INTRODUCTION

Delayed peripheral facial paralysis after uncomplicated stapedotomy surgery is well recognized in the otologic literature. Since first mentioned by Althaus and House in 1973, several other authors have described patients with similar clinical presentations. The typical scenario is a patient who has undergone an uncomplicated stapes operation for otosclerosis and develops an ipsilateral facial paresis/palsy several days to weeks later. Most patients eventually recover full facial function. This unusual event is reported in less than 0.004% of patients within the current clinical literature. To our knowledge, no imaging features of this entity have ever been reported.

Several hypotheses exist as to the etiology of this nerve palsy. These include edema of a deficient facial nerve, retrograde edema of the chorda tympani nerve, otitis media, and reactivation of a latent viral infection (i.e., Herpes simplex virus). Studies have added evidence to support the theory that reactivation of a latent herpes virus is the etiology for the facial nerve dysfunction. A similar theory has been attributed to the cause of idiopathic facial paralysis or Bell’s palsy. Many authors control that it is the reactivation of a latent viral infection that causes this idiopathic facial dysfunction.

In conjunction with this theory, several studies have demonstrated characteristic findings of gadolinium enhancement of the facial nerve on MRI in these patients. In patient #2, serial MRI scans with gadolinium were obtained on days 7, 49, and 86, periorbital and acyclovir. Patients were treated with a two-week course of prednisone (60 mg/day x 1 week followed by a one week taper) and acyclovir.

The MRI of patient #1, obtained on day 8 after onset of facial paralysis, also demonstrated a similar enhancement of the labyrinthine portion of the facial nerve with extension toward the fundus of the internal auditory canal. In patient #2, serial MRI scans with gadolinium were obtained on days 7, 49, and 86, after the onset of facial paralysis. Both patients demonstrated gadolinium enhancement of the labyrinthine portion of the facial nerve with extension toward the fundus of the internal auditory canal, with subsequent reduction in enhancement over time. (Fig. 2)

RESULTS

A total of two patients developed delayed facial paralysis after their stapes surgery out of 450, for an incidence of 0.004%.

Neither patient had a history of recent herpes simplex infection or varicella zoster reactivation.

In our cohort of patients, the incidence of delayed facial paralysis after stapedotomy was 2 in patients out of a series of 450 reviews, for an incidence of 0.004%. In the literature, the incidence of delayed facial paralysis after stapedotomy is 0.22% to 0.54%. The incidence appears to be slightly higher in patients who undergo stapes procedures. 5, 12. This latter finding is presumably due to thermalreactivation of latent herpes viruses.

The two patients in our study both demonstrated gadolinium enhancement of the labyrinthine portion of the facial nerve. This finding is characteristic of the changes that occur in Bell’s palsy. Although enhancement of the labyrinthine segment can be seen in other circumstances such as tumor, trauma, or inflammatory change, the fact that both groups of patients (i.e., those with delayed facial paralysis after stapes surgery and patients with Bell’s palsy) demonstrate identical MRI findings supports the theory that they share the same pathogenesis.

We do not understand the hypothesis that delayed facial paralysis after stapedotomy is caused by reactivation of a herpes virus as in Bell’s palsy, it would be logical to treat these patients with steroids and antiviral medications. Although the data is mixed on the effectiveness of steroids and acyclovir in the treatment of Bell’s palsy, substantial proof exists supporting the use of these medications in the treatment of idiopathic facial paralysis.

The appearance of facial paralysis after stapedotomy is an alarming clinical finding. The assessment of this patient by both physical examination and MRI provides important diagnostic information for the appropriate management of this patient. If the MRI demonstrates enhancement of the labyrinthine segment of the facial nerve, then the clinician can confidently treat this patient in a similar fashion. If MRI findings conflict with this assumption, then alternate etiologies need to be addressed.

DISCUSSION

In the rare complication of delayed facial paralysis after stapedotomy, MRI is a useful diagnostic tool to dictate the treatment strategy. The initial MRI demonstrates gadolinium enhancement of the labyrinthine segment, whereas the MRI scan demonstrates enhancement of the facial nerve in the labyrinth segment. Moreover, a history of HSV infection which is under treated stapes surgery should be prophylactically treated with acyclovir during the perioperative period.

CONCLUSIONS

In the rare complication of delayed facial paralysis after stapedotomy, MRI is a useful diagnostic tool to dictate the treatment strategy. A small group of patients may be treated medically when the MRI scan demonstrates enhancement of the facial nerve in the labyrinth segment. Moreover, a history of HSV infection which is under treated stapes surgery should be prophylactically treated with acyclovir during the perioperative period.

REFERENCES