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Case Report

Late Onset of Facial Nerve Palsy After Tympanomastoidectomy: HSV-1 Activation?

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ABSTRACT

We present a case of a right peripheral facial palsy occurring 7 days after an operation. A 42 year old female patient had an uneventful right tympanomastoidectomy in our clinic. She developed right Hause-Brackmann Grade II peripheral facial palsy postoperatively at the 7th day. A viral screen was performed using an Enzyme Immunoassay. Herpes Simplex Virus-1 specific antibody titer was determined on the 2nd day of facial palsy, confirming the viral etiology. She was commenced on steroid therapy. Her facial nerve functions recovered completely after one week.

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Introduction

Facial palsy is an uncommon complication of middle ear surgery. Its onset is usually immediate due to a trauma during surgery but there are a few cases in the literature of delayed facial palsy (DFP) following tympano-mastoid surgery [1-3]. In these cases, the etiology is not clear. However, in some papers, surgical stress is suspected to reactivate latent Herpes Simplex Virus Type-1 (HSV-1) in the geniculate ganglion [2].

Here we present a case with DFP on the 7th day after surgery, in which facial function recovered completely over a period of one week by medical treatment alone with methylprednisolone. We

would like to emphasize that facial nerve dysfunction after ear surgery is not only due to direct surgical trauma to the nerve but also due to secondary effects of the operation (surgical stress) that can cause viral reactivation resulting in DFP.

Case Presentation

A 42 year old female patient presented with a 2day history of weakness on the right side of her face, inability to close her right eye and asymmetrical appearance of the mouth while smiling, which was assessed as Hause-Brackmann

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Grade II Peripheral Facial palsy (Figure 1a). She had a right tympanomastoidectomy in our clinic nine days previously, and her symptoms developed on the 7th day after surgery. In the operation, the facial canal and chorda tympani were intact. Epitympanic recess and protubarium were invaded

with polypoid mucosa. There was no postoperative visible problem with facial nerve function immediately after surgery and during seven days postoperatively. The patient had an uneventful recovery following the procedure.

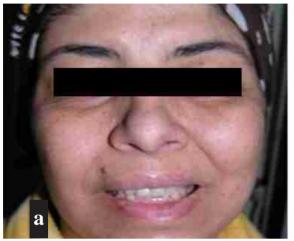




Figure 1. (a) She presented with a 2-days history of Hause-Brackmann Grade II Peripheral facial nerve palsy on the 9th day postoperatively. (b) The facial nerve functions recovered completely after one week, with high dose intravenous metilprednisolon for two days and 1mg/kg/day metilprednisolon by waning per 3 days. (The informed consent was obtained and pictures were used by the permission of patient)

Her past medical history was not significant for any systemic chronic disease or recent upper airway infection. But she had great emotional stress before and after the surgery. Medications at the time of admission were ciprofloxacin 750 mg/dose BID and naproxen sodium 500 mg/dose BID per oral.

She had a good general condition. On examination her temperature was 36.0°C, heart rate was 80 beats per minute, and blood pressure was 110/80 mm Hg. Her face had a normal tone and symmetry at rest. There was complete closure of the right eye with minimum effort and a slight asymmetry of the mouth. Her facial nerve function was assessed to be House Brackmann grade II. There were no visible vesicles to suggest a herpetic infection. The patient did not have otalgia, vertigo or dizziness. Therefore, the mastoid dressing was not removed.

Complete blood count and serum biochemistry, including creatinine, urea, glucose and electrolytes, were normal. A viral screen was performed using an Enzyme Immunoassay. HSV-1 specifie antibody titer was determined during the acute phase on the

2nd day of facial palsy, confirming the viral etiology.

The patient was started on a high dose (250 mg/day) of intravenous methylprednisolone sodium succinate for two days. She was then prescribed 1 mg/kg methylprednisolone (60 mg) for 3 days, followed by a taper to 10 mg per three days, for a total of 20 days. Also, acyclovir therapy was recommended but she rejected this after being informed about the side effects of the drug. Her facial nerve functions recovered completely after one week (Figure 1b).

Discussion

Delayed facial palsy (DFP) is defined as dysfunction occurring more than 72 hours postoperatively. JT Wrabec reported 7 cases of DFP after tympanomastoid surgery, which represents 1.4% of all cases (n,486) and 1.9% in revision cases (n,155) [1]. Viral reactivation may be an important etiological factor in the development of delayed onset facial nerve palsy.

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Any factors causing neural inflammation, e.g. direct thermal or mechanical injury to the facial nerve, local effects of blood breakdown products or any mediators causing vasospasm, can be encountered in the etiology of DFP [4].

Shea and Ge reported DFP in 0.22- 0.51% of patients after stapedectomy. They reported 11 cases of DFP. Six of them were evaluated serologically. Anti-HSV antibody titers were elevated in 5 of 6 patients. They focused on viral reactivation as heading the list for the most probable cause. Serologic investigations are suggested for diagnosis of the activation of latent herpes virus [5,6]. Murakami et al suggest that herpes simplex HSV-1 is active in idiopathic facial paralysis [7]. They suggested steroids and antiviral medication as the appropriate management strategy for the acute phase of the disease and proposethat the majority of patients will completely resolve their paralysis with no residual deficits. A new study in rats by Turner MT et al. provides evidence supporting the use of prophylactic antivirals for otologic surgeries associated with high rates of DFP [8].

Since we did not have an opportunity to test for viral DNA, we evaluated the patient serologically. De Diego et al reported a lesser degree of neural degeneration in Bell's palsy patients treated with low-dose prednisone (1 mg/kg body weight) within the first 96 hours, using acyclovir in controls [9]. Furthermore, an added beneficial effect of starting high dose steroid therapy early after palsy onset was suggested [10]. The dosage of prednisolone employed in different studies ranged between 216-760 mg/day. In this study, facial function recovered

completely over a period of one week by medical treatment, consistent with the literature.

Regarding the reports of DFP in stapedectomy operations, the cause of viral reactivation is attributed to the mechanical irritation of the facial or chorda nerve [5,11]. Since tympanomastoid surgery is a relatively more aggressive operation than stapedectomy, the surgical stress during tympanomastoid ectomy may also play a role in triggering viral reactivation.

"Is DFP after surgery coincidental?" is another question. If the association was coincidental, then the incidence of DFP would be expected to be the same as Bell's palsy, which was reported to be approximately 1:5000 persons per year [12]. As mentioned earlier, DFP is reported in 1.4% of cases after tympanomastoid surgery, which is well above the rate of Bell's palsy [1].

Conslusion

Any person undergoing ear surgery is a potential candidate for facial nerve dysfunction. This unfortunate event requires the physicianto decide whether a second operation is needed or to give medical treatment. DFP, as the name implies, is noticed more than 72 hours after surgery since facial nerve function is normal during this postoperative period. Thus it indicates that the facial nerve is anatomically intact in DFP, but that secondary events cause nerve palsy. Consequently, medical treatment (as with Bell's palsy) will be sufficientfor DFP.

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