swift control of the infection.

Factors implicated in the causes of brain abscesses

1. The immune status of the patient influences the microbiology of brain abscess. Multiple organisms have been reported in brain abscesses. Gram-positive organisms like *Streptococcus* and *staphylococcus* and gram-negative like *Escherichia coli* and *Proteus Miabilis*, *Klebsiella*, and *Pseudomonas* species are typically isolated from brain abscesses. *H. influenzae* is rarely found in otogenic brain abscesses.

2. Brain abscesses are likely to occur when the following conditions are present.
   (a) Contiguous spread of disease to surrounding structures.
   (b) Hematogenous spread from a distant focus like chronic pyogenic lung disease.
   (c) Trauma to the head or cranial/surgery on the ear.

Venous thrombophlebitis rather than direct extension of disease from the ear to the brain is the common cause of otogenic brain abscesses.

Surgical causes, i.e., mastoidectomy when an open mastoid cavity has been created but residual disease persists, can lead to brain abscess.

Pathophysiology

1. Osteitis or granulation tissue causes retrograde thrombophlebitis of dural venous sinuses that terminate in the white matter of the brain, causing encephalitis.
2. This progresses to necrosis and liquefaction of brain tissue (focal suppuration) with surrounding edema.
3. After approximately 2 weeks, an abscess capsule surrounded by granulation tissue forms. It has been observed that encapsulation is more well defined on the cortical side as compared with the ventricular side. This perhaps explains the propensity of abscesses to rupture medially into the ventricular system rather than into the subarachnoid space.

Clinical Presentation

Symptoms usually present for approximately 2 weeks before the brain abscess is fully formed. Brain abscess formation is indicated by the presence of (a) headache, (b) high-grade fever, and (c) focal neurologic deficits. Signs of meningitis are also usually present.

The patient is appears very "toxic" and drowsy and often complains of deep bony pain in the ear. Foul smelling, creamy otorrhea indicates a fulminant, destructive process. Location of the abscess can be predicted by the focal deficits, cerebellar abscesses present with dizziness, ataxia, nystagmus, and vomiting. Seizures are seen in temporal lobe lesions. Papilledema is a late sign seen in stage 3 of abscess formation.

Imaging

In computed tomographic scanning, brain abscess appears a hypodense area surrounded by an area of edema "ring sign." Serial scans can help in assessing the response of the treatment.

MRI (Figs. 34.2, 34.3, and 34.4) is more useful in checking the brain parenchyma and detecting the progress of abscess though it does not help in assessing the temporal bone for which a separate CT scan of the temporal bones may be needed.

Sequalae of Otogenic Brain Abscess

The brain abscess could rupture into the ventricle resulting in death. Brain abscesses can also cause herniation of the brain resulting in death. Brain asceses also can result in seizures, permanent neurologic deficits, and vegetative state.

Management

Management consists of antibiotic therapy and surgical management of the source of infection.

High-dose antimicrobial medication has to be started immediately. The decision to eradicate the infective focus from the ear will need to be performed at the earliest. Some prefer to operate at the earliest citing that once the focus of infection is eradicated, the infection in the brain will respond quickly to the antibiotics administered.

Neurosurgical intervention is required if the abscess is 2.5 cm or bigger.

The decision to excise or drain a brain abscess is controversial as improved radiological imaging and more effective antibiotics have radically improved the outcomes of such complications.

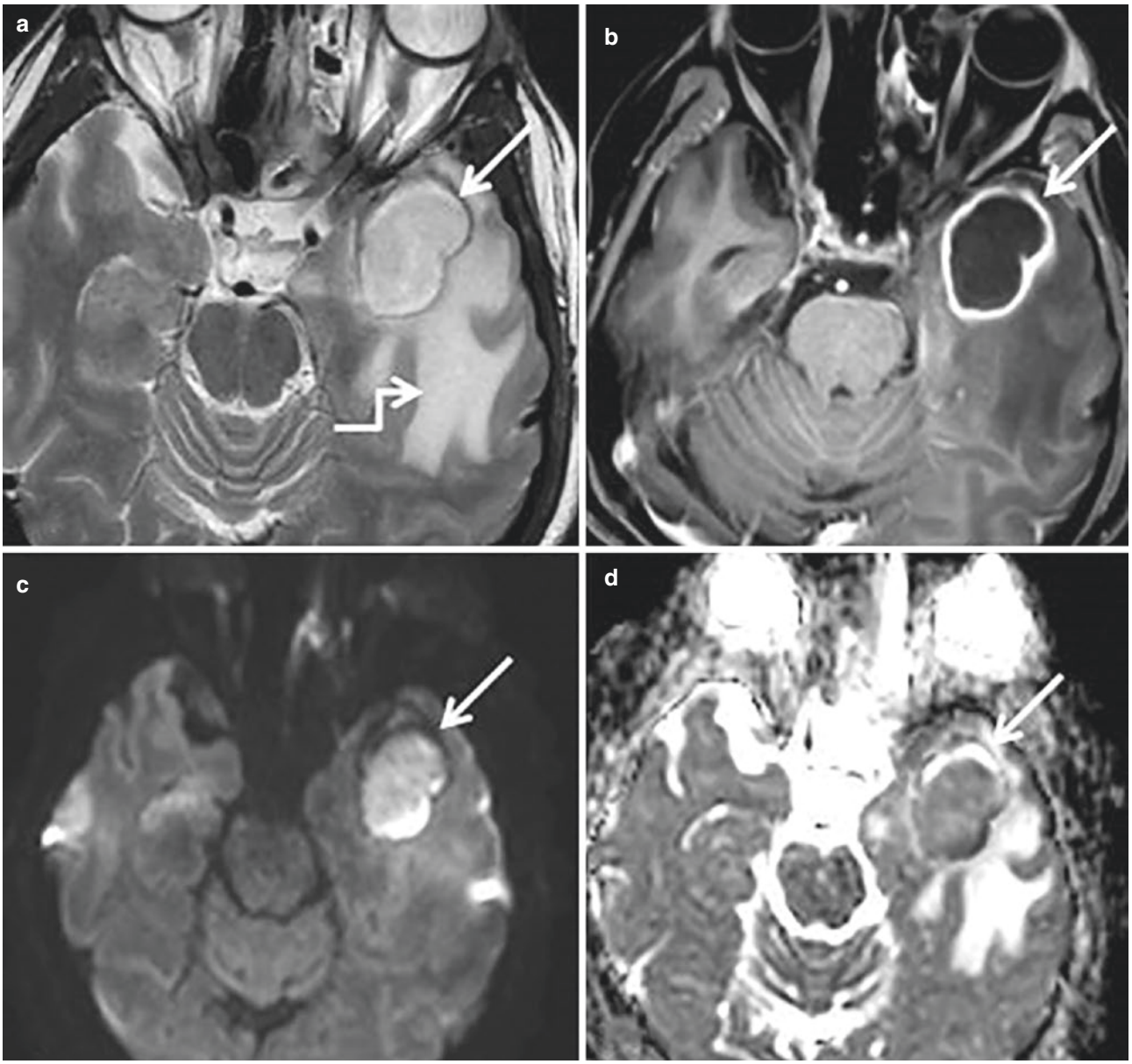

Fig. 34.2 (**a**) Axial T2-weighted image shows well-defined hyperintense lesion (arrow) with hypointense wall and perilesional edema (stepped arrow) in left temporal lobe, (**b**) axial T1W postcontrast image shows rim enhancement (arrow), (**c**) restriction on diffusion-weighted image (arrow), and (**d**) signal drop on ADC (arrow)

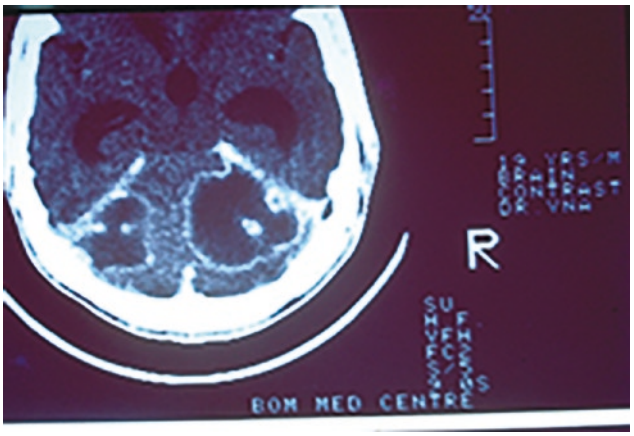

Fig. 34.3 Bilateral brain abscess of otogenic origin

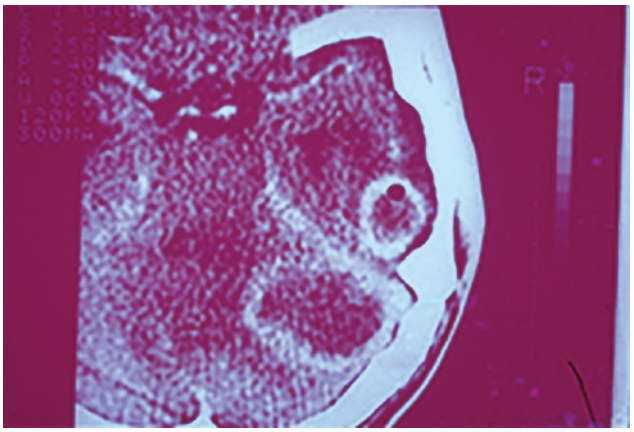

Fig. 34.4 Brain abscess of otogenic origin involving the temporal lobe and the cerebellum

Otogenic Suppurative Thrombophlebitis

The dural venous sinuses are blood vessels located between the endosteal and meningeal layers of dura mater that covers the brain. These venous sinuses receive blood from the cerebral veins and cerebrospinal fluid (CSF) from the subarachnoid space via arachnoid granulations. They drain into the internal jugular vein.

Suppurative thrombophlebitis of the sigmoid sinus is associated with both with acute and chronic otitis media [61–69].

Pathophysiology

Thrombosis occurs after the infection has spread to the intima of the vein. This results in edema resulting in sluggish blood flow in the lumen of the sinus resulting in a thrombus. This infected thrombus increases in size occluding the lumen of the vein. This can embolize downstream causing seeding of infection which can then result in generalized septicaemia and other problems like stroke.

Suppurative thrombus can propagate intracranially or into the jugular vein and onto the right atrium of the heart. Intracranial extension results in brain abscess and thrombophlebitis of other vessels in the cranial cavity. Intracardiac spread results in widespread spreading of infection and fulminant septicemia [70–73].

Bacteriology

Cultures have revealed the presence of polymicrobial infections. The B-hemolytic streptococcus, *Bacteroides* and *Streptococcus* species as well as gram-negative rods are seen. *Pseudomonas* and *Proteus* species have also been isolated.

Clinical Presentation

The patient will be seriously ill and restless and will complain of otalgia. Otalgia is described as a deep, boring pain, which usually heralds a worsening neurologic status. Otorrhea is foul smelling and usually blood-stained.

High-grade fever is a “picket fence” appearance or may be high grade without returning to baseline. Neck stiffness and papilledema are present. If beta-hemolytic streptococci are responsible for the infection, the patient may present with steadily worsening anemia, manifesting in pallor. Proptosis, ptosis, chemosis, and ophthalmoplegia are signs of the thrombus spreading to the cavernous sinus. Griesinger’s sign is usually present. Greisinger’s sign consists of tenderness, and edema over the mastoid is pathognomonic for suppurative thrombophlebitis of the sigmoid sinus and its cause is thrombosis of the mastoid emissary veins. Extension of the thrombus into the internal jugular vein causes it to become hard, cord-like, and very tender to palpation and results in a stiff neck. The cervical lymph nodes along the internal jugular vein are enlarged and tender. Otitic hydrocephalus is seen in involvement of the torcular and sagittal sinuses.

Imaging

MRI (Figs. 34.5, 34.6, 34.7, and 34.8) is the imaging tool of choice. MRI scans demonstrate venous sinus obstruction, and reversal of blood flow is seen. On gadolinium-enhanced MRI, the delta sign is observed. The thrombus is seen as a soft tissue signal with a bright appearance of the dural walls. It can help in differentiating an early and a late thrombus. The early thrombus is rich in deoxyhemoglobin; hence on the T1 weighted image, it has intermediate density and a low

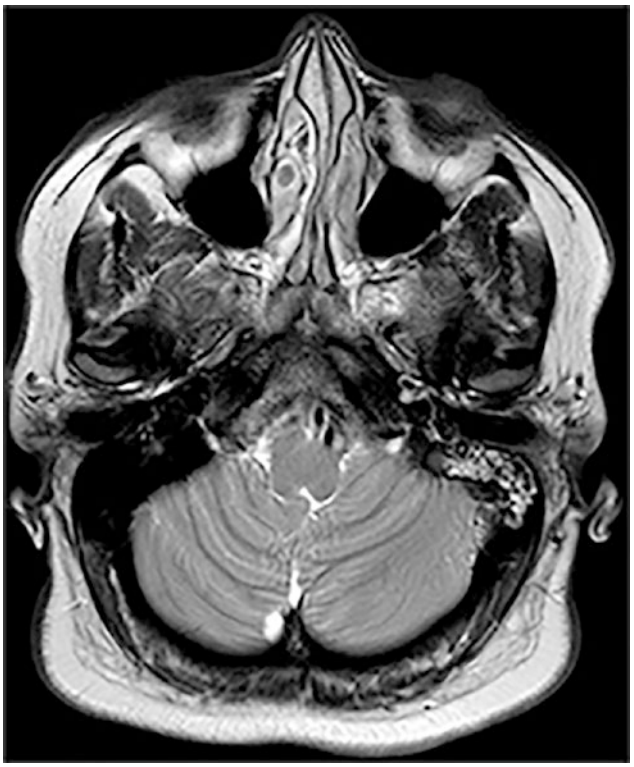

Fig. 34.5 MRI scan showing mastoiditis

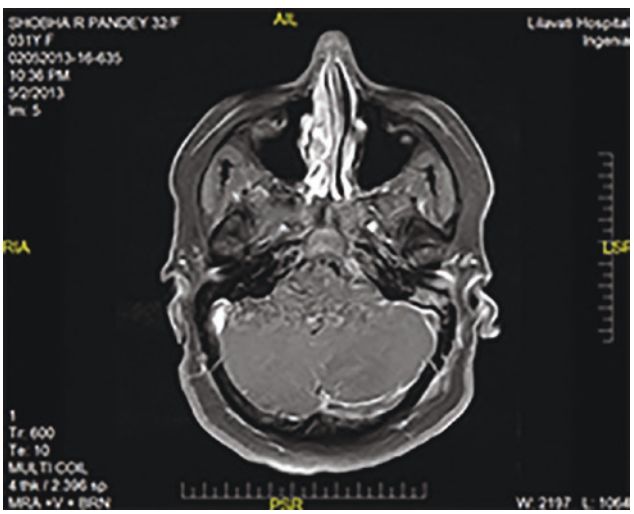

Fig. 34.6 MRI scan showing thrombus on the left side in the transverse sinus

intensity on the T2 image. In a mature thrombus, the clot appears hyperintense on both T1 and T2 images because of the formation of methemoglobin.

CT scans shows the "delta sign" clot surrounded by a high-intensity rim of contrast-enhanced dura.

Cerebral angiography is not indicated as it has a potential to dislodge the thrombus.

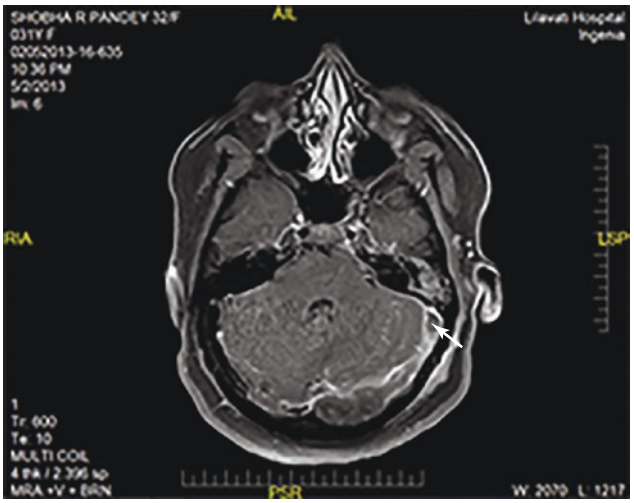

Fig. 34.7 MRI scan demonstrating sigmoid thrombophlebitis with typical "delta" sign

- **Queckenstedt's (or the Tobey–Ayer)** test is used to detect lateral venous sinus thrombosis: a spinal needle is placed in subarachnoid space and attached to a manometer, and the resting CSF pressure is measured. Pressure is applied to the IJV with fingers sequentially and then both at once. The test is considered positive if pressure fails to rise after compression of internal jugular vein (IJV) on the side of the diseased ear and fails to fall when the vein is released with a prompt contralateral response.

A false positive is seen mostly on the left side in cases where one sigmoid sinusis smaller than the other.

False-negative test is observed in cases in which there are well-developed collaterals around the sigmoid sinus through the mastoid emissary vein and the petrosal sinus.

Contraindications to lumbar puncture are high intracranial pressures are also contraindications for the performance of Queckenstedt's test.

With the advent of greatly improved radiological imaging techniques, Queckensted's test is now infrequently performed.

Blood cultures provide important information for the management of the sigmoid sinus thrombophlebitis. The microorganisms can be correctly identified, and the appropriate treatment can be initiated to deal with the infection.

Treatment

Prior to the antibiotic era, surgery on the ear was the focal point of treatment. A mastoidectomy with unroofing of the sigmoid sinus was carried out, and the perisinus abscess, if present, was drained. If the sinus demonstrated a thrombus, it would be removed. Scanty bleeding around the venous

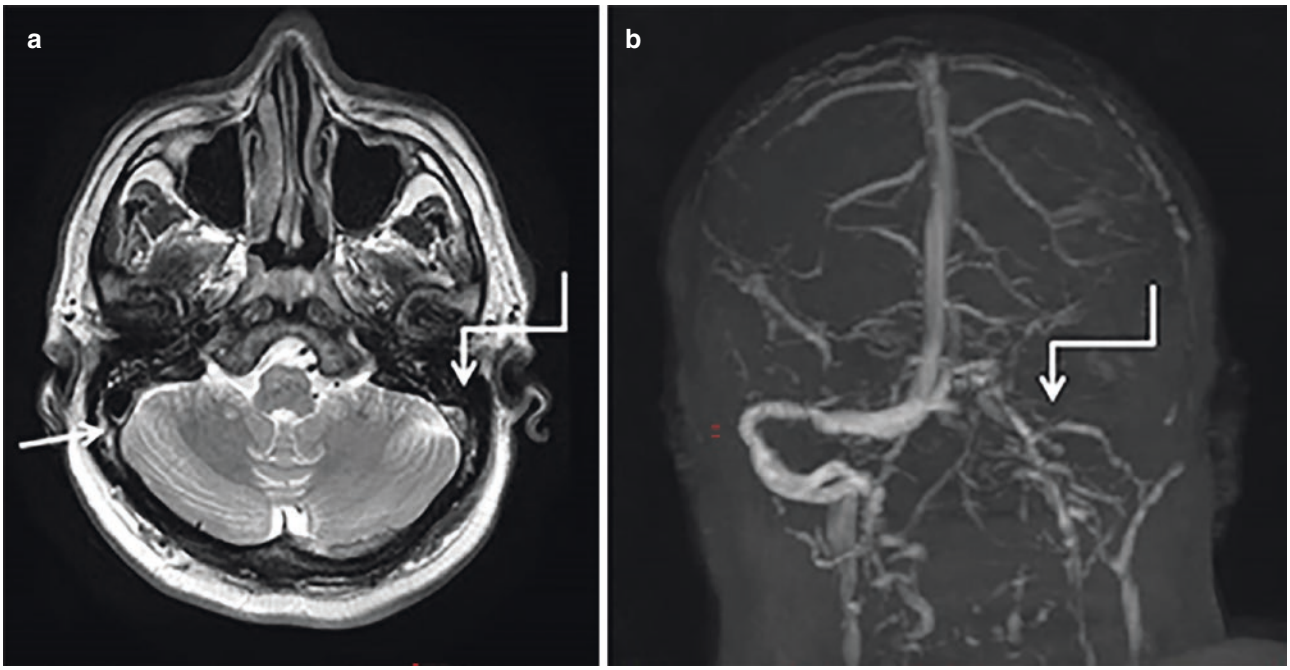

Fig. 34.8 (**a**) Axial T2W image shows the absence of flow void in left transverse sinus (stepped arrow) as compared to normal flow void on right side (arrow); (**b**) TOF venogram shows no flow-related enhancement in left transverse and sigmoid sinuses (stepped arrow)

sinus indicated the presence of an intraluminal clot. In such situations, the sinus should be opened very carefully to avoid inadvertent tearing of the medial dural wall. A tear in the medial dural wall would result in a CSF leak. The entire thrombus should be removed. In advanced thrombus formation, some authors state that the Internal Jugular Vein should be ligated to prevent passage of the thrombus into the heart.

In recent times, such extensive disease is rarely seen. High doses of appropriate antibiotics combined with complete evacuation of the disease from the middle ear and the mastoid is carried out. This should be adequate to halt the progress of disease and prevent worsening of the patient's condition. Drainage of the perisinus abscess and removal of the thrombus are included in the mastoidectomy procedure. Ligation of the internal jugular vein is rarely performed. It is carried out only in situations where the disease is advancing toward the heart in spite of the adequate and appropriate treatment. Anticoagulants are no longer recommended as they lead to increased chances of venous infarction.

Otitic Hydrocephalus

Otitic hydrocephalus or benign raised intracranial tension was first described by Quincke in 1897. It is a syndrome associated with otitis media characterized by intracranial pressure with normal CSF findings. It recovers spontaneously and is frequently associated with sigmoid sinus thrombophlebitis.

Pathophysiology

The precise mechanism underlying the development of otitic hydrocephalus is not known [74–77].

1. Sahs and Joynt [78] postulated that the hydrocephalus is secondary to brain edema as brain biopsies revealed interstitial edema.
2. Weed and Flexner [79] postulated disruption in venous circulation as a cause, since changes in CSF pressure are directly related to intracranial venous pressure.

Clinical Presentation

Patient presents with headache, drowsiness, vomiting, blurring of vision, and diplopia. Acute or chronic otitis media is usually present at the onset of otitic hydrocephalus.

Ophthalmic evaluation reveals the presence of papilledema and sixth cranial nerve palsy. If left untreated, optic atrophy can eventually occur.

Elevated CSF pressures with normal CSF biochemistry are the classic findings of otitic hydrocephalus.

Management

Ear pathology is eradicated, and the increased CSF pressure is assessed and addressed so that the hydrocephalus is reduced.

Drainage of cerebrospinal fluid with shunts have been recommended. Optic sheath decompression can be considered to prevent optic atrophy [80–83]. Medical therapy consists of corticosteroids, mannitol, diuretics, and acetazolamide which are also administered simultaneously.

Subdural Empyema

Collection of pus in the space between the dura mater and the arachnoid membrane is termed subdural empyema [81, 82].

Pathophysiology

The subdural spaces are anatomically confined spaces and can quickly develop into fatal mass lesion. It is a potential space than an actual one. It is divided into compartments by foramen magnum, tentorium cerebelli, base of the brain, and the falx cerebri.

Clinical Presentation

Sudden-onset severe headache is typical of subdural empyema. It is accompanied by fever and vomiting.

The rapid deterioration of the patient's conditions points to the presence of a subdural empyema.

Magnetic resonance imaging is the imaging modality of choice.

It can easily differentiate between epidural and subdural infection.

Multiple, discrete, and loculated subdural collections are seen. Magnetic resonance imaging is advantageous because of the absence of bone artifact, heightened contrast between bone, CSF, and brain parenchyma, as well as because of its multiplanar imaging capability.

Magnetic resonance imaging also allows differentiation of sterile, bloody, and infected collections.

Treatment

Immediate drainage of the abscess with complete removal of the focus of infection within the ear along with simultaneous administration of high-dose intravenous antimicrobial medication forms the core treatment modality.

Lumbar puncture is contraindicated as it may trigger herniation of the cerebellar tonsils.

Epidural (Extradural) Abscess

The epidural (extradural) space is the potential space between the dura mater and the bone of the intracranial cavity. Granulation tissue is seen in direct continuity with the suppurative process. Large accumulations of pus are rare. An epidural abscess usually precedes other intracranial complications, especially sinus thrombophlebitis and brain abscess. Sinus thrombophlebitis is the most complication coexisting with an epidural abscess.

Pathogenesis

Bone reabsorption in coalescent mastoiditis leads to bone giving way, especially in areas of sigmoid sinus, resulting in a pocket of granulation tissue or pus which infects the sigmoid sinus. It is mostly associated with chronic suppurative otitis media without cholesteatoma.

Chronic suppurative otitis media without cholesteatoma is usually associated with granulation tissue that invades the perisinus air cells.

Asymptomatic (silent) extradural granulation tissue if left untreated has the potential to progress into far more serious complications.

MRI is the best radiological imaging modality of choice for the diagnosis of epidural abscesses. Frequently, these epidural abscesses are found as incidental findings during surgery as they are rarely symptomatic unless very large.

Management

The presence of granulation tissue penetrating the bone along the sigmoid sinus indicates the presence of an epidural abscess. The surrounding bone should be opened, and the granulations should be removed and the abscess should be drained. Care should be taken that dura is not punctured as it can lead to CSF leak which can have catastrophic consequences.

How Common Are Intracranial Complications in Recent Times?

Complications have most certainly declined when looking at medical literature in recent times. However, reports advise against complacency. A report by Bales et al. [83] report complications occurring in developed nations where there is access to healthcare. Even in the antibiotic era, intracranial complications of otitis media still occur in developing nations. The reasons cited [84] for these problems are as follows:

1. Many patients do not seek medical care and delay treatment.

2. Poor access to healthcare.
3. Poor compliance with treatment. Patients either take inadequate treatment or stop treatment without the consultation of a medical person.
4. Healthcare centers are often so rudimentary that modern radiological imaging modalities are not available. This in turn impacts diagnosis and eradication of disease. This in turn impairs the detection of complications should they be within the temporal bone or on within the intracranial cavity.
5. Unlike developed nations where healthcare is covered by insurance, developing nations quite often do not have facilities available that are covered by healthcare insurance.
6. A large amount of the population has not received the pneumococcal vaccine. The protection offered by this vaccine is against the pneumococcal serotypes and Hemophilus serotypes. Both these bacteria are frequently implicated in complications caused by otitis media.

In today's times, are complications associated more frequently with acute otitis media or chronic otitis media? Recent literature [85, 86] indicates that chronic otitis media associated with cholesteatoma is the source. Literature cites the indolent manner in which chronic otitis media presents is the reason why patients often defer seeking treatment. This results in a complication which often takes a long time to diagnose causing a delay in treatment [87].

What are the frequently encountered complications in the antibiotic era in developed nations? The literature [88] cites meningitis as the commonest followed by brain abscess. These are the common intracranial complications. Temporal bone complications are subperiosteal abscess. Both intracranial complications and temporal bone complications are frequently associated with cholesteatoma. Though cholesteatoma is associated with a high incidence of complications, acute otitis media [89] when not diagnosed and treated in a timely and appropriate manner can result in an intracranial complications even in a developed nation.

One report [90] puts it very succinctly by stating the following,

(a) Complications of otitis media are still present even in the antibiotic era and in the age of awareness and information.
(b) Male children and adults are frequently affected. Chronic otitis media is responsible for most of the complications, approximately 80% of all intracranial complications.
(c) Acute otitis media causes complications below the age of 15 years. Indolent pediatric chronic otitis media when not correctly diagnosed results in complications.
(d) The most frequently encountered complications were in the order of frequency brain abscess with meningitis (78%), lateral sigmoid sinus thrombosis (13%), empyema (8%), and otitic hydrocephalus 1%.
(e) The organisms detected in these complications are *Proteus mirabilis*, *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Streptococcus pyogenes*. These organisms are commonly cited in nearly all literature as being responsible for infections associated with otitis media.

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