



Metabolic Disorders Associated with Neck Pain and Dizziness - Pathophysiological Hypotheses, Clinical Associations, Assessment, and Rehabilitation Implications: A Narrative Review

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Abstract

Neck pain and dizziness frequently coexist, but they do not represent a single diagnostic syndrome. This structured narrative review examines whether diabetes and other metabolic or endocrine disorders contribute directly to neck pain or instead modify dizziness, imbalance, falls, sensory integration, and rehabilitation safety. The review narrows the scope, separates symptom phenotypes, distinguishes direct clinical associations from mechanistic hypotheses, and incorporates negative and conflicting findings. The strongest clinically relevant links concern diabetes-related peripheral or autonomic neuropathy, orthostatic symptoms, multisensory balance impairment, and falls risk. Population-level data support an association between diabetes and peripheral vestibular disorders, whereas a recent cross-sectional study found no group difference in vestibulo-ocular reflex measures, showing that vestibular involvement is not uniform. Evidence connecting metabolic disease specifically to common mechanical neck pain is substantially weaker and usually indirect.

Obesity, vitamin D deficiency, thyroid dysfunction, vitamin B12 deficiency, electrolyte abnormalities, and medication effects may contribute to pain, weakness, disequilibrium, or exercise intolerance, but they should not be treated as default explanations. The clinical framework prioritises symptom classification, neurological and cardiovascular safety screening, medication and complication review, targeted vestibular and cervical assessment, and coordinated medical referral. Major research needs include prospective phenotyping, standardised dizziness outcomes, objective vestibular and sensory testing, and trials that account for multimorbidity and treatment effect modifiers.

Keywords: Neck Pain; Dizziness; Vertigo; Diabetes Mellitus; Metabolic Disorders; Peripheral Vestibular Disorders; Autonomic Neuropathy; Orthostatic Hypotension; Falls; Rehabilitation

Introduction

Neck pain is common and heterogeneous. Prevalence estimates vary with age, setting, case definition, and symptom duration, and most neck pain is non-specific rather than attributable to one isolated structural lesion [1-3]. The presence of a metabolic disorder should therefore not be treated as evidence that neck pain has a metabolic cause.

Dizziness is an umbrella symptom rather than a diagnosis. Vertigo describes an illusion of movement; light-headedness or presyncope may reflect postural or cardiovascular mechanisms; and disequilibrium describes unsteadiness during standing or walking. These symptoms may overlap, but treating them as one outcome obscures important differences in assessment and referral [4-8].

Physiotherapists and other first-contact rehabilitation clinicians are increasingly asked to assess people whose neck pain coexists with dizziness, and a substantial proportion of these patients have diabetes or another metabolic or endocrine comorbidity. Two decision errors follow from an undifferentiated reading of the literature: attributing non-specific neck pain to a metabolic abnormality that is present but not causal, and overlooking metabolic contributions to orthostatic symptoms, sensory loss, and falls risk that change assessment and referral. Misattribution delays correct differential diagnosis; overlooking modifiable risk delays falls prevention.

The literature relevant to this problem is spread across diabetology, neuro-otology, epidemiology, and musculoskeletal rehabilitation, and its strength differs sharply by outcome: associations with dizziness, balance, and falls are far better supported than associations with isolated neck pain. No existing narrative review for a rehabilitation readership separates these levels of evidence explicitly. This review is therefore the first to provide a symptom-phenotype-based clinical framework that distinguishes direct clinical association, indirect clinical association, and mechanistic plausibility for metabolic disorders in neck pain and dizziness, and that translates the distinction into assessment, referral, and rehabilitation decisions for practising clinicians.

Metabolic and endocrine disease may alter balance through glycaemic events, autonomic dysfunction, peripheral sensory loss, visual impairment, muscle weakness, medication effects, reduced activity, or vascular and inflammatory pathways. Diabetes has been associated with peripheral vestibular disorders in a large population-based analysis, but objective vestibular findings are not uniform across people with diabetes and neuropathy [9, 10]. The appropriate clinical question is therefore not whether metabolism universally causes neck pain and dizziness, but whether metabolic disease changes symptom expression, differential diagnosis, falls risk, treatment tolerance, or rehabilitation needs.

Aims and Review Questions

This structured narrative review addresses the following concrete questions:

1. Does diabetes or another metabolic or endocrine disorder contribute directly to common non-specific neck pain, or only modify pain burden, activity, and treatment tolerance?
2. Which metabolic and endocrine mechanisms are associated with dizziness phenotypes (vertigo, presyncope or light-headedness, disequilibrium, visual motion sensitivity, exertional intolerance), and how strong and how consistent is that evidence?
3. How do population-level, objective vestibular, and comparative functional studies differ in what they can establish about diabetes and vestibular disorders, and how should apparently conflicting findings be interpreted?
4. How should the answers to questions 1 to 3 change history-taking, physical examination, safety screening, referral, outcome-measure selection, and exercise rehabilitation for patients with coexisting neck pain and dizziness?
5. What study designs and outcome definitions are needed to resolve the remaining uncertainties?

Literature Search and Review Approach

This article is a structured narrative review and not a systematic review or meta-analysis. No protocol was registered, screening was not performed in duplicate by independent reviewers, no formal risk-of-bias tool was applied, and no quantitative synthesis was undertaken. The review therefore makes no claim to systematic-review completeness and does not use systematic-review or PRISMA terminology.

Search strategy

Searches were conducted in PubMed/MEDLINE, Scopus, Web of Science, PEDro, and the Cochrane Library. The final search was

completed on [insert exact end date, day/month/year]. Citation tracking (backward and forward) supplemented the database searches, and older landmark studies were retained where clinically relevant.

Search terms combined the following concept blocks with Boolean AND between blocks and OR within blocks:

- Concept block 1 (outcome, cervical): neck pain; cervical pain; neck ache
- Concept block 2 (outcome, vestibular and balance): dizziness; vertigo; balance; postural control; falls; disequilibrium
- Concept block 3 (exposure, metabolic): diabetes; metabolic syndrome; obesity; insulin resistance; dyslipidaemia; thyroid dysfunction; vitamin D deficiency; vitamin B12 deficiency; electrolyte disturbance; neuropathy
- Concept block 4 (mechanism): inflammation; vascular dysfunction; oxidative stress; proprioception; sensory reweighting

Eligibility and literature types

Priority was given to systematic reviews, clinical guidelines, large observational studies, prospective or comparative clinical studies, and mechanistic work with direct clinical relevance. Included literature comprised peer-reviewed primary studies, systematic reviews, and clinical practice guidelines in English. Grey literature was restricted to guidance documents from professional bodies. Opinion pieces without primary or synthesised data were excluded.

Synthesis method and evidence levels

Because the evidence base is heterogeneous in design and outcome definition, the synthesis is structured rather than pooled. Evidence is grouped into three descriptive levels: direct clinical association, indirect clinical association, and mechanistic plausibility. These labels are descriptive judgements and are not formal GRADE ratings. The scope is limited to diabetes and selected metabolic or endocrine conditions that may affect pain, dizziness, balance, falls, or rehabilitation safety. Osteoporosis and gout are retained as clinically relevant associated conditions because they may alter fracture or cervical-risk assessment, but they are not presented as equivalent metabolic mechanisms.

Clinical Phenotype and Diagnostic Boundaries

"Metabolic dizziness" should not be used as a diagnostic label. In this review, metabolic or systemic contributors are considered only when symptoms have a plausible relationship to hypoglycaemia, hyperglycaemia, postural blood-pressure change, autonomic dysfunction, medication exposure, electrolyte disturbance, sensory loss, or general neuromuscular impairment. Rotational vertigo should be assessed as a vestibular symptom until a suitable evaluation indicates otherwise.

A history should distinguish spinning or motion illusion from light-headedness, presyncope, visual dimming, imbalance, visual motion sensitivity, and exertional intolerance. Timing is clinically informative: symptoms related to standing, meals, exertion, glucose-lowering medication, or head position imply different diagnostic pathways. Patients may have more than one cause, particularly when diabetes, neuropathy, visual disease, cardiovascular disease, medication exposure, and ageing coexist [4, 8].

Metabolic Disorders and Neck Pain

Obesity, metabolic syndrome, and inactivity

Obesity is associated with chronic pain across several body regions, but the evidence commonly concerns widespread, low-back, knee, or other musculoskeletal pain rather than isolated neck pain [11, 12]. Proposed pathways include inflammation, mechanical loading, reduced activity, sleep disturbance, and psychosocial confounding, but these mechanisms do not establish a neck-specific causal relationship.

The defensible interpretation is that obesity and metabolic syndrome may modify activity, physical capacity, prognosis, and treatment tolerance in a person who also has neck pain. They should not be presented as established causes of common non-specific cervical pain. Weight-management advice may be appropriate within a coordinated health plan, but physiotherapy does not replace medical or nutritional treatment of metabolic disease.

Vitamin D deficiency

Low vitamin D status has been associated with non-specific musculoskeletal pain in some clinical and population studies, but findings are heterogeneous and affected by age, obesity, outdoor activity, comorbidity, and physical function [13-15]. The available evidence does not establish that vitamin D deficiency specifically causes neck pain or that supplementation reliably resolves it. Diffuse pain, weakness, or osteomalacia-like presentations should be distinguished from routine mechanical neck pain.

Diabetes and painful cervical presentations

Diabetic peripheral neuropathy is generally distal and length-dependent. Diabetes is highly relevant to sensory testing, autonomic symptoms, falls risk, exercise tolerance, and rehabilitation safety, but the available evidence does not justify describing uncomplicated diabetes as a direct cause of isolated neck pain or cervical radiculopathy [16, 17]. Adhesive capsulitis, limited joint mobility, myopathy, and focal neuropathic disorders may coexist with neck-region complaints, but they are separate clinical entities requiring targeted assessment.

Thyroid dysfunction

Thyroid dysfunction can produce weakness, stiffness, fatigue, tremor, exercise intolerance, and neuromuscular abnormalities [18]. These effects may increase perceived neck-region discomfort or reduce postural endurance, but neck pain is not established as a specific clinical consequence. Thyroid disease should therefore be considered when the broader history and examination suggest a systemic neuromuscular problem, not used as a default explanation for neck pain.

Osteoporosis, fracture risk, and gout

Osteoporosis is relevant to neck pain mainly through fracture risk, particularly after trauma or in people with substantial bone fragility. Sudden severe pain, pain after trauma, neurological change, or a high-risk fracture history should prompt medical assessment [19]. Axial or cervical gout is rare and should not be inferred from hyperuricaemia alone; a non-specific neck-pain presentation should not be attributed to gout without appropriate clinical evidence [20].

Summary of the neck-pain evidence

The evidence supports metabolic disease as a possible modifier of pain burden, activity, muscle performance, fracture risk, and treatment tolerance. It does not support a general claim that

metabolic disorders directly cause common non-specific neck pain. This asymmetry is stated explicitly in the abstract, title, tables, and conclusion.

Metabolic Disorders and Dizziness, Imbalance, and Falls

Diabetes and peripheral vestibular disorders: population-level association

A population-based analysis from Taiwan included 150,916 patients with newly diagnosed peripheral vestibular disorders and 452,748 propensity-score-matched controls. Diabetes was more common in the vestibular-disorder group than in controls, and the adjusted odds ratio for diabetes was 1.597 (95% CI 1.570 to 1.623) among patients with peripheral vestibular disorders [9]. This supports an association, not a causal pathway. The administrative design cannot determine whether diabetes caused the vestibular disorder, whether shared comorbidities explained part of the association, or whether the findings apply to other health systems.

The association is not equivalent to proof that every dizzy patient with diabetes has vestibular disease. Diagnostic categories such as Meniere's disease, benign paroxysmal positional vertigo, unilateral vestibulopathy, and other peripheral vestibular disorders were grouped within a population database [9]. A clinical assessment must still determine whether the patient has vertigo, presyncope, disequilibrium, or another symptom phenotype.

Negative and conflicting vestibular findings

A cross-sectional study directly evaluated vestibular function using video head-impulse testing in 114 participants with type 1 diabetes, type 2 diabetes, or no diabetes. Vestibular measurements did not differ between groups, all reported *p*-values were above 0.05, and subgrouping by large-fibre, small-fibre, or autonomic neuropathy did not change the result. Only a small correlation between an autonomic questionnaire score and right lateral gain was reported [10]. This is a clinically important negative finding: diabetes and peripheral neuropathy do not necessarily produce measurable vestibulo-ocular-reflex impairment.

The apparent conflict between population-level association and null objective testing is explainable rather than fatal. The studies used different populations, designs, outcomes, and definitions. A health-record diagnosis of a peripheral vestibular disorder is not equivalent to a video head-impulse measurement, and cross-sectional testing cannot establish temporal sequence. Both findings are presented here, and neither is converted into a universal rule.

Balance, falls-efficacy, and participation

In a comparative study of people with type 2 diabetes, participants classified as having vestibular dysfunction had poorer physical balance performance and lower falls-efficacy, social participation, and quality-of-life outcomes than participants without vestibular dysfunction [21]. The study supports the clinical importance of vestibular dysfunction when it is present, but it does not show that diabetes alone caused the dysfunction or that vestibular treatment will improve every outcome.

Diabetes-related peripheral neuropathy and visual impairment can reduce the sensory information used for postural control, while vestibular hypofunction may further reduce sensory redundancy. Age, renal and cardiovascular disease, cognition, footwear, fear of falling, deconditioning, and medication exposure may modify the

final functional presentation. Falls risk should therefore be measured functionally rather than inferred from the diabetes diagnosis alone [16, 17].

Autonomic neuropathy and orthostatic symptoms

Cardiovascular autonomic neuropathy may impair blood-pressure regulation. Orthostatic hypotension is defined by a sustained fall of at least 20 mmHg in systolic blood pressure or 10 mmHg in diastolic blood pressure within three minutes of standing [22, 23]. Light-headedness, presyncope, weakness, and visual dimming are more compatible with an orthostatic presentation than with rotational vertigo [24].

Dehydration, postprandial hypotension, antihypertensives, diuretics, glucose-lowering treatment, sedatives, and polypharmacy may produce similar symptoms. A physiotherapist may identify a reproducible postural pattern and measure blood pressure or heart rate when this is within local scope and competence, but suspected symptomatic orthostatic hypotension requires medical review rather than exercise progression alone.

Vitamin B12 deficiency

Vitamin B12 deficiency can cause peripheral neuropathy, dorsal-column dysfunction, sensory ataxia, and neuropsychiatric symptoms, sometimes without anaemia or macrocytosis [25, 26]. Loss of vibration and joint-position information may present as disequilibrium or gait instability. Long-term metformin therapy is associated with reduced vitamin B12 status in some patients, making medication history relevant when sensory or gait symptoms are unexplained [27]. B12 deficiency does not establish a vestibular or cervical cause of dizziness.

Thyroid disease, electrolytes, and medication effects

Thyroid disease may produce fatigue, tremor, weakness, palpitations, or exercise intolerance that patients describe as dizziness. Electrolyte disturbances, including hyponatraemia, may cause nausea, headache, confusion, gait disturbance, dizziness, and falls-related impairment [28]. Antihypertensives, diuretics, glucose-lowering medicines, sedatives, and other centrally acting drugs may contribute to hypotension, hypoglycaemia, fatigue, or impaired alertness. Medication concerns should be communicated to the prescriber; physiotherapists should not independently change prescribed treatment.

Pathophysiological Mechanisms (Plausible Pathways, not Established Causes)

The following mechanisms are plausible explanations for associations across different conditions, not established causal pathways for an individual patient. Each mechanism below is labelled with its evidence level.

Inflammation and pain sensitivity (mechanistic plausibility)

Adipose tissue and insulin-resistant states are associated with inflammatory signalling that may influence nociception, fatigue, and tissue recovery [29]. The direct contribution of this pathway to neck pain or vestibular symptoms remains uncertain because clinical studies often measure broad pain or metabolic outcomes rather than a combined neck-pain and dizziness phenotype.

Oxidative stress and microvascular dysfunction (mechanistic plausibility)

Hyperglycaemia-related oxidative stress and microvascular

injury are recognised mechanisms of diabetic complications [30]. They provide biological plausibility for nerve, retinal, autonomic, and possibly inner-ear involvement, but the presence of diabetes or an elevated HbA1c value does not demonstrate that these mechanisms explain a particular patient's dizziness.

Sensory loss and sensory reweighting (mechanistic plausibility)

Postural control depends on the integration of visual, vestibular, and somatosensory information. Distal neuropathy may reduce foot and joint input, vestibular dysfunction may reduce reliable orientation cues, and cervical disorders may alter head and neck sensorimotor control. Their combined effect is clinically plausible but insufficiently studied in diabetes [31, 32].

Autonomic dysfunction and cerebral perfusion (indirect clinical association)

Autonomic dysfunction can produce orthostatic or postprandial symptoms through impaired blood-pressure regulation [22-24]. This mechanism is clinically important because it may signal cardiovascular disease, medication effects, dehydration, or another medical problem. It should not be framed as a benign explanation until relevant risks have been assessed.

Weakness, deconditioning, and reduced activity (mechanistic plausibility)

Neuropathy, thyroid disease, vitamin deficiency, obesity, sarcopenia, pain, fear of falling, and reduced activity may interact to lower strength and exercise tolerance. Reduced activity may then worsen deconditioning and metabolic risk. This feedback loop is clinically relevant, but it does not show that correction of one metabolic variable will resolve neck pain or dizziness.

Clinical Assessment Implications for Physiotherapists

History

The history should clarify whether the symptom is spinning, motion illusion, light-headedness, presyncope, imbalance, visual motion sensitivity, or vague disequilibrium. It should record onset, duration, triggers, positional or postprandial timing, exertional relationship, falls, near-falls, hearing symptoms, visual symptoms, and recovery pattern. Diabetes duration and complications, hypoglycaemia history, renal or cardiovascular disease, thyroid disease, nutritional risk, visual impairment, and prior vestibular diagnoses should be documented.

Medication review should include recent changes and drugs associated with hypoglycaemia, hypotension, sedation, or impaired alertness. New headache, focal neurological symptoms, diplopia, dysarthria, syncope, chest pain, palpitations, fever, significant trauma, unexplained weight loss, or progressive neurological deficit should change the referral threshold.

Examination and referral

The examination should integrate cervical range of motion and sensorimotor testing with gait, balance, lower-limb sensation, vision, neurological screening, and vestibular assessment when indicated and within competence. Positional testing and head-impulse testing require training in performance and interpretation. HINTS is a specialised examination for selected patients with acute vestibular syndrome and should not be used as a generic dizziness screen by

Table 1: Outcome measures by clinical domain, with interpretation and limitations.

Domain	Candidate measures	Interpretation and limitation
Neck pain and disability	Numerical pain rating scale; Neck Disability Index	Measures pain or activity limitation; it does not identify a metabolic or dizziness cause [35].
Dizziness-related handicap	Dizziness Handicap Inventory	Captures perceived handicap and change; it does not identify the underlying cause [36].
Balance and mobility	Berg Balance Scale; Timed Up and Go; Functional Reach	Describes functional balance and mobility; selection should reflect setting and patient ability [37-39].
Sensory integration	Modified clinical test of sensory interaction on balance or comparable conditions	Helps describe reliance on visual, vestibular, and somatosensory inputs; it is not a metabolic diagnostic test [40].
Cervical sensorimotor function	Joint-position error and task-specific sensorimotor tests	May describe cervical sensorimotor impairment; it is not a stand-alone diagnosis of cervicogenic dizziness [32, 41].
Falls and participation	Falls diary, activity monitoring, exercise tolerance, and confidence measures	Captures meaningful risk and participation; results are influenced by comorbidity and environment.
Medical variables	HbA1c, glucose, electrolytes, thyroid and vitamin results	Record from medical documentation and communicate concerns; do not use as independent physiotherapy diagnoses.

untrained clinicians [33].

Supine and standing blood pressure and heart-rate responses may identify an orthostatic pattern when measurement is within local scope and equipment is available. Abnormal or symptomatic findings require communication with the medical team. Physiotherapists may record laboratory results from referral information but should not diagnose metabolic disease from symptoms or independently manage abnormal blood tests outside their scope.

Red flags

Urgent medical assessment is indicated for dizziness accompanied by new focal neurological signs, diplopia, dysarthria, severe or new headache, acute inability to walk, syncope, chest pain, or concerning cardiovascular symptoms. Neck pain with fever, malignancy history, unexplained weight loss, significant trauma, suspected fracture, or progressive neurological deficit also requires referral. Cervical vascular pathology cannot be excluded by a single provocative test; clinical reasoning and appropriate medical investigation are required [33, 34].

Outcome measures

Outcome measures should match the symptom phenotype and rehabilitation goal. None diagnoses a metabolic disorder. Table 1 summarises candidate measures, their interpretation, and their limitations.

Rehabilitation Implications

Management should be impairment-based and coordinated with medical care. For neck pain, education, exercise, and selected manual therapy may be used according to presentation and risk stratification [42, 43]. When peripheral vestibular hypofunction is established, vestibular rehabilitation has evidence and guideline support, but those recommendations should not be extrapolated automatically to non-specific dizziness or unconfirmed vestibular disease [44, 45].

Exercise planning for people with diabetes or other metabolic comorbidity should account for neuropathy, retinopathy, foot integrity, autonomic symptoms, cardiovascular risk, glucose-lowering medication, cognition, and falls history. Supervision, environmental safety, appropriate footwear, skin inspection when neuropathy is present, gradual position changes when orthostatic symptoms occur, and coordination with the medical team may be required. These are safety adaptations, not treatment of the underlying metabolic disease.

Balance, resistance, aerobic, and vestibular exercises may be combined when the examination supports them. Progression should use symptoms, blood-pressure response, perceived exertion, balance

quality, and functional goals rather than metabolic diagnosis alone. Weight-management and dietary advice should remain within professional scope and be coordinated with qualified medical or nutrition professionals.

Methodological Comparison of the Key Vestibular Evidence

The studies in Table 2 illustrate why the evidence should not be collapsed into one conclusion. They use different populations, designs, outcome definitions, and levels of vestibular measurement.

The methodological contrast is the central result of the comparison. Database studies can detect associations at scale but may lack symptom detail and physiological measurements. Objective vestibular studies can test a defined physiological function but may use smaller, selected samples. Comparative functional studies show patient impact but are vulnerable to residual confounding and cannot establish treatment effectiveness. This review preserves these distinctions instead of treating all vestibular evidence as interchangeable.

Research Gaps

Prospective studies should repeatedly measure diabetes status, medication exposure, neuropathy, autonomic function, visual disease, vestibular function, cervical sensorimotor control, physical activity, dizziness phenotype, and falls. This design would help establish temporal sequence and identify whether vestibular changes precede functional decline or simply coexist with it.

Future studies should separate vertigo, presyncope, light-headedness, disequilibrium, visual motion sensitivity, and exertional intolerance rather than use one undifferentiated dizziness item. They should report the diagnostic criteria and measurement properties of vestibular tests, distinguish type 1 from type 2 diabetes, and describe disease duration and complication burden.

Research should test the combined effects of distal sensory loss, visual impairment, vestibular function, and cervical sensorimotor impairment on balance using objective sensory-integration or posturographic methods. Studies should also control for age, renal disease, cardiovascular disease, obesity, cognition, medication burden, footwear, activity level, and fear of falling because these variables may explain part of the observed association.

Rehabilitation trials should prespecify metabolic phenotypes and report whether neuropathy, autonomic dysfunction, retinopathy, obesity, vitamin deficiency, or vestibular impairment modifies treatment response. Pragmatic multidisciplinary trials should

Table 2: Methodological comparison of the key studies of diabetes and vestibular dysfunction.

Study	Design and sample	Main finding	Methodological implication
Yang <i>et al.</i> , 2024 [9]	Population-based Taiwanese health-insurance database; 150,916 patients with newly diagnosed peripheral vestibular disorders and 452,748 propensity-score-matched controls. Administrative diagnoses of peripheral vestibular disorders and diabetes.	Diabetes was more common in the vestibular-disorder group; adjusted odds ratio for diabetes was 1.597 (95% CI 1.570 to 1.623).	Large sample and adjustment improve precision, but administrative diagnoses and cross-sectional association cannot establish causation or explain an individual dizziness phenotype.
Mahalingasivam <i>et al.</i> , 2024 [10]	Cross-sectional study of 114 participants with type 1 diabetes, type 2 diabetes, or no diabetes; vestibular function assessed by video head-impulse testing. Outcomes: vestibulo-ocular-reflex measurements, peripheral sensory testing, nerve conduction, autonomic questionnaire.	Vestibular measures did not differ between groups; all p-values were above 0.05, with only a small correlation between autonomic score and right lateral gain.	Direct objective testing supplies a negative finding, but cross-sectional data and the selected sample limit generalisation and cannot determine long-term falls consequences.
Komalasari <i>et al.</i> , 2024 [21]	Comparative study of 161 people with type 2 diabetes and vestibular dysfunction and 161 without; dysfunction assessed with head-impulse, Dix-Hallpike, and supine-roll tests. Outcomes: static and dynamic balance, falls-efficacy, social participation, quality of life.	The vestibular-dysfunction group had poorer balance performance, lower falls-efficacy, and lower social and quality-of-life outcomes.	Supports functional relevance when vestibular dysfunction is present, but group comparison cannot prove that diabetes caused the dysfunction or that treatment will improve all measured outcomes.

compare usual care with coordinated falls assessment, medication review, vestibular management, progressive exercise, and metabolic care, using falls, participation, quality of life, adverse events, and treatment adherence as outcomes.

Evidence is also limited by under-representation of younger adults, low- and middle-income settings, diverse ethnic groups, and people with multimorbidity. This is a limitation of the evidence base rather than evidence that a mechanism is absent.

Strengths and Limitations

This review narrows the clinical question, separates direct association from biological plausibility, includes a clearly negative vestibular finding, and places physiotherapy recommendations within diagnostic and referral boundaries. It also distinguishes evidence about neck pain from stronger evidence about dizziness, balance, autonomic symptoms, and falls.

Limitations remain. The review is narrative, has no registered protocol or formal risk-of-bias assessment, and synthesises heterogeneous definitions of metabolic disease, vestibular dysfunction, dizziness, balance, and neck pain. The original search approach was limited by its stated language and database boundaries, and the review cannot provide a pooled effect estimate. The evidence labels are descriptive rather than formal certainty ratings. The specific relationship between metabolic disease and common neck pain remains particularly under-studied.

Synthesis and Conclusion

Metabolic disorders are better supported as modifiers of dizziness, balance, falls risk, sensory integration, exercise tolerance, and rehabilitation safety than as direct causes of common non-specific neck pain. Diabetes is associated with peripheral vestibular-disorder diagnoses in population data, but objective vestibulo-ocular-reflex testing has also produced null group differences. The correct synthesis is therefore heterogeneous association, not a universal diabetes-to-vertigo pathway.

The clinical priority is to classify the symptom phenotype, review metabolic complications and medications, screen for neurological and cardiovascular danger signs, assess cervical, vestibular, neurological, visual, and balance systems within competence, and coordinate medical referral when indicated. Rehabilitation should target documented impairments and participation restrictions without

claiming to treat the underlying metabolic disorder. Prospective, phenotyped, multidisciplinary research is needed before stronger causal or treatment conclusions can be justified.

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Author Contributions

All author contributed equally to the conceptualization, drafting, and final approval of this letter .

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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