CASE REPORT

Facial Nerve Paralysis, Labyrinthine, Fistula and Brain Abscess due to Chronic Otitis Media

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Despite the availability of new antibiotics, chronic otitis media can still lead to major complications in developing countries. Except for mastoiditis, it is rare to see more than one complication in one patient simultaneously. We present the case of a 21 year old female who presented with facial nerve paralysis, labyrinthine fistula and brain abscess due to chronic otitis media. It is emphasized that preoperative findings such as facial nerve canal defect and labyrinthine fistula should raise the suspicion of the possibility of development of further and more serious complications such as abscess of the brain.

Development of new antibiotics reduced the incidence of chronic otitis media (COM) complications. However, in developing countries these complications are still frequent. These complications may cause symptoms that are similar to typical chronic otitis media symptoms and result in delays or difficulties in diagnosis.

CASE REPORT

A 21-year old female patient was referred to our clinic with left aural discharge, hearing loss, vertigo, headache and difficulty closing the left eye. She had a 15 year history of otologic discharge with difficulty hearing, 3 months history of vertigo and headache, and 1 week history of difficulty closing the left eye.

On physical examination, the patient was conscious and cooperative. We detected House - Brackmann Grade 5 left peripheral facial nerve paralysis. Otologic examination showed abundant discharge from the left ear. The tympanic membrane was retracted and there was a 2-3mm perforation at the attic filled with hemorrhagic granulation tissue. Fistula test was negative and no spontaneous nystagmus was detected. There was a left side lateralization at gait test. Rhomberg test was negative. Pure tone audiometry results of the left ear were 92 dB HL for air and 40 dB HL for bone conduction. This gap was greater at high frequencies. Radiologic examination with high resolution temporal bone tomography (CT) of the ear showed obliterated attic and antrum, destructed ossicular chain, defects on scutum, tegmen and osseous part of the horizontal semicircular canal (SSC) on the left side. The cranial parenchyma was normal (Figure 1).

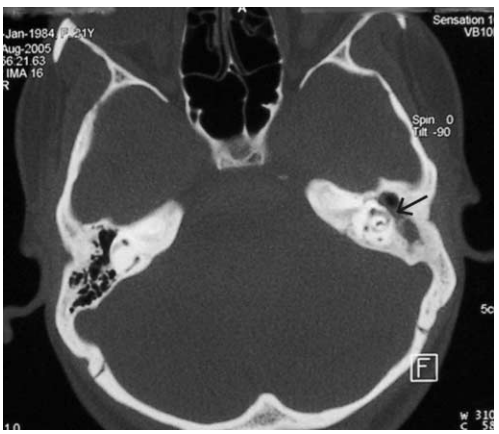

Figure-1: Destruction of the left horizontal semicircular canal on high resolution temporal bone tomography.

The patient was hospitalized and started on medical therapy with 20.000.000 u/day intravenous crystallized penicillin and 1mg/kg/day intramuscular dexhamethasone. The following day she underwent left radical mastoidectomy and transmastoid facial nerve decompression. During the operation, we observed that the mastoid antrum, aditus and epitympanum were covered with dense hemorrhagic granulation tissue. A tegmen defect approximately 5x10mm and a horizontal semicircular canal defect 2x3mm were detected. Ossicles were eroded except for the head of the malleus. Tympanic segment of the fallopian canal was eroded and the epineurium of the facial nerve was found to be edematous (Figure 2).

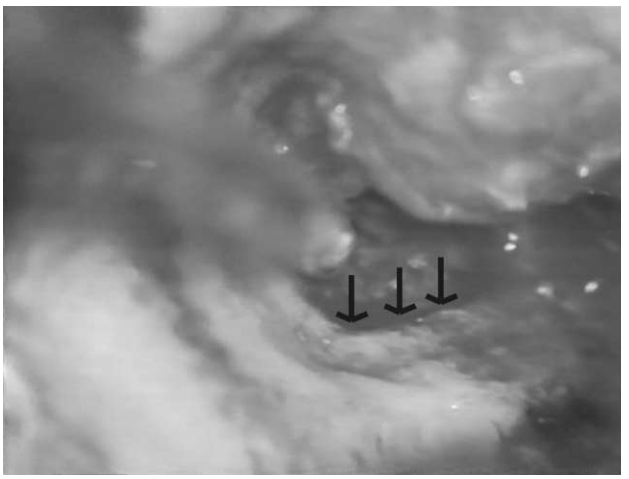

Figure-2: Defect on the tympanic segment of the facial nerve canal observed peroperatively. (Arrows)

Vertical segment of the canal was decompressed 5-6 mm up to the normal epineurium. Temporal fascia was laid over the facial nerve and the horizontal SSC defect. Tragal cartilage was used to support the defect at the tegmen. Antibiotic and steroid treatments were continued. Two days following the surgery, facial paralysis seemed to be partially improved; however the patient complained from vertigo attacks with 10-15 seconds of duration. Horizonto-rotatory nystagmus beating to the left side was observed. On postoperative day 5, facial paralysis was House-Brackmann Grade 2. Vertigo attacks continued. These attacks were thought to be related to the labyrinthine irritation which could have been due to the infection and surgery. On postoperative day 10, the vertigo symptoms were more stable and the patient was

discharged. She was scheduled for weekly controls of facial nerve functions, vertigo attacks and the radical cavity. During weekly controls, she complained of mild, intermittent vertigo without any aural discharge. On postoperative day 30, the patient was readmitted with sudden onset of fever, headache, nausea, vomiting and ataxia. CT revealed two adherent cystic lesions located at left cerebellar vermis and hemisphere (Figure 3). The neurosurgery team drained these abscesses using left paramedian suboccipital approach. Patient was discharged on postoperative day 7 with no sequel and is currently under control.

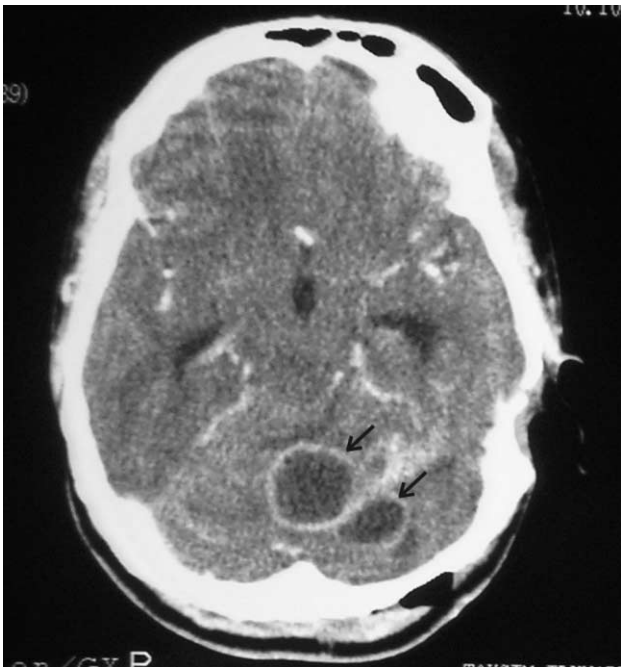

Figure-3: Cranial CT showing cystic lesions concordant with cerebellar abscess.

Discussion

Due to its complications, COM is a very serious disease. The most common intracranial complications include meningitis, brain abscess and lateral sinus thrombosis [1-3]. The most common extracranial complications include facial nerve paralysis, subperiosteal abscess and labyrinthitis. The prevalence of the intracranial and extracranial complications was reported to be between the range of 0.20-1.97 % and 0.45-1.35 %, respectively [1-3]. Except for mastoiditis, it is rare to see more than one complication in one patient simultaneously. In a retrospective study of 17144 chronic otitis media patients, the ratio of patients with three simultaneous complications was reported to be between 0,012% - 0,017%. [4].

Complications of COM are usually caused by progressive and relentless erosion of bone, thus exposing the risk of damage to the facial nerve, the labyrinth and the dura [1]. Generally bone destruction is due to granulation tissue formation and cholesteatoma [2-5].

Facial nerve paralysis, most likely, is the result of a combination of pathologies including osteitis, bone erosion, compression, direct inflammation and infection of the nerve itself [2-5]. The tympanic segment of the facial canal is the most common site of involvement. In our case, the primary cause of facial paralysis was possibly the granulation tissue which caused inflammation and pressure around the nerve.

Labyrinthine fistula has been long recognized as a complication of COM, which can result in progressive sensory hearing loss or permanent loss of inner ear function [6]. It represents an erosive loss of the endochondral bone overlying the semicircular canals without loss of the perilymph. Its prevalence was reported to be around 2,9 - 12,5% [6,7]. In nearly 90% of the patients, the fistula is located in the horizontal semicircular canal (HSCL), its dome being the most common site [7]. Preoperative detection of a fistula is based on history, clinical examination and radiographic findings. Patients recount relatively brief periods of imbalance, disequilibrium or vertigo but have normal equilibrium most of the time [7]. In the literature, the presence of vertigo ranges from 32 - 90% of patients [7,8]. The fistula test is the only method to detect a fistula preoperatively on physical examination. However, the fistula test results are reported as positive in only 22-72% of patients with LF [6,7]. Therefore, as we have experienced in our case, one should not rule out the possibility of a LF, even if the fistula test is negative. CT scanning provides preoperative evidence suggesting a LF convenience ratio as up to 90% [4].

The incidence of brain abscess has decreased significantly since the advent of antibiotics. Currently it is an uncommon complication of COM. Brain abscess begins when bacteria propagate in and around venous channels leading from the mastoid into the adjacent brain parenchyma [8]. The temporal lobe is most frequently and the cerebellum is the second most common involved site of the brain tissue [2]. Brain abscess develops through four of the following clinical stages over a period of weeks or months. Invasion; clinical symptoms are subtle during this stage and diagnosis may be missed. Localization; this stage may last for weeks and the patient may be asymptomatic. Enlargement; this is the manifest abscess period that results in mass effect. Termination; the abscess is ruptured in this stage. It is rapidly progressive and frequently fatal [2]. Cerebellar abscess is often accompanied by horizontal nystagmus, dysmetria, dysdiadokokinesia or action tremor [8]. In the manifest abscess stage, patients usually present with fever (62%), headache, nausea and vomiting (53%), papilledema (70%) and differing stages of impaired consciousness (56%) [2]. CT scan with intravenous contrast shows a hypodense area encircled by an enhancing ring. Magnetic resonance imaging (MRI) is helpful and more sensitive than CT [8].

In our case, when the patient was first referred to our clinic, there were no signs of cerebellar involvement at the CT scan. The abscess, which most likely had developed due to the defect of the tegmen, was probably at either the invasion or localization stage at this time. However, since we thought that the patient's symptoms were due to the defect of the horizontal SSC, we have not seen a need for a more detailed imaging technique preoperatively. Similarly, in the postoperative period, ongoing intermittent vertigo symptoms were attributed to the labyrinthine fistula and labyrinthitis; which estranged us from the possibility of the presence of another complication, and thus asking for an MRI.

Conclusion

If they are present together in one patient, it can be difficult to differentiate intracranial and extracranial complications of chronic otitis media; as they have similar symptoms and signs. The asymptomatic invasion and localization period of the brain abscess should be taken into account while treating extensive COM cases with bone destruction. These COM patients should be followed closely after the treatment and a control CT may be requested in the early postoperative period.

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