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Delayed facial nerve palsy following uncomplicated myringoplasty

Facial palsy occurring immediately after ear surgery is usually the result of direct injury to the facial nerve and is considered a serious complication. Facial weakness after surgery can be related to local anaesthetics. It occurs during or immediately after the operation and it resolves spontaneously within a few hours. Facial palsy may also develop several days after uneventful middle ear surgery. It is described as the facial weakness occurring at least 72 hours after the surgery. In most of reports, delayed facial palsy (DFP) occurs after acoustic neuroma surgery with the incidence ranging from 4.8 to 30% (2, 5, 7). Delayed facial palsy can also develop after stapes surgery. Althaus and House (1) described this phenomenon in five (0.2%) of 2,307 cases after stapedectomy. Facial weakness began 5 to 13 days after uneventful surgery and resolved completely within 8 weeks. They suggested that stretching the chorda tympani causes swelling that migrates to the facial nerve which in turn leads to facial weakness. The treatment they advocated is orally administered steroids and facial nerve decompression in patients not responding to conservative treatment. We report three cases of delayed facial palsy after uncomplicated middle ear surgery.

CASE REPORTS

Case 1. A 54-year-old male (W. T.) reported with a long history of chronic otitis media with recurrent discharge of his left ear. He had profound sensorineural hearing loss and a dry perforation in posterior quadrants of the drum in that ear. Endaural incision was used to perform myringoplasty with tragal perichondrium. The ossicular chain was intact. The healing was normal. The patient developed facial nerve palsy grade IV in House and Brackmann scale (H-B) on the seventh postoperative day. He received intravenous steroids (dexamathasone 8 mg per day) for 14 days. The symptoms gradually subsided and after 3 weeks the patient recovered completely.

Case 2. A 34-year-old female (M. R.) with history of bilateral chronic otitis media was referred to us with conductive hearing loss. She had a myringoplasty with a perichondrium graft performed 3 years before on her right ear due to dry subtotal perforation of the drum. No complications were noted after the first operation and the closure of the perforation was achieved. She had a dry posterior perforation in the second ear and 25 dB air-bone gap with normal bone conduction. The ear was operated under local anaesthesia and the perforation was closed with perichondrium. Ossicular chain was intact. Early postoperative period was uneventful. In the sixth postoperative day the patient reported otalgia, mild swelling of preauricular region and facial weakness. The facial nerve palsy progressed to grade V H-B within 3 days. She was treated in other hospital with antibiotics and group B vitamins without improvement. The pain and swelling subsided after 2 weeks, but facial weakness persisted. She was referred to us with facial nerve palsy grade VI. Because there was no improvement after 4 weeks of conservative treatment we decided to perform a decompression of the facial nerve. The middle ear was clear from any discharge. The nerve was decompressed in the mastoid and tympanic segment and the epineural sheath was incised. Facial nerve swelling was noticed. The patient received intravenous steroids for one week. Improvement

in the facial nerve function was noted 2 weeks later but grade II facial weakness persisted after 11-month follow-up.

Case 3. A 53-year-old female (J. Z.) with a history of recurrent discharge from the right ear, unilateral hearing loss and with epitympanic perforation in her right drum was operated in general anaesthesia. A granulation tissue and a small pearl of attic Cholesteatoma was removed and the tympanic membrane defect was closed with conchal perichondrium. Postoperative period was uneventful. She reported with a facial weakness grade II H-B on the 8th postoperative day. The patient received intravenous dexamethasone 8 mg per day for 9 days and group B vitamins. The facial palsy progressed to grade III H-B to the 10th postoperative day. Improvement in the facial movements was evident three days later and the facial nerve function recovered completely 2 months after the operation.

DISCUSSION

Most authors suggest viral aetiology of delayed facial palsy. Bonkowsky (3) presented seven patients with DFP after middle ear surgery. In four from five patients tested, HSV-1 particles were detected using polimerase chain reaction. They also noticed immunological changes typical of reactivation of herpes simplex virus. The authors suggested that reactivated viruses lead to an immune reaction in the facial nerve and cause the facial weakness.

The use of laser seems to enlarge the risk of delayed facial palsy after stapes surgery. Ng (10) reported two cases with DFP after KTP laser stapedotomy. The facial nerve function in this patient returned to normal within 3 months of conservative treatment. Similarly Mills et al. (9) reported six patients with DFP after the use of KTP laser. They conducted experiments showing that heating of the nerve by KTP laser is possible. This thermal effect may directly cause some damage to the facial nerve or can be a factor reactivating latent virus. Other types of lasers were also reported to involve single cases of DFP. Lesinski (8) reported one case after CO2 laser stapedectomy and Gerhini (4) another case after Argon laser stapes surgery.

Gyo and Honda (6) reported a case of DFP after open technique tympanplasty. The patient had previous ear surgery without complications. Facial weakness occurred on 14th postoperative day. This was associated with pain in the ear. The pain ceased two days later. Despite treatment with low molecule dextran and steroids facial palsy progressed and that ear was re-explored. Extensive bulging was fund during that operation and decompression of the nerve performed. Facial palsy improved from grade 6 to 3 in H-B scale. The authors performed PCR analysis of endoneurial fluid and found VZV specific DNA fragments. They concluded that reactivation of varicella-zoster virus was the cause of facial palsy. The second case presented by us had similar presentation and outcome. We have not performed any serological tests, but the patient also reported pain in the ear at the beginning of facial nerve symptoms. Re-exploration revealed no signs of bacterial infection in the middle ear and swollen facial nerve after incision of the epineural sheath. Those sings might suggest similar viral agent. The outcome of our patient and the one presented by Gyo and Honda (6) is similar. Both have residual facial weakness. This may be influenced by the same type of the virus or by management. Decompression of the facial nerve enables recovering but may also produce some damage to the nerve.

Vrabec (12) analysed a group of seven patients with DFP after tympanomastoid surgery. In two of them wound infection could be the cause of facial weakness. In the remaining five patients viral etiology was suggested. In four patients otalgia was a prominent symptom a few days before the facial palsy occurred.

Shea and Ge (11) reported 11 cases of DFP after stapedectomy. One case from his series developed DFP after first operation. The patient received antiviral agents before the second ear operation and facial palsy did not occur. The author suggested that prevention with antiviral drugs is possible. Our patient (M. R.) had operation on both sides and developed DFP only during second ear operation. No antiviral agents were used before the operation.

Some controversy relates to the treatment of DFP. Most authors suggest oral administration of steroids. Antiviral drugs have a potential role in reducing the duration of facial weakness. Aggres-

sive treatment with facial nerve decompression should be avoided because the outcome of DFP is favourable with conservative treatment. The operation performed in the presented patient may have been a factor preventing full recovery. However, some authors justify operation in cases not responding to treatment (1, 6).

Prophylactic treatment with steroid and antiviral drugs could be considered in cases with history of virus reactivation (11, 12). It is not indicated in other patients because of very low incidence of DFP after middle ear surgery.

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SUMMARY

Delayed facial nerve palsy after uncomplicated middle ear surgery is a rare event. We present three cases with facial palsy developed 6 and 7 days after uneventful myringoplasty. The facial weakness progressed to grade VI and resolved after treatment with steroids in two patients. The third patient was subjected to facial nerve decompression after four weeks of medical treatment. The operation revealed swelling of the nerve. Facial nerve function improved to grade II. We discussed possible etiologic factors and management of delayed facial nerve palsy after ear surgery.

Odległe porażenie nerwu twarzowego po niepowikłanej myringoplastyce

Odległe porażenie nerwu twarzowego po operacjach ucha środkowego występuje bardzo rzadko. Przedstawiamy trzech chorych, u których porażenie nerwu twarzowego wystąpiło w sześć i siedem dni po niepowikłanej myringoplastyce. Porażenie nerwu twarzowego pogłębiało się do stopnia VI (skala Hausa-Brackhmana) i cofnęło się po zastosowaniu sterydów u dwóch chorych. U trzeciego chorego wykonano dekompresję nerwu twarzowego po czterech tygodniach leczenia zachowawczego i stwierdzono rozległy obrzęk nerwu. Czynność nerwu twarzowego u tego chorego poprawiła się do II stopnia. Omówiono możliwą etiologię i postępowanie w odległym porażeniu nerwu twarzowego po operacji ucha środkowego.