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Main Article

Delayed facial nerve palsy following tympano-mastoid surgery: incidence, aetiology and prognosis

A Safdar, S Gendy, A Hilal, P Walshe, H Burns

Abstract

Objective: To establish the frequency of occurrence of delayed facial nerve paralysis following tympano-mastoid surgery in our department and to determine the aetiological factors and long term prognosis.

Setting: Tertiary care academic centre.

Materials and methods: A retrospective review of all patients who had undergone tympano-mastoid surgery in our department over the previous five years was carried out. A total of 219 patients were included in the study. Only two patients were identified as having delayed onset facial nerve palsy over this period of time. The patients' medical records were reviewed and the patients clinically assessed.

Results: The frequency of delayed onset facial nerve palsy following tympano-mastoid surgery in our series was 0.91 per cent. Facial weakness set in on day eight and day 14 in the two patients. Serological investigations in both patients revealed raised titres of immunoglobulin (Ig) M and IgG to varicella-zoster virus, confirming the presence of varicella-zoster infection. In our experience, the combined use of prednisone and acyclovir was an effective form of treatment for both patients, whose facial nerve function fully recovered within six months of onset.

Conclusion: The incidence of delayed facial nerve palsy following tympano-mastoid surgery is low. It can occur up to two weeks after the surgery. Our two cases confirm viral reactivation to be an important aetiological factor in the development of delayed onset facial nerve palsy. The overall prognosis for delayed facial nerve palsy following tympano-mastoid surgery appears to be good.

Key words: Otologic Surgical Procedures; Tympanoplasty; Facial Paralysis; Herpes Zoster; Complications

Introduction

Delayed onset facial nerve palsy (DFP) has been described after all forms of otologic and neuro-otologic procedures.[1–6] The pathogenesis and prognosis are of equal concern to the patient and surgeon. The relationship between surgery and the onset of facial nerve dysfunction implicates the procedure as an underlying cause, thus bringing the surgeon's technique into question. The differential diagnosis of post-operative facial nerve palsy cannot be made solely on the basis of iatrogenic injury; the patient's previous medical history and physical examination may influence the diagnosis, management and prognosis of this complication.

The incidence of DFP varies with the type of surgery. After stapedectomy, it has been reported to occur in 0.2 to 0.99 per cent of patients.[2–4] The incidence after surgery for acoustic neuroma ranges from 2.2 to 30 per cent[5–8] depending upon the criteria used to establish the diagnosis, while after cochlear implant surgery the incidence can be up to 4 per cent.[9,10]

Delayed onset facial nerve palsy after tympano-mastoid surgery has been infrequently reported. Vrabec *et al*. described seven patients within a series of 486 (1.4 per cent) who developed DFP,[11] while Bonkowsky *et al*. described seven cases (0.38 per cent) of DFP in a series of 1800 patients.[12]

A number of factors have been postulated in the pathogenesis of DFP, including neural oedema secondary to surgical trauma, local anaesthetic drugs and viral reactivation. Therefore, identifying the cause of this condition has important implications for both management and final outcome.

Material and methods

The medical records of all patients who underwent tympano-mastoid surgery in our department over a period of five years were reviewed (2000–2004). Patients who had undergone stapedectomy, tympanoplasty or myringoplasty were excluded. Patients with a facial weakness due to a suspected iatrogenic cause were also excluded. The definition used to

From the Department of Otolaryngology, Royal Victoria Eye and Ear Hospital, Dublin, Republic of Ireland.
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identify patients with DFP was a deterioration in facial nerve function noticed 72 hours after surgery, with a normal facial nerve function documented in the clinical records immediately following surgery. The patients' laboratory results were reviewed and patients were subsequently assessed clinically to document their facial nerve function.

Results

A total of 219 patients were included in the study. Details for all patients reviewed are shown in Table I. Only two patients were identified as having delayed onset facial nerve paralysis according to the established criteria; the rate of occurrence was therefore 0.91 per cent. Brief clinical descriptions of the two patients are given below.

Case one

A 16-year-old male patient underwent a right modified radical mastoidectomy for attico-antral cholesteatoma. The fallopian canal was found to be intact and facial nerve function was monitored throughout the surgery. Facial nerve function was documented to be normal immediately following surgery, and the patient made an uneventful recovery. The patient's mastoid cavity was assessed seven days post-operatively and was found to be satisfactory.

On the eighth post-operative day, the patient became increasingly dizzy, with third degree nystagmus, and a House–Brackman grade IV, right-sided facial weakness developed. This gradually became complete over the next 24 hours. There were no vesicles visible to suggest a herpetic infection. The patient was commenced on high dose oral steroids and intravenous ciprofloxacin. As no improvement resulted, the patient underwent re-exploration of the operated ear on day 13. The facial nerve was found to be intact. Viral screening was performed using enzyme immunoassay (EIA).

Enzyme immunoassay is a general term for an expanding technical arsenal of quantitative analyses for both antigen and antibodies. These tests use colour-changed products of enzyme–substrate interaction or inhibition in order to measure the antigen–antibody reaction. Examples of EIA procedures include Enzyme multiplied immunoassay (EMIT), enzyme-linked immunosorbent assay, MAC-ELISA defined as IgM antibody capture ELISA (MAC) and Microparticle enzyme immunoassay (MEIA).

TABLE I

CASES REVIEWED

Total mastoid operations in 5 years (*n*)	219
Average age of patients (years)	34.8
Males (*n*)	146
Females (*n*)	73
Modified radical mastoidectomies (*n*)	185
Radical mastoidectomies (*n*)	2
Tympano-mastoidectomies (intact canal wall) (*n*)	5
Cortical mastoidectomies (*n*)	4
Atticotomies (*n*)	4
Revision mastoidectomies (*n*)	19

The reference range for varicella-zoster immunoglobulin (Ig) G and IgM antibodies is: <0.8 index = negative; 0.8–1.0 index = borderline; and >1.0 index = positive.

The results of the patient's viral screen became available after exploration and showed levels of more than 1.0 of IgM and IgG against varicella-zoster virus, confirming a viral aetiology. The patient was commenced on oral acyclovir and discharged home two days later. He made a complete recovery of facial function within six months.

Case two

A 54-year-old man underwent a revision left modified radical mastoidectomy. Operative findings included an open middle ear, absent malleus and incus, low facial ridge, and previous exposure of the facial nerve above the oval window and in the pyramidal segment. Facial function was documented as normal immediately following surgery. The patient made an uneventful recovery following the procedure and the mastoid dressing was removed a week later.

Fourteen days after the surgery, the patient presented with a House–Brackman grade III left facial palsy. The mastoid cavity was found to be satisfactory and the patient was commenced on high dose oral steroids and intravenous broad spectrum antibiotics. The white cell count was normal. No vesicular eruption was seen. A viral screen was performed using EIA. This showed levels of more than 1.0 of IgG and IgM to varicella-zoster virus, confirming a viral aetiology. The patient was then commenced on acyclovir in addition to oral prednisone. His facial function recovered completely over a period of two months.

Discussion

The rate of DFP in our series was 0.91 per cent, which is slightly less than that reported by Vrabec *et al.*[11] We assume this to be due to the strict inclusion criteria that we used. However, we consider that the significantly higher incidence of DFP following acoustic neuroma surgery may be secondary to the increased manipulation of neural tissue at the time of surgery.

Immediate onset facial nerve palsy after tympanomastoid surgery requires surgical exploration. Deka *et al.* reported 10 patients with immediate post-operative facial nerve paralysis after tympanomastoid surgery, and surgical exploration demonstrated an insult to the facial nerve in all cases, ranging from oedema to transection.[13]

In traumatic injury, the nerve may be lacerated, transected or crushed, with resulting intraneural haematoma or oedema. In infectious cases, no disruption of the nerve fibres is found, but neural oedema or vascular thrombosis may still occur. Prognosis and management vary depending upon the mechanism of injury.

Recovery of facial nerve function is better in patients with DFP compared with those with immediate onset paralysis, and incomplete paralysis has a higher rate of normal recovery.[14]

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The potential causes of DFP after any otologic procedure include mechanical compression of the nerve,[13] bacterial infection and viral reactivation. Reactivation of the herpes virus after neurosurgical procedures has been well documented, especially in operations on the trigeminal nerve root.[15] Similarly, manipulation of the cervical, thoracic or lumbosacral nerve roots has been reported to cause herpetic viral reactivation and to produce lesions in the dermatome of the nerve manipulated during surgery.[16]

Viral reactivation as a cause of DFP following tympano-mastoid surgery has been reported infrequently in the literature. Gyo and Honda reported one case of delayed onset facial nerve palsy secondary to reactivation of varicella-zoster virus after middle-ear surgery.[17] Vrabec[11] and Bonkowsky *et al.*[12] reported five cases each of DFP due to reactivation of varicella-zoster virus following tympano-mastoid surgery.

In order for viral reactivation to occur, there must be latent virus within a sensory ganglion. Herpes simplex and varicella-zoster viruses have been isolated in the geniculate ganglion in a high percentage of autopsy cases with no antemortem manifestations of viral infection.[18] A preceding stress, such as an upper respiratory tract infection, fever or exposure to cold, is thought to act as a trigger. It has been reported that varicella-zoster virus specific T-cell response is correlated with the risk of herpes zoster infection and reactivation.[19]

The diagnosis of Ramsay–Hunt syndrome occurring after tympano-mastoid surgery is not very difficult when the pathognomonic auricular vesicles accompany the facial palsy. When facial nerve palsy develops without herpetic eruption, it is known as zoster sine herpete, as seen in both our patients. Serological tests can be helpful in demonstrating the presence of IgM antibody to varicella-zoster virus, indicating a recent infection or a significant change (greater than a twofold rise) in IgG.[20]

Gianoli *et al.* reported a significant post-operative rise of IgM titres to varicella-zoster virus, compared with pre-operative titre levels, in seven patients (out of 20) who developed DFP after excision of acoustic neuroma.[21]

The recent introduction of the polymerase chain reaction (PCR) technique allows early and definitive diagnosis of varicella-zoster infection in the exudate from the geniculate zone of the ear. Using PCR, Furuta *et al.* demonstrated the presence of varicella-zoster virus deoxyribonucleic acid in saliva from patients both with and without herpetic eruptions, suggesting that reactivated varicella-zoster virus in the geniculate ganglia may migrate into the oropharyngeal epithelium without producing zoster at the site.[20]

The use of steroids and antivirals in the treatment of Ramsay–Hunt syndrome is well proven. The largest published series showed a statistically significant improvement in patients treated with prednisone and acyclovir within three days of onset.[22] In our patients, the combined use of acyclovir and prednisone proved to be an effective treatment, evidenced by the complete recovery of facial nerve function.

It is thought that immediate facial nerve palsy following tympano-mastoid surgery is associated with a poorer prognosis and that incomplete DFP is more likely to resolve completely.[8,14] In our experience, as seen in our first patient, there was complete resolution of facial nerve function following complete paralysis. In view of its uncommon occurrence after tympano-mastoid surgery, it is difficult to establish firm prognostic criteria for DFP. Based on the experience with DFP following acoustic neuroma surgery, it may be reasonable to suggest that DFP following tympano-mastoid surgery may have a better prognosis.

Conclusion

In our experience, the risk of delayed facial nerve palsy after tympano-mastoid surgery is low and a reactivation of varicella-zoster infection is an important aetiological factor. Serological investigations, if performed early, can be valuable in proving a viral aetiology. Patients with a history of viral reactivation may be at risk of this complication after otologic surgery, and prophylaxis with an antiviral agent should therefore be considered.

- **This study aims to establish the frequency of occurrence of delayed facial nerve paralysis after tympano-mastoid surgery**
- **A retrospective review of 219 patients undergoing tympano-mastoid surgery over a five-year period was undertaken. Two patients were identified as having had delayed onset facial nerve palsy over this period of time**
- **Serological investigations in both patients revealed raised titres of IgM and IgG to varicella-zoster virus**
- **The combined use of prednisone and acyclovir was an effective form of treatment for both patients, who made a full recovery of their facial nerve function within six months of onset**

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Address for correspondence:
Mr Adnan Safdar,
27 Forster Walk,
Lucan,
Co Dublin, Republic of Ireland.

E-mail: adnan_safdar@hotmail.com

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