CASE REPORTS

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DELAYED-ONSET UNILATERAL FACIAL PARALYSIS
AFTER MASTOIDECTOMY: A CASE REPORT

Objective: To discuss the case of a 36-year-old man who presented with left unilateral facial paralysis 11 days after mastoidectomy.

Methods: Design: Case Report
Setting: Tertiary Government Training Hospital
Patient: One

Results: A 36-year-old man with recurrent left ear discharge of 30 years duration underwent left canal wall-down mastoidectomy and was discharged well after 3 days. On follow up after 8 more days, he was noted to have House-Brackmann IV left facial paralysis. Following 5 days methylprednisolone, neurologic evaluation and physical therapy rehabilitation, facial paralysis improved in the ensuing weeks until House-Brackmann I was achieved at week 12.

Conclusion: Delayed-onset Facial Palsy (DFP) following tympanomastoid surgery may be approached conservatively, including steroids, acyclovir, and, if with a history of herpes or varicella infection, immunization can be given. Prognosis for DFP is good especially when the facial nerve is identified intraoperatively during otologic surgeries.

Keywords: facial nerve; mastoidectomy; otologic surgeries, unilateral facial paralysis, viral reactivation

A thorough knowledge of the intricate, convoluted course of the facial nerve and its anatomic relationship to other vital structures in the temporal bone is essential to the otologic surgeon. Proper identification of the facial nerve intraoperatively is crucial for preventing facial paralysis. What if the complication was not present after surgery, but developed over a week later? We discuss one such case.
CASE REPORT

A 36-year-old man with recurrent left ear discharge of 30 years duration underwent an uneventful left canal wall-down mastoidectomy for chronic ototympanomastoiditis with cholesteatoma. He was discharged well after 3 days on oral antibiotics and a pain reliever. On follow up after 8 days, he had a House-Brackmann IV unilateral facial paralysis - drooling when he would drink liquids and being unable to close his left upper eyelid or puff his left cheek. (Figure 1) A review of the past medical history yielded childhood varicella infection but no other viral exanthems, no other drug intake or history of stroke. He was started on methylprednisolone 16 mg tab for 5 days and was referred to a neurologist and physical therapy for rehabilitation. No nerve conduction studies were done, but he was on weekly follow up with monitoring of his muscles of facial expression. (Figure 1) Facial paralysis improved week after week until he achieved House-Brackmann II - III on his 5th week and House-Brackmann I by the 12th week and beyond. (Figure 2)

DISCUSSION

Facial nerve paralysis as a complication of middle ear surgery is dreadful for the otologic surgeon. Usually caused by surgical trauma or local anesthetic use, it is uncommon to see onset of facial nerve palsy more than 72 hours following the surgery. In a meta-analysis of 14 patients by Bae and Kwak et al., the overall incidence of delayed facial palsy following middle ear surgery was 0.65% but had multiple etiologies and differed depending on the type of surgery. The mean time onset of facial palsy was 8.47± 3.98 days after surgery, and 95.3% of the patients completely recovered.3

Facial palsy that occurs immediately after middle ear surgery (stapedectomy, stapedotomy, and tympanoplasty) can be a consequence of local anesthetics and regresses completely within a few hours, while delayed facial palsy occurs several days or even weeks after uneventful surgery. The mechanism of the neural dysfunction is not readily defined. Surgical stress, intraoperative trauma, or laceration of the chorda tympani nerve with resultant retrograde facial nerve edema can all be provoking etiological factors. Our patient underwent mastoidectomy before the delayed-onset complication after eight days. Several studies identify tympanomastoidectomy as a high-risk factor due to the invasiveness and extent of the procedure.4,5,6

A 2019 review by Eckermann et al. of 10 studies involving 12,161 patients stated that the incidence of DFP after middle ear surgeries varies between 0.2 and 1.9%. In comparison, Bell's palsy occurs with an incidence of 0.02–0.03%.7 In Eckermann's review, the influence of the type of surgical technique on the incidence of DFP was included.
A comparison showed that the incidence of DFP was lowest in stapedectomy and stapedotomy with 0.43% in mean value and highest in tympanomastoid surgery with 1.18 in mean value. For the surgical technique of simple tympanoplasty, the mean incidence was 0.72% on average.3

Revesz et al. reported that out of 149 KTP laser stapedotomies performed at their department since 2006, 2 cases (0.01%) of delayed facial paralysis occurred.4 The first was a 52-year-old woman who had an uneventful postoperative period and was discharged 3 days after explorative tympanotomy, only to have House-Brackmann grade II facial palsy on the operated side 8 days postoperatively. The second was a 45-year-old man who also underwent an uneventful KTP laser stapedotomy who developed a House-Brackmann grade III facial palsy on the operated side 13 days post-operation.4 Neither patient had any active ear infections during their facial paralysis and both were managed with methyprednisolone and acyclovir, completely recovering (House-Brackmann grade I) several weeks after onset of facial palsy.4 In a study by Mills,5 the temperature in the facial canal was examined during stapedectomy using a laser or microdrill and a mean temperature elevation by 6.2 °C in the facial canal was observed when lasers were used.6 Thermal stress is assumed to be a possible factor for viral reactivation or edema. The use of lasers during middle ear surgery increases the risk of DFP.6

Since our patient had a history of childhood varicella, viral reactivation could be considered as another etiology. De Stefano et al. reported the case of a patient who had facial paralysis 11 days after a successful canal wall down mastoidectomy concluded that when an ipsilateral facial nerve palsy appears more than 72 hours after an uneventful middle ear procedure without symptoms of any infection, viral reactivation should be suspected.2

Bonkowski6 focused on viral reactivation and attempted to confirm this theory through various tests, identifying HSV-1 and VZV as two main representatives of viral reactivation. Furuta and others6,10 were able to detect the latent viral infection caused by HSV-1 and VZV in investigations of deceased patients who had an idiopathic acute facial paralysis, proving the site of origin of reactivation of latent viral infection in the geniculate ganglion on autopsy. Bonkowski6 opined that the viral reactivation due to surgical stress leads to an immune reaction, which can be responsible for the development of DFP. According to Gianoli et al., DNA detection by PCR should be sought if virus reactivation is suspected.11

Althaus12 cited the occurrence of neuronal edema as another reason for the development of DFP. In the examined articles, edema was postulated as a reason for the development of DFP in approximately 17% of the performed stapedotomies.12 These occurred intra- or immediately postoperatively. The causes were probably surgical stress and small injuries to the tissue.12 Edemas are caused by restricted venous drainage, vascular thrombosis, vasospasm, meningitis or fluid retention.12 Neuronal edema compresses the nerve and disrupts its supply because the facial nerve is located in a confined space.12 As a result, delayed paralysis of the nerve develops. Neuronal edema reaches its greatest extent on the 5th day, then subsides again, and is almost completely receded on the 14th day.13 Althaus12 cites manipulation or separation of the chorda tympani as another triggering cause for the development of neuronal edema. The resulting traumas develop edema that spread retrogradely and thus compress the facial nerve.12 This hypothesis of retrograde edema was also discussed by Blatt and Freeman.14

Patients with dehiscence of the facial canal have a significantly higher probability for a delayed facial palsy.3 Xu15 referred to a bony anomaly in his article wherein he cited facial canal dehiscence for the development of DFP. In his study, 9 of 15 patients with DFP (60%) had dehiscence of the facial canal. In comparison, 20% of patients who underwent surgery had dehiscence without DFP.15 Dehiscence, therefore, increases the risk of developing DFP by the open nerve canal and exposed nerve - in the case of our patient, no facial canal dehiscence was evident on his CT findings. Moreano et al.16 also recorded this problem in their study, where 1000 petrous bones were examined. Of the 1000 facial canals, 560 were dehiscent. The most common location of dehiscence was at the oval window, accounting for 73.5%.16

The recommended therapy of DFP based on the data of the therapy of Bell’s palsy, consists of steroid administration.2 For patients with a history of previous viral infections, antiviral prophylaxis is recommended.5 The meta-analysis of Bae et al.3 compared different therapeutic options for DFP after middle ear surgeries, finding no significant differences between steroids, antivirals, steroids plus antivirals, and no therapy. Glass et al.17 summarized the results of current reviews, meta-analyses and larger randomized trials dealing with Bell’s palsy therapy. An early steroid administration was recommended, with antiviral therapy only for patients with positive viral serology or anamnesis.17 Patients with IgM or IgG antibodies against HSV-1 or VZV for example, are treated with an antiviral such as acyclovir.17 In the study by De Stefano and colleagues,2 patients were treated with 2400 mg of acyclovir per day in addition to steroids if the viral serology was positive for 10 days. Acyclovir can also be administered prophylactically to prevent DFP if the patient had a positive viral anamnesis.4 Another prophylactic therapy is vaccination of high-risk patients; immunosuppressed patients in particular have an increased risk of herpes virus reactivation.18
Overall, DFP has a very good prognosis, with 95% healed completely following appropriate therapy. A review of the medical records of otologic surgeries in our hospital for the past 10 years (2010 to 2019) including interviews with otolaryngologists revealed several mastoid surgeries done but without any complication of delayed-onset facial paralysis on follow-up. Only one case was documented to have unilateral facial paralysis which the patient already had even before the surgery for mastoidectomy. After a search of HERDIN Plus and the ASEAN Citation Index (ACI), we found no locally published case of delayed-onset facial paralysis after otologic surgery; a study by Hardillo et al. of an unusual case of facial paralysis occurred with a pre-existing right sided paralysis and an ipsilateral infraauricular mass with ear discharge.

Our experience in this case teaches us that delayed-onset facial palsy (DFP) following tympanomastoid surgery may be approached conservatively, including steroids, acyclovir, and, if with a history of herpes or varicella infection, immunization can be given. It affirms the literature that the prognosis for DFP is good especially when the facial nerve is identified intraoperatively during otologic surgeries.

REFERENCES