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Vestibular evoked myogenic potentials and videonystagmography findings in type 2 diabetes mellitus patients with and without polyneuropathy

Rabab Ahmed Koura[1*], Mohamed Mohamed El-Badry[2], Asmaa Mohamed Othman[3], Rehab Hassan El Anwar[4], Sara Ahmed Mahmoud Elsharkawy[1] and Iman Mostafa Basiouny[1]

Abstract

Background Vestibular impairment is a common pathology in patients with type 2 diabetes mellitus (DM) due to ischemia of the vestibular end organs and lysis of the myelin of the vestibular nerve. We aimed to evaluate function of the vestibular end organs and vestibular nerve in patients with type 2 DM with polyneuropathy and compare results to those of the patients without polyneuropathy.

Method The participants consisted of three groups: thirty patients with type 2 diabetes mellitus without polyneuropathy (DM), thirty patients with type 2 diabetes mellitus with polyneuropathy (DPN), and thirty healthy non-diabetic individuals as the control group. Clinical examination, videonystagmography, cervical vestibular evoked myogenic potential(cVEMP), ocular vestibular evoked myogenic potential (oVEMP), Gans Sensory Organization Performance (SOP) test, and nerve conduction study were all performed on all groups.

Results P1 and N1 cVEMP latencies and n1 and p1 oVEMP latencies were statistically significantly delayed in both the DM and DPN groups than the control. Also, amplitudes of P1-N1 and n1-p1 were statistically significantly lower in the DM and DPN groups than the control group. DPN patients had longer latencies and lower amplitude of the cVEMP and oVEMP response compared to diabetes patients without neuropathy. There was a statistically significant prevalence of BPPV in the DPN group compared to the control and DM groups. DPN included the following: 4 (13.3%) had vestibular pattern, 12 had polyneuropathic pattern, and 5 (16.7%) had vestibular and polyneuropathic pattern as regards the Gans Sensory Organization Performance (SOP) test.

Conclusion VEMP is considered a promising objective tool in the assessment of the vestibular end organ disorders in patients with type 2 DM with and without polyneuropathy. Diabetics with prominent diabetic polyneuropathy showed higher vestibular impairment than diabetics without DPN, which may increase the risk of falling.

Keywords Diabetes mellitus, Diabetic neuropathy, Vestibular evoked myogenic potential, Videonystagmography, Gans Sensory Organization Performance

*Correspondence:
Rabab Ahmed Koura
rababkoura@yahoo.com
Full list of author information is available at the end of the article

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Background

Diabetes mellitus type 2 is a metabolic disorder with various etiologies. It is distinguished by chronic hyperglycemia caused by abnormalities in carbohydrate, protein, and fat metabolism as a result of deficiency of insulin secretion and resistance [1]. The etiology of DM type 2 is not fully understood; however, some factors, such as environmental, genetic, and behavioral factors, can enhance the probability that a person will develop the disease [2].

Chronic hyperglycemia causes micro and macrovascular changes, resulting in sensory and motor system abnormalities which could lead to impaired balance control and increase the incidence of risk of falling [3]. In diabetic patients, microangiopathy causes ischemia of the vestibular structures and changes the fluid metabolism in the inner ear, resulting in vestibular hypofunction [4].

Diabetic neuropathy (DPN) is a prevalent consequence of diabetes, with an incidence rate of 8% in patients with new diagnoses and more than 50% in patients with long-standing disease [5]. The main pathological process causing nerve damage in DM is thought to be oxidative stress [6].

VEMP is defined as a short-latency response. Based on where the recording electrodes are placed, VEMP measurements are categorized as cervical vestibular evoked myogenic potential (cVEMP) or ocular vestibular evoked myogenic potential (oVEMP). The oVEMP reflects the contralateral utricle and superior vestibular nerve function, whereas the cVEMP represents the ipsilateral saccule and inferior vestibular nerve activity [7, 8].

Rationale

Up to 50% diabetic people suffer from neuropathy. Vestibular dysfunction is a prevalent disease in diabetic individuals due to ischemia of the vestibular end organs and lysis of the vestibular nerve myelin. The primary goals of this study are to assess the function of the vestibular system in patients with type 2 DM with polyneuropathy and compare the results to those of patients without polyneuropathy. The proposed study's findings may offer information regarding the timing of vestibular affection in the DM clinical process and aid in the efficient treatment of DM patients.

Aim of the work

The aim of the work is to compare the function of the vestibular end organs and vestibular nerve in patients with type 2 diabetes and polyneuropathy to that of people without polyneuropathy.

Methods

This is a comparative study. The study group composed of thirty type 2 diabetes mellitus patients with polyneuropathy and thirty patients with type 2 diabetes mellitus without polyneuropathy. Patients were collected from adults who attended to the internal medicine outpatient clinic, Beni-Suef University Hospital. The audiological assessment was performed at the Unit of Audio-Vestibular Medicine in Beni-Suef University Hospital. The control group composed of 30 healthy non-diabetic individuals whose blood sugar was < 100 mg/dl after doing fasting and < 140 mg/dl 2 h postprandial, with age and gender matching to the study group [9]. The study started on June 2020 and finished within 24 months.

All participants underwent the following procedures:

1. Vestibular evoked myogenic potential testing: using Eclipse EP15 by Interacoustics. It included the following:

   A. Cervical vestibular evoked myogenic potential
   B. Ocular vestibular evoked myogenic potential

2. Videonystagmography was used to assess the following: spontaneous nystagmus, gaze-evoked nystagmus, smooth pursuit testing, the saccade testing, positional tests, and positioning test (Dix-Hallpike test). Water caloric tests were carried out, and each one of the ears was irrigated with water at temperatures ranging from 30 to 44 °C. Canal weakness was determined using Jongkees' formula [10]. Canal results of more than 25% have been declared abnormal.

3. Gans Sensory Organization Performance (SOP) test [11]

   • The SOP test is a test of postural stability and balance function.
   • The SOP test has seven conditions.
     Every condition took 20 s to complete. In conditions 1 through 4, the Romberg was applied, the Clinical Test for Sensory Integration Balance (CTSIB) was used in conditions 5 and 6, and the Fukuda was done in condition 5.
     Scoring and interpretation were done as the following.
     Conditions 1 through 4 (Romberg and tandem Romberg) will be assigned normal, sway, or fall. Designate a further direction R or L if the person's achievement has been classified as a fall. Normal, sway, or fall will be given to conditions 5 and 6 (CTSIB). Condition 7 (Fukuda) will have a score of normal, sway, fall, right, or left.

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- Pattern interpretation:

  a. Normal patterns.

  Normal people should perform or finish all conditions.
  b. Vestibular patterns (three options).
  A fall on condition 6 (CTSIB foam with eyes closed) is the condition with the highest risk of uncompensated peripheral vestibulopathy (right/left/(turn, drift, spin > 30°) or fall (Fukuda stepping)).
  c. Polyneuropathic pattern.
  Each of the three elements of peripheral neuropathy (Romberg, CTSIB, and Fukuda) could be abnormal.

4. Motor and sensory nerve conduction study (for patients only).

Sensory and motor nerve conduction studies using surface electrode for ulnar, peroneal, and tibial nerves bilaterally was carried out in Neuro-Diagnostic & Research Center (NDRC), Beni-Suef University.

For each response, parameters such as absolute distal latency, amplitude (peak to peak), and conduction velocity were measured. The results are as follows: mild PN—a mild reduction in sensory response amplitude; moderate PN causes a decrease in the amplitude of sensory and motor responses; marked PN—a considerable decrease in the amplitude of sensory and motor nerve conduction responses.

Statistical analysis

The Statistical Package for Social Sciences (SPSS, version 23; SPSS Inc., Chicago, IL, USA) was used for the analysis. Descriptive statistics were computed for quantitative data. When comparing more than two groups with quantitative data, the one-way ANOVA test was employed, and when comparing categorical data, the chi-square test (χ^2) was utilized. Pearson correlation analysis was used to compute the correlation coefficient. *P*-values less than 0.05 are regarded as significant.

Results

Each study group consisted of 30 subjects. There were 16 males (53%) and 14 females (47%) in the control group, 17 (57%) males and 13 (43%) females in the DM group, and 17 (57%) males and 13 (43%) females in the DPN group. The age of the study group ranged from 21 to 60 years, with an average of 41.8 years and SD of 11.4 years in the control group; ranged from 28 to 58 years, with an average of 43.1 years and SD of 8.3 in the DM group; and ranged from 23 to 60 years, with an average of 46.1 years and SD of 9.5 years in the DPN group. The duration of disease ranged from 2 to 13 years. The comparison between 3 groups was non-significant as regards the age and gender and was significant as regards the duration of disease, whereas DPN patients had longer disease duration (Table 1).

Table 2 shows the comparison between the 3 groups as regards the c-VEMP parameters. Both DM group and DPN group patients had statistically significantly longer P1 and N1 latencies than the control group, and the DPN group had prolonged P1 and N1 latencies than the control and DM groups.

As regards the P1-N1 amplitude, both DM and DPN patients had significantly lower amplitude than the control group, and DPN patients had significantly lower amplitude than the DM patients. No statistically significant difference was found as regards the P1-N1 amplitude AR% between the two ears among the three groups.

However, c-VEMP was absent in the 2 ears of the DPN group.

Table 3 shows that n1 and p1 latencies were statistically significantly delayed in both DM and DPN groups than the control group in both ears. Also, n1-p1 amplitude was statistically significantly lower in the DM and DPN groups than the control groups. Also, in the comparison between the 3 groups as regards n1-p1 asymmetric ratio (AR) between the RT and LR ears, there was a highly significant difference between the DM and DPN groups and between the DPN and control groups.

Table 4 shows that 13 (43.3%) of patients in the DM group and 20 (66.7%) of patients in the DPN group were complaining of dizziness, and the comparison between the 3 groups was statistically significant.

Table 1 Demographic features of the groups

		Control $n=30$	DM $n=30$	DPN $n=30$	*P* value
Age (year)		21–60 41.8 ± 11.4	28–58 43.1 ± 8.3	23–60 46.1 ± 9.5	$P=0.230$ $P_1=0.252$ $P_2=0.592$ $P_3=0.092$
Duration of disease (years)		-	2–13 6.5 ± 2.5	3–15 8.3 ± 3.2	0.018*
Gender	Male	16 (53%)	17 (57%)	17 (57%)	0.956
	Female	14 (47%)	13 (43%)	13 (43%)	

DPN diabetic polyneuropathy, *DM* diabetes mellitus, *SD* standard deviation

P: Comparison between all groups

P_1: Comparison between the DM and DPN groups

P_2: Comparison between the DM and control groups

P_3: Comparison between the DPN and control groups

*P-values less than 0.05 are regarded as significant

Table 2 c-VEMP parameters among the control, DM, and DPN groups

	cVEMP	Control n = 30	DM n = 30	DPN n = 30	P value
RT ear	P1 (ms)	13.03–15.36 14.0 ± 0.7	13.44–16.9 14.3 ± 1.0	13.54–18.43 14.4 ± 4.2	$P_1 = 0.929$ $P_2 = 0.003$** $P_3 = 0.002$**
	N1 (ms)	22.33–24.21 23.5 ± 0.5	23.06–25.91 23.8 ± 0.6	23.06–27.56 23.1 ± 6.4	$P_1 = 0.493$ $P_2 = 0.003$** $P_3 = 0.004$**
	Amplitude (µV)	215.5–325.8 278.0 ± 33.0	119.88–314.96 237.77 ± 54.98	0–297.06 175.2 ± 65.7	$P_1 = 0.001$** $P_2 = 0.002$** $P_3 = 0.001$**
LT ear	P1 (ms)	13.0–15.9 14.1 ± 0.8	13.5–17.1 14.3 ± 0.9	13.4–17.7 15.0 ± 1.3	$P_1 = 0.790$ $P_2 = 0.004$** $P_3 = 0.002$**
	N1 (ms)	22.7–24.2 23.6 ± 0.4	23.0–26.1 24.7 ± 0.4	23.3–27.4 24.7 ± 1.3	$P_1 = 0.001$** $P_2 = 0.002$** $P_3 = 0.001$**
	Amplitude (µV)	209.9–315.7 277.7 ± 30.0	132.5–300.5 239.3 ± 59.0	95.9–300.7 178.5 ± 55.0	$P_1 = 0.001$** $P_2 = 0.002$** $P_3 = 0.001$**
Asymmetry ratio (AR)		0.01–0.2 0.05 ± 0.05	0–0.16 0.06 ± 0.04	0–1 0.11 ± 0.24	$P_1 = 0.240$ $P_2 = 0.789$ $P_3 = 0.150$

P_1: Comparison between the DM and DPN groups

P_2: Comparison between the DM and control groups

P_3: Comparison between the DPN and control groups

**P-values less than 0.05 are regarded as highly significant

Table 3 O-VEMP parameters among the control, DM, and DPN groups

	O-VEMP	Control n = 30	DPN n = 30	DM n = 30	P value
RT Ear	N1 (ms)	10–12.73 10.9 ± 0.8	10.31–15.05 11.8 ± 1.3	10.05–13.66 11.6 ± 1.0	$P_1 = 0.392$ $P_2 = 0.025$* $P_3 = 0.002$**
	P1 (ms)	15–18.64 16.3 ± 0.8	15.43–20.11 16.9 ± 1.2	15.33–18.32 16.7 ± 0.9	$P_1 = 0.493$ $P_2 = 0.014$* $P_3 = 0.012$*
	Amplitude (µV)	5.56–13.76 8.8 ± 1.9	1.55–10.98 5.0 ± 3.1	2.06–12.97 7.8 ± 2.8	$P_1 = 0.001$** $P_2 = 0.156$ $P_3 = 0.001$**
LT Ear	N1 (ms)	10.3–12.6 11.1 ± 0.6	10.7–18.5 12.7 ± 2.1	10.5–15.0 11.6 ± 1.1	$P_1 = 0.003$** $P_2 = 0.013$* $P_3 = 0.001$**
	P1 (ms)	15.0–17.9 16.2 ± 0.8	15.1–20.1 17.4 ± 1.4	15.3–19.1 16.9 ± 1.0	$P_1 = 0.091$ $P_2 = 0.016$* $P_3 = 0.001$**
	Amplitude (µV)	5.1–13.0 9.1 ± 2.2	1.5–11.1 5.4 ± 3.4	2.8–12.0 8.1 ± 2.9	$P_1 = 0.001$** $P_2 = 0.184$ $P_3 = 0001$**
Asymmetry ratio (AR) (%)		0.01–0.22 0.09 ± 0.05	0.02–0.72 0.2 ± 0.2	0.01–0.22 0.1 ± 0.1	$P = 0.001$** $P_1 = 0.001$** $P_2 = 0.887$ $P_3 = 0.001$**

P_1: Comparison between the DM and DPN groups

P_2: Comparison between the DM and control groups

P_3: Comparison between the DPN and control groups

**P-values less than 0.05 are regarded as highly significant

Table 4 Comparison between all groups as regarding dizziness complaint

		Control n = 30	DM n = 30	DPN n = 30	P value
Dizziness complaint	No	30 (100%)	17 (56.7%)	10 (33.3%)	0.001**
	Yes	0 (0%)	13 (43.3%)	20 (66.7%)	

**P-values less than 0.05 are regarded as highly significant

In Table 5, the videonystagmography results showed that oculomotor testing was normal in the 3 groups. None of the patients had gaze-evoked nystagmus. They had normal saccadic velocity, latency, and accuracy. Smooth pursuit testing demonstrated normal gain to both sides. Optokinetic nystagmus had normal slow phase velocity and symmetrical between the two sides.

In the DM group, 2 patients (6.7%) had right posterior canal BPPV, and 1 patient (3.3%) had right horizontal canal BPPV. In the DPN group, 6 patients (20%) had posterior canal BPPV (4 in the right ear and 2 in the left ear), and 5 patients (16.6%) had horizontal canal BPPV (3 in the right ear and 2 in the left ear). Chi-square test revealed a statistically significant prevalence of BPPV in the DPN group compared to the control and DM groups.

For caloric test, 2 patients (6.7%) of the DM group had right canal weakness and 4 patients (13.3%) in the DPN had canal weakness (3 in the right ear and 1 in the left ear). Chi-square test revealed a statistically significant prevalence of caloric weakness in the DPN group compared to the control and DM groups.

Table 6 shows a comparison between the 3 groups as regards Gans test. None of the control subjects had abnormal pattern, while only 2 patients (6.7%) of the DM patients had vestibular pattern, and the rest had normal pattern. On the other hand, only 9 patients (30%) of the DPN group had normal pattern, while 4 (13.3%) had vestibular pattern, 12 had polyneuropathic pattern, and 5 (16.7%) had vestibular and polyneuropathic patterns.

Table 7 shows a comparison between the 3 groups in studying the amplitude of sensory and motor responses of nerve conduction in the upper limb (UL) and the lower limb (LL). The amplitude was significantly lower in the

Table 6 Comparison between all groups as regards (Gans SOP) test patterns

Pattern	Control n = 30	DM n = 30	DPN n = 30	P value
Normal	30 (100%)	28 (93.3%)	9 (30%)	0.001**
Vestibular	0 (0%)	2 (6.7%)	4 (13.3%)	
Polyneuropathic	0 (0%)	0 (0%)	12 (40%)	
Vestibular and polyneuropathic	0 (0%)	0 (0%)	5 (16.7%)	

**P-values less than 0.05 are regarded as highly significant

DPN patients compared to either the control group or the DM patients. Abnormal nerve conduction study in the DPN group was mild in 13 (43.3%) patients, moderate in 15 (50%) patients, and marked in 2 (6.7%) patients.

Figures 1, 2, 3, and 4 show that there was no effect of disease duration on P1 and N1 latencies of cVEMP, while P1-N1 amplitude decreased significantly as the disease duration increased. For the O-VEMP, disease duration had a significant effect on the latency and amplitude, whereas the n1 and p1 latency increased, and the amplitude decreased as the disease duration increased.

Discussion

Diabetes type 2 is a metabolic disorder resulting from defects in insulin caused by a variety of etiologies [12]. Diabetes can lead to a number of symptomatology, one of which is balance abnormality (dizziness and falls), which is frequently reported by diabetic patients. Dizziness is a clinical manifestation of a disturbed or worsened spatial orientation [13]. Diabetic neuropathy is caused by microvascular complications that influence the peripheral nerves (motor and sensory), and it affects more than 50% of diabetic patients [14].

In the current study, we found that as regard the c-VEMP parameters, both DM group and DPN group patients had statistically significantly delayed P1 and N1 latencies than the control group, and the DPN group had longer P1 and N1 latencies than the DM group. Regarding the P1-N1 amplitude, both DM and DPN patients had significantly smaller amplitude than the control group,

Table 5 Comparison between all groups as regarding videonystagmography findings

Positional test	NAD	30 (100%)	29 (96.7%)	25 (83.3%)	0.238
	Horizontal canal BPPV	0 (0%)	1 (3.3%)	5 (16.6%)	
Dix-Hallpike test	NAD	30 (100%)	28 (93.3%)	24 (80%)	0.021*
	Posterior canal BPPV	0 (0)	2 (6.7%)	6 (20%)	
Water caloric test	NAD	30 (100%)	28 (93.3%)	26 (86.6%)	0.03*
	Unilateral canal paresis	0 (0)	2 (6.7%)	4 (13.3%)	

*P-values less than 0.05 are regarded as significant

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Table 7 Comparison between all groups as regards nerve conduction study results

	Control *n* = 60	DM *n* = 60	DPN *n* = 60	*P* value
Motor UL amplitude (mV)	5–10.2 8.23 ± 1.19	5.1–10.2 8.14 ± 1.25	4.8–9.5 6.77 ± 1.08	$P = 0.001$** $P_1 = 0.001$** $P_2 = 0.693$ $P_3 = 0.001$**
Motor LL amplitude (mV)	4.1–8 5.49 ± 1.05	4.3–7.8 5.42 ± 0.81	0.12–8.6 4.38 ± 2.43	$P = 0.001$** $P_1 = 0.001$** $P_2 = 0.802$ $P_3 = 0.001$**
Sensory UL amplitude (mV)	12–45 23.24 ± 7.85	13.5–44 22.57 ± 6.21	5.6–29.4 14.28 ± 5.50	$P = 0.001$** $P_1 = 0.001$** $P_2 = 0.579$ $P_3 = 0.001$**
Sensory LL amplitude (mV)	12.5–23 16.34 ± 2.61	12.9–22.8 16.17 ± 2.16	4–23.9 8.32 ± 2.71	$P = 0.001$** $P_1 = 0.001$** $P_2 = 0.701$ $P_3 = 0.011$**

P: Comparison between all groups

*P*₁: Comparison between the DM and DPN groups

*P*₂: Comparison between the DM and control groups

*P*₃: Comparison between the DPN and control groups

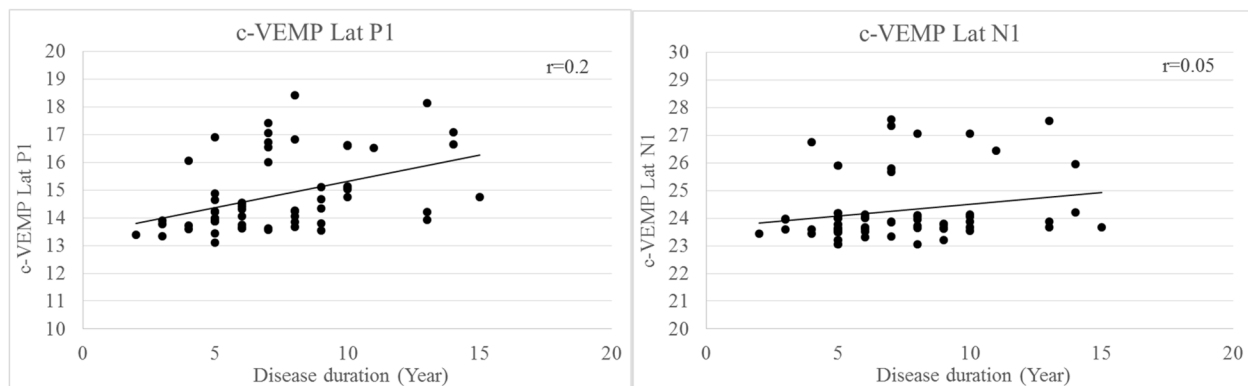**P-values less than 0.05 are regarded as highly significant

Fig. 1 Correlation between disease duration and c-VEMP P1 and N1latency (lat)

and DPN patients had significantly smaller amplitude than the DM patients. No statistically significant difference was found as regards the P1-N1 amplitude asymmetric ratio AR% between the two ears among the three groups (Table 2). Also, c-VEMP was absent in the 2 ears of the DPN group.

Similar to our results, Ren et al. [15] and Omar et al. [16] reported that patients with type 2 DM with polyneuropathy had longer c-VEMP P1 and N1 latencies, smaller P1 and N1 amplitudes, and lower P1-N1 amplitude AR% than people with type 2 DM without polyneuropathy. Such results of the current study suggest that neuropathy within the inferior vestibular nerve and the central

vestibular pathway is responsible for the c-VEMP reflex. The vestibular neuropathy is evidenced by the prolonged latency of the c-VEMP response. Moreover, the reduced c-VEMP amplitude is another manifestation of the inferior vestibular nerve neuropathy with possible saccular dysfunction as well. Consistent with our results, as per Agrawal et al. [17], patients with severe DPN have a 76% probability of developing vestibular dysfunction that could be due to neuropathy of the vestibular nerve.

In contrast to our findings, Kalkan et al. [18] demonstrated no significant difference between patients with type 2 DM with DPN and without DNP and control subjects as regards cVEMP P1 and N1 latencies. However,
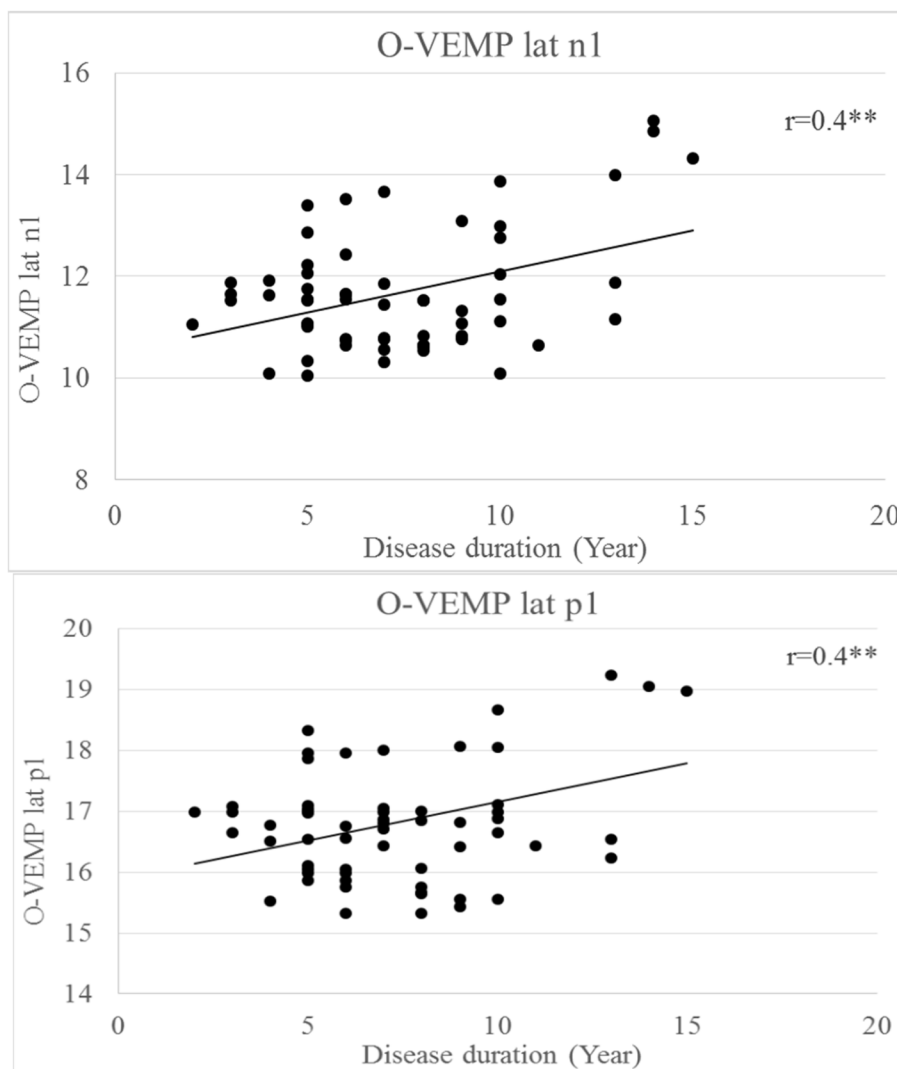
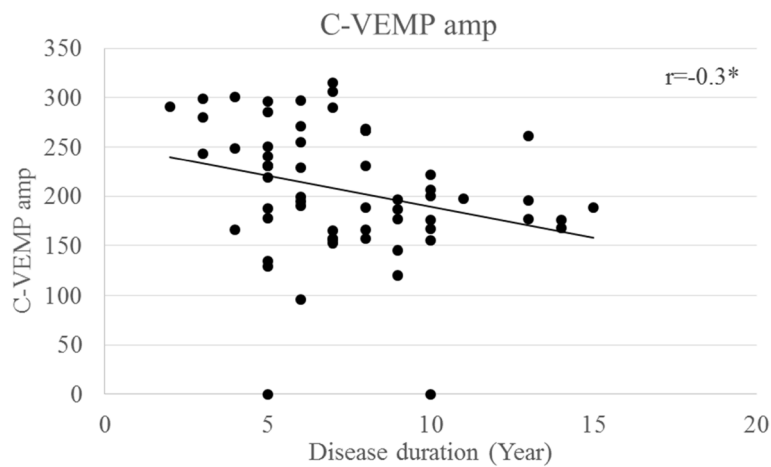**Fig. 2** Correlation between disease duration and O-VEMP lat n1 and p1 latency (lat)

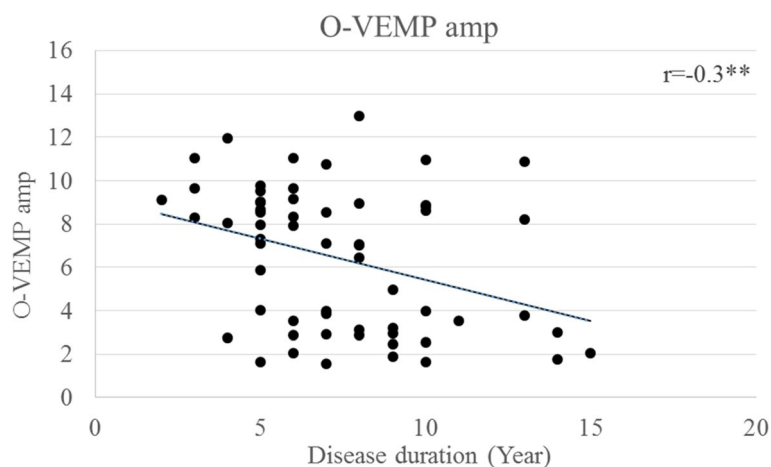**Fig. 3** Correlation between disease duration and c-VEMP amplitude (amp)

Fig. 4 Correlation between disease duration and O-VEMP amplitude (amp)

P1-N1 amplitude asymmetric ratio AR% was significantly higher in patients with type 2 DM with DPN or without DNP than the control group. Also, Zhang et al. [19] noticed no difference between the patients with type 2 DM and the control group regarding P1-N1 amplitudes.

Regarding o-VEMP parameters, n1 and p1 latencies were statistically significantly delayed in the DPN group than the DM group in both ears. Also, n1-p1 amplitudes were statistically significantly lower in the DPN group than the DM group. There was a highly significant difference between the DM and DPN groups and between the DPN and control groups as regards n1-p1 asymmetric ratio (AR) (Table 3). The O-VEMP results suggest neuropathic pathology within the superior vestibular nerve with possible utricular pathology as well.

The same oVEMP results in diabetic patients were reported by Zhang et al. [19] and El-seady et al. [20], while Ward et al. [21] stated that patients with DPN showed significant reduced n1 amplitude of oVEMP than the control group. On other hand, Minnar et al. [22] found no significant difference between cases with DPN and the control regarding latencies and amplitudes of oVEMP parameters.

Bayram [23] stated that Perez et al. [24] studied vestibular evoked potentials in an experimental model-induced type 2 DM and discovered that diabetic animals had significantly longer first wave latency and lower amplitude. As a result of these findings, the authors indicated that using an objective test in DM revealed vestibular disorder of the inner ear. VEMP testing was mostly used to investigate peripheral vestibular end organ abnormalities and to study the effect of DNP on vestibular system in diabetic patients. In cases of newly diagnosed disease, the occurrence of DNP is around 8%, while the percentage of DNP in DM with a long history is greater than 50% [5].

In the present study, we noticed that the DPN group had higher percentage of patients complaining of dizziness than the DM group (66.7% vs. 43.3% respectively) (Table 4). Insulin and glycemic rates in the blood have a significant impact on peripheral vestibular abnormalities. The greater these rates, the more likely the patient is to suffer from vestibular dysfunction, such as benign paroxysmal positional vertigo (BPPV), endolymphatic hydrops, and Ménière's disease [14].

As regards videonystagmography, we found that there was a statistically significant prevalence of BPPV and caloric weakness in the DPN group compared to the control and DM groups (Table 5). In accordance with our findings, Silva et al. [25] and Jáuregui-Renaud et al. [26] stated that BPPV is considered the most recurrent vestibular system disorder in patients with type 2 DM and explained that by the vascular role in the BPPV pathogenesis and frequency. Diabetes type 2 is linked to endothelial dysfunction in the inner ear, as well as probable labyrinthine ischemia, which promotes otoconial detachment and the development of BPPV [27].

Considering caloric findings, a recent study by Kuniyil et al. [28] found alterations in caloric test results which were significant when investigated in DM patients, while Di Nardo et al. [29] noticed no abnormal findings in electronystagmography (ENG) in type 2 DM patients with and without peripheral neuropathy. Overall, the horizontal canal and the superior vestibular nerve can be affected by the diabetes pathology similar to the cochlea, otolith organs, auditory nerve, and the inferior vestibular nerve.

As regards Gans SOP test patterns, we found that only two patients (6.7%) in the DM group had a vestibular pattern, while the rest had a normal pattern. Only 9 patients (30%) in the DPN group had a normal pattern, while

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4 (13.3%) had a vestibular pattern, 12 had a polyneuropathic pattern, and 5 (16.7%) had both vestibular and polyneuropathic patterns (Table 6). This could be explained by the fact that postural instability in DPN is caused by deficiencies in systems that work to maintain balance. Reduced proprioception feedback, combined with deterioration of the somatosensory, visual, and vestibular systems, causes postural instability and increased postural sway [30].

Dixit et al. [31] reported significant variations in eye-opened (EO) and eye-closed (EC) sway amplitude in the DPN group on firm foam conditions. Fahmy et al. [32] discovered that the DPN group had worse equilibrium scores, a lower Berg Balance Scale (BBS) balance score, and greater postural sway than the control group.

Agrawal et al. [17] used the Romberg Test of Standing Balance to collect data from adults with diabetes aged 40 and up. Because they had to attempt a step forward or open their eyes while standing on foam with their eyes closed, 54% of the diabetic individuals were found to be with vestibular impairment. Participants who had vestibular dysfunction and dizziness were 12 times more probably to fall than those who did not have either symptom.

In the current study, we discovered abnormal nerve conduction studies in the DPN group which were divided into mild in 13 (43.3%) patients, moderate in 15 (50%) patients, and marked in 2 (6.7%) patients. The amplitudes were significantly smaller in DPN patients than the DM group. Akaza et al. [33], when comparing the nerve conduction study results obtained from patients with type 1DM with that obtained from patients with type 2 DM, noticed that DPN appeared before symptoms like numbness and pain. Moreover, NCS can recognize peripheral nerve impairment even in the initial phases of illness and enable neuropathy to be quantified.

As regards the disease duration, we found that disease duration had no effect on P1 and N1 latencies of cVEMP, but P1-N1 amplitudes decreased significantly as disease duration increased. The disease duration had a significant effect on the latencies and amplitudes of the o-VEMP, as n1 and p1 latencies increased and amplitudes decreased as disease duration increased (Figs. 1, 2, 3, and 4).

Zhang et al. [19] noticed that o-VEMP and c-VEMP (n1, p1, P1, and N1 latencies respectively) had positive correlations with diabetes duration, However, El-seady et al. [20] found that there was no correlation between disease duration and o-VEMP latency. On the peripheral level of the vestibular system, diabetes may result in a reduction of type 1 hair cells in the saccule as well as demyelination of the vestibulocochlear nerve. There was an increase in lysosomes and lipid droplets, as well as an increase in extracellular matrix production, in the connective tissue of the utricle and saccule [34].

In the current work, we noticed that there was a significant prevalence of vestibular system impairment in the DPN group than the DM group, and this was confirmed by longer latencies and smaller amplitudes of oVEMP and cVEMP, high rate of abnormalities in videonystagmography results demonstrated by caloric weakness and presence of BPPV, and finally by Gans SOP test which revealed greater affection in vestibular and polyneuropathic patterns. We can deduce that vestibular system abnormalities are probably due to the angiopathy, neuropathy, or both.

Conclusions

Distinctive cochlear and vestibular end organ impairments were demonstrated in diabetic patients with DPN who showed more severe impairment than diabetic patients without polyneuropathy. Vestibular impairment may increase the risk of fall in individuals with diabetes with polyneuropathy. VEMP is considered a promising objective tool in the assessment of the vestibular end organ disorders in patients with type 2 DM with and without polyneuropathy. More research should be done with larger clinical groups in well-defined study participants.

Declarations

Ethics approval and consent to participate
All participants received their consent after the study was approved by the Beni Suef University's research ethics committee (approval no: FMBSUREC/03052020).

Consent for publication
Not applicable.

Competing interests
Dr Mohamed El badry is a co-author of this study and an Associate Editor for the journal. He has not involved in handling this manuscript during the submission and review processes. The rest of the authors have no conflict of interest to declare.

Author details
[1]Ear, Nose, Throat Department, Audiovestibular Unit, Faculty of Medicine, Beni-Suef University, Beni- Suef, Egypt. [2]Ear, Nose, Throat Department,

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Audiovestibular Unit, Faculty of Medicine, Minia University, Minia, Egypt.
[3]Internal Medicine Department, Faculty of Medicine, Beni-Suef University,
Beni- Suef, Egypt. [4]Neurophysiology Department, Faculty of Medicine, Beni-
Suef University, Beni- Suef, Egypt.

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