JBNC Brazilian Journal of Neurosurgery
Jornal Brasileiro de Neurocirurgia

Delayed facial nerve palsy after vestibular schwannoma surgery - case report

Paralisia facial tardia após cirurgia de schwanoma vestibular - relato de caso

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ABSTRACT

Background: *Facial nerve dysfunction may occur immediately after vestibular schwanoma surgery. Electromyographyc monitoring of motor cranial nerves during cerebellopontine angle surgery has become an essential tool. Although delayed onset of facial nerve dysfunction hours to months following vestibular schwanoma surgery are rare.* ***Case description:*** *We describe the case of a 70-years-old male who was admitted with a left side tinnitus and hearing loss of the last 3 years. Magnetic resonance imaging (MRI) T1-weighted demonstrated an isointensity lesion, 30mm in diameter, at the left cerebello-pontine angle with a small portion in the internal auditory canal. The patient was surgically treated by means of a standart suboccipital retrosigmoid approach. The facial nerve was monitored by continuously during surgery. Surgical removal was macroscopically complete. The facial nerve was well-preserved during surgery and showed at the end of the procedure normal electromyographic activity. The patient did well postoperatively and was discharged at the 4th postoperative day and facial function was normal (House-Brackmann grade I). On the 10th postoperative day he notices difficult closing his left eye that progressed to complete facial nerve palsy (House-Brackmann grade III). Steroid therapy was performed for five days associated with physical therapy. One month later his facial nerve function had completely recovered. After six months, the patient remains asymptomatic and neurologically intact. MRI obtained at the 16th postoperative day showed intense enhancement of the intracranial facial nerve segment and also demonstrated no residual tumor. Immunological study at the time of onset showed herpes simplex virus antibody titer normal as well as those for herpes zoster virus.* ***Conclusions:*** *Delayed facial nerve palsy remains an under reported and consequently not very known phenomenon in the neurosurgical practice and literature. Because of the favorable rate of recovery, patients should be reassured in the interim and should not undergo any corrective surgical procedures to improve facial nerve function. Delayed facial nerve palsy is uncommon after vestibular schwannoma surgery. Excellent recovery of facial nerve function to the original postoperative status nearly always occurs in those circumstances.*
Key-words: *Delayed facial palsy, vestibular schwannoma, monitoring.*

SUMÁRIO

Histórico: *A disfunção do nervo facial pode ocorrer imediatamente após a cirurgia de schwanoma vestibular ou mais raramente, horas ou dias depois da operação. Atualmente, a monitoração neurofisiológica intra-operatória é uma ferramenta importante para prevenir tal lesão nas intervenções do ângulo ponto-cerebelar.* ***Descrição do caso:*** *Paciente masculino, 70 anos, com diminuição da acuidade auditiva esquerda e zumbido há 3 anos. A ressonância magnética (RM) demonstrou lesão isointensa, hipercaptante de 30mm no ângulo ponto-cerebelar esquerdo, com uma pequena porção intracanalicular. O paciente foi operado por via suboccipital retrosigmóide, com monitorização intraoperatória do nervo facial por eletroneuromiografia. A remoção cirúrgica foi macroscopicamente completa. O nervo facial foi bem preservado durante o procedimento e ao término apresentava atividade elétrica normal. O paciente teve boa evolução e recebeu alta no 4º dia pós-operatório com a função do nervo facial normal (House-Brackmann grau I). No 10º dia pós-operatório iniciou com dificuldade de fechar o olho esquerdo, até paralisia completa do nervo facial (House-Brackmann grau III). O tratamento com corticóide foi realizado por 5 dias associado àfisioterapia. Em um mês houve recuperação completa da paralisia e*

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Recebido em junho de 2008. Aceito em agosto de 2008.

JBNC Brazilian Journal of Neurosurgery
Jornal Brasileiro de Neurocirurgia

após seis meses, o paciente estava assintomático e neurologicamente intacto. A RM realizada no 16º dia pós-operatório apresentou aumento de sinal em segmento do nervo facial e ausência de resíduos tumorais. O estudo imunológico no pós-operatório mostrou títulos normais de anticorpos séricos contra o vírus herpes simples e herpes zoster. ***Conclusões:*** *A paralisia facial tardia é um fenômeno raro, pouco relatado e conseqüentemente pouco conhecido na prática neurocirúrgica. Considerando os altos índices de recuperação da lesão do nervo facial, não se deve indicar precocemente o tratamento cirúrgico.* ***Palavras-chave:*** *Paralisia facial tardia, schwanoma vestibular, monitorização.*

Introduction

Electromyographyc monitoring of motor cranial nerves during cerebellopontine angle surgery has become an essential tool[10]. First introduced by Krauss[12] in the late 19th century for the monitoring of a cochlear neurectomy for an intractable tinnitus, it was further developed and perfected over the years. In 1979, Delgado et al.[3] introduced intraoperative facial nerve monitoring. This technique allowed the early identification and mapping of the facial nerve throughout the process of tumor resection. This has in turn led to successful preservation of the facial nerve during cerebello-pontine angle surgery[3,10]. Delayed onset of facial nerve dysfunction may occur hours to months following vestibular schwanoma surgery and the incidence has been reported to be between 11.7 to 41% of cases[1,5,13,16,19]: facial function recovers spontaneously without specific treatment in many cases[13,17]. However, it has not been found that recovery of the facial nerve function in patients with delayed facial palsy (DFP) is superior to that in patients without delayed facial palsy. Even though many etiologies, such as mechanical, vascular (ischaemia and vasospasm), chemical and inflammatory factors and viral reactivation have been postulated, none has been proved[5,13,17,21].

This clinical condition is an under reported phenomenon in the neurosurgical literature and is characterized by the spontaneous onset of partial to complete facial paralysis in a patient who has otherwise demonstrated excellent facial nerve function in the immediate postoperative period[9].

We describe an uncommon case of facial nerve dysfunction occuring 10 days after vestibular schwanoma (VS) surgery. Some aspects related to etiology, images and management are discussed.

Case report

This 70-year-old white male was admitted with left side tinnitus, hearing loss and episodes of mild headaches for the last 3 years. No abnormalities were observed on neurological examination, except left hipoacusia. Pure tone audiometry demonstrated left side neurosensorial deficit. Magnetic resonance imaging (MRI) T1-weighted demonstrated an isointense lesion, 30mm in diameter, in the left cerebello-pontine angle with a small portion inside the internal auditory canal. On T2-weighted images the lesion appeared isointense. Addition of gadolinium yielded a homogeneous enhancement of the lesion (Fig. 1).

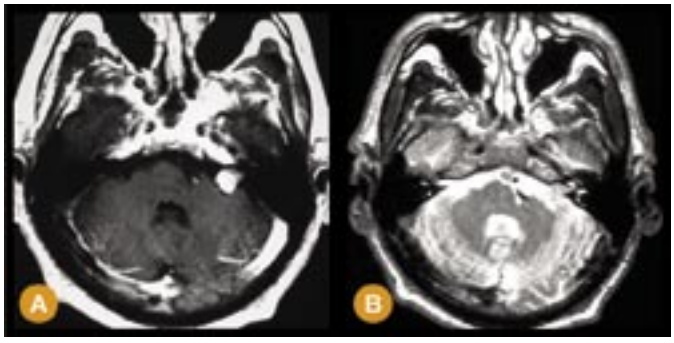

Figure 1: (A) T1-weighted MRI with addition of gadolinium : homogeneous enhancement of a lesion at the left cerebellopontine angle, (B) T2-weighted images.

The patient was surgically treated by means of a standard suboccipital retrosigmoid transmeatal approach. The facial nerve was continuously monitored by electromyography during surgery. Four pairs of needle electrodes were placed in the frontal, orbicularis oculi, orbicularis oris and platisma in the upper cervical region. The facial nerve was electrically stimulated by a monopolar probe with a 0.5-mm tip during and after tumor removal. This was performed in all nerve portions (upper, middle and lowers parts). The stimulation intensities ranged from 0.1 to 1 mA. The surgical removal was macroscopically complete. The facial nerve was well-preserved during surgery and showed at the end of the procedure normal electromyography activity. Histopathological diagnosis was schwannoma.

The patient did well postoperatively and was discharged at the 4th postoperative day with normal facial function (House-Brackmann grade I). On the 10th postoperative day he noticed difficulty in closing his left eye. This progressed during the subsequent 24 hours to clinically facial nerve palsy (House-Brackmann grade III (Fig. 2). Five days dexamethasone therapy and physical therapy during 30 days were prescribed. MRI obtained at the 16th postoperative day showed intense enhancement of the intracranial facial nerve segment and also demonstrated the absence of residual tumor (Fig. 3).

Figure 2: (A) Patient with facial palsy 15 days postoperatively, (B) Two months after, with complete recovery.

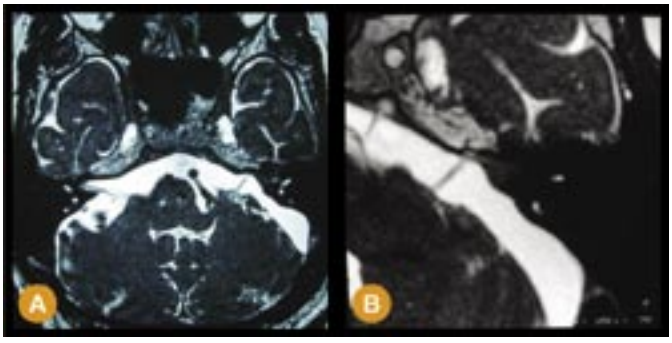

Figure 3: (A) Postoperative T1-weighted MRI showing no residual tumor. (B) Facial nerve preserved.

Immunological serologic study at the time of facial palsy onset showed normal herpes simplex viruses type 1 (HSV 1) and type 2 (HSV 2) antibody titers as well as those for varicella zoster virus (VZV).

Electroneuromyography demonstrated a complete recovery except to orbicularis oris muscle. One month later his facial nerve function had completely recovered (Fig. 2B). The patient remains asymptomatic and neurologically intact after two years of follow up.

Discussion

The improvement of the facial nerve monitoring systems may influence the functional prognosis in vestibular schwannoma surgery. An apparent intact facial nerve sheath could hide nonfunctioning or ruptured fibers[15]. This condition can explain the discrepancy between the intraoperative nerve aspect and facial function outcome[15]. It has been estimated that if 50% of fibers are not functioning, clinical evaluation can be normal if the remaining 50% are normally conducting[5]. The development of a deterioration of function after surgery indicates that the critical level of injured fibers has been surpassed[5].

The onset of DFP can be divided in early and late onset[13,16]. Sargent et al.[17] reported a distribution characterized by two peaks, the first one several hours to two days and the second peak between 8 and 17 days after surgery[17]. In the early onset group, the most likely etiology of DFP is neural edema, especially in the meatal foramen[13,16]. The fact that some surgeons have routinely decompressed the labyrinthine segment of the facial nerve and geniculate ganglion, and have achieved a better outcome, may provide indirect evidence supporting facial nerve edema as the etiology of early onset DFP[13].

Considering the late onset DFP, Sargent et al. suggest some mechanism as: progressive nerve injury by edema or vascular compromise; development of acute nerve compromise at the time of the palsy, perhaps due to the local effects of blood breakdown products that may cause vasospasm; traction on the nerve by shifting of the brain when patients become more upright and active[17].

Magliulo et al. report on a case of patient presenting with an acoustic neuroma operated on ,with immediate postoperative hearing and facial function preservation, although ten days postoperatively facial palsy and hearing loss occurred (Ramsay-Hunt syndrome)[13]. This patient had a history of previously diagnosed herpes zoster reactivation limited to the chest,supposedly to be a predisposing factor to the postoperative deficits. They propose antiviral therapy prophylaxis in these conditions.

Scheller et al. observed seven patients with DFP after acoustic neuroma surgery. The onset occurred between 2-5 days, following a 10 days treatment with vasoactive substances. They conclude that DFP following termination of vasoactive treatment points to a disturbed microcirculation of the nerve as the main pathophysiological feature[18].

Fenton et al. observed that eight of the 67 patients developed a worsening of facial function after the first postoperative day.

Darrouzet et al. studied 400 patients operated of VS and observed 12 (3%) of DFP occurring between the 6th and 10th postoperative days[2].

Franco-Vidal et al. studied in eight patients the role of herpes virus reactivation in the onset of DFP occurring three days after VS surgery[6]. The mean delay was 8.7 days and all patients had a House-Brackmann Grade 1 recovery: mean time to recovery was 40.4 days. A serologic search for specific antibodies (anti-HSV-1, anti- HSV-2 and anti-VZV) revealed either a high level of anti HSV or VZV antibodies, suggesting an HSV or VZV reactivation. They concluded that these reactivations might be responsible for most cases of DFP, thus recommending the use of immediate steroid and acyclovir administration. The viral reactivation mechanism is comparable to that already suspec-

ted in DFP occurring with the same delay in middle ear surgical procedures.

Furukawa et al. observed a case of DFP after microvascular decompression for hemifacial spasm seven days later[7]. Serum antibody of VZV was increased and MRI demonstrated an enhancement of a geniculate ganglion of the left facial nerve, indicating inflammation. Gianoli observed high IgM titers of HSV-1, HSV-2 and VZV antibodies in patients with DFP after SV surgery[8].

No patients had delayed facial palsy after radiosurgery in 45 patients treated of VS by Huang et al[11].

MRI of the facial nerve after surgery of VS has been rarely reported[16]. Ohata et al. demonstrated that surgical manipulation of the facial nerve in the auditory canal during VS surgery does not induce postoperative enhancement of the facial nerve at the labyrinthine segment if the facial function remains intact[16]. Thus, it is reasonable to predict that this findings when present are not caused by the operative procedure, but probably by the herpetic infection.

In the present case, it was observed facial nerve enhancement at the labyrinthine segment in MRI but the titer of serum herpes simplex was normal.

Normally, the prognosis for patients who develop DFP after VS resection is excellent and approximately 88% will recover to the initial grade or better within three months postoperatively and full recovery within one year[5,9].

Age, sex, tumor consistency and operative approach seem to be not significant factors in predicting postoperative facial nerve function[4], although Vabrec et al. observed different data of DFP according to operative approach (middle fossa 18%, translabyrinthine 11% and retrosigmoid 0%)[20].

DFP remains an undereported and consequently not very known phenomenon in the neurosurgical practice and literature[9]. Because of the favorable rate of recovery, patients should be reassured in the interim and should not undergo any corrective measures to improve facial nerve function.

DFP is uncommon after VS surgery and an excellent recovery of facial nerve function to the original postoperative status nearly always occurs in those circumstances.

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