

CORTICOSTEROIDS EFFECT ON VESTIBULAR NEURITIS SYMPTOM RELIEF

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ABSTRACT

Background and objectives: Vestibular neuritis is the second most frequent cause of peripheral vertigo. Adding corticosteroids to symptomatic treatment regimen is effective in shortening the symptomatic period of vestibular neuritis but there are controversies on administration of corticosteroids for this condition. The aim of this study was to investigate the corticosteroids effect on vestibular neuritis symptom relief.

Methods: In this double blind placebo controlled clinical trial on 40 patients with vestibular neuritis who presented at Hamedan Sina Hospital neurology ward, all subjects signed an informed consent. The participants were randomly assigned to intervention (n=20) and control group (n=20). The intervention group received dimenhydrinate 50 mg bid as well as dexamethasone 6mg tid for three days. Controls received deminhidrinat 50 mg bid plus placebo for three days. At 24, 48, and 72 hour the result of Caloric test and patients symptom were recorded.

Results: The mean duration of symptom relief had significant difference in two groups. At 24 hour of initiation of therapy vertigo, nystagmus and nausea were relived in 5, 16, 17 of intervention group and in 1, 5, 2 of control group, respectively (P<0.001).

Conclusion: Corticosteroids are effective in shortening the symptomatic period of vestibular neuritis.

KEY WORDS: Vestibular neuritis, Vertigo, Corticosteroids.

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INTRODUCTION

The vestibular system is one of three sensory system subserving spatial orientation and posture. The other two are the visual systems and the somatosensory systems. Disruption of these systems coordinations result in a number of conditions with vertigo being the most significant.¹ Vestibular neuritis is the

second most frequent cause of pripheral vertigo (first cause is benign paroxysmal positional vertigo). Of all patients who present at clinics specializing in management of dizziness, 70% have vestibular neuritis.² It has an incidence of about 3.5 in 100000 population.³ The key signs for diagnosis are the acute onset of sustained rotatory vertigo, postural imbalance with Romberg's sign (i.e., falls, with eyes closed, toward the affected ear) horizontal spontaneous nystagmus (toward the unaffected ear) and nausea. Caloric testing (stimulation of the ear with warm or cold water) is hypo-responsive or non-responsive.

It was believed that an inflammatory processor labyrinth ischemia cause the condition. More recently viral pathogens has been sought. The condition is most prevalent in young and middle aged which hinders social activities and increases the management costs.⁴⁻⁶ There has been reports of corticosteroid effect on the condition^{2,3,7} but its wide spread use as therapeutic choice to shorten the course of

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symptomatic disease is controversial. This study aims to investigate corticosteroids (dexamethasone) effects on vestibular neuritis symptomatic relief.

PATIENTS AND METHODS

This study is a double blind clinical trial. All patients participating in the trial signed an informed consent. Most of the cases presented at Hamedan Sina Hospital, neurology ward in 2000-2002 with vertigo. Other etiologies of true vertigo have been ruled out. All patients were referred for ENT consultation. After audiometric and other lab tests as well as blood pressure measurement, examining cardiac status, cerebral MR imaging and Caloric test a diagnosis of vestibular neuritis was established. Those who didn't give consent and patients with hearing loss or tinnitus as well as patients with diabetes or immune deficiency, psychiatric conditions, a blood pressure over 180/110 mmHg, peptic ulcer, hypersensitivity to corticosteroids, and pregnant women were excluded. The power of our study was 80% and 20 patients were calculated to be sufficient for each group. The intervention group received dimenhydrinate 50 mg bid as well as dexamethasone 6 mg tid for 3 days. Controls received deminhydrinate 50 mg bid plus placebo for 3 days. All patients underwent a Caloric test with hypo-responsive or non-responsive result. (The maximal slow-phase velocity during Caloric irrigation with water at 30°C and 40°C should be less than three degrees per second on affected side and asymmetry between the two sides should be more than 25 percent as measured with the use of Jongkee's formula for vestibular paresis.^{8,9} Jongkee's formula is as follows:

$$\frac{[(R_{30^{\circ}C} + R_{44^{\circ}C}) - (L_{30^{\circ}C} + L_{44^{\circ}C})]}{(R_{30^{\circ}} + R_{44^{\circ}} + L_{30^{\circ}} + L_{44^{\circ}})} \times 100$$
 for example, $R_{30^{\circ}}$ is the mean peak show phase velocity during Caloric irrigation of the right laboratory with water at 30°C (R. denotes right and L. left and 30° or 44° indicate water temperature).

Patients were randomly assigned to intervention and control group (based on their hospitalization date) and the attending staff and the

patient were not aware of the assignment. At 24, 48 and 72 hour of initiation of drug therapy, result of Caloric test, and the sign and symptoms were recorded through filling the questionnaire. All patients were hospitalized for 1-3 days. They were discharged when they were able to maintain their balance, or when they had no problem in maintaining their balance with closed eyes. The Caloric test results at discharge were compared with the initial results. The patients had follow up by neurologists. The mean values of intervention and control group was compared using t-test for independent groups and subgroup comparison was conducted using one-way variance analysis. For analysis EPI 2000 was used.

RESULTS

The 40 participants (14 male, aged 15-55) had an equivalent distribution in intervention and control groups in terms of age and sex. The disappearance of vertigo in intervention group were reported by 5 (25%) at 24 hours, by 12 (60%) at 48 hours and by 3 (15%) at 48 hours of dexamethasone administration while in control group 1 (15%) reported relieving of vertigo at 24 hours and 18 (90%) reported having vertigo at 72 hours. More or less the same findings were observed for nausea and nystagmus. In treatment group at 24 hours of initiation of therapy 17 (85%) and 16 (80%) reported relief of nausea and nystagmus, respectively, while 5 (25%) and 2 (10%) of control group reported similar results.

The mean intervals till symptoms relief (vertigo, nausea, nystagmus) were significantly different between two groups (Table-I).

Table-I: Mean of time for relief of vestibular neuritis symptom in treatment and control group

	Treatment group	Control group	P value
vertigo (mean ±SD) (hour)	45.6±15.3	68.4±11.7	<0.001
nausea (mean ±SD) (hour)	28.8±12.5	54±20.4	<0.001
nystagmus (mean ±SD) (hour)	28.8±9.8	63.6±16.1	<0.001

The mean intervals of symptoms relief were not significantly different in various age groups of control subjects. In intervention group the mean intervals till nausea and nystagmus relief were not different among various age groups while the difference was significant for vertigo with 26-35 year-age group having a better response. No significant difference was observed among the two sexes in terms of symptom relief and response to drug therapy.

DISCUSSION

Our results suggest that adding corticosteroids to routine symptomatic treatment had a significant effect on recovery of vestibular neuritis. These findings are consistent with the results of a study by Katahara et al. In a study of corticosteroid effects on clinical signs and symptoms of vestibular neuritis, they reported a significant reduction of duration in which the patients had nystagmus and vertigo following the movement of body or head in treatment group who received corticosteroids in acute phase of the disease in comparison with controls who didn't received the corticosteroids treatment.¹⁰ Another study in Japan on 111 patients with vestibular neuritis showed that 34 patients who received corticosteroids as part of their treatment had significantly better recovery of vestibular function.¹¹

Some researchers believe that there is an increasing evidence showing that steroids adjust those neurotransmitters activities that compensate for vestibular system dysfunction.^{12,13} Parnes et al showed that prednisolone and hydrocortisone can be traced in endolymph after systemic administration.¹⁴

No study of the effect of steroids on vestibular neuritis in terms of possible variations in different sex and age group was found in medical literature, while our study showed that younger patients had a better response in comparison with older ones. How this can be

explained is beyond the scope of our study and should be investigated through further studies. Our study shows that dexamethasone is an effective agent in shortening the duration of symptoms in patients with vestibular neuritis.

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