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Case Report

Facial Nerve Paralysis in a 54-Year-Old Woman with Exacerbated Chronic Suppurative Otitis Media and Chronic Mastoiditis: A Case Report

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Abstract: We present the case of a 54-year old woman diagnosed with chronic suppurative otitis media, who was admitted to the ENT Department with four-week-old, sudden-onset, left-sided facial nerve paralysis, and ipsilateral otalgia and hemicrania. Physical examination revealed positive signs of acute postaural inflammation. The patient's facial nerve paralysis was scored as VI, according to the House-Brackmann scale. A cranio-facial computer tomography examination revealed mastoid cavity opacification, mucosal hypertrophy, and signs of chronic osteitis, with minimal mucous accumulation. The patient underwent a radical modified mastoidectomy with type-I tympanoplasty to verify the presence of a cholesteatoma, and to remove the offending lesions. Post-operatively, patient evolution was favorable, and prognosis remained encouraging. The patient's evolution will be followed by check-ups every three months to assess progress and benefits of the treatment.

Keywords: facial nerve paralysis; chronic suppurative otitis; chronic mastoiditis

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Introduction

Chronic suppurative otitis media (CSOM) is the result of a persisting middle ear infection associated with otorrhea and tympanic perforation lasting greater than two weeks [1]. Patients suffering from CSOM develop a protracted inflammatory response to an improperly-treated acute otitis media, during which inflammatory tissue forms in the middle ear and may invade the mastoid cavity [2].

According to the World Health Organization, the global incidence of CSOM ranges from 1% to 46%, with the highest rates in Western Pacific Countries. The incidence rate in the Eastern Mediterranean is 1.4% [3].

CSOM is divided into tubo-tympanic and attico-antral disease types, according to the area most affected by chronic inflammation. In tubo-tympanic disease, a permanent tympanic membrane perforation and, frequently, ossicular chain erosion or fixation will lead to hearing loss. The process of ossicular erosion is regulated by tumour necrosis factor-alpha, interleukin-2, fibroblast growth factor, and platelet-derived growth factor, which promote hypervascularisation, osteoclast activation, and bone resorption. On the other hand, attico-antral CSOM usually involves a primordial perforation typically located within the pars flaccida of the tympanic membrane, as such perforations are highly more likely to result in cholesteatoma formation within and beyond the atticus. Given the location of the perforation, it is also more likely that a persistent (non-cholesteatoma) inflammatory response, such as granulation tissue, may also extend into the mastoid cavity via the antrum [4].

Furthermore, in cases with marginal perforations and subsequent cholesteatoma formation, it most commonly occurs when there is a perforation in the posterior superior, or anterior superior quadrants of the tympanic membrane. It allows stratified squamous epithelium from the external auditory meatus to migrate into the middle ear, subsequently forming a cholesteatoma [5]. Significant and numerous complications may arise from cholesteatoma, and may be divided into intracranial (dural, cerebral, or cerebellar abscess, lateral sinus thrombosis, and meningitis), and extracranial (facial nerve paralysis, labyrinthine fistula, osteitis, and neck abscesses) [6].

Facial nerve paralysis may be secondary to a plethora of causes, ranging from supranuclear, nuclear, or infranuclear lesions, to compressive masses at the cerebellopontine angle, or lesions of the internal auditory or facial canals. Head trauma may also lead to nerve paralysis, as fracture lines forming across the temporal bone compress and inflame the facial nerve. The most common infectious (viral) cause of facial nerve paralysis is herpes zoster oticus, while chronic suppurative (bacterial) otitis media, with or without cholesteatoma, may also lead to nerve paralysis. Patient suffering from acute otitis media with associated facial nerve paralysis generally fare better than chronic patients, rarely requiring surgical decompression of the nerve. As with most nerve lesions, prompt treatment provides the most favorable outcome [7].

Imaging studies such as a high-resolution computer tomography of the petrous part of the temporal bone, along with surgical decompression of the nerve, are recommended in patients with CSOM with facial nerve paralysis [8].

Patients whose facial nerve paralysis is determined to be secondary to CSOM and chronic mastoiditis are advised to undergo one of several types of mastoidectomy. This procedure allows for inspection of important structures in the middle ear and mastoid cavity, such as the ossicular chain integrity and mobility, the state of the mastoid cavity and attical mucosa, and the facial recess. These structures, and several others, provide evidence of the extent of tissue damage. Relevant tissue samples removed during the procedure are further reviewed by pathologists in order to exclude malignancy [8].

Resolution of facial nerve paralysis following mastoidectomy is variable. Patients undergoing surgery within four weeks of paralysis onset generally show good results (a House-Brackmann score of II), while patients receiving surgical treatment after more than four weeks show variable recovery (scale grade of II, III, or IV). However, even patients receiving delayed surgical treatment show improvement more readily than those receiving none at all. Post-operative corticotherapy is routinely administered in order to maximize paralysis improvement [9].

Case Report

A 54-year-old woman presented with complaints of four-week-long history of abrupt-onset, left-sided facial paralysis, with ipsilateral xerophthalmia, otalgia, initially abundant otorrhea, and periauricular hemicrania. Personal and family history revealed a life-long unilateral left eye convergent strabismus, ischemic cardiomyopathy treated with double anti-aggregant therapy, and a 15-year history of ipsilateral tympanic membrane perforation, marked hypoacusis, and recurring, fetid otorrhea, for which the patient had previously received symptomatic treatment.

The patient's chronic treatment included Nitroglycerin 2.6 mg, Trimetazidine 35 mg, Rosuvostatin 10 mg, with Aspirin 75 mg and Clopidogrel 75 mg for cardiovascular mortality risk prevention following three previous NSTEMI acute coronary syndrome episodes, with the latest 8 months prior to the onset of facial nerve paralysis. The patient also had been prescribed Loratadine 5 mg for her seasonal allergies.

Physical examination revealed an erythematous, tender postaural area of swelling. Otoscopy and microscopy revealed a subtotal perforation of a partially-calcified tympanic membrane, and presently dry middle ear mucosa, with minimal mucoid secretions. The patient's facial nerve paralysis showed asymmetry at rest, no discernable voluntary movements, and a complete inability to close the left eye.

Tonal audiogram showed complete hearing loss across all frequencies in the affected ear, and a moderate sensorineural hypoacusis in the right ear (see Figure 1). The patient also made use of an endaural hearing aid in the left ear.

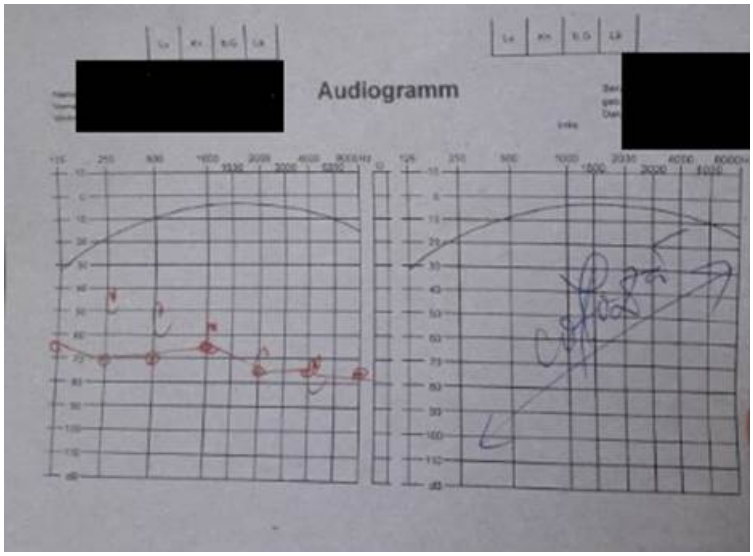

Figure 1. Pre-operative tonal audiogram, revealing moderate sensorineural hypoacusis of the right ear, and complete hearing loss in the left ear.

Cranio-facial computer tomography revealed an opacification of the left mastoid cavity, minimal mucoid secretion, and mucosal hypertrophy (see Figure 2).

Based upon the clinical and paraclinical findings, the diagnosis of exacerbated chronic suppurative otitis media with grade VI facial nerve paralysis on the House-Brackmann scale was reached, along with moderate right-sided sensorineural hypoacusis and severe left ear hearing loss.

Pre-operative treatment aimed to address the facial nerve paralysis included IV Hidrocortisone 200 mg b.i.d. and combined benfotiamine, cyanocobalamin, drotaverine, and

pentoxifilin administered once daily to improve cerebral circulation and hasten facial nerve recovery. Adjuvant drops with fluorometholone were applied to reduce xerophthalmia.

As per the pre-operative cardiology consult, Clopidogrel and Aspirin intake was interrupted 7 days and one day, respectively, while the patient was converted to Enoxaparin 4000 UI b.i.d. 3 days prior to surgery. Enoxaparin was restricted twelve hours prior to the mastoidectomy, and was resumed eight hours post-operatively, along with Clopidogrel and Aspirin. The overlapping therapy continued for the first 48 hours, under INR monitorization, until completion of the conversion.

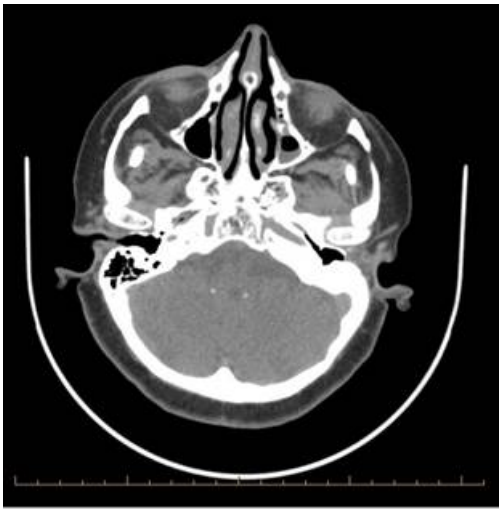

Figure 2. Transvese CT-section showing modifications of chronic mastoiditis, with cavity opacification and mucosal hypertrophy.

After seven days of pre-operative preparation, the patient's facial nerve paralysis and eye closure slightly improved, and facial expression was also partially regained. A new evaluation determined a pre-operative House-Brackmann score of V.

The patient then underwent a modified radical mastoidectomy with type I tympanoplasty. Intraoperative bone shaving indicated mastoid wall eburnation. The attical portion of the middle ear and mastoid cavity presented with minimal mucous accumulation and numerous granulation tissue deposits which were surgically removed. Middle ear inspection infirmed both osteitic lesions, as well as the presence of a cholesteatoma. Ossicular chain inspection revealed preserved mobility and continuity. The facial nerve was not exposed in the tympanic portion.

The surgery was carried out under general anesthesia with endo-tracheal intubation, with a grade III ASA risk score. The anesthetic approach was calculated in order to best avoid inducing increases in intracranial pressure, given that the mastoid cavity, prior to shaving, represents a closed cavity. Thus, one of the agents that remained unused was nitrous oxide.

Post-operative treatment added Ceftriaxone 1 g b.i.d. and routine analgesics to the previously mentioned medication. The selection of a third-generation, broad-spectrum cephalosporin was based upon its ability to permeate the blood-brain barrier, thus minimizing the risk of intracranial complications of exacerbated chronic otitis media and mastoiditis such as cerebral abscesses, as well as sepsis.

Over the course of the first post-operative week, the patient remained for monitoring and was treated with 200 mg Hidrocortisone IV t.i.d. for the first three days, along with previously mentioned treatments.

Upon discharge, the patient's symptoms of the initial presentation had completely subsided, save for the facial nerve paralysis, which still was rated as grade V on the

House-Brackmann scale. The IV corticotherapy was substituted by an oral, month-long step-down therapy with Prednisone.

Follow-ups at seven and twenty-one days showed only minor improvement, as expected. However, the patient's facial nerve paralysis three months after surgery demonstrated significant improvement in motor and sensory function, with a House-Brackmann rating of III.

The patient's intra- and post-operative evolution was favorable, and confidence in a favorable prognosis remained high.

Discussions

Pre-operative imaging studies are valuable tools used to decide which procedure may lead to the most favorable outcome for the patient. When performing a cranio-facial computer tomography to detect cholesteatoma, it is sensible to select a high-resolution CT with sections smaller than 150-micron sections in order to best differentiate the type of tissue formed as a result of chronic inflammation.

Intraoperatively, the patient's facial nerve was found to be unexposed, and the attico-antral and mastoid spaces showed minimal signs of erosion, indicating that the patient's facial nerve paralysis was most likely secondary to an acute (infectious) exacerbation of the patient's CSOM and chronic mastoiditis. Under the correct circumstances (i.e., a new bacterial infection of the middle ear), CSOM may be exacerbated, leading to the postaural inflammation and increased otorrhea described by the patient, as well as otogenic facial nerve paralysis. Given the patient's history of long-term use of endaural hearing aid and reduced medical attention before the exacerbation, it is arguably a likely course of the disease.

Surgical treatment delay of nearly a month might have led to a protracted convalescence, as it is usual for facial nerve paralysis secondary to. Follow-ups at one and three months, however, demonstrated improved motor and sensory function, with a House-Brackmann score of IV/V at the one-month check-up and an HB score of III after three months.

Generally, patients with non-cholesteatomatous CSOM and associated facial nerve paralysis fare better than those with cholesteatomatous CSOM and associated facial nerve paralysis. Similarly, cases in which surgical treatment is delayed by more than four weeks have a poorer prognosis of facial nerve recovery than those which receive early treatment [7].

Post-operative treatment of otogenic facial nerve paralysis includes a minimum of four weeks of high-dose corticotherapy, such as 25 mg prednisolone initially, followed by a step-down treatment plan. Further patient check-ups at one, three, and six month intervals are required to assess the effectiveness of both the surgical procedure and the post-operative recovery. The patient should be clinically examined during each follow-up to evaluate the remaining degree of facial paralysis and hypoacusis, and proper ear toilet should be observed [10].

At the present time, the six-month check-up has yet to occur, but confidence in a favorable outcome, based upon previous examinations, remains high.

Conclusions

Advances in Otorhinolaryngology combined with blanket-like public health-care systems can allow for complete control of CSOM, before intra- or extra-cranial complications appear. Given the socio-economic burden of CSOM for both the patient and the health-care infrastructure, primary disease prevention and patient education about risks must be at the forefront of the clinical approach.

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Conflicts of Interest: The authors have no conflict of interest.

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