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Delayed facial nerve paralysis post middle ear surgery: herpes simplex virus activation

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Abstract. *Delayed facial nerve paralysis post middle ear surgery: herpes simplex virus activation. Problem*: Facial nerve paralysis following middle ear surgery is a nightmare for the otology surgeon. Usually this is caused by surgical trauma or local anaesthetic use. It is uncommon to see onset of facial nerve palsy more than 72 hours following the surgery.
Methodology: We report a case of facial nerve paralysis appearing 11 days following a successful canal wall-down mastoidectomy.
Results: Viral screening for Herpes Virus type 1 confirmed the viral aetiology of the delayed facial paralysis.
Conclusion: When an ipsilateral facial nerve palsy appears more than 72 hours after an uneventful middle ear procedure, without symptoms of any infection, suspect a viral reactivation.

Introduction

Facial nerve paralysis is a well-known, albeit rare, complication of ear surgery.[1-3] In most cases, it is a secondary effect of local anaesthetics or trauma during surgery. The trauma is commonly related to difficulties during surgery, or anatomical anomalies. In the vast majority of cases, symptoms of the nerve injury occur immediately after surgery. Rarely, an ipsilateral delayed facial nerve paralysis (DFP) may also occur several days after a successful and uneventful ear surgery.[4]

Delayed facial nerve paralysis has been described after all types of otologic procedures.[1,2,4-6] The rate of incidence of DFP after an otherwise successful and uneventful surgery varies according to the type of surgery. DFP following middle ear surgery is very rare, and after tympanomastoid surgery it has been reported in few cases.[4,6] The rate of incidence of DFP after a successful and uneventful tympanomastoid surgery is summarised in Table 1.

Various pathophysiological mechanisms may explain DFP following middle ear surgery. Neural oedema is widely thought to be the primary cause. But neural oedema might, in fact, just be the common denominator of other mechanisms, including ischemia, vasospasm, neural devascularisation, immune reaction, and viral reactivation.[7] Various authors now suggest that viral reactivation is the true cause of DFP.[6,7] An increase in Varicella-Zoster Virus (VZV) and Herpes Simplex Virus (HSV) immunoglobulin M (IgM) and immunoglobulin G (IgG) titres is present in association with DFP.[2,8] There is currently little doubt that HSV and VZV are often present in a latent state in the human geniculate ganglion.[9,10] Viral reactivation is always thought possible, when the patient is subjected to surgical stress.[11]

Here we report a case of DFP presenting 11 days after a successful and uneventful canal wall-down tympanomastoidectomy. We discuss the possible mechanisms supporting the hypothesis of viral reactivation.

Case report

A 56-year-old man was referred to us with complaints of hearing loss and occasional right otorrhoea. His past medical history was unremarkable. Routine laboratory tests were within normal limits. At the time of presentation, there was no history of previous Bell's palsy. Clinical examination of the right ear revealed a sub-total perforation with large bone erosion in the epitympanum, and an associated cholesteatoma. There was some mucopurulent discharge in the external auditory canal. Facial

nerve functions were normal. A pure tone audiogram (PTA) showed a mixed hearing loss pattern in his right ear, with bone conduction thresholds at 60 dB, and air conduction thresholds at 90 dB. The hearing in his left ear was within normal limits. A Computed Tomography (CT) scan of the right petrous bone confirmed the diagnosis of chronic otitis media with cholesteatoma.

The patient underwent a right canal wall-down tympanomastoidectomy. Intra-operatively the facial canal was found to be intact. Cholesteatoma along with the hypertrophic mucosa of the middle ear was removed completely. Malleus and incus were eroded by the cholesteatoma, leaving behind only their remnants, which were therefore removed. The tympanic membrane was reconstructed with temporalis fascia resting over the stapes head. Facial nerve function immediately after surgery was documented as normal. The post-operative recovery was uneventful.

Eleven days after the surgery, the patient returned to us with the onset of right peripheral facial nerve palsy. On examination, the palsy was found to be of severity grade IV, according to House-Brackmann functional scale. There were no vesicles visible to suggest any herpetic infection. The patient was hospitalized and started on high dose oral steroids (1 mg/kg of prednisolone). Despite all these measures, the facial nerve palsy gradually worsened to become a complete paralysis of HB Grade VI, within 48 hours of presentation.

Viral screening for VZV antibodies was performed using Enzyme Linked Immuno Sorbent Assay (ELISA). Immunofluorescence was used for detection of HSV-1 and HSV-2 antibodies. ELISA examination was negative for VZV infection. However, immunofluorescensce showed levels of more than 1.1 for IgM and IgG against HSV-1 (IgM was 3.2, and IgG was 1.9) confirming the viral aetiology of the DFP. Based on these findings, the patient was started on oral acyclovir treatment. The dosage was 2400 mg (800 mg three times a day) for 10 days, in addition to oral steroids. The patient was advised to use eye protection, and given facial massage and exercises as part of the management.

At the end of medical treatment, the facial nerve function was grade II-III HB, and it recovered completely (HB Grade I) after three months.

Discussion

Facial nerve paralysis after middle ear surgery can be of two types: immediate and delayed. Immediate facial palsy is usually related to the local anaesthetic used during surgery, or it may be due to intra-operative trauma to the facial nerve.[1] Local anaesthetic may produce facial paralysis from the pre-operative infiltration. The instillation of a topical anaesthetic into the middle ear in the presence of facial nerve dehiscence may also produce such paralysis.[1] Traumatic facial nerve injury can occur from a curette, a drill, a chisel or any other surgical instrument during surgery. In such cases, the facial nerve may be lacerated, or crushed, resulting in intra-neural interruption, haematoma, or oedema.[11] Generally, if anaesthetics caused the paralysis, it resolves within a few hours. If the cause is surgical trauma to the nerve, the recovery takes a long time, and sometimes does not occur at all.

Delayed facial nerve paralysis after middle ear surgery is uncommon. It usually develops more than 72 hours after the surgery.[6] The nerve generally recovers its function rapidly and completely within a few months. In rare cases, recovery is incomplete, with synkinesis and spasm.[12,13]

Numerous mechanisms have been put forward to explain the genesis of DFPs after otologic surgery, including oedema, bacterial infection, and viral reactivation. Neural oedema is the primary pathology underlying such delayed facial nerve palsies.[6] A review of several published studies confirms neural oedema to be the most widely accepted pathophysiological mechanism of DFPs.[7,8,14] Why such oedema develops after a successful and uneventful middle ear surgery in some patients, is not yet clear. It is understandable that neural oedema can develop following surgical trauma. However, it seems unlikely that an oedema from trauma can unleash paralysis more than 72 hours after the injury. Bacterial infections are not commonly associated with facial nerve infection, but they can induce vascular thrombosis or oedema inside the nerve.[6] In cases of bacterial infection, a facial nerve palsy can occur a few days after the surgery. However, it is always associated with otorrhoea.[15] In our patient, the facial nerve palsy occurred 11 days after the surgery. There was no otorrhoea. This made us search for other possible reasons for the nerve paralysis.

Viral reactivation as a cause of DFP following uneventful middle ear surgery has been widely reported in the English litera-

ture.[1,2,4-6,11,13,16] Varicella zoster virus and HSV (type I and type II) are often implicated in reactivation after surgical intervention. Common examples include herpetic keratitis after corneal transplants, herpes pneumonitis after cardiac surgery, and herpes zoster after spinal procedures. Typically, there is a delay of few days before the manifestation of symptoms.[6] In 1992, Furuta documented the presence of VZV and HSV in the geniculate ganglion in a large percentage of autopsy studies.[9,10] In 1995, Sugita induced facial palsy by inoculating HSV-1 into the auricle of mice.[17]

The pathophysiology by which latent virus reactivation causes DFP is uncertain. McCormick[18] hypothesized that HSV takes up residence within the peripheral nerve axon. Facial palsy could result when HSV ruptures from the nerve axon, infects the Schwann sheath and progresses centripetally. This is possible even after surgical stress without direct damage on the facial nerve. In fact, Vrabec[6] demonstrated that a mechanical disturbance of the chorda tympani, and/or surgery close to the facial nerve, can result in a reactivation of HSV in the geniculate ganglia.

Pazin *et al*.[19] described a reactivation of HSV in 28 (50%) of 56 patients after decompression of the trigeminal nerve root. In this procedure, the nerve being compressed by a blood vessel is microsurgically decompressed. Micro injuries may occur, leading to the reactivation, but no macroscopic injury to the nerve occurs. A similar mechanism may be responsible in reactivating the HSV in facial nerve, resulting in DFP. In the present case, the high titre of IgM[2,3] and IgG[1,9] signalled the viral reactivation, leading us to an appropriate management of the patient.

The benefits of using steroids and antivirals are well proven. They not only reduce the duration of symptoms, but also prevent neural sequelae such as postherpetic neuralgia.[6] Aggressive surgical management such as exploration or nerve decompression is contraindicated in the management of this uncommon condition.[6] Prophylactic treatment with antivirals (during the days preceding the otologic surgery and after the procedure for a week) to prevent viral reactivation is possible. However, it must be based on a history of herpetic lesions.[13]

Conclusion

The risk of DFP after an uneventful middle ear surgery is variable but tends to be very low. However, a history of herpetic lesions greatly increases the possibility of DFP, and these patients should be treated with an antiviral prophylaxis before and after surgery. When an ipsilateral facial nerve palsy appears more than 72 hours after an uneventful middle ear procedure, without symptoms of infection, a viral reactivation must be suspected. Conservative management with antivirals and steroids should be employed without awaiting the results of serological tests. This rapid management usually results in complete recovery of the nerve function.

Table 1

Reported incidence of DFP after uneventful and successful tympanomastoid surgery

Author	Year	Patients with DFP	% of Incidence of DFP
Vrabec[6]	1999	7	**0,8% - 1,4%**
Gyo *et al*[16]	1999	1	
Safdar *et al.*[11]	2006	2	
Kithara *et al*[4]	2006	3	
This Study	***2007***	***1***	

DFP: Delayed Facial Paralysis.

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