Evaluation of Auditory and Vestibular Function in Patients with Idiopathic Intracranial Hypertension

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Abstract

Background: Idiopathic Intracranial Hypertension (IIH) is characterized by increased intracranial pressure (ICP) without a clear etiology. Up to 50% of patients report audiovestibular symptoms, such as pulsatile tinnitus and dizziness, yet few studies have assessed the objective impact on auditory and vestibular systems. **Objective:** to evaluate the effect of elevated ICP on auditory and vestibular function in patients with IIH using audiological and neuro-otological test batteries. **Methods:** This case-control study included 25 adult females with IIH and 25 age-matched healthy female controls. Participants underwent comprehensive auditory evaluation (pure tone audiometry, speech audiometry, tympanometry), cervical and ocular vestibular evoked myogenic potentials (cVEMP, oVEMP), and videonystagmography (VNG). **Results:** Significant differences were observed between groups in pure tone thresholds (250–8000 Hz) and speech recognition thresholds. cVEMP showed prolonged P13 and N23 latencies and reduced amplitudes; responses at 500 Hz were absent in 9 ears. oVEMP results revealed absent responses in 10 ears, delayed P15 latency, and reduced N10–P15 amplitudes. VNG revealed spontaneous nystagmus (24%), oculomotor test abnormalities (20%), and caloric weakness (32%). Positional testing identified BPPV in 5 cases. Audiovestibular symptoms included dizziness (44%), pulsatile tinnitus (40%), and ear fullness (28%). **Conclusions:** IIH significantly affects both auditory and vestibular systems, often subclinically. Auditory involvement is reflected in pure tone thresholds, while vestibular impairment includes both peripheral and central features, evidenced by VEMP and VNG abnormalities. Audiovestibular testing should be routinely considered in IIH management.

Keywords: Idiopathic Intracranial Hypertension, Audiovestibular Dysfunction, VEMP, VNG, Pulsatile Tinnitus, Dizziness, Hearing Loss.

Introduction

Idiopathic intracranial hypertension (IIH) is a disease characterized by increased ICP without any apparent cause, such as hydrocephalus. It is frequently detected in young obese females. A lot of terms are utilized to define this state (IIH, benign intracranial hypertension, pseudotumor cerebri). Currently, IIH is the preferred term.[1] Typical manifestations of IIH involve headache, papilledema, diplopia, transient visual obscurations, and divergence insufficiency.[2]

It is known that the intracranial and intralabyrinthine spaces are firmly connected via several connections, comprising the cochlear duct and vestibular aqueduct, such connections are thought to account for some auditory and vestibular symptoms and also such connections. May favor the use of IIH as a model to study the effect of ICP alterations on the inner ear.[3]

Vestibular evoked myogenic potentials (VEMPs) are short-latency muscle potentials produced by vestibular system excitation by using a loud sound.[4] The most used types

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are cervical VEMPs, measured over contracted anterior neck muscles, primarily the sternocleidomastoid muscle (SCM), and ocular VEMPs (oVEMPs), measured over contracted extraocular muscles mainly inferior oblique recorded below the eye contralateral to the triggered ear by using surface electrodes.[5]

In IIH, hearing loss (HL) is a symptom recorded in about 57.5% of cases.[6] Previous studies on cases with assumed pseudotumor cerebri, comprising those with IIH mimics, characterised the HL as sensorineural, affecting the lower frequencies,[7] whereas another new study conducted on IIH cases displayed asymmetry of the frequencies affected in each ear.[2]

Vestibular dysfunction in IIH patients was reported in about 23%–50% of patients,[8] proof of impaired central vestibular function is defined, with a single study of 20 cases displaying proof of central pathology on electronystagmography, comprising optokinetic asymmetry, and tracking test changes in ten cases of the study group.[3]

Various mechanisms were suggested to clarify otological manifestations in IIH. Transmitted pressure into the inner ear from the subarachnoid space through the vestibular aqueduct or from elevated perilymphatic pressure through the cochlear duct is suggested.[9,10] Changes in perilymphatic and endolymphatic pressures by increased ICP, look like to endolymphatic hydrops, is thought,[8] although about 50% of the cases report audio vestibular symptoms such as dizziness very and pulsatile tinnitus a limited number of studies are present assessing audio vestibular functions.[10] So, we aimed to assess the auditory and vestibular functions in patients with IIH.

Patients and Methods

This case-control study was held at audiology unit, Mansoura University Hospital, Egypt and included 25 adult patients diagnosed with IIH (fulfilling thier diagnostic criteria at neurology department) as case group and a control group of 25 normal adults with no audiolovestibular or neurological complaint. But we exclude patients with any age less than 18 years or more than 40 years, patients with any other neurological disease or diseases causing peripheral or central hearing loss. Both the groups had normal middle ear functions.

Ethical Consideration

Following the Helsinki Declaration, informed written consent was obtained from both groups involved in this study. The study was conducted according to the Mansoura ORL Department Ethical Committee standards and was approved by the Faculty of Medicine IRB.

Equipment

Instrumentation for basic audiological evaluation included Immittancemetry (interacoustics, Titan, Denmark), two channel audiometer (Interacoustic, AC40 diagnostic audiometer, version 1.48 (Denmark)), videonystagmography (Micromedical, Spectrum, Visual eye, version 6.1. (Unites States of America)), and (Interacoustic, eclipse EP15 Auditory Evoked Potential).

Methods

All subjects were subjected to complete audiological history, otological assessment, and basic audiological assessment comprising pure tone audiometry, speech audiometry and immittancemetry measurement including tympanometry. Videonystagmography (VNG), where the patient was prepared by being instructed not to eat for four hours or more before the test, evade consuming caffeine for 72 hours prior to the test, and also discontinue taking some drugs, such as sedatives, tranquilizers, and anti-vertiginous. The goggles were placed on the participant's head and over his eyes, providing a totally dark visual environment. The patient's eyes were fitted with infrared glasses that were firm but comfortable. At the start of the VNG testing, calibration was done.

VNG test approaches involve spontaneous nystagmus, oculo-motor tests (Smooth pursuit, Saccade testing, Gaze fixation, Optokinetic stimulation), positioning testing (Dix—Hallpike maneuver, supine roll test, and head hanging maneuver), positional testing (Sitting, supine, head right, head left correct lateral and left lateral positions) and bithermal open loop water caloric tests.

cVEMP and oVEMP, respectively were done at frequencies of 500 and 1000 Hz. To decrease electrode impedance, the skin near the electrode locations was cleaned using gauze soaked in alcohol. Disposable electrodes were utilized.

For cVEMP, on each side, 2 active electrodes were positioned on the middle 1/3 of the contracted ipsilateral SCM. The reference electrodes were positioned on the upper sternum and the ground on the forehead. The case was asked to rotate his head to the other side of recording with flexing the head about thirty degrees forward to achieve adjustable contraction of the SCM active contraction range default set for range 50-150 micro volt root mean square, monitor turns green when patient contracts within pre-defined range. From all recorded traces, the positive and the negative peaks were recognized based on their latencies, followed by measuring the wave amplitude from the beak to the trough (p13-n23). At least 2 successive averages were recorded from both sides to confirm reproducibility. The Asymmetry Ratio was calculated.

With regard to oVEMP, 2 active electrodes were positioned below the center of the lower eyelid. The reference electrodes were positioned over the lower forehead ground on the forehead just above the reference. The subjects were instructed to sit and maintain a 30-degree upward gaze and keep gazing upward at a fixed mark in the ceiling while hearing the stimulus. The first negative peak happening around a latency of ten ms was marked as N1, and the following positive peak was marked as P1 occurring around a latency of 15ms. The peak latency of both N10 and B15 and peak to peak amplitude (n10-p15) were measured from all participants. Asymmetry Ratio and frequency tuning were calculated in the same pattern as cVEMP

Sample Size

The calculation was based on the difference between cases with increased intracranial hypertension and control groups regarding VEMP records retrieved from previous research.[2] Using G power program version 3.1.9.4 to calculate sample size based on expected difference in of 35%, using 2-tailed test, α error =0.05 and power = 80.0%, allocation ration N2/N2=1, the total calculated sample size will be 22 in each group and by adding 10% to compensate for possible drop out the sample size will be 25 in each group.

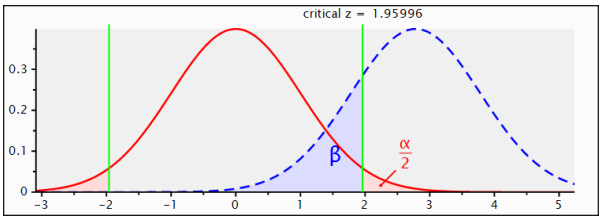

Figure 1: Visualization of Type I (α) and Type II (β) Errors in Hypothesis Testing.

Statistical Analysis

Data analysis was performed using SPSS software, version 26 "(PASW statistics for windows version 26. Chicago: SPSS Inc."). Qualitative data were described by number and percent while Quantitative data were defined using median for non-normally distributed data and mean±SD for normally distributed data following assessing normality using Kolmogrov-Smirnov test. The significance of the results was set at the (0.05) level. Chi-Square and Fisher exact test were utilized to compare qualitative data between groups. U test was utilized to compare between two studied groups for non-normally distributed data. Student t test was utilized to compare two independent groups for normally distributed data. ROC curve was utilized to measure validity of continuous variables with calculation of best cutoff point.

Results

Table 1 shows that there was insignificant difference between the study group and the control group regarding age distribution; both groups consisted of adult females. Concerning the PTA, a significant difference was observed across the range of 250 to 8000 Hz and in the speech recognition threshold (SRT). Conversely, both groups exhibited no significant difference in the speech discrimination score.

The cVEMP response at 500 Hz was absent in 9 of the 50 ears tested. A statistically significant difference was observed within the study group regarding the P13 latency and N23 latency and P13-N23 amplitude. Among the 19 cases with a bilaterally present response, 2 cases exhibited an abnormal asymmetry ratio. Among the 40 ears that demonstrated a response at both 500 and 1000 Hz, 4 ears showed evidence of frequency tuning at 1000 Hz. 3 of the 50 tested ears displayed a response at 1000 Hz while having an absent response at 500 Hz.

The oVEMP response at 500 Hz was absent in 10 ears of the 50 tested ears. At 500Hz There was a statistically significant difference between the control and study groups regarding the N10 l and p15 latency and N10-P15 amplitude. Among the 19 cases with a bilateral presence of response, 4 cases exhibited an abnormal asymmetry ratio. Among the 39 ears with a response at 500 and 1000 Hz, 3 displayed the presence of frequency tuning at 1000 Hz. Among the 50 ears tested, 2ears displayed a presence of response at 1000 Hz and absence at 500 Hz.

Table 1: Demographic Characteristics, Pure tone Audiometry Threshold, cVEMP and oVEMP of the Studied Groups.

Items	Study Group	Control Group	Test of Significance
Age/Years			t=1.19
Mean ±SD	34.80±7.54	32.0±8.92	p=0.236
Sex			
Females	50(100%)	50(100%)	P=1.0
Pure Tone Audiometry Threshold			
250 Hz	20.80±7.85	11.40±5.81	Z=6.81, P=0.001*
500 Hz	18.8±6.74	9.70±5.66	Z=7.31, P=0.001*
1000 Hz	14.70±6.58	6.10±6.22	Z=6.68, P=0.001*
2000 Hz	15.3±6.8	5.90±6.75	Z=6.93, P=0.001*
4000 Hz	15.10±56.93	8.1±6.14	Z=5.80, P=0.001*
8000 Hz	17.60±7.23	11.60±6.18	Z=4.46, P=0.001*
SRT	18.30±6.19	7.90±5.25	Z=9.05, P=0.001*
Speech recognition score	N (%) 25 (100)	N (%) 25 (100)	
cVEMP			
p13 latency	14.31±1.34	13.51±0.94	t=3.32, p=0.0001*
n23 latency	23.03± 1.19	21.70± 1.12	t=5. 45, p<0.001*
p13-n23 amplitude	131.86±48.94	157.72±34.02	t=2.97, p=0.004*
Asymmetry Ratio	N=19 *case*		
Normal Abnormal	17 2	89.5% 10.5%	
Frequency tuning	N=40 ear 4	10%	
present response at 1000 Hz and absent at 500 Hz	3ear	6%	
Absent response at 500Hz	9 ear	18%	
oVEMP			
N10 latency	10.99±1.01	10.96±0.84	Z=0.131, P=0.896
p15 latency	16.15±0.99	14.81±0.78	t=7.17, p<0.001*
n10-p15 amplitude	5.34±2.52	6.64±2.39	Z=2.46, P=0.016*
Asymmetry Ratio	N of cases=19		
Normal Abnormal	14(78.0) 4(21.0%)	N A	
Frequency tuning	N=39 ear 3(7.6%)	0	
Absence of response at 500 Hz and presence of response at 1000 Hz.	3(6%) ears	0	
Absent response at 500Hz	10(20%) ears	0	

t: Student t test

Table 2 presents the audiological and vestibular complaints among the study group. Table 3 illustrates the VNG results among the study versus the control group oculomotor test abnormality was found in five cases, one case showed abnormalities in random sacchadic testing in the form of prolonged latency and smooth pursuit trackins in the form of cogwheel (sacchadic) appearance and optokinetic nystagmus asymmetry testing, one case showed decreased gain in smooth pursuit and OKN.

One case showed cog wheel (sacchadic) appearance in smooth pursuit tracking and assymetrical OKN. Two cases showed abnormality in saccadic accuracy in the form of overshoot and decreased gain in OKN .11 cases showed abnormal posional and poisoning test. Two of them had posterior canal BPPV and three of them had lateral canal BPPV.

Table 2: Audio-vestibular Symptoms in the Study Group.

Items	Number (N)	Percentage (%)
Pulsatile tinnitus	10	40.0
Non-pulsatile tinnitus	4	16.0
Hearing loss	3	12.0
Ear fullness	7	28.0
Imbalance	11	44.0
Sense of rotation	10	40.0

Table 3: Videonystagmography (VNG) Results of the Study and Control Groups.

VNG	Study Group		Control Group	
	N of Case	%	N of Cases	%
Spontaneous Nystagmus				
Normal	19	76.0	25	100.0
Abnormal	6	24.0	0	0.0
Oculomotor Test				
Saccades				
Normal	22	88.0	25	100.0
Abnormal	3	12.0	0	0.0
Smooth Pursuit				
Normal	22	88.0	25	100.0
Abnormal	3	12.0	0	0.0
Optokinetic Test (OKN)				
Normal	20	80	25	100.0
Abnormal	5	20	0	0.0
Gaze Test				
Normal	25	100.0	25	100.0
Abnormal	0	0.0	0	0.0
Positional and Positioning Test				
Normal	14	56	25	100.0
Abnormal	11	44.0	0	0.0
Caloric Test Results				
Normal	17	68.0	25	100.0
Right weakness	5	20.0	0	0.0
Left weakness	3	12.0	0	0.0
Fixation Index				
Normal	25	100.0	25	100.0
Abnormal	0	0.0	0	0.0
Directional Preponderance				
Normal	25	100.0	25	100.0
Abnormal	0	0.0	0	0.0

Table 4 shows regarding cVEMP, the best detected cut of point, Area under curve (AUC) for p13 sounds to be fair for P13 and N23 latency and p13-N23 amplitude. Regarding oVEMP, the best detected cut of point, Area under curve (AUC) sounds to excellent for P15 latency and fair for N10-P15 amplitude and poor for N10 latency.

Table 4: ROC Curve of the cVEMP Parameters (p13 latency - N23 latency – P13 –N23 amplitude) and oVEMP parameters (N10 latency–P15 latency–N10-P15 amplitude).

		AUC (95%CI)	P value	Cut off point	Sensitivity	Specificity
Cervical VEMP	p13 latency	0.678 (0.566-0.790)	0.004*	≥13.45	73.2	52
	N23 latency	0.781 (0.689-0.873)	<0.001*	≥21.95	85.0	54.0
	p13-N23amplitude	0.660 (0.540-0.780)	0.009*	≤122.50	51.2	86
Ocular VEMP	N10 latency	0.516 (0.390-0.542)	0.800	≥11.3150	62.2	50.0
	p15latency	0.842 (0.761-0.923)	<0.001*	≥15.25	85.0	62.0
	N10-P15amplitude	0.697 (0.577-0.817)	0.002*	≥5.3350	64.9	82.0

Table 5 displays descriptive analysis of the number of cases of abnormal oculomotor test and caloric weakness and absent CVEMP and oVEMP and the number of combined cases of more than abnormal test results. Table 6 displays the number of abnormal Oculomotor test, Caloric weakness, Absent cVEMP and Absent oVEMP in relation to dizziness versus non dizziness cases. There was significant difference between dizziness and non-dizziness cases regarding oVEMP (p=0.1).

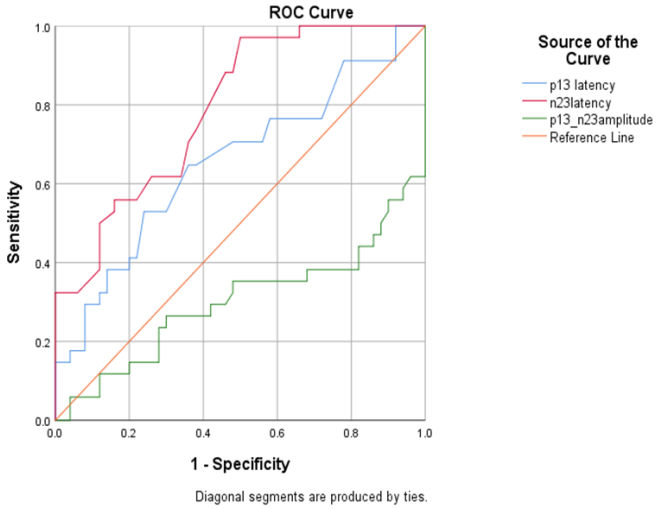

Figure 2: Displays the Receiver Operating Curve (ROC) of the cVEMP Parameter (p13 latency - N23 latency – P13 –N23 amplitude).

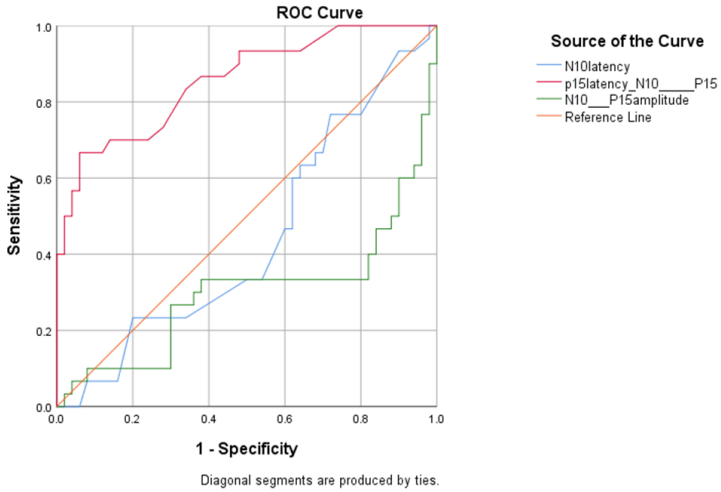

Figure 3: Shows the Receiver Operating Curve (ROC) of the oVEMP Parameter (N10 latency – P15 latency – N10–P15 amplitude).

Table 5: Oculomotor Test and Caloric Weakness.

Test Used	Number of Cases	%
Abnormal Oculomotor test	5	20. 0
Caloric weakness	8	32.0
Absent cVEMP	6	24.0
Absent oVEMP	6	24.0
Abnormal Oculomotor test and absent Cvemp	1	4.0
Abnormal Oculomotor test and absent oVEMP	1	4.0
Caloric weaknes and absent cVEMP	3	12.0
Caloric weaknes and absent oVEMP	5	20.0
Abnormal Oculomotor test and absent cVEMP and absent oVEMP	1	4.0
Caloric weaknes and absent cVEMP and absent oVEMP	2	8.0

Table 6: Oculomotor Test and Caloric Weakness in Relation to Dizziness Versus Non Dizziness Cases.

Test Used	Dizziness (15 case)	Non Dizziness(10 case)	P value
Abnormal Oculomotor test (5)	4 (26.7)	1(10)	0.307
Caloric weakness (8 cases)	6 (40)	2(20)	0.293
Absent cVEMP (6cases)	4(26.7)	2 (20)	0.702
Absent oVEMP (7 cases)	6 (40)	0	0.01*

Discussion

Idiopathic intracranial hypertension (IIH) is a disease of unknown etiology, characterized chiefly by headaches and vision disorders because of elevated ICP. Its clinical picture is highly diverse; however, sometimes the first presentation is just pulsatile tinnitus or dizziness. This is not a surprising finding due to the strong intracranial and intralabyrinthine connections via the cochlear duct and vestibular aqueduct; such connections are thought to account for some auditory and vestibular symptoms. Also, such connections may favor using IIH as a model to study the effect of ICP changes on the inner ear.[3] Although about 50% of cases report audiovestibular symptoms which include dizziness and pulsatile tinnitus, very few studies evaluate audiovestibular functions.[10]

So, the rationale for this work was to evaluate the effect of increased intracranial pressure on the auditory and vestibular systems. The current study included 50 adult females divided equally into study group and control group. The IIH is often detected in young overweight females based on Andrews *et al*.[11] and McCluskey *et al*.[12]. The female-male ratio differs between 2:1 and 8:1 also our small sample size may be a causative factor that all study group are of adult females. The age distribution is between 3 and 58 years, with the median age of 30. So, the demographic character of this study matches both the age and the sex of the highest incidence of IIH, and accordingly, the control group was chosen to match the study in age and sex for accurate test results.

In this study, 40% of the study group reported experiencing pulsatile tinnitus (PT). According to Sismanis *et al*.[13] pulsatile tinnitus is considered the most prevalent otological symptom in idiopathic intracranial hypertension (IIH), affecting approximately 52–61% of IIH patients. The hypothesized mechanism behind PT involves the transmission of systolic vascular pulsations from the cerebrospinal fluid (CSF) to the transverse and sigmoid venous sinuses, causing intermittent compression and turbulent blood flow in structures nearby to the ears. Application gentle digital pressure to the internal jugular vein could reduce or totally alleviate PT in cases with IIH, seemingly by disturbing the mechanisms that induce intermittent venous sinuses compression.

Also, six patients within the study group experienced unilateral PT, whereas four patients presented with bilateral PT. These findings contrast with those reported by Sismanis *et al*.[13] who noted that PT is most often bilateral and relatively symmetric , According to King *et al*.[14] the laterality of PT directly correlates with the dominance pattern of the dural venous sinus system; unilateral PT is linked to stenosis in the ipsilateral dominant sinus, whereas bilateral PT is associated with bilateral stenosis within a codominant system. Similar variations are also observed within the jugular bulb and its relationship to the sigmoid sulcus.

Continuous investigation revealed that 16% of the study group reported experiencing nonpulsatile tinnitus (NPT), as indicated by Funnell *et al*.[15]. The elevation of intracranial pressure (ICP) could have a significant role in the development of endolymphatic hydrops (ELH), thereby elucidating the heightened prevalence of NPT within the idiopathic intracranial hypertension (IIH) population. Corroborating findings from Ranieri *et al*.[8] proposed the causative influence of elevated ICP on the mechanisms of ELH, potentially mediated through direct pressure transmission to the inner ear fluids, which parallels the observation that 28% of the study group experienced a sensation of aural fullness.

There was a significant difference in the averaged pure tone audiometry (PTA) thresholds across the tested frequency range of 250 to 8000 Hz and the speech recognition threshold (SRT) between the study and control groups. This came in agreement with Çoban *et al*.[2] who identified a significant difference in the tested frequency range in both ears. However, they diverge from the earlier study by Sismanis *et al*.[13] which indicated that patients with idiopathic intracranial hypertension (IIH) are more likely to experience low-frequency hearing loss. These discrepancies may be attributed to the masking effect of pulsatile tinnitus, which can result in pseudo sensorineural low-frequency hearing loss, thereby leading to an overestimation of low-frequency sensorineural hearing loss (SNHL). Consequently, the tension on the basilar membrane increases, and the mobility of the stapes footplate is impeded, leading to hearing loss.[16]

In the present study, the results of speech discrimination scores were found to be comparable between the two groups. This finding can be attributed to the minimal degree of hearing impairment observed, particularly within speech frequencies, which did not ascend to a level adequate to disrupt the speech discrimination scores. Furthermore, this limited degree of hearing impairment may also explain the low percentage of hearing loss sensation (12%) recorded within the study group.

The VNG results of the study compared with the control group showed six patients (24%) exhibited spontaneous nystagmus. These findings diverge from the earlier study conducted by Sismanis[7] who evaluated vestibular function in individuals with IIH utilizing electronystagmography (ENG) on a cohort of 20 patients with benign intracranial hypertension, all of whom did not display spontaneous nystagmus.

Within our study, spontaneous nystagmus may be attributed to an irritative effect induced by the elevation of both perilymphatic and endolymphatic pressures resultant from ICP akin to endolymphatic hydrops. An alternative explanation may involve the compression or stretching of the vestibulocochlear nerve resulting from elevated ICP of the six patients, four patients showed horizontal nystagmus and two showed mixed up beating and horizontal nystagmus, The noticed increase of number of horizontal nystagmus may be explained by the orientation of lateral canal orientation (horizontal plane) makes it more sensitive to pressure changes of

endolymph, the weaker structural support compared to other canals and the vestibular afferent dominance of the superior vestibular nerve supplying the superior and lateral canal this dominance may also explain the presence of combined up beating and horizontal nystagmus due to irritation of the afferent pathway of superior vestibular nerve.[17]

Abnormalities in oculomotor testing were identified in the current study as following one case showed abnormalities in random sacchadic testing in the form of prolonged latency and smooth pursuit trackins in the form of cogwheel (sacchadic) appearance and optokinetic nystagmus asymmetry testing, one case showed decreased gain in smooth pursuit and OKN .One case showed cog wheel (sacchadic) appearance in smooth pursuit tracking and assymetrical OKN. Two cases showed abnormality in saccadic accuracy in the form of overshoot and decreased gain in OKN, these results matches with Çoban *et al*.[2] and can be explained by Raised ICP is postulated to affect brainstem and cerebellum where the oculomotor nucelii present also abducent nerve palsies leading to lateral rectus muscle impairment and horizontal gaze abnormalities.[16]

Abnormalities in positional and positioning tests were noted in 11 cases (44%), with two patients diagnosed with posterior canal BPPV and three patients identified with lateral canal BPPV. Repositioning maneuvers were administered to these patients, leading to an improvement in their signs and symptoms. Bithermal caloric irrigation was conducted, revealing that five patients demonstrated right caloric weakness while three patients exhibited left caloric weakness, with no instances of bilateral weakness or abnormal fixation index identified. These findings align with the observations made by Çoban *et al*.[2] who reported caloric test abnormalities in three of their 20 patients, two unilateral and one bilateral. The noted abnormalities may be explained by increased tension within the vestibular nerve or the endolymphatic system, which is acknowledged as one of the primary pathophysiological explanations. A potential mechanism for vestibular symptoms may include malabsorption of endolymphatic fluid, resulting from direct compression or diminished vascular supply to the endolymphatic sac, situated between the dural leaflets.[18]

Regarding comparison of cervical Vestibular Evoked Myogenic Potential (VEMP), results between the study group and the control group aligned with the findings of Uyaroğlu *et al*.[3]. Although vestibular symptoms are prevalent in IIH, there is a paucity of VEMP research. This study documented the investigation of cervical VEMP (cVEMP) and ocular VEMP (oVEMP). Notably, cVEMP abnormalities were identified, including an absent response at 500 Hz in six cases (three bilateral and three unilateral) and a significant variance in p13 and N23 latencied and p13-n23 amplitude between the groups. Two cases within the study population demonstrated abnormal asymmetry ratios.

Our findings correlate with those of Çoban *et al*.[2] who reported that eight individuals (40%) in their study exhibited various pathological signs in VEMP responses. Three cases did not yield VEMP responses, which included one bilateral case. Uyaroğlu *et al*.[3] recorded cVEMP responses in 30 patients diagnosed with IIH prior to lumbar puncture. In contrast to the 30 healthy controls, all of whom produced bilateral responses, 49 out of 60 tests (81%) were successfully conducted in patients with IIH. Five patients exhibited absent bilateral responses, and one patient displayed an absent unilateral response. The patients' response latency and amplitude values did not demonstrate statistically significant differences compared to healthy controls.

We focused on another feature of the cVEMP measurement identified as cVEMP tuning. The cVEMP "tuning" refers to the amplitude difference between p13 and n23 at 1,000Hz divided by the amplitude difference of identical peaks at 500Hz within the same ear. In cases of Meniere's disease, the cVEMP 1000/500 Hz amplitude ratio has been recorded to be abnormal. It can be utilized in the differential diagnosis of non- Meniere's disease vertigo.[19] Based on the most accepted pathophysiological mechanism underlying audiovestibular symptoms in IIH, which involves increased perilymphatic and endolymphatic pressures due to ICP, akin to endolymphatic hydrops, we aimed to assess cVEMP tuning in IIH. This study found that abnormal cVEMP tuning was present in 4 ears of three different cases of the study group. Furthermore, we identified 3 ears with an absent response at 500 Hz and a present response at 1000 Hz. These results may elucidate the pathophysiological mechanism of the transmission of increased CSF pressure to the cochlear aqueduct and internal acoustic canal, leading to compression or increased tension in the endolymphatic system and subsequent endolymphatic hydrops.[20] To our knowledge, cVEMP tuning has not been studied previously and warrants further research.

In the present study, oVEMP abnormalities were documented as follows: there was a lack of response at 500 Hz in six cases, four of which were bilateral and two were unilateral. A significant difference was observed in the P15 latency and N10-P15 amplitude between the study and control groups. Within the study group, four cases demonstrated an abnormal asymmetry ratio.

In a manner akin to cVEMP, the tuning characteristics of oVEMP were examined. Tuning refers to the amplitude difference between N10 and P15 at 1000Hz, divided by the amplitude change of identical peaks at 500Hz. The study findings revealed that 3 ears, constituting 7.6% of the study group, exhibited abnormal oVEMP tuning. Furthermore, three ears were identified, representing 6%, that displayed an absent response at 500 Hz yet a present response at 1000 Hz, suggesting a similar underlying mechanism as speculated for cVEMP.

Gürkov *et al*.[10] investigated the effects of alterations in CSF pressure induced by lumbar puncture and whether

this intervention could change the parameters of oVEMP, for both air conduction stimulation (ACS) and bone conduction stimulation (BCS). The findings indicated that, for ACS oVEMPs, the mean amplitudes exhibited a statistically significant increase from 5.0 (±2.4) μV to 8.2 (±5.1) μV following the spinal tap. Conversely, for BCS, the mean amplitudes of BCS oVEMPs were measured as 36.3 (±17.6) μV and remained essentially unchanged post-lumbar puncture as 39.0 (±13.4) μV. Additionally, it was found that ACS oVEMPs are inhibited by an increasing ICP to a significantly higher degree compared to BCS oVEMPs. Thus, it is plausible to suggest that an increase in ICP, whether caused experimentally or associated with IIH, contributes to a rise in intraotic pressure through the vestibular and cochlear aqueduct, thereby exerting slight pressure on the stapes at the oval window outward, and this could be accompanied by an increased stiffness of the sound transmission system exhibits reduced efficiency in transferring air-conducted acoustic energy from the external ear canal to the otolithic afferents in the vestibulum. In contrast, bone-conducted vibrations don't mainly depend on this sound transmission system and are consequently not considerably inhibited by elevated intracranial pressure.

Conclusion

Idiopathic intra cranial hypertension affects both the auditory and the vestibular system. Auditory system affection manifests itself in pure tone audiometry by subclinical abnormalities across of whole tested frequency range. Vestibular system abnormalities involve both central and peripheral abnormalities in the form of abnormal oculomotor tests and abnormal caloric weakness respectively in the videonystagmography tests.

Recommendations

We recommended performing routine audio-vestibular test battery in IIH patients, to compare the audio- vestibular function in IIH patients before and after therapeutic maneuver, to increase the sample size of both the study and control group and to study the relation between audio- vestibular and MRI, MRA, and MRV and CSF flowmetry in patients with IIH.

Conflict of Interest

None.

Funding

None.

Reviewer Disclosures

None.

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