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Evaluation of audiovestibular functions in patients with posterior circulation cerebrovascular stroke

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Abstract

Background It has been found that 25% of posterior circulation stroke patients experience vertigo. Sudden hearing loss due to a vascular source is typically caused by infarction in the anterior inferior cerebellar artery territory. However, it is uncommon in the posterior inferior cerebellar artery territory infarction.

Aim of the work The objective of the proposed study is to evaluate the function of the auditory and vestibular system in patients with posterior circulation cerebrovascular stroke.

Methods Fifty patients with posterior circulation stroke, diagnosed by MRI at least 3 months after onset, were included. The controls consisted of fifty healthy people. The average age in cases was 58.68 ± 8.60 years, while in controls, it was 55.44 ± 11.72 . The Health Stroke Scale (NIHSS) was used to conduct clinical assessments. All study groups were investigated using pure tone audiometry, auditory brain stem-evoked potential (ABR), videonystagmography (VNG), and vestibular-evoked myogenic potential (VEMP).

Results MRI findings revealed that pontine infarction was the most frequent lesion in 36 (72%) patients. The latencies of ABR waves I, III, V, I–III, I–V, and III–V were all considerably longer in cases than controls. Cervical VEMP P1 latency was considerably delayed in cases compared to controls, as the amplitude was lower. VNG data found abnormalities in all test parameters, indicating central disease.

Conclusion Posterior circulation stroke causes acute sensorineural hearing loss with varying degrees. Videonystagmography is an accurate assessment of oculomotor function that can be combined with vestibular-evoked myogenic potential to objectively assess posterior circulation stroke patients.

Keywords Hearing loss, Auditory brainstem-evoked potential, Vestibular function test, Posterior circulation stroke

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Background

Posterior circulation stroke (PCS) is characterized as an acute neurological impairment persisting 24 h and imaging of the brain demonstrating parenchymal infarction located in the vertebrobasilar area [1]. Infarcts are classified as proximal, intermediate, or distal to the posterior circulation [2]. Sixty-two percent of individuals suffering from vertebrobasilar infarction experienced more than one episode of solitary vertigo, and 19% acquired vertigo as the first symptom [3].

Acute vestibular syndrome (AVS) is defined by acute onset persistent vertigo or dizziness that continues for at least 24 h [4]. AVS of central etiology has been seen more frequently in strokes affecting the caudal cerebellum or the dorsal parts of the lower brainstem [5]. This syndrome is caused by a static and dynamic asymmetry in the release of both sides' vestibular systems [6].

About 10% of all cerebellar infarctions are accompanied by solitary spontaneous vertigo and disequilibrium in the absence of other evident cerebellar manifestations. The medial branch of the posterior inferior cerebellar artery (PICA) territory (about 96%) is the most usually implicated vascular territory, which is followed by the anterior inferior cerebellar artery (AICA) territory (approximately 4%). Because AICA provides the peripheral labyrinth and central vestibular structures, AICA stroke generally appears by sudden audiovestibular loss [7].

A stroke frequently affects the auditory system, which leads to unilateral or bilateral hearing impairment, peripheral and/or central hearing loss, and cortical impairment. Certain disorders are obscure and require careful psychoacoustic and electrophysiological testing to detect [8]. The major cause of sudden hearing loss following stroke is anterior inferior cerebellar artery (AICA) stroke, which accounts for 83% of cases, whereas posterior inferior cerebellar artery (PICA) stroke accounts for 12% of cases [9].

Aim of the work

The objective of the proposed study is to evaluate the function of the auditory and vestibular system in patients with posterior circulation cerebrovascular stroke.

Methods

This was a case-control study. The study started in January 2021 and finished in June 2023. It entailed 50 patients with posterior cerebrovascular stroke. Age ranged from 18 to 55 years old. Patients were recruited from the neurology outpatient clinic, Beni-Suef University Hospital. The audiovestibular assessment was performed at the unit of audio-vestibular medicine. The control group constituted 50 individuals who were age and gender matched

with the study group. Subjects with transient ischemic attack (TIA), concomitant anterior circulation infarction, neurodegenerative diseases like Parkinson's disease and Alzheimer's disease, and metabolic illness known to affect hearing or ear pathologies causing conductive hearing loss were excluded. The study was approved by the ethical committee of the Faculty of Medicine, Beni-Suef University. Informed written consent was obtained from all participants.

All participants were subjected to the following procedures:

1. History taking: focusing on vascular risk factors including age, body mass index (BMI), smoking, diabetes mellitus (DM), hypertension (HTN), drug abuse, and history of recurrent stroke in addition to audiovestibular symptoms and posterior circulation stroke symptoms.
2. Neurological assessment:
 - National Institute of Health Stroke Scale (NIHSS) [10] was used to objectively quantify the functional impairment caused by stroke. The NIHSS is composed of 11 items; each of them has a score between 0 and 4. For each item, a score of 0 typically indicates normal function, while a higher score is indicative of some level of impairment. The maximum possible score is 42, with the minimum score being 0.
3. Radiological assessment:
 - a. Magnetic resonance imaging (MRI brain): It was done for all included patients to detect site and size of the infarction.
 - b. Vertebro basilar duplex: to detect atherosclerosis or stenosis of vertebrobasilar arteries.
4. Basic audiological evaluation including:
 - Pure tone audiometry: including air and bone conduction (PTA "AD 629 by Intreacoustics").
 - Speech audiometry: including Speech Reception Threshold (S.R.T), using Arabic spondaic words [11] and word discrimination score (WDS %), using Arabic phonetically balanced (PD) [12].
 - Immittance: including tympanometry and acoustic reflex threshold (Tympanometry "AT 235 by Interacoustics").
5. Brainstem auditory-evoked potential using Intera-coustic Eclipse "EP15". Click presented at a rate of 21.1 and 61.8 stimuli per second in alternating polarity at intensity of 80 dB nHL. Averaged potentials to 1000–2000 clicks were obtained. Two record-

ings were obtained to ensure the replicability of the waveforms. The analyzed parameters were absolute latencies of waves I, III, and V, inter-peak latencies (I–III, III–V, and I–V), inter-aural latency difference of waves III and V, and the effect of high rate (61.8 vs. 21.1).

6. Vestibular system evaluation by using:
 - a. Videonystagmography (VNG“ICS Chart 200 VNG/ENG”)
 - b. Vestibular-evoked myogenic potential testing using Eclipse EP15 by Interacoustics. It included:
 1. Cervical vestibular-evoked myogenic potential (cVEMP).
 2. Ocular vestibular-evoked myogenic potential (oVEMP).

Statistical analysis

Categorical variables were reported as numbers and percentages of patients, which were then compared across groups using Pearson’s chi-square test or Fisher exact (where more than 20% of cells had an anticipated count less than 5). To determine group differences in continuous variables, means, standard deviations, and medians (interquartile range) were calculated using normal distribution. We compared two groups using the independent *T*-test or the Mann-Whitney *U* test, depending on their normalcy. For our analysis, we employed IBM’s SPSS version 27 statistical software, with an alpha of 5%. Thus, a *P*-value of 0.05 was deemed statistically significant.

Results

This study included 100 subjects: 50 cases with posterior circulation cerebrovascular stroke and 50 healthy controls. The mean age in cases was 58.68 ± 8.60 years and in controls was 55.44 ± 11.72. Males were 58% of cases and 52% of controls vs. 48% and 42% females in both groups, respectively.

Table 1 shows that the mean NIHSS score was 4.1 ± 1.96. The selected cases had a mean onset of attack of 11.9 ± 9.3 months. There were 26 (52%) of cases with bilateral diffuse atherosclerotic changes of carotid arteries with increased resistive index of both vertebral arteries. MRI findings revealed that pontine infarction was the most common lesion in 36 (72%) patients.

Table 2 shows that all the latencies of waves I, III, V, I–V, and III–V of auditory-evoked potential response test (ABR) were significantly prolonged in cases than controls (*P*-value < 0.001).

Table 1 Clinical and radiological characteristics of patients

	Cases (no. = 50)	
	No	%
NIHSS		
Mean ± SD	4.1 ± 1.96	
Median (min–max)	4.0 (1.0–12.0)	
Onset of attack	11.9 ± 9.3	
	10.0 (3.0–48.0)	
Side of lesion		
Lt	23	46.0
Rt	27	54.0
Vertebro basilar duplex findings		
Bilateral diffuse atherosclerotic changes of carotid arteries with increased resistive index of both vertebral arteries	26	52.0
Normal cerebral angiography	24	48.0
MRI findings		
Cerebellar infarction	5	10.0
Medullary infarction	4	8.0
Occipital infarction	5	10.0
Pontine infarction	36	72.0

Table 2 Comparison between cases and controls regarding auditory brainstem evoked potential response test (ABR) latencies

	Controls (no. = 50) Mean ± SD	Cases (no. = 50) Mean ± SD	<i>P</i> -value
Wave I latency	1.33 ± 0.15	1.46 ± 0.25	< 0.001*
Range (min–max)	1.00–1.73	1.00–1.93	
Wave III latency	3.38 ± 0.13	3.49 ± 0.22	< 0.001*
Range (min–max)	3.03–3.69	3.07–4.20	
Wave V latency	5.37 ± 0.22	5.57 ± 0.25	< 0.001*
Range (min–max)	5.00–5.93	5.00–6.20	
I–III latency	2.01 ± 0.14	2.05 ± 0.17	0.109
Range (min–max)	1.63–2.50	1.71–2.63	
I–V latency	4.03 ± 0.24	4.09 ± 0.16	0.037*
Range (min–max)	3.55–4.67	3.71–4.54	
III–V latency	1.98 ± 0.23	2.08 ± 0.16	< 0.001*
Range (min–max)	1.50–2.62	1.38–2.60	
V high rate [median (IQR)]	5.4 (5.27–5.6)	5.53 (5.40–5.73)	0.336
Range (min–max)	5.00–5.87	5.00–6.20	

* *P*-value is significant

Table 3 shows that the AC cervical vestibular-evoked myogenic potential (cVEMP) P1 latency was statistically significant delayed in cases than the control, and the amplitude was significantly lower in cases than in controls (*P*-value < 0.001), but the N1 latency did

Table 3 Comparison between cases and controls regarding AC cervical and ocular vestibular-evoked myogenic potential (VEMP)

	Controls (no. = 50)	Cases (no. = 50)	P-value
AC cervical VEMP			
Mean ± SD			
P1 latency	14.15 ± 1.04	14.7 ± 1.4	0.005*
Range (min–max)	11.67–16.00	11–19.33	
N1 latency	24.11 ± 1.12	24.28 ± 2.31	0.527
Range (min–max)	21.33–26.67	11.67–29.33	
Amplitude	72.95 ± 9.63	64.88 ± 17.48	0.001*
Range (min–max)	59.35–98.50	18.93–95.67	
IAAD [median (IQR)]	2.54 (1.18–4.58)	4.49 (2.21–33.63)	<0.001*
Range (min–max)	.07–10.43	(0.02–51.99)	
AC ocular VEMP			
mean ± SD			
N1 latency [median (IQR)]	10.83(10.0–11.67)	10.67(10.0–11.33)	0.143
Range (min–max)	(9.00–112.67)	(9.33–13.00)	
P1 latency	15.46 ± 0.96	15.33 ± 1.10	0.356
Range (min–max)	(13.00–17.67)	(13.00–18.00)	
Amplitude	11.87 ± 4.68	12.0 ± 5.0	0.771
Range (min–max)	(4.36–25.67)	(3.19–24.97)	
IAAD [median (IQR)]	5.25 (3.68–8.81)	10.06 (3.53–12.23)	<0.001*
Range (min–max)	(.51–12.29)	(0.56–59.39)	

* P-value is significant

not differ significantly between groups. The AC ocular (oVEMP) N1 and P1 latencies, and the amplitude did not differ significantly between cases and controls (P -value < 0.001). Seven cases with pontine stroke had absent cVEMP, and three cases also with pontine lesion showed lost oVEMP.

Table 4 demonstrates that there was spontaneous nystagmus in twenty cases, distributed as follows: six patients showed torsional downbeat nystagmus, while four had pure upbeat nystagmus and ten with horizontal nystagmus with slow peak velocity (SPV) (ranging from 8 to 11°). The smooth pursuit test's gain was considerably lower in cases than controls at all testing frequencies. All saccade test parameters were distorted in all patients; however, gaze-evoked nystagmus was only affected in four cerebellar stroke cases. Positional tests revealed torsional downbeat nystagmus in 6 cases, horizontal nystagmus in 10 cases, and pure upbeat nystagmus in 4 cases. Some cases had direction-changing nystagmus 5 (10%) during positional testing. The water caloric test revealed canal weakness in 39 (78%) of the patients.

Discussion

Dizziness or vertigo can be one of the most prevalent symptoms of a posterior circulation stroke [13]. Vertigo has been found to affect 25% of patients with posterior circulation strokes [14]. Recent researches have resulted in the conclusion that a stroke impacting the brainstem

or cerebellum can cause solitary vertigo, dizziness, or imbalance [5].

In the present study, in the 50 patients with posterior circulation stroke, the most common site of infarction was paramedian pons 36 (72%), medulla 4(8%), occipital lobe 5 (10%), and cerebellar 5 (10). Physical examination was performed by the National Institute of Health Stroke Scale (NIHSS), and its score was 4.1 ± 1.96 which means that all patients suffered from mild degree of impairment (Table 1).

Detection of posterior circulation strokes may be missed or postponed [15]. Approximately 28–59% of cerebellar strokes are ultimately misdiagnosed [16]. Clinical deterioration might result from stroke extension, brain stem compression due to posterior fossa edema, or recurrent stroke [17]. Recognizing posterior circulation stroke symptoms requires a more sophisticated physical examination, as the National Institutes of Health Stroke Scale (NIHSS) is biased towards anterior circulation. Patients with posterior circulation strokes may have an NIHSS of zero but still experience debilitating abnormalities [18].

Fifty-two percent of cases in the study group suffered from sensorineural hearing loss (SNHL) with different degrees, 27% had mild SNHL, 14% had moderate SNHL, 4% had moderately severe SNHL, and 7% had severe SNHL (Fig. 1). According to Kim and Lee [19], hearing loss caused by non-anterior inferior cerebellar artery (AICA) territory posterior circulation ischemic stroke is

Table 4 Video nystagmography (VNG) findings in cases and controls

	Controls (no. = 50)	Cases (no. = 50)	P-value
Spontaneous nystagmus			
Nystagmus	0 (100%)	20 (40%)	0.02*
No nystagmus		30 (60%)	
Smooth pursuit test gain			
0.2 kHz	0.89±0.07	0.74±0.12	0.001*
Range (min–max)	(0.68–1.10)	(0.40–0.95)	
0.4 kHz	0.88±0.08	0.69±0.15	0.001*
Range (min–max)	(0.64–1.10)	(0.34–0.94)	
0.7 kHz	0.81±0.09	0.59±0.17	0.001*
Range (min–max)	(.60–0.98)	(0.25–0.86)	
Saccade test			
Latency	217.76±7.78	221.74±7.66	0.011*
Range (min–max)	(205–232)	(207–232)	
Velocity	371.60±28.79	346.00±25.43	0.001*
Range (min–max)	(336–436)	(320–432)	
Accuracy %	92.96±2.53	82.30±8.152	0.003*
Range (min–max)	(89.09–98.53)	(68.41–96.36)	
Gaze-evoked nystagmus (GEN)			
No nystagmus	0 (0%)	46 (92%)	<0.001*
lt. GEN		2 (4.0%)	
rt. GEN		2 (4.0%)	
Positional test			
Head down and up			
No nystagmus	0 (0%)	30 (40.0%)	0.02*
Torsional down beating nystagmus		6 (12%)	
Horizontal nystagmus		10 (24%)	
Pure upbeating nystagmus		4 (8%)	
Unilateral canal weakness	0(0%)	39(78%)	<0.001*
Range (min–max)			

* P-value is significant

Hearing sensitivity in cases

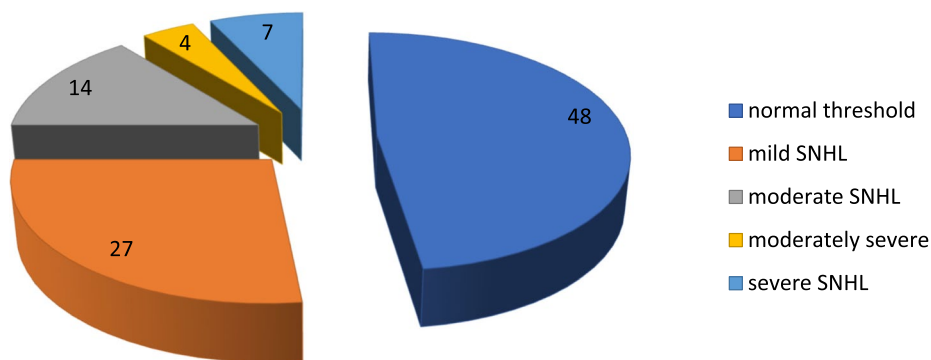


Fig. 1 Hearing sensitivity among patients with posterior circulation cerebrovascular stroke

likely due to injury to the peripheral auditory system that nourishes the inner ear via the internal auditory artery, mainly from the PICA.

In the current study, the latencies of waves I, III, and V of the auditory-evoked potential response test (ABR) were considerably delayed in cases compared to controls (Table 2). These results agreed with Kim et al. [20] who stated that waveform I, III, and V latencies showed delays in patients who had lower functional and neurological scores compared to those who had a more favorable outcome at discharge. On the other hand, Rollnik [21] found that prolonging the latency of waveform III alone in ABR is related to bad outcome for early rehabilitation.

Protuberance features can cause the disappearance of the waveforms III, IV, and V, as well as extended III–V and I–V intervals of ABR. Once brain damage occurs, ABR may reveal the lack of all waves except the wave I that is normally present bilaterally [22]. Alpini et al. [23] described the value of ABR in the identification of vascular disease affecting the brain stem; they highlighted the diagnostic and prognostic importance of this approach.

Regarding VEMP findings, we noticed a statistically significant delay in the latency of P1 and a significant decrease in P1–N1 amplitude value for AC cVEMP in patients with posterior circulation stroke (PCS). But there was any statistically significant difference between cases and control concerning AC oVEMP results (Table 3). Calic et al. [24] found abnormal cVEMPs in 59% of PCS patients, which is consistent with our findings.

Weng and Young [25] discovered in their study that cVEMPs were delayed or missing in 36% of PICA and 75% of AICA patients as a result of lower brainstem lesions. Lesions in the cerebellum can trigger abnormal cVEMP results. Out of 27 patients, 11 (41%) showed a higher inter-aural amplitude difference (IAD) ratio, delayed or absent responses, and no cVEMPs was obtained in the lesion side [26].

The cVEMP study indicates that the peripheral vestibular system is critical in creating an abnormal response in cVEMP during AICA stroke [27]. Furthermore, the absence of cVEMP symmetry could mean the presence of crossed or bilateral otolithic vestibular regulating circuits within the cerebellum [26].

On the other hand, Calic et al. [24] reported that 5% of PCS patients showed abnormal oVEMP findings. According to Weng and Young [25], abnormal oVEMPs were detected among PCS patients (PICA and AICA) as a result of upper brainstem strokes. Over 30% of unilateral cerebellar infarct participants also showed abnormal oVEMP findings, indicating a cerebellar function in controlling the oVEMP pathway.

The VNG findings showed that there was upbeat- ing, torsional down beating, and horizontal nystagmus

in observation for spontaneous nystagmus and in positional testing in twenty (40%) PCS cases, with slow peak velocity (SPV) (ranging from 8 to 11°). Nham et al. [28] mentioned that in the PCS study, 63.6% of patients with horizontal nystagmus had “peripheral-appearing” nystagmus, which does not change direction or is triggered by gaze. Primary position vertical or torsional nystagmus, typically associated with central nystagmus, was uncommon in PCS patients, contributing to fewer than 13% of all nystagmus examinations.

Concerning the smooth pursuit test gain and all saccade test parameters in our study, they were altered in all PCS cases. Calic et al. [24] found no significant difference in saccade occurrence for horizontal semicircular canals (SCCs) in PCS patients compared to controls. The level of involvement in the labyrinth, vestibular nucleus, or flocculus after stroke lesions was responsible for a decline or difference in the vestibulo ocular reflex (VOR) gain results [29].

In the present study, we found gaze-triggered nystagmus in four cerebellar stroke cases. Leigh and Zee [30] noticed that gaze-evoked nystagmus can occur in PICA territory stroke locations such as the lateral medulla, uvula, and tonsils.

As regards the water caloric test, there was canal weakness in 38 (76%) of the patients. In a research conducted by Weng and Young [25], 64% of PICA stroke patients experienced either canal paresis, caloric areflexia, or hyperactive responses to irrigation as a result of diminished VOR following brainstem infarction.

Conclusion

Posterior circulation stroke causes unilateral and/or bilateral acute sensorineural hearing loss with or without vestibular dysfunction. Auditory brain stem-evoked potentials are rarely employed as regular diagnostic tools in stroke diagnosis, but they are extremely valuable in determining complications, anticipating stroke evolution, and identifying stroke disability. Videonystagmography (VNG) is an effective approach for assessing nystagmus. VNG is an accurate assessment of oculomotor function that can be combined with VEMPs to objectively assess central vestibular dysfunction like posterior circulation stroke patients who had delayed neurological signs and normal imaging results during the beginning of the condition.

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Authors' contributions

RAK had an important role in developing the study's concept. ME was critical to the analysis, interpretation, and drafting of the manuscript. MR independently collected study data from the selected participants. MH has a substantial impact on data collecting and analysis. FA made major contributions to

the data analysis. RY made important contributions to the writing and editing of the publication. All authors reviewed and approved the final manuscript.

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Availability of data and materials

The datasets during and/or analyzed during the present study are available from the corresponding author.

Declarations

Ethics approval and consent to participate

All participants received their consent after the study was approved by Beni-Suef University's Research Ethics Committee (Approval No.:FMBSUREC/03012021/Radwan).

Consent for publication

Not applicable.

Competing interests

Mohamed Mohamed El-Badry is a co-author of this study and a Co-Editor of the journal. He has not been involved in handling this manuscript during the submission and review processes. The rest of the authors have no conflict of interest to declare.

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