Complications of otitis media: A review
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Otitis media is a potentially serious disease chiefly because of its complications, which may threaten life and health. This fact has been recognized from antiquity, when Hippocrates, about 460 B.C noted that "acute pain of the ear with continued high fever is to be dreaded for the patient may become delirious and die." Complications of suppurative otitis media develop if infection spreads form the middle ear cleft to structures from which this mucosa-lined space is usually separated by bone. Before antibiotics were regularly available, these complications followed acute suppuration more often than chronic middle ear disease. Nowadays chronic middle ear infection is the greater hazard.

The complication to be discussed fall into two main categories:

1. Those within the cranial cavity
   - Extradural abscesses
   - Subdural abscesses
   - Sigmoid sinus thrombophlebitis
   - Meningitis
   - Brain abscess
   - Otitic hydrocephalus

2. Those within the temporal bone/Extracranial
   - Subperiosteal abscesses
   - Facial paralysis
   - Labyrinthine infections

Pathways of spread in the production of a complication:
- Extension by osteothrombophlebitis
- Extension by bone erosion
- Extension by preformed pathway

Extradural abscesses
The bone erosion of coalescent mastoiditis frequently exposes the sigmoid portion of the lateral sinus to produce a perisinus abscess. The bone erosion of a cholesteatoma more often exposes the dura of the middle cranial fossa to produce an extradural abscess. In either case, protective granulations always form between the pus and the dura.

Symptoms
The majority of perisinus and extradural abscesses produce no symptoms and are a chance finding at operation. Their presence may be suspected when:
1. There is profuse or markedly intermittent otorrhea.
2. There are marked pulsations of the purulent discharge.
3. There is a low grade fever of unknown origin following acute otitis media.
4. There is persistent headache on the side of otitis media.
5. There are symptoms and signs of localized protective meningitis.
6. The X-ray shows an unusually large coalescent or cholesteatoma cavity. Computed tomographic scan shows an epidural collection with a contrastenhancing periphery.

Occasionally a very large extradural abscess may cause symptoms of cerebral compression, with somnolence, projectile vomiting, papilloedema, and slowed pulse, resembling, but with out the localizing symptoms of a brain abscess.

Treatment
The treatment of perisinus or extradural abscess is surgical. If the symptoms and finding suggest a possible extradural or perisinus abscess, the tegmen and sinus plates should be removed at operation and the dura exposed.
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In addition to the wide exposure of an extradural or perisinus abscess, the causative coalescent or cholesteatomatous bone eroding disease must be dealt with by the appropriate mastoidectomy.

**Subdural abscess (empyema)**

Clinical features

The development of a subdural empyema is heralded by the development of severe headache, fever and drowsiness, which is followed by the onset of focal neurological symptoms, both irritative as fits and paralytic. The course is much more rapid than that of a brain abscess. Drowsiness may develop over a few hours and proceed quickly to coma. Paralysis of one upper or lower limb may rapidly extend to hemiplegia. Hemianopia and hemianesthesia occur and if the lesion is on the dominant side, aphasia develops. Epileptiform fits of Jacksonian type, starting locally and spreading to affect one side of the body, may precede the weakness. These fits sometimes increase in frequency, and are probably the result of the cortical thrombophlebitis. Papilloedema is uncommon, as are cranial nerve palsies but they have been described in the fully developed picture. The site of the fits, and the pattern of weakness indicate the position of the empyema.

**Diagnosis**

Meningism may accompany the headache. The clinical picture can usually be distinguished from that of meningitis by virtue of the characteristic neurological localizing features. The rate of development, over hours rather than days, is much faster than would be expected from a typical brain abscess. Nowadays, a definitive diagnosis relies on enhanced CT scanning but pictures may be equivocal or normal and MRI may prove to be more reliable. Lumbar puncture is helpful, but risky. The cerebrospinal fluid pressure is raised, but its sugar content is normal, and cultures are sterile. The fluid may occasionally be turbid if there is marked pleocytosis.

**Treatment**

This complication must be managed in closed cooperation with a neurosurgeon. Treatment comprises the administration of massive doses of systemic antibiotic, removal of the subdural fluid and treatment of the ear disease.

**Lateral sinus thrombophlebitis**

Symptoms

The most constant, characteristic and sometimes the only symptom of lateral sinus thrombophlebitis is fever. Typically the fever is septic in type with wide swings in the temperature curve producing a "picket fence" temperature chart. Chills usually precede the sharp rises in temperature and profuse sweats accompany the downward swings. Each rise in temperature corresponds to the escape of a fresh batch of organisms into the systemic circulation, occurring at irregular intervals but averaging once or twice in 24 hours. The temperature curve resembles that of malaria but lacks its regularity.

Between the bouts of fever, the patient with lateral sinus thrombophlebitis is alert with a sense of well being out of proportion to the serious illness and the prostration of the patient with meningitis. Progressive anemia is the third symptom of infection of the lateral sinus.

Progressive emaciation is the fourth symptom. Less common symptoms of lateral sinus thrombophlebitis are oedema over the posterior aspect of the mastoid emissary vein, known as Griesinger's sign: tender, enlarged cervical glands along the internal jugular vein on the involved side: extension to the cavernous sinus via the superior and the inferior petrosal sinus, with the usual symptoms of cavernous sinus thrombosis, namely chemosis, proptosis, fixation of the eyeball, and papilloedema: and pain when there is dural inflammation.

**Diagnosis**

A positive blood culture is very strong evidence in favor of lateral sinus thrombophlebitis. However, a negative blood culture on one or
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several occasions does not rule out lateral sinus thrombophlebitis.

Treatment

The aim of therapy today is the control of infection, not the reestablishment of blood flow through the sinus. Intravenous antibiotics are started to sterilize the blood and to prevent mastoidectomy, with wide exposure of the sigmoid sinus. The mastoidectomy may be either an open cavity or an intact canal wall technique. After exposing the bony sigmoid sinus, the dura of the sinus and the posterior fossa must be explored. Granulation tissue is bluntly dissected from the dura and any abscesses are drained. Bone is removed from the dura and sinus until normal dura is visualized all around the area of phlebitis. If the sinus is found to be normal in appearance and easily compressible no further dissection is indicated.

If the sinus wall is markedly thickened, inflamed and not compressible, the status of blood flow through its lumen should be assessed. This assessment may be done by inserting a small gauge needle into the lumen and aspirating blood. If blood is found, no further surgery need be done. However, if blood is not aspirated, the sinus should be opened. If a fibrotic mural thrombus is found, no additional dissection is indicated. However, if an infected thrombus or mass of granulation tissue is found it should be removed through gentle dissection and suction.

Otitic Hydrocephalus

Symptoms

In 1927, Symonds described a syndrome of increased intracranial pressure without a brain abscess following several weeks or more after acute otitis media. The most constant symptom is headache, often with sixth cranial nerve paralysis on the same side and sometimes with vomiting. Otherwise the patient looks and feels quite well. The most constant findings are papilloedema and a spinal fluid pressure exceeding 300 ml of water. Unlike localized meningitis, the spinal fluid is clear without increase in cells or protein. In contrast to brain abscess, there are no localizing neurologic changes and ventriculography of computed tomographic scan does not show a space occupying lesion.

Diagnosis

The diagnosis of otitic hydrocephalus is made by lumbar puncture, showing a normal spinal fluid under markedly increases pressure, in a patient who develops headache and papilloedema with or without sixth cranial nerve paralysis 1 to several weeks or more after otitis media.

Treatment

The treatment of otitis hydrocephalus is to reduce the increased intracranial pressure by repeated lumbar or ventricular punctures and use of diuretics and if necessary to perform a subtemporal decompression, until the condition subsides, so as to prevent optic atrophy from prolonged papilledema. After weeks or months, the increased cerebrospinal fluid pressure slowly return to normal.

Brain abscess

Otitis media, despite the benefits of antibacterial therapy, continues to be a frequent cause of brain abscess. Otitic brain abscess occurce in the temporal lobe twice as often as in the cerebellum. As a rule, otitic brain abscess is single. In rare cases, otitic brain abscess may be multiple, it may become multilocular and involve the parietal and occipital lobes and it sometimes occurs primarily in the parietal lobe rather than in the temporal lobe or cerebellum.

Symptoms and Signs

The symptoms of the first stage of initial encephalitis rarely last more than a few days and are generally mild, but they are sufficiently characteristic to alert the keen clinician to the possibility of beginning brain involvement in a patient with otitis media. A chill or chilly sensation, following by a slight or moderate rise in temperature lasting several days frequently heralds the invasion of the brain. Headache and nausea, sometimes with non projectile
vomiting, are often present. If observed closely, the patient may appear apathetic, drowsy, or irritable. In children a convulsion may be the first evidence of cerebral involvement.

As a rule, there are no localizing neurologic changes in the stage of initial encephalitis. Should there be a localized meningeal reaction, there may be slight stiffness of the neck and the spinal fluid will show a slight to moderate increase in cells and protein but without organisms and with a normal glucose content. The symptoms of the second latent or quiescent stage, as its name implies, are minimal or absent in many cases. In other cases malaise, poor appetite, intermittent headache, and slight temperature elevation may persist with listlessness, drowsiness, slowed cerebration and fretfulness or, irritability due to continued encephalitis. The latent or quiescent stage lasts from 10 days to several week or rarely, several months. As a rule, there are no neurologic signs and the number of cells in the spinal fluid diminishes or returns to normal.

The symptoms and signs of the third stage of manifest expanding abscess are due to cerebral compression caused more edema and encephalitis around. As the encephalitis and edema fluctuate from hour to hour and day to day, so the symptoms and signs may come and go. Therefore repeated frequent examination of the patient is often necessary to make the diagnosis of a brain abscess.

The symptoms and signs of an expanding brain abscess are of two types: those due to generalized increased intracranial pressure and those due to localized pressure on brain centers. The most constant generalized symptom of brain abscess is severe and usually continuous headache. Projectile vomiting is common and characteristic. Intermittent slowing of the pulse due to pressure on the vagus center in the brain stem is more common than Cheyne Stokes respiration due to pressure on the respiratory center. The temperature may be slightly elevated, normal, or subnormal. Apathy and drowsiness may be accompanied by disorientation. Jacksonian convulsions and ocular paralysis with pupillary changes may occur.

The most constant signs of increased intracranial pressure are in the eyegrounds, occurring in about half the cases, with blurring of the disc margins, hyperaemia or papilloedema. The spinal fluid is rarely normal in brain abscess and usually sows a slight increase in cells and protein. Spinal fluid should be withdrawn by lumbar puncture cautiously and in small amounts in cases of suspected temporal lobe abscess and only by ventricular puncture in case of a suspected cerebellar abscess to avoid herniation of the brain stem into the foramen magnum.

Symptoms and signs of increased intracranial pressure are more constant and definite in cerebellar than in temporal lobe abscess because of the restricted space of the posterior fossa and its proximity to the brain stem. In addition to the slowed pulse and cheyne stokes respiration, there may be elevation of blood pressure due to pressure on the brain stem.

The most constant symptom of a left temporal lobe abscess in a right handed patient is aphasia, usually of the naming variety, so that the patient cannot remember the name of a familiar object, such as a pencil or pen, although he or she knows perfectly well its purpose and quickly recognizes the correct name when he or she hears it. The next most common focal symptom is paresis of the face and mouth on the opposite side. Visual field defects may be found, although these are not easy to demonstrate in a confused or lethargic patient.
The most constant symptom and sign of a cerebellar abscess is ataxia on the same side, most easily demonstrated for the hand and arm. The patient does poorly on the finger to nose test, there is decreased ability to alternate movements rapidly (adiadochokinesia) and there may be a coarse intention tremor. Muscular hypotonia and weakness on the involved side are present without paralysis. The gait is ataxic with a tendency to fall toward the diseased side. Especially characteristic is a spontaneous nystagmus that fluctuates in degree and direction and is often vertical or oblique. A striking symptom of cerebellar abscess is rapid emaciation of the patient despite a fair appetite, probably owing to pressure on vegetative centers in the brain stem.

**Diagnosis**
The suspicion of brain abscess or lateral sinus thrombosis arises in any patient with chronic suppurative otitis media with cholesteatoma who has a chill followed by a low grade fever and headache. If after a latent period symptoms and signs of increased intracranial pressure develop, a brain abscess probably exists. When localizing symptoms and signs occur there is almost the certainty of an abscess. In some cases, a latent brain abscess becomes manifest soon after a radical mastoidectomy with severe headache and fever followed by localizing symptoms. For this reason, a computed tomographic scan of the brain should be obtained in any case in which there is a suspicion of an intracranial complication. This is particularly true in case in which there is a suspicion of an intracranial complication. Computed tomographic scanning is currently the most valuable diagnostic aid for the detection of intracranial space occupying lesions. Computed tomographic scans are performed with and without contrast enhancement.

**Treatment**
The patient is admitted to the hospital and a computed tomographic scan is obtained. When the diagnosis is confirmed, the patient is given intravenous antibiotic therapy. The choice of antibiotic is based on isolated organisms along with the presumption that anaerobic bacteria are involved. Intravenous dexamethasone is given to reduce brain edema, the ear is cleaned and antibiotic steroid drops instilled into the canal. Neurosurgical consultation is obtained.

The patient is taken to surgery as soon as possible. The abscess is approached through a craniectomy rather than through the ear. The abscess cavity is aspirated, and a specimen is submitted for culture and sensitivity and gram's stain. The cavity is then gently irrigated with saline and antibiotic solution. Some controversy exists as to the best management of the abscess cavity. The preferred method is to perform repeated aspirations and irrigations of the abscess cavity until resolution occurs. Alternatives include open incision and drainage of the abscess cavity and resection of the abscess in toto. Total resection is accompanied by higher incidence of post surgical seizures. These decisions are best made in conjunction with the neurosurgical consultant. When the patient has stabilized from the abscess drainage, the ear is approached surgically. This surgery is usually 3 to 4 days after the craniectomy although it can be sooner, depending upon clinical circumstances. Surgery is aimed at cleaning infection rather than reconstruction of hearing at this stage. Antibiotic therapy is usually continued for 4 to 6 weeks.

**Meningitis (leptomeningitis)**
This is a major and serious complication of middle ear infection and probably still the commonest intracranial complication.

**Clinical features**
The two most constant and reliable early clinical features are headache and neck stiffness. At first the headache may be localized to the side of the infected ear but soon becomes generalized and bursting. There is malaise and pyrexia often to 39oC. Initial neck stiffness shows as resistance to flexion, later rigidity or retraction develop. Mental hyperactivity,
fretfulness in children. Anxiety, punctuated by periods of drowsiness is usual in adults. At this stage, the tendon reflexes may be exaggerated. Photophobia is a constant characteristic symptom and before neck stiffness is marked, the patient may lie curled up away from the light. Vomiting, caused by raised intracranial pressure is also a feature.

As the condition proceeds, all these symptoms become more severe. The headache may be excruciating, and neck rigidity is marked with a positive Kering's sign, retraction and later on opisthotonus. The temperature remains uniformly raised with none of the swinging pattern, which used to characterize lateral sinus thrombosis. Gradually, the tendon reflexes become less marked and the abdominal reflexes may be lost.

Deterioration is marked by alternating delirium and stupor, passing finally into coma. The tendon reflexes disappear, and cranial nerve palsies develop. Eventually cheyne stokes respiration follows, with fixed dilated pupils, then coma and death.

Any local neurological signs, especially in the early stage should raise suspicion of cerebral abscess. Similarly epileptic fits do not occur with otherwise uncomplicated meninitis. The neck stiffness, which is so typical of the disease may be delayed for several days from the onset, especially if the first accumulation of exudate is vertical rather than in the basal cisterns.

**Diagnosis**

The diagnosis is made by examination of cerebrospinal fluid. Any patient with middle ear infection headache and neck stillness must undergo a lumbar puncture. At the same time, suspicion of other complications must always be entertained, and possible brain abscess and subdural empyemas need to be excluded, preferably by CT scanning. In the earliest stages of otogenic meningitis the only abnormality on lumbar puncture is a rise in fluid pressure above the normal 100-150 mmHg. As the infection proceeds white cells accumulate in the cerebrospinal fluid and the fluid becomes cloudy and then turbid in appearance. On cytological inspection, most of these cells will be found to be polymorphonuclear leucocytes, which are not normally present in cerebrospinal fluid. They increase in number to reach the range of 0.1-10 x 10^9/l (100-10000/mm^3), although with Staphylococcus epidermidis, count below 0.1 x 10^9/l may be met. Thus the protein content may rise from a normal 150-400 mg/l to a raised level of 2-3 g/l. The chloride content may fall from the normal 120 mmol/l to 80 mmol/l. The appearance of bacteria in the cerebrospinal fluid is accompanied by a fall of cerebrospinal fluid glucose levels from the normal value of 1.7-3.0 mmol/l to zero. Bacteriological examination of cerebrospinal fluid is first undertaken by direct examination after gram staining, and then by culture of the fluid. Despite positive diagnostic findings on cellular and biochemical testing, positive bacteriological diagnosis is by no means the rule, and so treatment cannot wait for, nor depend upon, it. The lumbar puncture findings are decisive when no mass shows on CT scanning, even without bacteriological identification. There is no other complication in which the cerebrospinal fluid sugar level is lowered, and few in which the white cell count is so high. A brain abscess, if leaking into the subarachnoid space may cause a huge rise of cerebrospinal fluid white cell count even to more than 50 x 10^9/l (50000/mm^3) and a subdural abscess to counts over 0.1 x 10^9/l In both of these the cerebrospinal fluid pressure may be raised but in neither is the cerebrospinal fluid sugar level reduced. In the presence of cerebrospinal fluid pleocytosis. CT scanning should exclude either form of abscess leaving a diagnosis of meningitis unchallenged. MRI may show typical signal changes in meningitis, although a CT scan will be normal.

**Treatment**

**Surgical**

As with most other complications, treatment of the intracranial sepsis should take precedence
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over management of the otitis media. Medical treatment of the meningitis is of paramount importance and any operation for the ear condition should, if possible, be deferred from several days until the patients general condition has improved. Years ago, before antibiotics offered hope of cure, appropriate ear surgery was undertaken as soon as the diagnosis had been made. Nowadays, urgent surgical intervention should be advised only if the expected response to treatment does not appear. Certainly deterioration or failure of response over 48 hours implies loculated infection in the mastoid, needing surgical drainage.

Medical

Because of its frequent role as a causative agent, haemophilus influenza must be a target of any regimen, and since more and more strains are becoming resistant to ampicillin, chloramphenicol is considered the first choice combined with ampicillin or penicillin.

Agents like to be effective against gram negative organisms must also be considered when the infection is secondary to chronic middle ear disease. In these categories are azlocillin, ticarcillin and some newer cephalosporins such as ceftazidine. All are less toxic than aminoglycosides like gentamicin.

Systemic therapy must be continued for at least 10 days after apparent clinical recovery. If bacteroides are found on anaerobic culture, metronidazole should be administered in a dose of 400 mg 8 hourly. Dexamethasone is now a days considered to be useful adjunctive therapy in the treatment of bacterial meningitis. The lumbar puncture used for diagnosis may be repeated several times to reduce intracranial pressure, possibly a second time in the first 24 hours and then daily until improvement is assured.

Subperiosteal abscess³

- Postauricular abscess: lies over the external surface of mastoid and is the commonest type, especially in children. The auricle is displaced outwards, forwards, and downwards. The postauricular sulcus tends to be retained. Pus has often tracked outwards through minute vascular channels in the suprameatal (Macewen's) triangle.
- Zygomatic abscess: pus escaping from zygomatic cells near the squama forms an abscess deep to the temporal muscle and makes a swelling above and in front of the ear. This may be confused with a parotid swelling. Pus escaping from more distal cells in the process forms an abscess superficial to the temporal muscle. Pus tracking outwards under the periosteum of the roof of the bony canal can reach the subtemporal position (Luc's abscess).
- von Bezold's abscess: perforation of the tip or inner surface of the mastoid may give rise to an abscess in the sternomastoid muscle (von Bezold's) or in the digastic triangle (Citelli's)
- Pharyngeal abscess: pus tracking from peritubal cells may form a parapharyngeal or retropharyngeal abscess.

Treatment

Mastoid exploration and drainage of abscess.

Facial paralysis

*In acute otitis media*

Facial palsy occurs in acute otitis media in that small proportion of patients (less than 10%) with a congenital dehiscence of the thin bony wall normally separating the horizontal part of the facial nerve canal from the middle ear mucosa.
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Treatment
The affection of the nerve is invariably a neuropraxia and full recovery of facial muscle function is to be expected after recovery from the preceding infection. This can usually be achieved by appropriate systemic antibiotic treatment but occasionally myringotomy or more rarely, cortical mastoidectomy could be needed.

In chronic otitis media
In chronic destructive middle ear disease, the facial nerve trunk may be exposed if its bony covering is eroded by cholesteatoma or by granulation tissue and osteitic disease in ears without cholesteatoma.

Treatment
Urgent operative exploration of the middle ear and mastoid region is mandatory to treat the chronic middle ear disease, proceeding if necessary to a radical mastoidectomy. Than facial nerve should be carefully exposed.

Suppurative labyrinthitis and serous labyrinthitis

Clinical features
A patient suffering from acute or chronic middle ear infection presents with violent prostrating vertigo and vomiting. Severe hearing loss of a sensorineural type is to be expected but will be adumbrated by the severe disabling vertigo, especially if there has been a preceding conductive impairment from middle ear disease. The patient lies immobile, on the side with the infected labyrinth upwards, avoiding any head movement.

Diagnosis
The clinical pattern described above is the same as that of sudden vestibular failure from any cause and suspicion of suppurative labyrinthitis primarily rests with precise recognition of the underlying middle ear infection.

Management
Treatment of suppurative labyrinthitis requires complete bed rest. Head movements should be avoided as much as possible. Vertigo and vomiting may be controlled by parenteral prochlorperazine or cinnarizine. If vomiting prevents hydration, Intravenous fluids must be infused. It is has been customary to recommend the administration of parenteral antibiotics.

Reference