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Review article

Recent advances in central acute vestibular syndrome of a vascular cause

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ABSTRACT

Acute vestibular syndrome (AVS) is characterized by acute onset of spontaneous prolonged vertigo (lasting days), spontaneous nystagmus, postural instability, and autonomic symptoms. Peripheral AVS commonly presents as vestibular neuritis, but may also include other disorders such as Meniere's disease. Vertigo in central AVS due to vertebrobasilar ischemic stroke is usually accompanied by other neurological dysfunction. However it can occur in isolation and mimicking peripheral AVS, particularly with cerebellar strokes. Recent large prospective studies have demonstrated that approximately 11% of patients with isolated cerebellar infarction presented with isolated vertigo mimicking peripheral AVS, and the bedside head impulse test is the most useful tool for differentiating central from peripheral AVS. Herein we review the keys to the diagnosis of central AVS of a vascular cause presenting with isolated vertigo or audiovestibular loss.

Contents

1. Introduction

Acute vestibular syndrome (AVS) is characterized by acute onset of spontaneous prolonged vertigo (lasting days), spontaneous nystagmus, postural instability, and autonomic symptoms [1,2]. AVS can be divided into peripheral (i.e., inner and vestibular nerve) and central (i.e., brainstem and cerebellum) causes. The peripheral causes of AVS included acute vestibular neuritis (VN), Meniere's disease, and migraine. Vertebrobasilar ischemic stroke (VBIS) can also cause isolated prolonged vertigo mimicking peripheral AVS [3–5]. Recent studies have shown that cerebellar infarction simulating peripheral AVS is more common than previously thought and the bedside head impulse test (HIT) is the most useful tool for differentiating central AVS from other more benign disorders involving the inner ear [4,5]. Clinically, it is important to differentiate central AVS of a vascular cause from

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peripheral AVS because therapeutic strategy and prognosis are different in the two conditions. Early recognition of the central AVS of a vascular cause may allow specific management. This review aims to highlight recent advances in central AVS of a vascular cause presenting with isolated vertigo or audiovestibular loss and to address their clinical significance.

2. Possible anatomical structures responsible for central AVS of a vascular cause

Vertigo is resulted from imbalance of tonic discharge of the vestibular systems arising from the inner ears on both sides. The origin of vertigo may be peripheral or central. When the vertigo occurs as a symptom of VBIS, it is usually associated with other neurological symptoms or signs [6]. Three possible structures responsible for central AVS of a vascular cause are the nodulus, root entry zone of the eighth nerve in the pontomedullary junction, and vestibular nucleus (Fig. 1). Theoretically, a small infarct localized to these structures can cause vertigo with no accompanying other neurological symptoms or signs since all of these structures receive afferent vestibular inputs from the inner ear (Fig. 2). Because none of these structures are known to be more sensitive to ischemia than other surrounding structures, the incidence of central isolated vertigo associated with ischemic stroke is low. Rarely, lesions involving the flocculus lobe or dorsal insular cortex can also cause isolated vertigo (Fig. 2) [7–9]. Vertigo due to a lesion involving the dorsal insular cortex is usually not associated with nystagmus and a flocculus lesion is commonly associated with other central signs with gaze-evoked nystagmus and asymmetrical oculomotor dysfunction [7,9]. Therefore, in such cases, clinicians may conclude that vertigo is caused by damage to the central vestibular structure.

3. Classification of central AVS

3.1. Cerebellar ischemic stroke

3.1.1. Frequency, pattern of involved vascular territory, and associated vestibular dysfunction

Vertigo is one of the commonest symptoms in patients with cerebellar stroke syndrome. Among cerebellar stroke syndrome, cerebellar ischemic stroke probably ranks first as central AVS of a vascular cause. A small retrospective study showed that as many as 25% of patients with vascular risk factors who presented to an emergency medical setting with isolated severe vertigo, nystagmus, and postural instability have a cerebellar infarction in the territory of the medial branch of the PICA (mPICA) [10]. A recent large prospective study on clinical findings of 240 patients with isolated cerebellar infarction also demonstrated similar results. In this study, about 11% (25/240) with isolated cerebellar infarction had isolated vertigo only and most (24/25: 96%) patients with isolated vertigo had an infarct in the territory of the mPICA including the nodulus [5]. Another more recent study using diffusion-weighted imaging found that 75% of patients with at least one vascular risk factor who presented with acute isolated vertigo had acute stroke, mostly involving the caudal cerebellum in the mPICA territory [4]. In PICA territory cerebellar infarction, the key structure responsible for vertigo is the nodulus. The nodulus is strongly connected to the ipsilateral vestibular nucleus and receives direct projections from the labyrinth [11,12]. Functionally, nodulovestibular Purkinje fibers have an inhibitory effect on the ipsilateral vestibular nucleus [11,12]. A predominant involvement of mPICA territory cerebellar infarction associated with central AVS may be explained in several ways. First, the mPICA usually supplies the nodulus, a part of the vestibulocerebellum [13]. Thus, infarction in the territory of the medial PICA can cause severe vertigo. Second, dysmetria, a major finding of the cerebellar lesion, may be minimal or absent after a cerebellar infarction in the territory of the mPICA if the size of an infarct is not large [5,14,15]. Third, gaze-evoked asymmetrical nystagmus, which commonly occurred in central vestibulopathy of cerebellar origin, is sometimes absent in the PICA territory cerebellar lesion [5,14–18]. Finally, hearing loss that is generally considered a peripheral sign commonly accompanies anterior inferior cerebellar artery (AICA) stroke, not PICA stroke [19]. Since the superior cerebellum supplied by the superior cerebellar artery (SCA) does not have significant vestibular connections, cerebellar infarction in the SCA territory rarely causes vertigo [20,21]. The vestibulo-ocular portion of the cerebellum is located primarily in the flocculonodular lobes, which are supplied by branches of the AICA and PICA. The low incidence of vertigo in SCA distribution may be a useful clinical distinction from PICA or AICA cerebellar infarction in patients with acute vertigo and limb ataxia [20,21].

In PICA territory cerebellar infarction, the direction of nystagmus and degree of postural instability were variable. The prominent cerebellar signs, particularly severe axial instability and direction changing gaze-evoked nystagmus (occurring in 71% and 54%, respectively, in the aforementioned series [5]), can help in the differential, but these findings are less reliable.

Similarly, perverted head shaking (mostly downbeating) and positional downbeating nystagmus as important signs of central vestibular dysfunctions are found in only half of the cases with cerebellar infarction [22]. In contrast, the vestibular dysfunction in some patients with PICA territory cerebellar infarction is similar to that in those with VN. For example, spontaneous unidirectional, ipsilesional nystagmus and mild postural instability with standing or walking independently could be seen in cases with PICA territory cerebellar infarction (occurring in 17% and 29%, respectively, in the aforementioned series [5]). The mechanism of spontaneous unidirectional, ipsilesional nystagmus may have

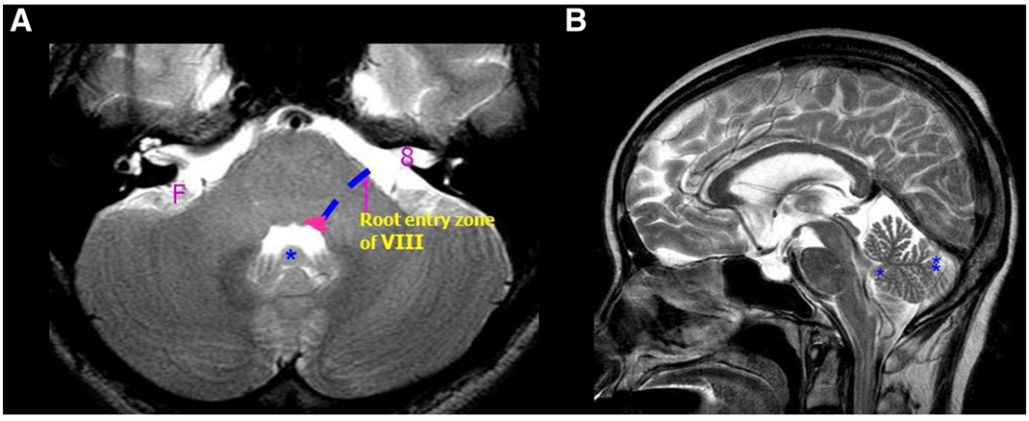

Fig. 1. Structures related to the central isolated vertigo syndrome. F = flocculus, 8 = vestibule-cochlear nerve, ⌐ = intraparenchymal portion of vestibular nerve (i.e., vestibular fascicle), ◄ = vestibular nucleus, * = nodulus, ** = oculomotor vermis.