

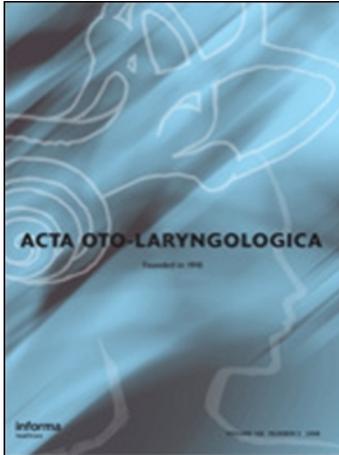
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ORIGINAL ARTICLE

Correlation between enhanced MRI and surgical findings in herpes zoster oticus

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Abstract

Conclusion: This study demonstrates good correlation between enhanced MRI and surgical findings. **Objectives:** This study investigated the reliability of enhanced magnetic resonance imaging (MRI) to make a surgical decision on the strategy for facial nerve decompression in herpes zoster oticus, by determining the degree of correlation between contrast enhancement in MRI and the pathologic change in the facial nerve. **Subjects and methods:** This retrospective study of 13 patients, who underwent facial nerve decompression with herpes zoster oticus, was designed to compare gadolinium-enhanced segment of facial nerve on MRI and the pathologically changed segment confirmed by surgical exploration, grouping them by the timing of operation after onset of facial paralysis. **Results:** Commonly enhanced segments on MRI were the labyrinthine, intracanalicular, and geniculate ganglion, found in 84%, 69%, and 69% of all patients, respectively. The most common pathologic segment was the labyrinthine segment (92%), followed by the geniculate ganglion (84%).

Keywords: *Herpes zoster oticus, magnetic resonance imaging, MRI, facial nerve, decompression*

Introduction

The treatment of patients with facial paralysis caused by varicella zoster virus greatly depends on the MRI examination for the inspection of the degree and the site of pathologic change along the facial nerve [1,2].

The enhanced segment of facial nerve on MRI is not equal to the pathologic segment of facial nerve because the enhancement of facial nerve is affected by the anatomical blood supply [3], histological structure of the facial nerve [4], and the surrounding bony and neural structures along its course [5]. This means that the sensitivity or specificity of MRI examination could vary considerably depending on the segment of facial nerve studied [6].

Demonstrating the correlation between the enhancement on MRI and the actual pathologic change of facial nerve is very difficult or even impossible until direct surgical exploration is conducted. Although the surgical exploration of facial nerve is limited to patients with severe facial paralysis, the comparison of pathologic segment of facial nerve with the enhanced segment of facial

nerve on MRI could be valuable in deciding the surgical strategies of facial nerve decompression.

Unfortunately, previous studies concerning the relationship between contrast enhancement seen on an MRI and real pathologic changes in the facial nerve have not achieved consensus and therefore provide little guidance as to the appropriate surgical approach.

Therefore, in this study, we attempted to confirm the pathologic change of the facial nerve by surgical exploration in the segment that was enhanced on MRI, and determine the correlation between contrast enhancement in MRI and the pathologic change of facial nerve that was caused by herpes zoster oticus alone (not Bell's palsy or post-traumatic facial paralysis), considering many unique biologic properties and clinical characteristics.

Subjects and methods

Subjects

Thirteen patients with herpes zoster oticus who underwent facial nerve decompression via the mid-

dle fossa approach or combined transmastoid approach between 1996 and 2007 were included in this study. The patients included eight men and five women, aged from 16 to 63 years. None of them had a history of infection, trauma, or neurologic disorder, but all had clinically complete facial paralysis on the day of admission. The clinical diagnosis of herpes zoster oticus was made on the basis of the classic triad of ear pain, facial paralysis, and herpetic rash of the external auditory canal or pinna. Confirmation of an active herpes zoster infection was made by laboratory parameters (elevated IgM or IgG antibodies against varicella zoster virus). The facial paralysis was graded according to the House-Brackmann (H-B) classification system [7] and facial nerve function was investigated by electro-neurography (ENoG) within 3 weeks after onset of facial paralysis. MRI was performed for the accurate identification of involved lesion of facial nerve. The mean interval between the onset of paralysis and MRI was 25.3 days (range 6–59 days). For the surgical decompression, the patients met the following criteria in this study: (1) worst score of facial palsy, H-B grade V or VI, (2) degree of degeneration as measured by ENoG exceeded 90%, (3) no improvement after steroid treatment and anti-viral agent, and (4) no systemic disease. The patients were classified into three groups depending on the timing of performance of the operation after onset of facial paralysis: (1) within 3 weeks after the onset of symptoms, (2) between the 3rd and 4th week, (3) after the 8th week.

Methods

MRI scans were obtained with a 1.5 T imager (GE Medical System, Philips) in coronal, axial, and sagittal planes; T1 and T2 sequences; and T1-weighted post-contrast examination immediately after intravenous Gd-DTPA injection. The sequences were performed with a slice thickness of 1 mm. The following segments of the facial nerve were analyzed: intracanalicular, labyrinthine, geniculate ganglion, tympanic, and mastoid segment, as shown in Figure 1. The pattern of enhancement was visually assessed by two experienced neuroradiologists who were blinded to the clinical presentation and each other's opinion in order to judge contrast enhancement as definite or not. The final judgment was reached by consensus. Contrast enhancement of the non-paralyzed facial nerve was compared with that of the paralyzed nerve.

Using the middle cranial fossa approach, the facial nerve was decompressed from the intracanalicular segment to the proximal tympanic segment. A combined transmastoid approach was performed if

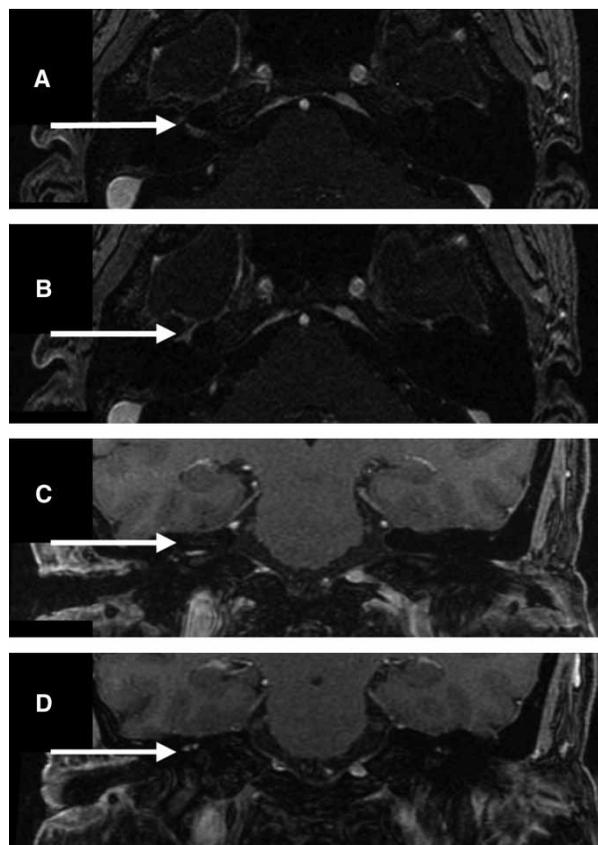


Figure 1. Gadolinium-enhanced MRI of a 33-year old woman with Ramsay-Hunt syndrome on the right side. (A) Axial T1-weighted enhanced MRI shows the intracanalicular segment. (B) Axial T1-weighted enhanced MRI shows the labyrinthine segment, geniculate ganglion, and tympanic segment. (C) Coronal T1-weighted enhanced MRI shows the intracanalicular segment. (D) Coronal T1-weighted enhanced MRI shows the labyrinthine and tympanic segments.

contrast enhancement was found in the tympanic and mastoid segment.

During surgery, the surgeon assessed and recorded the pathologic change of facial nerve into three groups under an operating microscope: i.e. normal, swelling, and pale or shrunken status of the intracanalicular, labyrinthine, geniculate ganglion, tympanic, and mastoid segment, immediately after the facial canal was opened.

Although the judgment of pathologic change was made subjectively by the surgeon's view, the reliability of the judgment was ensured by other neurosurgeons, by re-examining the photograph, and digital video demonstrations that were taken during the operation (Figure 2).

To calculate the relationship of MRI and surgical findings of the facial nerve in Ramsay-Hunt syndrome, the positive predictive value of MRI was analyzed at each segment depending on the timing of the operation after onset. In addition, Fisher's exact

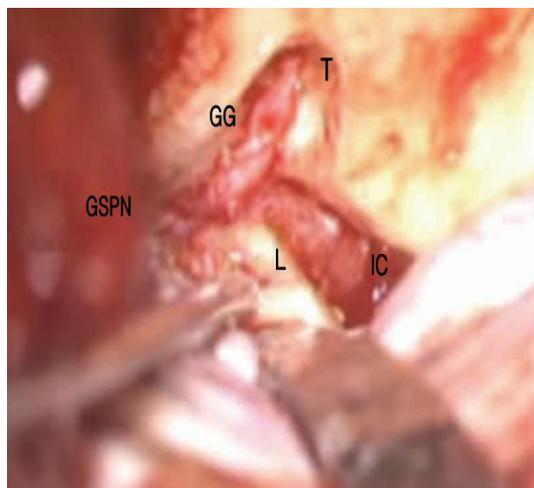


Figure 2. A 43-year-old man with Ramsay-Hunt syndrome on the right side. The pathological findings of facial nerve in a middle cranial fossa approach after incising the nerve sheath. Hyperemic and edematous swelling changes are observed in the intracanalicular segment, labyrinthine segment, and geniculate ganglion of the facial nerve. T, tympanic segment; GG, geniculate ganglion; GSPN, greater superficial petrosal nerve; L, labyrinthine segment; IC, intracanalicular segment.

test was used to compare the results of MRI and surgical findings statistically.

Results

Contrast enhancements of the facial nerve on MRI were present in five segments of one patient, four segments of three patients, three segments of six patients, two segments of two patients, and no enhanced segment at all for one patient. There was no correlation between the extent of involvement of facial nerve on MRI and the course of facial paralysis.

Degeneration of the facial nerve was present on surgical exploration in four segments of two patients, three segments of five patients, two segments of three patients, and one segment of three patients, respectively. The average time from the onset of facial paralysis to facial nerve surgery was 36.3 days (Table I).

Contrast enhancement findings for each segment of facial nerve on MRI

Abnormal enhancement of the contralateral non-paralyzed facial nerve was not found in any patient. Contrast enhancement of the intracanalicular and the labyrinthine segment was found in 69% and 84% of the patients, respectively. Contrast enhancement of the geniculate ganglion, tympanic segment, and mastoid segment were found in 69%, 53%, and 15%, respectively (Figure 3).

Pathologic findings for each segment of facial nerve confirmed by surgical exploration

The pathologic changes in facial nerves in the 13 patients are summarized in Table I. The most common pathologic segment was the labyrinthine segment (92%), followed by the geniculate ganglion (84%) (Figure 4). Hyperemic, edematous swelling or vascular engorged changes of facial nerve were observed after the neural sheath was opened. But, occasionally, in the labyrinthine segment, pale or shrunken change of facial nerve was found in cases where surgery was performed a long time after onset.

Correlations between contrast-enhanced MRI and surgical findings

The positive predictive value of contrast enhancement was assessed for each segment and discrepant incidence between MRI findings and operative findings was charted, dividing them into three groups according to the surgical timing after onset of facial paralysis (Figure 5).

In the group of patients who underwent surgical decompression within 3 weeks after onset of facial paralysis ($n=3$), the positive predictive value in all segments reached 100% except for the intracanalicular segment. Therefore, it can be concluded that good correlation between enhanced MRI and pathological change is present in the acute stage of facial paralysis.

In the group of patients who underwent surgical decompression between 3 and 4 weeks after the onset of facial paralysis ($n=5$), the discrepancy in the intracanalicular segment increased. Enhancement of the intracanalicular segment and mastoid segment on MRI that was actually not involved was observed in this subacute stage. But enhancement of the labyrinthine segment, geniculate ganglion, and tympanic segment still maintained high positive predictive value, which could mean that enhancement in these segments can be trusted in making a decision for decompression surgery.

In the group of patients who underwent surgical decompression over 8 weeks after onset of facial paralysis ($n=5$), overestimation of the actual lesion by MRI enhancement was remarkable in the intracanalicular and tympanic segments. However, high positive predictive value was observed constantly in the labyrinthine segment and geniculate ganglion.

There was no difference in the MRI enhancement between nerve swelling and nerve shrinkage shown in the labyrinthine segment in cases where surgery was performed a long time after onset of paralysis.

The recovery of the facial nerve function of the patients after facial nerve surgery was evaluated using the H-B system. When we checked the grade

Table 1. The data for 13 patients with herpes zoster oticus who underwent facial nerve surgery.

Case no.	Gender/age (years)	Timing of operation after onset of paralysis	Facial function (H-B grade system)		Gd-enhanced MRI/pathologic change confirmed by surgery				
			Preoperative	Postoperative	Intracanalicular segment	Labyrinthine segment	Geniculate ganglion	Tympanic segment	Mastoid segment
1	M/63	24	6	2	-/N	-/SW	-/N	-/N	-/ND
2	M/22	23	6	2	+ /SW	+ /SW	-/N	+ /SW	-/ND
3	F/50	28	5	2	+ /N	+ /SW	+ /SW	+ /SW	+ /N
4	M/47	56	5	1	+ /N	+ /SH	+ /SW	+ /SW	-/ND
5	M/43	26	5	2	+ /SW	+ /SW	+ /SW	-/N	-/ND
6	F/57	22	6	2	+ /N	-/N	+ /SW	-/N	-/ND
7	F/33	58	6	3	+ /SW	+ /SW	+ /SW	+ /SW	-/ND
8	M/44	62	5	3	+ /N	+ /SH	+ /SW	+ /SW	-/ND
9	M/16	52	5	3	-/N	+ /SW	+ /SW	+ /N	-/ND
10	F/34	70	5	3	-/N	+ /SW	+ /SW	+ /SW	-/ND
11	M/33	18	5	4	-/SW	+ /SW	-/N	-/SW	+ /ND
12	F/24	13	6	1	+ /N	+ /SW	-/N	+ /N	-/ND
13	M/22	21	5	2	-/N	+ /SW	+ /SW	+ /N	-/ND

+, enhancement; -, no enhancement in MRI; SW, swelling; SH, shrinkage; N, normal in surgery; ND, not determined.

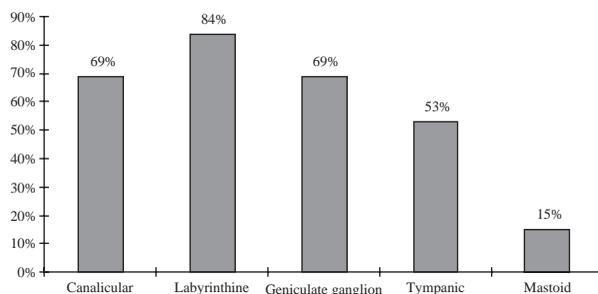


Figure 3. Distribution of gadolinium-enhanced segments of facial nerve.

of facial nerve function 6 months after operation, two of the patients were H-B grade I, six were grade II, three were grade III, and one was grade IV. All of them were shown to have improved facial nerve function, even though four patients had facial nerve decompression delayed over 8 weeks after onset.

Discussion

In our study, the enhancement of facial nerve on MRI was inhomogeneous in herpes zoster oticus, and the predictive value of the enhancement was different according to the site and the timing.

As elicited by other reports, the anatomic distribution of the arteriovenous plexus partially explains the difference in the enhancement in normal facial nerve [8,9] and the inflammation caused by varicella zoster virus can change the permeability of the blood-nerve barrier, leading to abnormal contrast enhancement [10] and difficulty in explaining the enhancement on MRI, and so in 1992 Gebarski et al. reported the limitations of facial nerve enhancement [6].

Daniels et al. reported that facial nerve enhancement on MR images proved to be a nonspecific finding and that further work was required to determine the diagnostic importance of facial nerve enhancement on MRI [11].

However, in our study, we found that there was good correlation between the enhancement on MRI

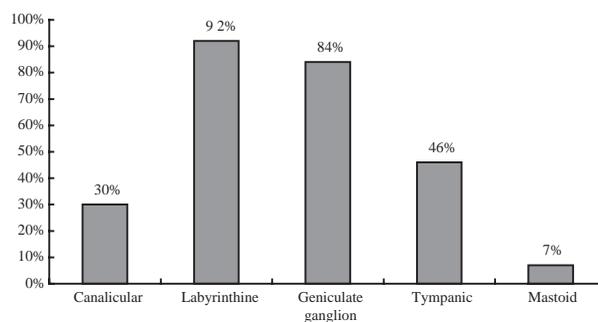


Figure 4. Distribution of pathologic segments of facial nerve.

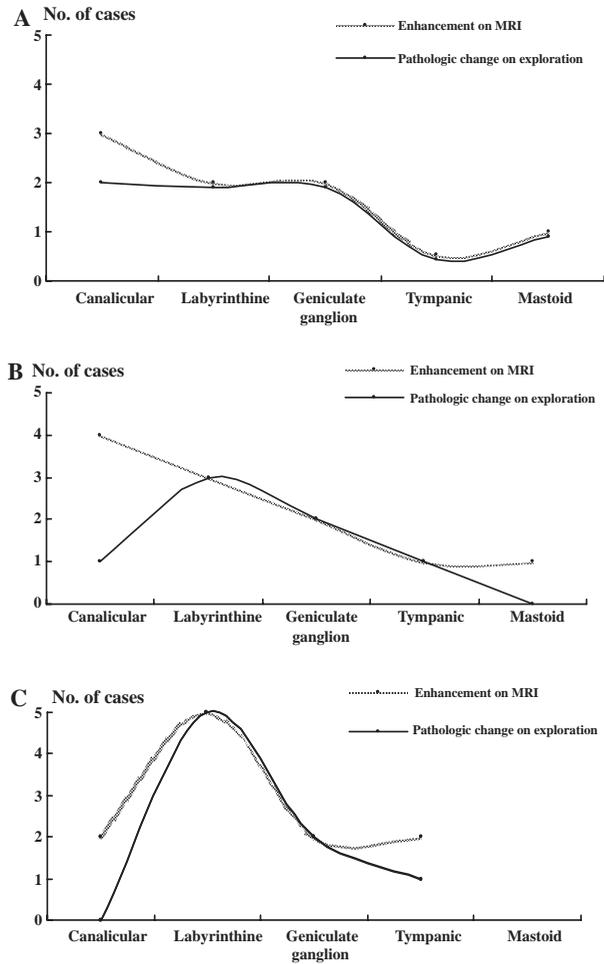


Figure 5. The number of cases of degenerated segments that was confirmed on surgical exploration among the enhanced segments of facial nerve on MRI. This graph shows positive predictive value indirectly for A the group that had surgical decompression within 3 weeks of the onset of symptoms n3, B the group that had surgical decompression between 3 and 4 weeks after onset n5, and C the group that had surgical decompression over 8 weeks after onset n5.

and the pathologic change, especially at the labyrinthine segment and geniculate ganglion in the acute stage (defined previously as ‘within 3 weeks after onset’).

In cases of facial paralysis, there have been many reports concerning the enhancement pattern on MRI. Schwaber et al. [12], studied 17 Bell’s palsy cases, demonstrating facial nerve enhancement in the distal auditory canal, labyrinthine/geniculate segment, and less often in other segments. This is in agreement with our study, which found enhancement of the intracanalicular, labyrinthine, and geniculate ganglion in 69%, 84%, and 69% of the patients, respectively. This can be explained by the reports by O’Donoghue et al. [13] and Sartoretti-Schefer et al. [10] stating that viral inflammation of

the geniculate ganglion causes enhancement of the peri-geniculate ganglion on MRI.

The present study focused on the reliability of MRI and the real pathologic neural change in herpes zoster oticus. We used the middle fossa approach to decompress the facial nerve from the intracanalicular segment to the tympanic segment, and if strong enhancement of mastoid segment was definite, a combined transmastoid approach was performed; the transmastoid approach was not always performed alone. So it is impossible to confirm the correlation between MRI and surgical findings of the mastoid segment in this study.

This is the optimal approach for decompression of the facial nerve centering around the geniculate ganglion, as J Ramsay Hunt suggested [14], and hyperemic, edematous swelling or vascular engorged changes of facial nerve were observed after the neural sheath was opened in the labyrinthine segment (92%), geniculate ganglion (84%), and tympanic segment (46%), in order of frequency. Although many authors have questioned the validity of the geniculate ganglion theory [15], the results of our study that demonstrated pathologic change of the facial nerve support Hunt’s suggestion.

In two cases where surgery was performed a long time after onset, palely shrunken nerves were observed in the labyrinthine segment, which had been enhanced on preoperative MRI. It is thought that the lasting blood–nerve barrier breakdown gives rise to gadolinium uptake on enhanced MRI, despite the sparse vascularity in the labyrinthine segment [16] with the lapse of time.

The serial changes in the correlation between enhancement on MRI and surgical findings in our study, even if they have no statistical significance due to a limited number of cases, helped to determine that the high positive predictive value was found in the labyrinthine segment and geniculate segment regardless of the timing of MRI examination.

The anatomical characteristics of the labyrinthine segment include the fact that it is the narrowest part of the facial canal, especially its proximal portion [17] and that it has a sparse blood supply. So the enhancement of this segment on MRI despite poor blood supply could be reliable evidence of pathological change in the labyrinthine segment, without false positive results.

The vascularity of the arteriovenous plexus is lush in the geniculate ganglion, the tympanic segment, and the mastoid segment [10], with the capillary component especially prominent in the geniculate ganglion. Unlike the tympanic or mastoid segment, cytopathologic change in the geniculate ganglion, as in Hunt’s theory, added to the high positive predictive value of enhancement.

There has been much debate as to the criteria for the optimal timing of surgery in surgical decompression of the facial nerve. Although Fisch [18] recommended that immediate middle fossa decompression should be performed only in patients with degeneration of 90% or more within 2 weeks of the onset of the paralysis for Bell's palsy, we have conducted facial nerve decompression in five patients with enhancement on MRI and complete paralysis continuing for 8 weeks. As it was reported by Honda et al. [19] that facial nerve swelling had persisted even beyond the 16th week after onset in herpes zoster oticus, the persistence of nerve swelling was also found beyond the 8th week after onset, in the geniculate ganglion in five patients, the labyrinthine segment in three patients, and the tympanic segment in three patients in our study. The relief of entrapment of the facial nerve performed a long time after onset of paralysis should be investigated further in comparison with non-surgical treatment for complete facial paralysis. However, in our study, all of the patients showed improved facial nerve function better than H-B grade III, even though it could not be proven whether the decompression was beneficial.

Conclusion

This study demonstrates good correlation between enhanced MRI and surgical findings. The enhanced MRI may be useful in the acute stage of herpes zoster oticus and the enhancement of the labyrinthine segment and the geniculate ganglion was reliable regardless of onset period.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

References

- [1] Osumi A, Tien RD. MR findings in a patient with Ramsay-Hunt syndrome. *J Comput Assist Tomogr* 1990;14:991-3.
- [2] Li J, Xiong L, Jinkins JR. Gadolinium-enhanced MRI in a patient with AIDS and the Ramsay-Hunt syndrome. *Neuroradiology* 1993;35:269.
- [3] Kishimoto S, Saito H. Blood distribution in the facial nerve in temporal bones acquired from cases with subarachnoid hemorrhage. *Laryngoscope* 1998;108(4 Pt 1):576-9.
- [4] Lundborg G. Structure and function of intraneural microvessels as related to trauma, edema formation, and nerve function. *J Bone Joint Surg* 1975;57A:938-48.
- [5] Blunt MJ. Ischemic degeneration of nerve fibers. *Arch Neurol* 1960;2:528-36.
- [6] Gebarski SG, Telian SA, Niparko JK. Enhancement along the normal facial nerve in the facial canal: MR imaging and anatomic correlation. *Radiology* 1992;183:391-4.
- [7] House JW. Facial nerve grading system. *Laryngoscope* 1993; 13:1056-69.
- [8] Balkany T, Fradis M, Jafet BW, Rucker NC. Hemangioma of the facial nerve: role of the geniculate capillary plexus. *Skull Base Surg* 1991;1:59-63.
- [9] Balkany T, Fradis M, Jafet BW, Rucker NC. Intrinsic vasculature of the labyrinthine segment of the facial nerve: implications for site of lesion in Bell's palsy. *Otolaryngol Head Neck Surg* 1991;104:20-3.
- [10] Satoretti-Schefer S, Wichmann W, Valvanis A. Idiopathic, herpetic, and HIV-associated facial nerve palsies. Abnormal MR enhancement patterns. *AJNR* 1994;15:479-95.
- [11] Daniels DL, Czervionke LF, Millen SJ, Haberkamp TJ, Meyer GA, Hendrix LE, et al. MR imaging of facial nerve enhancement in Bell palsy or after temporal bone surgery. *Radiology* 1989;171:807-9.
- [12] Schwaber MK, Larson TC III, Zealer DL, Creasy J. Gadolinium-enhanced magnetic resonance image in Bell's palsy. *Laryngoscope* 1990;100:1264-9.
- [13] O'Donoghue GM, Michaels L. Histopathological aspects of Bell's palsy. In: Portmann M, editor. *Proceedings of the Fifth International Symposium on the Facial Nerve*. New York: Masson Publishing; 1985. p. 248-52.
- [14] Hunt JR. On herpetic inflammations of the geniculate ganglion. A new syndrome and its complications. *J Nerv Ment Dis* 1907;34:73-96.
- [15] Guldborg-Moller J, Olsen S, Kettel K. Histopathology of the facial nerve in herpes zoster oticus. *Arch Otolaryngol* 1959; 69:266-75.
- [16] Balkany T, Fradis M, Jafek BW, Rucker NC. Intrinsic vasculature of the labyrinthine segment of the facial nerve: implications for site of lesion in Bell's palsy. *Otolaryngol Head Neck Surg* 1991;104:20-3.
- [17] Eicher SA, Coker NJ, Alford BR, Igarashi M, Smith RJ. A comparative study of the fallopian canal at the meatal foramen and labyrinthine segment in young children and adults. *Arch Otolaryngol Head Neck Surg* 1990;116: 1030-5.
- [18] Fisch U. Surgery for Bell's palsy. *Arch Otolaryngol* 1981; 107:1-11.
- [19] Honda N, Yanagihara N, Hato N, Kasaki H, Murakami S, Gyo K. Swelling of the intratemporal facial nerve in Ramsay Hunt Syndrome. *Acta Otolaryngol* 2002;122:348-52.