

CASE REPORTS

Do Not Dismiss the Dizzy Patient: A Case of Wallenberg Syndrome Highlighting Red Flags for Central Vertigo in Primary Care

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Background

Wallenberg syndrome, or lateral medullary syndrome, is a type of posterior circulation stroke that is often mistaken for peripheral vestibular disease because there is no muscle weakness. Family practitioners and emergency doctors should be aware of the key signs to prevent diagnostic errors.

Case Description

A 70-year-old man with coronary artery disease, heart failure with reduced EF, hypertension, and hyperlipidemia who came into a Houston area ED with vertigo, right-sided facial paresthesias, difficulty swallowing, and imbalance. The first set of CT and MRIs was interpreted as normal; however, a neurology-directed reassessment led to the diagnosis of an acute non-hemorrhagic infarct involving the dorsolateral aspect of the right medulla and a thrombosis of the right vertebral artery. His treatment included dual antiplatelet therapy and statins, as well as physiotherapy.

Discussion

The case highlights three key learning points for general medicine: (1) ipsilateral cranial nerve dysfunction, ataxia, and dysphagia along with vertigo serve as key red flags necessitating immediate MRI scanning despite CT results; (2) HINTS is an evidence-based method of differentiating between central and peripheral vertigo at the bedside; and (3) lack of adequate control over cardiovascular risk factors, represented by an LDL level of 139 mg/dL and the absence of established general medical care, may have played a role in the etiology of the stroke.

Conclusion

Diagnosis of Wallenberg's syndrome needs to be considered for any patients complaining of acute vertigo and other neurological symptoms. Family doctors are very important in controlling risk factors, organizing care after strokes, and recognizing central vertigo syndromes.

Brief Background

Recognizing the differences between central and peripheral vertigo represents a fundamental competence of family medicine practitioners. Vertigo is among the most frequent complaints seen by primary care providers, contributing to about five percent of all outpatient visits in the United States.¹ Most cases are benign and peripherally mediated, including benign paroxysmal positional vertigo (BPPV) and labyrinthitis. Yet, a crucial number of presentations have central pathology, which poses an immediate threat to the patient's life, such as a stroke in the posterior circulation. Posterior circulation strokes account for around 20% of all ischemic strokes and are three times more likely to be overlooked initially than anterior circulation strokes, often during initial assessment in primary care or emergency departments.^{2,3}

Wallenberg syndrome, also known as lateral medullary syndrome, is the most frequent posterior circulation ischemic stroke syndrome and refers to infarction of the lateral medulla oblongata caused by occlusion of the intracranial vertebral artery (VA) and its branches.^{4,5} The risk factors for this syndrome include hypertension, diabetes mellitus, hyperlipidemia, and atherosclerosis.⁴ Typical manifestations involve ipsilateral numbness of the face and ataxia, contralateral hemibody loss of pain and temperature sensation, and hemiatrophy of the tongue, vertigo, nystagmus, dysphagia, hoarseness, and Horner's syndrome (ipsilateral ptosis, miosis, and anhidrosis). These manifestations typically present acutely at stroke onset. Because Wallenberg syndrome does not cause limb weakness, it can be mistakenly attributed to a benign peripheral vestibular disorder, such as BPPV or labyrinthitis, where limb weakness is similarly absent.

Dysphagia is one of the serious complications in this syndrome that is found in about 51% of the patients and is seen in the most severe form when compared to other forms of stroke. This is caused by injury to the nucleus ambiguus and the swallowing centers in the brainstem.^{6,7} This report reviews a case encountered at a Houston-area hospital in which the clinical hallmarks of central vertigo were initially hidden by a benign-appearing presentation, illustrating essential diagnostic red flags and management principles directly applicable to family medicine practice.

Case Report

A 70-year-old male with coronary artery disease (CAD) status post percutaneous coronary intervention (PCI) to the left anterior descending (LAD) artery, chronic systolic heart failure with reduced ejection fraction (HFrEF; ejection fraction [EF] 25–30% approximately 10 years prior), hypertension, and hyperlipidemia presented to the emergency department at a Houston-area hospital. He did not have a primary care physician. He

reported a gradual-onset right-sided headache followed by acute vertigo, right facial numbness, nausea, dysphagia, and inability to ambulate. He denied prior vertigo episodes or focal limb weakness.

Vital signs were stable. Neurological examination revealed central red flags: right partial ptosis, nystagmus, right facial paresis with nasolabial fold flattening, reduced light touch in the right V2 distribution, right-sided dysmetria, and right-sided ataxia with inability to stand. Notably, muscle strength was 5/5 bilaterally. Physical therapy assessment confirmed moderate assistance required for transfers and ambulation, with rightward listing and right-beating nystagmus on head thrust testing.

Laboratory studies revealed notable cardiovascular risk markers: sodium 140 mEq/L, creatinine 0.8 mg/dL, hemoglobin 15.5 g/dL, international normalized ratio (INR) 1.0, troponin I 9 ng/L, low-density lipoprotein (LDL) 139 mg/dL (elevated), and hemoglobin A1c 6.1% (consistent with pre-diabetes). These findings indicated untreated dyslipidemia and pre-diabetes, early risk factors for posterior circulation stroke. Initial CT and CT angiography (CTA) of the head and neck were read as unremarkable. Brain MRI without contrast was also initially interpreted as normal; upon neurologist-directed review and prompted re-read, the amended report confirmed (1) acute nonhemorrhagic dorsolateral right medullary infarct and (2) intracranial right vertebral artery occlusion. The CTA similarly revealed right V4 occlusion not identified on the original read. Transthoracic echocardiography (TTE) showed EF 35–39%, moderately dilated left atrium, and a finding that could not exclude an apical thrombus, raising the possibility of a cardioembolic etiology in addition to atherosclerotic vertebral artery disease. The patient was diagnosed with Wallenberg syndrome secondary to right VA thrombosis.

For secondary prevention of ASCVD, dual antiplatelet therapy (DAPT) was initiated with aspirin 81 mg daily and clopidogrel (Plavix) 300 mg loading dose then 75 mg daily for 30 days, followed by aspirin monotherapy.

Atorvastatin (Lipitor) 80 mg daily was initiated for secondary prevention of atherosclerotic cardiovascular disease (ASCVD). Pregabalin (Lyrica) 25 mg twice daily and baclofen (Lioresal) 10 mg every eight hours as needed were added for central pain management. A modified barium swallow study (MBSS) confirmed severe oropharyngeal dysphagia; enteral nutrition was initiated via Dobhoff tube. Endoscopic percutaneous gastrostomy (PEG) on hospital day 9 was unsuccessful due to superior stomach positioning, requiring general surgery consultation. Physical therapy (PT), occupational therapy (OT), and speech-language pathology (SLP) were engaged from hospital day two. Cardiology managed HFrEF with sacubitril/valsartan (Entresto) and carvedilol (Coreg), and case management coordinated inpatient rehabilitation facility (IRF) placement.

Discussion

The present case has immediate applicability to family medicine physicians in that vertigo without peripheral weakness does not necessarily indicate a peripheral origin of vertigo. Vertigo, nausea, and facial paresthesia in this particular case pointed to the possible diagnosis of peripheral vestibular disorders like BPPV and labyrinthitis, which are easily diagnosed and treated without urgent imaging in the outpatient clinic. However, this patient suffered a brain stem stroke. A reminder that the absence of limb weakness does not exclude a central etiology. It is imperative that any physician diagnosing a vertiginous syndrome be familiar with the clinical signs of central vertigo. Typical peripheral vertigo is characterized by positional nausea, horizontal uni-directional nystagmus, fatigable and extinguishable by fixation, and absence of other neurologic symptoms. Central vertigo is indicated by the triad of HINTS (Head Impulse test, Nystagmus type, Test of Skew) examination findings, direction-changing or vertical pure nystagmus, new ataxia, new cranial nerve sign, or “first or worst ever” sudden, severe headache.⁸ In the outpatient setting, ipsilateral facial paresthesia, ptosis, dysmetria, and dysphagia along with vertigo should prompt immediate referral to the emergency department rather than scheduling outpatient workup, as these features are inconsistent with benign peripheral vestibular disease.

The other equally important lesson from this case is the key drawback of CT as well as the absolute need for MRI in assessing suspected cases of posterior circulation stroke. MRI with diffusion-weighted imaging is the gold-standard method to make the diagnosis of lateral medullary syndrome. CT often does not detect brainstem infarcts.^{2,4} In this case, both the initial CT and CTA scans were normal. It was only after the neurologist reviewed the results that he noticed a dorsolateral right medullary infarct. Notably, the initial MRI was also interpreted as normal prior to neurologist-directed re-review. When imaging findings are this subtle, clinicians, particularly in the ED or inpatient setting, should explicitly communicate their clinical suspicion for posterior infarct to the radiologist before imaging is performed, so the radiologist knows exactly what to look for. If initial imaging is unremarkable but clinical suspicion remains high, a formal neuroradiology over-read should be requested. For the primary care physician, the practical implication is simpler: if central vertigo is suspected, do not wait for outpatient imaging, send the patient to the ED immediately and convey your suspicion to the receiving team. This is one phenomenon that has been well documented: posterior fossa susceptibility artifacts reduce early sensitivity to lateral medullary infarcts on MRIs and even more so on CT scans.^{2,4,5} For the practicing family physician, this implies that a normal CT scan should not be reassuring in cases where the presentation of a posterior stroke is highly suggestive.

These cardiovascular risks posed by this patient (i.e., hypertension, dyslipidemia, and pre-diabetes) indicate the problem of another sort, namely the consequences of unmanaged cardiovascular risks, which become catastrophic events. It was important to note in this case that the patient did not have any primary care physician, and that this was more an issue of healthcare access and not physician mismanagement. There are two lessons for family medicine physicians here, and these include ensuring that patients at high risk in your practice panel do not slip between the cracks, and advocating for public health initiatives in those communities that lack primary care services due to their high cardiovascular risk. This patient had no primary care physician, representing a gap in healthcare access rather than physician negligence. His untreated dyslipidemia and pre-diabetes (noted in his labs) represent unrecognized and unmanaged risk factors for posterior circulation stroke. Family physicians play a critical preventive role by proactively engaging high-risk unattached patients and advocating for public health outreach in communities with poor primary care access. The VA atherosclerotic disease is the most common cause of lateral medullary syndrome.

Inpatient Management

Dysphagia emerged as the primary inpatient management challenge and remains relevant to primary care during outpatient follow-up. It develops in approximately 51% of Wallenberg syndrome cases and represents one of the most severe and long-lasting stroke complications, arising from damage to the nucleus ambiguus.^{6,7} When dysphagia requires surgical gastrostomy, antiplatelet medications must be temporarily discontinued and cardiac clearance obtained, adding complexity to the perioperative course, a consideration that family physicians should be aware of when coordinating follow-up care.

Return to Primary Care

Family physicians serve as the longitudinal care coordinator for post-stroke patients discharged from rehabilitation facilities. Familiarity with the expected trajectory of Wallenberg syndrome, including residual gait ataxia, dysphagia, and central neuropathic pain, enables family physicians to anticipate ongoing needs and guide appropriate specialist referrals, including neurology for stroke follow-up, speech-language pathology for continued dysphagia management, physical and occupational therapy for functional rehabilitation, and cardiology for HFrEF and secondary stroke prevention. Patient and caregiver resources for long-term functional recovery should be provided at outpatient visits. Secondary prevention measures, including dual antiplatelet therapy for 30 days followed by aspirin monotherapy, intensive statin therapy, and blood pressure management per American Heart Association and American Stroke Association (AHA/ASA) secondary prevention guidelines, should be continued and monitored in the primary care setting.⁹⁻¹² The 2026

AHA/ASA Guideline for the Early Management of Acute Ischemic Stroke further endorses dual antiplatelet therapy as preferred over thrombolysis for patients with non-disabling deficits, expands endovascular thrombectomy eligibility for posterior circulation large vessel occlusions, including basilar artery occlusion within 24 hours of onset, and underscores the importance of high-intensity statin therapy and blood pressure control as cornerstones of secondary prevention.^{11,13}

This case highlights the importance of Wallenberg Syndrome as a diagnosis despite its rarity. The moment a patient is seen complaining of a sudden onset of vertigo with signs such as ipsilateral cranial nerve dysfunction, dysphagia, or ataxia, the practitioner should not get anchored on an outer ear condition. Timely identification, referral to an emergency department, and coordination of care are essential components in the management of Posterior Circulation Stroke.

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