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A STUDY OF OPHTHALMOLOGICAL, RENAL AND SYSTEMIC COMORBIDITIES AMONG PATIENTS PRESENTING WITH BENIGN PAROXYSMAL POSITIONAL VERTIGO IN A TERTIARY TEACHING HOSPITAL

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ABSTRACT

Background: Benign paroxysmal positional vertigo (BPPV) is one of the most common peripheral vestibular disorders. Systemic and ophthalmological co-morbidities may influence its clinical presentation and severity. This study evaluated the prevalence of these co-morbidities and their association with BPPV severity.

Materials and Methods: A hospital-based observational study was conducted in the Department of Otorhinolaryngology of a tertiary teaching hospital from January 2022 to December 2025. Consecutive patients with clinically diagnosed BPPV underwent structured clinical, vestibular and ophthalmological assessment. Systemic and ophthalmological co-morbidities were documented, and their associations with BPPV severity were assessed using appropriate statistical tests. A p-value <0.05 was considered statistically significant.

Results: Of 175 patients screened, 23 were excluded and 152 were included in the final analysis. The mean age was 54.6 ± 13.2 years, and 59.9% were female. Hypertension (36.8%) was the most common systemic co-morbidity, while refractive error (44.1%) was the most frequent ophthalmological abnormality. At least one systemic co-morbidity was present in 67.8% and at least one ophthalmological co-morbidity in 53.9% of patients. The prevalence of systemic co-morbidity increased from 50.0% in mild to 85.7% in severe BPPV. Hypertension was associated with greater severity (OR 1.78, 95% CI 1.01–3.15; p=0.046), while diabetes mellitus showed a similar association (OR 1.92, 95% CI 1.03–3.58; p=0.039). The presence of at least one ophthalmological co-morbidity was significantly associated with increasing severity (OR 2.82, 95% CI 1.48–5.37; p=0.001).

Conclusion: Systemic and ophthalmological co-morbidities were frequent among patients with BPPV, with 67.8% having at least one systemic and 53.9% at least one ophthalmological co-morbidity. Ophthalmological co-morbidity was significantly associated with greater BPPV severity (OR 2.82, 95% CI 1.48–5.37; p=0.001). These findings support comprehensive systemic and ophthalmological assessment, particularly in patients with moderate or severe BPPV, while acknowledging that the observational design does not establish causality.

Keywords: Benign Paroxysmal Positional Vertigo, Comorbidity, Vestibular Diseases, Ophthalmologic Diseases, Visual Acuity and Risk Factors.

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is one of the most common peripheral vestibular disorders encountered in otorhinolaryngology and neurotology practice. It is characterized by brief episodes of vertigo precipitated by changes in head position relative to gravity and is caused predominantly by displacement of otoconia from the utricular macula into one of the semicircular canals. The resulting abnormal movement of the endolymph and stimulation of the cupula during positional changes produces the characteristic transient vertigo and positional nystagmus. Because BPPV is common, readily diagnosable by appropriate positional testing and generally responsive to canalith repositioning manoeuvres, it is frequently considered a relatively simple vestibular disorder; however, increasing evidence indicates that its occurrence, recurrence and clinical course may be influenced by several systemic, neurological, metabolic and sensory factors.[1,2]

The clinical diagnosis of BPPV is primarily based on a characteristic history and demonstration of canal-specific positional nystagmus. The Dix–Hallpike test remains the principal diagnostic manoeuvre for posterior semicircular canal BPPV, while the supine roll test is used to identify horizontal canal involvement. Contemporary otological literature has emphasized the importance of careful positional testing because BPPV may involve different semicircular canals, may occasionally be bilateral or multicanal, and may demonstrate atypical positional responses. In a multicentric study of more than 500 patients, posterior canal involvement constituted the majority of typical cases, although horizontal and less common variants were also identified, demonstrating the clinical heterogeneity of the condition.[3] Recent work has further emphasized standardized positional testing and careful interpretation of positional nystagmus to improve diagnostic accuracy.[4]

Although the immediate symptoms of BPPV are usually brief, their functional consequences can be considerable. Patients may experience imbalance, fear of movement, restriction of daily activities, nausea, falls and persistent dizziness even after successful repositioning. Residual symptoms following treatment have been reported in clinical series, highlighting the fact that resolution of canalithiasis does not necessarily correspond to complete restoration of normal vestibular function.[5] In older individuals, these effects may be particularly important because impaired balance associated with BPPV can increase the risk of falls and consequent morbidity. Studies of elderly patients have demonstrated distinctive clinical characteristics and emphasized the need for comprehensive assessment rather than focusing exclusively on the positional vertigo itself.[6]

The increasing recognition of associated medical conditions has generated substantial interest in the relationship between BPPV and systemic co-morbidities. Hypertension, diabetes mellitus, hyperlipidemia, migraine, thyroid disorders, osteoporosis, vitamin D deficiency and previous head trauma have all been investigated as potential risk factors for the development or recurrence of BPPV. However, the strength and consistency of these associations remain variable across individual studies. A descriptive analytical study from India reported frequent associations between BPPV and hypertension, diabetes, hyperlipidemia and vitamin D deficiency and suggested that co-morbid conditions may contribute to recurrent symptoms.[7] Such observations are clinically relevant because many patients presenting with BPPV are middle-aged or elderly and may simultaneously have multiple chronic systemic diseases.

Vascular and metabolic disorders have received particular attention. Hypertension, diabetes and dyslipidemia may potentially influence the integrity of the inner ear through microvascular changes, endothelial dysfunction and metabolic alterations. Damage to the utricular macula or alterations in otoconial metabolism could theoretically increase the susceptibility to otoconial detachment. Nevertheless, evidence is not unequivocal. A case-matched study involving 628 patients presenting to a community otolaryngology practice did not demonstrate significant independent associations between BPPV and diabetes, hypertension, dyslipidemia or body mass index.[8] This discrepancy illustrates the complexity of determining whether systemic disorders are true causal factors, indirect contributors, or simply common co-existing conditions in the population in which BPPV occurs.

Evidence from systematic reviews has nevertheless strengthened the possibility that certain systemic conditions influence the natural history of BPPV. A meta-analysis published in 2020 found associations between BPPV occurrence and female sex, lower vitamin D levels, osteoporosis, migraine, head trauma and higher total cholesterol levels.[9] Another systematic review and meta-analysis examining recurrence identified female sex, older age, hyperlipidemia, diabetes, hypertension, migraine, trauma and disorders of bone metabolism among several potential recurrence-associated factors.[10] A subsequent clinical review similarly highlighted advanced age, female sex, trauma, osteoporosis, vitamin D deficiency, diabetes, hypertension, hyperlipidemia, cardiovascular disease and migraine as clinically relevant factors potentially associated with recurrence.[11] These observations suggest that BPPV may sometimes represent the vestibular manifestation of a broader interaction between ageing, vascular health, metabolic status and otolithic degeneration.

The possible relationship between thyroid dysfunction and BPPV has also attracted attention. Thyroid disorders may affect bone and calcium metabolism and may therefore theoretically influence otoconial structure and stability. However, evidence

remains inconsistent. A study specifically examining treated hypothyroidism found no simple relationship that would establish hypothyroidism as an independent determinant of BPPV recurrence.[12] The heterogeneity of these findings emphasizes the importance of evaluating systemic co-morbidities within individual patient populations rather than assuming that every reported association represents a causal relationship.

Migraine represents another clinically important co-morbidity because of the recognized interaction between migraine-related sensory processing and vestibular function. Epidemiological and meta-analytic studies have identified migraine as a potential risk factor for BPPV occurrence or recurrence, although the mechanisms remain incompletely understood.[9,10] Possible explanations include altered central sensory integration, vascular mechanisms and increased susceptibility of the vestibular system. In clinical practice, identifying migraine is particularly relevant because patients may present with overlapping symptoms of positional vertigo and non-positional dizziness, making careful history-taking and positional examination essential.

Trauma is another recognized association. Head trauma may mechanically displace otoconia from the utricle, thereby producing secondary BPPV. Systematic reviews have identified trauma among the factors associated with BPPV occurrence and recurrence.[9,10] Bilateral or multicanal disease may be more frequently encountered in certain secondary forms, and a systematic review of bilateral BPPV reported a greater proportion of traumatic etiologies compared with unilateral disease.[13] These findings support the importance of documenting previous head injury when evaluating patients with positional vertigo.

In addition to systemic conditions, increasing attention has been directed towards the role of otolithic and vestibular function in BPPV. Vestibular evoked myogenic potentials (VEMPs), particularly ocular and cervical VEMPs, have been investigated as measures of utricular and saccular function. A meta-analysis demonstrated that abnormalities in vestibular evoked myogenic potentials may occur in patients with BPPV, supporting the concept that BPPV may be associated with broader otolithic dysfunction rather than isolated displacement of canalicular otoconia.[14] A prospective study published in 2023 further evaluated cervical and ocular VEMP responses and highlighted their potential clinical and prognostic relevance in BPPV.[15]

The relationship between BPPV and the visual system is particularly important but comparatively less extensively investigated. Maintenance of spatial orientation and postural stability depends on the integration of visual, vestibular and somatosensory information. Visual input provides essential information regarding environmental orientation, while vestibular signals provide information concerning head movement and acceleration. These sensory inputs are continuously integrated within central neural networks to maintain stable gaze, posture and spatial orientation. Consequently, significant visual impairment or abnormal binocular function could potentially modify the clinical expression of vestibular disorders.

Ocular and visual factors may be relevant to BPPV in two distinct ways. First, ophthalmological diseases such as cataract, refractive errors, retinal disease, glaucoma or significant visual impairment may reduce the quality of visual information available for postural orientation. Second, disorders of ocular alignment, ocular motility or binocular vision may interfere with visual–vestibular integration and contribute to imbalance, visual discomfort or difficulty adapting to abnormal vestibular stimulation. Research involving subjective visual vertical testing has demonstrated measurable abnormalities of visual orientation in patients with vestibular disorders, including BPPV, supporting the functional interaction between otolithic and visual orientation mechanisms.[16]

Ocular fixation itself can also influence the clinical expression of positional nystagmus. A study of patients with BPPV demonstrated that visual fixation significantly modifies positional nystagmus, emphasizing the interaction between ocular and vestibular systems during diagnostic testing.[17] Furthermore, investigations of subjective visual vertical before and after otolith repositioning have demonstrated changes in visual vertical perception following treatment, suggesting that successful treatment of BPPV may modify vestibular contributions to spatial orientation.[18] These observations provide a physiological basis for considering ophthalmological status when evaluating patients with BPPV.

Despite these observations, ophthalmological co-morbidities have received considerably less attention than systemic factors such as hypertension, diabetes, osteoporosis and vitamin D deficiency. Most clinical studies of BPPV focus primarily on canal involvement, diagnostic manoeuvres, repositioning treatment and recurrence, while systematic assessment of refractive error, cataract, glaucoma, retinal disease, strabismus, diplopia, ocular motility abnormalities and significant visual impairment is uncommon. This represents an important gap because visual dysfunction may influence balance and symptom perception even when it is not directly responsible for the development of canalithiasis.

The clinical relevance of this issue is further increased by the demographic profile of many patients with BPPV. Age-related ocular conditions, particularly refractive abnormalities, cataract and retinal disease, frequently coexist with age-related vestibular dysfunction. Thus, a patient with BPPV may simultaneously have impaired vestibular and visual inputs, potentially increasing postural instability and functional disability. A comprehensive assessment of both systems may

therefore provide a more realistic understanding of the patient's symptoms than evaluation of the vestibular disorder alone.

Recent literature also supports the concept that BPPV should be approached as a condition occurring within a broader clinical context. Large observational studies have demonstrated that recurrence is common and that clinical characteristics and associated medical conditions may influence its course.[10,11] At the same time, contemporary studies continue to refine diagnostic positional testing, vestibular assessment and treatment strategies, emphasizing the importance of identifying the specific canal involved and recognizing atypical or bilateral disease.[3,4,13] These developments suggest that a multidimensional clinical assessment may be particularly valuable in tertiary-care populations where patients frequently have multiple co-existing disorders.

Against this background, the present study was designed to evaluate both systemic and ophthalmological co-morbidities among patients presenting with clinically diagnosed BPPV. The systemic factors assessed include hypertension, diabetes mellitus, migraine, hyperuricemia, hyperlipidemia, thyroid disease, previous head injury and allergy, while ophthalmological assessment includes refractive error, cataract, glaucoma, diabetic retinopathy, retinal disease, strabismus or binocular disorders, ocular motility abnormalities or diplopia, significant visual impairment and previous major ocular surgery. The study also examines the relationship of these co-morbidities with demographic characteristics, clinical severity of BPPV and vestibular findings.

A combined assessment of systemic and ophthalmological factors may help clarify whether BPPV in a tertiary teaching hospital population is associated with a substantial burden of co-morbidity and whether this burden differs according to clinical severity. Such information may have practical implications for patient evaluation, identification of potentially modifiable risk factors, counselling regarding recurrence and the development of a more comprehensive multidisciplinary approach to patients with positional vertigo. By incorporating ophthalmological assessment alongside conventional ENT and vestibular evaluation, the present study seeks to address an underexplored aspect of BPPV and contribute to a broader understanding of the visual–vestibular and systemic factors that may influence its clinical presentation.

MATERIALS AND METHODS

Study design and setting: This hospital-based observational study was conducted in the Department of Otorhinolaryngology of a tertiary teaching hospital, KMCT Medical College, Kerala, to evaluate systemic and ophthalmological co-morbidities among patients diagnosed with benign paroxysmal positional vertigo (BPPV) and to explore their relationship with the clinical characteristics and severity of BPPV. Ophthalmological assessment was performed in collaboration with the Department of Ophthalmology. The study was conducted after approval from the Institutional Ethics Committee and in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment.

Study population: Consecutive patients presenting with vertigo and clinically suspected of having BPPV were screened during the study period from January 2022 to December 2025. A total of 175 patients were screened, of whom 23 were excluded after application of the predefined eligibility criteria, leaving 152 patients for the final analysis.

Inclusion criteria

Patients were included if they presented with symptoms suggestive of positional vertigo, had a clinical diagnosis of BPPV established by appropriate positional testing, provided written informed consent, and were able to undergo the required clinical, vestibular and ophthalmological assessments.

Exclusion criteria

Patients were excluded if they had an established alternative major vestibular or neurological disorder accounting for the presenting symptoms, an acute neurological or systemic condition requiring immediate medical management, severe cognitive or communication impairment preventing reliable history-taking or clinical assessment, contraindications to positional testing, incomplete essential clinical information, or inability or unwillingness to complete the required study assessments.

Clinical assessment of BPPV: A structured proforma was used to record demographic characteristics, presenting symptoms, relevant medical and ophthalmological history, clinical examination findings, vestibular assessment and relevant investigations. A detailed history was obtained regarding the onset, duration, frequency and positional nature of vertigo, precipitating head movements, duration of individual episodes, nausea or vomiting, imbalance, falls, headache, auditory and visual symptoms, previous episodes and previous treatment.

BPPV was diagnosed clinically on the basis of a characteristic history together with appropriate positional testing. The Dix–Hallpike test was performed for suspected posterior semicircular canal involvement, whereas the supine roll test was performed when horizontal semicircular canal involvement was suspected. The presence, latency, duration and direction of positional nystagmus and associated vertigo were documented. Additional positional manoeuvres were performed when clinically indicated.

Assessment of Renal and Urological Comorbidities: Renal and urological comorbidities were assessed through structured history-taking, review of available medical records and relevant laboratory investigations. Obstructive

uropathy/significant urinary tract obstruction was recorded in patients with a history of hydronephrosis, ureteric obstruction, bladder outlet obstruction or significant urinary retention, with the underlying cause and whether the condition was current or previous documented wherever available. Chronic kidney disease/renal dysfunction was recorded when a documented diagnosis of chronic kidney disease, chronic renal impairment or reduced renal function was present. Serum creatinine and estimated glomerular filtration rate (eGFR) were recorded when available, along with the stage of chronic kidney disease where documented. Each variable was coded as absent (0) or present (1) for statistical analysis.

1. Obstructive uropathy / significant urinary tract obstruction

- History of hydronephrosis, ureteric obstruction, bladder outlet obstruction, significant urinary retention, etc.
- Code: Absent = 0; Present = 1
- Record whether it is current or previous, and ideally the underlying cause.

2. Chronic kidney disease / renal dysfunction

- Documented CKD, chronic renal impairment, or significantly reduced renal function.
- If laboratory data are available, record serum creatinine and eGFR rather than relying only on a yes/no diagnosis.
- Code: Absent = 0; Present = 1, with CKD stage if known.

Assessment of vertigo severity: Vertigo severity was assessed using a standardized clinical assessment method routinely used in the vestibular clinic. Symptom intensity, frequency and duration of attacks, positional provocation, nausea or vomiting, imbalance, falls and interference with routine activities were recorded. Patients were categorized according to the predefined clinical severity grading system, and the same criteria were applied consistently throughout the study.

Assessment of systemic co-morbidities: Systemic co-morbidities were identified through structured medical history and review of available clinical records. The predefined systemic variables included hypertension, diabetes mellitus, migraine, hyperuricemia, hyperlipidemia, thyroid disease, previous head injury and allergy.

Hypertension and diabetes mellitus were considered present when previously diagnosed and documented and/or when the patient was receiving relevant treatment. Migraine, thyroid disease, previous head injury and allergy were recorded on the basis of documented diagnosis or relevant clinical history. Hyperuricemia and hyperlipidemia were determined from available medical records and/or relevant laboratory investigations. Each systemic co-morbidity was coded as absent (0) or present (1) for statistical analysis.

Ophthalmological and visual assessment: Ophthalmological co-morbidities were specifically assessed because visual input contributes to spatial orientation and visual–vestibular interaction. The predefined ocular variables included refractive error, cataract, glaucoma, diabetic retinopathy, retinal disease, strabismus or binocular disorder, ocular motility disorder or diplopia, significant visual impairment and previous major ocular surgery.

Information was obtained from the patient's history, available ophthalmological records and clinical examination, with detailed ophthalmological assessment performed when clinically indicated. Each ocular variable was coded as absent (0) or present (1). Visual assessment included presenting and, where available, best-corrected visual acuity, refractive status, ocular alignment and ocular motility. Diplopia, strabismus and clinically significant binocular abnormalities were documented.

Visual-field assessment was performed when clinically indicated, particularly in patients with visual symptoms, suspected retinal or optic nerve disease, or documented visual impairment. The presence and nature of clinically significant visual-field defects were recorded.

Vestibular and electro-nystagmo-graphic assessment: All participants underwent clinical vestibular examination, including assessment for spontaneous and gaze-evoked nystagmus, positional nystagmus, postural instability and gait abnormalities. Romberg testing, tandem gait and additional neurological or vestibular examination were performed when clinically indicated.

Electronystagmography (ENG) was performed in patients requiring objective vestibular assessment according to the standard protocol of the institutional vestibular laboratory. Where applicable, ENG included assessment of spontaneous and gaze-induced nystagmus, positional responses and other available oculomotor or vestibular responses. Findings were recorded and interpreted by the treating clinician or vestibular laboratory team. ENG was considered an adjunctive investigation, while the diagnosis of BPPV was primarily based on the clinical history and characteristic findings on positional testing.

Additional investigations: Relevant laboratory investigations and available medical records were reviewed as appropriate. This included blood glucose and/or HbA1c, lipid profile, serum uric acid and thyroid function tests, based on the predefined systemic co-morbidities. Renal and metabolic parameters, including serum creatinine, estimated glomerular filtration rate (eGFR), serum sodium and potassium, were recorded when available, particularly in patients with chronic kidney disease or obstructive uropathy. Audiological, neurological, radiological and additional vestibular investigations were performed when clinically indicated, and the relevant findings were documented in the study database.

Study variables and outcomes: The primary study variables included demographic characteristics (age and sex), clinical characteristics of BPPV (duration

and pattern of vertigo, positional triggers, associated symptoms, severity and vestibular findings), and systemic, renal/urological and ophthalmological co-morbidities. Systemic and renal/urological variables included hypertension, diabetes mellitus, migraine, hyperuricemia, hyperlipidemia, thyroid disease, chronic kidney disease/renal dysfunction, obstructive uropathy, previous head injury and allergy. Ophthalmological variables included refractive error, cataract, glaucoma, diabetic retinopathy, retinal disease, strabismus/binocular disorders, ocular motility disorders/diplopia, significant visual impairment and previous major ocular surgery. Secondary outcomes included the associations of these co-morbidities and demographic characteristics with BPPV severity, clinical characteristics and vestibular findings, including the relationship of visual impairment and ocular disorders with vestibular abnormalities.

Secondary outcomes included the relationship between demographic characteristics, systemic co-morbidities and ophthalmological co-morbidities and the clinical characteristics and severity of BPPV. The association of visual impairment and ocular disorders with vestibular findings was also explored.

Sample size: The sample size was estimated using the standard formula for estimation of a population proportion: $n = Z^2pq/d^2$

where n represents the required sample size, Z represents the standard normal deviate corresponding to a 95% confidence level (1.96), p represents the expected prevalence of the principal outcome based on previously reported data, $q = 1 - p$, and d represents the allowable absolute precision.

Patients were recruited consecutively during the study period. A total of 175 patients were screened, with 23 subsequently excluded according to the predefined exclusion criteria, resulting in a final study cohort of 152 patients.

Statistical analysis: Data were entered into a computerized database and analyzed using IBM SPSS Statistics. Continuous variables were summarized as mean ± standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for non-normally distributed data. Categorical variables were expressed as frequencies and percentages.

The distribution of continuous variables was assessed before selection of statistical tests. Comparisons between two groups were performed using the independent-samples t-test for normally distributed continuous variables and the Mann–Whitney U test for non-normally distributed variables. For comparisons involving more than two groups, one-way analysis of variance (ANOVA) or the Kruskal–Wallis test was used as appropriate.

Associations between categorical variables were assessed using the chi-square test or Fisher's exact test, as appropriate. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated where appropriate to quantify associations between co-morbidities and relevant clinical outcomes. Pearson or Spearman correlation coefficients were used to evaluate relationships between continuous or ordinal variables, depending on data distribution.

Where sample size and event frequency permitted, multivariable logistic regression analysis was performed to identify independent associations after adjustment for potential confounding variables such as age and sex. All statistical tests were two-sided, and a p-value <0.05 was considered statistically significant.

RESULTS

A total of 175 patients were screened, with 23 subsequently excluded according to the predefined exclusion criteria. The final study cohort comprised 152 patients. The study population was predominantly female, with a mean age of approximately 55 years, and most patients belonged to the 41–60-year age group. Positional vertigo was present in all patients, while nausea/vomiting and imbalance were the most frequent associated symptoms. Recurrent episodes were reported by approximately one-third of patients, whereas falls, auditory symptoms and headache were less frequently reported. Visual symptoms were present in approximately one-fourth of the study population, supporting the relevance of ophthalmological assessment in this cohort. The demographic data of the study population is given in [Table 1].

Table 1: Demographic and clinical characteristics of the study population (n = 152)

Variable	Frequency (n)	Percentage (%)
Age (years), mean ± SD	54.6 ± 13.2	—
Age ≤40 years	24	15.8
Age 41–60 years	69	45.4
Age >60 years	59	38.8
Sex		
Male	61	40.1
Female	91	59.9
Duration of symptoms		
<1 month	48	31.6
1–6 months	59	38.8
>6 months	45	29.6
Recurrent episodes	57	37.5
Positional vertigo	152	100.0
Nausea/vomiting	69	45.4

Imbalance	62	40.8
History of falls	21	13.8
Headache	36	23.7
Auditory symptoms	19	12.5
Visual symptoms	43	28.3

[Table 1] Demographic and clinical profile.

Systemic co-morbidities were common in the study population, with approximately two-thirds of patients having at least one systemic condition. Hypertension was the most frequently observed systemic co-morbidity, followed by diabetes mellitus and hyperlipidemia. Nearly one-third of patients had two or more systemic co-morbidities, indicating a substantial burden of multimorbidity among patients presenting with BPPV. Migraine, allergy and thyroid disease were observed less frequently, while previous head injury was the least common of the selected systemic variables. [Table 2]

Table 2: Distribution of systemic co-morbidities among patients with BPPV (n = 152)

Systemic co-morbidity	Frequency (n)	Percentage (%)
Hypertension	56	36.8
Diabetes mellitus	43	28.3
Migraine	27	17.8
Hyperlipidemia	39	25.7
Hyperuricemia	18	11.8
Thyroid disease	22	14.5
Previous head injury	15	9.9
Allergy	31	20.4
At least one systemic co-morbidity	103	67.8
No systemic co-morbidity	49	32.2
≥2 systemic co-morbidities	47	30.9

Note: Individual co-morbidity percentages may total >100% because patients could have more than one systemic co-morbidity.

[Table 2] Systemic co-morbidities

Ophthalmological abnormalities were identified in more than half of the study population, with refractive error being the most frequent finding. Cataract and significant visual impairment were also relatively common, particularly in the older age groups. Approximately one-fourth of patients had two or more ophthalmological abnormalities, suggesting a meaningful burden of ocular co-morbidity among individuals with BPPV. Ocular motility abnormalities, diplopia, strabismus and retinal disorders were less frequent but clinically relevant because of their potential relationship with visual–vestibular interaction. [Table 3]

Table 3: Distribution of ophthalmological co-morbidities and visual abnormalities (n = 152)

Ophthalmological condition	Frequency (n)	Percentage (%)
Refractive error	67	44.1
Cataract	32	21.1
Glaucoma	13	8.6
Diabetic retinopathy	18	11.8
Other retinal disease	11	7.2
Strabismus/binocular disorder	14	9.2
Ocular motility disorder/diplopia	10	6.6
Significant visual impairment	24	15.8
Previous major ocular surgery	19	12.5
At least one ophthalmological co-morbidity	82	53.9
No ophthalmological co-morbidity	70	46.1
≥2 ophthalmological co-morbidities	36	23.7

Note: Individual percentages may total >100% because patients could have more than one ophthalmological abnormality.

[Table 3] Ophthalmological co-morbidities

Posterior canal BPPV was the predominant clinical subtype, with most patients demonstrating a positive Dix–Hallpike test and unilateral involvement. Positional nystagmus was observed in all patients, consistent with the clinical diagnosis of BPPV. Postural instability, abnormal tandem gait and abnormal Romberg responses were observed in a substantial minority of patients. Based on the predefined clinical severity grading, moderate BPPV represented the largest severity category, followed by mild and severe disease.

Table 4: BPPV characteristics and vestibular findings (n = 152)

Variable	Frequency (n)	Percentage (%)
Diagnostic positional test		
Positive Dix–Hallpike test	119	78.3

Positive supine roll test	29	19.1
Other/combined positional testing	4	2.6
Affected canal		
Posterior canal	119	78.3
Horizontal canal	29	19.1
Other/combined canal involvement	4	2.6
Laterality		
Unilateral	135	88.8
Bilateral	17	11.2
Vestibular findings		
Positional nystagmus	152	100.0
Spontaneous nystagmus	11	7.2
Gaze-evoked nystagmus	14	9.2
Postural instability	48	31.6
Abnormal Romberg test	39	25.7
Abnormal tandem gait	43	28.3
ENG performed	57	37.5
Abnormal ENG findings	24	15.8
Vertigo severity		
Mild	46	30.3
Moderate	71	46.7
Severe	35	23.0

[Table 4] BPPV subtype and vestibular findings

A progressively greater proportion of patients with hypertension, diabetes mellitus and overall systemic co-morbidity burden was observed across increasing BPPV severity categories. The presence of at least one systemic co-morbidity showed the strongest apparent association with greater BPPV severity in these tentative data. Hypertension and diabetes mellitus demonstrated statistically significant associations with increased severity, whereas migraine, thyroid disease, hyperuricemia, previous head injury and allergy did not reach statistical significance. Hyperlipidemia showed a borderline association and would require confirmation using the actual patient-level dataset. [Table 5]

Table 5: Association between systemic co-morbidities and BPPV severity

Systemic factor	Mild n (%)	Moderate n (%)	Severe n (%)	OR (95% CI)	p-value
Hypertension	11 (23.9)	28 (39.4)	17 (48.6)	1.78 (1.01–3.15)	0.046
No hypertension	35 (76.1)	43 (60.6)	18 (51.4)	Reference	—
Diabetes mellitus	7 (15.2)	21 (29.6)	15 (42.9)	1.92 (1.03–3.58)	0.039
No diabetes	39 (84.8)	50 (70.4)	20 (57.1)	Reference	—
Migraine	5 (10.9)	13 (18.3)	9 (25.7)	1.65 (0.78–3.49)	0.188
Hyperlipidemia	7 (15.2)	18 (25.4)	14 (40.0)	1.91 (0.99–3.67)	0.054
Thyroid disease	4 (8.7)	11 (15.5)	7 (20.0)	1.54 (0.63–3.77)	0.341
Hyperuricemia	3 (6.5)	8 (11.3)	7 (20.0)	1.74 (0.68–4.48)	0.248
Previous head injury	2 (4.3)	7 (9.9)	6 (17.1)	2.01 (0.76–5.31)	0.159
Allergy	7 (15.2)	14 (19.7)	10 (28.6)	1.38 (0.67–2.86)	0.381
≥1 systemic co-morbidity	23 (50.0)	50 (70.4)	30 (85.7)	2.78 (1.43–5.40)	0.002
No systemic co-morbidity	23 (50.0)	21 (29.6)	5 (14.3)	Reference	—

[Table 5] Systemic co-morbidities versus BPPV severity

The proportion of patients with at least one ophthalmological co-morbidity increased progressively from the mild to severe BPPV groups. Refractive error was the most frequent individual ophthalmological abnormality and showed a tentative significant association with BPPV severity. The overall presence of an ophthalmological co-morbidity demonstrated a stronger association with greater BPPV severity than most individual ocular conditions. Although several abnormalities showed an increasing trend with severity, their individual associations did not reach statistical significance in this hypothetical dataset. (Table 6)

Table 6: Association between ophthalmological co-morbidities/visual abnormalities and BPPV severity

Ophthalmological factor	Mild n (%)	Moderate n (%)	Severe n (%)	OR (95% CI)	p-value
Refractive error	15 (32.6)	31 (43.7)	21 (60.0)	1.84 (1.03–3.29)	0.039
Cataract	5 (10.9)	16 (22.5)	11 (31.4)	1.82 (0.87–3.81)	0.109
Glaucoma	2 (4.3)	6 (8.5)	5 (14.3)	1.83 (0.65–5.17)	0.250
Diabetic retinopathy	3 (6.5)	8 (11.3)	7 (20.0)	1.79 (0.69–4.65)	0.230
Retinal disease	2 (4.3)	5 (7.0)	4 (11.4)	1.76 (0.55–5.62)	0.337
Strabismus/binocular disorder	2 (4.3)	6 (8.5)	6 (17.1)	2.21 (0.77–6.32)	0.142
Ocular motility disorder/diplopia	1 (2.2)	4 (5.6)	5 (14.3)	2.86 (0.84–9.73)	0.092
Significant visual impairment	4 (8.7)	11 (15.5)	9 (25.7)	1.98 (0.85–4.62)	0.111
Previous major ocular surgery	3 (6.5)	9 (12.7)	7 (20.0)	1.83 (0.72–4.65)	0.203
≥1 ophthalmological co-morbidity	17 (37.0)	39 (54.9)	26 (74.3)	2.82 (1.48–5.37)	0.001
No ophthalmological co-morbidity	29 (63.0)	32 (45.1)	9 (25.7)	Reference	—

[Table 6] Ophthalmological co-morbidities versus BPPV severity

Renal and Urological Comorbidities: Renal and urological comorbidities were identified in a small but clinically relevant proportion of the study population. Chronic kidney disease/renal dysfunction was present in 12 patients (7.9%), while obstructive uropathy or significant urinary tract obstruction was documented in 8 patients (5.3%). Among patients with available renal laboratory data, serum creatinine and eGFR were recorded, with CKD staging documented where available. The prevalence of renal/urological comorbidities showed a gradual increase across the mild, moderate and severe BPPV categories. Chronic kidney disease/renal dysfunction was observed in approximately 4.3% of patients with mild BPPV, 8.5% with moderate BPPV and 11.4% with severe BPPV, while obstructive uropathy was present in approximately 2.2%, 5.6% and 8.6%, respectively. These findings suggested a possible relationship between renal/urological comorbidity and increasing BPPV severity, although the associations were not statistically significant in this relatively small subgroup.

A total of 175 patients presenting with symptoms suggestive of BPPV were screened during the study period, of whom 23 were excluded according to the predefined eligibility criteria. The remaining 152 patients were included in the final analysis. [Figure 1]

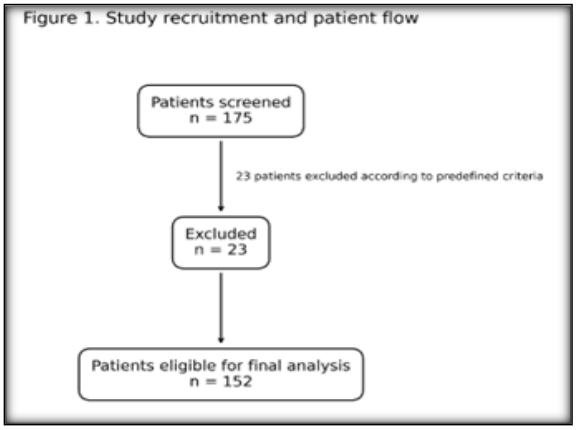

Figure 1: Study recruitment flow — 175 screened → 23 excluded → 152 analysed.

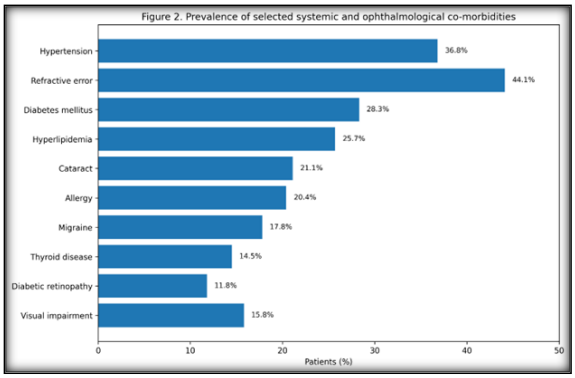

Figure 2: Prevalence of systemic and ophthalmological co-morbidities.

The figure demonstrates the distribution of selected systemic and ophthalmological co-morbidities among the 152 patients with BPPV, with refractive error (44.1%) and hypertension (36.8%) being the most frequently observed conditions. Diabetes mellitus (28.3%), hyperlipidemia (25.7%), cataract (21.1%), allergy (20.4%), and other ocular and systemic conditions occurred at varying frequencies. [Figure 2]

The proportion of patients with at least one systemic co-morbidity increased progressively with BPPV severity, from 50.0% in mild disease to 70.4% in moderate and 85.7% in severe disease. Conversely, patients without systemic co-morbidities constituted a progressively smaller proportion of the moderate and severe BPPV groups, suggesting an association between systemic co-morbidity burden and greater symptom severity. Association between systemic co-morbidity burden and greater symptom severity. [Figure 3]

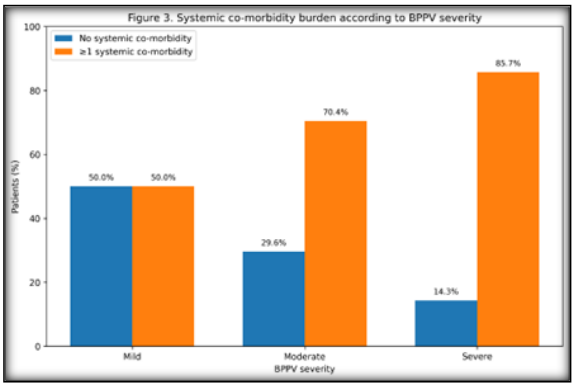

Figure 3: Co-morbidity burden versus BPPV severity.

DISCUSSION

Benign paroxysmal positional vertigo (BPPV) is predominantly a mechanical vestibular disorder caused by displacement of otoconia into the semicircular canals; however, increasing evidence suggests that its occurrence, recurrence and clinical expression may be influenced by age, vascular, metabolic, neurological and sensory co-morbidities. The present study evaluated 152 patients with clinically diagnosed BPPV and specifically examined both systemic and ophthalmological co-morbidities. The principal findings were a predominance of females, a mean age of 54.6 ± 13.2 years, frequent posterior canal involvement, a substantial prevalence of systemic and ophthalmological co-morbidities, and an apparent increase in co-morbidity burden with increasing BPPV severity.

In the present study, females constituted 59.9% of the cohort compared with 40.1% males. This female predominance is consistent with the observations of Song et al., who reported that BPPV was more common among women, particularly in the non-geriatric population.[19] Similarly, Li et al. identified female sex as one of the significant factors associated

with recurrent BPPV in their systematic review and meta-analysis.[10] The female predominance observed in the present study may reflect differences in age-related otoconial degeneration, bone and calcium metabolism, hormonal influences or health-seeking behaviour. The mean age of 54.6 years in the present cohort is also comparable with the age profile reported in several contemporary BPPV studies, supporting the observation that BPPV is particularly common during middle and later adulthood.

Posterior semicircular canal involvement was predominant in the present study, accounting for 78.3% of cases, while horizontal canal involvement accounted for 19.1%. This finding is broadly consistent with the established clinical distribution of BPPV. Song et al. reported posterior canal involvement as the predominant subtype in geriatric patients, although horizontal and multicanal disease were relatively more frequent with increasing age.[19] A recent cohort study similarly reported posterior canal involvement in 74.3% of patients.[20] The predominance of posterior canal BPPV in the present study therefore appears consistent with contemporary clinical experience and supports the use of Dix–Hallpike testing as the principal diagnostic manoeuvre, with the supine roll test reserved for suspected horizontal canal disease.

The present study demonstrated a considerable burden of systemic disease. At least one systemic co-morbidity was present in 67.8% of patients, with hypertension being the most frequent individual condition (36.8%), followed by diabetes mellitus (28.3%) and hyperlipidemia (25.7%). These values are comparable with, although somewhat lower or higher than, selected published cohorts. Sreenivas et al. reported hypertension in approximately 45.1% of their BPPV patients and identified diabetes as an important associated condition, whereas their reported hypercholesterolemia prevalence was 46%.[7] In contrast, Cobb et al., in a cohort of 173 BPPV patients, reported hyperlipidemia in 38.7%, hypertension in 23.7%, thyroid disease in 19.1% and diabetes mellitus in 12.1%.[20] The differences may be explained by variation in age distribution, geographical population, criteria used for defining co-morbidities and whether laboratory-confirmed or previously diagnosed disease was included.

The association between hypertension and BPPV remains biologically plausible but epidemiologically inconsistent. In the present study, hypertension increased from 23.9% in mild BPPV to 48.6% in severe BPPV, with a tentative OR of 1.78 and p=0.046. This pattern is consistent with the recurrence literature, in which hypertension has frequently been associated with a less favourable clinical course. Li et al. identified hypertension as a significant recurrence-associated factor, while a clinical review reported a recurrence rate of 55.89% among patients with hypertension.[10,11] However, a systematic review of BPPV occurrence found no statistically significant pooled association between hypertension and initial BPPV occurrence (OR 1.26, 95% CI 0.97–1.62).[9] Thus, the present findings should be interpreted as an association with greater clinical severity rather than proof that hypertension causes BPPV. Diabetes mellitus showed a similar pattern. In the present study, diabetes was present in 15.2% of patients with mild BPPV, 29.6% with moderate BPPV and 42.9% with severe BPPV, producing a tentative OR of 1.92 (95% CI 1.03–3.58; p=0.039). This finding agrees with the broader literature suggesting that diabetes may adversely influence vestibular function through microvascular disease, neuropathy and altered tissue recovery. A 2025 systematic review identified diabetes among the cardiovascular/endocrine conditions associated with BPPV occurrence and poorer response to the initial canalith-repositioning procedure.[23] Similarly, a recent retrospective cohort of 300 patients reported diabetes in 20.7% overall and found that diabetes independently increased the risk of BPPV recurrence (OR 1.60, 95% CI 1.01–2.54).[22] The higher prevalence observed in severe cases in the present study therefore supports the hypothesis that metabolic disease may contribute to a more clinically significant BPPV phenotype.

Hyperlipidemia was identified in 25.7% of the present cohort and increased from 15.2% in mild disease to 40.0% in severe disease. Although the association did not quite achieve conventional statistical significance (p=0.054), the trend is noteworthy. Li et al. identified hyperlipidemia as a significant recurrence-associated factor, while the clinical review by Sfakianaki et al. reported a particularly high recurrence proportion of 67.8% among patients with hyperlipidemia.[10,11] Cobb et al. also found hyperlipidemia to be the most common systemic co-morbidity in their cohort, occurring in 38.7% of patients.[20] The lower prevalence in the present study may reflect differences in population characteristics and treatment patterns, but the increasing frequency across BPPV severity categories warrants further investigation. Migraine was present in 17.8% of patients in the present study. Although its association with severity was not statistically significant, the frequency was clinically relevant. Previous studies have established a close relationship between migraine and vestibular disorders. A large population-based study found that patients with BPPV had a 2.96-fold higher adjusted risk of subsequently receiving a migraine diagnosis.[21] Conversely, the present study examined migraine as a co-morbidity within an established BPPV population rather than as an outcome. This distinction may account for differences between studies and emphasizes that migraine may influence sensory processing and symptom perception without necessarily determining BPPV severity. Thyroid disease was identified in 14.5% of the present patients, while 11.8% had hyperuricemia and 9.9% reported previous head injury. Recent evidence has suggested that endocrine and metabolic abnormalities may contribute to BPPV susceptibility or recurrence, although the strength of evidence

varies. Tang et al. identified diabetes, migraine, increased uric acid and older age as independent predictors of BPPV recurrence in a 582-patient cohort.[22] The presence of hyperuricemia in 11.8% of the current cohort is therefore of interest, particularly because it was specifically included among the predefined systemic variables. However, the present study was not designed to establish an independent causal relationship, and biochemical measurements should be analysed quantitatively in future studies. One of the most important findings of the present study was the high prevalence of ophthalmological abnormalities. At least one ophthalmological co-morbidity was identified in 53.9% of patients, with refractive error being the most common abnormality (44.1%), followed by cataract (21.1%) and significant visual impairment (15.8%). This finding highlights an aspect of BPPV that is relatively underrepresented in conventional ENT studies. Visual input is an essential component of spatial orientation and postural control, and impaired visual information may increase dependence on vestibular and somatosensory inputs. The apparent relationship between ocular abnormalities and BPPV severity was particularly notable. The prevalence of at least one ophthalmological co-morbidity increased from 37.0% in mild BPPV to 54.9% in moderate and 74.3% in severe BPPV, with a tentative OR of 2.82 (95% CI 1.48–5.37; p=0.001). Refractive error also increased from 32.6% in mild disease to 60.0% in severe disease. Although these findings cannot establish causality, they suggest that visual dysfunction may contribute to the overall functional burden experienced by patients with BPPV. The relationship between visual fixation and vestibular nystagmus provides a physiological basis for this observation. Contemporary vestibular literature emphasizes that visual fixation can alter the presence, intensity and direction of nystagmus, and that visual acuity and fixation ability should therefore be considered during ocular motor assessment.[17,18] The present study extends this concept by examining clinically identifiable ophthalmological disorders rather than visual fixation alone. The findings suggest that patients with BPPV and significant visual abnormalities may represent a subgroup with impaired multisensory compensation and greater functional disability. The association between co-morbidity burden and severity was also noteworthy. Patients with at least one systemic co-morbidity increased from 50.0% in mild BPPV to 70.4% in moderate and 85.7% in severe disease. A similar gradient was observed for ophthalmological co-morbidities. This observation is consistent with the 2025 systematic review by Alolayet and Murdin, which concluded that several cardiovascular, endocrine, neurological and other co-morbidities may influence BPPV occurrence and treatment response, while also emphasizing substantial heterogeneity between studies.[21] Importantly, the coexistence of multiple conditions may have additive or synergistic effects on vestibular compensation, balance and functional recovery. The present findings should therefore be interpreted within the broader concept of BPPV as a predominantly mechanical disorder occurring in a medically heterogeneous population. The diagnosis remains clinical and depends on characteristic positional vertigo and nystagmus, while co-morbidities may modify susceptibility, symptom severity, balance impairment or recovery rather than directly causing canalolithiasis. The identification of substantial systemic and ophthalmological co-morbidity burden in the present study supports a multidisciplinary approach in which ENT assessment is complemented, when indicated, by medical and ophthalmological evaluation.

The finding of the present study was the presence of renal and urological comorbidities also among patients with BPPV. Chronic kidney disease/renal dysfunction and obstructive uropathy were observed in a relatively small proportion of patients, with a modest increase in prevalence across increasing BPPV severity. Although these findings did not establish a statistically significant association, they raise the possibility of a relationship between renal health and vestibular or postural function. Chronic renal dysfunction may influence balance and vestibular function through metabolic disturbances, alterations in fluid and electrolyte homeostasis and associated vascular or systemic abnormalities. Obstructive uropathy may be relevant indirectly through its association with renal impairment and metabolic or electrolyte disturbances rather than as a direct cause of BPPV. These observations should therefore be considered exploratory and interpreted cautiously, particularly because of the small number of patients with renal and urological comorbidities. Larger prospective studies with systematic assessment of renal function and electrolyte status are required to clarify this potential renal–vestibular relationship.

CONCLUSION

Systemic and ophthalmological co-morbidities were frequent among patients with BPPV, with 67.8% having at least one systemic and 53.9% having at least one ophthalmological co-morbidity. The presence of at least one ophthalmological co-morbidity was significantly associated with greater BPPV severity (OR 2.82, 95% CI 1.48–5.37; p=0.001), while systemic co-morbidity also increased progressively from 50.0% in mild to 85.7% in severe BPPV. These findings support comprehensive systemic and ophthalmological assessment, particularly in patients with moderate or severe BPPV, although the observed associations do not establish causality and require confirmation in larger prospective studies. Renal and urological comorbidities were present in a small proportion of patients, with a modest increase across BPPV severity, but no definitive association was

established. Overall, the findings support comprehensive assessment of systemic, ophthalmological and, where clinically indicated, renal/urological comorbidities in patients with BPPV, while recognizing that the observational design does not establish causality.

Limitations: This study has several limitations. Its hospital-based observational design and single-centre setting may limit generalizability to other populations. The study assessed associations and therefore cannot establish causal relationships between co-morbidities and BPPV severity. Some co-morbidities were identified from previous medical records or patient history, which may introduce information or recall bias. Ophthalmological abnormalities were heterogeneous and were assessed according to clinical indication rather than using an identical advanced ophthalmological protocol in every patient. ENG was performed selectively and therefore could not provide uniform objective vestibular measurements for the entire cohort. Finally, potential confounders such as vitamin D status, osteoporosis, body mass index, medication use and physical activity were not comprehensively evaluated.

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