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Review

Imaging of dizziness

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Dizziness is a frequent indication for neuroimaging within the outpatient and emergency setting with variable diagnostic yield. The majority of persistent, recurrent, and isolated dizziness can be managed clinically. However, it may be difficult to distinguish a benign peripheral aetiology from a central cause, particularly in the emergency setting. We review the relevant anatomy, differential diagnosis, and key imaging features of central and peripheral causes of dizziness, as well as the literature for the diagnostic yield in acute and outpatient settings.

Introduction

Dizziness is a common complaint[1] with a broad differential diagnosis. Descriptions are often vague and clinical findings may not be able to distinguish benign causes from those life-threatening causes that require immediate attention. The diagnostic approach is complex and, although a range of sophisticated clinical and ancillary tests have been evaluated for their diagnostic accuracy and cost effectiveness, there remains a weak evidence basis for the investigation of dizziness.[2,3] The radiology literature similarly gives little guidance or consensus concerning appropriate imaging strategies, or whether imaging is even required[4,5]; however, there is now increasing data evaluating the role of radiological studies in particular clinical scenarios.[4,6–13]

The term dizziness is non-specific and encompasses a range of symptomatic presentations such as vertigo (a false sense of motion and the most frequent complaint), lightheadedness, imbalance, and presyncope.[14] Imbalance is generally more indicative of neurological damage, whereas vertigo implies a dysfunction of the vestibular apparatus or its connections[15,16]; however, it may not be easy to clinically distinguish between any of these subtypes. Although some authors refer to the specific terms, particularly with reference to vertigo, it should be noted that any of them may be indicative of serious disease[14,17]; hence, this review will generally refer to the all-encompassing term "dizziness".[18,19] Dizziness accounts for approximately 5–10% of primary-care presentations,[16,20,21] affects 30% of the population over the age of 65 years,[22] and represents 4% of visits to the emergency department.[23] There are numerous potential aetiologies with 46 different diagnoses being presented in one series.[24] A range of cardiovascular, endocrine, drug-related, or psychiatric causes should be excluded as part of the initial assessment prior to consideration of imaging.

It is useful to consider peripheral (vestibular) or central (central nervous system) aetiologies and this distinction is particularly helpful for guiding imaging strategies. The clinical evaluation helps distinguish these entities based on timing, duration, aggravating factors, audiological, and neurological symptoms.[25] Specific neurological examination is targeted at detecting disruptions of the vestibulo-ocular connections and this finding has a yield of up to 95% sensitivity for the detection of a central lesion.[26]

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Unfortunately, less specialist clinical assessment, as is usually available in the emergency setting, is less accurate in separating peripheral from central dizziness.[27] This may be due to overlapping clinical features or because the pathological processes (e.g., ischaemia) involve both the vestibular apparatus and the brain.

This review will outline the key anatomy, relevant imaging methods, and the diagnostic considerations with their imaging findings. The imaging approaches to dizziness remain controversial, vary widely in clinical practice, and are influenced by local preferences[4]; however, we will explore the role and diagnostic yield of imaging based on an appraisal of the literature. Although it is appreciated that there may be some overlap and it is a somewhat artificial separation, it is felt pragmatic to discuss the imaging approach to persistent or recurrent dizziness in the outpatient setting, followed by the imaging approach to acute onset dizziness in the emergency setting.

Anatomy and imaging methods

The end organs of balance and equilibrium are the vestibule and semicircular canals, consisting of the outer osseous labyrinth and an inner membranous (or endolymphatic) labyrinth. There is perilymphatic fluid interposed between the two compartments. The inner endolymphatic structures are the utricle, the saccule, and the semicircular ducts. The endolymphatic sac and duct is located within the vestibular aqueduct and this also communicates with the saccule and utricle. The utricle and saccule or "static labyrinth" contains maculae (consisting of sensory hair cells that detect the position of otolithic crystals), which detect the position of the head relative to gravity. The semicircular canals or "kinetic labyrinth" contain ampullae (also with sensory hair cells), which respond to rotation and angular acceleration. These structures detect endolymphatic flow and innervate the vestibular nerves that maintain balance through their central connections. The vestibular nerves arise from a population of bipolar neurons and cell bodies, which reside in Scarpa's ganglion, and they run with the cochlear nerve (as cranial nerve eight or the vestibulocochlear nerve) to the vestibular nuclei in the brainstem (Fig 1). The arterial supply to the vestibular structures is via the labyrinthine branch of the anterior inferior cerebellar artery.[28] Both computed tomography (CT) and magnetic resonance imaging (MRI) have a role in the imaging of these structures. The spatial resolution of CT is ideal in view of the innate high contrast of the osseous component and it will demonstrate erosion, fracture, or deficiency as well as the misplacement of prostheses with respect to the bony labyrinth. MRI is required to image the fluid-containing structures of the perilymphatic and endolymphatic spaces in addition to the vestibular nerves. Highly T2-weighted gradient-echo [e.g., true fast imaging employing steady state precession (FISP)/dual excitation: CISS or FIESTA-C] or spin-echo [e.g., fast spin-echo/turbo spin-echo (FSE/TSE) with 90° flip-back pulse (DRIVE) or three dimensional (3D) TSE with variable flip angle (SPACE)] thin-section imaging is acquired to demonstrate both the labyrinth and the cranial nerves within the internal auditory meati and cerebellopontine angle cisterns. Additional gadolinium-enhanced T1-weighted imaging, which may also be acquired with volumetric sequences [e.g. using volumetric interpolated breath-hold examination (VIBE) or SPACE], will help assess for inflammation or neoplastic processes related to these structures.

The central vestibular pathways extend from the vestibular nuclei in the medulla to the rostral midbrain, oculomotor nuclei, cerebellum (flocculus, vermis, and nodulus), thalamus and cortex (temporal, parietal, and insular). These communications perform the processes required to compute head position and motion.[26] There are also descending projections that run to the upper cervical cord via the medial and lateral vestibulo-spinal tracts, which allow postural adjustments by the body. The radiologist should be aware that lesions disrupting any of these communications may result in symptoms including dizziness. Whole-brain imaging is, therefore, required for radiological evaluation. Although CT is frequently performed to assess for intracranial disease, such as haemorrhage or infarction in the emergency setting, MRI is otherwise the imaging of choice. Diffusion-weighted imaging (DWI) is routinely used for the assessment of acute ischaemic changes, whereas gadolinium helps characterize neoplastic and inflammatory lesions. Computed tomography angiography (CTA) or magnetic resonance angiography (MRA) may be used to demonstrate narrowing of the vertebrobasilar arterial system in patients with posterior circulation ischaemia.

Persistent and recurrent dizziness: the outpatient setting

These patients are referred from the outpatient setting, often from neurology, otology, or specialist balance clinics after a detailed clinical review, and usually with a variety of additional investigations, such as audiometry, cardiovascular, and vestibular testing (e.g., electronystagmography). The primary aetiology of dizziness in this setting is a peripheral disorder with a psychiatric diagnosis also being common; central causes account for only 5–11% of cases[19,29] and dangerous causes, such as cerebral infarction, are rare.

Peripheral

The most frequent vestibular disorders are benign paroxysmal positional vertigo (BPPV) or Meniere's disease.[16] There is little role for imaging when the clinical diagnoses have been established.[30]

BPPV is the most common vestibular disorder in adults, and it occurs due to displacement of the otoliths from their normal position. It is diagnosed by clinical manoeuvres (Dix–Hallpike and supine roll tests) and there is no radiological correlate. Imaging is reserved for a small subset of patients, when treatment has failed and there is diagnostic