



# Differential diagnosis of acute vascular vertigo

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## Purpose of review

The current review covers recent advances in vascular vertigo in terms of diagnostic strategies, clinical/laboratory features, pathophysiology, and differential diagnosis.

## Recent findings

Acute strokes presenting with isolated dizziness/vertigo without other obvious symptoms or signs of central nervous system involvements may be easily mistaken as peripheral vestibulopathy. For correct diagnosis of vascular vertigo, the importance of clinical history (timing and triggers) and targeted bedside examination cannot be overemphasized. In addition to Head Impulse-Nystagmus-Test of Skew, several differential strategies have been advanced by adopting a combination of clinical history, bedside or laboratory examination, and imaging for diagnosis of vascular vertigo. Circumscribed cerebellar and brainstem lesions may cause isolated central vestibular syndromes with characteristic vestibular and ocular motor manifestations. Recognition of these findings would aid in localizing the lesions and understanding the function of each central vestibular structure. Central positional nystagmus (CPN) may mimic benign paroxysmal positional vertigo (BPPV), but additional oculomotor or neurological findings mostly permit differentiation of CPN from BPPV.

## Summary

In acute vestibular syndrome, discriminating vascular causes is still challenging especially when other central symptoms and signs are not evident. An integrated approach based on understanding of clinical features, laboratory findings, speculated mechanisms, and limitations of current diagnostic tests will lead to better clinical practice.

## Keywords

dizziness, nystagmus, stroke, vertigo, vestibulo-ocular reflex

## INTRODUCTION

Vascular compromise is one of the leading causes of central vertigo and is mostly due to strokes [1]. Vascular vertigo especially requires early diagnosis and proper treatments given its potentially grave prognosis. However, vascular vertigo can be easily mistaken as more benign disorders involving the inner ear when other symptoms and signs of central vestibular involvements are not obvious [2].

Vascular vertigo should be a prime suspicion in patients with acute vestibular syndrome and vascular risk factors even though confirmation of a stroke is mostly based on the findings of neurotological examination such as HINTS (Head Impulse-Nystagmus-Test of Skew) and brain imaging [2,3]. Various diagnostic strategies have been introduced, especially in the emergency setting, which will be summarized in this review.

In addition to vascular vertigo, various disorders involving the central vestibular structures may present with acute vertigo and imbalance [4]. Thus, proper diagnosis and treatments of vascular vertigo begin with correct understanding of the symptoms

and signs of central vestibular dysfunction. We will also review and summarize recent findings in clinical and laboratory aspects of vascular vertigo.

## MISDIAGNOSIS OF VASCULAR VERTIGO

A population-based cohort study found that patients discharged from the emergency department (ED) with a diagnosis of benign dizziness have

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## KEY POINTS

- History taking on timing and triggers of dizziness/vertigo and targeted bedside examination are crucial for the correct diagnosis of vascular vertigo.
- A focused bedside examination, represented by Head Impulse-Nystagmus-Test of Skew, is more useful than brain imaging in distinguishing acute vestibular syndrome due to strokes from vestibular neuritis.
- Recognition of vestibular and ocular motor syndromes from circumscribed brainstem and cerebellar lesions allows better understanding of the function of each central vestibular structure, and accurate localization of the lesions in patients with dizziness/vertigo.
- For differentiation of CPN from BPPV, characteristics of nystagmus, additional oculomotor or neurological findings, and response to the canalith repositioning maneuvers are important.

a 50-fold increased risk of hospitalization due to stroke within 7 days of discharge compared with the propensity score-matched controls [5]. A retrospective population-based study also showed that dizzy patients having visited the ED had a seven-fold higher risk of strokes than the general population, especially when they have stroke risk factors such as atrial fibrillation and diabetes mellitus [6]. Thus, dizziness and vertigo are the symptoms most tightly linked to missed stroke [7<sup>■</sup>,8].

Misdiagnosis of stroke as a cause of dizziness/vertigo is not uncommon in the ED even with modern neuroimaging [7<sup>■</sup>]. A systematic review including 15 721 patients on the accuracy of ED diagnosis for ischemic stroke, transient ischemic attack (TIA), or subarachnoid hemorrhage showed

that roughly 9% of cerebrovascular events are missed at the initial ED presentation. The risk for misdiagnosis was much greater when the presenting neurologic symptoms are mild, nonspecific, or transient (range 24–60%). Thus, posterior circulation strokes are especially at higher risk of misdiagnosis since those often present with dizziness/vertigo [9].

## DIAGNOSTIC STRATEGY FOR ACUTE VASCULAR VERTIGO

### Timing, triggers, and targeted examination

For differential diagnosis of dizziness/vertigo, the importance of clinical history and bedside examination cannot be overemphasized. Dizziness/vertigo may be classified into three types, acute, episodic, and chronic according to the presentation patterns [10,11<sup>■</sup>,12]. A new approach divides dizzy patients into three key categories, guiding a differential diagnosis and targeted bedside examination protocol (TiTrATE): acute vestibular syndrome where bedside physical examination differentiates vestibular neuritis from stroke; spontaneous episodic vestibular syndrome where associated symptoms help differentiate vestibular migraine from TIAs; and triggered episodic vestibular syndrome where the Dix-Hallpike and supine roll tests help differentiate benign paroxysmal positional vertigo (BPPV) from central positional nystagmus (CPN) due to posterior fossa lesions. All these three vestibular syndromes may be caused by vascular diseases such as TIAs, ischemic strokes, and hemorrhage (Table 1) [11<sup>■</sup>].

Vascular vertigo commonly represents an acute vestibular syndrome even though various disorders involving the brain may present with acute vertigo

**Table 1.** Vascular causes linked to four acute dizziness/vertigo syndromes

Syndrome	TIA	Ischemic stroke	Hemorrhage
t-EVS (brief, repetitive)	Rotational vertebral artery syndrome	CPPV from small ischemic strokes near the fourth ventricle	CPPV from small hemorrhages near the fourth ventricle
s-EVS (<24 h) <sup>a</sup>	PICA-isolated vertigo; AICA-vertigo ± tinnitus or hearing loss <sup>a</sup>	Small ischemic strokes presenting transient symptoms	Subarachnoid hemorrhages mimicking TIA
t-AVS (>24 h)	Overlap syndrome with trauma and vertebral artery dissection/TIA	Overlap syndrome with trauma and vertebral artery dissection/stroke	Overlap syndrome with trauma and traumatic hemorrhage (subdural, subarachnoid, etc.)
s-AVS (>24 h) <sup>a</sup>	Not yet reported (would be difficult to differentiate from vestibular migraine)	PICA-isolated vertigo; AICA-vertigo ± tinnitus or hearing loss <sup>a</sup>	Small to medium-sized cerebellar hemorrhages

AICA, anterior inferior cerebellar artery; CPPV, central paroxysmal positional vertigo; PICA, posterior inferior cerebellar artery; s-AVS, spontaneous acute vestibular syndrome; s-EVS, spontaneous episodic vestibular syndrome; t-AVS, traumatic/toxic acute vestibular syndrome; t-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack. Adapted from [11<sup>■</sup>].

<sup>a</sup>Common cerebrovascular presentations; the other combinations are rare.

**Table 2.** Diagnostic algorithms proposed for acute vascular dizziness or vertigo

Indicator	Contents of each indicator	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Areas under ROC curve
Based on clinical and neurological examination						
HINTS [3]	Head Impulse-Nystagmus-Test of Skew	100	96	99	100	0.995
STANDING [13]	SponTaneous and positional nystagmus, evaluation of the Nystagmus Direction, the head Impulse test, evaluation of equilibrium	95	87	48	99	NC
PCI score (ABCD <sup>2</sup> score) [18]	PCI score comprises high BP, diabetes, ischemic stroke, rotating and rocking, difficulty in speech, tinnitus, limb and sensory deficit, gait ataxia, limb ataxia; ABCD <sup>2</sup> is composed of age, BP, clinical features, duration of symptoms, diabetes	NC	NC	NC	NC	0.82
TriAGe+ score [19]	No triggers (2), AF (2), male sex (1), BP $\geq$ 140/90 (2), brainstem or cerebellar dysfunction (1), focal weakness or speech impairment (4), dizziness (3), no history of dizziness/vertigo or labyrinth/vestibular disease (2)	NC	NC	NC	NC	0.82
DEFENSIVE stroke scale [20 <sup>*</sup> ]	DisEquilibrium, Floating sEnsation, Non-Specific dizziness, Imbalance, Vertigo	94.8	83	58.6	98.4	NC
Based on neurological and laboratory test						
NLR > 2.8 and the absence of HN [21]	Neutrophil-to-lymphocyte ratio	NC	NC	NC	NC	0.84
Based on sonographic finding						
Abnormal VAECCS [29]	Vertebral Artery Extracranial Color-Coded duplex Sonography	54	95	75	88	NC
Abnormal ultrasound [30]	Presence of stenotic or occlusive disease in intracranial or extracranial segments of the VB circulation	41	100	100	84	NC

AF, atrial fibrillation; BP, blood pressure; HN, horizontal nystagmus; NC, not calculated; NLR, neutrophil-to-lymphocyte ratio; NPV, negative predictive value; PCI, posterior circulation ischemia; PPV, positive predictive value; ROC, receiver operating characteristic; VB, vertebrasilar. Modified from [34].

and imbalance [4]. A focused bedside examination, represented by HINTS (Table 1), has higher sensitivity and specificity than diffusion-weighted imaging (DWI) in patients with acute spontaneous prolonged (>24 h) vertigo with at least one stroke risk factor when there is no hearing loss [3].

### Other diagnostic approach to vascular vertigo in acute or emergency setting

In addition to HINTS, several algorithms have been advanced for diagnosis of vascular vertigo. Evaluation of balance and gait could be a key to rule out cerebellar strokes in patients with acute vertigo

(Table 2) [13]. From a review of 16 relevant studies, normal gait examination cannot exclude a cerebellar stroke, but the presence of abnormal gait can be a finding of cerebellar stroke [14<sup>\*</sup>].

ABCD<sup>2</sup> score, originally adopted to determine the risk of stroke after a TIA [15], may be applied in diagnosing strokes [16]. However, HINTS outperforms ABCD<sup>2</sup> for stroke diagnosis in ED patients with acute vestibular syndrome [17]. The posterior circulation ischemia score system has nine variables including the risk factors for strokes [high blood pressure (BP), diabetes mellitus, ischemic stroke, difficulty in speech, limb and sensory deficit, gait and limb ataxias] and the factors for peripheral

dizziness/vertigo (rotating and rocking sense, and tinnitus) [18]. A modified system was reported to outperform the ABCD<sup>2</sup> score for identifying strokes in ED patients with dizziness/vertigo [19] when scores from eight variables [no triggers (2), atrial fibrillation (2), male sex (1), BP > 140/90 (2), brainstem or cerebellar dysfunction (1), focal weakness or speech impairment (4), dizziness (3), and no history of dizziness/vertigo or labyrinthine/ vestibular diseases (2)] with a cutoff value at 10 points. Another stroke scale based on the presence of disequilibrium, floating sensation, nonspecific dizziness, imbalance, and vertigo [20<sup>a</sup>] showed a sensitivity of 100% and a decreased rate of improper diagnosis of stroke in an ED setting [20<sup>a</sup>].

Some laboratory findings have been reported helpful for diagnosis of strokes among patients with dizziness. Neutrophil-to-lymphocyte ratio has been used as a marker for various conditions. A combination of neutrophil-to-lymphocyte ratio more than 2.8 and absence of horizontal nystagmus was specific for acute infarction in ED patients with acute vertigo [21]. Increased neuron specific enolase was also independently associated with cerebral infarction in patients with dizziness/vertigo [22].

Stroke risk factors are always important. Isolated vertigo patients are at a higher risk of central vertigo when they have more than three risk factors (man, age >60, hypertension, diabetes mellitus, smoking, and stroke history) [23]. A recent study for identifying acute ischemic strokes in 77 993 ED patients with dizziness, vertigo, and imbalance found a positive association with admission presentation of imbalance, African-American race, history of hypertension, diabetes mellitus, hypercholesterolemia, tobacco use, atrial fibrillation, and prior acute ischemic stroke due to extracranial artery atherosclerosis [24<sup>a</sup>]. Factors negatively associated with an acute ischemic stroke included admission presentation of vertigo, female sex, age more than 81, history of anemia, coronary artery disease, asthma, depressive disorders, and anxiety disorders.

### Imaging studies

Given their low sensitivity, computed tomography scans have a limited role in identifying acute ischemic strokes particularly in the posterior circulation territory [11<sup>a</sup>,25]. Even MRI with DWI misses about 15–20% of acute posterior circulation infarctions within 24–48 h of symptom onset [10,26]. Another study found an even higher chance (about 50%) for misdiagnosis when the strokes are small (less than 10 mm in diameter) [26,27].

Perfusion-weighted imaging (PWI) may contribute to identification of ischemic strokes especially in

those with initially negative DWI. In a prospective study comprising a large number of patients with acute ischemic strokes, PWI revealed a decreased perfusion in 12 of the 26 patients with posterior circulation strokes and negative DWI. The sensitivity of systematic clinical evaluation adopting neurological examination, HINTS plus, and assessment of equilibrium was 83% for prediction of a stroke and 100% when combined with PWI. An integrated approach using systematic neurological and neurootological examination (neurologic examination + HINTS plus + equilibrium) combined with PWI accurately diagnoses posterior circulation strokes presenting with acute dizziness/vertigo [28<sup>a</sup>].

Neurosonology may offer a noninvasive method to study the cervicocephalic circulation when a vascular cause is suspected. In a study having adopted vertebral artery duplex sonography, 15 (75%) of 20 patients with abnormal findings and 13 (12%) of 106 with normal results had a final diagnosis of vertebrobasilar stroke. Thus, the sensitivity and specificity were 53.6 and 94.9% (odds ratio = 21.5, Table 2) [29]. In another study, neurosonological assessment showed an excellent specificity (100%), positive predictive value (100%), and negative predictive value (83.5%) even though the sensitivity was relatively low (40.7%, Table 2) [30].

### VESTIBULAR AND OCULAR MOTOR FINDINGS IN VASCULAR VERTIGO DUE TO FOCAL BRAINSTEM OR CEREBELLAR LESIONS

Circumscribed cerebellar and brainstem lesions provide an opportunity for defining the vestibular and ocular motor syndrome from damage to central vestibular structures [31]. Recognition of isolated central vestibular syndromes from brainstem or cerebellar lesions would aid in localizing the lesion responsible for various vestibular and ocular motor manifestations (Table 3).

Acute unilateral flocculus infarction resulted in spontaneous nystagmus beating to the lesion side, impaired ipsilesional smooth pursuit, contraversive ocular tilt reaction (OTR), and tilt of subjective visual vertical (SVV), and increased vestibulo-ocular reflex (VOR) gains during lower frequency and decreased gains during higher frequency stimulations [32]. Another patient with an isolated unilateral flocculus infarction showed similar findings, but initially ipsilesional caloric paresis, and reduced head impulse VOR gain only for the contralesional horizontal canal [33]. The caloric responses became normalized within a few weeks, but the abnormal head impulse test (HIT) persisted for months [33].



**Table 3.** Neuro-ophthalmologic findings in isolated central vestibular lesions

	Vestibular nucleus	NPH	Flocculus	Tonsil	Nodulus	ICP
SN	Contralesional	Ipsilesional	Ipsilesional	Ipsilesional	PAN or ipsilesional	Ipsilesional
GEN	Bilateral (contra>)	Bilateral (ipsi>)	Bilateral (ipsi>)	Bilateral (ipsi>)	None	None
OTR	Ipsiversive	Ipsiversive or contraversive	Contraversive	None	Contraversive	Contraversive
SVV tilt	Ipsiversive	Ipsiversive or contraversive	Contraversive	Contraversive	Contraversive	Contraversive
Pursuit	Impaired, ipsilesional	Impaired, ipsilesional	Impaired, ipsilesional	Markedly impaired, ipsilesional	Normal	Impaired, ipsilesional
<b>VOR</b>						
Caloric	Ipsilesional paresis	Normal	Normal	Normal	Normal	Normal
Bedside HIT	Impaired, ipsilesional	Impaired, contralesional	Impaired, ipsilesional	Normal	Normal	Normal
HIT-MSc	Impaired, bilateral	Impaired, contralesional	Impaired, bilateral	Normal	Normal	Normal
Saccades	Normal	Normal	Normal	Normal	Normal	Normal

Contralesional > greater amplitude during contralesional gaze; ipsilesional > greater amplitude during ipsilesional gaze. GEN, gaze-evoked nystagmus; HIT, head impulse test; ICP, inferior cerebellar peduncle; MSC, magnetic search coil; NPH, nucleus prepositus hypoglossi; OTR, ocular tilt reaction; PAN, periodic alternating nystagmus; SN, spontaneous nystagmus; SVV, subjective visual vertical; VOR, vestibulo-ocular reflex. Adapted from [31].

The midline vestibulocerebellum, especially the nodulus, provides inhibitory projections to the vestibular nuclei and modulates the velocity storage mechanism [34]. Tilt suppression of postrotatory nystagmus is impaired in about one-third of the patients with cerebellar infarctions mostly involving the nodulus and uvula [35<sup>■</sup>]. Transient downbeat nystagmus is usually found after horizontal head-shaking (perverted nystagmus) in association with cerebellar dysfunction, probably due to an enhanced central processing of the signals from the anterior semicircular canal. Along with positional downbeat nystagmus and impaired tilt suppression, this cross-coupled head-shaking nystagmus appears to indicate midline cerebellar dysfunction [35<sup>■</sup>]. Nodular lesions also generate CPN, especially apogeotropic horizontal and/or downbeat nystagmus [36].

The middle cerebellar peduncle (MCP) is a major conduit for the cortico-ponto-cerebellar fibers that convey information related to eye movements. Patients with acute strokes restricted to unilateral MCP usually present acute vertigo or imbalance, and frequently show abnormal eye movements that include spontaneous horizontal/torsional nystagmus, gaze-evoked nystagmus, OTR, abnormal HIT, and bilaterally impaired horizontal smooth pursuit. These ocular motor abnormalities are primarily caused by damage to the central vestibular structures and by disruption of the neural pathways responsible for eye-position stabilization and smooth pursuit [37].

Strokes involving the medial longitudinal fasciculus (MLF) may cause diverse ocular motor and vestibular abnormalities in addition to internuclear ophthalmoplegia (INO) [38<sup>■</sup>,39,40]. These include upbeat or jerky seesaw nystagmus, contraversive OTR

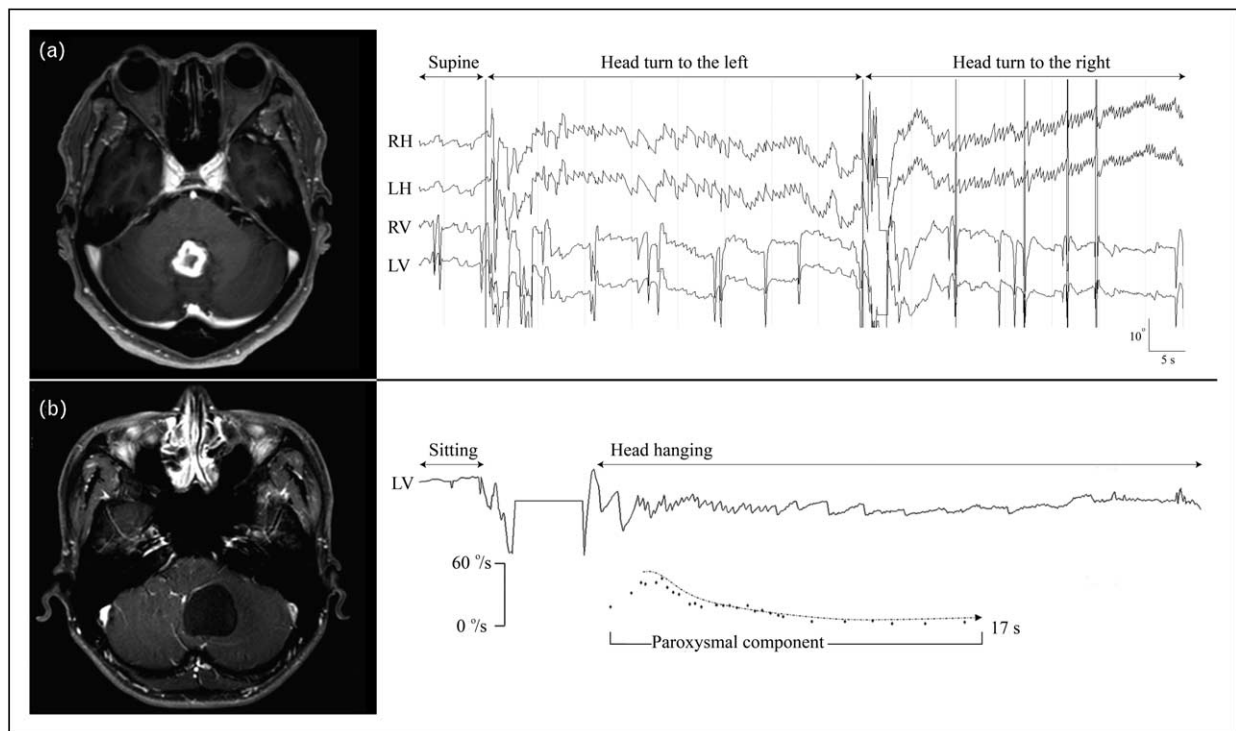
and SVV tilt, impaired vertical VOR and smooth pursuit, and abnormal ocular vestibular evoked myogenic potentials (VEMPs) and cervical VEMPs [38<sup>■</sup>,39,40]. A recent study also confirmed a prominent decrease in the VOR gain for the contralesional posterior canal during HITs in patients with unilateral INO due to strokes [38<sup>■</sup>]. This suggests that the MLF serves as a main passage for the high acceleration VOR from the contralateral posterior canal.

Lateral medullary infarction (LMI) may present unidirectional horizontal nystagmus and abnormal horizontal HIT, which mimics peripheral vestibulopathy. Transient peripheral vestibular symptoms and signs were ascribed to transient ischemia of the vestibular afferents from the horizontal semicircular canal [41]. LMI rarely presents presyncope due to orthostatic hypotension in the absence of vertigo or Horner's syndrome [42].

Labyrinthine infarction is a cause of acute audio-vestibulopathy, but can be diagnosed only in association with concurrent cerebral infarctions since current imaging techniques mostly fail to visualize an infarction confined to the labyrinth. A recent study described 10 patients with embolic labyrinthine infarction and introduced a diagnostic algorithm [43<sup>■</sup>]. All patients developed acute audio-vestibulopathy in association with an obvious source of embolism and concurrent acute embolic infarctions in the territories other than those supplied by the anterior inferior cerebellar artery.

### CENTRAL POSITIONAL NYSTAGMUS

Strokes involving the brainstem or cerebellum may give rise to positional nystagmus (Fig. 1). Even though



**FIGURE 1.** (a) A patient with a tuberculoma involving the cerebellar nodulus shows persistent apogeotropic nystagmus. (b) Another patient with a midline cerebellar tumor develops paroxysmal downbeat nystagmus on straight head hanging. LH, horizontal position of the left eye; LV, vertical position of the left eye; RH, horizontal position of the right eye; RV, vertical position of the right eye.

isolated positional vertigo is rare in strokes, CPN due to strokes should be differentiated from BPPV.

A systematic review of 28 studies characterized various patterns of CPN, but the most salient feature distinctive from BPPV is atypical direction of nystagmus for the canal stimulated in 97.5% of patients during Dix–Hallpike and 54.5% upon supine roll testing. CPN was paroxysmal (<60 s) in 85% of patients on straight head hanging, 63.9% on Dix–Hallpike maneuver, and 37.5% on supine roll test, and had a latency less than 3 s upon positioning in 94.7%. Concurrent vertigo was present in 63.4% of patients and other neurological signs were present in 48.8%. Imaging documented cerebellar involvements in 74.4%, isolated brainstem lesions in 8.5%, and fourth ventricle involvements in 14.6% [44]. Persistent geotropic nystagmus has also been reported in unilateral cerebellar infarctions, especially in the territory of medial posterior inferior cerebellar artery [45].

In apogeotropic CPN, the lesions were also mostly overlapped in the vestibulocerebellum (nodulus, uvula, and tonsil). It was postulated that the estimate of gravity direction is erroneously biased away from the true vertical when the tilt-estimator circuit in the brain malfunctions due to lesions involving the vestibulocerebellum. According to direction of the bias in each position, various

patterns of positional nystagmus may be generated [46,47].

Compared with those of apogeotropic BPPV, the slow phase velocity was lower, and the amplitude was smaller in apogeotropic CPN due to cerebellar lesions [48]. Furthermore, the intensity of apogeotropic CPN peaks initially and then decreases with time [46]. Along with these characteristics of CPN, additional oculomotor or neurological findings are the most important features for differentiation of CPN from BPPV.

## CONCLUSION

Vascular vertigo may have diverse clinical manifestations including acute vestibular syndrome mimicking vestibular neuritis, spontaneous episodic vestibular syndrome mimicking Meniere's disease or vestibular migraine, and central positional vertigo mimicking BPPV. Thus, discriminating vascular causes in patients with dizziness/vertigo is still challenging especially when other symptoms and signs of central nervous involvements are not evident. The importance of history taking on timing and triggers of dizziness/vertigo and targeted bedside examination such as HINTS cannot be overemphasized. Recognition of responsible central vestibular and ocular motor manifestations in isolated central

vestibular syndromes from brainstem or cerebellar lesions would be also important. Overall, an integrated approach based on understanding of clinical features, laboratory findings, speculated mechanisms, and limitations of current diagnostic tests would lead to better clinical practice.

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### Conflicts of interest

There are no conflicts of interest.

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