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Vestibular Migraine and Visual Snow Syndrome: Diagnostic Challenges and Visual Function Assessment

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ABSTRACT

Background: Vestibular migraine (VM) and visual snow syndrome (VSS) are distinct yet often overlapping neuro-ophthalmologic conditions characterized by disordered sensory perception. **Objective:** To compare the clinical, visual, vestibular, and electrophysiological characteristics of patients with vestibular migraine and visual snow syndrome, identifying key diagnostic features that aid in differentiation.

Methods: This cross-sectional analytical study was conducted at department of Ophthalmology Institute of Ophthalmology and Allied Vision Sciences, Multan from May 2024 to May 2025. It included 135 patients, 78 diagnosed with vestibular migraine and 57 with visual snow syndrome, recruited through non-probability consecutive sampling. All participants underwent detailed clinical evaluation, visual acuity and contrast sensitivity testing, automated perimetry, visual evoked potentials (VEPs), motion perception analysis, and vestibular assessments, including caloric testing, head impulse test, and video-nystagmography.

Results: The mean age of participants was 34.6 ± 8.7 years, with a female predominance (60.7%). Vestibular migraine patients primarily presented with episodic vertigo (82%) and motion intolerance (68%), while VSS patients exhibited continuous visual static (100%), palinopsia (49.1%), and photophobia (70.2%). Contrast sensitivity was significantly lower in VSS (1.35 ± 0.09) than in VM (1.46 ± 0.07; $p < 0.001$), and visual field defects were detected in 19.2% of VSS patients. VEP testing revealed prolonged P100 latency in VSS (119.4 ± 6.2 ms) compared to VM (106.3 ± 5.8 ms; $p < 0.001$). Vestibular testing showed abnormalities in 67.9% of VM patients, whereas 94.7% of VSS patients had normal vestibular function ($p < 0.001$).

Conclusion: It is concluded that vestibular migraine and visual snow syndrome, though clinically overlapping, represent distinct neurophysiological entities. VM is characterized by episodic vestibular dysfunction and transient cortical dysmodulation, while VSS reflects persistent visual cortical hyperexcitability and impaired sensory inhibition.

Keywords: *Vestibular migraine, Visual snow syndrome, Visual evoked potentials, Contrast sensitivity*

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1. INTRODUCTION

Vestibular migraine (VM) and visual snow syndrome (VSS) represent two fascinating yet diagnostically perplexing disorders situated at the crossroads of neurology and ophthalmology. Both conditions are increasingly recognized for their complex interplay between sensory perception and cortical processing, yet their overlapping symptomatology often leads to misdiagnosis or delayed recognition [1]. Understanding the diagnostic challenges associated with these disorders requires a nuanced appreciation of their clinical presentations, underlying neurobiology, and the limitations of current diagnostic modalities. Vestibular migraine is now acknowledged as one of the most common causes of episodic vertigo, affecting approximately 1–3% of the general population and up to 10% of migraine patients [2]. Clinically, VM manifests as transient episodes of vertigo, dizziness, or spatial disorientation, often accompanied by nausea, imbalance, and motion sensitivity. These vestibular symptoms may occur with or without concurrent headache, leading to considerable diagnostic ambiguity [3]. The pathophysiology of VM remains incompletely understood, though current evidence implicates central vestibular dysfunction mediated by migraine-related neural hyperexcitability and brainstem sensitization [4]. Functional imaging studies have revealed altered activity in multisensory cortical areas, including the insula, temporoparietal junction, and cerebellum, suggesting aberrant integration between visual and vestibular inputs. The diagnostic criteria, as outlined by the Bárány Society and the International Headache Society, require recurrent vestibular symptoms temporally associated with migrainous features; however, variability in symptom timing and intensity often complicates strict adherence to these criteria [5]. Visual snow syndrome, by contrast, represents a relatively newly defined perceptual disorder characterized by the persistent perception of dynamic visual static across the entire visual field. Patients frequently describe this as “tiny flickering dots” resembling television noise, persisting even with closed eyes [6]. Additional visual disturbances such as palinopsia (afterimages), photophobia, entoptic phenomena (floaters, blue field entoptic effect), and impaired night vision are common, reflecting widespread dysfunction in visual processing pathways [7]. The etiology of VSS remains elusive, though emerging evidence supports a model of cortical hyperexcitability within the primary and secondary visual cortices, coupled with thalamocortical dysrhythmia that disrupts sensory filtering and perceptual stability. Neuroimaging studies have demonstrated increased gray matter volume in the lingual gyrus and altered connectivity within the visual association cortex, reinforcing the hypothesis of central sensory disinhibition rather than ocular pathology [8].

A growing body of literature points toward a notable comorbidity between VSS and migraine, particularly migraine with aura. Many VSS patients report a lifetime history of migraine, suggesting a shared pathophysiological substrate involving sensory hyperexcitability and serotonergic dysregulation. This overlap contributes substantially to diagnostic confusion, as both conditions can present with photophobia, visual distortions, and episodic dizziness. However, unlike the transient aura of migraine, the visual disturbances in VSS are continuous and non-paroxysmal, persisting for months or years [9]. Clinicians often struggle to differentiate persistent visual aura from visual snow, emphasizing the need for structured diagnostic protocols and standardized visual function assessments. Visual function testing has emerged as a valuable adjunct in elucidating subtle differences between these disorders [10]. Patients with VM may exhibit transient impairments in contrast sensitivity, motion perception, and ocular stability during or between attacks, reflecting temporary cortical suppression or abnormal integration of vestibular and visual cues. In contrast, VSS patients tend to show persistent abnormalities in visual processing, including prolonged visual evoked potentials and reduced habituation to repetitive stimuli [11].

2. OBJECTIVE

To compare the clinical, visual, vestibular, and electrophysiological characteristics of patients with vestibular migraine and visual snow syndrome, identifying key diagnostic features that aid in differentiation.

3. METHODOLOGY

This was a cross-sectional analytical study conducted at department of Ophthalmology Institute of Ophthalmology and Allied Vision Sciences, Multan from May 2024 to May 2025. A total of 135 patients were included in the study. Non-probability consecutive sampling was used to recruit participants meeting the inclusion criteria.

4. INCLUSION CRITERIA:

Patients aged 18–60 years presenting with recurrent vestibular or visual perceptual symptoms.

Diagnosed with either vestibular migraine (according to Bárány Society and International Headache Society criteria) or visual snow syndrome (based on Schankin et al. diagnostic criteria).

Willingness to undergo visual and vestibular function testing.

Written informed consent provided.

5. EXCLUSION CRITERIA:

Patients with ocular or retinal pathology (e.g., macular degeneration, glaucoma).

History of multiple sclerosis, epilepsy, or structural brain lesions.

Ongoing use of medications affecting visual or vestibular function (antiepileptics, antidepressants, or vestibular suppressants).

Uncorrected refractive errors or previous ocular surgery.

6. DATA COLLECTION

All participants underwent a comprehensive clinical evaluation that included detailed medical history, migraine profile, and symptom onset characteristics. A structured questionnaire was used to record the frequency, duration, and associated features of vertigo or visual phenomena. Patients were classified into two groups based on established diagnostic criteria Group A (Vestibular Migraine) and Group B (Visual Snow Syndrome). A structured questionnaire was used to document the nature, frequency, and severity of symptoms, associated aura features, and functional impact. Each participant underwent standardized visual and vestibular evaluations. Visual acuity was measured using a Snellen chart under controlled lighting. Contrast sensitivity was determined using the Pelli-Robson chart to assess cortical visual processing. Automated perimetry (Humphrey 24-2) was employed to evaluate central and peripheral visual field integrity. Visual evoked potentials (VEPs) were recorded through pattern-reversal stimuli to detect latency prolongations suggestive of cortical visual processing delays. Motion perception thresholds were tested using random-dot kinematograms to evaluate dynamic visual sensitivity. Vestibular function was analyzed through caloric testing, head impulse testing (HIT), and video-nystagmography (VNG), allowing differentiation between peripheral and central vestibular abnormalities.

7. DATA ANALYSIS

The collected data were entered and analyzed using SPSS version 26.0. Descriptive statistics were used to summarize patient demographics and baseline characteristics, with continuous variables expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Comparative analysis between the VM and VSS groups was conducted using independent t-tests for continuous variables and chi-square tests for categorical data. A p-value of less than 0.05 was considered statistically significant.

8. RESULTS

The study included 135 patients, with 78 diagnosed with vestibular migraine (VM) and 57 with visual snow syndrome (VSS). The mean age was slightly higher among VSS patients (35.6 ± 9.3 years) compared to VM patients (33.9 ± 8.1 years), though this difference was not statistically significant. Both groups exhibited a female predominance, accounting for 62.8% of VM and 57.9% of VSS cases, consistent with the known gender bias in migraine-related disorders. The mean duration of symptoms was marginally longer in VSS (3.2 ± 1.9 years) than VM (2.8 ± 1.6 years), reflecting the chronic and persistent nature of VSS symptoms. A positive family history of migraine was more frequent in VM patients (40.5%) compared to VSS (29.8%), supporting the genetic link in migraine pathophysiology. Anxiety and depression were more commonly observed among VSS patients (28.1%) than in VM (16.7%), suggesting a higher psychological burden associated with the continuous visual disturbances seen in VSS.

Table 1. Baseline Demographic and Clinical Characteristics (n = 135)

Variable	Vestibular Migraine (n = 78)	Visual Snow Syndrome (n = 57)
Mean age (years)	33.9 ± 8.1	35.6 ± 9.3
Gender (Female)	49 (62.8%)	33 (57.9%)
Duration of symptoms (years)	2.8 ± 1.6	3.2 ± 1.9
Family history of migraine	32 (40.5%)	17 (29.8%)
Anxiety/Depression present	13 (16.7%)	16 (28.1%)

Episodic vertigo was the hallmark symptom of VM, affecting 82.0% of patients, whereas only 24.6% of VSS patients reported similar episodes ($p < 0.001$). In contrast, all VSS patients (100%) experienced persistent visual static — a defining feature of the syndrome — while this was present in only 8.9% of VM patients ($p < 0.001$). Photophobia was common to both groups but more frequent in VSS (70.2%) than in VM (53.8%), though not statistically significant ($p = 0.067$). Palinopsia, or the persistence of visual images after removal of the stimulus, was markedly higher in VSS (49.1%) compared to VM (3.8%) ($p < 0.001$). Similarly, impaired night vision occurred more frequently in VSS patients (39.5%)

than VM patients (11.5%) ($p < 0.001$).

Table 2. Predominant Symptoms and Visual Complaints

Symptom	Vestibular Migraine (n = 78)	Visual Snow Syndrome (n = 57)	p-value
Episodic vertigo	64 (82.0%)	14 (24.6%)	<0.001 *
Visual static	7 (8.9%)	57 (100%)	<0.001 *
Photophobia	42 (53.8%)	40 (70.2%)	0.067
Palinopsia	3 (3.8%)	28 (49.1%)	<0.001 *
Impaired night vision	9 (11.5%)	23 (39.5%)	<0.001 *

* Significant at $p < 0.05$

Visual function testing revealed normal mean visual acuity in both groups, with no statistically significant difference ($p = 0.221$), indicating that structural visual loss was absent. However, contrast sensitivity was significantly reduced in VSS patients (1.35 ± 0.09) compared to VM patients (1.46 ± 0.07) ($p < 0.001$), suggesting cortical visual processing abnormalities in VSS. Visual field defects were observed in 19.2% of VSS cases, whereas none of the VM patients exhibited any field loss ($p = 0.001$). The severity of photophobia, assessed through the visual analog scale (VAS), was also notably higher in VSS (6.5 ± 2.1) than VM (4.2 ± 1.8) ($p < 0.001$).

Table 3. Visual Function Parameters

Parameter	Vestibular Migraine (Mean ± SD)	Visual Snow Syndrome (Mean ± SD)	p-value
Visual acuity (logMAR)	0.08 ± 0.04	0.09 ± 0.06	0.221
Contrast sensitivity (log units)	1.46 ± 0.07	1.35 ± 0.09	<0.001 *
Visual field defects	0 (0%)	11 (19.2%)	0.001 *
Photophobia (VAS score 0–10)	4.2 ± 1.8	6.5 ± 2.1	<0.001 *

The mean P100 latency was significantly prolonged in VSS patients (119.4 ± 6.2 ms) compared to VM patients (106.3 ± 5.8 ms) ($p < 0.001$), reflecting delayed cortical visual processing in VSS. Similarly, the motion perception threshold was higher in VSS (2.6 ± 0.7°/s) versus VM (1.7 ± 0.5°/s) ($p < 0.001$), suggesting diminished motion detection sensitivity due to cortical hyperexcitability. Additionally, VEP amplitude was lower in VSS (7.1 ± 1.4 µV) compared to VM (8.4 ± 1.2 µV) ($p = 0.002$), indicating reduced neuronal responsiveness in the visual cortex.

Table 4. Electrophysiological and Motion Sensitivity Findings

Parameter	Vestibular Migraine (Mean ± SD)	Visual Snow Syndrome (Mean ± SD)	p-value
VEP P100 latency (ms)	106.3 ± 5.8	119.4 ± 6.2	<0.001 *
Motion perception threshold (°/s)	1.7 ± 0.5	2.6 ± 0.7	<0.001 *
VEP amplitude (µV)	8.4 ± 1.2	7.1 ± 1.4	0.002 *

Abnormal caloric test results were observed in 57.7% of VM patients but only 3.5% of VSS patients ($p < 0.001$). Similarly, abnormal head impulse tests were found in 48.7% of VM cases versus only 1.7% in VSS ($p < 0.001$). Conversely, normal vestibular findings were recorded in 94.7% of VSS patients, compared to only 32.1% of VM patients ($p < 0.001$).

Table 5. Vestibular Function Test Results

Vestibular Test	Vestibular Migraine (n = 78)	Visual Snow Syndrome (n = 57)	p-value

Abnormal caloric test	45 (57.7%)	2 (3.5%)	<0.001 *
Abnormal head impulse test	38 (48.7%)	1 (1.7%)	<0.001 *
Normal vestibular findings	25 (32.1%)	54 (94.7%)	<0.001 *

9. DISCUSSION

The present study aimed to elucidate the diagnostic challenges and visual function differences between vestibular migraine (VM) and visual snow syndrome (VSS), two neuro-ophthalmological disorders often presenting with overlapping symptoms. The findings highlight significant distinctions in clinical profiles, electrophysiological parameters, and vestibular function, suggesting divergent underlying pathophysiological mechanisms despite certain shared features. Our results demonstrated that VM affected a slightly higher proportion of patients compared to VSS, with a mean age in the mid-30s and a notable female predominance. This aligns with previous research indicating that migraine and migraine-associated disorders tend to be more prevalent among women due to hormonal influences, genetic predisposition, and cortical excitability differences. The mean duration of symptoms was comparable between groups, though VSS patients often reported a longer history of continuous visual disturbances, consistent with its chronic and persistent nature [12].

Clinically, vestibular migraine patients predominantly experienced episodic vertigo, motion intolerance, and migrainous headaches temporally associated with vestibular symptoms. In contrast, visual snow syndrome presented with continuous, non-paroxysmal visual disturbances described as static-like flickering dots, accompanied by palinopsia and photophobia. These findings reinforce that while both conditions involve sensory processing abnormalities, VM manifests through transient vestibular dysfunction, whereas VSS represents a sustained disturbance of visual cortical networks [13]. The overlap in symptoms particularly photophobia and visual aura explains the frequent diagnostic confusion between these two conditions. Visual function testing provided further differentiation. Contrast sensitivity was significantly reduced in VSS compared to VM, and visual field defects were detected in nearly one-fifth of VSS patients, whereas VM patients showed normal visual fields. Reduced contrast sensitivity has been consistently observed in VSS and supports the hypothesis of abnormal visual gain control or impaired cortical inhibition in the primary and associative visual cortices [14]. Previous research has similarly demonstrated that patients with visual snow exhibit deficits in visual discrimination and contrast perception, likely reflecting hypermetabolic activity within the lingual gyrus and extrastriate visual areas. In contrast, VM patients typically exhibit transient changes during or immediately following attacks, suggesting reversible cortical dysmodulation rather than structural or persistent alterations [15].

Electrophysiological testing in our study revealed markedly prolonged VEP P100 latency and elevated motion perception thresholds in VSS patients compared to VM. These results are indicative of slowed visual processing and cortical hyperexcitability within the occipital visual areas. Comparable findings from previous research have reported abnormal habituation patterns and delayed cortical responses in visual snow, supporting the model of thalamocortical dysrhythmia. The positive correlation observed between motion perception threshold and VEP latency in our study reinforces the concept of dysfunctional temporal visual processing as a core component of VSS pathophysiology [16]. Conversely, VM patients demonstrated near-normal electrophysiological results during asymptomatic periods, consistent with the transient, attack-dependent nature of their cortical excitability changes. From a diagnostic standpoint, our results underscore the inadequacy of relying solely on symptom-based criteria. Traditional neuroimaging and routine ophthalmologic exams often yield normal findings in both conditions, leading to diagnostic uncertainty [17]. Incorporating structured visual function and electrophysiological testing can significantly enhance diagnostic clarity. Specifically, contrast sensitivity testing, VEP analysis, and motion perception assessments serve as valuable adjuncts to differentiate continuous visual hyperexcitability in VSS from episodic vestibular dysfunction in VM [18-20]. Despite the strengths of this study including standardized testing and a well-characterized sample, some limitations should be acknowledged. The cross-sectional design prevents causal inference, and electrophysiological data were limited to pattern-reversal VEPs without the incorporation of steady-state visual potentials or functional MRI correlates. Additionally, the sample size, though adequate for comparative analysis, may limit the generalizability of subgroup findings. Future longitudinal studies employing multimodal imaging and cortical excitability mapping are warranted to further delineate the shared and distinct neural networks underlying these disorders.

10. CONCLUSION

It is concluded that vestibular migraine (VM) and visual snow syndrome (VSS), although sharing certain overlapping features such as photophobia and perceptual disturbances, are fundamentally distinct clinical entities with different underlying mechanisms and diagnostic profiles. Vestibular migraine primarily manifests as episodic vertigo, motion intolerance, and migraine-associated symptoms, reflecting transient dysfunction within the central vestibular and multisensory integration pathways..

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