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Prognostic Factors in Herpes Zoster Oticus (Ramsay Hunt Syndrome)

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Objectives: To determine if an accurate prognosis can be made in patients with Herpes zoster oticus (HZO), facial nerve outcomes were assessed at 1-year after onset and compared with symptoms and signs at presentation.
Study Design: Individual retrospective cohort study of 101 records in a case series (level of evidence: Level 2b).
Methods: Symptoms, signs, audiology, and treatment records were analyzed to determine their association with facial nerve outcome at 1 year.
Results: Mean improvement at 1 year for the 101 patients was 3 House-Brackmann (HB) grade units. Initially, severity ranged from HB III to HB VI. Mean recovery was significantly greater for those patients who were initially more affected, although at 1 year, they had still not recovered to the same grade as those initially less affected. Having both incomplete eye closure and a dry eye was associated with less recovery at 1 year. The use of prednisone combined with an antiviral agent, and begun at or after Day 5 of the illness, was related to a better facial nerve outcome. No other symptom, sign, or audiologic feature was of prognostic value.
Conclusion: All patients with HZO improved facial function to some degree, with the mean gain at 1 year after onset being 3 HB grade units. Improvement was less for patients who initially had both incomplete eye closure and dry eye. The group who received a combination of an antiviral medication with steroids given after 5 days had the best facial nerve outcome.
Key Words: Antiviral therapy—Facial nerve—Facial paralysis—Herpes zoster oticus—Prednisone—Ramsay Hunt syndrome—Varicella zoster virus.
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Herpes zoster oticus (HZO), also known as Ramsay Hunt syndrome, Hunt's syndrome, or geniculate ganglion herpes, is a viral illness that can cause serious and permanent damage to the facial and other cranial nerves (CNs) (1,2). In addition to unilateral facial nerve paralysis (FNP), it may present as a polycranial neuropathy that includes symptoms of hearing loss, vertigo, speech disturbance, and swallowing abnormalities. HZO accounts for 2% to 10% of all cases of FNP. Unlike Bell's palsy (3,4), there are currently few reports of prognosis or of the effect of commonly prescribed treatments on HZO.

HZO is part of the Varicella zoster virus (VZV) group. VZV is a herpes virus that causes 2 distinct clinical syndromes. The primary infection is Varicella (chickenpox virus) that is common and extremely contagious. VZV establishes latency in the dorsal root ganglia (5). Reactivation of latent VZV results in herpes zoster (shingles), which can affect both spinal and CNs. HZO is caused by the reactivation of latent VZV in the geniculate ganglion, thereby affecting the facial (CN VII) and vestibulocochlear (CN VIII) nerves that lie in proximity (6). The subsequent dysfunction of these CNs is thought to be mainly caused by VZV neuritis and, to a lesser extent, by inflammatory edema (7). Maximal facial paralysis usually occurs within 1 week of symptom onset, and thereafter, prognosis for recovery is similar, although not as good as following Bell's palsy (8). Unlike Bell's palsy, late degeneration of the facial nerve function up to 21 days from the onset of symptoms has been described (2).

HZO is commonly characterized by a viral prodrome, such as an upper respiratory tract infection, followed by severe pain in and around the ear (9). Vesicles then erupt, principally involving the pinna, external canal, and drum head. These vesicles may extend to the face and neck down as far as the shoulder and also may appear on the tongue, palate, larynx, or buccal mucosa. The distribution of vesicles follows a dermatomal pattern that depends on the sensory afferent fibers involved. Other clinical manifestations include tinnitus, unilateral sensorineural hearing loss (10), vertigo, nausea, and vomiting (11) It is thought that the type of symptom and the onset sequence may reflect the severity of the individual HZO case and thus the prognosis.

The annual incidence of herpes zoster is estimated to be 1.5 to 4 cases per 1,000 persons (12,13), and the estimated

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incidence of HZO is 5 cases per 100,000, with a significantly increased incidence in patients older than 60 years (14). The condition is less frequent and less severe in children (6,15).

In addition, chronic ganglionitis caused by the VZV may cause post-herpetic neuralgia (16), which is defined as pain persisting at 120 days after VZV onset. This is commonly associated with hypesthesia, which may begin at the time of onset of the disease. The incidence and duration of pain correlates directly with age, becoming worse in older patients (13,17).

Following a diagnosis of HZO, both the medical practitioner and the patient need accurate prognostic information. Although Yeo et al. (18), in a study of 26 patients with HZO, found age, diabetes mellitus, essential hypertension, and vertigo to be the prognostic factors for HZO, there are no current studies with large samples that have considered the prognostic factors influencing outcome in HZO. Accordingly, the purpose of the current study is to assess a large cohort and to examine the demographics, symptoms, and signs of patients with HZO to evaluate the outcome at 1 year and identify prognostic indicators.

MATERIALS AND METHODS

A 20-year retrospective review of a prospectively maintained data base of 1,144 patients with facial nerve disorders was undertaken. The files collected were from 1988 to 2008 and were held in the clinical practice of the consultant otolaryngologist (G. R. C.). The 3 entry criteria for patients in this study were as follows: 1) having vesicles on the pinna, oral cavity, or both; 2) having facial nerve palsy; and 3) a complete set of records including a final House-Brackmann (HB) grade score given at 12 months after onset (19). One hundred forty-eight patients were identified with HZO, of which, 101 had complete records. The most common cause for exclusion was no final HB grade being recorded because of the patient being lost to follow-up at 12 months.

The symptoms noted for each case were recorded, including the presence of pain, facial numbness or paresthesia, excessive tearing, dry eye, tongue numbness, dysgeusia, diplopia, decreased hearing, tinnitus, hyperacusis, vertigo, swallowing dysfunction, hoarseness, and facial tightness. In addition, the sequence of onset of symptoms was recorded to determine whether pain, facial paralysis, or vesicles were noted as the first symptom.

The clinical signs noted were the distribution of vesicles, and patients also were classified as having vesicles either in the ear and/or in the oral cavity. An HB grade was assigned, and examination of other CNs was undertaken.

Audiology results also were examined, and any asymmetrical sensorineural hearing loss involving the affected ear was noted.

Patients with HZO were referred to the clinic for a number of purposes. Seventy-three patients were sent acutely for diagnosis and treatment. Seventeen were sent for prognostication after prolonged paralysis, and 11 were sent for consideration of reanimation for long-standing paralysis.

The 73 patients who were seen in the acute phase were treated with a regimen of prednisone 1 mg/kg orally daily until Day 14 of their illness, then a decreasing dose of 10 mg per day until 0 mg. Acyclovir (200 mg 5 times per day orally) also was given in years before 1998, when famciclovir (250 mg 3 times per day orally) was substituted until Day 21 of the illness. The pharmacologic treatment is continued until Day 21 of the illness to cover the concept of late neural degeneration after HZO (2,20). The retrospective design of this study meant that the treatment given by other medical practitioners could vary to that described above given by the treating author (G. R. C.). The treatment given to those patients who were sent for prognostication (n = 17) and those for reanimation (n = 11) was either no medication, steroids alone, antivirals alone, or a combination of both drugs.

We divided the treated patients into those whose treatment began before Day 5 of the illness (early treatment group) and those whose treatment began after Day 5 (late group). The allocation of patients into early and late groups reflected their eligibility for subsidized pharmaceutical prescription of an antiviral agent (vesicles of <5-d duration) and those who presented later than 5 days, who received no subsidy for antivirals (Pharmaceutical Benefits Scheme Australia). The effects associated with the timing and type of medication were analyzed in relation to the number of days since the onset of FNP and in relation to the initial and final HB gradings.

Statistical Method

The improvement between presentation and 12 months after onset in terms of HB grade units was determined by comparing the HB grade at presentation with the mean HB grade at 12 months. The change in HB grade was examined for any relationship with time and symptom factors, in the same manner as was done by Lo et al. (21).

Thus, each symptom was examined for its effect on the amount of observed improvement. Likewise, the distribution of vesicles and the presence or absence of hearing loss, as determined by audiology, were assessed for their association with improvement in terms of HB grade units.

To conduct a combined analysis of the signs and symptoms, a multivariate analysis was performed, with the amount of improvement as the dependent measure.

Finally, the impact of receiving either prednisone alone, prednisone and an antiviral agent, an antiviral agent alone, or no treatment in relation to time of administration was tested to determine the impact on observed HB grade improvement at 12 months.

RESULTS

One hundred one patients (70 male and 31 female subjects) fitted the entry criteria for the study. Fifty-eight patients had right-sided HZO, and 43 had left. The ages of the patients ranged from 11 to 85 years, with a mean of 49.4 years and a median age of 49 years (Table 1).

The most common symptom reported was pain in and around the ear (90%), followed by decreased hearing on the affected side (43%). Facial pain other than in the periauricular region (37%), imbalance (33%), dysgeusia (31%), and tinnitus (20%) also were common symptoms at presentation (Table 1). The sequence of symptoms revealed that 54.5% had only pain as the first symptom, 22.8% had facial paralysis as the first symptom, and 2% had vesicles as their first presenting symptom. Analysis of the vesicle distribution showed that 86 patients had vesicles in the ear alone, 7 had vesicles in the oral cavity alone, and 8 patients had vesicles in both the ear and oral cavity. The sequence of onset of initial symptom, whether it was pain, paralysis, or vesicles, had no relationship to outcome as measured using the HB grade difference (all $p > 0.21$).

TABLE 1. *Mean House Brackmann grades at onset and at 1 year and the difference between these for various sub-classifications of the patient group. Classification was made according to symptoms, signs, age decade, gender, side-affected, and House Brackmann grade at onset*

Attribute at diagnosis	No. of affected (N = 101)	Mean HB at onset	Mean HB at 1 yr	Mean difference	95% Confidence interval for the difference
HB grade					
I	0	—	—	—	—
II	0	—	—	—	—
III	11	3	1.2	1.8	1.2–2.4
IV	13	4	1.8	2.2	1.7–2.7
V	30	5	2.0	3.0	2.7–3.3
VI	47	6	2.6	3.4	3.1–3.7
Side					
Right	58	5.1	2.2	2.9	2.6–3.2
Left	43	5.1	2.1	3	2.6–3.4
Sex					
Male	29	4.6	1.7	2.9	2.4–3.4
Female	70	5.3	2.3	3.0	2.7–3.3
Age (yr)					
0–10	0	0	0	0	0
11–20	5	4.8	1.4	3.4	2.3–4.5
21–30	11	5.5	2.2	3.3	2.6–4.0
31–40	14	5.3	1.9	3.4	2.8–3.9
41–50	21	4.8	2.1	2.7	2.2–3.2
51–60	15	5.2	2.1	3.1	2.5–3.7
61–70	20	5.4	2.4	3.0	2.5–3.5
71–80	11	5.1	2.5	2.6	1.9–3.3
81–90	4	4.5	2	2.5	0–5.0
Symptoms					
Bell's pain[a]	90	5.2	2.2	3.0	2.8–3.2
Decreased Hearing	43	5.3	2.2	3.1	2.7–3.5
Other pain	37	5	2.1	2.9	2.5–3.3
Imbalance	33	5	2	3.0	2.6–3.4
Dysgeusia	31	5.1	2.1	3.0	2.6–3.4
Tinnitus	20	5	2	3.0	2.6–3.4
Vertigo	18	5.4	2.4	3.0	2.5–3.5
Facial numbness	17	4.8	1.4	3.4	2.8–4.0
Hyperacusis	12	4.8	2.1	2.7	1.9–3.5
Dry eye	5	5.4	3.4	2.0	1.1–2.9
Excessive tearing	5	5.4	3.2	2.2	1.6–2.8
Tongue numbness	5	4.8	2.4	2.4	1.0–3.8
Swallowing dysfunction	3	4.7	2.7	2.0	0–5.0
Diplopia	2	4.5	3	1.5	0–5.0
Signs					
Incomplete eye closure	86	5.4	2.3	3.1	2.9–3.3
Nystagmus	5	5	1.8	3.2	1.6–4.8
Vesicles	75	5.1	2.2	2.9	2.6–3.2
Audiometric status					
No audiometric deficit	36	5.1	1.9	3.2	2.8–3.6
Sensorineural hearing loss	51	5.1	2.0	3.1	2.7–3.5

HB indicates House-Brackmann.

[a]May, M. (1986a) Ch. 9. Differential diagnosis by history, physical findings, and laboratory results. Idiopathic Bell's palsy and herpes neuropathy. In: *The Facial Nerve*. New York: Thieme. p.197.

Eighty-nine patients had undergone audiometry, and of these, 51 patients had an asymmetrical sensorineural hearing loss associated with HZO. Table 1 shows the various treatment regimes that the patients received. When the 38 patients whose audiometry tests showed no hearing loss were compared with the 51 with diagnosed sensorineural hearing loss, the amount of improvement at 1 year was not significantly different ($p = 0.57$).

The facial function scores at presentation are shown in Table 1, along with the facial function at 12 months. The mean HB score of all patients at presentation was 5.12 HB grade units, and that at 1 year was 2.16 HB grade units. Thus, the mean improvement at 1 year after the onset of HZO in this patient cohort was 2.96 or close to 3 HB units.

Demographics, symptoms, signs, audiology, and treatment outcomes were examined to determine any deviation from the overall improvement of 3 HB units. There was no significant difference ($p = 0.58$) in the outcome for the 29 male subjects (mean improvement, 2.86 HB units; SD, 1.30) and the 70 female subjects (mean improvement, 3.0 HB units; SD, 1.06). Similarly, there was no significant difference ($p = 0.90$) in the mean improvement for the 58 patients with right-sided paralysis (mean improvement,

2.95 HB grade units; SD, 1.05) when compared with the 43 patients with left-sided paralysis (mean improvement, 2.98 HB grade units; standard deviation [SD], 1.22).

Table 1 shows the relationship between age and severity of onset. There was no significant association between the age at onset and the severity of facial nerve dysfunction at presentation ($p = 0.82$). Although the 2 smallest amounts of improvement for any age group were for patients in their seventh and eighth decades, the linear function relating increasing age decade with decreasing HB grade improvement was not significant ($p = 0.07$).

The relationship between severity of facial paralysis at onset and amount of improvement was analyzed. There was a significant difference in the recovery gradient between those who had more severe facial dysfunction at onset compared with those with less severe initial dysfunction ($p = 0.03$). Whereas patients presenting with HB grades V and VI tended to have an HB grade II or III recovery, patients presenting with HB grades III and IV dysfunction usually achieved an HB grade I or II recovery (Fig. 1).

Stepwise multiple regression was conducted using the improvement in terms of HB grade points as the dependent measure and the signs and symptoms listed in Table 1 as predictor variables. The regression result was significant ($F_{2,93} = 12.25$, $p < 0.01$), with a combination of the presence of 2 predictor variables, incomplete eye closure and dry eye, being associated with a smaller amount of improvement and accounting for 19% of the variance in improvement scores. The way in which the presence of these eye attributes combined to affect improvement at 1 year is shown in Table 2.

The pharmacologic treatment modalities that patients underwent and their associated outcomes are shown in Table 3. The amount of change in HB grade units was examined by planned contrasts conducted within analysis of variance. These contrasts examined the outcomes associated with a single drug used early or late, as well as for the drug combination used early or late. Finally, groups given a single drug treatment and those receiving the drug combination were compared with those who received no drug treatment and with each other. Whereas outcome after receiving a single-drug treatment was not significantly different from no treatment ($p = 0.93$) in terms of HB grade improvement at 1 year, the combination of antivirals and steroids was associated with significantly better outcome than no treatment ($p = 0.046$) and was better than the single-drug regimes ($p = 0.019$). Furthermore, when the combination of antivirals and steroids was examined for outcome depending on whether drug administration was early or late outcome associated with the later use of steroids was significantly better ($p = 0.005$). The time of antiviral administration did not significantly alter the drug combination effect ($p = 0.62$).

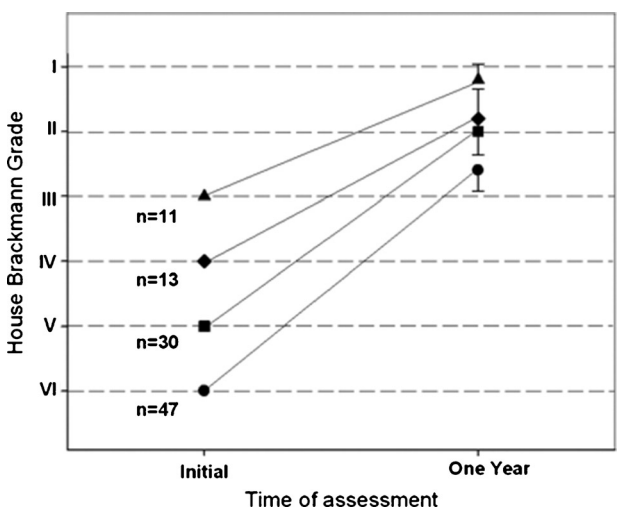

FIG. 1. Outcome in House-Brackmann grade units for the initial grade subgroups at 1 year after HZO. The number of patients in each grade subgroup at initial assessment is shown on the left of the figure. The error bars show the limits of the 95% confidence interval for the grade subgroup mean values at 1-year assessment.

TABLE 2. *Mean House-Brackmann grades at onset and the outcome at 1 year for subgroups experiencing incomplete eye closure and/or dry eye*

Incomplete eye closure	Dry eye	n	HB at onset	HB at 1 yr	HB diff	(95% CI)
No	No	15	3.3	1.3	2.0	1.7–2.3
Yes	No	81	5.4	2.2	3.2	3.0–3.4
Yes	Yes	5	5.4	3.4	2.0	1.1–2.9

CI indicates confidence interval; Diff, difference; HB, House Brackmann.

DISCUSSION

Patients with HZO in this study experienced a global improvement of 3 HB grades over 1 year. Fifty-nine percent of the patients achieved a satisfactory recovery (HB grade I or II), and this is similar to the 60% value previously published by Shaitkin et al. (11). The initial severity of HZO for the individual patient was related to both the amount of subsequent improvement and the final HB grade. Although the most affected patients improved more, at 1 year, they had still not reached the level of those who were less affected at onset. Although there was less gain in HB grade points made by patients in their seventh and eighth decades, patient age as an overall trend was not significantly related to amount of improvement.

No individual symptom or pattern of onset of symptoms affected recovery; however, the presence of diplopia or swallowing difficulty suggested a trend toward worse outcome. Diplopia and swallowing disorder are uncommon symptoms, and low numbers of patients with these symptoms in this study precluded statistical analysis. This poor prognosis, however, does suggest the possibility of a more widespread herpetic polyneuropathy and brainstem involvement by the zoster virus, although this is yet to be confirmed by specific magnetic resonance imaging during the acute illness. Similarly, patients with incomplete eye closure and a dry eye, indicating secretomotor involvement in the inflammatory process involving the greater

TABLE 3. *Mean House-Brackmann (HB) grades at onset and the outcome at 1 year for subgroups experiencing different pharmacologic treatment*

Pharmacologic treatment	No. of treated/101	Mean HB at onset	Mean HB at 1 yr	Mean difference	95% CI for the difference
No treatment	15	5.2	2.6	2.6	2.0–3.2
Antiviral only, given early	7	5.1	3.1	2.0	1.2–2.8
Antiviral only, given late	10	5.0	2.0	3.0	2.1–3.9
Steroids only, given early	5	4.8	2	2.8	1.8–3.8
Steroids only, given late	14	5.2	2.5	2.7	2.4–3.0
Antivirals early + steroids early	22	5.1	2.2	3.0	2.5–3.5
Antivirals early + steroids late	4	5.0	1.3	3.8	0.8–5.0
Antivirals late + steroids early	6	5.0	2.5	2.5	1.6–3.4
Antivirals late + steroids late	18	5.2	1.3	3.8	3.3–4.3
Total	101	5.1	2.2	3.0	2.8–3.2

Early ≤5 days after onset, late ≥5 days after onset.

superficial petrosal nerve, represent a more severely affected group with a poorer prognosis.

Although sensorineural hearing loss and vestibular disturbance are common in HZO (10), and have been linked previously to a poor facial nerve prognosis (18), data from this study have not supported this observation. We found the prognosis of HZO not to be significantly related to cochleovestibular symptoms.

Patients with HZO are commonly treated with prednisone and antiviral medications. Uscategui et al. (7) reviewed randomized controlled trials in which antiviral agents alone or in combination with other therapies were given in HZO. They found 1 prospective study using steroids and antiviral drugs in a cohort of 15 people recorded in the literature. Following a reanalysis, they concluded that antiviral agents had no beneficial effect on outcomes in HZO. However, there are numerous recommendations regarding the use of steroids and antivirals in the literature (7,8,14), none of which have been tested by randomized placebo-controlled trials. In the current retrospective study, those who received a combination of both antivirals and steroids recovered significantly more than those who had only one or no pharmacologic treatment. Furthermore, within the group who received the drug combination, those who received the steroid component after 5 days did significantly better than those who received it at less than 5 days after onset. This finding is consistent with previous reports of better outcome associated with the use of the drug combination for the treatment of HZO (7). One possible explanation for this finding is that the anti-inflammatory action of the steroids needs to be present at the time of greatest inflammation for maximum effectiveness. Also, prolonged administration of prednisone and an antiviral may prevent late degeneration of the facial nerve. A drug trial that compares single with combined regimes and varies the time of administration of the drugs is needed in the future.

CONCLUSION

The most significant factor found to influence outcome after HZO was the severity of facial impairment at presentation as measured using the HB grading system. All patients with HZO improved to some degree, and the mean improvement was 3 HB grades. Patients with HB grades V and VI presentations usually achieved an HB II or III recovery, whereas those with HB III or IV at presentation achieved HB grade I or II recovery. Although treatment regimens for HZO remain untested in prospective, randomized, placebo-controlled trials, the findings of this retrospective study showed that the combination of an antiviral with a steroid, with the steroid given after 5 days, was the most effective pharmacologic management.

REFERENCES

1. Crabtree J. Herpes zoster oticus. *Laryngoscope* 1968;78:1853–78.
2. Robillard R, Hilsinger RJ, Adour K. Ramsay Hunt facial paralysis: clinical analyses of 185 patients. *Otolaryngol Head Neck Surg* 1986; 95:292–7.
3. Engstrom M, Berg T, Stjemquist-Desatnik A, et al. Prednisolone and valaciclovir in Bell's palsy: a randomised double-blind, placebo controlled, multicentre trial. *Lancet Neurology* 2008;7: 993–1000.
4. Sullivan FM, Swan IRC, Donnan PT, et al. Early treatment with prednisolone or acyclovir in acute Bells Palsy. *N Engl J Med* 2007; 357:1598–607.
5. Gnann JW Jr, Whitley RJ. Clinical practice. Herpes zoster. *N Engl J Med* 2002;347:340–6.
6. Sweeney C, Gilden D. Ramsay Hunt syndrome. *J Neurol Neurosurg Psychiatry* 2001;71:149–54.
7. Uscategui T, Doree C, Chamberlain I, Burton M. Antiviral therapy for Ramsay Hunt syndrome (Herpes zoster oticus with facial palsy) in adults. *Cochrane Database Syst Rev* 2008;(4): CD006851.
8. Uri N, Meyer W, Greenberg E, Kitzes-Cohen R. Herpes zoster oticus: treatment with acyclovir. *Ann Otol Rhinol Laryngol* 1992;101: 161–2.
9. Furuta Y, Ohtani F, Fukuda S, Inuyama Y, Nagashima K. Reactivation of Varicella-zoster virus in delayed facial palsy after dental treatment and oro-facial surgery. *J Med Virol* 2000;62: 42–5.
10. Schreiber BE, Agrup C, Haskard DO, Luxon LM. Sudden sensorineural hearing loss. *Lancet* 2010;375:1203–11.
11. Schaitkin B, May M, Podvinec M, Ulrich J, Pietersen E, Klein S. Ideopathic (Bell's) palsy, herpes zoster cephalicus and other facial nerve disorders of viral origin. In: May M, Shaitkin B, eds. *The Facial Nerve*. 2nd ed. New York: Thieme, 2000:330–1.
12. Donahue JG, Choo PW, Manson JE, Platt R. The incidence of herpes zoster. *Arch Intern Med* 1995;155:1605–9.

13. Ragozzino MW, Melton LJ III, Kurland LT. Population-based study of herpes zoster and its sequelae. *Medicine* 1982;61:310–6, 344.
14. Murakami S, Hato N, Horiuchi J, Honda N, Gyo K, Yanagihara N. Treatment of Ramsay Hunt syndrome with acyclovir-prednisone: significance of early diagnosis and treatment. *Ann Neurol* 1997;41: 353–7.
15. Hato N, Kisaki H, Honda N, Gyo K, Murakami S, Yanagihara N. Ramsay Hunt syndrome in children. *Ann Neurol* 2000;48:254–6.
16. Gilden D, Cohrs R, Hayward A, Wellish M, Mahalingam R. Chronic varicella-zoster virus ganglionitis—a possible cause of post herpetic neuralgia. *J Neurovirol* 2003;9:404–7.
17. Choo PW, Galil K, Donahue JG, Walker AM, Spiegelman D, Platt R. Risk factors for postherpetic neuralgia. *Arch Intern Med* 1997;157: 1217–24.
18. Yeo SW, Lee DH, Jun BC, Changa KH, Park YS. Analysis of prognostic factors in Bell's palsy and Ramsay Hunt syndrome. *Auris Nasus Larynx* 2007;34:159–64.
19. House JW, Brackmann DE. Facial nerve grading system. *Otolaryngol, Head Neck Surg* 1985;93:146–7.
20. May M. *The Facial Nerve*. New York: Thieme, 1986:247.
21. Lo YL, Fook-Chong S, Leoh TH, Dan YF, Gran HY, Chan LL. High-resolution ultrasound in the evaluation and prognosis of Bell's palsy. *Europ J Neurol* 2010;17:885–9.