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Vascular vertigo and dizziness: Diagnostic criteria

Consensus document of the committee for the classification of vestibular disorders of the Bárány society

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Abstract. This paper presents diagnostic criteria for vascular vertigo and dizziness as formulated by the Committee for the Classification of Vestibular Disorders of the Bárány Society. The classification includes vertigo/dizziness due to stroke or transient ischemic attack as well as isolated labyrinthine infarction/hemorrhage, and vertebral artery compression syndrome. Vertigo and dizziness are among the most common symptoms of posterior circulation strokes. Vascular vertigo/dizziness may be acute and prolonged ($\geq$24 hours) or transient (minutes to $<$24 hours). Vascular vertigo/dizziness should be considered in patients who present with acute vestibular symptoms and additional central neurological symptoms and signs, including central HINTS signs (normal head-impulse test, direction-changing gaze-evoked nystagmus, or pronounced skew deviation), particularly in the presence of vascular risk factors. Isolated labyrinthine infarction does not have a confirmatory test, but should be considered in individuals at increased risk of stroke and can be presumed in cases of acute unilateral vestibular loss if accompanied or followed within 30 days by an ischemic stroke in the anterior inferior cerebellar artery territory. For diagnosis of vertebral artery compression syndrome, typical symptoms and signs in combination with imaging or sonographic documentation of vascular compromise are required.

Keywords: Vertigo, dizziness, imbalance, infarction, stroke, brainstem, cerebellum

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1. Introduction

The Bárány Society, representing the international community of basic scientists, otolaryngologists, and neurologists committed to vestibular research, mandated a Committee for an International Classification of Vestibular Disorders (ICVD) [12].

Vertigo/dizziness is one of the most common symptoms of posterior circulation stroke [29, 53, 106]. Its onset is typically acute and may be prolonged ($\geq$ 24 hours, acute prolonged vertigo/dizziness) or transient (<24 hours, transient vertigo/dizziness) [109, 131]. Transient vertigo/dizziness may recur in episodes (recurrent spontaneous vertigo/dizziness). An isolated positional vestibular syndrome (or recurrent positional vertigo/dizziness) due to vascular vertigo/dizziness is rare. Vertigo/dizziness in cerebrovascular disorders is usually accompanied by other neurological symptoms and signs [49, 74, 95, 157]. Recent advances in clinical neuro-otology/neuro-ophthalmology and neuroimaging have led to a consensus that strokes involving the brainstem or cerebellum can also present with isolated vertigo/dizziness or imbalance [106]. Finally, transient vertigo/dizziness is also one of the most common manifestations of vertebrobasilar ischemia and is occasionally isolated [22, 54, 55, 83].

It is important to differentiate isolated vertigo/dizziness of a vascular cause from non-vascular disorders, e.g., acute unilateral vestibulopathy (AUVP)/vestibular neuritis, involving the labyrinth or vestibular nerve since therapeutic strategies and prognosis differ in these two conditions [109]. Misdiagnosis of acute stroke may result in loss of effective treatment opportunities, which may increase morbidity and mortality, while over-diagnosis of vascular vertigo/dizziness would lead to unnecessary costly work-ups and medication [30, 152–154]. Depending on the underlying etiology, more aggressive treatments including thrombolysis or endovascular intervention as well as dual antiplatelet therapy or anticoagulation may be indicated to treat a stroke and prevent recurrences of stroke in vascular vertigo/dizziness [39, 40, 122]. Finally, from a scientific point of view, detailed evaluation of patients with infarctions restricted to specific vestibular structures also allows a better understanding of the function of each vestibular structure and definition of various ischemic vestibular syndromes [92, 93].

To develop the diagnostic criteria for vascular vertigo/dizziness, creation of the subcommittee was initiated by the members of the ICVD Committee of the Bárány Society in Uppsala, 2012. They selected a chairperson (JSK) to choose subcommittee members representing different subspecialities from three different continents. Diagnostic criteria were developed through discussions among the subcommittee members. Draft criteria were presented to the ICVD committee of the Barany Society in November 2020 and then modified based on comments. A revised draft became available for comments by the Bárány Society membership in July 2021.

2. Diagnosis of vascular vertigo/dizziness

Patients with vascular vertigo/dizziness typically present with *acute vestibular syndrome (AVS), which* refers to the acute onset of vertigo or dizziness with nausea or vomiting, head-motion intolerance, and unsteadiness [11, 154]. Depending on the presentation, vascular vertigo/dizziness can be divided into acute prolonged vascular vertigo/dizziness and transient vascular vertigo/dizziness. *Acute prolonged vascular vertigo/dizziness* refers to symptoms lasting 24 hours or more. When a patient presents with a previous episode of vertigo/dizziness of less than 24 hours, the term *transient vascular vertigo/dizziness* may be used [155]. In addition, the term *acute vascular vertigo/dizziness in evolution* may be applied when a patient with acute vertigo/dizziness is being evaluated within 24 hours from symptom onset.

2.1. Diagnostic criteria for acute prolonged vascular vertigo/dizziness

2.1.1. Acute prolonged vascular vertigo/dizziness

Criteria A-C should be fulfilled to make the diagnosis of acute prolonged vascular vertigo/dizziness.

A) Acute vertigo, dizziness, or unsteadiness lasting for 24 hours or more[1)]
B) Imaging evidence of ischemia or hemorrhage in the brain or inner ear, which corresponds to the symptoms, signs and findings[2)]
C) Not better accounted for by another disease or disorder

2.1.2. Probable acute prolonged vascular vertigo/dizziness

Criteria A-C should be fulfilled to make the diagnosis of probable acute prolonged vascular vertigo/dizziness.

A) Acute vertigo, dizziness or unsteadiness lasting for 24 hours or more[1)]
B) At least one of the following:
   1. Focal central neurological symptoms and signs, e.g., hemiparesis, sensory loss, dysarthria, dysphagia, or severe truncal ataxia/postural instability[3)]
   2. At least one component of central HINTS [normal head impulse test, direction-changing gaze-evoked nystagmus, or pronounced skew deviation][4)]
   3. Other central ocular motor abnormalities, e.g., central nystagmus, impaired saccades, or impaired smooth pursuit[5)]
   4. Increased risk for vascular events (e.g., $ABCD^2$ score of 4 or more, or atrial fibrillation)[6)]
C) Not better accounted for by another disease or disorder

2.1.3. Notes

1) An acute prolonged vestibular syndrome consists of continuous vertigo/dizziness, imbalance, oscillopsia, vegetative symptoms such as nausea and vomiting, or head motion intolerance lasting more than 24 hours [64, 166].
2) Even though a diagnosis of acute stroke is primarily based on the findings of neuroimaging, initial MRIs, including diffusion-weighted images (DWI), are falsely negative in 12–50% within the first 48 hours [21, 73, 75, 154] in patients who are eventually determined to have had a stroke. This high false negative rate has two implications: First, if the initial MRI is normal, serial radiological evaluation is required to identify an acute lesion in these patients. Second, it increases the importance of a systematic clinical examination, which has a higher sensitivity during the acute phase than imaging. Further, as the internal auditory artery (IAA), usually a branch of the anterior inferior cerebellar artery (AICA), supplies the inner ear [125], acute vertigo and hearing loss also can be due to labyrinthine or very rarely eighth cranial nerve infarction [36, 81, 90]. As isolated labyrinthine damage may precede ponto-cerebellar involvement in AICA infarction, recognizing audio-vestibular loss may provide an opportunity to prevent progression to a more widespread infarction involving the posterior circulation, mainly in the AICA territory [88, 102].

   A very rare entity is labyrinthine hemorrhage. It may occur spontaneously, but more frequently it occurs in association with head trauma or bleeding disorders [97, 175]. It shares with labyrinthine infarction the frequent association of vertigo and hearing loss but without brainstem involvement [175]. In labyrinthine hemorrhage, the vertigo is often severe and hearing loss is profound with a poor prognosis [175]. Labyrinthine hemorrhage may be identified by a hyperintense signal in the labyrinth on T1 or fluid-attenuated inversion recovery (FLAIR) MRIs, although this signal can also be caused by inflammatory disorders [175].
3) Severe truncal ataxia or postural instability is defined by a patient being unable to maintain an upright sitting or standing posture without support [16, 106, 174]. In a 2016 study, none of the patients with AUPV/vestibular neuritis showed severe truncal ataxia or postural instability [16] However, mild to moderate truncal ataxia or postural instability does not necessarily exclude a central lesion [16]. Thus, evaluation of upright balance function enhances the detection of central lesions in acute prolonged vertigo/dizziness [16, 174]. For patients who are too symptomatic to walk, postural stability can be assessed by asking the patient to sit upright in a stretcher with their arms crossed [16].
4) In a study of 101 patients (69 ischemic strokes, 4 hemorrhages, and 28 non-strokes), a refined bedside examination protocol that incorporates HINTS performed by a clinical expert showed an up to 100% [69/69 with ischemic strokes, 95% confidence interval (CI) = 95–100%] sensitivity and 96% (24/25 with acute peripheral vestibulopathy, 95% CI = 80–100%) specificity, giving a positive likelihood ratio of 25 (95% CI = 3.66–170.59) and a negative likelihood ratio of 0.00 (95% CI = 0.00–0.11), compared with delayed MRI in identifying ischemic strokes in patients with acute prolonged vertigo of more than 24 hours and one vascular risk factor, whereas initial DWIs were normal in 12% (8/69 ischemic strokes) [73]. Another report on 20 patients with acute pure vestibular syndrome (10 with strokes and 10 with vestibular neuritis) also found diagnostic utility of the signs including normal horizontal head impulse tests (HIT), skew deviation (SD), abnormal vertical smooth pursuit, and central type nystagmus at the bedside [19]. Since a

mild degree of SD may go unnoticed during bedside examination and gaze-evoked nystagmus may be absent in cerebellar strokes [106], bedside HIT is a good tool for differentiating isolated vertigo due to cerebellar strokes from AUVP/vestibular neuritis. Indeed, of the three bedside signs of 'HINTS', the horizontal HIT had the greatest combined sensitivity (0.85, 95% CI = 0.79–0.91) and specificity (0.95, 95% CI = 0.90–1.00) for central causes [166]. Since pathological HIT and SD can be seen in either peripheral or central lesions, these tests are complementary in diagnosing central vestibular disorders [37, 56, 73, 96, 98].

5) The HINTS may not be sufficiently robust to detect an AICA infarction since the HIT is mostly pathological in this disorder [67]. Patients with AICA territory infarction may develop isolated vertigo with negative HINTS (pathological HIT in the absence of gaze-evoked nystagmus and SD), mimicking AUPV/vestibular neuritis [105]. Since the AICA supplies the inner ear, the signs of an AUVP/vestibular neuritis may overshadow the central signs and HINTS may be negative in AICA territory infarctions [34]. Indeed, about 5% of patients with AICA territory ischemic strokes presented acute prolonged vertigo and canal paresis without hearing loss, mimicking acute peripheral vestibular syndrome [105]. Another study also showed negative HINTS in 5 of 17 patients (29.4%) with AICA infarction [67]. In those with negative HINTS, the addition of horizontal head shaking, hearing test with finger rub (HINTS plus), and examination of stance and gait may aid in detecting a central lesion [16, 67, 139, 154].
6) The $ABCD^2$ score (age, blood pressure, clinical features, duration, and presence of diabetes) was originally developed to estimate the future risk of stroke in patients with a transient ischemic attack (TIA) [69]. However, the $ABCD^2$ score may be used to predict stroke in patients presenting with acute vertigo/dizziness [177]. In a study on patients having visited emergency department with dizziness, vertigo or imbalance, either prolonged or transient, only 1.0% (5/502) with an $ABCD^2$ score of 3 or less had a stroke compared with 8.1% (25/369) in those with a score of 4 or more. Notably, 27% (7/26) of the patients with a score of 6 or 7 suffered from strokes [130]. A prospective study of emergency department dizziness presentations from a single center found that the $ABCD^2$ score as a continuous variable was an independent predictor of acute infarct on MRI [odd ratio (OR) = 1.74, 95% CI = 1.20–2.51] adjusting for findings on the general neurologic and oculomotor examination [75].

2.2. Diagnostic criteria for transient vascular vertigo/dizziness and acute vascular vertigo/dizziness in evolution

2.2.1. Transient vascular vertigo/dizziness or acute vascular vertigo/dizziness in evolution

Criteria A-C should be fulfilled to make the diagnosis of transient vascular vertigo/dizziness or acute vascular vertigo/dizziness in evolution.

A) Acute spontaneous vertigo, dizziness, or unsteadiness lasting less than 24 hours[1)]
B) Imaging evidence of ischemia or hemorrhage in the brain or inner ear, which corresponds to the symptoms, signs and findings[2)]
C) Not better accounted for by another disease or disorder

2.2.2. Probable acute vascular vertigo/dizziness in evolution

Criteria A-C should be fulfilled to make the diagnosis of probable acute vascular vertigo/dizziness in evolution.

A) Acute spontaneous vertigo, dizziness, or unsteadiness for more than 3 hours, but that has not yet lasted for at least 24 hours when seen.
B) At least one of the following:
    1. Focal central neurological symptoms and signs, or severe truncal ataxia/postural instability
    2. At least one component of central HINTS (normal head impulse tests, direction-changing gaze-evoked nystagmus, or pronounced skew deviation)[3)]
    3. Other central ocular motor abnormalities, e.g., central nystagmus, impaired saccades or impaired smooth pursuit)
    4. New onset of moderate to severe craniocervical pain[4)]
    5. Increased risk for vascular events, e.g., $ABCD^2$ score of 4 or more, or atrial fibrillation

6. Significant (>50%) narrowing of an artery of the vertebrobasilar system[5)]

C) Not better accounted for by another disease or disorder[6)]

2.2.3. Probable transient vascular vertigo/dizziness

Criteria A–C should be fulfilled to make the diagnosis of probable transient vascular vertigo/dizziness.

A) Acute spontaneous vertigo, dizziness, or unsteadiness lasting less than 24 hours[1)]
B) At least one of the following:
   1. Focal central neurological symptoms or severe postural instability during the attack
   2. New onset of moderate to severe craniocervical pain during the attack[4)]
   3. Increased risk for vascular events, e.g., $ABCD^2$ score of 4 or more, or atrial fibrillation
   4. Significant (>50%) narrowing of an artery of the vertebrobasilar system[5)]
C) Not better accounted for by another disease or disorder[6)]

2.2.4. Notes

1) Many patients develop acute vertigo/dizziness or imbalance lasting less than a day, which may be termed transient vestibular syndrome [22], even though the National Institute of Neurological Disorders and Stroke (NINDS) III Classification and the European Stroke Organization (ESO) Executive Committee and the ESO Writing Committee do not embrace isolated vertigo as a symptom of TIA involving the vertebrobasilar territory (VB-TIA) [51]. The use of the previous terminology "vertebrobasilar insufficiency" is not recommended [18]. Transient vestibular syndrome frequently occurs in VB-TIA [55, 148]. Indeed, isolated episodic vertigo was the only manifestation in 21% (6/29) of patients with a presumptive diagnosis of VB-TIA [54], and 62% (29/42) of the patients with vertigo due to VB-TIA and 29% (12/42) of patients with vertebrobasilar infarction had a history of isolated episodic vertigo [55]. Other studies reported that preceding transient isolated brainstem symptoms are common in patients with a completed stroke in the vertebrobasilar territory [148]. The episodic vertigo is typically spontaneous in onset and lasts for minutes in VB-TIA [55, 64]. Despite detailed neuro-otologic examination and neuroimaging studies including MRIs with DWI and perfusion imaging, underlying etiologies remained unknown in more than half of the patients with transient vestibular syndrome [22]. Transient vestibular syndrome may be attributed to rapid resolution of some peripheral vestibular disorders such as benign paroxysmal positional vertigo (BPPV) or Menière's disease during their first attack. Transient brainstem hypoperfusion may be another possibility since perfusion imaging has limitations in detecting a small perfusion defect restricted to the brainstem.
2) A study found stroke in 27% [23/86, cerebral infarction in 15% (13/86) and cerebellar hypoperfusion in 12% (10/86)] of patients referred to the emergency department with transient vestibular syndrome [22].
3) Whereas application of HINTS has greatly enhanced the diagnosis of stroke in acute prolonged vascular vertigo, the diagnostic utility of HINTS/HINTS plus examination and MRIs was limited in transient vestibular syndrome. HINTS plus could not be applied to the majority of patients with transient vestibular syndrome since the vestibular symptoms or signs had already resolved by the time of evaluation in about 73% (63/86) of the patients [22].
4) Even though headache is a common symptom, moderate to severe craniocervical pain is very rare in peripheral vestibular disorders. Thus, when patients experience the new onset of moderate to severe craniocervical pain along with acute vestibular symptoms, vascular dissection or posterior circulation strokes including hemorrhages should be suspected, especially when migraine or vestibular migraine is unlikely. Indeed, associated craniocervical pain was a clue for strokes with an OR of 15.2 (95% CI = 2.5–93.8, multivariate logistic regression analysis) in a study of 86 patients who were diagnosed with transient vestibular syndrome [22].
5) In a previous study on transient vestibular syndrome [22], eight of the 10 patients with unilateral cerebellar hypoperfusion only on perfusion images without an infarction on DWI showed a focal stenosis or hypoplasia of the corresponding vertebral artery (VA). The results of multivariate logistic regression analysis showed

that VA stenosis or hypoplasia (OR = 7.0, 95% CI = 1.7–29.4) is a risk factor for strokes in patients with transient vertigo/dizziness [22]. Besides atherosclerotic stenosis or occlusion, hypoplasia of the VA may be a predisposing factor for posterior circulation stroke especially when vascular risk factors coexist [2, 71]. Vascular investigations may reveal reversal of flow in one VA [149], or other kinds of steal phenomena [172]. Reversal of VA flow is often asymptomatic or may be associated with carotid territory symptoms as it is with vertebrobasilar territory symptoms [7]. For this reason, subclavian steal syndrome is not included in this classification as a distinct entity for vascular vertigo/dizziness [48, 150].

6) Even though a vascular origin is a serious concern in patients with new onset transient vertigo/dizziness and vascular risk factors, other diagnoses such as vestibular migraine or Menière's disease are more likely in patients with episodes of vertigo/dizziness that have been occurring for many months or years [116, 131].

2.3. Diagnostic criteria for vertebral artery compression syndrome (VACS)

2.3.1. Vertebral artery compression syndrome (VACS)

Criteria A-D should be fulfilled to make the diagnosis of VACS.

A) Vertigo with or without tinnitus provoked by a sustained eccentric neck position, especially in an upright body position[1)]
B) Presence of nystagmus with the symptoms during an attack[2)]
C) Either 1) or 2) during the provoking head motion[3)]
   1) Documentation of VA compression using dynamic angiography
   2) Demonstration of decreased blood flow in the posterior circulation using transcranial Doppler
D) Not better accounted for by another disease or disorder[4)]

2.3.2. Previously used terms

Bow hunter's syndrome, rotational VA syndrome, rotational VA compression syndrome, rotational VA occlusion syndrome.

2.3.3. Notes

1) Episodic vertigo, nystagmus, and syncope rarely may occur due to mechanical compression of the VA induced by horizontal or diagonal neck rotation, tilt or extension [99, 171, 187]. Tinnitus develops several seconds after the onset of vertigo and nystagmus, which suggests that the vestibule is more sensitive to ischemia than the cochlea [28, 164].
2) Oculographic analyses reveal various patterns of nystagmus during attacks in the VACS [32, 123, 164]: the initial nystagmus is mostly downbeat, with the horizontal and torsional components beating either toward the compressed VA side, indicating a transient excitation of the labyrinth [164], or directed away [32]. Patients may show spontaneous reversal of the nystagmus, or markedly diminished or absence of nystagmus when the provocative neck rotation is repeated [32].
3) Patients with VACS usually have one hypoplastic or stenotic VA, or the VA terminating as the posterior inferior cerebellar artery (PICA), and a contralateral, dominant, VA compressed or occluded mostly at the atlantoaxial junction when the head is turned away from the compression side [28, 32, 137]. VACS is confirmed if angiography documents compression of a dominant VA when vertigo occurs during head rotation or tilt [28], or by demonstrating a head rotation/tilt-induced decrease of blood flow in the posterior circulation using transcranial Doppler [156].

3. Lesion sites responsible for isolated vascular vertigo

In a study performed in the emergency department of a tertiary referral hospital, 47 (13.4%) of 351 patients with acute isolated vestibular or ocular motor symptoms of unclear etiology showed acute unilateral stroke on MRIs [189]. Volumetric analyses showed that medial cerebellar strokes are associated with vertigo, lateral cerebellar strokes with dizziness, and pontomesencephalic strokes with double vision [189]. In contrast, cerebral cortical lesions are rare and present with milder symptoms of shorter duration [119, 189].

3.1. Brainstem

In brainstem lesions, vertigo/dizziness is commonly associated with other neurological symptoms

and signs, but some patients with an isolated vestibular syndrome show a small lesion confined to the vestibular nuclei or root entry zone of the eighth cranial nerve at the pontomedullary junction [50, 169], dorsolateral medulla [86, 113, 170], pontine or midbrain tegmentum [46, 185, 189], or cerebellar peduncles [9, 23, 111, 154],

Patients with an infarction involving the caudal lateral medulla may present with isolated imbalance, probably due to interruption of the lateral vestibulospinal tract or dorsal spinocerebellar tract. In a study of 105 patients with AVS and at least one stroke risk factor from a single academic medical center, approximately 15% (15/105) of the patients with a stroke had isolated AVS from a small ($\leq$10 mm) infarction, and 11 of them (11/15, 73%) showed a lesion involving the inferior cerebellar peduncle, mostly in the lateral medulla (9/11, 82%) [154]. Only one patient showed an isolated small infarction in the cerebellum [154], which is known as one of the most common sites causing AVS [21, 106]. The inferior cerebellar peduncle carries various input and output fibers to and from the cerebellum, which are mainly concerned with integrating proprioceptive sensory inputs with vestibular signals important for balance. Proprioceptive information from the body is carried to the cerebellum via the posterior spinocerebellar tract in the inferior cerebellar peduncle. The vestibulocerebellum also receives mossy fiber inputs from the vestibular nuclei and nerve, and projects efferent fibers to the vestibular nuclei via the inferior cerebellar peduncle. Thus, an infarction involving the inferior cerebellar peduncle may result in imbalance with vertigo and nystagmus [23]. Since the medial vestibular nucleus is more vulnerable to ischemia than other structures in the brainstem or cerebellum according to a animal study [107], ischemia of the dorsolateral medulla where the vestibular nuclei are located may be a mechanism of isolated vascular vertigo. Indeed, several studies described isolated vertigo from infarctions restricted to the vestibular nuclei [86]. Rarely, cerebral hemispheric infarctions involving the vestibular cortices can cause isolated vertigo with spontaneous nystagmus and subjective visual vertical (SVV) tilt [1, 13, 176].

However, since previous reports on isolated vestibular syndrome of vascular cause are mostly limited to anecdotal case reports, small case series from a single center, and specific subtypes of posterior circulation ischemia, the overall frequency and the structures involved remain to be determined in strokes presenting with isolated vestibular syndrome.

3.2. Cerebellum

The frequency of acute isolated vascular vertigo and the structures involved were analyzed in 132 prospectively recruited consecutive patients with posterior circulation infarctions in a referral Stroke Center [21]. This study found that approximately 26% (34/132) of patients with posterior circulation infarction present with isolated vestibular syndrome: cerebellar infarction (67.6%) was most frequent, mostly in the territory of medial PICA. These results are consistent with those of previous and recent studies that showed a high frequency of medial PICA infarction in patients presenting with acute isolated vascular vertigo, and frequent isolated vertigo in medial PICA infarction [106, 189]. Indeed, dysmetria, a major sign of cerebellar dysfunction, may be minimal or absent in cerebellar infarctions involving the territory of medial PICA, especially when the infarction is not large. In the cerebellum, the nodulus and ventral uvula may cause isolated vestibular syndrome when damaged [104, 127]. A study of eight patients from a single center also showed that isolated nodular infarction mostly presents with isolated vertigo and imbalance without other neurological deficits, mimicking AUVP/vestibular neuritis [127]. The flocculus and parafloccculus may be other neural structures leading to isolated vestibular syndrome [110, 145, 182]. They participate in the control of smooth tracking, gaze-holding, and eye movements induced by vestibular stimulation. Experimental lesions cause gaze-evoked nystagmus, downbeat nystagmus, post-saccadic drift, impaired smooth pursuit, and impaired cancellation of the vestibulo-ocular reflex (VOR) [188]. However, since the flocculus is supplied by a branch from the AICA, which also supplies the dorsolateral pons and inner ear, an infarction involving the flocculus usually accompanies other brainstem signs or hearing loss [4]. Studies have also suggested the inferior cerebellum as a lesion site responsible for isolated vascular vertigo. In AVS due to stroke, the lesions are mostly found in the cerebellum, usually in the territory of PICA [106]. In a retrospective study of 240 patients with a cerebellar infarction in a single center, isolated vestibular syndrome mimicking AUVP/vestibular neuritis was found in 10% (25/240) of patients [106].

3.3. Inner ear

Ischemia of the inner ear may cause isolated vascular vertigo/dizziness due to its requirement for high-energy metabolism and absence of collateral

circulation [90, 138]. The labyrinth and its individual components appear to be vulnerable to ischemia because the IAA is an end artery with minimal collaterals from the otic capsule [125], such that blockage of the IAA leads most often to a severe peripheral vestibular deficit and loss of hearing (see above). By contrast, the vestibulocochlear nerve is less vulnerable to ischemia based on the arterial system of the internal auditory canal [125]. It is nearly impossible to document isolated labyrinthine infarction, labyrinthine component infarction, or vestibulocochlear nerve infarction without a pathologic study [36, 90, 115].

4. Epidemiology of vascular vertigo/dizziness

Approximately 20% of ischemic events are known to involve the neural structures supplied by the posterior (vertebrobasilar) circulation, and vertigo/dizziness is one of the most common symptoms of vertebrobasilar diseases [148, 157]. Recent large database prospective studies also reported dizziness as a presenting symptom in 47–75% of patients with posterior circulation stroke [3, 159]. In the USA, dizziness and vertigo account for 3.3% to 4.4% of visits to emergency departments [132], and stroke is responsible for 3–4% of these presentations [74, 75, 132]. Furthermore, those patients hospitalized with isolated vertigo have a 3 times higher risk for stroke (95% CI, 2.20–4.11; $p < 0.001$; absolute risk, 6.1% vertigo group vs 1.9% comparison group) than a comparison group of patients hospitalized for appendectomy during the 4-year follow-up [101]. Nearly all of the excess risk for stroke occurred in patients with vertigo who also had vascular risk factors. In particular, those patients with three or more risk factors had a 5.51-fold higher risk for stroke (95% CI, 3.10–9.79; $p < 0.001$) than those without risk factors [101]. Overall, patients with vertigo/dizziness showed a 2-fold higher risk of stroke or cardiovascular events (95% CI, 1.35–2.96, $p < 0.001$) than a non-dizziness comparison group during a follow-up of 3 years after adjusting for confounding and risk factors [100]. Thus, even if we accept that a proven cerebrovascular cause is rare in patients with isolated vertigo in unselected samples and the risk of future stroke is low, the future risk of stroke is considerably higher in those patients with vertigo/dizziness as compared with those patients with non-dizziness visits [5], especially when several vascular risk factors are present. Furthermore, there has been an accumulation of evidence indicating that posterior circulation ischemia can present with isolated vertigo without other focal signs [21, 106].

Vertebrobasilar ischemia is also a serious concern when patients present with acute transient vertigo [49, 54, 55].Vertigo typically occurs abruptly and usually lasts several minutes to hours [22, 49]. In a study of 84 patients with vertigo due to vertebrobasilar ischemia, 62% had at least one isolated episode of vertigo, and 19% developed vertigo as the initial symptom [55]. In another study, 21% of 29 patients with VB-TIA reported episodic vertigo as the only symptom for at least 4 weeks [54]. In a study of patients with posterior circulation stroke, 22% reported subtle transient neurological symptoms in the 90 days preceding their stroke, most frequently vertigo [148]. Patients with infarction in the territory of the AICA can also experience recurrent vertigo in combination with fluctuating hearing loss and/or tinnitus as the initial symptoms 1–10 days prior to an infarction [103]. Thus, to prevent future strokes, it is crucial to identify those patients presenting with vertiginous episodes as a symptom of a TIA [83, 173].

5. Evaluation of vascular vertigo/dizziness

When acute vertigo/dizziness accompanies other neurological symptoms and signs, diagnosis of central, most often vascular vertigo is straightforward in most cases even without documentation of a stroke with neuroimaging. Even though introduction of DWI has greatly enhanced detection of infarctions in patients with vascular vertigo/dizziness, especially due to compromised posterior circulation or atrial fibrillation, bedside neuro-otologic evaluation by experts has been more sensitive than acute MRI including DWI in detecting acute small infarctions seen on a delayed MRI as the cause of spontaneous vertigo lasting more than 24 hours, especially during the first 48 hours [21, 73, 134, 154].

Thus, vascular vertigo should be strongly suspected in patients with an AVS and vascular risk factors even though confirmation of a stroke is mostly based on the findings of the neurotological examination and imaging of the brain and cerebral vasculature [178]. Vascular causes should also be suspected in non-positional episodic vestibular syndrome, especially when the dizzy spells last only minutes in patients with risk factors for stroke [55]. Brain imaging assists in determining the involved territories and stroke etiology.

5.1. Clinical evaluation

Despite the marked progress in laboratory medicine and neuroimaging, systematic history taking and bedside examination provide the foundation for accurate diagnosis of vestibular disorders [41, 66, 75, 174]. Patients with vascular vertigo/dizziness invariably present with acute vestibular symptoms, either transient or prolonged [55]. Patients often have vascular risk factors or atrial fibrillation and, in most cases, present with other neurological symptoms and signs. History may disclose preceding attacks of vertigo/dizziness suggestive of VB-TIA [49, 55]. Patients with vertigo/dizziness should undergo a bedside evaluation for ocular misalignment including SD as a component of the ocular tilt reaction (OTR), spontaneous and gaze-evoked nystagmus, HIT, and gait and balance function [37, 43, 66]. Positional testing and examination of head-shaking nystagmus (HSN), saccades, and smooth pursuit may provide additional support in discriminating a central from a peripheral vestibular lesion [26, 37, 67].

5.1.1. Ocular tilt reaction (OTR) and tilt of the subjective visual vertical (SVV)

The OTR refers to the triad of head tilt, SD, and ocular torsion [14, 60, 181]. The OTR and SVV tilt may be attributed to unilateral lesions involving the pathways from the otolithic organs or semicircular canals [14, 52, 60]. SD indicates vertical misalignment of the eyes in the absence of an extraocular muscle palsy or strabismus. The presence of SD or other ocular misalignment should be determined with the alternating cover test. In OTR, the head tilt and ocular torsion occur toward the lower eye. Even though a small SD is also observed in peripheral vestibular disorders [56, 80, 96] (e.g., in 24% according to a study on 53 patients with acute unilateral vestibulopathy [96]), it has been included as a part of an ocular motor assessment to discriminate central from peripheral causes of AVS [37, 73]. Pronounced SD may be specific (98.1%) for stroke detection when larger than 3.3° [96], but is not very sensitive since SD is found in only about one third of patients with acute unilateral brainstem infarctions (31%, $n = 111$) [14]. Pathological SVV tilts (94%, $n = 111$) and ocular torsion (83%, $n = 111$) are the most sensitive signs of vestibular imbalance in the roll plane in patients with acute unilateral brainstem infarction [14], but these signs do not discriminate between a peripheral and central lesion [190]. SVV can be measured easily in the emergency department by the bucket test [190]. The measurement of ocular torsion requires magnified fundoscopy, fundus photography or a scanning laser ophthalmoscope [14, 44, 60, 190].

5.1.2. Spontaneous, gaze-evoked, head-shaking and positional nystagmus

Spontaneous nystagmus: Patients with AUVP/vestibular neuritis show spontaneous horizontal-torsional nystagmus that beats away from the lesion side [43]. The nystagmus is unidirectional and maximal when looking in the direction of the fast phases of nystagmus (Alexander's law). In contrast, pure downbeat, upbeat, or torsional nystagmus is well recognized in central vestibular lesions [114]. Marked suppression by visual fixation has been considered a hallmark of peripheral nystagmus [61]. The effects of visual fixation on spontaneous nystagmus are variable in central lesions [63, 121]. Failure of fixation suppression is observed in about 50% of patients with cerebellar infarctions, especially when the nodulus and flocculus are affected [84]. Thus, proper observation of nystagmus requires the use of Frenzel's goggles or M glasses [163]. Fixation may evoke nystagmus or augment spontaneous nystagmus in central lesions [27, 165].

Gaze-evoked nystagmus: Integrity of the central neural network can be evaluated by inducing eccentric gaze [114]. Direction-changing gaze-evoked or gaze-holding nystagmus in the horizontal or vertical plane is generally considered a sign of impaired neural integration from lesions involving the brainstem and cerebellum [43]. However, with a peripheral lesion, a reversal of the nystagmus direction can occur with gaze in the direction opposite to the spontaneous slow phase possibly based on a leaky neural integrator induced by an acute peripheral vestibular asymmetry [76, 151].

Head-shaking nystagmus (HSN) and positional nystagmus: Both HSN and positional nystagmus may give additional information. HSN may be ipsi- or contralesional according to the location and extent of central lesions [31, 65, 67]. However, vigorous HSN (> 50°/s) or HSN with cross-coupling (mostly downbeat after horizontal head shaking) should be considered a central sign [184]. Vigorous horizontal HSN is typically observed in patients with lateral medullary infarction [31]. HSN with cross-coupling has been reported in strokes involving the cerebellum or brainstem [25, 65, 67, 82, 87], and has been explained by enhanced responses of the anterior semi-

circular canal pathway due to cerebellar dysfunction [25].

Positional maneuvers can evoke nystagmus or modulate a spontaneous nystagmus in central as well as peripheral vestibular disorders. Central positional vertigo and nystagmus may be paroxysmal (< 1 minute in duration) or persistent [15, 24, 26]. Since the paroxysmal and persistent forms of central positional nystagmus may mimic the positional nystagmus of BPPV [15, 91], a central lesion should be suspected in patients with positional nystagmus atypical for BPPV, mimicking multi-canal BPPV, or positional dizziness and nystagmus refractory to repeated treatment maneuvers [91, 129]. Vascular causes are very rare in pure positional vertigo/dizziness, and there have been no convincing cases of a central lesion causing a nystagmus pattern typical of posterior canal BPPV: upward/torsional nystagmus with a transient crescendo-decrescendo pattern, elicited on the Dix-Hallpike/diagnostic Sémont maneuver to the affected side. Geotropic or apogeotropic central positional nystagmus can also be differentiated from BPPV involving the horizontal semicircular canal by the temporal profile of the positional nystagmus, associated central symptoms and signs, and no response to repeated canalith repositioning maneuvers [24, 26, 33]

5.1.3. Head impulse test (HIT)

The bedside HIT is a useful tool for differentiating central vascular vertigo from disorders of peripheral vestibular structures [37, 73, 133], but it has a low sensitivity and specificity to diagnose a vestibular deficit [186]. Therefore, whenever possible, the video-HIT should be used (see below) [118, 136]. Pathological HIT with corrective catch-up saccades due to a reduced gain of the VOR is generally considered as localizing to peripheral vestibular structures, particularly the vestibular nerve or labyrinth [59]. In contrast, a bilaterally normal HIT indicates that the peripheral vestibular function is intact, and therefore is suggestive of a central lesion in patients with AVS [73]. Indeed, bedside HIT was normal in 24 patients with isolated vertigo from cerebellar infarction involving the medial PICA territory [106]. However, the HIT may also be pathological in patients with cerebellar or brainstem strokes (3/34, 9%) [133]. Several studies documented pathological HIT in patients with lesions involving central vestibular structures such as the root entry zone of the vestibular nerve, vestibular nucleus [86], flocculus [145], and nucleus prepositus hypoglossi (NPH) [94]. In unilateral lesions involving the flocculus or NPH, pathological HIT may be more prominent when the head is turned to the intact side. Bedside HIT was also positive during contralesional head rotation in about 20% of patients with PICA or superior cerebellar artery territory infarction (4 of 20) [20]. Thus, while normal HIT is a strong indicator of central vestibular dysfunction in patients with AVS, pathological HIT does not necessarily indicate a peripheral lesion. Furthermore, bedside HIT may be normal (false negative), especially when the vestibular deficits are partial, e.g., in Menière's disease with a low-frequency hearing deficit during an attack, or when the corrective saccades occur during the head impulse (covert saccades) [37, 112, 126, 179]. Up to 12% of patients with AUPV/vestibular neuritis may have an isolated inferior divisional involvement and thus show normal horizontal HIT. [6, 89, 108].

The video-HIT can provide objective measurements of VOR gains and also document isolated vertical canal involvement. [8, 117]. The sensitivity of clinical HIT for identifying vestibular hypofunction at the bedside ranges widely between 35% and 71% depending on the test technique and the extent of vestibular loss [10, 158, 186]. Examiner skill plays an important role in detecting an abnormal result [70], raising questions about whether inexperienced examiners should use the clinical HIT to make high-stakes triage decisions about stroke in acute dizziness in the emergency department [38, 143]. Eye and head movements can be recorded at the bedside using lightweight portable video goggles with an integrated high-speed infrared camera [8, 117]. The video-HIT (see below) can assist clinicians to correctly perform a standardized HIT and facilitate interpretation of the test results [128]. Portable VOG can be used in the emergency department in real time to help differentiate brain infarction from AUVP/vestibular neuritis in patients with AVS [120, 135].

5.1.4. Central ocular motor signs

Saccades: Abnormal saccades may be a feature of central lesions. Slow vertical saccades in association with or without a gaze palsy indicate a lesion in the midbrain affecting the rostral interstitial nucleus of the medial longitudinal fasciculus and slow horizontal saccades indicate a lesion involving the paramedian pontine reticular formation [114]. Hypermetric saccades suggest lesions involving the cerebellum (fastigial nucleus) or lateral medulla, and hypometric saccades imply lesions involving the dorsal ocular motor vermis [114].

Smooth pursuit: Impaired smooth pursuit is also considered a central sign and is considered a hallmark of cerebellar disorders though it is not specific for cerebellar dysfunction. Smooth pursuit is typically less relevant in identifying acute vascular vertigo and should be interpreted with caution since it could be affected by various factors including an underlying spontaneous nystagmus, medications, and cognition.

5.1.5. Posture and gait

Patients with vascular vertigo should have an evaluation of balance while standing and balance during gait. Severe postural instability is a predictor of a central lesion [57, 106, 174, 183].

5.2. Laboratory tests

5.2.1. Examination of the vestibular, ocular motor, and auditory systems

VOG may aid in documenting and characterizing SD, spontaneous and triggered nystagmus, and other ocular motor function including saccades and smooth pursuit [96, 136]. Caloric tests are mostly normal and are of limited value in assessing the horizontal VOR in central vestibular disorders. Ocular and cervical vestibular evoked myogenic potentials (o/cVEMPs) have been used to evaluate the function of the otolithic pathways in central as well as peripheral vestibular disorders [140]. In patients with isolated vestibular nucleus infarction, o/cVEMPs were diminished or absent during stimulation of the ipsilesional ear [86]. Above the vestibular nucleus level, oVEMPs are impaired in lesions involving the medial longitudinal fasciculus [85, 141]. In unilateral cerebellar infarction, o/cVEMPs are frequently impaired [35]. Thus, abnormalities of VEMPs are not helpful in differentiating peripheral from central vestibular disorders [47].

Ocular torsion can be measured with magnified fundoscopy, fundus photography or scanning laser ophthalmoscope [44, 60]. Patients may have quantitative evaluation of the SVV, for instance using the Bucket Test or more specialized equipment. A deviation of SVV is found acutely in more than 90% (48/51) of patients with unilateral vestibular lesions [80], but it does not discriminate between a peripheral and central lesion [190].

In patients with acute auditory symptoms, pure tone and speech audiometry can aid in documenting hearing loss, in particular if an AICA-infarction or Menière's disease are suspected [105].

5.2.2. Blood tests

In general, routine laboratory studies including complete blood counts, electrolytes, and thyroid function tests have a very low yield in diagnosing a cause of dizziness. In a meta-analysis, only 26 of 4,538 patients (0.6%) had laboratory abnormalities that could explain their dizziness [62]. The ischemic stroke guidelines also recommend a limited number of hematologic, coagulation, and biochemistry tests during the initial emergency evaluation, and only the assessment of blood glucose must precede the initiation of intravenous recombinant tissue plasminogen activator [68]. If giant cell arteritis is a concern, inflammatory markers should be checked. The diagnostic value of serum biomarkers in differentiating central from peripheral causes remains to be elucidated [77, 162].

5.2.3. Cardiovascular work-up

Extra- and transcranial Doppler/duplex sonography, ECG monitoring, and echocardiography are recommended in patients with acute ischemic stroke [68].

5.3. Imaging

Neuroimaging studies are essential in the evaluation of stroke. CT has a limited value in detecting acute posterior circulation infarction, and is only recommended to detect hemorrhages or other pathologies [17, 42]. Introduction of DWI has greatly enhanced detection of infarctions in patients with isolated vascular vertigo. However, even DWI may miss up to one in five strokes occurring in the posterior fossa when performed during the first 24–48 hours, though this may relate in part to the MRI protocol of slice thickness and gaps [21, 45, 73, 142]. False negative initial MRIs (6–48 hours) were more common with small ($\leq$10 mm) strokes than larger ones (53% vs 7.8%, $p < 0.001$) [154]. Furthermore, current imaging technique cannot detect isolated labyrinthine infarction that may progress to involve the portions of the brainstem and cerebellum supplied by the AICA [36, 88]. Thus, serial evaluation should be considered in patients with suspected vascular vertigo when the initial DWIs are normal [21, 73].

It is challenging to visualize an image correlate of acute isolated vascular vertigo/dizziness when MRI, including DWI, does not show any evidence of acute infarction. Since perfusion CT or MRI can detect hypoperfusion and potentially reversible injury to the brain, they may aid in detection of stroke in patients with isolated

vascular vertigo/dizziness especially when MRI, including DWI, is normal [79, 161, 167]. Perfusion imaging mostly has been used to assess the risks or benefits of stroke intervention or to predict the outcome of an infarction [167]. However, perfusion imaging does not readily detect small perfusion reductions in the brainstem, and thus, the diagnostic yield of perfusion imaging still needs to be validated in isolated vascular vertigo/dizziness [124].

Imaging of the cerebral vasculature using CT/CT-angiography or MRI/MR-angiography can be considered in patients with suspected vascular vertigo/dizziness. However, the evidence base for vascular interventions in the posterior circulation is substantially less than that for interventions in the anterior circulation [109]. Unilateral cerebellar hypoperfusion mostly is caused by stenosis or occlusion of the ipsilesional VA or proximal PICA. In one study, 80% (8/10) of patients with cerebellar hypoperfusion on perfusion CT or MRI showed a luminal irregularity or hypoplasia of the corresponding VA [22]. VA hypoplasia may be a risk factor for posterior circulation ischemia, especially when other vascular risk factors coexist [72, 146]. A study using perfusion CT revealed that VA hypoplasia can lead to a relative regional hypoperfusion in the territory of PICA [168]. In one study, patients with vertigo/dizziness of unknown etiology had a higher prevalence of VA hypoplasia than that in a control group [144]. A unilateral hypoplastic VA may rarely cause recurrent isolated vertigo and subsequent cerebellar infarction [2]. In patients with cerebellar hypoperfusion on perfusion CT or MRI in the presence of a normal VA on the corresponding side, focal atherosclerosis or dissection of the PICA should be suspected, which may require conventional digital subtraction angiography for documentation [79, 147].

Although current imaging cannot detect isolated labyrinthine infarction, observation of a hypersignal in the labyrinth on pre-enhanced T1 or FLAIR MRIs suggests the rare diagnosis of labyrinthine hemorrhage [78, 160, 180]. Vascular imaging of the neck can be used to diagnose VA dissection, in particular in the setting of neck pain or trauma [58].

Overall, the rate of false negative imaging in vascular vertigo/dizziness further supports the relevance of a systematic patient history and bedside as well as laboratory evaluation of vestibular and ocular motor function to look for central signs, which permits a differentiation of acute vascular vertigo/dizziness from AUVP/vestibular neuritis with a sensitivity and a specificity of about 90%.

6. Conclusion

Determining the characteristics of vestibular symptoms, associated central symptoms, and vascular risk factors should be the first step in establishing a diagnosis of vascular vertigo/dizziness. A systematic examination focused on central vestibular and ocular motor signs, especially HINTS and evaluating postural instability provides more accurate diagnostic information than early (< 48 hrs.) imaging in patients with vascular vertigo/dizziness. Video-HIT and video-oculographic recording of eye movements may help to increase diagnostic accuracy. Identifying the underlying etiology of vascular vertigo/dizziness is essential for guiding the selection of appropriate management options, including both acute treatment and secondary prevention of future stroke.

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