A 43-year-old female presents to the emergency department with a three-day history of vertigo with nausea and vomiting. She states that the onset was sudden and severe, and denies any history of similar incidents. She does not report any headache and has not noticed a change in her hearing or the presence of tinnitus. Neurologic exam was unremarkable, but she displayed gait instability to the left side. There is a horizontal right beating nystagmus present. Bedside caloric test reveals reduced response in her left ear.

What is the most likely diagnosis?

A. Isolated inferior cerebellar stroke
B. Episode of benign paroxysmal positioning vertigo
C. Vestibular neuronitis
D. Multiple sclerosis
E. Ménière’s disease
Answer: C, vestibular neuronitis

Overview

Vertigo is the illusory sense of movement of self or the environment. The etiology is notoriously difficult to diagnose, due in part to high levels of semantic confusion in the patient-physician interaction. ‘Dizziness’ accounts for approximately 4% of emergency department (ED) visits, but the physician must determine whether the sensation is true vertigo, presyncope, or imbalance due to another etiology—whether cardiogenic, neurologic or even psychiatric.

While oto-vestibular causes make up the single largest category (32.9%) of ED ‘dizziness’ diagnoses, the emergent nature of central causes of vertigo warrant careful clinical consideration. Epidemiological data indicates that the most common oto-vestibular cause of dizziness in the ED is vestibular neuronitis/labyrinthitis, followed by benign paroxysmal positional vertigo (BPPV) and Ménière’s disease.

Central vs peripheral vertigo

Central signs that indicate a neurologic cause include ataxia, weakness or paralysis, cranial nerve abnormalities, and central pattern nystagmus. Central nystagmus may be vertical, torsional or horizontal, is often multidirectional, and is not inhibited by visual fixation. Other eye movement abnormalities may be present. Peripheral signs that indicate involvement of the vestibular organ include hearing loss, tinnitus, and horizontal nystagmus, with the exception of nystagmus due to BPPV which is torsional. Peripheral nystagmus is characterized by inhibition with visual fixation and is consistent with Alexander’s law, in which nystagmus increases when the gaze is in the direction of the fast component. Positive bedside tests such as the Dix-Hallpike or head thrust manoeuvres can also help distinguish peripheral from central causes. The ‘HINTS’ test, consisting of a normal head impulse test, gaze evoked nystagmus, or positive skew has been shown to be up to 100% specific and 96% sensitive for vertebrobasilar stroke, with fewer false negatives than magnetic resonance imaging (MRI). Likewise, severity of symptoms provide diagnostic clues. The sensation of movement and subsequent nausea is often more severe in peripheral than in central causes of vertigo. Additionally, while the vertigo patient will almost always feel off balance, peripheral causes will rarely cause imbalance severe enough to cause functional limitations in ambulation.

The differential of peripheral pathologies can often to be narrowed to the three most common oto-vestibular causes of vertigo. Vestibular neuronitis, Ménière’s disease and BPPV can be differentiated based on history, duration of symptoms, and auditory abnormalities. Episodes lasting seconds to minutes suggest BPPV, while minutes to hours suggest Ménière’s, and hours to days, vestibular neuronitis. Ménière’s becomes less likely in the absence of sensorineural hearing loss or tinnitus and more likely with a history of recurrent episodic vertigo, especially when associated with fluctuating hearing loss. BPPV becomes more likely with a history of positional triggers, absence of auditory symptoms and positive Dix-Hallpike. Finally, vestibular neuronitis becomes more likely with a history of upper respiratory infection and in the absence of auditory symptoms.

Imaging and special tests

In the case of acute vertigo, the clinical likelihood of a central cause will influence what special tests, if any, are ordered. While unlikely to present without additional neurologic symptoms, the clinical similarity of isolated inferior cerebellar stroke and vestibular neuritis may warrant further investigation. Computed tomography or MRI is indicated for patients presenting with any positive neurological signs or stroke risk factors to rule out cerebellar or brainstem involvement. If the cause is vestibular, audiometry is recommended to rule out causes like Ménière’s or acoustic neuroma. Dix-Hallpike, head thrust, and caloric test can all be useful bedside investigations to supplement history and physical exam, and these tests are often sufficient to make a clinical diagnosis.

Management and prognosis

Vestibular neuronitis is one of the most common oto-vestibular pathologies to present to the emergency room physician and otolaryngologist. While commonly thought to occur after or during a viral process, the majority of patients do not present with this history. The typical patient is middle-aged and presents with acute vertigo lasting hours to days. Viral/immunologic mechanisms have been proposed, but the pathophysiology remains largely unknown. Once diagnosed, the treatment of vestibular neuronitis is supportive and concerned with the alleviation of symptoms. Use of anti-emetics and vestibular suppressants are recommended for acute phase only, as prolonged use may interfere with the process of central compensation. Glucocorticoids may have some benefit if administered within three days of onset; the use of antivirals rarely improves outcomes. For most patients, acute symptoms will resolve within two days. Recurrence of vestibular neuronitis is rare, but some research suggests
that after an episode of vestibular neuronitis, patients may later have a higher risk of BPPV. Treatment of BPPV and Ménière’s disease are similarly focused on supportive treatment; however, further management may differ depending on severity. Progressive Ménière’s disease may warrant intratympanic gentamicin injection or surgical therapy if conservative measures and corticosteroids fail; hearing loss is often progressive. BPPV, though distressing, has a better prognosis and is usually self limited. It can be treated with the Epley manoeuvre, which can be performed in office by the specialist or general practitioner and is highly effective in inducing a remission.

References