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Original Research Article

Vestibular Dysfunction in Post-Viral Syndrome: Clinical Trajectories and Neurophysiologic Correlates

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Abstract:

Background: Vestibular symptoms are commonly reported following viral infections, but the clinical course and objective neurophysiologic correlates that distinguish persistent post-viral vestibular dysfunction from transient vestibular complaints remain incompletely characterized.

Objective: To define clinical trajectories of vestibular symptoms after viral illness and to identify neurophysiologic markers that predict persistence versus recovery.

Methods: In a prospective longitudinal cohort study, adults presenting within 3 months of a documented viral illness with new vestibular symptoms (vertigo, imbalance, oscillopsia; n = 72) were enrolled and followed for 12 months. Baseline assessment included standardized symptom scales (Dizziness Handicap Inventory, Vertigo Symptom Scale), bedside neuro-otologic exam, video head-impulse testing (vHIT), caloric testing, cervical and ocular vestibular-evoked myogenic potentials (cVEMP/oVEMP), computerized dynamic posturography, and videonystagmography (VNG). A matched control group without vestibular complaints after viral illness (n = 30) underwent the same baseline testing. Participants were reassessed at 3, 6 and 12 months. Trajectories were defined by symptom resolution, intermittent symptoms, or persistent disabling dysfunction. Multivariable models examined baseline neurophysiologic predictors of persistent dysfunction.

Conclusions: Post-viral vestibular complaints follow heterogeneous clinical trajectories. A subset of patients develops persistent, objectively demonstrable vestibular dysfunction, with specific neurophysiologic abnormalities (reduced vHIT gain, abnormal VEMPs, caloric asymmetry) at presentation that predict poor recovery. Early multimodal vestibular testing can identify high-risk patients who may benefit from targeted vestibular rehabilitation and closer follow-up.

Keywords: Post-Viral Syndrome, Vestibular Dysfunction, vHIT, Vestibular-Evoked Myogenic Potentials, Caloric Testing, Posturography, Longitudinal.

Introduction

Vestibular disturbances are increasingly recognized as a significant component of post-viral syndromes, particularly in the aftermath of respiratory and neurotropic viral infections. Patients frequently report vertigo, chronic dizziness, imbalance, motion sensitivity, and cognitive–vestibular complaints such as “brain fog” associated with multisensory disequilibrium. While acute viral neuritis and labyrinthitis are well-established causes of transient vestibular dysfunction, a growing body of evidence suggests that a subset of individuals develop persistent or fluctuating vestibular symptoms that extend well beyond the acute illness. These prolonged manifestations impose substantial functional limitations, affect return to work, and diminish overall quality of life.

The mechanisms underlying post-viral vestibular dysfunction remain incompletely defined. Proposed pathophysiologic pathways include direct viral injury to vestibular hair cells or neurons, immune-mediated inflammation of the vestibular nerve, disruption of central vestibular processing, autonomic instability, and alterations in sensory integration. However, objective neurophysiologic markers capable of distinguishing transient post-infectious vestibular involvement from chronic post-viral sequelae remain limited. Standard clinical evaluations often fail to fully capture the complexity

of vestibular impairment, underscoring the need for multimodal and longitudinal assessment.

Recent advances in vestibular testing—such as video head-impulse testing (vHIT), vestibular-evoked myogenic potentials (VEMPs), video nystagmography (VNG), and computerized dynamic posturography—allow precise characterization of peripheral and central vestibular function. Applying these tools in a longitudinal framework may help delineate clinical trajectories and identify early predictors of persistent dysfunction.

This study investigates the natural course of vestibular symptoms following viral illnesses and evaluates neurophysiologic correlates associated with recovery versus persistent disability. By integrating clinical, functional, and neurophysiologic data over a 12-month follow-up period, we aim to improve understanding of post-viral vestibular syndromes and to inform early identification and targeted rehabilitation strategies for high-risk patients.

Material and Methods

Study Design: A prospective longitudinal cohort study was conducted over 18 months at a tertiary neurotology center. At Darbhanga Medical College and Hospital Laheriasarai. A total of 120 consecutive adult patients presenting with new-onset vestibular symptoms within 3 months of a documented viral illness were enrolled. A parallel control group of 40 age- and sex-matched individuals with recent viral illness but no vestibular complaints was also recruited for baseline comparison**.** Ethical Approval The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants.

Inclusion Criteria

- Age 18–70 years.
- Laboratory-confirmed or clinically diagnosed viral illness (respiratory, gastrointestinal, neurotropic, or systemic) within the preceding 12 weeks.
- New vestibular symptoms: vertigo, imbalance, unsteadiness, oscillopsia, motion sensitivity, spatial disorientation.
- Ability to complete serial follow-up assessments.

Exclusion Criteria

- Pre-existing vestibular disorders (Ménière's disease, BPPV, vestibular migraine).
- History of otologic surgery or significant hearing loss.
- Neurologic disorders affecting balance (stroke, demyelinating disease, peripheral neuropathy).
- Structural abnormalities on MRI/CT.
- Uncontrolled systemic illness limiting follow-up.

Clinical Assessment: At baseline and at 3, 6, and 12 months, the following evaluations were performed:

- Symptom scales:
  - Dizziness Handicap Inventory (DHI).
  - Vertigo Symptom Scale (VSS).
  - Fatigue Severity Scale (FSS).
- **Bedside neuro-otologic examination:**
  - Spontaneous/gaze-evoked nystagmus.
  - Head impulse test.
  - Romberg and tandem gait testing.

Neurophysiologic Testing

All patients and controls underwent standardized vestibular function testing:

1. Video Head Impulse Testing (vHIT)
   - Horizontal and vertical canal gains measured.
   - Presence and characteristics of corrective saccades documented.
2. Caloric Testing (ENG/VNG-calorics)
   - Unilateral weakness calculated using Jongkees' formula.
   - Directional preponderance recorded.
3. Vestibular-Evoked Myogenic Potentials (VEMPs)
   - cVEMP for saccular/inferior vestibular nerve function.
   - oVEMP for utricular/superior vestibular nerve function.
   - Latency, amplitude, and asymmetry ratios analyzed.
4. Computerized Dynamic Posturography
   - Sensory Organization Test (SOT).
   - Composite equilibrium scores.
   - Sensory integration patterns (somatosensory, visual, vestibular ratios).
5. Video nystagmography (VNG)
   - Smooth pursuit, saccades, optokinetic testing.
   - Positional testing for central and peripheral signs.

Classification of Clinical Trajectories: Based on symptom and functional trends at follow-up, patients were grouped into:

1. Rapid recovery: symptom resolution by 3 months.

2. Delayed recovery: improvement between 3–12 months.
3. Persistent dysfunction: significant symptoms and abnormal testing at 12 months.

Statistical Analysis

- Continuous variables analyzed via ANOVA or Kruskal–Wallis test.
- Categorical variables via chi-square or Fisher's exact test.
- Multivariable logistic regression to identify predictors of persistent dysfunction.
- Longitudinal changes assessed using mixed-effects models.
- Significance threshold set at $p < 0.05$.

Results

Participant Characteristics: Of the 120 enrolled patients, 114 completed the 12-month follow-up (6 lost to follow-up). The mean age was 42.3 ± 11.7 years, with a slight female predominance (56%). The most common antecedent viral illnesses were upper respiratory infections (48%), COVID-19 (31%), gastrointestinal infections (12%), and other systemic viral syndromes (9%). The control group (n = 40) showed no baseline vestibular abnormalities.

Clinical Trajectories

Three distinct outcome trajectories were identified:

1. Rapid recovery (n = 52; 45.6%).
    - Significant symptom resolution by 3 months.
    - Mean DHI dropped from 38.2 ± 14.7 at baseline to 8.4 ± 5.2 at 3 months.
2. Delayed recovery (n = 40; 35.1%).
    - Gradual improvement from 3 to 12 months.
    - Mean DHI decreased from 42.7 ± 13.5 to 12.6 ± 7.8 at 12 months.
3. Persistent dysfunction (n = 22; 19.3%)
    - Symptoms persisted at 12 months.
    - Baseline DHI was highest (56.1 ± 15.9) with only modest improvement to 34.8 ± 12.4 at 12 months ($p < 0.001$ vs other groups).

Patients with persistent dysfunction also reported higher fatigue, motion sensitivity, and oscillopsia scores across all time points.

Objective Neurophysiologic Findings

1. Video Head Impulse Testing (vHIT).
    - Reduced horizontal canal gains (<0.8) were noted in:
        - 18% of rapid recovery group.
        - 42% of delayed recovery group.
        - 73% of persistent dysfunction group.
    - Covert and overt corrective saccades were significantly more frequent in the persistent group ($p < 0.01$).
2. Caloric Testing
    - Clinically significant unilateral weakness (>25%) was observed in:
        - 21% of rapid recovery.
        - 38% of delayed recovery.
        - 64% of persistent dysfunction.
    - Directional preponderance >30% was also most prevalent in the persistent group.
3. Vestibular-Evoked Myogenic Potentials (VEMPs)
    - Absent or markedly attenuated cVEMP responses:
        - 12% in rapid recovery.
        - 28% in delayed recovery.
        - 59% in persistent dysfunction.
    - oVEMP abnormalities:
        - 16% in rapid recovery.
        - 35% in delayed recovery.
        - 68% in persistent dysfunction.

Both cVEMP and oVEMP abnormalities showed strong correlation with high DHI and poor functional outcomes.

4. Posturography (SOT Scores)
    - Composite scores were lowest in the persistent group (mean 51.6 ± 9.3) vs.
        - delayed (62.4 ± 7.8).
        - rapid recovery (72.8 ± 6.5).
    - Patients with persistent dysfunction demonstrated specific deficits in vestibular and visual-vestibular integration.
5. Video nystagmography (VNG)

- Abnormalities in smooth pursuit and saccades suggestive of central involvement were seen in 27% of persistent dysfunction patients, significantly higher than in rapid or delayed recovery groups ($p < 0.05$).
- Persistent positional nystagmus was more common in the delayed and persistent groups.

Predictors of Persistent Dysfunction: Multivariable logistic regression identified the following independent predictors at baseline:

Predictor	Adjusted OR	95% CI	p-value
vHIT horizontal gain < 0.8	3.9	1.8–8.2	<0.01
Absent cVEMP	4.6	2.0–10.2	<0.001
Caloric weakness > 25%	3.2	1.4–7.0	<0.01
High baseline DHI (>50)	2.8	1.3–6.1	0.02

Combined presence of vHIT reduction + absent cVEMP predicted persistent symptoms with 81% sensitivity and 78% specificity.

Longitudinal Changes

- Rapid and delayed recovery groups showed progressive normalization of vHIT gains and improved SOT scores by 12 months.
- Persistent dysfunction group showed minimal improvement, with stable or worsening VEMP abnormalities indicating chronic vestibular hypofunction.

Discussion

This prospective study provides a comprehensive evaluation of vestibular dysfunction following viral illnesses, highlighting the heterogeneous clinical trajectories and identifying neurophysiologic biomarkers that distinguish patients who recover from those who progress to chronic disability. With a cohort of 120 patients monitored for 12 months, the findings contribute to a growing recognition that post-viral vestibular syndromes extend beyond classical acute neuritis or labyrinthitis and may represent a spectrum of peripheral and central vestibular involvement triggered by viral or immune-mediated processes.

Heterogeneity of Clinical Trajectories: Three distinct outcome trajectories—rapid recovery, delayed recovery, and persistent dysfunction—underscore the variability of post-viral vestibular sequelae. Nearly one-fifth of patients manifested persistent symptoms at one year, consistent with emerging literature on post-viral and post-COVID vestibular disorders. These individuals presented with more severe symptoms at baseline, greater functional impairment, and limited improvement over time, indicating an early clinical phenotype of high-risk patients.

Pathophysiologic Insights: The neurophysiologic patterns identified suggest that persistent dysfunction is primarily associated with chronic peripheral vestibular hypofunction, involving both superior and inferior vestibular pathways. Reduced horizontal vHIT gains and abnormal caloric responses indicate impaired semicircular canal function, while absent or attenuated cVEMP and oVEMP responses reflect utricular and saccular involvement. This multisite deficit pattern may reflect viral-induced neuroinflammation, selective neural vulnerability, or persistent immune-mediated injury.

In addition, the higher prevalence of VNG abnormalities in pursuit and saccades among the persistent group suggests a subset of patients with central vestibular integration deficits, which may contribute to multisensory disorientation, cognitive-vestibular complaints, and delayed compensation.

Functional Correlates and Sensory Integration: Posturography findings demonstrate compromised vestibular reliance and sensory reweighting in patients with persistent symptoms. Unlike the rapid and delayed recovery groups—who showed gradual normalization of sensory integration—the persistent dysfunction group exhibited stable low composite scores and impaired vestibular ratios, emphasizing ongoing difficulty in adapting to vestibular deficits.

These findings underscore the importance of treating post-viral vestibular dysfunction not solely as a peripheral injury but as a condition affecting dynamic interaction between peripheral input and central processing.

Predictive Value of Early Neurophysiologic Testing: Among all measured variables, the strongest predictors of long-term dysfunction were:

- reduced horizontal vHIT gain,
- absent cVEMP responses,
- significant caloric weakness, and
- high baseline DHI scores.

The combination of vHIT and VEMP abnormalities provided the highest predictive accuracy, suggesting that multimodal vestibular testing early after symptom onset is crucial for prognosis. These objective markers can assist clinicians in identifying patients unlikely to recover spontaneously, and who may benefit from early vestibular rehabilitation, customized compensation strategies, and closer follow-up.

Clinical Implications: The study highlights several key implications for practice:

1. Routine evaluation of post-viral dizziness should include vHIT and VEMP testing rather than relying on bedside examinations alone.
2. Patients with multiple neurophysiologic abnormalities should be flagged for early vestibular rehabilitation therapy, as central mechanisms of compensation may be limited without guided intervention.
3. Recognition of central signs in VNG may direct multidisciplinary management, including neurorehabilitation and cognitive-vestibular therapy.

4. Understanding trajectory subtypes can improve patient counseling and help set realistic recovery expectations.

Limitations: Several limitations should be acknowledged. The study did not include viral subtype-specific analyses, which may reveal differences in neurotropism or immune mechanisms. While the follow-up period of 12 months captures major recovery patterns, longer-term data may identify late compensation in some individuals. Rehabilitation was not standardized across participants and may have introduced variability in outcomes.

Conclusion

This prospective study demonstrates that vestibular dysfunction following viral illness presents with heterogeneous clinical trajectories, ranging from rapid recovery to persistent long-term disability. Approximately one-fifth of patients experienced sustained vestibular symptoms at one year, characterized by significant functional impairment and limited physiologic compensation. Comprehensive neurophysiologic testing revealed that persistent dysfunction is associated with multisystem vestibular involvement, including reduced semicircular canal function, otolith pathway abnormalities, and impaired sensory integration.

Key baseline biomarkers—particularly reduced horizontal vHIT gains, absent or attenuated VEMP responses, and significant caloric weakness—emerged as strong predictors of chronic symptoms. When combined with high initial symptom burden, these markers reliably identified individuals at highest risk of poor recovery.

The findings underscore the clinical value of early multimodal vestibular assessment in patients with post-viral dizziness and highlight the need for timely, tailored vestibular rehabilitation strategies. Recognizing at-risk patients early in the course of illness may improve functional outcomes, reduce chronic disability, and guide patient-centered management in the growing population affected by post-viral syndromes.

Table 1: Baseline Demographic and Clinical Characteristics (N = 120)

Variable	Total Cohort (N = 120)	Rapid Recovery (n = 52)	Delayed Recovery (n = 40)	Persistent Dysfunction (n = 22)	p-value
Age, years (mean ± SD)	42.3 ± 11.7	41.1 ± 10.9	43.8 ± 12.2	44.6 ± 12.5	0.42
Female sex, n (%)	67 (56%)	28 (54%)	23 (57%)	13 (59%)	0.88
Recent viral illness type					0.71
- Respiratory	58 (48%)	25 (48%)	18 (45%)	11 (50%)	
- COVID-19	37 (31%)	16 (31%)	12 (30%)	7 (32%)	
- Gastrointestinal	14 (12%)	6 (12%)	5 (12%)	2 (9%)	
- Other systemic viral	11 (9%)	5 (9%)	5 (13%)	2 (9%)	
Time from infection to symptom onset (days)	14.2 ± 8.4	12.6 ± 7.8	15.4 ± 8.3	17.1 ± 9.2	0.09
Baseline DHI score	44.7 ± 16.1	38.2 ± 14.7	42.7 ± 13.5	**56.1 ± 15.9**	<0.001
Oscillopsia (n, %)	44 (37%)	13 (25%)	16 (40%)	**12 (55%)**	0.01

Table 2: Neurophysiologic Test Abnormalities Across Clinical Trajectories

Test and Parameter	Rapid Recovery (n = 52)	Delayed Recovery (n = 40)	Persistent Dysfunction (n = 22)	p-value
vHIT				
Horizontal canal gain < 0.8	9 (18%)	17 (42%)	**16 (73%)**	<0.001
Corrective saccades present	12 (23%)	21 (52%)	**18 (82%)**	<0.001
Calorics				
Unilateral weakness > 25%	11 (21%)	15 (38%)	14 (64%)	<0.001
Directional preponderance > 30%	6 (12%)	11 (28%)	10 (45%)	0.003
VEMPs				
Absent/low amplitude cVEMP	6 (12%)	11 (28%)	13 (59%)	<0.001
Absent/abnormal oVEMP	8 (16%)	14 (35%)	15 (68%)	<0.001
Posturography (SOT)				
Composite equilibrium score	72.8 ± 6.5	62.4 ± 7.8	51.6 ± 9.3	<0.001
Vestibular ratio deficit	14%	36%	68%	<0.001
VNG				
Central pursuit/saccade abnormalities	4 (8%)	8 (20%)	6 (27%)	0.04

Table 3: Multivariable Logistic Regression Predictors of Persistent Vestibular Dysfunction at 12 Months

Predictor	Adjusted Odds Ratio (OR)	95% CI	p-value
Horizontal vHIT gain < 0.8	**3.9**	1.8–8.2	<0.01
Absent/attenuated cVEMP	**4.6**	2.0–10.2	<0.001
Caloric weakness > 25%	**3.2**	1.4–7.0	<0.01
Baseline DHI > 50	**2.8**	1.3–6.1	0.02
Age > 50 years	1.4	0.6–3.1	0.31
Female sex	1.1	0.5–2.2	0.80

Table 4: Longitudinal Changes in Key Outcomes Over 12 Months

Parameter	Baseline	3 Months	6 Months	12 Months	p-value (trend)
Rapid Recovery Group					
DHI score	38.2 ± 14.7	8.4 ± 5.2	6.1 ± 4.3	4.3 ± 3.8	<0.001
vHIT gain	0.88 ± 0.09	0.92 ± 0.06	0.95 ± 0.05	0.96 ± 0.04	<0.001
SOT composite	68.1 ± 7.2	72.8 ± 6.1	75.2 ± 5.4	76.6 ± 4.9	<0.001
Delayed Recovery Group					
DHI score	42.7 ± 13.5	28.4 ± 10.2	18.6 ± 8.3	**12.6 ± 7.8**	<0.001
vHIT gain	0.83 ± 0.11	0.86 ± 0.09	0.90 ± 0.08	0.92 ± 0.07	<0.001
SOT composite	55.8 ± 8.7	59.9 ±			

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