INTRACRANIAL COMPLICATIONS AS A MANIFESTATION OF CLINICAL ONSET IN A PATIENT WITH PROLONGED INSIDIOUS EVOLUTION OF EAR PATHOLOGY

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ABSTRACT

Infectious processes and ear cholesteatoma may have prolonged, insidious evolution (for years), leading in some cases to life threatening intracranial complications and/or severe neurologic risk (1). The pathways for spreading the infection to the intracranial structures are: directly by bone erosion, by blood or by preformed anatomical spaces (2).

The cases in which the insidious middle ear and mastoid pathology is revealed directly by the manifestations of intracranial complications require unhesitating interdisciplinary therapeutic approach through collaboration between ENT, neurology, neurosurgery, radiology and infectious diseases (1).

We present the case of a 58 years old patient who presented complex neurological deficits as a way of clinical onset for an insidious suppurative middle ear and mastoid pathology.

Key words: middle ear and mastoid suppuration, cholesteatoma, intracranial complications, neurological deficits

INTRODUCTION

Intracranial complications of middle ear and mastoid suppurations are a real, life threatening pathology; the diagnosis and treatment of these pathology requires the collaboration of several medical and surgical specialties: ENT, neurology, neurosurgery, infectious diseases and radiology (2).

In 2013 in the Institute of Phonoaudiology and Functional ENT Surgery „Prof. Dr. D. Hociotă"-Bucharest, we recorded a record number of 7 patients presenting with intracranial complications for middle ear and mastoid suppurations. It was the highest rate recorded in the last 20 years. Of the 7 cases recorded, we found that the most common intracranial complication was meningitis (6 cases), followed by lateral sinus thrombosis (5 cases), cerebellitis (3 cases), CSF fistula (3 cases) and osteonecrosis with dural necrosis (1 case illustrated in Figure 1). In 2013, unlike previous years, we recorded no otogenic brain abscess.

Patient outcomes after medical and surgical treatment was favorable in all cases without neurological deficits.

CASE REPORT

We present the case of a 58 years old patient, known with neglected hypertension, as well as bilateral hearing loss for many years, uninvestigated, who presented to the neurology emergency room the following signs and symptoms: diffuse headache, stiff neck, confusion, psychomotor agitation, left peripheral facial palsy, flaccid tetraparesis and abolished osteo-tendinous reflexes. The headache had started 3days before and the remaining symptoms set up a few hours before presenting to the emergency room. The cranio-cerebral CT scan performed in the emergency department ruled out the presence of recently constituted cerebrovascular lesions. Subsequently, the results of laboratory tests, including lumbar puncture, set the diagnostic

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of meningoencephalitis. On admission in the infectious diseases service the patient presented the above-mentioned symptoms without fever, which shows no immunological reactivity; spontaneous breathing with oxygen mask, SpO2 99% polypnea; vesicular murmur present bilaterally, without superimposed crackles, blood pressure of 167/116 mmHg, heart rate of 115 bmp; mobile abdomen with breathing, no signs of peritoneal irritation; present diuresis, of approximately 3,000 ml (by Catheter).

Biology: leukocytosis (21,600/mmc) with neutrophilia, inflammatory syndrome (fibrinogen 1006 mg / dl), mild thrombocytopenia (140000/mmc), urea 74.4 mg / dl, creatinine 1.3 mg / dl, mild hepatic cytolysis (TGP 74 U / l SGOT 117 U / l), cholestasis (total bilirubin 3.6 mg / dl, 285 GGT U / l alkaline phosphatase, 256 U / l) negative HIV serology, anti-HCV Ac negative, anti-HBs Ac positive. Blood cultures taken - negative. Ear discharge smear describes Gram positive cocci in diplo, but cultures revealed no pathogenic characters. Lumbar puncture performed on admission: muddy-looking LCR with „cabbage soup" aspect, > 10,000 elements/ mmc (acetic acid) with 85% neutrophils, 5% monocytes, 10% lymphocytes, polymorphic, round-elongated Gram positive cocci in diplo- with polymorphic aspect, some of them encapsulated; Pandy reaction 3 + + + proteinorachia 1,647 mg / dl, glicorachia 13 mg / dl.

Abdominal ultrasound: gallstones (gallbladder lithiasis echo of about 1.5 cm) mild splenomegaly.

CXR: bilateral bronchopneumonia outbreaks, enlarged aortic configured heart.

Antibiotic treatment was initiated with Meropenem 2g/8h, Vancomycin 500mg/6h, Dexamethasone 10mg/6h, stress ulcer prophylaxis, anticoagulant treatment for lateral sinus thrombosis, antihypertensive medication (Clonidine, Enalapril), fluid resuscitation, enteral and parenteral nutrition and symptomatic treatment.

The **second day** after admission neurological examination shows: + + + stiff neck, swinging movements of the eyeballs, intermediate pupils; persistance of the flaccid tetraparesis, persistance of left peripheral facial paresis, abolished osteo-tendinous reflexes. Also, the patient presented minimum left otorrhea.

The patient required 24 hours of endotracheal intubation and mechanical ventilation.

On the 5th day of hospitalization and treatment, the clinical and biological evolution was improving progressively but the patient was still presenting minimum otorrhea of the left ear and mild left peripheral facial paralysis. Lumbar puncture: no. of elements 450/mmc, 90% neutrophils, albuminorachia 163mg/dl, glicorachia 11mg/dl.

ENT consult was required and revealed a rough, bony, almost complete stenosis of the left external ear canal and minimal purulent otorrhea, without being able to see the eardrum or middle ear structures. A CT scan of the temporal bone was performed which revealed suppurated left mastoiditis, that had perforated intracranialy, sigmoid sinus thrombosis and left cerebellitis, raising the suspicion of otogenic meningoencephalitis. Based on the clinical and CT findings, we establish the indication for surgery and a left radical mastoidectomy was performed.

Intraoperatory findings: an osteoma on the anterior wall of the left external ear canal which caused an almost complete obstruction of the meatus; a large mass of cholesteatoma and polyps, as well as abundant, fetide, purulent secretions, in the middle ear and mastoid; the absence of the tympanic membrane and ossicular chain due to infectious necrosis; osteitis extended to the mastoid; in the posterior cerebral fossa: bone necrosis of approx 1.5/2 cm; underneath it there was an area of dural necrosis, with a minimal CSF fistula.

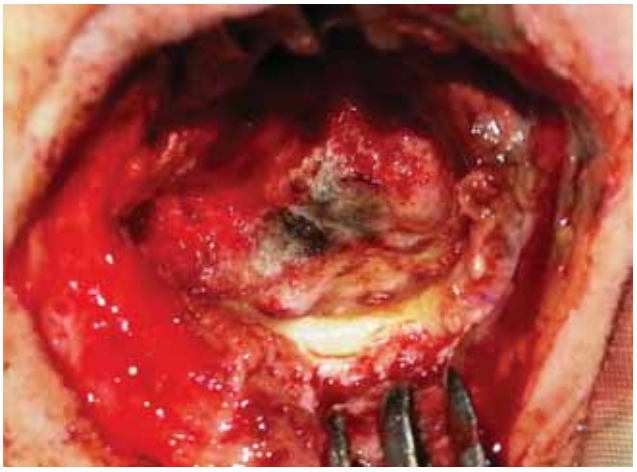

FIGURE 1. *Intraoperative image after the excision of pathological lesions: left tympanic-mastoid dissection cavity presented osteonecrosis and dural necrosis in the posterior cranial fossa.*

Postoperative care: daily otomicroscopic examination and aspiration until the epithelialization of the cavity was complete and continous medical treatment as mentioned above, for 8 more weeks.

The evolution of the patient's condition was slowly favorable until the neurological symptoms and signs remitted.

We mention that the patient was treated by a physiokinetotherapist in order to recover his neurologic deficits.

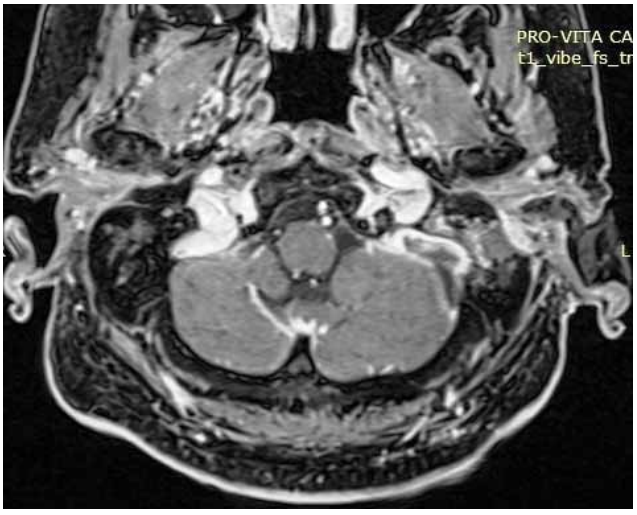

FIGURE 2. *3 days after surgery the MRI showed persistent, left sinus thrombosis and left cerebellitis.*

DISCUSSIONS

In this case the ear lesions evolved slowly, destructive, to the intracranial structures, without early signs or symptoms. This situation was the result of the existence of the bone malformation of the external ear, which determined the ear canal stenosis and did not allowed the drainage of the secretion and cholesteatoma. 4 of the 7 cases had no clinical manifestation until intracranial complications occurred.

The evolution of the neurological manifestations was oscillating during the medical and surgical treatment.

Fortunately, in this case, collaboration between specialists in neurology, neurosurgery, infectious diseases, imaging and ENT led to the healing of the middle ear and mastoid suppuration, cholesteatoma excision and intracranial complications remission. Various authors describe cases of permanent neurological sequelae and mortality caused by the middle-ear and mastoid suppurations (3).

CONCLUSIONS

Cholesteatoma associated with middle ear and mastoid suppuration can evolve for long periods of time (years) with few or without any symptoms at all and can manifest clinically only in the phase of intracranial complications (4). Therefore, any sign or symptom of ear pathology must require a prompt ENT consult; also certain neurological signs and symptoms may require thorough ENT examination.

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