Vestıbular Evoked Myogenıc Potentıals ın Benıgn Paroxysmal Posıtıonal Vertıgo

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ABSTRACT

Objective: The aim of this study is to investigate the vestibular evoked myogenic potentials (VEMP) findings in posterior semicircular canal benign paroxysmal positional vertigo (BPPV) patients.

Methods: Twenty patients with posterior semicircular canal BPPV and 20 healthy control subjects were recruited to participate in this study. VEMP was performed at diagnosis and after treatment.

Results: All the volunteers in the control group and 15 patients in BPPV group showed a VEMP response in both ears. The P1, N1 latencies showed a significant difference between BPPV patient's affected ears and control subjects. There were no significant differences between the unaffected ears of BPPV patients and control subjects with respect to P1, N1 latencies. P1, N1 latencies showed no significant difference between affected and unaffected ears in BPPV patients. No significant difference could be found between the pre and post treatment values of P1, N1 latencies in BPPV patients.

Conclusions: Our findings showed significantly more abnormal VEMP results in the BPPV than in the control subjects.

Keywords: Benign paroxysmal positional vertigo, treatment, vestibular evoked myogenic potential

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INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is one of the most common vestibular disorders (1). BPPV is idiopathic in most cases and characterized by episodes of vertigo (2). Head motion causes BPPV and results in temporary vertigo due to free-floating debris in the semicircular canal (SCC) or attachment of otolithic debris to the cupula (3). Although BPPV can affect each of the three SCC (posterior, horizontal, superior), posterior SCC is the most frequently affected (4).

BPPV, which is one of the commonly seen vestibular disorders, occurs as temporary attacks of vertigo caused by head movements (1, 2). These attacks result from debris floating freely in the semicircular canal (SCC) or adhering to the cupula (3). Although BPPV affects posterior, horizontal and superior SCC, the most affected canal is posterior (4).

Various pathophysiological mechanisms including canalithiasis or cupulolithiasis have been proposed to explain BPPV (5). In canalithiasis, debris floats freely between the ampulla and common crus of posterior SCC (6). Canalithiasis can occur in any SCC (7). All of the features of typical nystagmus provoked by Dix Halpike maneuver in BPPV patients may be explained by canalithiasis (8). The nystagmus usually develops with a brief latency of several seconds, although cases with greater than 10-second latency have been described. It resolves within 1 minute. The nystagmus diminishes with repeated maneuvers (9).

In cupulolithiasis, the particles are adherent to the cupula (10). According to the cupulolithiasis theory, a cupular deposit would induce a gravitational effect on the crista (11). In cupulolithiatic type of posterior SCC BPPV, nystagmus has shorter latency and longer time constant than canalithiasis (12).

Vestibular evoked myogenic potential (VEMP) has been described as a useful clinical test of the vestibular system (5). Cervical VEMP is short-latency myogenic potentials evoked by high-level acoustic stimuli (14).-The VEMP test measures a vestibulo-spinal reflex mediated

through the saccule and the inferior vestibular nerve (6, 7). Clinical and neurophysiological studies have suggested that VEMP originates from saccular afferents (13, 17). The posterior SCC and the saccule are innervated by inferior vestibular nerve (18-21). Gacek (22) has suggested that BPPV is due to recurrent neuritis of the inferior vestibular nerve. In BPPV, the degenerative process can also affect the macula of the saccule (8). Patients with BPPV may exhibit abnormalities in VEMP (9).

Several researchers have published the results of VEMP in BPPV patients (8, 9, 10-15). Some of these studies have investigated the results of VEMP in BPPV and another vestibular disorders (8, 10), while other studies (9, 12) have investigated the results of VEMP of BPPV involvement in different canal type. In two of these studies (11, 15) VEMP responses of patients only with unilateral posterior semicircular canal BPPV have been investigated. In one study (11), 15 patients with BPPV had canalithiasis and 4 BPPV patients had cupulolithiasis. Another study (15) has not specified canalithiasis or cupulolithiasis of BPPV patients. The aim of this study is to investigate the cervical VEMP results in patients with canalithiasis form of idiopathic BPPV affecting the posterior canal unilaterally, and examine the results in comparison with that of healthy individuals

DISCUSSION

The aim of this study is to investigate the VEMP findings in idiopathic unilateral BPPV patients with posterior semicircular canalithiasis form,and compare the results with that of healthy individuals. We only evaluated VEMP P1 and N1 latencies in this study. Amplitude could be easily affected by several factors such as muscle activity and patient's position. Therefore, we did not evaluate VEMP amplitude.

All of the control subjects showed VEMP responses in this study. Also the VEMP responses were obtained in all unaffected ears in patients with BPPV. However, 5 patients (25%) in BPPV group showed no response to VEMP in the affected ears. Our results showed significantly more abnormal VEMP results in the BPPV patients than in the control subjects. This result is consistent with several previous surveys findings (11, 12, 15). Researchers (11, 15) reported that abnormal VEMP recordings were statistically higher in BPPV patients than in controls. Aguirre et al. (12) found a lack of VEMP response in 52 % of the ears with BPPV.

BPPV is a disease attributed to the appearance of lithiasic material from the otolithic maculae of the saccule and the utricle in the SCC (17). Various pathophysiological mechanisms including canalithiasis or cupulolithiasis have been proposed to explain BPPV (18). In canalithiasis, debris floats freely between the ampulla and common crus of posterior SCC (19). All of the features of typical nystagmus provoked by Dix Halpike maneuver in BPPV patients may be explained by canalithiasis (20). The nystagmus usually develops with a brief latency of several seconds, although cases with greater than 10-second latency have been described. It resolves within 1 minute. The nystagmus diminishes with repeated maneuvers (16).

In cupulolithiasis, the particles are adherent to the cupula (21). In cupulolithiatic type of posterior SCC BPPV, nystagmus has shorter latency and longer time constant than canalithiasis (22). Regarding physiopathology of BPPV, the role of the utricle is widely accepted because of its anatomical proximity with ampulla of the posterior SCC (23).

Welgampola et al. (23) pointed to the possibility of saccular involvement in BPPV. These degenerative processes, which affect the macula of the utricle, might also affect the macula of the saccule, resulting in abnormal VEMP (14).

The VEMP test evaluates the pathway from the saccule to the inferior vestibular nerve, vestibular nucleus, lateral vestibular nucleus, lateral vestibulospinal tract, and sternocleidomastoid muscle (8, 14).

The absence of VEMP, sign of sacculo-collic reflex pathway lesion, has been correlated to a degeneration of the saccular macula (9). The reason for this appears to be that the absence of VEMP of the affected ears is due to involvement of the inferior vestibular nerve or involvement of the structures that it innervates (24). The posterior SCC and the saccule are innervated by inferior vestibular nerve (8, 10-12). The absence of VEMP in BPPV patient's affected ears means that there was more extensive damage that affects the macula of the saccule (9). Gacek (25) has suggested that BPPV is due to recurrent neuritis of the inferior vestibular nerve.

We found that the mean P1 and N1 latencies showed statistically significant difference between BPPV patients' affected ears and that of control subjects ($P < .05$). No statistically significant differences were found between the unaffected ears of BPPV patients and control subjects with respect to the VEMP mean P1 and N1 wave latencies ($P > .05$). In those patients in whom responses were obtained in both ears, there were significant differences in terms of response to P1 and N1 latencies between the affected ear and the unaffected ear ($P < .05$). These results are similar to those obtained by Akkuzu et al. (8). They found delayed latencies in some of their BPPV patients. Yang et al. (9) also found that BPPV patients showed prolonged P1 and N2 latencies compared to the control group, in agreement with our results. Contrary to our findings, Korres et al. (11) reported that P1 and N1 latencies are not statistically different between BPPV patients and controls. In addition, Aguirre et al. (12) found no statistically significant difference between affected and unaffected ear with respect to VEMP p13 and n23 wave latencies in BPPV patients.

Abnormalities of VEMP (prolonged latencies and absent responses), indicate that lower brainstem and vestibulospinal tract are affected in BPPV patients (13). The prolongation of P1 or N1 latency in affected ears of patients with BPPV could be correlated to the degeneration of vestibular ganglion cells (15). Gacek (25) examined temporal bones in BPPV patients and

suggested that there is a significant decrease in the superior and inferior ganglion vestibular cells. The existence of degeneration of the macula and of the inferior vestibular nerve ganglion cells has been documented in BPPV patients (25). We found that the mean P1 and N1 latencies were statistically significant delayed in BPPV patients' affected ears ($P < .05$).

We could not find any significant difference between pre and post treatment values of P1 and N1 latencies in BPPV patients. This result is similar to those obtained by Yang et al. (9). This finding implies irreversible neuronal degenerative changes (9).

CONCLUSION

Our findings showed significantly more abnormal VEMP results in the BPPV than in the control subjects. Also, we found delayed latencies in BPPV patients' affected ears. The VEMP test provides valuable information regarding the inferior vestibular nerve, lower brainstem and vestibulospinal tract. Therefore, VEMP can be used to aid in the diagnosis and follow-up of patients with BPPV.

AUTHORS' NOTE

There is no conflict of interest.

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Table 1: Comparison of VEMP latencies in the affected ear of BPPV patients and in controls.

	Control (mean±SD)	Affected ear (mean±SD)	p
P1 latency	13.90±.74	14.62±.82	.003
N1 latency	20.32±1.11	21.53±1.74	.022

VEMP; vestibular evoked myogenic potentials, BPPV; benign paroxysmal positional vertigo

Table 2: Comparison of VEMP latencies in the unaffected ear of BPPV patients and in controls.

	Control (mean±SD)	Unaffected ear (mean±SD)	p
p1 latency	13.90±.74	13.80±.55	.597
N1 latency	20.32±1.11	20.33±.1.23	.980

VEMP; vestibular evoked myogenic potentials, BPPV; benign paroxysmal positional vertigo

Table 3: Comparison of VEMP latencies in the affected and unaffected ears of BPPV patients.

	Affected ear (mean±SD)	Unaffected ear (mean±SD)	p
P1 latency	14.62±.82	13.80±.55	.003
N1 latency	21.53±1.74	20.33±.1.23	.023

VEMP; vestibular evoked myogenic potentials, BPPV; benign paroxysmal positional vertigo

Table 4: Comparison of VEMP latencies pre and post treatment in BPPV patients

	Pre treatment median (min-max)	Post treatment median (min-max)	p
P1 latency	13.98 (12.27-15.95)	14.50 (12.83-15.43)	.566
N1 latency	20.53 (18.16-24.80)	21.78 (18.45-24.80)	.572

VEMP; vestibular evoked myogenic potentials, BPPV; benign paroxysmal positional vertigo