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## REMOTE COMPLICATIONS OF OTITIS

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### Abstract

Otitis is an inflammation of the mucous membrane of the air chambers of the ear. The disease begins with the fact that the resistance of the immune system drops. In 80% of cases, a virus can act as a trigger, then it creates favorable conditions for bacteria to join and inflammation occurs. If a person observes such symptoms as: sharp pains in the ear of a shooting nature, which can cause pain in the temporal part of the head and jaw; increase in body temperature; hearing loss; pus from the ear, you need to see a doctor immediately. It cannot be allowed to reach the chronic stage, because the hearing may not recover because the auditory nerve is damaged. Otogenic complications occur when the infection spreads beyond the tympanic cavity.

**Keywords:** otitis, complications, diagnosis, treatment.

Otogenic complications can be divided into intratemporal and intracranial.

Most complications occur as a result of chronic otitis media. An exception is the relatively frequent inflammation of the mastoid in the course of otitis media in infants.

Intratemporal complications include mastoiditis, petrositis (inflammation of the pyramid of the temporal bone), labyrinthitis, damage to the facial nerve

Intracranial complications include epidural abscess, subdural abscess, brain abscess, sigmoid sinus thrombosis, serous and purulent meningitis, otogenic hydrocephalus.

Since the beginning of the widespread use of antibiotics, otogenic complications have become rare. However, they continue to threaten the life and health of children in clinical practice, so knowledge of them is important.

### Intratemporal complications Mastoiditis

This is most often a complication of acute, less often chronic otitis media. Mastoiditis most often occurs when inflamed tissues block the connection between the tympanic cavity and the cells of the mastoid process. Discharges from the process cannot be drained through the auditory tube or through perforation of the tympanic membrane. The inflammatory process spreads to the periosteum of the appendage (periostitis). A bone with the development of destruction (osteitis) can be involved in the process. Eventually, the pus may evacuate toward the planum mastoideum, apex of the pyramid (Betzold's abscess), or zygomatic process, forming an abscess. Sometimes it penetrates to the middle cranial fossa, sigmoid sinus or labyrinth. It can also eventually penetrate toward the apex of the pyramid, causing petrositis.

**Diagnosics.** Clinically, strong soreness during palpation of the mastoid process, especially in the area

of the apex, is characteristic. In the anamnesis — purulent otorrhea and hearing loss. A characteristic picture, especially in infants, is the protrusion of the auricle on the affected side and the overhang of the back-upper wall of the auditory canal in its bony part. An abscess may form in the area of the formed fistula. In case of suspicion, a CT scan of the temporal bone or an X-ray examination according to Schuller should be performed, which will show complete darkening of the cells of the process. In the case of acute otitis media, the pneumatization is correct, and in the case of chronic otitis media, the process is, as usual, sclerotic.

**Treatment.** The basis of the treatment of each mastoiditis is the use of broad-spectrum antibiotics. In acute cases, antibiotic therapy should cover the most frequent pathogens that occur in acute otitis media, namely: *Haemophilus influenzae*, *Streptococcus pneumoniae*, *Staphylococcus aureus*, and anaerobes. In cases of chronic otitis media, staphylococcal infection and strains of *Pseudomonas aeruginosa*, *Proteus*, and anaerobes should be taken into account. It is indicated to quickly take a culture for the purpose of further appointment of targeted therapy.

Mastoiditis with chronic otitis media requires surgical treatment in addition to antibiotic therapy. It consists at least in a wide mastoidectomy with the removal of the focus of inflammation from the spaces of the middle ear. The principle is to create a wide drainage of the mastoid cavity to the tympanic cavity through thorough cleaning and expansion of the aditus ad antrum. The creation of an additional ventilation path through the opening of the recess of the facial nerve should be considered. External drainage is also important. It is obtained by introducing drainage into the mastoid process and a typical ventilation shunt into the tympanic cavity (Bluestone, 1988). In the case of cholesteatoma, the surgical principles are discussed above.

In the case of mastoiditis, which is not complicated by bone involvement, fistula or abscess, conservative tactics are widely used in children. Intravenous antibiotic therapy and paracentesis or tympanotomy are prescribed. If antibiotic therapy is ineffective, a wide mastoidectomy should be performed and drains should be inserted. It is not recommended to leave the mastoid cavity wide open after mastoidectomy until healing by secondary tension "per secundam intentionem". This exposes the small patient to unnecessary many weeks of patience associated with dressings of the wound behind the ear. Inserting a drain and suturing the wound, as usual, creates sufficient drainage and ensures effective treatment of the disease.

A special form of mastoiditis, which occurs practically only in children, is the so-called latent mastoiditis (mastoiditis latens), which can be the result of untreated acute otitis media or the result of conservatively treated acute mastoiditis (Soboczyński, 1962; Paparella, 1980; Wickham, 1990). It is characterized by retention of fever and dull pain in the ear, as well as tenderness during palpation in the projection of the mastoid process with a normal tympanic membrane. Damage to the facial nerve occurs (Fukuda, 1998). The diagnosis is established on the basis of history, clinical manifestations and radiological examination of the temporal bone. Treatment is surgical.

#### **Petrositis (inflammation of the top of the pyramid)**

The cells of the apex of the pyramid can also become inflamed during otomastoiditis. Although this complication is very rare, it is still one of the biggest problems of otology. The diagnosis is established on the basis of clinical manifestations — acute or chronic otitis media with otorrhea, hearing loss, and Gradenigo's syndrome (pain behind the eyeball on the affected side, damage to the afferent nerve, otorrhea).

*Treatment* consists of intensive multi-week targeted intravenous antibiotic therapy, radical mastoidectomy and drainage of the top of the pyramid (transmastoid approach) may be required. Radiological examination (CT and MRI) of the temporal bone is indispensable for diagnosis and possible planning of the operation.

#### **Labyrinthitis**

A distinction is made between serous (non-purulent) and purulent labyrinthitis.

With serous labyrinthitis, bacterial toxins enter the inner ear, causing cochlear and vestibular disorders. With purulent labyrinthitis, bacteria enter the inner ear. Pathogens penetrate through a round or oval window, a congenital defect or a fistula on the bony labyrinth.

It can also occur after fractures of the temporal bone that pass through the inner ear.

*Labyrinthitis can be diagnosed* when dizziness, nystagmus, and peripheral balance disorders appear during otitis media, as well as varying degrees of sensorineural deafness. Diagnosis is especially difficult in young children. In such cases, objective hearing and balance studies play an important role. The appearance of manifestations from the side of the vestibular apparatus and sensorineural deafness is an indication for immediate treatment, since non-purulent labyrinthitis can

turn into a purulent form with complete loss of inner ear function.

*In differential diagnostics*, the most important thing is the assessment of hearing and the function of the labyrinth. With purulent labyrinthitis, the neurosensory organ of hearing is always destroyed, and the function of the labyrinth is lost. An important manifestation that indicates the progression of the disease is a change in the direction of nystagmus. Initially, it has a direction towards the diseased ear (irritation), and when it changes to the side of the diseased ear, this indicates a loss of function of the diseased labyrinth. An important symptom is the detection of nystagmus and dizziness, which appear when the pressure changes in the auditory canal (fistula symptom). This may indicate a bony labyrinth fistula. This is not a clear manifestation, as it can be both false positive and false negative. Therefore, it is important to confirm the destruction in the CT study of the temporal bones (photo 21).

In case of suspicion of purulent labyrinthitis, it is necessary to perform a spinal tap and examine the cerebrospinal fluid. Purulent labyrinthitis is especially dangerous, because the infection can spread along the aqueduct of the cochlea, pinna or internal auditory canal and turn into purulent meningitis.

*Treatment* should at least include mastoidectomy with ear drainage and broad-spectrum antibiotics to prevent disease progression and intracranial complications. Labyrinthectomy is indicated only in cases of complete loss of function of the inner ear and confirmation of the spread of infection in the skull cavity, which appeared despite the prescribed treatment (detection of pleocytosis in the cerebrospinal fluid).

#### **Paresis and paralysis of the facial nerve**

It is most often a complication of chronic otitis media with cholesteatoma. It also occurs relatively quickly in the case of tuberculosis of the middle ear, less often occurs in acute otitis media. The cause is swelling of the nerve in places of destruction of its bone canal by cholesteatoma or granulations, or in places of congenital defects of the bone canal.

With acute otitis media, as usual, paracentesis or ventilation shunt is sufficient, as well as antibiotic therapy, which covers *H. influenzae*, *Str. pneumoniae* and *Moraxella catharalis*. The appearance of a complication in chronic otitis media requires urgent surgical intervention with cleaning and decompression of the involved part of the nerve.

#### **Intracranial complications**

Before the era of antibiotics, intracranial complications occurred in 2.3% of otitis cases. Two-thirds of them occurred as a result of chronic otitis media (Turener, Reynolds, 1931). Currently, the most common intracranial complication in children is meningitis. In a group of 29 children operated on for intracranial complications, Juselius (2004) found meningitis in 14, epidural abscess in 8, thrombophlebitis of the sigmoid sinus in 5, and temporal lobe abscess in 2.

#### **Meningitis**

It can occur both with acute and chronic otitis media. The infection can spread from the ear:

- Due to the contact of inflamed tissues with the meninges and the subarachnoid space

- From another complication (epidural abscess, brain abscess, phlebitis of the sigmoid sinus, purulent labyrinthitis)

- Hematogenous
- After fractures of the temporal bone

In case of acute otitis media, the infection to the meninges most often penetrates hematogenously. Eavey (1985), examining 16 temporal bones from 8 children who died of meningitis, testified the coexistence of acute otitis media in 14 of them. However, he found no evidence that the infection had traveled to the meninges directly from the middle ear. Djerić et al (1994), examining the ears of children who died of purulent meningitis with acute otitis media and comparing them with a control group of children with only otitis media, demonstrated inflammatory changes in the middle ear and in the perilymphatic spaces and cochlear aqueduct. This suggests that this route of infection often occurs in children.

Children with congenital defects of the middle ear and facial skull are especially prone to meningitis during otitis media, as they often have pathological connections between the middle ear and the middle or back cranial fossa.

In meningitis, which occurs with acute otitis media, the most common pathogens in children are *Haemophilus influenzae* type b and *Streptococcus pneumoniae*, in chronic otitis media — *Proteus*, *Pseudomonas aeruginosa*, *Escherichia coli*, and anaerobes.

**Diagnosics.** Regardless of the etiology, the manifestations of meningitis are similar - fever, stiffness of the muscles of the back of the head and impaired consciousness. Focal manifestations in the form of paresis, aphasia, or damage to the cranial nerves often appear. For diagnosis, examination of cerebrospinal fluid and detection of pleocytosis, elevated protein level, and reduced glucose level are mandatory. It is necessary to perform a radiological examination — CT scan of the temporal bones, which makes it possible to assess bone destruction and the spread of inflammatory changes in the ear, as well as MRI, which makes it possible to exclude the coexistence of other complications and to assess the presence of inflammatory changes in the soft tissues of the brain.

**Treatment.** Treatment begins with intravenous empiric antibiotic therapy in maximum doses. It should be changed to a target one after receiving the results of a bacteriological examination (culture of blood, secretions from the ear, cerebrospinal fluid). In the case of acute otitis media, a wide mastoidectomy with drainage of the middle ear spaces should be performed. In children with chronic otitis media, even radical surgery combined with labyrinthectomy may be necessary if purulent labyrinthitis is detected.

#### **Epidural abscess**

As usual, it is a consequence of the destruction of the bone adjacent to the meninges and the cause of which is a cholesteatoma or granulation. Then inflammation occurs and pus accumulates between the dura mater and the adjacent temporal bone.

An epidural abscess can be asymptomatic. If there are manifestations, then they are uncharacteristic and consist of varying degrees of pain in the ear and head

in the temporal region, as well as subfebrile fever. An abscess is often discovered accidentally during a planned mastoidectomy during the treatment of chronic otitis media. Analysis of cerebrospinal fluid — without deviations. The diagnosis is based on a radiological examination of the ear — CT of the temporal bone with contrast or MRI of the head.

**The treatment** is surgical and consists in performing mastoidectomy with elimination of the focus of inflammation and wide opening of the dura mater to healthy limits with drainage of the abscess.

#### **Subdural abscess**

It is formed by direct spread of infection or by thrombophlebitis of small veins. It consists in the accumulation of pus in the hypothetical space between the dura mater and the arachnoid meninges.

Unlike an epidural abscess, a child with a subdural abscess has a pronounced dynamic course of infection. As usual, the fever is high against the background of the poor general condition of the child. Focal manifestations resemble the manifestations of a rapidly growing brain tumor. There is a severe headache in the temporal-parietal region. Focal manifestations may include convulsions, paraparesis, pathological drowsiness, impaired sensitivity, dysphagia, nystagmus of the central type, and rigidity of the muscles of the back of the head.

**Diagnosis** is confirmed by radiological examination (CT, MRI). Moderate pleocytosis with a predominance of neutrophilic granulocytes with normal glucose values is found in the cerebrospinal fluid. The pressure of the cerebrospinal fluid is increased.

**Treatment** consists of intensive intravenous antibiotic therapy and neurosurgical drainage of the abscess. Elimination of the inflammatory focus and drainage of the mastoid process can be performed simultaneously or postponed until the patient's condition improves after neurosurgical intervention. The mortality rate is high, and more than half of the children have neurological deficits of various degrees after treatment (Bluestone, 1988).

#### **Brain abscess**

Szmeja et al (1991) reported that among 75 patients operated on for brain abscess, 19 were children. In this group, 3 of them arose as a complication of chronic cholesteatoma otitis media, and the rest - acute otitis media. Among all age groups, brain abscesses are most often found in infants and young children (Brewer, 1975). In children, as in adults, 2/3 of abscesses are localized in the temporal lobe, and only 1/3 — in the cerebellum (Bordley et al, 1985)

A brain abscess forms directly around the inflammatory process in the temporal bone or during the spread of another intracranial complication. Over time, the initial inflammatory swelling is replaced by liquid necrosis, and in a few weeks, a clear capsule of the abscess is formed. The natural course of the abscess in the worst case scenario ends with its spontaneous breakthrough to the ventricles of the brain or to the subarachnoid space.

**Manifestations.** The classic 4 periods of brain abscess development (initial, hidden, overt and final) are now rarely observed, as the clinical picture has changed significantly due to widespread antibiotic therapy.

Manifestations of spread to the central nervous system usually appear about 1 month after acute otitis media or an exacerbation of chronic otitis media. General manifestations, such as fever and convulsions, are uncharacteristic and may not be present at all. When the intracranial pressure increases, severe headache, nausea, vomiting, and congestion of the optic nerve appear. There may be bradycardia and heart rhythm disturbances. Convulsions and loss of consciousness occur. Inflammatory manifestations depend on the location of the abscess. Abscess of the temporal lobe — aphasia, visual impairment, paraparesis, severe disorders of the process of reading, counting, and writing. Manifestations are more pronounced when localized in the dominant hemisphere. With cerebellar abscess - dizziness, imbalance, central type nystagmus, directed towards the abscess. Violations of muscle tone and coordination of movements are also observed.

**Diagnosics.** Currently, radiological methods of examination — MRI of the head and CT with contrast — play a decisive role in diagnosis. If an abscess is suspected, these examinations should be repeated, because the formation of an abscess is a dynamic process, and the formation of an abscess capsule takes several weeks.

**Treatment.** Abscess treatment consists of broad-spectrum antibiotics, neurosurgery to remove or drain the abscess, and often-simultaneous otosurgery to remove and drain pathology in the middle or inner ear spaces. Only conservative treatment is rarely used.

**Forecast.** The prognosis for abscesses is uncertain, and neurological deficits often remain after treatment.

#### **Thrombophlebitis of the cavernous sinus**

It can be a consequence of both acute and chronic otitis media. It occurs when the infection spreads from the adjacent mastoid process. Inflammation gradually covers all layers of the sinus wall. The inflammatory condition initially causes the formation of a wall thrombus, which, gradually growing, can completely close the lumen of the sinus. A thrombus can become infected, and particles that break off from it can cause sepsis and abscesses in distant parts of the body.

**Typical manifestations** are septic fever, soreness when pressing on the neck on the affected side, swelling and soreness of the top of the mastoid process at the exit of the mastoid emissary vein (Griesinger's symptom). Positive sign of Queckenscheidt (increased intracranial pressure when pressing on the jugular vein on the opposite side).

**The diagnosis** is established based on the clinical picture and radiological examination (CT of the temporal bones and MRI with contrast or vascular MRI).

**Treatment.** Conservative treatment consists in prescribing parenteral antibiotic therapy. In some cases, anticoagulants are also indicated. Surgical treatment consists of at least a wide mastoidectomy with exposure of the sinus walls and drainage of the perisinus abscess. Removal of the infected thrombus and ligation of the jugular vein is rarely necessary and indicated in cases of metastatic abscesses (Shambaugh, Glasscock 1980; Ooi, 2003).

#### **Ear hydrocephalus (otitic hydrocephalus, pseudo-tumor cerebri)**

Symonds first described this disease in 1931. He testified that the presence of an inflammatory focus in the temporal bone could be the cause of the so-called ear hydrocephalus. The etiology of the disease is still unclear. It is characterized by high intracranial pressure (>200 mm Hg). Complaints include severe headache with nausea and vomiting, as well as visual disturbances (congestive disc of the optic nerve). In the absence of treatment, there is a risk of blindness.

**The diagnosis** is established based on history, clinical manifestations (coexistence of ear disease and increased intracranial pressure), as well as radiological examination in order to exclude other causes of the disease.

Antibiotic therapy, drugs that reduce intracranial pressure, as well as surgical removal of the focus in the temporal bone (at least mastoidectomy and drainage) are used in the treatment.

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