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PROSPECTIVE STUDY OF EFFECT OF GLUCOCORTICOIDS IN THE TREATMENT OF VESTIBULAR NEURONITIS

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ABSTRACT

Objective: To study the efficacy of glucocorticoid treatment in acute vestibular neuronitis on recovery of vestibular function and the duration of hospital stay. **Study Design:** Prospective, comparison of consecutive case series with historic controls. **Setting:** Government Dharmapuri medical college hospital. **Patients:** Patients with acute vestibular neuronitis. One group treated with glucocorticoids within 3 days after the onset of symptoms[n=30] and the other group of untreated historic controls[n=40]. **Interventions:** Oral Prednisolone at a dose of 50 mg/day for the first 5 days followed by tapering of the doses for the next 5 days, alternatively with intravenous Betamethasone 8 mg on the first 1-2 days if nauseated. Main Outcome **Measures**: Extend of unilateral vesibular paresis (%) in the caloric test at presentation and at 12months of follow up. Duration of hospital stay (days). **Results**: The initial vestibular paresis value was same for the treatment group and the control group. A lower value (22.8% versus 47.2%) and greater improvement was seen in the treatment group on follow up. About 70% of the treatment group had a normal caloric test result compared to only 34% of the of the control group. The mean hospital stay of the treatment group was significantly shorter than that of the control group (1.8 versus 3.0 d, p=0.001).

KEYWORDS: Clinical medicine-Treatment outcome-vertigo-vestibular function tests.

Vestibular neuronitis (VN) or vestibular neuritis or sudden unilateral vestibular loss is characterized by sudden onset of prolonged vertigo, unsteadiness nausea and vomiting without other symptoms. (1) The clinical findings include horizontal – torsional spontaneous nystagmus with the quick phase beating towards the intact side, a tendency to fall toward the side of lesion, and a pathologic head impulse or head thrust test toward the side with lesion. (1)

A sudden unilateral loss of vestibular function is responsible for vestibular neuronitis. Most commonly, the functions of the organs that are innervated by superior vestibular nerve, the superior and lateral semicircular canals and the utricle, are affected but and the functions of the organs that are innervated by the inferior vestibular nerve, the posterior semicircular canals, and the saccule are spared.

The actual cause of VN is not known. Many researchers have shown that the reactivation of neurotrophic viruses such as Herpes simplex type 1, in the vestibular ganglion leads to an inflammation in the nerve causing swelling, entrapment and subsequent loss of function, but the evidence is circumstantial.

Central vestibular compensation occur over time, leading to restitution of vestibular function and the symptoms and findings will gradually disappear. However only one third of the patients will have normal caloric test results.

Previous open and recent placebo-controlled, double blind studies have shown that there is faster improvement and recovery of vestibular function with early treatment of VN with high dose of glucocorticoids. The aim of the present study was to report our results of glucocorticoid treatment of VN, compared with untreated historic controls, concerning the recovery of vestibular function and the duration of hospital stay.

MATERIALS AND METHODS

GLUCOCORTICOID TREATMENT GROUP

Since January 2017, the Department of Oto-Rhino-Laryngology, Government Dharmapuri Medical College Hospital, Dharmapuri has offered glucocorticoid treatment to patients with patients with VN who presented within 3 days after the onset of symptoms. Acute VN was diagnosed based on a history of sudden onset of vertigo in the absence of auditory symptoms or other neurologic symptoms. The clinical findings include contralesional spontaneous horizontal tortional - nystagmus that do not change thew direction with gaze

and increase when visual fixation is abolished and an ipsilesional pathologic head impulse test.

Patients were treated with oral prednisolone at dose of 50 mg/d for the first 5 days and then 40, 30, 20, 10, and 5 mg during the next 5 days. The patients who could not tolerate oral medication because of nausea or vomiting were treated with intravenous Betamethasone 8mg administered once daily during the first day or second day. Nausea was treated with Dimenhydrinate 100 mg rectally once or twice daily during the first 1-3 days. As soon as possible the patient was mobilized and instructed to perform vestibular rehabilitation exercise. (1)

The diagnosis of acute VN was confirmed by testing all the patients with bithermal, 30 and 44 degree celcius water caloric tests[15], cervical vestibular evoked myogenic potentials[17], test of subjective visual horizontal and vertical[14], saccadic and smooth pursuit eye movements, and pure tone audiometry. In a minority of patients, magnetic resonance imaging of brain was done.

Follow up with caloric test was performed 3 months after the onset of symptoms. The patients with an abnormal canal paresis value (higher than 22% in the formula of Jongkees et al) were subjected to second caloric test, conducted 12 months after the onset of VN.

All the patients of the treatment group were consecutively observed by
The group comprised 30 patients (16 men and 14 wemen) with a mean age of 57 year (range, 17-85). VN was on the right side in 13 patients and on the left side in 17patients.

CONTROL GROUPS

The first control group of patients with VN was observed at our department. Acute VN was diagnosed based on the same symptoms and findings as in the treated patient group, but head impulse test, cervical vestibular myogenic potential and tests of subjective visual horizontal and vertical were not used. Bithermal 30 and 44 degree celcius water caloric tests and test of voluntary eye movement s (saccades and smoothy pursuit eye movements) were performed during the acute stage and at follow up 12 months after onset of vestibular neuritis. The untreated control group consisted of 41 patients (18 men and 23 women) with a mean age of 41 years.VN was on the right side in 25 patients and on the left side in 16 patients.

STATISTICAL ANALYSES

The data of the treated group was compared with that of the control group s. $p<0.05$ was considered to be statistically significant.

RESULTS

RECOVERY OF VESTIBULAR FUNCTION

The Patients treated with prednisolone were significantly older than the control group. The canal paresis value was almost same for both groups at presentation. at follow up, the treated group showed significantly lower canal paresis value and significantly larger improvement than the control group. In the treatment group,70%(23/33) of the patients had normal caloric test result (canal paresis value <22%) at follow up. No major or unexpected adverse effect of corticosteroid treatment were recorded.

Hospital stay

The prednisolone – treated group had shorter duration of hospital stay compared to the control group.

DISCUSSION

The study shows that administration of glucocorticoids within 3 days after the onset of VN improves the long time recovery of vestibular function. Both the patients of treatment group and control group of 1981 to 1986 were consecutively and prospectively assessed. The statistics of vestibular paresis values of the control group of 1981 to 1986 both at presentation and follow-up were same as to of untreated and the placebo treated groups. This report show that treatment of VN with corticosteroids is of substantial benefit for the patients.

Our treatment regimen was 50mg of prednisolone daily for the fiorst five days followed by tapering doses in the next five days. Higher doses and longer duration with 100 mg of prednisolone was tried and the effect did not differ from to lower dose. There are many similarities between VN and Bell's palsy.reactivation of neurotropic virus has been suggested as a cause of both conditions. Herpes simplex virus DNA is detected by PCR in approximately two-thirds of both human vestibular and facial ganglia and the latency associated transcript is found in approximately 70% of human vestibular ganglia.

The bony canal of superior vestibular nerve is longer and narrower than that of inferior vestibular nerve and hence the superior vestibular nerve is more susceptible to inflammatory swelling of the nerve. Glucocorticoids reduce the inflammation in the nerve with less edema and less swelling of the nerve decreasing the entrapment and diminishing the nerve damage. An improved central vestibular compensation can be another mechanism of glucocorticoids treatment of VN and might explain the fast recovery and shorter hospital stay.

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