Review Article
http://mjiri.iums.ac.ir
Medical Journal of the Islamic Republic of Iran (MJIRI)

Med J Islam Repub Iran. 2026 (15 Jul);40.75. https://doi.org/10.47176/mjiri.40.75

Mechanisms of Vestibular System Damage in Diabetes Mellitus: Implications for Benign Paroxysmal Positional Vertigo Pathogenesis and Recurrence

Seyedeh Nazanin Hajjari[1], Fatemeh Jafarlou[1]*

Received: 14 Dec 2025 Revised: 14 Jun 2026 Accepted: 20 Jul 2026 Published: 15 Jul 2026

Abstract

Background: Diabetes mellitus (DM) is increasingly recognized as an independent risk factor for falls and balance disorders in elderly populations. While diabetic neuropathy and retinopathy are well-established complications, the role of vestibular system dysfunction has been underappreciated. Benign paroxysmal positional vertigo (BPPV), the most common peripheral vestibular disorder, demonstrates markedly increased prevalence and recurrence in diabetic patients, yet the underlying mechanisms remain incompletely understood. Therefore, the aim of the present review was to investigate the mechanism and pathophysiology of vestibular system damage in diabetes and its association with increased risk of BPPV recurrence.

Methods: We conducted a narrative review by searching PubMed, Scopus, and Web of Science databases using keywords related to diabetes mellitus, hyperglycemia, vestibular system, and BPPV. Our initial search yielded 847 articles; after screening and manual reference review, 12 studies (4,937 total participants) meeting inclusion criteria were selected for detailed analysis. Study designs included histopathological analyses, cross-sectional studies, retrospective cohorts, and one randomized controlled trial.

Results: Evidence identifies four convergent mechanisms: selective saccular hair cell loss, nerve demyelination, disrupted calcium/vitamin D metabolism causing otoconia fragility, and microangiopathy. DM independently predicts BPPV onset (OR 1.5–2.1) and recurrence (>50% vs. 27%), with VEMP latencies correlating with HbA1c levels.

Conclusion: Diabetes induces multifactorial vestibular damage, significantly increasing BPPV occurrence and recurrence. Management requires metabolic optimization, vestibular screening with VEMP, vitamin D supplementation, and integration of vestibular rehabilitation.

Keywords: Diabetes mellitus, Hyperglycemia, Vestibular system, Benign paroxysmal positional vertigo, Vestibular evoked myogenic potentials

This work has been published under CC BY-NC-SA 4.0 license.

Copyright© Iran University of Medical Sciences

Cite this article as: Hajjari SN, Jafarlou F. Mechanisms of Vestibular System Damage in Diabetes Mellitus: Implications for Benign Paroxysmal Positional Vertigo Pathogenesis and Recurrence. *Med J Islam Repub Iran.* 2026 (15 Jul);40:75. https://doi.org/10.47176/mjiri.40.75

Introduction

Diabetes mellitus (DM) is a global health crisis affecting over 500 million people, with type 2 (T2DM) accounting for 90% of cases; beyond classic complications, it significantly impairs balance and increases fall risk (1) and increases fall risk through both neuropathy and underappreciated vestibular dysfunction (2). Benign paroxysmal positional vertigo (BPPV) arises from pathological displacement of calcium carbonate crystals (otoconia) from the utricular macula into the semicircular canals (3).

Although BPPV is mechanically treatable using canalith repositioning procedures (CRP) (4), its high recurrence rate poses a significant challenge. Growing evidence

Corresponding author: Dr Fatemeh Jafarlou, Jafarlouf@tbzmed.ac.ir

1. Department of Audiology, Faculty of Rehabilitation Sciences, Tabriz University of Medical Sciences, Tabriz, Iran

Key Messages:

↑What is "already known" in this topic:

Diabetes mellitus is associated with falls and balance disorders in older adults. While neuropathy and retinopathy are well-recognized complications, vestibular dysfunction has received less attention. Benign paroxysmal positional vertigo (BPPV) appears more frequent in diabetic patients, but its underlying mechanisms and clinical implications remain unclear.

→What this article adds:

This review clarifies multifactorial mechanisms linking diabetes to vestibular damage, including hair cell loss, microangiopathy, demyelination, and calcium dysregulation. It confirms diabetes as an independent predictor of BPPV onset and recurrence, underscoring the need for metabolic control, vestibular screening, and preventive strategies.

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

shows markedly elevated BPPV prevalence and recurrence in diabetic patients (5–9) , suggesting a direct link between metabolic dysfunction and vestibular pathology.

Systematic reviews identify DM as an independent risk factor for BPPV (OR 1.5–2.1) (10, 11). These reviews have largely focused on epidemiological associations rather than mechanistic understanding. The precise cellular and molecular mechanisms by which chronic hyperglycemia and hyperinsulinemia damage the inner ear remain incompletely characterized (12, 13). Furthermore, while diabetic patients demonstrate dramatically higher BPPV recurrence rates (>50%) compared to non-diabetic individuals (approximately 27%) (6, 9), the optimal management strategies for this high-risk population have not been systematically evaluated. Critical questions remain: Which specific vestibular structures are most vulnerable to diabetic damage? Can subclinical vestibular dysfunction be detected before symptomatic BPPV develops? What role do comorbid conditions such as hypertension, hyperlipidemia, and vitamin D deficiency play in modulating this risk (5, 14–16)?

This narrative review synthesizes evidence on the mechanisms of diabetic vestibular damage, focusing on BPPV pathogenesis and recurrence. This review advances beyond previous work by: (1) integrating histopathological evidence (13,17–19) with electrophysiological findings (20–22) and clinical outcomes (6, 9); (2) highlighting the underappreciated role of hyperinsulinemia mediated through insulin receptors in the endolymphatic sac (9, 13); (3) synthesizing evidence on vestibular evoked myogenic potentials (VEMP) testing for early detection of subclinical vestibular neuropathy (20, 21); (4) critically evaluating multimodal management strategies including vestibular rehabilitation therapy (23); and (5) systematically addressing contributions of comorbid conditions as modulating factors (5, 14–16, 24). Our specific aims are to: (1) elucidate cellular, molecular, and vascular mechanisms underlying diabetic vestibulopathy; (2) evaluate electrophysiological and clinical assessment modalities; (3) analyze risk factors specific to diabetic populations; and (4) propose evidence-based, multimodal management strategies.

Methods

Study Design and Rationale

A narrative approach was adopted to integrate diverse evidence ranging from histopathological to clinical data. This design was chosen due to significant study heterogeneity, which precluded a formal meta-analysis.

Search Strategy

We conducted a systematic literature search in three major bibliographic databases (PubMed/MEDLINE, Scopus, and Web of Science) from January 2000 to January 2025. The search strategy combined MeSH terms and keywords: (“diabetes mellitus” OR “hyperglycemia”) AND (“vestibular system” OR “vestibular dysfunction”) AND (“benign paroxysmal positional vertigo” OR “BPPV”) AND (“vestibular evoked myogenic potentials” OR “VEMP”).

Screening and Selection Process

Our initial database search identified 847 articles. After removal of 312 duplicates, 535 unique articles underwent title and abstract screening performed independently by two reviewers. Of these, 487 were excluded as not relevant, leaving 48 articles for full-text review. An additional 36 articles were excluded after full-text review for the following reasons: not focused on DM and vestibular dysfunction (n=18), review articles without primary data (n=8), insufficient methodological detail (n=6), and non-English language (n=4). This resulted in 12 studies meeting all inclusion criteria. Figure 1 shows the flow diagram of the study selection process.

Inclusion Criteria

(1) Original research (observational, clinical trials, or histopathology), (2) Human participants with diagnosed DM (Type 1 or 2), (3) Evaluation of vestibular function or BPPV, (4) Peer-reviewed, English-language articles.

Exclusion Criteria

(1) Reviews, editorials, or case reports (n < 5), (2) Gestational or secondary diabetes, (3) Animal-only studies, (4) Unpublished data or abstracts.

Data Extraction

Two reviewers independently extracted data using a standardized form, including study design, sample characteristics, assessment methods, BPPV criteria, main outcomes, and limitations.

Quality Assessment

While no formal risk-of-bias tools were used due to study heterogeneity, we qualitatively assessed methodological rigor. Criteria included sample size adequacy, use of control groups, adjustment for confounders, and reporting transparency.

Results

Study Characteristics

Table 1 summarizes 12 studies (4,937 participants), including histopathological (13, 18), 6 cross-sectional (8, 21, 22, 25–27), 3 retrospective cohort (6, 9, 14), and 1 randomized controlled trial (23). While ten studies evaluated DM-BPPV prevalence and treatment outcomes (6,8,9,14,23),two specifically analyzed saccular histopathology in diabetic temporal bones (13, 18).

PRIMARY OUTCOME: Mechanisms of Vestibular System Damage

Epidemiological Profile and Diabetes as a Key Risk Factor

Type 2 diabetes affects 9.3% of the US population and by 2050 is predicted to affect 1 in 3 persons (28). Animal models demonstrate that diabetes predominantly affects the saccule, causing type 1 hair cell loss (17). Clinical evidence confirms that utricular and saccular function is markedly disturbed in diabetic individuals compared to healthy controls (21). Potentially facilitating otoconia de-

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

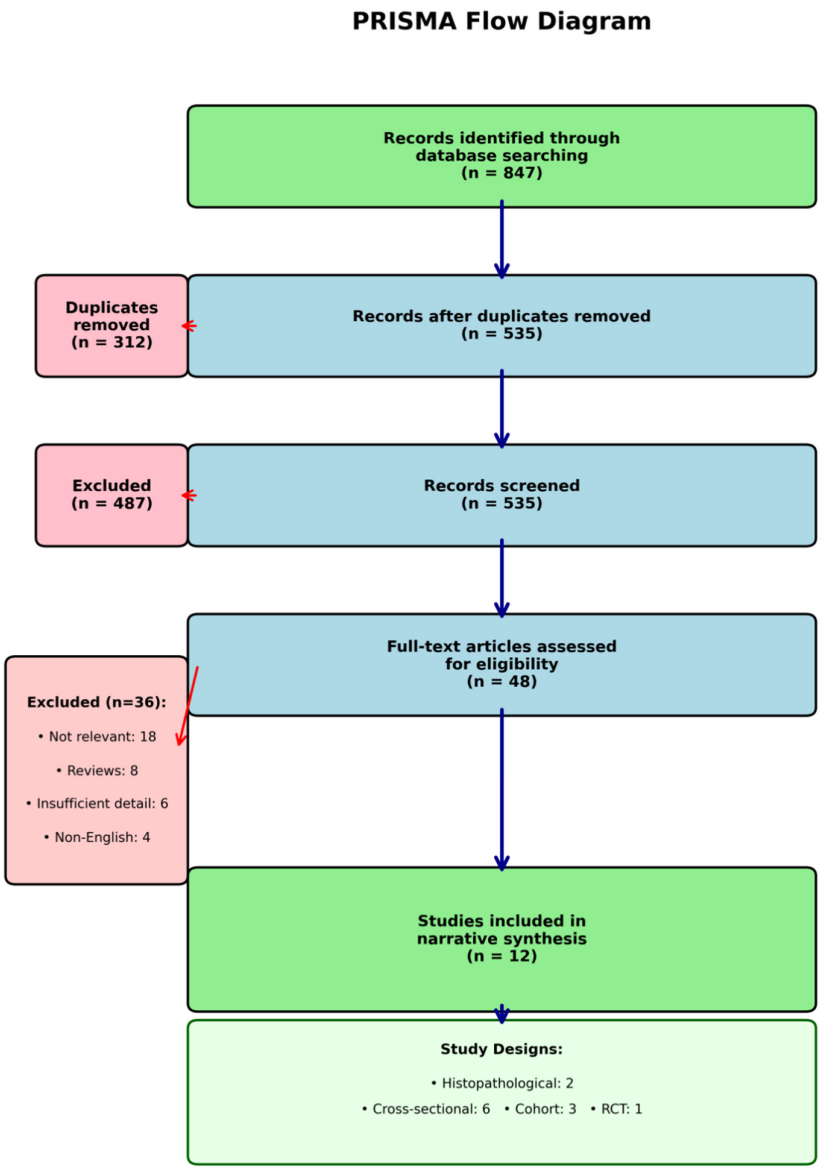

Figure 1. PRISMA flow diagram shows study selection process. From 847 initial records, 12 studies (4,937 participants) were included after removing duplicates and applying inclusion/exclusion criteria. This figure is original.

tachment and BPPV (4,29). Consequently, diabetes serves as a significant independent factor in BPPV pathology rather than a mere comorbidity (7).

Increased Prevalence and Risk of Onset

Large retrospective studies confirm that BPPV prevalence is higher in T2DM patients than in non-diabetic counterparts (7). establishing diabetes as an independent predictor regardless of age. One study reported BPPV in 46% of T2DM patients compared to 37% in non-diabetics (14), significantly exceeding general population rates (8). Hyperinsulinemia and hyperglycemia are significant risk factors;specifically, high insulin receptor density in the endolymphatic sac makes the inner ear highly sensitive to plasma insulin fluctuations (9). Moreover, diabetes rarely occurs alone; it frequently coexists with other metabolic and cardiovascular risk factors including hypertension (HTN), hyperlipidemia (HLD), and obesity. These conditions—themselves known as risk factors for BPPV—are very common in the diabetic population (15).

Diabetes as a Major Determinant of BPPV Recurrence

Clinical evidence identifies diabetes as a strong, independent predictor of BPPV recurrence following successful initial treatment. Systematic reviews and meta-analyses consistently report high odds ratios (ORs) for

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]
[DOI: 10.47176/mjiri.40.75]

Table 1. Summary of 12 included studies (2 histopathological, 6 cross-sectional, 3 cohort, 1 RCT) examining vestibular dysfunction in diabetes mellitus. Abbreviations defined in table footnote.

Study	Study Design	Participants (N)	Age Range	Key Findings
Bazoni et al. 2020	Cross-sectional	Elderly with DM (109)	>65 years	Association between BPPV, bone mineral density, and vitamin D deficiency in elderly with DM
Cohen et al. 2004	Cross-sectional	BPPV patients (176)	20-88 years	Prevalence of DM unusually high in BPPV patients (14% vs 4% general population)
D'Silva et al. 2017	Cross-sectional	DM, BPPV, controls (71)	40-65 years	BPPV and DM independently affect otolith function without cumulative effect
D'Silva et al. 2016	Cohort	Type 2 DM with HTN (3933)	>18 years	Hypertension mediates pathway by which diabetes affects vestibular system
D'Silva et al. 2017	Cohort	BPPV with/without DM (50)	40-80 years	No difference in CRP efficacy between diabetic and non-diabetic BPPV patients
Kocdor et al. 2016	Histopathology	Type 2 DM vs controls (63)	18-95 years	Selective type I hair cell loss in saccule, vestibulocochlear nerve demyelination
Konukseven et al. 2014	Cross-sectional	Diabetic, prediabetic, controls (91)	Mean 43.9-46.4 years	Prolonged VEMP latencies indicating subclinical vestibular neuropathy
Degerman et al. 2013	Histopathology	Human saccule analysis	Adult	Type I hair cell vulnerability to glucose toxicity due to glucose uptake differences
Ward et al. 2015	Cross-sectional	Type 2 DM vs controls (50)	>50 years	Poorer vestibular function in both SCC and otolith organs in diabetes
Webster et al. 2015	Cohort	Idiopathic BPPV (72)	18-65 years	Both hyperglycemia and hyperinsulinemia are risk factors for BPPV recurrence.
Jeong et al. 2013	Cross-sectional	BPPV vs controls (292)	Mean 60-61 years	Association between BPPV and decreased serum vitamin D levels
Abu Shaphe et al. 2023	RCT	Type 2 DM with BPPV (30)	40-65 years	VRT showed a trend toward greater improvement compared to the Epley maneuver in diabetic BPPV

Abbreviations: DM = Diabetes Mellitus; BPPV = Benign Paroxysmal Positional Vertigo; VEMP = Vestibular Evoked Myogenic Potentials; VRT = Vestibular Rehabilitation Therapy; CRP = Canalith Repositioning Procedure; SCC = Semicircular Canal; HTN = Hypertension; RCT = Randomized Controlled Trial; HbA1c = Hemoglobin A1c

recurrence in diabetic patients (10, 11). Long-term follow-up studies showed that the recurrence rate of BPPV in patients with coexisting BPPV and DM may be higher than 50 percent (6). This suggests that while CRP resolves mechanical detachment, they do not address the underlying metabolic factors. Consequently, persistent otoconia instability creates a continuous risk for repeated detachment and recurrence. Importantly, this risk is not fixed but dynamically differs based on the quality of metabolic control. In particular, elevated hemoglobin A1c (HbA1c), an established indicator of long-term glycemic control, as well as hyperinsulinemia, have been independently correlated with an increased probability of disease recurrence (9).

Molecular and cellular pathophysiology of diabetic vestibulopathy

While the precise pathophysiology is not fully understood, the vestibular organs are vulnerable due to high metabolic demands and dependence on endolymphatic fluid balance. So, the vestibular system is highly susceptible to the vascular and neuropathic complications of diabetes (12, 14). While microangiopathy affects broader labyrinthine circulation (7, 10, 30), direct vascular changes in sensory maculae remain unobserved in human models (13, 18). Instead, pathology is characterized by selective degeneration of saccular type I hair cells, likely due to cell-specific glucose toxicity and inadequate metabolic regulation rather than direct microvascular obstruction (13, 18). In other words, cells that are unable to limit glucose influx under hyperglycemic conditions appear to be partly prone to glucose toxicity and damage. Glucose uptake in most tissues is regulated through cell surface glucose transporters (31). There are multiple glucose isoforms, each of which exhibits distinct kinetic characteristics. However, the specific glucose transporter isoforms expressed in vestibular neuroepithelial cells have not been fully elucidated, although existing evidence indicates that the saccule is a target of insulin-mediated signaling pathways (13). The increased susceptibility of the inner ear in diabetes is thought to be because of multiple factors including labyrinth-specific microangiopathic, neuropathic, and metabolic-osteogenic insults. Experimental animal studies have shown that metabolic stress induced by hyperglycemia can lead to the loss of type 1 hair cells in the saccule (7) and demyelination of the vestibulocochlear nerve (19). Additionally, structural changes, such as the excessive production of extracellular matrix and increased lysosome activity and lipid droplets in the connective tissue of the utricle and saccule, have been documented (32). In accordance with these findings, Yoda et al. analyzed 28 temporal bones from 14 patients with type 2 dia-

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

betes and compared them with 56 normal temporal bones using histopathology. Their results indicated an increased frequency of cup-shaped and free-floating deposits within the lateral and posterior semicircular canals among patients with type 2 diabetes relative to healthy control temporal bones (33).

Diabetic Microangiopathy and Ischemic Vestibular Neuropathy

The inner ear's blood supply, derived from the labyrinthine artery, lacks collateral support, making its microcirculation uniquely susceptible to diabetic microvascular pathology where local damage remains uncompensated. The molecular mechanisms underlying diabetic microangiopathy—the core of the pathologic process—is well characterized: 1: Increased oxidative stress: Chronic hyperglycemia saturates metabolic pathways, increasing polyol pathway activity andthe generation of reactive oxygen species (ROS), leading to oxidative damage to cellular DNA and lipid membranes (30). 2: formation and accumulation of advanced glycation end-products (AGEs)-non-enzymatic glycation of proteins results in the formation of AGEs, which accumulate in the basilar membranes of cochlear and vestibular capillaries. This process causes thickening and stiffness of the microvascular walls and restricts the exchange of nutrients and oxygen and promotes chronic inflammatory activity through the receptor for AGE (RAGE) (10). These microvascular alterations produce ischemia in critical structures of the inner ear. The dark cellular region - responsible for endolymph generation and maintenance of ionic balance (K+ gradient)- undergoes metabolic dysfunction and ultimately disrupts the ionic composition of the endolymph (7).

Metabolic Bone Disorders and Otoconia: The Role of Calcium Homeostasis

Otoconia are composed of calcium carbonate biocrystals and play a main role in the pathophysiology of BPPV (34). The integrity and maintenance of otoconia are intrinsically linked to systemic calcium homeostasis and bone metabolism. This regulatory system is significantly compromised by diabetes mellitus. A central element of this impairment is the co-occurrence of severe vitamin D deficiency with both diabetes and BPPV (16, 35). Vitamin D is not only essential for calcium absorption at the systemic level but is also critical for the local homeostatic regulation of Ca2+ ions in the endolymphatic sac (36). Vitamin D deficiency leads to defective or incomplete organic matrix synthesis, compromising otoconia mineralization. This results in the formation of fragile crystals prone to detachment from the utricle (24). This pathology is further exacerbated by the association of diabetes with osteoporosis and high bone turnover (5), as otoconiashare structural and biochemical similarities with bone tissue (37). Elevated serum Otolin-1 (sensitivity 78%, specificity 82% at >2.5 ng/mL) provides molecular evidence of ongoing otoconia degeneration in BPPV patients (25). However, data on its diagnostic performance specifically within diabetic populations are still limited and require further investigation. The relationship between insulin resistance and vitamin D deficiency is bidirectional: insulin resistance impairs hepatic 25-hydroxyvitamin D synthesis and increases vitamin D sequestration in adipose tissue, while vitamin D deficiency worsens insulin sensitivity by reducing insulin secretion from pancreatic β-cells (38). This creates a vicious cycle that further compromises calcium homeostasis and otoconia integrity in diabetic patients.

Hypertension as a Precipitating and Mediating Factor

The elevated prevalence of BPPV in individuals with diabetes is not merely due to the coexistence of two common chronic diseases. In fact, the co-presence of hypertension acts as a synergistic mediator, making this association statistically stronger and more significant. When a patient presents with both diabetes and hypertension, the two diseases concurrently affect the inner ear and can ultimately precipitate or exacerbate BPPV. Vascular damage from hypertension acts synergistically with diabetic microangiopathy. That is, both are more deleterious when they occur simultaneously. Diabetic microangiopathy occurs due to the accumulation of AGEs and oxidative stress in diabetes (39). The combination of these two factors results in a severe and rapid reduction in perfusion to the inner ear, which causes ischemic damage to the utricle epithelium and its nerve fibers (37). Emerging literature indicates that hypertension is a significant determinant in the recurrence of BPPV. In patients with hypertension, the recurrence rate of this disease has been reported to exceed 55 percent (5). Figure 2 shows a schematic representation of the multifactorial pathways linking chronic hyperglycemia to vestibular structural defects and BPPV recurrence.

The Role of Hyperlipidemia and Metabolic Syndrome

While diabetes and hypertension have been well-characterized as risk factors for BPPV, the contribution of hyperlipidemia and broader metabolic syndrome requires attention. Recent studies indicate that the prevalence of hyperlipidemia among BPPV patients is significantly elevated compared to the general population. Meta-analyses show hyperlipidemia doubles BPPV recurrence risk (OR 2.347) (40),with recurrence rates reaching 67.80%—the highest among metabolic comorbidities (6). This disparity may be attributed to microvascular damage linked to dyslipidemia, which can result in displacement or dysfunction of otoliths. Elevated serum lipids can alter endolymph viscosity and ionic composition, disrupting the delicate biomechanical properties required for normal otolith function. Additionally, lipid peroxidation products generated under conditions of dyslipidemia can damage the organic matrix of otoconia, compromising their structural integrity and increasing susceptibility to detachment (41). Metabolic syndrome—the constellation of insulin resistance, obesity, hypertension, and dyslipidemia—creates a synergistic pro-inflammatory and pro-oxidative milieu that amplifies vestibular damage beyond the effects of any single component (10, 15). Patients with metabolic syndrome demonstrate higher BPPV recurrence rates compared to those with diabetes alone, suggesting that

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

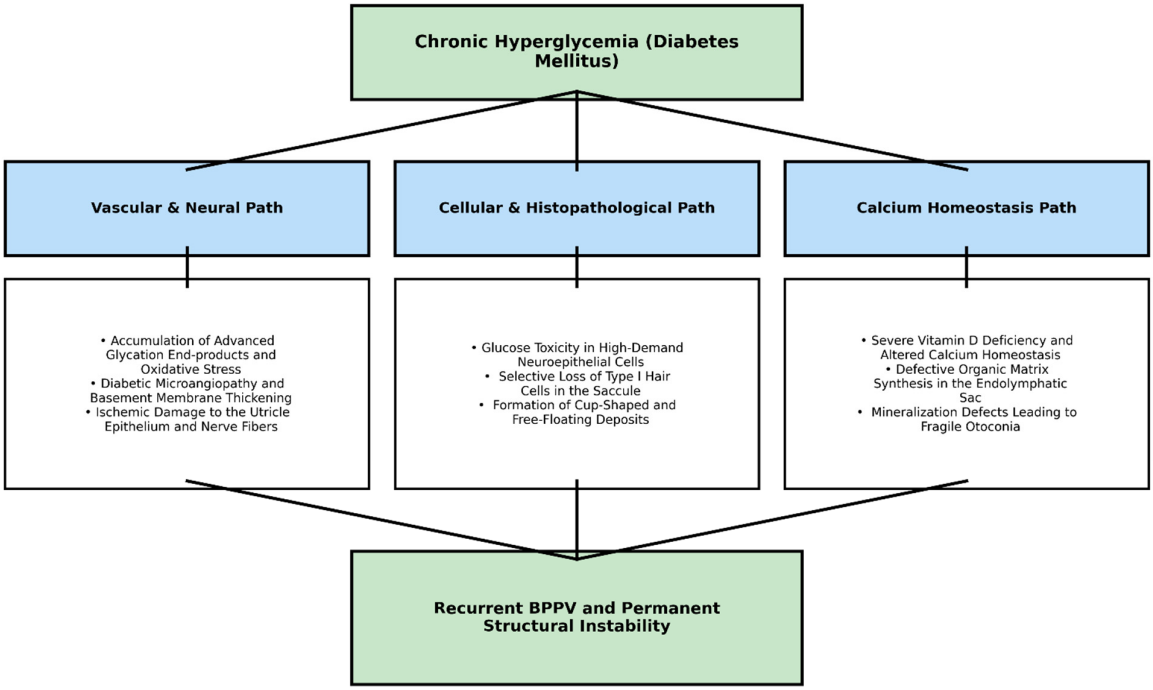

Figure 2. Schematic representation of three convergent pathways linking chronic hyperglycemia to BPPV recurrence: vascular/neural, cellular/histopathological, and calcium homeostasis pathways. Thisfigure is original.

comprehensive metabolic management addressing all components may be necessary to optimize outcomes.

Auditory and Vestibular Assessment

Objective electrophysiological tests provide quantitative and rigorous evidence of vestibular pathology associated with diabetes. Videonystagmography (VNG) represents a comprehensive battery of tests used to evaluate the vestibular system and central pathways controlling eye movements. VNG employs infrared cameras mounted in goggles to precisely record eye movements, eliminating the artifacts associated with traditional electronystagmography (ENG) (25). The VNG battery typically consists of several components: (1) oculomotor testing, which assesses saccades, smooth pursuit, and optokinetic nystagmus to evaluate central vestibular pathways and cerebellar function; (2) positional testing, which involves placing the patient in various head and body positions to provoke nystagmus characteristic of BPPV or central lesions; and (3) caloric testing, which involves irrigating each ear with warm and cool air or water to assess horizontal semicircular canal function and detect unilateral weakness (26). In diabetic populations, VNG abnormalities may include impaired smooth pursuit (suggesting cerebellar involvement), spontaneous or positional nystagmus (indicating peripheral vestibular dysfunction), or unilateral caloric weakness (reflecting damage to the lateral canal or superior vestibular nerve) (25, 26).

Although diabetic hearing loss is well-documented, vestibular studies remain limited. Gawron et al. reported central ENG abnormalities, including impaired optokinetic responses and positional nystagmus, in 95 T2DM patients (25). Conversely, Klagenberg et al. identified peripheral vestibular dysfunction in 60% of their cohort (18/30 cases), suggesting a significant peripheral involvement in diabetes (26). Furthermore, Rigon et al., in their analysis of a cohort of 19 individuals with type 2diabetes, observed some ENG changes in 7 cases (37%), all of whom exhibited definite manifestations of peripheral vestibular dysfunction. These initial findings suggested the need for more precise tools such as VEMP (27).

Electrophysiological Assessment of Otolith Neuropathy

VEMPs are the most sensitive modality for detecting subclinical diabetic vestibular neuropathy. (Ocular VEMP)oVEMP assesses the utricle/superior nerve, while (cervical VEMP) cVEMP evaluates the saccule/inferior nerve. The evaluation of otolith function in the saccule with cVEMP and in the utricle with oVEMP is a major advancement, analogous to the advent of the caloric test, because the findings of these tests can render the diagnosis and prognosis of many vestibular disorders more accurate and reliable (42).

BPPV diagnosis relies on provocative maneuvers to induce canal-specific nystagmus. The Dix-Hallpike maneuver, the gold standard for posterior canal BPPV, involves moving the patient to a supine-head-extended position to elicit torsional upbeat nystagmus (3, 4). Similarly, the supine roll test identifies horizontal canal involvement, with nystagmus characteristics differentiating canalithiasis

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

from cupulolithiasis (3). Systemic assessment is crucial in diabetic patients to detect subclinical multi-canal involvement (4).

Consistently, diabetic patients show prolonged cVEMP (P13, N23) and oVEMP (N10, P15) latencies compared to controls (36,43). Konukseven et al. reported significantly longer latencies in T2DM versus prediabetic and control groups (P<0.001) (21). The authors further observed that the latencies of both cVEMP and oVEMP waves exhibited a positive correlation with glycosylated hemoglobin (HbA1c) levels. In a similar vein, Kamali and colleagues documented prolonged p1 and n2 latencies among patients with diabetic neuropathycompared to normal subjects. These latencies indicate a deceleration in nerve conduction velocity, which is a hallmark of vestibulopathy/diabetic neuropathy (20). Moreover, D'Silva et al. reported a significant association between longer p1 and n1 latencies and higher HbA1c levels (P<0.03) (22). Kalkan et al. also reported that there was a statistically significant difference in the amplitudes of cVEMPs and oVEMPs between polyneuropathy patients (lower amplitudes) and control groups (higher amplitudes) (44). This finding suggests a decrease in excitability or complete degeneration of saccule hair cells or nerve terminals (45).

In the context of diabetic vestibulopathy,VEMP emerges as the primary electrophysiological assessment modality for several reasons. First, VEMP specifically evaluates otolith organ function (saccule via cVEMP, utricle via oVEMP), which are the structures most vulnerable to diabetic damage and most directly relevant to BPPV pathogenesis (13, 17, 21). Second, VEMP testing can detect subclinical abnormalities (prolonged latencies, reduced amplitudes) in asymptomatic diabetic patients, enabling early detection before symptomatic BPPV develops (20, 21). Third, VEMP parameters—particularly wave latencies—show strong correlation with metabolic control markers (HbA1c), providing both diagnostic and prognostic information (21, 22). Fourth, VEMP is relatively quick, non-invasive, and well-tolerated compared to rotational chair testing or other comprehensive vestibular batteries (42).

However, VEMP should not be used in isolation. Comprehensive vestibular assessment in diabetic patients should employ a tiered approach: (1) clinical examination including Dix-Hallpike and positional tests (essential for BPPV diagnosis) (3); (2) VEMP testing (primary electrophysiological tool for otolith assessment) (42); (3) caloric testing or vHIT (to evaluate semicircular canal function, particularly in symptomatic patients or when central pathology is suspected) (46, 47); and (4) VNG with oculomotor battery (when central vestibular or cerebellar involvement is suspected based on atypical nystagmus patterns or neurological symptoms) (25, 26). This hierarchical approach balances diagnostic yield with practical constraints of time and resources in clinical settings.

Semicircular Canal Function and Vestibular-Ocular Reflex Integration

Assessing semicircular canals and the vestibulo-ocular reflex (VOR) complements VEMP metrics by utilizing high-acceleration Video Head Impulse Tests (vHIT) and low-frequency Caloric irrigation (46, 47). In diabetes, these interpretations are complex as otolith damage often precedes or exceeds semicircular canal impairment (48, 49). Consequently, vHIT function and lateral canal VOR gain often remain normal in asymptomatic diabetic patients (50, 51). This finding was corroboratedby Ward et al.; they observed that the susceptibility to impairment was higher for the superior and lateral semicircular canals, as well as the utricle and saccule, while the posterior semicircular canal remained relatively unaffected. The authors further hypothesized that this observation might be attributable to the longer and more narrow bony canal traversed by the superior vestibular nerve in contrast to the innervation pathway of the posterior semicircular canal (17). In addition, in a recent study, Jáuregui-Renaud et al. reported that the utricle response to unilateral centrifugal stimulation was reduced in type 2 diabetic patients. This reduction was observed compared to controls. Interestingly, horizontal canal function was still preserved in these patients (P=0.01). They argued that the discrepancy in the response of the otoliths and semicircular canals in the early stages of diabetes may be related to their distinct metabolic and vascular characteristics (12). On the other hand, the Caloric test, which is sensitive to low-frequency damage and an indicator of vestibular tonic function, often exhibits abnormal results in symptomatic diabetic patients. Unilateral weakness (UW) indicates hypofunction of the horizontal canal and possibly the vestibular nerve (21).

The vHIT-Caloric dissociation pattern (normal vHIT/abnormal caloric) typically reflects incomplete peripheral compensation or central adaptation deficits in these patients (52, 53).

In some diabetic persons, oculomotor abnormalities could be caused by persistent hyperglycemia, impaired microvascular circulation and neural conduction. Oculomotor abnormalities may manifest as reduced saccadic accuracy, increased latency of saccadic eye movements, or reduced smooth pursuit performance, even in the absence of clear ophthalmic complications. Such findings suggest that oculomotor abnormalities may be an early indicator of central and peripheral neurodegenerative processes associated with type 2 diabetes, emphasizing the need for a comprehensive assessment of neuro-ophthalmological evaluation in this population (1).

Functional Balance Deficits and Fall Risk

Diabetic sensory deficits impair postural stability; vestibular dysfunction alone doubles fall risk (54). Posturography studies have reported that diabetic patients fail to maintain control of balance when the vestibular or visual system is challenged. This confirms that the central nervous system of diabetic patients has difficulty in effectively reweighting and integrating impaired sensory inputs (especially vestibular and proprioceptive) (22, 55). Even mild vestibular dysfunction in these individuals can elevate the risk of falls, suggesting an important functional relevance.

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

Targeted Clinical Management and Tailored Treatment Strategies

Diabetic BPPV management requires a paradigm beyond mechanical maneuvers, addressing systemic abnormalities and neurological damage alongside particle displacement (56). Given the high recurrence and treatment resistance, multidisciplinary care is essential, as simple mechanical stabilization is insufficient long-term.

The Vital Role of Vestibular Rehabilitation Therapy

While the Epley maneuver (CRP) is the gold-standard first-line treatment for acute BPPV (3, 4), emerging evidence suggests that vestibular rehabilitation therapy (VRT) may offer additional benefits for diabetic patients, particularly those with recurrence or underlying neuropathy. Evidence for VRT in diabetic BPPV, however, remains extremely limited. In the only randomized trial for diabetic BPPV (n=30), Abu Shaphe et al. (2023) found that VRT significantly improved vertigo scores (VSS-sf, P=0.03) with a positive trend in balance (Berg Balance Scale, P=0.51) compared to CRP alone (23). However, critical limitations include the small sample (n=15 per group), short 4-week follow-up, and lack of long-term recurrence or objective VEMP data. The theoretical rationale for VRT in diabetic patients is sound. Based on neural adaptation and sensory substitution, VRT addresses VOR and postural deficits exacerbated by diabetic neuropathy, enhancing central neuroplasticity to compensate for weakened peripheral signals.Given the current state of evidence, we propose that VRT should not replace CRP as primary treatment, but rather should be considered as a complementary therapy in specific clinical contexts: (1) patients with persistent balance deficits after successful CRP; (2) patients with recurrent BPPV despite optimal metabolic control (9); (3) patients with evidence of peripheral vestibular neuropathy on VEMP testing (20,21); and (4) patients with coexisting diabetic peripheral neuropathy affecting proprioception (2). The question of whether VRT provides superior outcomes compared to CRP in diabetic populations remains open and requires larger, well-designed randomized controlled trials with adequate sample sizes, extended follow-up periods (≥6 months), and standardized outcome measures including both subjective symptoms and objective vestibular function parameters.

Modifying Systemic Risk Factors

Reducing BPPV recurrence rates is contingent upon controlling the physical and systemic factors that cause otoconia instability. Strong glycemic control and achieving an optimal HbA1c level are the paramount treatment (57). Precise control of high blood glucose attenuates the progression of microangiopathy, thereby protecting the inner ear from damage. This slows the rate of progressive otoconia loss (58). Vitamin D and calcium supplementation are a critical preventive measure. In patients with BPPV who are vitamin D deficient (<30 ng/mL), these supplements significantly reduce the recurrence rate (24, 59). This helps strengthen the structure of the otoconia and promotes the formation of more stable calcium crystals. Rigorous management of comorbid hypertension and hyperlipidemia is also essential. By stabilizing and controlling blood pressure, synergistic vascular damage to the labyrinth is reduced, and one of the main mediators of BPPV in diabetes is mitigated (14, 60).

Proposed Clinical Algorithm for Management of BPPV in Diabetic Patients

We propose the following stepwise clinical approach for the management of BPPV in diabetic patients, integrating both mechanical and systemic interventions:

Initial Presentation (First Episode): 1. Confirm BPPV diagnosis with Dix-Hallpike or supine roll test (3). 2. Perform standard CRP (Epley or Semont maneuver) (4). 3. Assess metabolic parameters: HbA1c, fasting glucose, insulin levels, vitamin D, calcium (9,35). 4. Initiate metabolic optimization: target HbA1c <7%, vitamin D >30 ng/mL 5. Provide patient education on recurrence risk and preventive strategies (57, 61).

Early Recurrence (within 3 months): 1. Repeat CRP (4). 2. Perform comprehensive vestibular assessment including VEMP testing (21, 42). 3. If VEMP abnormalities are present (prolonged latencies >2.5 SD above normal, reduced amplitudes <50% of control values), consider early initiation of VRT (20, 21). 4. Optimize vitamin D and calcium supplementation if deficient (24, 57). 5. Intensify glycemic control efforts in consultation with endocrinology (61).

Persistent or Frequent Recurrence (>2 episodes within 6 months): 1. Initiate formal VRT program (minimum 6 weeks, 2-3 sessions per week) (23). 2. Prescribe home exercise program for gaze stabilization and balance (62, 63). 3. Consider referral to endocrinology for advanced metabolic management (61). 4. Re-evaluate for other vestibular pathology (Ménière's disease, vestibular migraine, bilateral vestibulopathy). 5. Address comorbidities aggressively (hypertension, hyperlipidemia) (15, 58).

Chronic/Refractory Cases: 1. Multidisciplinary management involving otology, neurology, and endocrinology (54). 2. Long-term VRT with periodic reassessment (23). 3. Consider emerging therapies or clinical trial enrollment. 4. Regular monitoring of vestibular function with serial VEMP testing (21, 42).5. Fall prevention strategies and assistive devices as needed (1).

This algorithm is based on synthesis of available evidence and expert opinion, and should be validated in future prospective studies. Individual patient factors, preferences, and local resource availability should guide clinical decision-making within this framework.

Limitations and Future Directions: The Need for Advanced Imaging

Current imaging modalities have significant limitations in visualizing the fine structures of the inner ear and detecting subtle vascular or cellular pathology. Standard MRI (1.5T or 3T) can visualize gross anatomical abnormalities such as endolymphatic hydrops or vestibular schwannomas, but lacks the resolution to detect microangiopathic changes in labyrinthine vessels (diameter <100 µm) or cellular-level alterations in the macula. Histopatho-

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

logical studies remain the gold standard for detecting type I hair cell loss and nerve demyelination (13, 19), but these are only feasible post-mortem. This diagnostic gap means that much of diabetic vestibulopathy remains subclinical and is only detected through functional testing (VEMP) rather than structural imaging (20, 21).

Emerging technologies such as ultra-high-field MRI (7 Tesla), which offers superior spatial resolution, and optical coherence tomography (OCT) adapted for inner ear imaging, hold promise for non-invasive in vivo visualization of labyrinthine microstructure. Such advances could enable earlier detection of diabetic vestibular damage and monitoring of disease progression or treatment response. These imaging capabilities would complement electrophysiological assessment and could potentially identify patients at highest risk for BPPV before symptomatic episodes occur.

Discussion

Our review reveals that diabetes affects the vestibular system through mechanisms that extend beyond simple metabolic dysregulation. The finding that type I hair cells in the sacculeare selectively vulnerable to glucose toxicity (13, 18) helps explain why BPPV occurs more frequently in diabetic patients. Unlike type II hair cells, type I cells appear to lack effectiveglucose regulation mechanisms, making them particularly susceptible to hyperglycemic damage. This selective vulnerabilityhas important clinical implications, as it suggests that even well-controlled diabetes may still pose risks to vestibular function over time. The strong correlation between VEMP latency prolongation and HbA1c levels (21, 22) provides a valuable clinical tool. VEMP testing could identify at-risk patients before symptomatic BPPV develops, potentially allowing earlier intervention. However, the lack of standardized VEMP protocols across different laboratories remains a significant limitation. Establishing universal cut-off values for diabetic populations should be a research priority. The recurrence rate exceeding 50% in diabetics (6, 9) compared to 27% in non-diabetics cannot be attributed solely to otoconia fragility; hyperinsulinemia acts as an independent risk factor affecting the endolymphatic sac beyond glucose metabolism alone (9). This finding challenges the traditional focus on glycemic control and indicates that comprehensive metabolic management addressing both glucose and insulin levels may be necessary. The role of vitamin D deficiency deserves particular attention. While vitamin D supplementation has shown promise in reducing BPPV recurrence in the general population (24, 57), its specific efficacy in diabetic patients remains understudied. Given the high prevalence of vitamin D deficiency in diabetes (16, 35), thisrepresents a relatively simple intervention that warrants investigation in randomized controlled trials specifically targeting diabetic BPPV patients. The evidence for VRT in diabetic BPPV is limited to a single small trial (23), yet the theoretical rationale is compelling. Diabetic patients often have impaired central vestibular compensation mechanisms (55, 56), which may explain why standard repositioning maneuvers provide only temporary relief. Larger studies comparing VRT with standard care in diabetic populations are needed before definitive clinical recommendations can be made. Several limitations must be acknowledged. First, most included studies were observational rather than interventional, limiting causal inferences. Second, the heterogeneity in diabetes severity, duration, and treatment across studies makes it difficult to identify which diabetic patients are at highest risk. Third, the two histopathological studies (13, 18) examined only small numbers of temporal bones, and findings may not generalize to all diabetic patients. Finally, most studies focused on type 2 diabetes, and whether similar mechanisms apply to type 1 diabetes remains unclear. Despite these limitations, the evidence consistently points to diabetes as an independent and modifiable risk factor for BPPV. The practical implication is clear: diabetic patients with balance complaints warrant thorough vestibular evaluation, and those with recurrent BPPV may benefit from interventions beyond mechanical repositioning maneuvers.

Conclusion

Diabetes mellitus represents a significant and independent risk factor for both BPPV occurrence and recurrence through convergent pathophysiological mechanisms including selective type I hair cell loss, vestibular nerve demyelination, and otoconia fragility secondary to impaired calcium homeostasis. The dramatically elevated recurrence rate in diabetic patients reflects persistent metabolic disruption that mechanical repositioning alone cannot address. Clinical management should integrate VEMP screening for subclinical neuropathy, rigorous glycemic control, vitamin D supplementation, and consideration of vestibular rehabilitation therapy as complementary treatment. Future research priorities include identifying molecular biomarkers of otoconia degeneration, developing standardized VEMP protocols, and conducting adequately powered trials of multimodal interventions specifically in diabetic populations. A proactive approach addressing systemic metabolic factors alongside vestibular rehabilitation may reduce the substantial burden of recurrent BPPV and fall risk in diabetic patients.

Acknowledgment

None.

Conflict of Interests

The authors declare that they have no competing interests.

Authors' Contributions

Seyedeh Nazanin Hajjari: Conceptualization, Data curation, Investigation, Writing – original draft, Writing – review & editing.

Fatemeh Jafarlou: Supervision, Methodology, Validation, Writing – review & editing, Project administration.

Ethical Considerations

Not applicable.

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

Funding Support

This study was supported by Tabriz University of Medical Sciences, Tabriz, Iran (Grant Number: 79120).

Data Availability

Not applicable (no new data were generated or analyzed in this review; all data are from previously published studies cited in the references).

AI Use Statement

The authors did not use artificial intelligence or AI-assisted technologies in the preparation of this manuscript.

References

1. D'Addio F, Pastore I, Loretelli C, Valderrama-Vasquez A, Usuelli V, Assi E, et al. Abnormalities of the oculomotor function in type 1 diabetes and diabetic neuropathy. Acta Diabetol. 2022;59(9):1157-1167. doi:10.1007/s00592-022-01911-1.
2. Deli G, Bosnyak E, Pusch G, Komoly S, Feher G. Diabetic neuropathies: diagnosis and management. Neuroendocrinology. 2013;98(4):267-280. doi:10.1159/000358728.
3. Yetişer S. Review of the pathology underlying benign paroxysmal positional vertigo. J Int Med Res. 2020;48(4):0300060519892370. doi:10.1177/0300060519892370.
4. Bhattacharyya N, Gubbels SP, Schwartz SR, Edlow JA, El-Kashlan H, Fife T, et al. Clinical Practice Guideline: Benign Paroxysmal Positional Vertigo (Update). Otolaryngol Head Neck Surg. 2017;156(3 Suppl):S1-S47. doi:10.1177/0194599816689667.
5. Chen J, Zhang S, Cui K, Liu C. Risk factors for benign paroxysmal positional vertigo recurrence: a systematic review and meta-analysis. J Neurol. 2021;268(11):4117-4127. doi:10.1007/s00415-020-10175-0.
6. Sfakianaki I, Binos P, Karkos PD, Dimas GG, Psillas G. Risk Factors for Recurrence of Benign Paroxysmal Positional Vertigo: A Clinical Review. J Clin Med. 2021;10(19):4372. doi:10.3390/jcm10194372.
7. D'Silva LJ, Staecker H, Lin J, Maddux Ch, Ferraro J, Dai H, et al. Otolith dysfunction in persons with both diabetes and benign paroxysmal positional vertigo. Otol Neurotol. 2017;38(3):379-385. doi: 10.1097/MAO.0000000000001309
8. Cohen HS, Kimball KT, Stewart MG. Benign paroxysmal positional vertigo and comorbid conditions. ORL J Otorhinolaryngol Relat Spec. 2004;66(1):11-15. doi: 10.1159/000077227.
9. Webster G, Sens PM, Salmito MC, Cavalcante JD, Santos PR. Hyperinsulinemia and hyperglycemia: risk factors for recurrence of benign paroxysmal positional vertigo. Braz J Otorhinolaryngol. 2015;81(4):347-351.
10. Li Y, Liu Y, Liu SW, Gao M, Wang W, Chen K, et al. Diabetic vascular diseases: molecular mechanisms and therapeutic strategies. Signal Transduct Target Ther. 2023;8(1):152. doi: 10.1038/s41392-023-01400-z.
11. Wen Y, Fan Y, Jian B. Identifying key risk factors for the recurrence of benign paroxysmal positional vertigo following successful canalith repositioning maneuvers: a meta analysis. Eur J Med Res. 2025;30:262. doi:10.1186/s40001-025-02482-x.
12. Jáuregui-Renaud K. Diabetes Mellinus in the Inner Ear. Med (Buenos Aires). 2016;76(1):60–63.
13. Kocdor P, Kaya S, Erdil M, Cureoglu S, Paparella MM, Adams ME. Vascular and neuroepithelial histopathology of the saccule in humans with diabetes mellitus. Otol Neurotol. 2016;37(5):553-557. doi:10.1097/MAO.0000000000001018.
14. D'Silva LJ, Lin J, Staecker H, Whitney SL, Kluding PM. Impact of diabetic complications on balance and falls: contribution of the vestibular system. Phys Ther. 2016;96(3):400-409. doi:10.2522/ptj.20140604.
15. Messina A, Casani AP, Manfrin M, Guidetti G. Italian survey on benign paroxysmal positional vertigo. Acta Otorhinolaryngol Ital. 2017;37(4):328-335. doi:10.14639/0392-100X-1121.
16. Bazoni JA, Mendes WS, Meneguello J, Brandão RA, Menezes AS, Pereira LV, et al. Vitamin D deficiency and benign paroxysmal positional vertigo: a systematic review and meta-analysis. Braz J Otorhinolaryngol. 2020;86(6):795-805. doi: 10.1016/j.mehy.2012.11.029.
17. Ward BK, Wenzel A, Kalyani RR, Agrawal Y, Feng AL, Polydefkis M, et al. Characterization of vestibulopathy in individuals with type 2 diabetes mellitus. Otolaryngol Head Neck Surg. 2015;153(1):112-118. doi:10.1177/0194599815576717.
18. Degerman E, Rauch U, Lindberg S, Caye-Thomasen P, Hultgårdh A, Magnusson M. Expression of insulin signalling components in the sensory epithelium of the human saccule. Cell Tissue Res. 2013;352(3):469-478. doi:10.1007/s00441-013-1614-x.
19. Lasagni A , Giordano P , Lacilla M , Raviolo A , Trento M , Camussi E , et al. Cochlear, auditory brainstem responses in Type 1 diabetes: relationship with metabolic variables and diabetic complications. Diabet Med. 2016;33(9):1260–1267. doi: 10.1111/dme.13039
20. Kamali B, Hajiabolhassan F, Fatahi J, Nasli-Esfahani E, Sarrafzadeh J, Fallah S. Effects of type 1 diabetes mellitus with or without neuropathy on vestibular evoked myogenic potentials. Acta Med Iran. 2013;51(2):107-112.
21. Konukseven O, Polat SB, Karahan S, Konukseven E, Ersoy R, Cakir B, et al. Electrophysiologic vestibular evaluation in type 2 diabetic and prediabetic patients: air conduction ocular and cervical vestibular evoked myogenic potentials. Int J Audiol. 2015;54(8):536–543. doi: 10.3109/14992027.2014.971887
22. D'Silva LJ, Staecker H, Lin J, Maddux C, Ferraro J, Dai H, et al. Retrospective data suggests that the higher prevalence of benign paroxysmal positional vertigo in individuals with type 2 diabetes is mediated by hypertension. J Vestib Res. 2015;25(5-6):233-239. doi:10.3233/VES-150563 .
23. McDonnell MN, Hillier SL. Vestibular rehabilitation for unilateral peripheral vestibular dysfunction. Cochrane Database Syst Rev. 2015;1(1):CD005397. doi:10.1002/14651858.CD005397.pub4.
25. Gawron W, Pospiech L, K Orendorz-Fraczkowska K, A Noczynska A. Are there any disturbances in vestibular organ of children and young adults with Type I diabetes? Diabetologia. 2002;45(5):728–734. doi: 10.1007/s00125-002-0813-x.
26. Klagenberg KF, Zeigelboim BS, Jurkiewicz AL, Martins-Bassetto J. Vestibulocochlear manifestations in patients with type 1 diabetes mellitus. Braz J Otorhinolaryngol. 2007;73(3):353-358. doi: 10.1016/S1808-8694(15)30079-3.
27. Rigon R, Rossi AG, Casali CP. Otoneurologic findings in patients with type 1 diabetes mellitus. Braz J Otorhinolaryngol. 2007;73(1):100-105. doi: 10.1016/s1808-8694(15)31130-7.
28. Boyle JP, Thompson TJ, Gregg EW, Barker LE, Williamson DF. Projection of the year 2050 burden of diabetes in the US adult population: dynamic modeling of incidence, mortality, and prediabetes prevalence. Popul Health Metr. 2010;8:29. doi:10.1186/1478-7954-8-29.
29. Von Brevern M, Schmidt T, Schönfeld U, Lempert T, Clarke AH. Utricular dysfunction in patients with benign paroxysmal positional vertigo. Otol Neurotol. 2006;27(1):92-96. doi: 10.1097/01.mao.0000187238.56583.9b.
30. Elangovan S, Spankovich C. Diabetes and auditory vestibular pathology. Semin Hear. 2019;40(4):292-299. doi: 10.1055/s-0039-1697033.
31. Thorens B, Mueckler M. Glucose transporters in the 21st century. Am J Physiol Endocrinol Metab. 2010;298(2):E141-E145. doi:10.1152/ajpendo.00712.2009.
32. Fife TD. Benign paroxysmal positional vertigo. Semin Neurol. 2009;29(5):500–508. doi: 10.1055/s-0029-1241041.
33. Yoda S, Cureoglu S, Yildirim-Baylan M, Morita N, Fukushima H, Harada T,et al. Association between type 1 diabetes mellitus and deposits in the semicircular canals. Otolaryngol Head Neck Surg. 2011;145(3):458-462. doi: 10.1177/0194599811407610.
34. Nikolenko V, Proshkina A, Zharikov Y, Rizaeva N, Lapina T, Sharifullina P, et al. Vestibular Otolithic Receptors: Association Between Molecular Mechanism of Degeneration and Benign Paroxysmal Positional Vertigo. Current Behavioral Neuroscience Reports. 2025;12(1):1. doi: 10.1007/s40473-025-00299-w.
35. Jeong SH, Kim JS, Shin JW, Kim S, Lee H, Lee AY, et al. Decreased serum vitamin D in idiopathic benign paroxysmal positional vertigo. J Neurol. 2013;260(3):832-838. doi: 10.1007/s00415-012-6712-2.
36. Kumar P, Singh NK, Raveendran RK, Apeksha K, Ghosh V, Bhanukumar M. The functioning of otolith organs in individuals with type-II diabetes mellitus. Hearing Research. 2025; 467, 109412. https://doi.org/10.1016/j.heares.2025.109412.
37. Yetişer S. Secondary benign paroxysmal positional vertigo. J Int Adv

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]

Otol. 2019;15(3):451.
38. Lips P, Eekhoff M, van Schoor NM, Oosterwerff M, de Jongh RT, Krul-Poel YH, et al. Vitamin D and type 2 diabetes. J Steroid Biochem Mol Biol. 2017;173:280-285. doi:10.1016/j.jsbmb.2016.11.021.
39. Tan KCB, Chow WS, Ai VHG, Metz Ch, Bucala R, Lam KS. LAdvanced glycation end products and endothelial dysfunction in type 2 diabetes. Diabetes Care. 2002 Jun;25(6):1055-9.doi: 10.2337/diacare.25.6.1055.
40. Li S, Wang Z, Liu Y, Cao J, Zheng H, Jing Y, et al. Risk factors for the recurrence of benign paroxysmal positional vertigo: a systematic review and meta-analysis. Ear Nose Throat J. 2022;101(3):NP112-NP134. doi: 10.1177/0145561320943362.
41. Cao W, Geng Y, Chang J, Li F. Risk factors for benign paroxysmal positional vertigo and construction of a nomogram predictive model. Am J Transl Res. 2024;16(6):2435-2444. doi: 10.62347/DHAJ4799.
42. Magliulo G, Iannella G, Gagliardi S, Re M. A 1-year follow-up study with C-VEMPs, O-VEMPs and video head impulse testing in vestibular neuritis. Eur Arch Otorhinolaryngol. 2015;272(11):3277-3281. doi:10.1007/s00405-014-3404-9.
43. Kamali B, Hajiabolhassan F, Fatahi J, Nasliesfahani E, Sarafzadeh J, Faghihzadeh S. Comparing the vestibular evoked myogenic potentials in patients with type I diabetes mellitus and normal people. Aud Vestib Res. 2017;22(2):94-103.
44. Kalkan M, Bayram A, Gökay F, Cura HS, Mutlu C. Assessment of vestibular-evoked myogenic potentials and video head impulse test in type 2 diabetes mellitus patients with or without polyneuropathy. Eur Arch Otorhinolaryngol. 2018;275(3):719-724. doi: 10.1007/s00405-018-4873-z.
45. Zhang J, Ye L, Bai X, Huang Y, Lin J, Huang H. Cervical and ocular vestibular evoked myogenic potentials in patients with Diabetic Peripheral Neuropathy. Diabetol Metab Syndr. 2023;15(1):100. doi: 10.1186/s13098-023-01068-z.
46. MacDougall HG, Weber KP, McGarvie LA, Halmagyi GM, Curthoys IS. The video head impulse test: diagnostic accuracy in peripheral vestibulopathy. Neurology. 2009;73(14):1134-1141. doi:10.1212/WNL.0b013e3181bacf85.
47. Shepard NT, Jacobson GP. The caloric irrigation test. In: Furman JM, Lempert T, editors. Handbook of Clinical Neurology. Vol. 137. Amsterdam: Elsevier; 2016: 119-131. doi:10.1016/B978-0-444-63437-5.00009-1.
48.Nourizadeh N, Jahani M, Jafarzadeh S. Auditory and Vestibular Assessment of Patients with Type Two Diabetes Mellitus: A Case-Control Study. Iran J Otorhinolaryngol. 2021 Sep;33(118):263–269. doi: 10.22038/ijorl.2021.55334.2899.
49. Tahraband MH, Heidari F. Clinical Results of Peripheral Vestibular Function in People With Diabetes Mellitus. Sci J Rehabil Med. 2022;10(6):1124–39. DOI:10.32598/SJRM.10.6.14.
50. Mahalingasivam AA, Jespersen AK, Ejskjaer N, Hougaard DD, Vestergaard P, Rasmussen NH, et al. The co-existence of peripheral and vestibular neuropathy in diabetes: a cross-sectional study. Eur Arch Otorhinolaryngol. 2024;281(2):663-672. doi:10.1007/s00405-023-08130-6.
51. Nguyen D, Hoang N, Huynh L, Huynh Y, Phan H, Nguyen H. Vestibular function in type 2 diabetes: A vHIT-based comparative study. 2025; Journal of the Neurological Sciences, 480, Article 125435.
52. Bakkali S, Taeymans J, Osafo Sasu Senior C, Dirinck E, Vereeck L, Vissers D. Does diabetes mellitus type 2 affect vestibular function? A systematic review and meta-analysis. Diabetes Epidemiol Manag. 2021;4:100035. doi:10.1016/j.deman.2021.100035.
53. Hannigan IP, Welgampola MS, Watson ShRD. Dissociation of caloric and head impulse tests: a marker of Meniere's disease? J Neurol. 2021; 268(2):431-439. doi: 10.1007/s00415-019-09431-9.
54. Agrawal Y, Carey JP, Della Santina CC, Schubert MC, Minor LB. Disorders of balance and vestibular function in US adults: data from the National Health and Nutrition Examination Survey, 2001-2004. Otol Neurotol. 2009;25;169(10):938-44. doi: 10.1001/archinternmed.2009.66.
55. Hamed SA, ElHadad AF, Oseily AM. Vestibular Function in Children with Type 1 Diabetes: Videonystagmography Testing. Audiol Neurootol. 2021;26(6):470-478. doi: 10.1159/000516204.
56. Balatsouras DG, Koukoutsis G, Fassolis A, Moukos A, Apris A. Benign paroxysmal positional vertigo in the elderly: current insights. Clin Interv Aging. 2018 Nov 5;13:2251-2266. doi: 10.2147/CIA.S144134.
57. S. Savitha S, Kirthiga A, Sathishwari S. Effectiveness of Vestibular Rehabilitation for Diabetic Patients in Improving Balance and Quality of Life. International Journal of Current Science Research and Review. 2925; 8 (5): 2192-2199. DOI: 10.47191/ijcsrr/V8-i5-28.
58. Webster G, Sens PM, Salmito MC, Cavalcante JDR, Santos PR, Silva ALM, et al. Glucose and insulin profiles in patients with benign paroxysmal positional vertigo. Braz J Otorhinolaryngol. 2015;81(2):180-187. doi:10.1016/j.bjorl.2014.09.008.
59. Wang Z, He G, Chen Z, Jie H, Zeng Q. Effect of calcium and vitamin D supplementation on recurrence of benign paroxysmal positional vertigo in osteoporotic patients. Am J Transl Res. 2026 Jan 15;18(1):432-443. doi: 10.62347/YFAH9697.
60. Doğan B, Baklacı D. Association between diabetes mellitus, hypertension, migraine, and recurrence risk in benign paroxysmal positional vertigo: a retrospective cohort study. Front. Neurol. 2026;17:1785013. doi: 10.3389/fneur.2026.1785013.
61. Savitha S. Effectiveness of Vestibular Rehabilitation for Diabetic Patients in Improving Balance and Quality of Life. International Journal of Current Science Research and Review. 2025;8(05). doi:10.47191/ijcsrr/v8-i5-28
62. Gaur S, Awasthi SK, Bhadouriya SK, Saxena R, Pathak VK, Bisht M. Efficacy of Epley's Maneuver in Treating BPPV Patients: A Prospective Observational Study. Int J Otolaryngol. 2015:487160. doi: 10.1155/2015/487160.
63. Shaphe MA, Alshehri M, Alajam RA, Ali Beg R, Hamidi NA, Nanjan S, et al. Effectiveness of Epley–Canalith Repositioning Procedure versus Vestibular Rehabilitation Therapy in diabetic patients with posterior benign paroxysmal positional vertigo: a randomized trial. Life (Basel). 2023;13(5):1169. doi:10.3390/life13051169.

[Downloaded from mjiri.iums.ac.ir on 2026-09-08]

[DOI: 10.47176/mjiri.40.75]