The Effect of Intravenous Dexamethasone on the Nausea Accompanying Vestibular Neuritis: A Preliminary Study

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ABSTRACT

Purpose: We undertook a preliminary assessment of the efficacy of administering intravenous dexamethasone (DEX) for relieving the nausea and dizziness accompanying vestibular neuritis (VN).

Methods: Between November 2013 and October 2014, 26 patients with VN were prospectively enrolled in this study. The patients were randomly assigned to treatment with a combination of 20 mg/d of intravenous metoclopramide, 100 mg of oral dimenhydrinate, and 5 mg/d of intravenous DEX or 20 mg/d of intravenous metoclopramide, 100 mg of oral dimenhydrinate, and intravenous normal saline as a placebo therapy. Patients' subjective assessments of the severity of their nausea and dizziness were recorded using a visual analog scale on the day of admission and 2 days, 3 days, 1 month, and 3 months thereafter. Bedside examinations consisted of spontaneous nystagmus (SPN) assessment, the head shaking nystagmus test, and the head impulse test, which were performed at every follow-up visit.

Findings: The severity of nausea and dizziness was significantly reduced over time (both $P < 0.05$). However, there was no significant effect of DEX injection on the severity of nausea or dizziness ($P > 0.05$). The presence of SPN was solely associated with nausea (hazard ratio = 3.34; 95% CI, 1.85–6.02).

Implications: The administration of intravenous DEX did not relieve nausea or dizziness any better than a placebo treatment. However, further research is required to confirm whether there is a dose-dependent effect of DEX on the control of nausea or dizziness in VN. (*Clin Ther*. 2015;37:2536–2542)

Key words: dexamethasone, dizziness, nausea, vestibular neuritis.

INTRODUCTION

Nausea and/or vomiting and the acute onset of vertigo are the most common symptoms in patients with vestibular neuritis (VN).[1] In the acute stage, symptomatic therapy via intramuscular or intravenous drug administration is preferred over oral drug administration due to severe nausea and reduced gastric motility.[2] Dimenhydrinate is one of the drugs most commonly prescribed to suppress these symptoms in the acute phase.[3]

Although antihistamines have been the mainstream treatment for vestibular disorders, diverse antiemetics such as metoclopramide, promethazine, droperidol, and serotonin receptor antagonists (eg, ondansetron) are also frequently prescribed.[4] Metoclopramide has both antidopaminergic and prokinetic effects, and it is relatively inexpensive. However, extrapyramidal effects have been reported, and prolonged use should be avoided.[4] Administration of a serotonin receptor antagonist was reported to be more effective than metoclopramide in shortening hospital stays and reducing the time to first unassisted walking in VN, although the mechanism of action was not described.[5] The use of a serotonin antagonist has generally been restricted to postoperative nausea or chemotherapy-induced emesis (CIE), however, according to governmental insurance policy.

Corticosteroid therapy is another potential treatment option and was recently reported to be associated with earlier recovery after VN, although it may not influence long-term outcome and there is still insufficient evidence to support its use for VN according to a recent Cochrane review.[6–8] In light of their

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antiemetic action, corticosteroids also have a high therapeutic index for CIE and can be used as a single agent or in combination with a serotonin receptor antagonist.[9] In addition, the effect of dexamethasone (DEX) on reducing postoperative nausea and vomiting is well-known.[10] However, to the best of our knowledge, the role of corticosteroids in the control of nausea accompanied by VN has rarely been studied.

In this study, we hypothesized that an additional intravenous DEX injection in the acute phase of VN may result in the earlier resolution of nausea. To investigate this hypothesis, we undertook a preliminary assessment of the efficacy of additional DEX for relieving the nausea accompanies VN.

MATERIALS AND METHODS

Ethical Considerations

The Institutional Review Board of Eulji University Hospital approved this study (EMC-2013-09-0009-001). Written informed consent was obtained from all patients. The study was registered at http://cris.nih.go.kr (registration number: KCT0001079).

Patients

Between November 2013 and October 2014, patients who visited the dizziness clinic and emergency department at a university hospital with nausea and/or vomiting as well as the acute onset of vertigo and with a diagnosis of VN were screened. The inclusion criteria were (1) admission to the hospital and willingness to participate in the study, (2) acute onset of sustained vertigo, (3) unidirectional mixed horizontal-torsional spontaneous nystagmus (SPN) as determined by infrared video goggle examination, (4) a positive head impulse test (HIT) result, and (5) no additional neurologic signs or symptoms suggestive of central lesions.[11,12]

Patients who met any of the following exclusion criteria were not enrolled in the study: (1) uncontrolled diabetes mellitus and/or hypertension; (2) younger than 18 years of age; (3) poor general condition suggesting a potential inability to tolerate DEX, such as acute peptic ulcer, glaucoma, intestinal obstruction, pregnancy, immunocompromised state, or an allergy to DEX; (4) currently using steroids or a medical history of steroid use within the 3 months before the initial visit; and (5) a previously diagnosed psychogenic disorder.

Otoneurologic Examination

To detect SPN, patients sat in the upright position and were instructed to look straight ahead, and infrared goggles were used to evaluate the presence of SPN. To assess head-shaking nystagmus (HSN), the patient's head was tilted forward at 30 degrees, and then shaken 20 times over 10 sec at a speed of 2 Hz. An increased frequency of preexisting SPN was considered a positive result. For HIT, patients were asked to focus their gaze on the examiner's nose while their head was rotated 20 degrees to the right and left. Corrective saccades were considered a positive result.[13] Bithermal caloric tests were performed using warm (44°C) and cold (30°C) water and recorded by videonystagmography software (CHARTR VNG; ICS Medical, Schaumburg, Illinois).[13,14] Canal paresis was calculated using Jongkees' formula.

For the measurement of cervical vestibular evoked myogenic potentials (cVEMPs), the patient's head was tilted to the opposite side in the supine position with an active electrode on the midpoint of the sternocleidomastoid muscle, a reference electrode on the sternum, and a ground electrode placed centrally on the forehead. To calculate cVEMP response, Navigator Pro software (Bio-logic Systems Corp, Mundelein, Illinois) was used and VEMP amplitudes were measured by playing a 500-Hz tone burst at 95 dB nHL through a headphone. The stimulation pattern used had a rise/fall time of 4 msec, and a plateau time of 2 msec. cVEMP asymmetry exceeding 30% or no cVEMP response was considered to be pathologic.

Treatment Protocols

All patients were hospitalized. Before enrollment, prerandomization with 40 sample numbers was performed using an Internet-based randomization service by an otolaryngologist who was not involved in the study. The allocation procedure was concealed from patients and clinicians. For patients in the study group, 5 mg/d of intravenous DEX for 2 days, 20 mg/d of intravenous metoclopramide for a maximum of 5 days, and 100 mg/d of oral dimenhydrinate during same period were administered. For the control group, 1 mL/d of intravenous normal saline for 2 days, 20 mg/d of intravenous metoclopramide for a maximum of 5 days, and 100 mg/per of oral dimenhydrinate were administered. All patients were instructed to perform a vestibular-ocular reflex