EPISODIC VESTIBULAR SYNDROME

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Episodic Vestibular Syndrome [a.k.a. Transient Spontaneous Dizziness]

Overview

The *episodic vestibular syndrome* (EVS) is a clinical condition characterized by recurrent dizziness or vertigo that develops acutely (over seconds, minutes, or hours); may be accompanied by nausea/vomiting, gait instability, nystagmus, or head-motion intolerance; disappears clinically within 24 hours; and is not linked to head motion, head position/posture, or body position/posture. While many patients with this clinical syndrome suffer from Menière disease, vestibular migraine, or reflex/vasovagal (pre-)syncope, some instead harbor dangerous disorders such as life-threatening cardiac arrhythmia or vertebrobasilar transient ischemic attack (TIA).

Patients with TIAs resulting from tight vertebral or basilar artery stenosis may present with repeated, episodic vertigo that is 'isolated' (without any other neurologic symptoms such as numbness, weakness, etc.) over a period of weeks, months, or occasionally years before suffering a completed stroke.[1] Dizziness is the most commonly encountered symptom in basilar artery occlusion,[2] and isolated dizziness is the initial complaint in roughly 20% of cases.[3] Since vertebro-basilar occlusion is associated with serious morbidity or mortality,[4] there is a high premium on disease recognition and stroke prevention prior to occlusion.

Epidemiology of EVS

Dizziness/vertigo is the third most common major medical symptom reported in general medical clinics[5] and accounts for roughly 3-5% of visits across clinical settings.[6] This translates to ~10 million ambulatory visits per year in the US for dizziness[7] with ~25% of these visits to emergency departments (ED).[6] It is likely that ~70-80% of patients present with episodic dizziness, but it remains unknown what fraction of these patients have EVS, as opposed to triggered positional or postural dizziness symptoms. Based on data from a German population-based survey, the 1-year prevalence of recurrent vestibular vertigo lasting less than 24 hours is approximately 4%; about 40% had brief, positional dizziness symptoms, so perhaps 2.5% have EVS.[8] Generalizing these data to the US population, we estimate that there are over 7.5 million people in the US every year who suffer from EVS, and it is likely that many of these patients visit an ED or ambulatory care physician.

Differential Diagnosis of EVS

We are unaware of any studies in unselected patients with EVS (often referred to by names such as "recurrent attacks of vertigo"[9]). The differential diagnosis for transient non-positional dizziness or vertigo includes both vestibular disorders (e.g., Menière disease, vestibular migraine, vertebrobasilar TIA) and non-vestibular disorders manifesting vestibular symptoms (e.g., reflex pre-syncope, cardiac arrhythmia, panic disorder, hypoglycemia). The precise relative prevalence of these disorders is unknown, though reflex (vasovagal) syncope[10] and vestibular migraine[11] are likely among the most common causes. *See the "New Approach" handout for a differential diagnosis of common and dangerous causes*.

Predictors of TIA in EVS – The History – Type, Timing, & Triggers

The *type* or *quality* of dizziness is often the main focus for directing diagnostic inquiry in patients with dizziness.[12] Classic teaching[13] and current US practice[14] subdivides dizziness into four types based on symptom quality, each

said to predict the underlying etiology: (i) vertigo (false sense of spinning or motion), (ii) presyncope, (iii) unsteadiness, and (iv) nonspecific/other dizziness.[15;16] Despite the fact that EVS has sometimes been referred to as "recurrent attacks of vertigo,"[9] the type of dizziness in EVS is not likely a strong predictor of underlying etiology under most circumstances. Studies in patients with acute dizziness indicate that the type of dizziness is an imprecise, unreliable metric.[17] These results accord with disease-based studies indicating different or overlapping types of dizziness are present in patients with disorders known to cause EVS such as migraine, syncope, panic disorder, and hypoglycemia (reviewed in Newman-Toker et al.[17]). Even patients with primary cardiac diseases such as arrhythmia, myocardial infarction, and aortic dissection often present with transient vertigo as opposed to presyncope or other types of dizziness.[18;19] Transient vertigo is apparently a predictor of stroke in the general population (relative incidence ratio 2.5), while non-rotatory dizziness is not.[20] However, about 10% of patients with cerebral hemispheric infarction report transient, non-rotatory dizziness at onset[21] and perhaps as many as 32% of patients with vertebrobasilar insufficiency present with presyncope.[22] Thus, the type of dizziness is not helpful.

The *timing* of dizziness or vertigo helps define the EVS category, since disorders such as vestibular neuritis or completed posterior fossa stroke generally produce a prolonged period of continuous dizziness or vertigo lasting days to weeks known as the *acute vestibular syndrome* (AVS) (*see AVS handout for details*). Disorders causing AVS are not generally important considerations in EVS patients, though vestibular neuritis is preceded by a single, transient episode of dizziness in about 25% of cases,[23] and completed stroke in either the anterior[24] or posterior circulation[25] may be associated with transient dizziness or vertigo that resolves within minutes to hours. Within the 24-hour time frame, some disorders are probably excluded by symptoms lasting hours. For example, dizziness from cardiac arrhythmia or hypoglycemia is more likely to last seconds or a few minutes than to persist for hours, since stable cerebral hypoperfusion or metabolic insufficiency without brief loss of consciousness or frank coma is unusual. Unfortunately, disorders such as migraine and TIA are known to produce symptoms with almost perfectly overlapping *episode duration* profiles. Vestibular migraines last <5 minutes (18%), 5-60 minutes (33%), and 1 hour to 1 day (21%)[11] while vertiginous TIAs last seconds (10%), minutes (80%), or hours (10%).[1] *Abrupt-onset* of symptoms may be a clue to an ischemic etiology, though this has not been studied prospectively. The *number of episodes* and *total illness duration* are likely predictors of the underlying etiology, though this also has not been prospectively studied. Although it is clear that the increased risk of stroke after TIA occurs predominantly during the first 3 months after the initial event,[26] patients with vertiginous TIA have been reported to have multiple recurrences of isolated vertigo for up to 2 years leading up to a completed stroke.[1] Patients with numerous, stereotyped episodes of isolated vestibular or combined audiovestibular symptoms over a period of 5 or more years are presumably more likely to have vestibular migraine or Menière disease than TIA.

The *triggers* for most patients with transient dizziness are positional or postural, and disorders causing a *positional vestibular syndrome* (PVS), particularly benign paroxysmal positional vertigo (BPPV) and orthostatic hypotension, are considered elsewhere. Triggers among EVS patients are usually absent or less tightly linked to the onset of vestibular symptoms. For example, patients with vestibular migraine or Menière disease may report food (e.g., red wine), hormonal (e.g., menstrual), sensory (e.g., visual motion), environmental (e.g., barometric pressure change), or lifestyle (e.g., stress) triggers for their symptoms. Similarly, patients with reflex syncope or panic disorder may report specific triggers (e.g., micturition, pain/sight of blood, crowded elevator). When triggers are clearly present, they usually support a benign diagnosis over a dangerous one since disorders such as TIA, cardiac arrhythmia, and hypoglycemia (insulinoma) tend to be unprovoked.[18] Exceptions include exertional triggers, use of hypoglycemic medications, exposure to carbon monoxide, and, rarely, horizontal head turning (rotational vertebral artery syndrome[27]). Care must be taken to distinguish between symptoms which are provoked (triggered) by head movements, and those exacerbated by head movements. All dizziness of vestibular origin is exacerbated by head movement, regardless of peripheral (benign) or central (malignant) localization, in the symptomatic phase of the illness. During a posterior circulation TIA (e.g., in the cerebellum), a patient may well note that their symptoms, which began spontaneously, are exacerbated by head movement; this fact does *not* imply an inner ear or benign cause.[28] Visual stimuli as a trigger[29] may be diagnostic of migraine.

Predictors of Stroke in EVS – The History – Associated Symptoms & Risk Factors

Neurovegetative (nausea with or without vomiting) and gait or postural symptoms are frequent in EVS, regardless of the underlying cause. The most common accompanying *neurologic symptoms* in patients with vertebrobasilar insufficiency are visual disturbances (69%), drop attacks (33%), incoordination (21%), weakness (21%), confusion (17%), headaches (14%), syncope (10%), numbness (10%), and dysarthria (10%).[1] Visual disturbances include diplopia, illusions, hallucinations, field defects, and blindness.

When *"focal" motor neurologic symptoms* such as dysarthria, diplopia, or weakness accompany transient dizziness or vertigo, the correct diagnosis is usually TIA. Although visual (99%) and somatosensory (31%) auras are not uncommon among migraineurs, motor auras occur infrequently (6%).[30] This is in contrast to TIAs, where spinal motor manifestations are prototypical — among all comers with transient neurologic symptoms,[31] and those specifically with vertigo,[32] motor symptoms increase the odds of a cerebrovascular cause roughly 15-fold. Although most patients with vertigo have visual complaints that may include a disturbing sense of visual motion (oscillopsia),[33] a clear co-morbid complaint of transient binocular double vision (implying ocular misalignment), in a patient with episodic vertigo should raise immediate concerns over possible brainstem or cerebellar TIA, since frank diplopia is rare among typical vestibular migraine patients, occurring in only about 1%.[34] Differential diagnostic possibilities include basilar migraine[35] or hypoglycemia,[36] which may present with focal motor findings.

A history of co-morbid *afferent visual symptoms* such as bilateral central blur/dimming or hemianopsia probably do not differentiate effectively between posterior-circulation TIA and migraine, although a classic visual aura contemporaneous with the vestibular episode would certainly make migraine the most likely diagnosis. Co-morbid *confusion* likely broadens the differential diagnosis to include intermittent carbon monoxide exposure, as well as migraine, TIA, and hypoglycemia. Co-morbid *sensory symptoms* such as distal or cheiro-oral paresthesias probably do not differentiate well among TIA, migraine, panic/hyperventilation, and hypoglycemia.

A history of associated *cardiorespiratory symptoms* (e.g., palpitations, chest pain, dyspnea), should prompt suspicion of underlying cardiac pathology, even in the presence of true, spinning vertigo, which, contrary to conventional wisdom, is frequent in patients with primary cardiovascular disease.[18;19] Dizziness or vertigo followed by true *syncope* is probably a predictor of non-vestibular disease in most cases. Patients with reflex syncope or cardiac arrhythmias frequently lose consciousness. By contrast, patients with Menière disease (72%) experience short-lasting drop attacks,[37] but syncope is unusual. Patients with critical basilar stenosis are more likely to have sudden falls *without* loss of consciousness (i.e., drop attacks) than true loss of consciousness (14% vs. 5%).[1]

Unlike syncope, *drop attacks* or *room tilt illusions* generally point to a vestibular cause, and should generally be assumed to indicate brainstem ischemia until proven otherwise. Drop attacks are common in patients with Menière disease[37] and occasionally seen in patients with other inner ear diseases.[38-40] However, in the absence of a clear Menière disease history (i.e., onset before age 50, years of repeated dizziness/vertigo episodes associated with fluctuating tinnitus and/or hearing loss with most attacks, progressive low-frequency hearing loss), they should spark concern for an underlying cerebrovascular[41;42] or cardiovascular[43] etiology for dizziness/vertigo. Although drop attacks are also seen in epilepsy and narcolepsy-cataplexy, in such cases, they are generally not associated with dizziness. Room tilt illusions (where the entire room briefly appears tilted at some fixed angle or even inverted 180 degrees) can be seen in patients with Menière and other inner ear diseases,[44] but, in the absence of a convincing history of Menière disease or symptoms suggesting otic involvement (e.g., symptoms provoked by noise [Tullio phenomenon], valsalva, or vibration), a cerebrovascular cause should be surmised.[45;46]

Although *tinnitus or hearing loss* accompanying vertigo usually reflects an inner ear localization (and is therefore sometimes thought of as benign and comforting as a symptom), the blood supply to the inner ear derives from the posterior circulation.[47] Thus, mixed audio-vestibular symptoms lasting seconds or minutes, (whether unilateral or bilateral) should spark concern for TIA in the labyrinth and cochlea.[48;49] At least 10-20% of patients with vertebrobasilar insufficiency have transient tinnitus or hearing loss.[1] Transient dizziness, hearing loss, and/or tinnitus may occur as a premonitory TIA preceding acute audio-vestibular loss due to AICA infarction in up to 42% of cases.[48;50] Similar transient auditory symptoms occur in Menière disease,[51] and other less common otologic disorders such as autoimmune inner ear disease and perilymph fistula.[9] Although Menière disease can occur *de novo* in older patients,[52] great caution should be exercised before applying a new Menière disease diagnosis in an acute care setting, since TIAs in the basilar territory may mimic the disorder precisely (vertigo, tinnitus, hearing loss, drop attacks).[1;53] Approximately 20% of patients with migraine may experience tinnitus, hearing loss, or pitch distortion.[54] Patients with true vertigo of cardiac cause ('cardiogenic vertigo') may experience auditory symptoms such as transient bilateral reduction of hearing (things sounding 'far away' or 'muffled') immediately prior to syncope, but they may also experience tinnitus.[55] Aural symptoms such as a feeling of fullness, pressure, or discomfort in the ear are probably less specific, occurring in migraine,[56] Menière disease, other otologic disorders, and perhaps non-otologic conditions such as TIA.

When a patient with EVS has associated *craniocervical* pain, the differential diagnosis primarily includes migraine, TIA (especially associated with vertebral artery dissection), and, rarely, carbon monoxide intoxication. Because migraine is so common, it often represents the default explanation in young patients with unexplained transient

neurologic symptoms.[57] By contrast, TIA is rarely considered in a patient under age 50 unless they are known to have significant personal or familial vascular risk factors for early atherosclerosis. Unfortunately, when young people *do* suffer TIAs and strokes, the most common identifiable cause is not atherosclerosis, but cervicocranial arterial dissection,[58] and this holds true for infratentorial (posterior fossa) infarcts.[59] After head or neck pain, vertigo is the most common presenting symptom of vertebral artery dissection,[60] and resulting cerebellar TIAs produce short-lived vertiginous episodes.[61] Vertebral artery dissection can be mistaken for migraine[62;63] because at least 70% of cases are associated with headache or neck pain.[64] Although we often think of arterial dissection as a traumatic disorder, many cases are "spontaneous."[65] The majority of vertebral artery dissections seem to occur *without* an identifiable history of trauma,[64;66] and, even among those *with* such a history, the "trauma" is often trivial or minor (e.g., holding head in awkward posture, chiropractic neck manipulation).[67] Although the presence of clear food or visual triggers for dizziness, associated photo- or phono- phobia, or classic visual aura can sometimes suggest migraine as the cause, a non-trivial fraction of migraine vertigo patients lack these telltale symptoms (e.g., 30% have no photophobia, and 64% have no visual or other auras[33]).

The *nature of the headache or neck pain* may be helpful diagnostically in distinguishing migraine from TIA due to vertebral artery dissection. The pain of migraine is typically gradual in onset, building to peak intensity over 30 minutes or more in more than three fourths of cases.[68] By contrast, the pain of vertebral dissection is abrupt in onset in about half of cases.[64;66;69] Furthermore, for reasons that remain unclear, headaches associated with *vestibular* migraine tend not to be severe (12%),[34] while those associated with dissection are severe in more than half.[66;69] Migraine is a medically-benign, episodic disorder characterized by episodic neurologic symptoms lasting minutes to hours, in association with *episodic* cranial or cervical pain, lasting up to 72 hours.[70] By contrast, dangerous acute or subacute diseases of extra- and intra-cranial blood vessels (e.g. dissection or vasculitis) tend to produce episodic neurologic symptoms lasting minutes to hours in association with *persistent*[60] cranial or cervical pain, lasting days or weeks, up to 1-3 months.[65] Thus, the presence of persistent pain, particularly posterior cervical or retro-auricular in location,[65] in a patient with vertigo lasting minutes to hours should spark concern for underlying vertebral artery dissection. Although sustained pain greater than 72 hours is a red flag, the converse is not true — pain that remits within 72 hours cannot reliably be considered benign, since about half of vertebral dissection patients have headaches that last three days or less.[65] Thus, sudden, severe, or sustained (>72 hours) pain should spark concern for vertebral artery dissection rather than vestibular migraine.

Unfortunately, as mentioned above, TIAs resulting from tight vertebral or basilar artery stenosis may present with repeated, episodic vertigo that is 'isolated' (without associated neurologic symptoms) for weeks, months, or even years before suffering a completed stroke.[1] Dizziness is the most commonly encountered symptom in basilar artery occlusion[2] and with posterior circulation ischemia in general,[3] and isolated dizziness is the initial complaint in roughly 20% of cases.[3] When there is no headache or neck pain (as occurs in about 30% of cases for both migraine[34] and vertebral dissection[64]) or the pain has abated,[65] migraine and TIA can be challenging to differentiate.[71-73] In such cases, consideration of typical vascular risk factors (e.g., chronic hypertension, diabetes mellitus, hypercholesterolemia, cigarette use) is obviously important.

When obtaining the medical history from a patient with EVS, one should also strive to identify any major known, *concurrent illnesses* (e.g., cardiac disorders, hypercoagulable state, multiple sclerosis) or *potentially-relevant exposures* (e.g., ear surgery, bacterial otitis media, ototoxic drugs, chemotherapy, or viral syndrome). It is also probably useful to seek a detailed personal and family history of similar episodes or migraine. However, virtually nothing is known about the predictive value of these historical elements for the underlying cause of EVS.

Predictors of Stroke in EVS – The Examination

The predictive value of physical examination in diagnosing patients with EVS has not been formally studied. Most patients are examined during the asymptomatic phase between episodes and have either normal examinations or non-specific abnormalities such as mildly reduced vestibulo-ocular reflex responses on one side or vibration-induced nystagmus. Vestibular migraine patients have been examined during the acute phase, and oculomotor findings suggest central localization (50%), peripheral localization (15%), or unknown localization (35%).[56] Patients with rare otologic disorders such as superior canal dehiscence syndrome may have pathognomonic findings such as inducible torsional eye movements during valsalva maneuver,[76] but this is the exception, rather than the rule. Thus, examination is unlikely to offer many important insights into the underlying etiology of EVS except under conditions where TIA is suspected and clear neurologic signs are still evident.

Other components of the exam might serve to identify disease risk factors such as post-surgical changes of the ear or impaired distal pulses, irregular heart rhythm, and carotid bruits.

Neuroimaging in EVS

Neuroimaging by MRI with diffusion-weighted imaging (DWI) has not been studied in patients with EVS, but may be able to identify minor strokes in those with transient dizziness or vertigo that resolved quickly.[25] In patients with transient clinical symptoms, DWI may be positive in up to 40%,[77;78] although this estimate is likely to be somewhat lower in the posterior fossa (*see AVS handout*). Thus, the presence of a normal MRI with DWI in a patient with EVS is insufficient to exclude a cerebrovascular cause. A full TIA workup may therefore be required to adequately stratify the patient's risk of a subsequent, more devastating vascular event.

Prognosis of AVS and Consequences of Diagnostic Failure

While details of management and treatment of EVS are beyond the scope of this course, a brief discussion of prognosis and the relevance of diagnostic failure is warranted. Dizziness is the ED symptom most often linked to missed diagnosis of stroke,[79] with 35% of cerebrovascular events in patients with *any* dizziness (and 44% in those with *isolated* dizziness) missed at first medical contact.[32] While the real-world impact of these errors on patient outcomes is incompletely known, available data suggest those misdiagnosed are probably at high risk for adverse outcomes (*see AVS handout*). Those presenting initially with unimpressive symptoms suggesting migraine rather than TIA can progress rapidly to cerebellar infarction followed by life-threatening obstructive hydrocephalus[80] or brainstem compression.[81] Patients mistakenly believed to have vestibular disease on the basis of symptoms being "vertiginous" may instead harbor life-threatening cardiovascular disorders such as complete heart block[18] or aortic dissection.[28] Those thought to have panic disorder or hyperventilation may instead have hypoglycemia.

Summary of EVS

EVS is likely an extremely common presentation across healthcare settings, and potential etiologies range from benign to deadly. Initial evaluation should include history emphasizing duration and possible triggers for episodes, accompanying symptoms, and relevant past medical history (especially vascular risk factors). Examination is generally unhelpful but should be focused on identifying general neurologic signs that suggest TIA. Imaging, if readily available and indicated, should be by brain MRI with DWI, since CT scans are grossly inadequate for ischemic stroke detection, particularly with minor strokes in the posterior fossa. Visual triggers suggest migraine; spontaneous (untriggered) episodes are worrisome; focal motor symptoms suggest TIA; sensory symptoms are only helpful if there is classic visual aura; auditory symptoms are only benign if the history clearly suggests Menière disease; and sudden, severe, or sustained pain is worrisome for vertebral artery dissection. A TIA, cardiac, and metabolic workup should be considered for anyone with EVS whose symptoms do not match vestibular migraine, Menière disease, vasovagal/reflex syncope, or panic disorder.

When true syncope is absent, the main differential diagnosis is between vestibular migraine/Menière disease and TIA. For vestibular migraine, visual motion (or food/hormonal) triggers, classic visual aura, photophobia, and/or a long (>5 year) personal history are most likely to confirm the diagnosis. For Menière disease, a typical history of fluctuating tinnitus, hearing loss, and aural fullness that evolve over minutes in association with vertigo spells, a long (>5 year) personal history, and gradually progressive auditory loss are most likely to confirm the diagnosis. TIA should be considered the leading diagnosis in anyone with a short total illness duration (< 6 months), vascular risk factors, recent trauma, or sudden, severe, or sustained (>72 hours) craniocervical pain.

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