CLINICAL VIGNETTE

Weak and Dizzy: What Goes Around Comes Around

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Case Presentation

A 66-year-old male with history of tobacco use and hypertension presented to the Emergency Department (ED) with a chief complaint of weakness and dizziness for 5 days. He described a sensation that the room was spinning. Symptoms worsened when standing or moving his head. Symptoms were sudden onset, and when they first occurred he sought care at an outside ED where a non-contrast computed tomography (CT) scan of the brain revealed no acute intra-cranial findings and he was discharged home. However, symptoms persisted so he presented to a different ED. At the second ED, review of systems revealed mild headache, double vision, hoarse voice, right facial anhidrosis, and decreased cold sensation over the right face and left upper extremity.

Initial vital signs were: temperature 36.4°C, pulse 107 beats/ min, blood pressure 157/96 mm Hg, respiratory rate 20 breaths/ min, and oxygen saturation 99% on room air. Physical exam was notable for mild right sided ptosis. The primary gaze of the right eye was slightly depressed and extroverted. There was vertical nystagmus on upward gaze. Sensory exam showed decreased sensation to temperature over the right face and left upper extremity. The remainder of the exam was grossly unremarkable including normal strength, coordination, finger to nose, and rapid alternating movements.

Laboratory values including unremarkable complete blood count, chemistry panel, and troponin. Magnetic resonance imaging (MRI) of the brain revealed subacute diffusion positive infarct involving the right posterolateral medulla, and small subacute infarct along the inferior aspect of the right cerebellar hemisphere. The patient was diagnosed with Wallenberg syndrome (lateral medullary infarction) and was admitted to a telemetry bed and started on aspirin, and high dose atorvastatin and clopidogrel, in addition to his chronic amlodipine. His symptoms gradually improved after 5 days. He was transferred to acute rehab and his diplopia and sensory deficits continued to improve, and after 35 days was discharged home. At eleven week follow up, the patient continued to improve with resolution of sensory deficits, and further decreased vertigo, though he continued to have mild ptosis and diplopia.

Discussion

The chief complaint of "dizziness" poses a challenge to any clinician. Its vague meaning usually can be more narrowly divided into presyncope, vertigo, and disequilibrium, though a significant amount of overlap exists between these categories and a broad non-specific dizziness category still exists.[1,2] In our case above, the patient's presentation is characteristic of vertiginous dizziness, i.e. the false perception of movement of self or environment usually in a spinning manner. Vertigo caused by an asymmetric activity within the vestibular system including the inner ear, vestibular nerve, medulla, and cerebellum.

The Emergency Department evaluation of vertigo includes assessment of peripheral versus central causes of vertigo.[1] An overwhelming majority of patients presenting to the ED with vertigo will have a peripheral etiology. Kerber et al. found only 2.7% of patients presenting to the ED with symptoms of vertigo were diagnosed with stroke or transient ischemic attack (TIA).[3] Nonetheless, because of the potential for permanent morbidity or mortality, it is essential to evaluate for central causes.

Classically, peripheral vertigo is severe, sudden onset, and improves with time. It is usually worse with position change. Central vertigo may be sudden or gradual, mild or severe, and may occasionally worsen with head position. Obviously these historical features overlap substantially. Perhaps the most important distinction is that peripheral vertigo is likely to occur in isolation or with associated hearing loss or tinnitus. Central vertigo, is likely to have other associated posterior circulation findings such as ataxia and the "Deadly D's": diplopia, dysmetria (finger to nose), dysdiadochokinesia (rapid alternating movements), dysphagia, dysaesthesia, and dysphonia.[2,4] Lastly, in peripheral vertigo, nystagmus should be unidirectional and is usually horizontal. Vertical nystagmus is highly suggestive of a central cause. When central vertigo is suspected MRI is the preferred imaging modality. CT scan is of limited utility due to poor visualization of the posterior fossa and low sensitivity for acute ischemia/infarction.[5]

Lateral medullary infarction (Wallenberg syndrome) is a long recognized cause of central vertigo and the most common presentation of occlusion of the intracranial vertebral arteries. Risk factors for developing Wallenberg syndrome include hypertension, smoking, and diabetes. Vertebral artery dissection from neck manipulation or in patients with Marfan syndrome or Ehlers Danlos syndrome can also lead to Wallenberg syndrome.[6] Vertigo is nearly always present in Wallenberg syndrome because the vestibular nuclei of the medulla are almost always involved. The most common sensory

finding is loss of temperature and pain in the contralateral limbs and ipsilateral face.[7] Dysphonia (hoarseness in this case) and dysphagia may also be present due to involvement of cranial nerves IX and X. Horner's syndrome (miosis, ptosis, and anhidrosis) may be present when the ipsilateral descending sympathetic nervous system is involved.[8] Although headache is rarely present in stroke, it is commonly seen in posterior circulation infarction as in this case. Treatment for lateral medullary infarction is the same as management for any acute ischemic stroke. The overall prognosis for patients with Wallenberg syndrome is better than most other stroke syndromes and most patients can return to satisfactory levels of function.[6]

This case highlights key points in the evaluation of a patient with weakness and dizziness in the ED. First, characterizing subjective dizziness as vertigo is important in narrowing the differential diagnosis and initiating the evaluation. Second, careful evaluation for associated signs and symptoms may strongly implicate central pathology. Many of these findings, however, such as miosis, anhidrosis, vertical nystagmus, crossed neurological findings and isolated loss of temperature sensation are rarely encountered in daily practice. Further complicating matters is that classic features such as positionality do not effectively rule in peripheral causes, and readily available imaging such as CTs do not effectively rule out central causes. These issues lead to frequent missed or delayed diagnoses for posterior circulation syndromes.[9] The solution to detecting central vertigo in general, and Wallenberg syndrome specifically is not simply "maintaining a high index of suspicion". The solution is a careful history, thorough neurologic/ cerebellar exam, and targeted use of MRI.

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