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REVIEW

Progress in the Study of Diagnostic Methods for Central Acute Vestibular Syndrome of a Vascular Cause

Jing Zhao[1], Yuan Xu[2], Jia Chen[3], Chong Shi[3], Xuefang Liu[1], Guihua Wang[1], Hongmei Zhang[1], Shaochen Ma[1], Paolo Candelaresi[4], Jilai Li[1], Peifu Wang[1], Jichen Du[1], Zhirong Wan[1]

[1]Department of Neurology, Aerospace Center Hospital, Beijing, 100049, People's Republic of China; [2]Department of Neurology, The First Affiliated Hospital of Jinzhou Medical University, Jinzhou, 121001, People's Republic of China; [3]Department of Traditional Chinese Medicine, Aerospace Center Hospital, Beijing, 100049, People's Republic of China; [4]Neurology and Stroke Unit, Azienda Ospedaliera Di Rilievo Nazionale Antonio Cardarelli, Napoli, Italy

Correspondence: Jichen Du; Zhirong Wan, Email djc189@126.com; wanzhirong@asch.net.cn

Abstract: Acute vestibular syndrome (AVS) is characterized by the sudden onset of dizziness or vertigo, accompanied by nausea, vomiting, gait instability, and nystagmus, lasting for more than 24 hours and often persisting for several days to weeks. Central AVS primarily involves central vestibular structures, such as the brainstem and cerebellum, and is most commonly caused by ischemic stroke in the posterior circulation. When acute posterior circulation infarction presents solely with isolated dizziness or vertigo, without other symptoms of central nervous system damage, it is often misdiagnosed as a peripheral vestibular disorder, this can lead to serious consequences. Therefore, distinguishing between central AVS and peripheral AVS in clinical practice is crucial, as the treatment strategies and prognosis differ significantly. Early identification of central AVS helps in adopting specific diagnostic and therapeutic measures. With advancements in vestibular and oculomotor theories, as well as neuroimaging, it is now possible to rapidly identify and diagnose central AVS of a vascular cause. This article summarizes recent diagnostic strategies, and discusses the progress in clinical and laboratory examinations for central AVS of a vascular cause presenting as isolated vertigo.

Keywords: acute vestibular syndrome, posterior circulation infarction, vascular vertigo, nystagmus

Introduction

Etiology of acute vestibular syndrome (AVS) can be classified into peripheral vestibular and central lesions. The former primarily involves the peripheral vestibular structures (inner ear and vestibular nerve), such as vestibular neuritis and migraine, accounting for over 75% of AVS cases.[1] The latter may affect central vestibular structures, including the vestibular nerve nuclei, the root of the eighth cranial nerve at the ponto-medullary junction, the cerebellar flocculus, and the nodulus. These structures participate in controlling the perception of head and body movements, generating vestibular-driven eye movements, processing visual signals, and maintaining balance and posture.[2,3] Therefore, dysfunction of the central vestibular system can lead to dizziness, vertigo, oculomotor disturbances, and postural instability. Central AVS (eg, stroke) account for approximately 20% of all AVS cases.[4] Central AVS is most commonly vascular in origin, primarily caused by ischemic stroke within the vertebrobasilar artery system,[5] and it can often present solely as isolated vertigo, making it difficult to recognize during initial diagnosis.[6] This article provides a detailed overview and summary of the recent diagnostic strategies and advances in clinical and laboratory testing for central AVS of a vascular cause, aiming to enhance clinicians' understanding of such disease and improve their ability to make differential diagnoses.

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Risk Factors of Central AVS

In neuro-otology outpatient clinics, approximately one-quarter of patients with vertigo have central AVS.[7] Central AVS constitutes 20%-30% of posterior circulation ischemic strokes, with vertigo or dizziness as the dominant manifestation.[8] While cases with neurological signs are easily recognized, isolated vertigo poses diagnostic challenges due to its mimicry of peripheral vestibular disorders (eg, vestibular neuritis).[9–12] Notably, 19%-42% of posterior circulation ischemia cases present with isolated vertigo,[13,14] and 20% lack focal neurological signs.[14] In emergency departments, central vascular lesions account for approximately 3% of AVS patients.[15] Comorbidities like atrial fibrillation or diabetes increase stroke risk sevenfold.[16] Despite extensive emergency evaluations,[17] emergency evaluations miss 35% of strokes,[18,19] partly due to false-negative MRI-DWI results in 50% of minor stroke-related isolated vertigo cases.[20]

Patients diagnosed with peripheral vertigo had a significantly higher risk of ischemic stroke than propensity score-matched controls with renal colic (50-fold higher at 7 days; RR=9.3 at 30 days).[21] And 0.4%-1.5% experiencing ischemic events within a year.[22] Overreliance on dizziness classification systems and imaging,[23] coupled with unstandardized bedside exams,[23] perpetuates diagnostic errors. These findings underscore isolated vertigo as a critical “red flag” for central AVS, demanding vigilance even without classic signs or positive initial imaging.[14,20,24]

Diagnostic Strategies for Central AVS

Clinical History Characteristics Aid in Diagnosis

For the differential diagnosis of central AVS, it is essential to thoroughly collect the patient’s clinical history and conduct detailed neurological examinations along with bedside tests. Traditionally, excessive emphasis has been placed on categorizing dizziness into specific types—such as vertigo (vascular), presyncope (cardiovascular), episodic (neurological), and nonspecific (psychiatric, metabolic)—to guide diagnosis. This approach has led to numerous cases of central AVS being missed or misdiagnosed.[25]

Recently, a rapid bedside diagnostic approach, the Triage, Timing, Targeted Examination algorithm (TiTrATE) method, has been proposed.[26] It classifies dizziness and vertigo based on the timing of onset (episodic or continuous), trigger factors (such as positional changes), and then conducts a Targeted Exam of eye movements to differentiate between peripheral and central AVS. In emergency patients presenting with intermittent or continuous dizziness, based on the timing and triggering factors from the clinical history, three syndromes may arise: 1) AVS: bedside examination helps differentiate vestibular neuritis from stroke. 2) Spontaneous episodic vestibular syndrome: Clinical history aids in distinguishing vestibular migraine from transient ischemic attack. 3) Triggered episodic vestibular syndrome: the Dix-Hallpike and roll test can be used to differentiate benign paroxysmal positional vertigo (BPPV) from central positional nystagmus caused by posterior fossa lesions.

All these three vestibular syndromes can be caused by vascular diseases, such as transient ischemic attack, ischemic, and hemorrhagic stroke[27,28] (Table 1).

Table 1 Vascular Factors Leading to Four Types of Acute Vestibular Syndromes

Syndrome	Syndrome	Ischemic Stroke	Hemorrhagic Stroke
Spontaneous AVS (>24h)	No reports (difficult to differentiate from vestibular migraine)	PICA – Isolated vertigo; AICA – Vertigo with or without tinnitus and hearing loss	Small to medium cerebral hemorrhage
Traumatic/Toxic AVS (>24h)	Overlapping syndrome with trauma and vertebral artery dissection/TIA	Overlapping syndrome with trauma and vertebral artery dissection/infarction	Overlapping syndrome with trauma and traumatic hemorrhage (subdural, subarachnoid)

(*Continued*)

Table 1 (Continued).

Syndrome	Syndrome	Ischemic Stroke	Hemorrhagic Stroke
Spontaneous episodic VS (<24h)	PICA – Isolated vertigo; AICA – Vertigo with or without tinnitus and hearing loss	Small infarcts with transient symptoms	Subarachnoid hemorrhage mimicking TIA
Triggered episodic VS	Vertebral artery rotation syndrome	CPPV from small infarct near the fourth ventricle	CPPV from small hemorrhagic foci near the fourth ventricle

Abbreviations: VS, vestibular syndrome; AVS, acute vestibular syndrome; CPPV, central paroxysmal positional vertigo; PICA, posterior inferior cerebellar artery; AICA, anterior inferior cerebellar artery; TIA, transient ischemic attack.

Vestibular and Oculomotor Bedside Examinations

When a patient presents with AVS accompanied by other neurological symptoms and signs, stroke can be diagnosed in most cases, even without neuroimaging. However, central AVS with subtle neurological deficits may be undetectable by MRI, presenting a diagnostic challenge even for specialists.[29] Recent advances in clinical neurology suggest that systematic bedside evaluation is more advantageous for identifying AVS caused by posterior circulation ischemia than neuroimaging.[30] Key vestibular and oculomotor examinations include various nystagmus examinations, the head impulse test (HIT), HINTS bedside test, and other diagnostic approaches.

Various Nystagmus Examinations

Simple downbeat, upbeat, or torsional nystagmus is a significant feature of central vestibular lesions. Other forms of central nystagmus include periodic alternating nystagmus,[31] see-saw or hemi-see-saw nystagmus,[32,33] and acquired pendular nystagmus.[34] Gaze-evoked nystagmus, which changes direction in the horizontal or vertical plane, often indicates impaired integration within the central nervous system network.[35] Intense, paradoxical downbeat nystagmus following horizontal head shaking is commonly seen in central lesions, such as stroke, degenerative diseases, and drug intoxication.[36] However, the direction of head-shaking nystagmus (HSN) depends on the location and extent of the central lesion, either ipsilateral or contralateral.[37,38] Intense horizontal HSN is frequently observed in lateral medullary infarctions.[39] A recent study suggests that enhanced anterior semicircular canal pathway responses may contribute to paradoxical downbeat HSN, representing one of the mechanisms of central lesions.[40]

In both central and peripheral vestibular diseases, positional changes can induce or modulate spontaneous nystagmus. Since central positional nystagmus can resemble the positional nystagmus of BPPV,[41] central lesions should be suspected in patients with persistent positional nystagmus despite repeated canalith repositioning maneuvers.[42] Particularly in cases of nodular and uvular damage, secondary vestibular neurons' responses to irregular afferent signals may be enhanced, leading to increased post-rotational signals and resulting in paroxysmal positional nystagmus.[43]

Head Impulse Test (HIT)

The bedside HIT is an effective tool for distinguishing central AVS from benign inner ear diseases.[44,45] It tests the vestibulo-ocular reflex (VOR) by turning the head rapidly to one side. A positive HIT, characterized by reduced VOR gain with corrective saccades, indicates peripheral vestibular hypofunction.[46] Lee et al[47] found that patients with isolated cerebellar infarctions consistently had normal bedside HIT results. However, when lesions involve specific central vestibular structures such as the vestibular nuclei,[48,49] flocculus,[50,51] or nucleus prepositus hypoglossi (NPH),[52] a positive HIT may be observed. Involvement of the unilateral flocculus or NPH often results in more prominent VOR gain reduction on the healthy side than on the affected side[52,53] (Table 2). A recent report indicated that approximately 20% of patients with posterior inferior cerebellar artery or superior cerebellar artery infarctions exhibited decreased VOR gain on the side contralateral to the lesion, with bilateral mean HIT gain of around 0.75, and 80% of these patients had abnormalities.[54] Therefore, although a negative HIT strongly suggests central AVS, a positive HIT is not an absolute marker of peripheral vestibular lesions. Moreover, other abnormal HIT patterns indicating central lesions include hyperactivity (increased gain with reverse corrective saccades) and paradoxical responses (upward eye

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Table 2 HIT Manifestations in Central Vestibular Structure Lesions

Lesion	Ipsilateral	Contralateral
Vestibular nucleus	P	N
Medial longitudinal fasciculus	N/P	N/P
Nucleus prepositus hypoglossi	N	P
Flocculus	N/P	P
Tonsil/nodulus/uvula	N	N
Diffuse cerebellar lesions	N/H/ cross-coupling	N/H/ cross-coupling

Abbreviations: HIT, head impulse test; P, Positive; N, Normal; H, Hyperactive.

movements during head turns with downward corrective saccades), previously reported in patients with diffuse cerebellar dysfunction.[40,55]

HINTS Bedside Test

The HINTS examination includes HIT, nystagmus assessment, and the test of skew.[44] Several studies have demonstrated that HINTS results are highly specific for distinguishing central from peripheral vertigo and are even more sensitive than early MRI-DWI, particularly in diagnosing lacunar infarction.[56] A study by Choi et al in Korea reported the use of HINTS in 34 patients with AVS caused by lacunar infarction, identifying central eye movement abnormalities in 33 cases, even though initial MRI-DWI scans in six patients did not detect lesions.[57] That same year, Kim et al conducted HINTS examinations on 91 AVS patients, with 7 of 8 stroke patients displaying central HINTS findings.[58]

It's noteworthy that HINTS is limited in its ability to detect anterior inferior cerebellar artery (AICA) infarction, with a false-negative rate of 17–29%.[59] This is because AICA supplies the inner ear and structures like the flocculus, and infarction in this region can result in both central and peripheral vestibular deficits.[60] A previously reported case of isolated unilateral flocculus infarction showed an increase in low-frequency horizontal VOR gain and decreased VOR gain during high-frequency stimuli. Despite HINTS often being normal in patients with central vestibular lesions, a positive HIT does not rule out cerebellar involvement affecting the flocculus.[61] Therefore, in elderly patients with sudden onset of unilateral hearing loss and vertigo, particularly in the presence of vascular risk factors, isolated labyrinth infarction should be considered. Incorporating horizontal head-shaking and finger-rubbing hearing tests (HINTS plus) is helpful in detecting central lesions, proving more convenient and effective than repeat MRI scans.[57]

Other Diagnostic Approaches

In addition to HINTS, other methods for diagnosing central AVS have been proposed. A retrospective study found that assessing gait and balance is crucial for ruling out AVS.[62] Although normal gait does not exclude cerebellar infarction, abnormal gait is a significant clue for diagnosing cerebellar stroke.[63] The ABCD2 score—accounting for age, blood pressure, clinical features, duration, and diabetes—is used to predict stroke risk in AVS patients. It was shown that 8.1% of AVS patients with a score ≥4 developed a stroke, increasing to 27% with a score of6−7.[64] However, HINTS is superior to the ABCD2 score in evaluating stroke risk in AVS patients.[65] Additionally, the recently reported the Triage plus Gait (TriAGe+) score, which includes eight variables [no triggers (2), atrial fibrillation (2), male (1), blood pressure >140/90 (2), brainstem or cerebellar dysfunction (1), focal weakness or speech disturbance (4), dizziness (3), no history of dizziness/vertigo or labyrinth/vestibular disease (2)], has proven to be more sensitive than the ABC Vestibular and oculomotor bedside examinations D2 score. With a score of 10, the sensitivity reaches 83.4%.[66] A Posterior Circulation Ischemia (PCI) Risk Score specifically designed to assess the risk of posterior circulation stroke was developed in 2018 with higher sensitivity and specificity, and is particularly well used in patients with dizziness as a primary symptom. The PCI scale diagnosed posterior circulation stroke with a much higher sensitivity and area under the receiver operating characteristic curve value (0.82) than the ABCD2 score (0.69).[67] Another scale that screens AVS symptoms such as imbalance, floating sensation, nonspecific dizziness, unsteadiness, and vertigo showed a sensitivity of 100%, significantly reducing the risk of misdiagnosis in AVS.[68] A new scale was recently developed. The Sudbury Vertigo Risk Score [Male (1), Age>65 (1), Diabetes (1), Hypertension (3), Motor/sensory (5), Cerebellar (6), BPPV diagnosis (−5)] effectively identifies the risk of a serious diagnosis in patients with dizziness. The risk of a serious diagnosis ranged from 0% for a score of <5 to 16.7% for a score >8. Sensitivity for a serious diagnosis was 100% and specificity was 69.2% for a score <5.[69]

Certain laboratory tests have been reported to be significant indicators for diagnosing AVS patients. The neutrophil-to-lymphocyte ratio (NLR) has become a widely used marker. An NLR >2.8 combined with the absence of horizontal nystagmus is a specific indicator for diagnosing stroke in AVS patients.[70] Elevated neuron-specific enolase levels in AVS patients are also independently associated with stroke.[71]

For years, the relationship between stroke risk factors and AVS has been a research focus. Univariate analysis has shown that age, diabetes, coronary artery disease, atrial fibrillation, and a history of dizziness are statistically significant.[72] Bi et al also proposed that AVS patients with three or more risk factors (male, age >60, hypertension, diabetes, smoking, and a history of stroke) have a significantly higher risk of posterior circulation infarction.[17] In addition to these factors, Kim et al demonstrated that in multivariate models, imbalance and extracranial atherosclerosis are independent risk factors for posterior circulation infarction[73] (Table 3).

Imaging Diagnosis

The sensitivity of computed tomography (CT) in detecting posterior circulation infarction is relatively low, only 16%.[76,77] Diffusion-weighted imaging (DWI) in MRI can detect 80% of infarct lesions. However, it is important to note that within 24–48 hours of onset, 15–20% of posterior circulation infarctions may be missed on MRI scans.[78] Perfusion-weighted imaging (PWI) helps identify posterior circulation ischemia, especially in patients with initial DWI-negative results. In a prospective cohort study, 12 out of 26 posterior circulation infarction patients presenting with AVS who were DWI-negative had decreased PWI perfusion. The sensitivity of combining HINTS plus balance testing reached 83%, and further integration with PWI improved the sensitivity to 100%. This shows that the combination of

Table 3 Diagnostic Methods for Central AVS

Test	Content	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Area Under ROC Curve
HINTS[44]	Head Impulse, Nystagmus, Test of Skew	100	96	99	100	0.995
STANDING[62]	Spontaneous and positional nystagmus, head impulse, balance	95	87	48	99	NC
ABCD2[64]	Age, blood pressure, clinical features, duration, diabetes	61[74]	62[74]	NC	NC	0.61[74] 0.82[63]
TriAGe+ Score[66]	No trigger, atrial fibrillation, sex, blood pressure, brainstem or cerebellar dysfunction, focal weakness or speech disturbance, dizziness, no history of vestibular disease	For a cutoff value of 10 points: 78	For a cutoff value of 10 points: 72	NC	NC	0.82
PCI score[67]	Hypertension, diabetes, a history of ischemic stroke, gait ataxia, limb sensory deficits, limb ataxia, vertigo, oscillopsia, speech difficulty, and tinnitus.	For a cutoff value of 0 points: 94	For a cutoff value of 0 points: 41	NC	NC	0.82
Sudbury Score[69]	Male, Age>65, Diabetes, Hypertension, Motor/sensory, Cerebellar, BPPV diagnosis	For a cutoff value of 5 points:100	For a cutoff value of 5 points:69	NC	NC	0.95
DEFENSIVE[68]	Instability, floating sensation, nonspecific dizziness, vertigo	94.8	83	58.6	98.4	NC
NLR >2.8 and no HN[70]	Neutrophil/lymphocyte ratio	NC	NC	NC	NC	0.84
Ultrasound[75]	VB intracranial and extracranial stenosis or occlusion	41	100	100	84	NC

Abbreviations: HINTS, Head Impulse, Nystagmus, Test of Skew; PCI score, posterior circulation infarct score; HN, horizontal nystagmus; NLR, neutrophil-to-lymphocyte ratio; PPV, positive predictive value; NPV, negative predictive value; NC, not calculated; VB, vertebrobasilar artery; ROC, receiver operating characteristic.

neurological and neuro-otological assessments (neurological examination + HINTS plus + balance test) with PWI can accurately identify posterior circulation infarction in patients presenting with AVS.[79]

When a vascular cause is suspected, neurosonography offers a non-invasive method to assist in diagnosis. In a study using duplex ultrasound of the vertebral artery, 25% (27/108) of AVS patients were diagnosed with posterior circulation infarction through ultrasound, showing that while the sensitivity of neurosonography is not high (40.7%), its specificity (100%), positive predictive value (100%), and negative predictive value (83.5%) are favorable[75] (Table 3).

Conclusions

Central AVS remains a diagnostic challenge, particularly when presenting as isolated vertigo without overt neurological deficits. This review highlights the critical importance of integrating clinical history, targeted bedside examinations (eg, HINTS, nystagmus assessments), and multimodal imaging to differentiate central AVS from peripheral vestibular disorders. Key findings include: 1) Bedside evaluation superiority: systematic oculomotor testing (eg, HINTS) demonstrates higher sensitivity than early MRI-DWI for detecting posterior circulation strokes, especially in lacunar infarctions. 2) Pitfalls in imaging: up to 20% of posterior circulation infarctions may be missed on initial MRI-DWI, necessitating adjunctive techniques like PWI or neurosonography when clinical suspicion persists.3) Complex vestibular pathways: lesions in specific central structures (eg, vestibular nuclei, flocculus) can mimic peripheral vestibular dysfunction, underscoring the need for nuanced interpretation of tests like the HIT. 4) Risk stratification tools: Scores such as TriAGe+ and PCI scale improve early identification of stroke risk in AVS patients, complementing traditional ABCD2 assessments. Although posterior circulation strokes account for the majority of central AVS cases, anterior circulation involvement (particularly insular, frontal, or parietal lesions) may rarely present with isolated vertigo, posing diagnostic challenges. AVS is not only seen in the posterior circulation but can also be seen in the anterior circulation.

It is critical to prioritize bedside examinations despite negative imaging, maintain high suspicion for central AVS in patient population at high-risk of stroke, and adopt a stepwise diagnostic algorithm to optimize outcomes. Moving forward, a multidisciplinary approach—combining advanced neuroimaging, laboratory biomarkers (eg, NLR), and dynamic follow-up—is essential to reduce misdiagnosis rates. Future research should focus on validating rapid diagnostic protocols and exploring the role of emerging technologies (eg, AI-assisted oculomotor analysis) in AVS management.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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