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Steroids for Acute Vestibular Neuronitis—the Earlier the Treatment, the Better the Outcome?

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Objective: To present findings that suggest steroid treatment within 24 hours of onset of vestibular neuronitis results in better restitution of vestibular function than treatment between 25 and 72 hours.

Patients: Thirty-three consecutive patients (17 men, 16 women, mean age 57 yr, range 17–85 yr) with acute vestibular neuronitis and treated with steroids within 72 hours after symptom onset. Patients were divided into two groups depending on if they were treated within the first 24 hours or not.

Interventions: Oral prednisolone 50 mg/d for 5 days with tapering of doses for the next 5 days, or combined with initial intravenous betamethasone 8 mg the first 1 to 2 days if the patient was nauseous.

Main Outcome Measures: Proportion of patients with normal caloric test result (canal paresis value < 32%) at follow-up after 3 or 12 months.

Results: All 9 patients (100%) treated within 24 hours from onset of vestibular neuronitis had normal caloric test results at follow-up after 3 months, as compared with 14 of 24 (58%) of the patients treated between 25 and 72 hours ($p < 0,05$, Fisher's exact test).

Conclusions: The timing of steroid treatment of vestibular neuronitis may be of importance for subsequent vestibular restitution, and hence, for both time to recovery and late symptoms according to the literature. **Key Words:** Steroids—Vestibular function—Vestibular neuronitis.

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Vestibular neuronitis (VN) is caused by a sudden unilateral loss of peripheral vestibular function. It is, after benign paroxysmal vertigo, the second most common cause of peripheral vertigo (1). The aetiology of VN is not known. The most prevailing theory suggests that there is a reactivation of neurotropic viruses (herpes simplex virus type 1) in the vestibular ganglion which leads to an inflammation in the nerve with swelling, entrapment and loss of function (2–4). The evidence is only circumstantial and specific treatment of the supposed viral infection does not seem to have an effect (5,6).

The recovery after VN normally takes several weeks and sometimes months and is a combination of restitution of peripheral labyrinthine function and gradual central vestibular compensation (7). However, only one-third of

patients recover to such an extent that their caloric test becomes normal without treatment (8). At long-time follow-up, the findings of a more pronounced canal paresis in the caloric test and a pathologic head impulse test are related to patients having more symptoms of dizziness, fatigue, and anxiety (9,10).

Previous placebo-controlled, double-blinded studies have shown that early treatment of acute VN with high doses of glucocorticoids speeds up and improves the recovery of vestibular function (6,11–14), although some reports have not shown the same beneficial effects (15,16).

Similar to VN, acute idiopathic peripheral facial palsy, “Bell palsy” is a diagnosis with unknown aetiology. The most established theory of the pathophysiology is the same as for VN. Studies on Bell palsy (5,17,18) have shown that patients, who received treatment with glucocorticoids within 48 hours from onset, had a significantly higher complete recovery and less synkinesis score compared with no glucocorticoid treatment. The time to complete recovery was significantly shorter if treatment started within 48 hours after onset (17). One smaller study (18) reports better long-term recovery if treatment started within 24 hours from onset. The aim of this report is to present findings that suggest that the timing of treatment of vestibular neuronitis with glucocorticoids may affect long-time vestibular restitution.

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The authors disclose no conflicts of interest.

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METHODS

Since July 2004 glucocorticoid treatment has been offered to patients with acute VN who presented to the emergency department of the Skåne University Hospital, Lund, Sweden within 3 days after the onset of symptoms. The diagnosis of acute VN was based on a history of sudden onset of vertigo, without auditory or neurological symptoms. The clinical findings comprised of spontaneous contralesional horizontal-torsional nystagmus that did not change direction with gaze and increased without visual fixation and an ipsilesional pathologic head impulse test.

All the patients were seen by one of the authors (M.K.) at our secondary referral center from July 2004 to December 2005. The group comprised 33 patients (17 men and 16 women) with a mean age of 57 years (range 17–85 yr). Nine patients were treated within 24 hours of symptom onset and 24 patients were treated after 25 to 72 hours. The patient group is the same as presented previously by Karlberg and Magnusson in 2011 (19).

All patients had tests of bithermal, 30°C and 44°, water caloric tests, cervical vestibular evoked myogenic potentials, test of subjective visual horizontal and vertical, saccadic and smooth pursuit eye movements to confirm the diagnosis of acute VN. Follow-up caloric tests were performed 3 months later. If the caloric test was abnormal after 3 months (>32% canal paresis in Jongkees formula (20)), patients were readmitted for another caloric test 12 months after the onset of the acute VN.

The patients were treated with oral prednisolone 50 mg (equivalent to 50 mg prednisone) daily for 5 days, and then tapering by 10 mg/d for the next following 5 days. If the patient were nauseous and due to vomiting could not tolerate oral medication, intravenous betamethasone 8 mg (equivalent to 50 mg prednisone and 8 mg dexamethasone) was instead given once daily for 1 to 2 days. Patients were informed about the importance of vestibular exercises and instructed to initiate rehabilitation as soon as possible.

STATISTICAL ANALYSES

The proportion of patients with normal caloric test results at the follow-up in the group treated within 24 hours after onset of symptoms and in the group treated

within 25 to 72 hours were compared with Fisher’s exact test. A *p* value of <0.05 was considered to be statistically significant.

RESULTS

All 9 patients (100%) treated within 24 hours after the onset of acute VN had a normal caloric test result at the 3 month follow-up, as compared with 14 of 24 (58%) of the patients treated between 25 and 72 hours (*p* < 0,05) (Fig. 1). Eight of the 10 patients with abnormal caloric reaction (all in the group treated between 25 and 72 h) at the first follow-up were readmitted after 12 months (2 patients declined participation). A further two patients recovered their caloric reactivity thus leaving 6 out of 22 (27%) with an abnormal caloric reaction after 1 year.

DISCUSSION

The findings suggest that there is a critical time slot in which steroid treatment could be effective. Patients treated within 24 hours after symptom onset had a better restitution of vestibular function compared with the patients who received treatment after 25 to 72 hours. This is in line with results found when treating Bell’s palsy; better results if steroids are administered within 48 hours of onset (17) and may be even better within 24 hours (5). If the effects of steroids primarily are anti-inflammatory, and thus reduce swelling and oedema in the nerve, then it is logical that the effect has potential to be greater in Bell’s palsy due to the long and narrow facial bone canal. The bony canal of the superior vestibular nerve is longer and more narrow than that of the inferior vestibular nerve (4). Thus the superior vestibular nerve might be more liable to entrapment due to the inflammatory swelling of the nerve. This can explain why the superior vestibular nerve is almost always (97.7%) affected in patients with vestibular neuritis

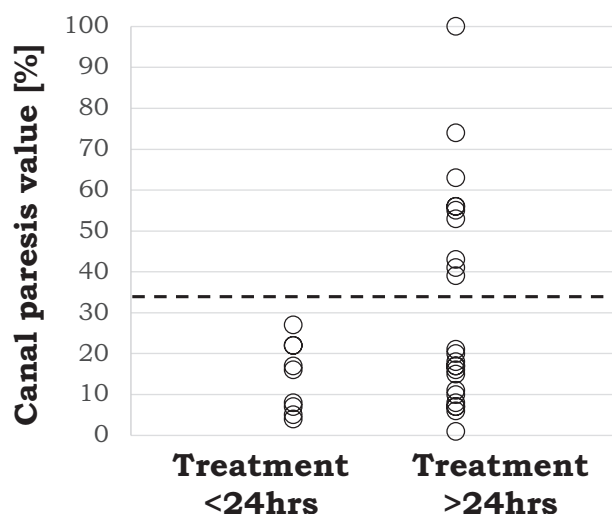


FIG. 1. Caloric responses at follow-up after 3 months for treatment within 24 hours versus after 24 hours. Dashed line indicates cutoff for normal caloric response. (20).

and the inferior vestibular nerve is less affected (58.1%), and this is why steroids could be as effective as treatment for VN as for Bell's palsy (21).

Based on currently available data there is not sufficient evidence for treatment with corticosteroids for acute VN (22), and there is no previous report for the effectiveness of early treatment. Many of these studies though have only few patients and too wide inclusion criteria. There are many limitations to this study; the groups of patients are very small and heterogeneous, but although being a small sample open study, the results do suggest that the time elapsed from onset to start of treatment might explain the results of studies in which steroid treatment was less beneficial. The present study may indicate that patients with early intervention versus late intervention might skew the outcome and thus, the interpretation of a possible benefit of steroids in VN. Another limitation is that the only outcome measured was the result of the caloric test, which does not necessarily correspond to symptomatology. Large-scale, adequately powered, randomized controlled trials are necessary to verify the effectiveness of treatment, and are currently being performed on our clinic (clinicaltrials.gov, ref. NCT02912182). In the absence of such studies, one might recommend the earliest possible start of steroid treatment, if such is considered.

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