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Case Report

Status vestibular migrainosus as a cause for acute vestibular syndrome

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ABSTRACT

Acute vestibular syndrome (AVS) is characterized by sudden-onset, continuous vertigo lasting more than 24 hours, accompanied by nausea, vomiting, and worsening with head movement. Common etiologies include vestibular neuritis, labyrinthitis, and posterior circulation stroke. Because of overlapping neurovascular supply, peripheral and central vestibular disorders can share clinical signs such as hearing loss, nystagmus, abnormal vestibulo-ocular reflex (VOR), and unstable gait. Status vestibular migrainosus, an extended episode of vestibular migraine (VM) with persistent vertigo and disequilibrium for over 24 hours-may mimic AVS and is often difficult to distinguish from vestibular disorders. We report a patient with status vestibular migrainosus whose features mimicked AVS. The clinical picture fulfilled the diagnostic criteria for AVS, with features typically attributed to peripheral vestibular pathology. Comprehensive evaluation, however, supported a diagnosis of status vestibular migrainosus, highlighting the diagnostic challenge posed by overlapping peripheral and central vestibular signs. Status vestibular migrainosus can present as AVS and closely resemble peripheral vestibular disorders. Awareness of this entity and careful clinical assessment are critical to avoid misdiagnosis and prompt recognition is essential for accurate management and prognosis.

Keywords: Acute vestibular syndrome, Vertigo, Status vestibular migrainosus, Vestibular migraine

INTRODUCTION

Vestibular migraine (VM) is diagnosed clinically in patients who experience both migraine and vertigo, based on specific criteria established in the International Classification of Headache Disorders, 3rd edition (ICHD-3).[1,2] VM is recognized as the second most common etiology of recurrent vertigo and accounts for approximately 6-7% of individuals evaluated in dizziness clinics and about 9% of patients seen in migraine clinic populations.[3] Patients who present headache associated with dizziness are often diagnosed with VM, reflecting its significant impact. According to the current diagnostic framework of the Bárány Society and the ICHD-3, acute vestibular episodes attributed to VM are limited to a duration of 5 to 72 hours. Episodes persisting beyond this timeframe are, by definition, excluded from the present classification of VM.[4] Several researchers have highlighted that this presentation is considered conceptually comparable to status migrainosus, but with vestibular symptoms predominating, leading to the suggested term "status vestibular migrainosus."[5,6]

Status vestibular migrainosus is an emerging, descriptive term used by clinicians for a prolonged, severe attack of VM in which vertigo and other vestibular symptoms persist beyond the usual 5-72 hour window defined in current diagnostic criteria, often overlapping with status migrainosus of headache (lasting more than 72 hours).[5] It is increasingly identified as a leading non-ischemic cause of acute, spontaneous vertigo that persists for several hours to multiple days and can manifest with features of an AVS. Although exact data on the occurrence of status vestibular

migrainosus is not recognized its often underdiagnosed cause of AVS. Individuals may experience sudden-onset vertigo either spontaneous or position-triggered that can resemble other acute vestibular conditions such as labyrinthitis, vestibular neuritis or Meniere's disease. Accompanying symptoms often include sensitivity to light and sound and migraine-type headache, although vertigo may also occur in the absence of headache.

Proposed pathophysiological mechanisms for status vestibular migrainosus include transient hypoperfusion of the inner ear from labyrinthine artery vasospasm, abnormal activation of brainstem vestibular circuits, cortical spreading depression involving the vestibular cortex, and possible ion-channel dysfunction or inflammatory processes within the inner ear.[7,8]

Clear diagnostic guidelines for "status vestibular migrainosus" have yet to be defined, but the widely accepted standards for diagnosing VM, including its prolonged form is considered when the current episode occurs in the background of following criteria having been met: At least 5 episodes with vestibular symptoms of moderate or severe intensity, lasting 5 min to 72 hours. Current or previous history of migraine with or without aura according to the ICHD-3.

One or more migraine features with at least 50% of the vestibular episodes: headache with at least two of the following characteristics: one sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity, photophobia and phonophobia and visual aura.

Not better accounted for by another vestibular or ICHD diagnosis.

CASE REPORT

Description of episode and medical history

A gentleman in late 20's came with complaints of imbalance, nausea and epigastric discomfort on moving head. The patient was a surgical resident and reported that his symptoms started after finishing a night shift. He fell asleep at midnight and was awakened at 2:00 a.m. by a duty phone call that seemed unusually loud. After providing call orders, the patient returned to sleep. On waking at 6:15 a.m., the patient felt off-balance.

The patient left at 7:00 a.m. for early morning rounds. At 8:30 a.m., while speaking with colleagues and turning the head to the left, the patient experienced sudden imbalance, epigastric discomfort, and a brief spinning sensation. His symptoms worsened with head movements, but he was able to complete rounds. By 9:30 a.m., the combination of epigastric pain and imbalance had intensified to an intolerable level. Intramuscular prochlorperazine 12.5 mg was given at 10:30 am. Despite partial relief, he had a further episode of external vertigo at 15:30 pm the same day.

The patient reported a prior episode of imbalance lasting three days in 2011. Since childhood, there has been a history of motion sickness and intolerance to theme-park rides. Family history is notable for motion sickness in a sibling and episodic headaches in the mother. The patient experiences headaches triggered by sleep deprivation and missed meals.

Investigations

Magnetic resonance imaging (MRI) of the brain with contrast revealed no significant abnormalities.

A detailed vestibular evaluation was performed to assess vestibular function. The following equipment's were used during the assessment: cranio-oculography (COG)-EquiCOG (Equidor MedTech, LLP, Bangalore), functional head impulse test (Equi fHIT Equidor MedTech, LLP, Bangalore), computerized static posturography- Equidor MedTech, LLP, Bangalore, vestibular evoked myogenic potentials (VEMP)-Labat EcoVEMP and video head impulse test (vHIT)-ICS impulse system. Comprehensive cranio-oculography (COG) was performed to evaluate eye and head movements. Table 1 provides an overview of the patient's follow-up visits and cranio-oculography findings.

Both vHIT and fHIT were conducted to evaluate all semi-circular canals. The results of vHIT and fHIT testing are presented in Table 2. Improvement of vHIT test results in shown Figure 1 and fHIT test results in Figure 2.

Computerized static posturography assessment conducted over the course of the patient's follow-up demonstrated progressive and significant improvement in vestibular, somatosensory, and tandem scores, reflecting enhanced balance and postural control from the 1st visit to the 7th visit. The individual scores for each visit are presented in detail in Table 3.

Differential diagnosis

We considered the possibility of vestibular neuritis but was ruled out in view of absence of spontaneous nystagmus, normal VEMPS and classical hypofunction of semi-circular canal hypofunction in vHIT/fHIT. We also considered the possibility of first attack of Meniere's disease but there were no features of unilateral aural fullness or fluctuating hearing loss associated with the vertigo. Normal imaging further diminished the possibility of a vascular etiology that could explain the patient's symptomatology.

Treatment outcome and follow-up

The patient was given intravenous methylprednisolone 1g for 2 days to relieve the acute symptoms. Simultaneouly, he received oral flunarizine 10 mg twice daily for 2 months, and oral magnesium threonate with vitamin D3

once daily for 3 weeks. Following treatment, the patient noted substantial improvement in clinical symptoms over a period of one month.

Follow-up vestibular evaluation over course of a month revealed significant and progressive improvement across all domains. Specifically, vHIT and fHIT demonstrated recovery of semi-circular canal functions, COG showed normalized patterns, and posturography revealed marked gains in vestibular, somatosensory, and tandem scores. Collectively, these findings indicate substantial recovery of vestibular function from 1st-7th visit.

Table 1: Summary of follow-up visits and cranio-oculography test findings.

Visits	Tests	Findings
1st	Vertical saccades (0.3 Hz)	Slowing of saccades
	Horizontal smooth pursuit (0.2 Hz)	Low gain pursuit with compensatory catch-up saccades
	High-frequency head shaking	Biphasic nystagmus-initial left-beating followed by right-beating
	Clinical chair rotation	Prolonged post-rotatory left-beating nystagmus after rightward rotation.
3rd	High-frequency head shaking	No significant nystagmus
	Clinical chair rotation	No significant nystagmus
	McClure-Pagnini test	Left-beating nystagmus- all positions
	Dix Hallpike test (Right and left)	No significant nystagmus
4th	High-frequency head shaking	Right beat nystagmus.
	McClure-Pagnini test	Left-beating nystagmus-all positions
	Head positioning test	Head-yaw to the right and left side produced right torsional nystagmus, both without a crescendo–decrescendo pattern
	Pupillometry-Hippus	Negative
5th	Clinical chair rotation test	No significant nystagmus
6th	High-frequency head shaking	Right beat nystagmus
	Clinical chair rotation	No significant nystagmus
	Positional tests	No significant nystagmus
7th	Vertical saccades (0.3 Hz)	Normal
	Horizontal smooth pursuit (0.2 Hz)	Normal
	High-frequency head shaking	No nystagmus
	McClure-Pagnini test	No nystagmus
	Dix Hallpike test (Right and left)	No nystagmus
	Head positioning test	No nystagmus
	Clinical chair rotation	No nystagmus

Table 2: vHIT and fHIT findings across all semi-circular canals.

Visits	vHIT		fHIT	
	(Gain)		(Canal functioning)	
1st	Right lateral	0.77	Right lateral	Hypofunctional
	Left lateral	0.1	Left lateral	Hypofunctional
	LA	0.43	LA	Hypofunctional
	RP	0.52	RP	Hypofunctional
	RA	0.24	RA	Normal
	LP	0.59	LP	Hypofunctional
	Impression: All semi-circular canals were hypofunctional		Impression: All semi-circular canals were hypofunctional except the right anterior canal.	
3rd	Right lateral	0.85	Right lateral	Hypofunctional
	Left lateral	0.91	Left lateral	Normal
	LA	0.8	LA	Normal
	RP	0.79	RP	Borderline
	RA	0.69	RA	Borderline
	LP	0.8	LP	Hypofunctional
	Impression: Normal functioning of all semi-circular canals		Significant improvement seen with respect to the previous visits.	
7th	Right lateral	0.89	Right lateral	Borderline
	Left lateral	0.79	Left lateral	Normal
	LA	0.77	LA	Hypofunctional
	RP	0.74	RP	Borderline
	RA	0.79	RA	Normal
	LP	0.63	LP	Borderline
	Impression: Normal functioning of all semi-circular canals		Significant improvement seen with respect to the previous visits.	

*LA: Left anterior, RP: Right posterior, RA: Right anterior, LP: Left posterior.

Table 3: Posturography scores over seven follow-up visits.

Visits	Posturography		
	Vestibular (out of 100)	Somatosensory (out of 100)	Tandem (out of 100)
1st	36-Low	79.6-Normal	86.9-Normal
2nd	67-Borderline	76.9-Normal	100-Normal
3rd	60-Borderline	85-Normal	100-Normal
7th	88-Normal	85-Normal	100-Normal

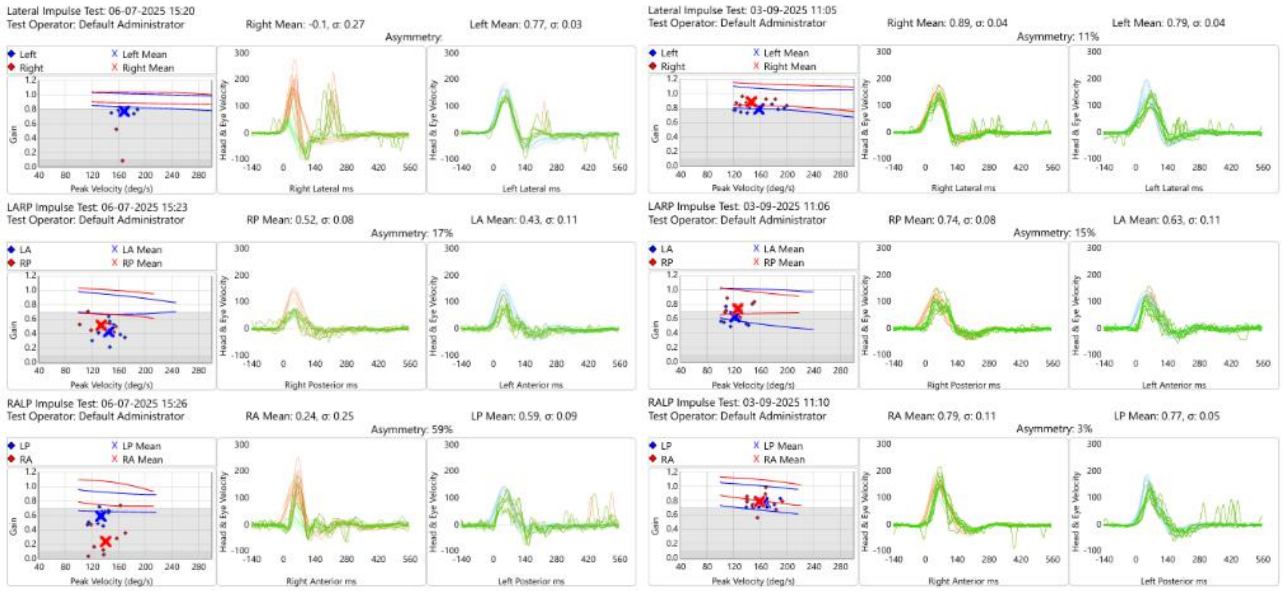

Figure 1: Evolution of vHIT test results from baseline to last visit.

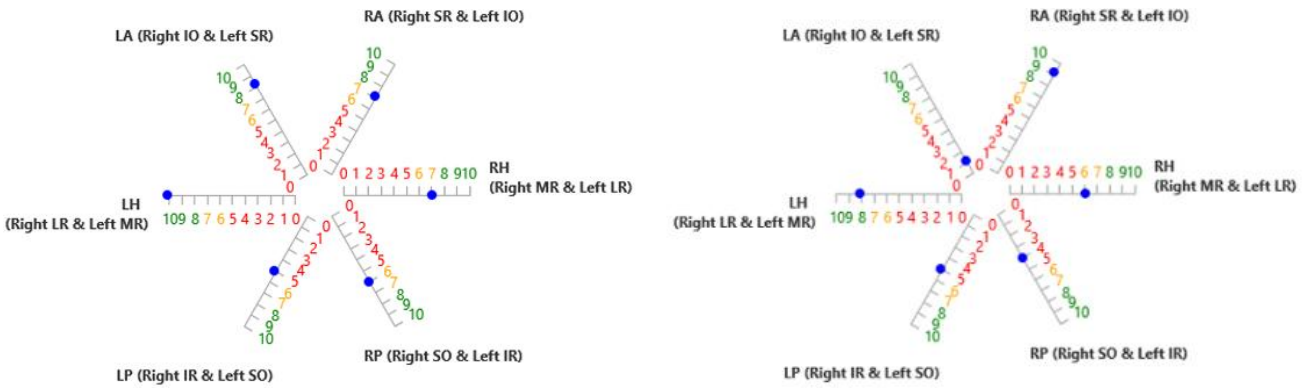

Figure 2: Evolution of fHIT test results from baseline to last visit.

DISCUSSION

Status vestibular migrainosus, although not yet a formally codified diagnostic entity, offers a useful framework to describe unusually prolonged, severe VM attacks that present as AVS. Current consensus criteria characterize VM as an episodic disorder, with discrete vertigo episodes of moderate to severe intensity lasting from 5 minutes to 72 hours. However, several observational series and expert reviews have identified a subset of patients whose vestibular symptoms persist beyond 72 hours or even become near-continuous, suggesting a chronic or status-like VM phenotype that challenges the existing time-bound definitions. Within this spectrum, the term "status vestibular migrainosus" has been proposed by analogy with status migrainosus in headache medicine, to denote a prolonged VM attack with sustained vertigo, motion intolerance, and migrainous features that fail to remit over days, yet lack evidence of vestibular pathology A history of repeated vestibular episodes accompanied by migraine symptoms is the key.

From a neuro-otological perspective, status vestibular migrainosus is important because VM is now recognized as one of the most frequent causes of spontaneous episodic vertigo and a leading vestibular mimic of AVS. Patients may present with continuous dizziness or vertigo, imbalance, nausea, and nystagmus, closely resembling vestibular neuritis or posterior circulation stroke, particularly when headache is minimal or absent. Nevertheless, careful evaluation often reveals features more typical of VM, including a history of migraine, photophobia or phonophobia, visual aura, motion sensitivity, or visually induced vertigo, as well as positional nystagmus that is with pattern a small shimmering movement (comes and goes off) multiple epochs of short-lasting nystagmus evoked in certain positions not corresponding to any canals. Reports of positional nystagmus in VM describe short-lasting, recurrent, and frequently canal-noncorresponding patterns, which differ from the stereotyped responses seen in benign paroxysmal positional vertigo and support a central, migraine-related mechanism when occurring in the context of AVS. Brain MRI with diffusion-weighted imaging can help confirm or exclude stroke, but early scans may yield false-negative results, underscoring the need for a thorough clinical evaluation.

Emerging pathophysiological work provides a plausible substrate for such status-like presentations. VM is thought to arise from abnormal interactions between the trigeminovascular system, vestibular nuclei, and multisensory cortical networks, leading to transient but sometimes prolonged dysregulation of vestibulo-thalamocortical processing. Experimental and clinical data indicate that trigeminal innervation of labyrinthine and brainstem structures, neurogenic inflammation, and reversible vasospasm of the internal auditory artery or its branches may contribute to acute vestibular and auditory symptoms during migraine attacks, while central sensitization and altered functional connectivity in temporo-parietal, insular, and limbic regions can perpetuate dizziness and vertigo.[7]

Clinically, recognizing status vestibular migrainosus as a cause of AVS has significant diagnostic and therapeutic implications. First, it underscores the need to consider VM in the differential diagnosis of acute, continuous vertigo once stroke, demyelination, and overt peripheral vestibulopathies (such as classic vestibular neuritis or initial Ménière's disease) have been reasonably excluded through neuroimaging, audiovestibular testing, and bedside oculomotor examination. Second, it justifies management strategies that mirror those used for severe migraine states, including the use of intravenous anti-migraine therapies or short steroid courses in the acute phase, followed by prophylactic agents such as calcium-channel blockers (e.g., flunarizine), beta-blockers, antiepileptic drugs, or antidepressants, alongside lifestyle and trigger modification. Narrative and practical reviews on VM treatment report that flunarizine and other migraine prophylactics can reduce vertigo burden and improve patient-reported outcomes, supporting a migraine-directed approach in prolonged attacks that present as AVS.[9,10]

CONCLUSION

The diagnosis of vestibular migrainosus is challenging due to the lack of specific biomarkers and significant overlap of its symptoms with other vestibular disorders. Excessive dependence on symptom patterns or positional triggers may lead to misdiagnosis. Additionally, vestibular function tests in these patients are frequently normal or yield nonspecific findings and there may be temporal discordance in findings depending on the status of migraine activity, in contrast to certain other vestibular conditions. A precise diagnosis depends on taking a detailed clinical history, ruling out other possible causes, and assessing for migraine-related features. Status vestibular migrainosus should be included in the differential diagnosis of AVS, particularly in patients with a history of migraine or concurrent migraine-type headache.

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