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Vestibular Abnormalities in Pseudotumor Cerebri

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OBJECTIVE: To identify the vestibular function in patients diagnosed with pseudotumor cerebri and determine whether pseudotumor cerebri is accompanied by vestibular system malfunction.

METHODS: Fifteen patients (12 women and 3 men; age range, 23-36 years [mean, 31.5 years]) with benign intracranial hypertension and true vertiginous attacks were referred to the vertigo clinic of Cairo University in Egypt for further evaluation and management. A complete neuro-otological history was taken from all subjects, with special emphasis on vertigo history. Subjects with a known history of peripheral or central vestibular system involvement and those with present predisposing factors for vestibular system involvement were excluded from this study. All patients underwent basic audiologic evaluation, and vestibular system testing by videonystagmography.

RESULTS: The entire study group had normal hearing threshold levels with pure tone average 20 dB and normal middle ear function, evidenced by type A tympanogram with preserved acoustic reflexes. Four subjects (26.6%) had normal videonystagmography test battery. Saccadic tracking was normal in the whole study group. No spontaneous or gaze-evoked nystagmus were detected in the 15 subjects. Three subjects (20%) showed reduced pursuit and optokinetic gain with normal symmetry. The same 3 subjects showed normal caloric responses with abnormal fixation index. Dix-Hallpike tests elicited robust positioning nystagmus in 2 subjects (13.4%). Unilateral canal weakness and directional preponderance were found in 5 subjects (33.4%) and 1 subject had bilateral canal weakness (6.6%).

CONCLUSIONS: These findings confirm that vestibular abnormalities are common in patients with pseudotumor cerebri.

Idiopathic intracranial hypertension and benign intracranial hypertension are considered synonymous for the following: symptoms and signs restricted to those of elevated intracranial pressure; normal findings on neuroimaging study, excluding nonspecific findings of raised intracranial pressure; and increased cerebrospinal fluid (CSF) pressure with a normal composition.[1,2] Current theories concerning the pathogenesis of elevated intracranial pressure include increased resistance to CSF outflow at the arachnoid granulations that line the dural venous sinuses and through which CSF reabsorption is thought to occur by bulk flow[3]. Studies of American-based populations have estimated that the incidence of pseudotumor cerebri ranges from 0.9-1.0 per 100,000 in the general population. This incidence increases to 1.6-3.5 per 100,000 in women, and 7.9-19.0 per 100,000 in women who are overweight. The incidence of pseudotumor cerebri is variable from country to country, and its occurrence varies according to the incidence of obesity in the respective region[3,4]. A study performed in Libya demonstrated an incidence of 2.2 per 100,000 in general population, 4.3 per 100,000 in women, and 7.9 per 100,000 in women who were overweight.[5]

Dizziness is a common complain among patients diagnosed with pseudotumor cerebri.[6,7] Dizziness is a generic designation, which includes all circumstances involving a feeling of disturbance of the relations between the patient and space.[8] Often it is very difficult to distinguish between true vertigo and dizziness. Vertigo is the consequence of any type of vestibular lesion, defined by an illusionary sensation of motion: spinning, whirling, or linear.[9]

The objective of this study is to evaluate vestibular function in patients diagnosed with pseudotumor cerebri.

MATERIALS AND METHODS

This study was conducted during a 2-year period (2003-2005) in the hearing and balance disorders unit of Cairo University. Fifteen patients (12 women and 3 men) with age range 23 to 36 years (mean, 31.5 years) diagnosed with benign intracranial hypertension and complaints of true vertiginous attacks were included. A complete neuro-otologic history was taken from all subjects. Subjects with a known past history of peripheral or central vestibular system involvement and those with a present history of predisposing factors for vestibular system involvement, such as upper respiratory tract infection, head trauma, or chronic ear infection, were excluded from this study to eliminate any factor that might affect the vestibular system other than pseudotumor cerebri. All subjects underwent pure-tone audiometry, speech audiometry, and tympanometry (Audiometer Siemens SD 50 and Impedance Audiometer Interacoustic AZ26, Siemens Corp, Siemens, Germany). Vestibular testing was performed using computed videonystagmography (VNG; Hortmann Video CNG Analyser, GN Otometrics, Taastrup, Denmark). Saccadic eye movements, smooth pursuit, optokinetic nystagmus, and any spontaneous and positional nystagmus were recorded. Bithermal caloric testing was also performed with suppression fixation. Saccades were tested for accuracy, velocity, and latency. Smooth-pursuit and optokinetic tracking were analyzed for symmetry and gain.

The gaze test was performed by recording eye movements as the patient gazes 30° in 4 directions (right, left, up, and down), holding each gaze position for at least 20 seconds. Spontaneous nystagmus was examined while the patient was in the sitting position and his/her eyes were in the neutral position. Positional and positioning (Dix-Hallpike) tests were performed to determine if the vestibular system responded normally and symmetrically to changes in head position. Findings for spontaneous and positional nystagmus were considered abnormal if the slow phase velocity (SPV) was > 5°/sec. Air-caloric irrigation was performed to evaluate the response of the lateral semicircular canals. The produced caloric nystagmus was analyzed for presence and symmetry. Visual fixation was evaluated during the period of nystagmus response to caloric stimulation. Bilateral canal weakness was confirmed by ice water caloric test. Normal values are shown in Table 1.

Table 1. Normal values used in vestibular testing*

Saccades	Mean latency 180 (± 25) Mean accuracy ≥75% Mean velocity 533 ± 88)
Smooth Pursuit	Symmetry < 15%, gain > 0.8
Optokinetic	Symmetry < 15%, gain > 0.8
Unilateral canal weakness	Right - left ear asymmetry < 22%
Bilateral canal weakness	Sum of peak warm and peak cool response < 12deg/sec
Directional preponderance	Right beating-left beating asymmetry < 30%
Fixation suppression index	≤ 60%

*Vestibular lab of S. Fakeeh for age-matched group.

RESULTS

A total of 15 subjects diagnosed with benign intracranial hypertension were examined in this study. The entire study group had normal hearing threshold levels with pure tone average 20 dB and normal middle ear function, as proved by the presence of type A tympanograms and preserved acoustic reflexes.

Four subjects (26.6%) had normal VNG test battery. Saccadic tracking was normal in the whole study group. No spontaneous or gaze-evoked nystagmus was detected in all subjects. Three subjects (20%) showed reduced pursuit and optokinetic gain at 40°/sec and 60°/sec, with normal symmetry and abnormal fixation index (mean value 41.7 ± 2.9). The same 3 subjects showed normal caloric responses. Dix-Hallpike test elicited robust positioning nystagmus in 2 subjects (13.4%). The nystagmus was horizontal and ageotropic, with longer duration, almost no latency, and absence of fatigability.

Unilateral canal weakness and directional preponderance were found in 5 subjects (33.4%). Only 1 subject (6.6%) had bilateral canal weakness, with sum of peak warm and peak cool response 7 degrees/sec. The abnormal vestibular test findings are shown in Table 2.

DISCUSSION

Although dizziness is not a rare complaint in patients with benign intracranial hypertension, the effect of pseudotumor cerebri on the peripheral and central

Table 2. Abnormal vestibular findings in the study group.

Smooth pursuit and optokinetic nystagmus	**Pursuit gain**	**Optokinetic gain**
Subject 1	0.6	0.5
Subject 2	0.7	0.6
Subject 3	0.6	0.5
Caloric responses	**Right-left ear asymmetry**	**Right beating-left beating asymmetry**
Subject 1	30%	37%
Subject 2	30%	40%
Subject 3	34%	43%
Subject 4	32%	35%
Subject 5	36%	43%

vestibular system is not well established in the literature.[6,7] Four subjects in our study had no abnormalities in the VNG test battery, but this is not absolutely indicative of normal vestibular function, as the VNG tests only a limited portion of the vestibular system (ie, lateral canals) at a small range of frequencies.[10] Unfortunately, further vestibular evaluation by rotatory chair and/or posturography were not available in our facility.

Three subjects had central vestibular system involvement without apparent peripheral vestibular system involvement, as proved by the abnormal findings in the pursuit and optokinetic tracking test, as well as the fixation suppression. Defective pursuit is a particularly sensitive sign of central nervous system dysfunction.[11] The increase in the intracranial hypertension can cause ischemia of the brain stem, which might be the cause of central vestibular system dysfunction. Abnormal pursuit without saccadic abnormality supports the assumption that pursuit tracking is more sensitive in detecting central vestibular dysfunction.[12] The absence of spontaneous and gaze-evoked nystagmus may be explained by the slow increase in the intracranial hypertension, which allows for central compensation.

Five subjects showed unilateral canal weakness and 1 showed bilateral canal weakness documented by the bithermal caloric irrigation and the ice-water test. Vestibular paresis may be caused by pontine lesions affecting the intrapontine vestibular nerve fascicle or medial and lateral vestibular nucleus, but normal fixation suppression in those patients favors involvement of the intrapontine vestibular nerve fascicle (or more peripheral), as nuclear lesions usually show little or no inhibitory effect of visual fixation on caloric nystagmus. Increased intracranial hypertension can cause ischemia of the anterior inferior cerebellar artery, which supplies the region of intrapontine vestibular nerve. However, there were no other manifestations of this type of disorder.

Two subjects showed robust positioning nystagmus during the Dix-Hallpike maneuver. It is not uncommon to see benign paroxysmal positional vertigo in a patient that has ischemic involvement in the posterior circulation. This may be supported by the features of the produced positioning nystagmus, which are compatible with horizontal canal cupulolithiasis. Schuknect proposed that vascular involvement of the superior labyrinth might release otoliths from the degenerating utricular macula, which could then lodge on the posterior canal crista.[13]

No patient demonstrated any kind of reduced hearing sensitivity. This supports the theory that the vestibular portion of the inner ear is more sensitive to vascular ischemia than the cochlear one[14] or that the ischemic theory was not relevant in those cases.

CONCLUSION

Abnormal vestibular findings are common in patients with pseudotumor cerebri. Patients with true vertiginous attacks who match criteria for the diagnosis of pseudotumor cerebri, such as overweight women of child-bearing age, should be referred for complete neurologic evaluation.

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