Acute Vertigo — Identifying Stroke Risk:

Recognising Posterior Circulation Stroke in the Dizzy Patient

Vestibular Medicine for General Clinicians

Topic 3 of 14

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How to Use This Review

This literature review is part of the Vestibular Medicine for General Clinicians series published by the Australian Dizziness Clinics Education Hub. It is written for general practitioners, hospital generalists, nursing, and allied health staff who assess and manage patients presenting with dizziness.

The review is designed to be read in a single 20–30 minute sitting, or used as a desktop reference. It is supported by an A4 one-page cheat sheet, short-form clinician videos, and audio episodes that cover the same material.

Callout Box Guide

□ **Key Point:** Foundational concepts and summary statements that anchor the core clinical content of each section.

□ **Clinical Insight:** Clinically relevant observations for direct application in assessment and management.

□ **Clinical Pearl:** High-yield memorable clinical points — the take-home messages most likely to change practice.

□ **Important:** Red flags, emergencies, and critical safety points requiring immediate action.

Table of Contents

I. The Problem — Stroke Hiding as Vertigo

II. Anatomy of Posterior Circulation — What Gets Infarcted

III. The Acute Vestibular Syndrome

IV. ABCD2 — Limited Utility in Vertigo

V. HINTS-Plus in the Acute Vertigo Patient

VI. Isolated Vertigo as Stroke — The Hardest Cases

VII. When to Refer to ED Immediately — Clinical Algorithm

VIII. Stroke Mimics and Vestibular Conditions That Feel Like Stroke

IX. Treatment and Disposition Principles

X. Safety-Netting, Documentation, and Key Messages

References

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I. The Problem — Stroke Hiding as Vertigo

Posterior circulation stroke is one of the most frequently missed diagnoses in emergency medicine. Large population-based studies estimate that 35–60% of patients with posterior circulation stroke present with dizziness or vertigo as a dominant symptom, and approximately 20% present with isolated vertigo without other focal neurological signs [1,2]. The diagnostic error rate for posterior circulation stroke presenting with dizziness is in the order of 30–50% at initial encounter — substantially higher than for anterior circulation stroke [3,4].

The consequences of a missed posterior circulation stroke are severe. Patients discharged with a misdiagnosis of "vestibular neuritis" or "benign vertigo" have a 7-fold increased risk of stroke or death within 30 days of the index visit [4]. Cerebellar stroke can cause malignant oedema with herniation within 24–72 hours, and early decompressive surgery is life-saving in carefully selected patients [5].

> □ **Important:**
> A patient with acute continuous vertigo is in the highest-risk diagnostic category in emergency medicine. Vestibular neuritis and cerebellar stroke can be clinically indistinguishable without a structured bedside examination [1,3].

Equally, the opposite error — over-investigation — generates its own harms. The vast majority of patients with acute dizziness in primary care do not have stroke, and CT scanning is both expensive and insensitive for posterior fossa lesions. The clinical challenge is not to image everyone; it is to identify the small group at high risk using history and examination, and to escalate them appropriately.

> □ **Key Point:**
> The question is not "does this patient have stroke?" but "is this acute vestibular syndrome peripheral or central?" Answering the second question correctly answers the first.

II. Anatomy of Posterior Circulation — What Gets Infarcted

The vertebrobasilar system supplies the brainstem, cerebellum, thalami, occipital lobes, and inner ear. The three branches most relevant to vertigo are PICA, AICA, and the superior cerebellar artery (SCA).

Posterior Inferior Cerebellar Artery (PICA)

PICA arises from the vertebral artery and supplies the inferior cerebellum (including the vermis) and the lateral medulla. Infarction of the medial PICA territory commonly presents with isolated vertigo, imbalance, and direction-changing nystagmus — the classic "pseudo-labyrinthitis" of stroke. The lateral medullary (Wallenberg) syndrome includes vertigo, Horner's syndrome, ipsilateral facial sensory loss, contralateral body sensory loss, dysphagia, and hoarseness [6].

Anterior Inferior Cerebellar Artery (AICA)

AICA arises from the basilar artery and supplies the lateral pons, middle cerebellar peduncle, flocculus, and — critically — the inner ear via the internal auditory artery. AICA infarction may therefore present with vertigo, ipsilateral sensorineural hearing loss, and ipsilateral facial weakness. This is the archetypal stroke mimic for labyrinthitis and is missed on history alone [7].

Superior Cerebellar Artery (SCA)

SCA territory infarction more commonly presents with gait ataxia, dysarthria, and limb ataxia, with less prominent vertigo. It is less commonly mistaken for a peripheral vestibular process.

Table 1 — Vascular Territories and Their Vertigo Presentations

Artery	Territory	Typical vertigo-relevant features
PICA (medial)	Inferior cerebellar vermis	Isolated vertigo, truncal ataxia,

		direction-changing nystagmus
PICA (lateral)	Lateral medulla	Wallenberg syndrome — crossed sensory findings, Horner's, dysphagia
AICA	Lateral pons, flocculus, inner ear	Vertigo + unilateral hearing loss + facial weakness — labyrinthitis mimic
SCA	Superior cerebellum	Gait ataxia, limb ataxia, dysarthria
Basilar (top-of-basilar)	Thalamus, midbrain, occipital	Altered consciousness, visual disturbance, vertical gaze palsy
Vertebral artery dissection	Variable	Neck or occipital pain, vertigo, PICA / AICA territory signs

Knowledge of vascular territory explains why "isolated vertigo" does not exclude stroke — PICA-medial and AICA infarction can present without any other focal signs detectable on routine examination [6,7].

> □ **Clinical Insight:**
> Vertebral artery dissection should be suspected in any patient under 50 with vertigo and neck or occipital pain, particularly after cervical manipulation, trauma, or unusual head positioning. Up to 20% of posterior circulation strokes in young adults are due to dissection [8].

III. The Acute Vestibular Syndrome

Acute vestibular syndrome (AVS) is the clinical syndrome of acute-onset, continuous vertigo lasting more than 24 hours, accompanied by nystagmus, nausea or vomiting, and gait unsteadiness [9]. It is the syndrome most commonly confused with stroke. The two leading causes are vestibular neuritis (peripheral) and cerebellar/brainstem stroke (central).

Within AVS, the peripheral-to-central ratio varies by population. In a general primary care population, vestibular neuritis is far more common. In an emergency department population with vascular risk factors, the pre-test probability of stroke is 20–25% — a prevalence that makes structured risk stratification essential [1,2].

Distinguishing Features — History

- **Onset: vestibular neuritis tends to build over hours to peak; stroke is often abrupt at maximum.**
- **Associated features: new headache (particularly occipital or neck), diplopia, dysarthria, dysphagia, limb weakness, hearing loss — all raise stroke probability.**
- **Vascular risk factors: age over 50, hypertension, diabetes, atrial fibrillation, smoking, hypercholesterolaemia, prior TIA / stroke.**
- **Triggering event: neck manipulation, trauma, sustained neck extension — consider vertebral artery dissection.**

> □ **Key Point:**
> In AVS, the history alone is inadequate to exclude stroke. Vestibular neuritis and cerebellar stroke can be indistinguishable on history. Bedside examination — not history alone — is the diagnostic step.

IV. ABCD2 — Limited Utility in Vertigo

The ABCD2 score was developed and validated to predict early stroke risk after TIA. Its components are: Age ≥60 (1 point), Blood pressure ≥140/90 (1 point), Clinical features (unilateral weakness 2, speech disturbance without weakness 1), Duration (≥60 min 2, 10–59 min 1), and Diabetes (1 point). A total of 4 or more is conventionally considered high-risk [10].

ABCD2 has been evaluated in the acute dizziness population, with disappointing results. In patients presenting with acute vertigo, ABCD2 has a sensitivity of approximately 61% and specificity of 62% for stroke — substantially inferior to HINTS (sensitivity 97%, specificity 95%) in the same population [11]. Its principal limitation is the weighting toward unilateral weakness and speech disturbance, which are often absent in posterior circulation stroke.

> □ **Important:**
> A low ABCD2 score does not exclude stroke in the acute vertigo patient. Relying on ABCD2 alone to discharge a patient with AVS is unsafe [11,12].

> □ **Clinical Insight:**
> ABCD2 retains utility as a communication tool — a score ≥4 supports escalation — but it cannot substitute for HINTS-plus in the acute vertigo cohort. Where AVS is the presentation, HINTS-plus is the primary risk stratification tool.

Table 2 — Comparative Performance of Stroke Risk Tools in Acute Vertigo

Tool	Sensitivity	Specificity	Comment
HINTS-plus (AVS only)	~99%	~97%	Highest-yield tool; requires AVS and active nystagmus
ABCD2 (all dizziness)	~61%	~62%	Under-performs HINTS; do not use in isolation
Early MRI-DWI (<48h)	~80%	~99%	May miss small brainstem strokes in first 48 hours
CT brain	~16%	~98%	Rules out haemorrhage only; poor for posterior fossa ischaemia
PCS score (clinical)	~80%	~80%	Emerging alternative when HINTS not feasible [12]

Performance data synthesised from Newman-Toker et al. [11], Saber Tehrani et al. [13], and GRACE-3 [14].

V. HINTS-Plus in the Acute Vertigo Patient

HINTS-plus combines the three-component HINTS examination (Head Impulse, Nystagmus, Test of Skew) with a bedside hearing screen. In adequately trained hands, it has a sensitivity approaching 100% and specificity above 95% for central cause in AVS — outperforming early MRI-DWI [11,14].

The Central Pattern (INFARCT)

- **I — Impulse Normal: no corrective saccade on head impulse testing.**
- **N — Fast-phase Alternating nystagmus: direction-changing gaze-evoked nystagmus.**
- **F — Refixation on Cover Test: skew deviation present.**
- **Plus — new unilateral hearing loss: raises concern for AICA territory.**

Any one of these findings, or the presence of new unilateral hearing loss, constitutes a "central" HINTS-plus and mandates urgent neuroimaging with MRI-DWI, ideally with MR angiography of the posterior circulation [14,15].

Operator Dependence and the Training Gap

HINTS has excellent performance in the hands of trained vestibular physicians and neuro-ophthalmologists. The sensitivity falls substantially when performed by emergency clinicians without structured training — to around 83% in some studies, with inter-rater agreement below 50% [16]. This is the single largest barrier to real-world implementation.

> □ **Clinical Pearl:**
> A HINTS examination that the clinician is not confident interpreting should not be used as the basis for discharge. An equivocal HINTS is not a reassuring HINTS. If in doubt, image or refer.

> □ **Key Point:**
> HINTS-plus in AVS: if all three components are clearly peripheral AND hearing is unchanged AND the clinician is experienced — peripheral cause is likely and imaging may be avoided. Any deviation from these conditions tips toward imaging.

VI. Isolated Vertigo as Stroke — The Hardest Cases

The greatest diagnostic challenge is the patient with acute vertigo and a near-normal examination. Up to 20% of posterior circulation strokes present this way [2]. Several scenarios should prompt concern:

Red-Flag Patterns in Isolated Vertigo

- **Age over 50 with vascular risk factors: raises pre-test probability substantially. A 65-year-old hypertensive smoker with new AVS has a 30–40% probability of stroke [2,11].**
- **Sudden onset at maximal intensity: vestibular neuritis typically builds over hours; abrupt onset favours vascular.**
- **Severe occipital or neck pain: dissection or cerebellar infarction; pain is rare in vestibular neuritis.**
- **Inability to stand or walk despite willingness to try: truncal ataxia is a cerebellar sign.**
- **New unilateral hearing loss with vertigo: AICA territory until proven otherwise; cannot exclude stroke with HINTS alone.**
- **Direction-changing nystagmus or vertical nystagmus: central, regardless of other findings.**

> □ **Important:**
> The "normal examination in an actively vertiginous patient" is itself a red flag. Patients with vestibular neuritis have florid peripheral findings — severe unidirectional nystagmus, obvious head impulse deficit. An absence of clear peripheral findings in a patient who is prostrate with vertigo should be treated as central until excluded [3].

Imaging in Isolated Vertigo

Non-contrast CT has a sensitivity of approximately 16% for acute posterior fossa ischaemia and should not be used to exclude stroke in this context; it remains useful only to exclude haemorrhage [13]. MRI with diffusion-weighted imaging is the imaging modality of choice, but within the first 24–48 hours it may miss up to 20% of small brainstem strokes [13]. In these cases, repeat imaging at 72 hours, or expert clinical review, is required.

> □ **Clinical Insight:**
> An MRI-DWI within 48 hours of symptom onset that is read as normal does not conclusively exclude a small brainstem stroke. If HINTS-plus is central or clinical suspicion remains high, escalate to vestibular physician review and consider repeat imaging at 72 hours or MR angiography [13,15].

VII. When to Refer to ED Immediately — Clinical Algorithm

The decision to refer urgently to emergency care is driven by history features, examination findings, and the patient's risk profile. The algorithm below synthesises the evidence-based approach recommended by GRACE-3 [14] and the Australian Stroke Foundation acute management guidelines.

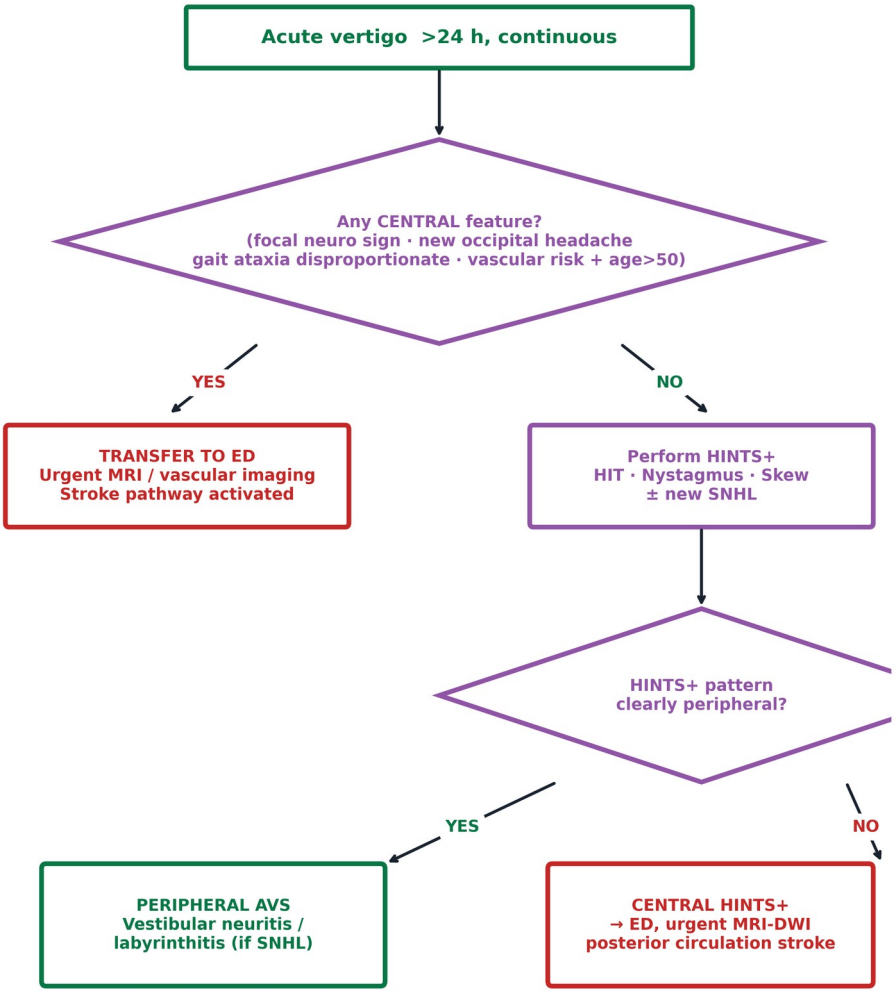

Figure 1. Acute Vertigo Stroke-Risk Triage Algorithm — bedside risk stratification for the patient with acute continuous vertigo.
Source: Australian Dizziness Clinics — clinical algorithm.

> □ **Key Point:**
> Any central HINTS finding, any new unilateral hearing loss with vertigo, any truncal ataxia, any new focal neurological sign — direct transfer to ED, not a routine referral.

VIII. Stroke Mimics and Vestibular Conditions That Feel Like Stroke

Just as stroke can mimic vestibular disease, vestibular disease can feel like stroke to the patient. Distinguishing mimics reduces unnecessary imaging without missing genuine pathology.

Vestibular Migraine Presenting Acutely

Vestibular migraine can present as acute severe vertigo with headache, photophobia, and visual aura. Episodes typically last minutes to 72 hours. A personal or family history of migraine, typical migrainous features, and spontaneous resolution without residual neurological signs are reassuring. However, a first episode in an older patient with vascular risk factors cannot be distinguished from TIA without examination and, often, imaging [17].

Ménière's Disease

Ménière's presents with episodic vertigo lasting 20 minutes to several hours, with associated aural fullness, tinnitus, and fluctuating low-frequency hearing loss. Between episodes, examination is generally normal. A first episode without established pattern may require the same triage as new-onset episodic vertigo.

BPPV Misinterpreted as Stroke

BPPV can feel catastrophic to the patient, particularly during the first episode. The key discriminator is episode duration — seconds — and a clear positional trigger. A structured Dix-Hallpike in primary care will confirm the diagnosis without imaging in the vast majority.

Functional (Psychogenic) Dizziness

Functional dizziness presents with chronic symptoms and is rarely the acute presentation. It is a diagnosis of inclusion based on positive features (inconsistency, distractibility, symptoms triggered by complex visual environments), not exclusion of other causes.

Table 3 — Mimics: How to Distinguish from Posterior Circulation Stroke

Condition	Discriminating features
Vestibular neuritis	Unidirectional horizontal nystagmus; positive head impulse; no hearing loss; gradual onset over hours
Labyrinthitis	Like neuritis plus hearing loss; must exclude AICA stroke (HINTS-plus + hearing + imaging)
Vestibular migraine	Known migraine; episodic; spontaneous resolution; typical migrainous features
Ménière's	Episodic 20 min–several hours; aural fullness + fluctuating low-frequency hearing loss
BPPV	Episodes of seconds; clear positional trigger; Dix-Hallpike positive; fatigable nystagmus
Vasovagal / orthostatic	Lightheadedness rather than vertigo; pallor, diaphoresis, orthostatic BP drop
PPPD	Chronic (>3 months); visually induced; no acute onset

The discriminating features listed assume bedside examination at the time of symptoms. All of these conditions can present atypically — the examination result, not the history alone, should drive the decision to image.

> □ **Clinical Pearl:**
> In a patient with acute vertigo, the question "what else could this be?" is always relevant, but the question "is this central?" takes precedence. Answering the second question correctly protects the patient irrespective of which peripheral mimic is ultimately responsible.

IX. Treatment and Disposition Principles

A patient identified as having posterior circulation stroke should receive stroke-pathway care: immediate transfer to a stroke service, early anti-thrombotic therapy when appropriate, consideration of thrombolysis or thrombectomy within therapeutic windows, and admission to a stroke unit.

Time Windows for Intervention

- **IV thrombolysis (alteplase or tenecteplase): within 4.5 hours of symptom onset in eligible patients.**
- **Endovascular thrombectomy: up to 24 hours in selected posterior circulation large-vessel occlusion with favourable imaging.**
- **Basilar artery occlusion: evolving evidence supports thrombectomy up to 24 hours; mortality exceeds 80% untreated [18].**

Cerebellar Oedema and Neurosurgical Considerations

Large cerebellar infarcts may swell over 24–96 hours, with risk of fourth-ventricular obstruction, hydrocephalus, and brainstem compression. Decompressive suboccipital craniectomy is life-saving in selected patients. Any confirmed cerebellar infarct requires admission to a stroke / neurosurgical service, not an outpatient pathway [5].

Management of Confirmed Vestibular Neuritis

Once central cause is excluded, vestibular neuritis is managed with brief symptomatic control (short course of vestibular suppressant for ≤72 hours, anti-emetics) followed by early mobilisation and vestibular rehabilitation. Prolonged vestibular suppressant use prevents central compensation and worsens long-term outcomes [19]. Corticosteroids within 3 days of onset may accelerate peripheral recovery, though evidence for long-term benefit is modest [20].

> □ **Important:**
> Never prolong vestibular suppressants (prochlorperazine, promethazine, betahistine sedation) beyond 72 hours in vestibular neuritis. Early mobilisation and vestibular rehabilitation exercises are the evidence-based treatment [19].

> □ **Clinical Insight:**
> Disposition is as important as diagnosis. A patient appropriately diagnosed with vestibular neuritis needs a 24–48 hour safety-net review, early rehabilitation referral, and clear advice on red-flag symptoms. A diagnosis without a follow-up plan is an incomplete encounter.

X. Safety-Netting, Documentation, and Key Messages

Because posterior circulation stroke can evolve over hours, and because a subset of small brainstem strokes are missed on initial imaging, every patient discharged with a peripheral-cause diagnosis in acute vertigo should receive structured safety-net advice.

Safety-Net Advice to the Patient

- **Return immediately (by ambulance) if new headache, neck pain, double vision, slurred speech, weakness or numbness, trouble swallowing, or inability to walk develops.**
- **Return if symptoms worsen rather than gradually improve over 48–72 hours.**
- **Return if hearing changes, ringing in one ear, or ear fullness develops.**
- **Planned review at 24–48 hours with the primary clinician or service.**

Documentation Essentials

- **Syndrome classification (AVS / t-EVS / s-EVS / chronic).**
- **Full HINTS-plus components and interpretation (if AVS).**
- **Gait, tandem gait, focal neurological examination result.**
- **Risk factors assessed (age, hypertension, diabetes, AF, smoking, prior stroke, recent trauma or neck manipulation).**
- **Red-flag history elements explicitly addressed (headache, neck pain, audiological, neurological).**

- **Diagnostic decision and reasoning, including reason imaging was or was not obtained.**
- **Disposition plan and safety-net advice given.**

> □ **Key Point:**
> The medico-legally defensible note in acute vertigo documents both what was found and what was considered and excluded. "HINTS examined: head impulse abnormal on right, unidirectional nystagmus, no skew, normal finger rub both sides — peripheral pattern" is far stronger than "vertigo, likely peripheral".

Six Take-Home Messages

- **Posterior circulation stroke is a leading cause of diagnostic error in acute vertigo.**
- **Up to 20% of posterior circulation strokes present with isolated vertigo.**
- **HINTS-plus, in trained hands, outperforms early MRI-DWI for stroke detection in AVS.**
- **ABCD2 is insufficient in the acute vertigo cohort.**
- **Any central HINTS finding, truncal ataxia, new hearing loss, or focal neurological sign mandates immediate ED referral.**
- **A normal CT does not exclude posterior circulation stroke; MRI-DWI with MRA is the imaging standard.**

> □ **Clinical Pearl:**
> If the examination is confidently peripheral and the history has no red flags, treat and reassure. If anything is equivocal — equivocal HINTS, equivocal neurological examination, equivocal history — the correct next step is escalation, not reassurance.

References

[1] Tarnutzer AA, Berkowitz AL, Robinson KA, Hsieh YH, Newman-Toker DE. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. CMAJ. 2011;183(9):E571–E592.

[2] Lee H, Sohn SI, Cho YW, et al. Cerebellar infarction presenting isolated vertigo: frequency and vascular topographical patterns. Neurology. 2006;67(7):1178–1183.

[3] Newman-Toker DE, Moy E, Valente E, Coffey R, Hines AL. Missed diagnosis of stroke in the emergency department: a cross-sectional analysis of a large population-based sample. Diagnosis. 2014;1(2):155–166.

[4] Atzema CL, Grewal K, Lu H, Kapral MK, Kulkarni G, Austin PC. Outcomes among patients discharged from the emergency department with a diagnosis of peripheral vertigo. Ann Neurol. 2016;79(1):32–41.

[5] Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. J Neurol. 1999;246(4):257–264.

[6] Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain. 2003;126(Pt 8):1864–1872.

[7] Lee H, Kim HA. Anterior inferior cerebellar artery infarction presenting as acute vestibular syndrome. Curr Opin Neurol. 2015;28(1):82–85.

[8] Debette S, Leys D. Cervical-artery dissections: predisposing factors, diagnosis, and outcome. Lancet Neurol. 2009;8(7):668–678.

[9] Hotson JR, Baloh RW. Acute vestibular syndrome. N Engl J Med. 1998;339(10):680–685.

[10] Johnston SC, Rothwell PM, Nguyen-Huynh MN, et al. Validation and refinement of scores to predict very early stroke risk after transient ischaemic attack. Lancet. 2007;369(9558):283–292.

[11] Newman-Toker DE, Kerber KA, Hsieh YH, et al. HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness. Acad Emerg Med. 2013;20(10):986–996.

[12] Carmona S, Martinez C, Zalazar G, et al. The diagnostic accuracy of truncal ataxia and HINTS as cardinal signs for acute vestibular syndrome. Front Neurol. 2016;7:125.

[13] Saber Tehrani AS, Kattah JC, Mantokoudis G, et al. Small strokes causing severe vertigo: frequency of false-negative MRIs and nonlacunar mechanisms. Neurology. 2014;83(2):169–173.

[14] Edlow JA, Carpenter C, Akhter M, et al. Guidelines for Reasonable and Appropriate Care in the Emergency Department (GRACE-3): Acute Dizziness and Vertigo. Acad Emerg Med. 2023;30(5):442–486.

[15] Kattah JC, Talkad AV, Wang DZ, Hsieh YH, Newman-Toker DE. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. Stroke. 2009;40(11):3504–3510.

[16] Ohle R, Montpellier RA, Marchadier V, et al. Can emergency physicians accurately rule out a central cause of vertigo using the HINTS examination? A systematic review and meta-analysis. Acad Emerg Med. 2020;27(9):887–896.

[17] Lempert T, Olesen J, Furman J, et al. Vestibular migraine: diagnostic criteria. J Vestib Res. 2012;22(4):167–172.

[18] Tao C, Nogueira RG, Zhu Y, et al. Trial of endovascular treatment of acute basilar-artery occlusion. N Engl J Med. 2022;387(15):1361–1372.

[19] Strupp M, Magnusson M. Acute unilateral vestibulopathy. Neurol Clin. 2015;33(3):669–685.

[20] Fishman JM, Burgess C, Waddell A. Corticosteroids for the treatment of idiopathic acute vestibular dysfunction (vestibular neuritis). Cochrane Database Syst Rev. 2011;(5):CD008607.

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