Clinical and Therapeutic Aspects of Vertigo

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Abstract: Dizziness is a symptom frequently encountered in clinical practice, especially during emergency department visits and primary care visits. We can classify dizziness in one of four categories: vertigo, presyncope, disequilibrium and lightheadedness. Vertigo is the most common cause of dizziness and very often represents a diagnostic challenge for the clinician. Although appropriate clinical approach and history taking, the final diagnosis is unidentified in about 20 percent of the patients complaining of dizziness. The aim of this review is to provide an update on the main differential diagnoses of dizziness, with particular attention to the different otologic, vestibular and neurologic causes of vertigo, covering epidemiology, pathophysiology, clinical presentation, and therapy. We will discuss in detail the most common causes of vertigo, such as benign paroxysmal positional vertigo, labyrinthitis, Meniere's syndrome, vestibular neuritis, vestibular migraine and brainstem lesions. Special emphasis will be placed on the usefulness of diagnostic tests such as the Dix-Hallpike maneuver, the head impulse test and the electronystagmography. We will underline the need to perform a complete physical and neurological examination in order to correctly choose the appropriate diagnostic tests. Finally, we will review the different treatments currently available. It is extremely important to prescribe a proper treatment for dizziness, because this can dramatically improve the quality of life of these patients.

Keywords: Dizziness, vertigo, vestibular system, VIII cranial nerve, Meniere's syndrome.

INTRODUCTION

Dizziness is one of the most frequently encountered symptoms in clinical practice, especially among patients visited by neurologists, primary care physicians, otolaryngologists and during emergency department visits [1]. The 20-30% of people in the general population suffer from dizziness [2], however the term "dizziness" can refer to multiple sensations, such as vertigo, presyncope, disequilibrium and lightheadedness [3], each having different etiologies and requiring specific treatments. Vertigo is the most common type of dizziness and can be defined as a sensation of false movement (illusion or hallucination of movement), most frequently described as rotation, but sometimes as a sensation of tilt [4]. Signs and symptoms that associate with vertigo and dizziness depend primarily on their etiology. Nystagmus, nausea, vomiting, facial weakness, hearing loss and pain can help differentiate the cause of vertigo [5] and must be carefully searched during anamnesis and clinical evaluation. In this review we discuss the most common causes of vertigo, the diagnostic tests and the different treatments currently available.

THE VESTIBULAR SYSTEM

The vestibular system consists of a peripheral section and a central component. The peripheral section is made up of the membranous labyrinth of the inner ear and the vestibular nerve. The labyrinth contains the cochlea, the three semicircular canals (posterior, superior and lateral) and the otolithic organs (saccule and utricle). The three semicircular canals transduce rotational head movements, while the saccule and utricle respond to linear acceleration and gravity. The membranous labyrinth contains endolymph. The endolymph is produced in the endolymphatic sac and is in continuity between vestibular and cochlear systems. Each semicircular canal presents at its anterior end a dilatation called "ampulla", where sensory cells named "hair cells" are situated on a structure termed "crista". The stereocilia of the hair cells are embedded in a gelatinous mass called "cupula". In saccule and utricle, the hair cells can be found in the "macula" and their cilia are embedded in the "otolithic membrane", composed by a gelatinous layer with calcium carbonate crystals called statoconia or otoliths. The neural output from semicircular canals and otolithic organs are transmitted through the vestibular nerve which forms, together with the cochlear nerve, the eighth cranial nerve. Axons of the vestibular nerve synapse in the vestibular nuclei, localized in the pons and medulla. There are four main vestibular nuclei (superior, medial, lateral, and descendent) [6] that present reciprocal ipsilateral and contralateral connections and numerous connections with the cerebral cortex, the cerebellum, the spinal cord and other brainstem nuclei.

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Table 1: In this Table are Summarized the Most Common Causes of Vertigo

Peripheral	Central	Other causes of dizziness
Meniere's syndrome	Migrainous vertigo	Cardiac arrhythmias
Benign paroxysmal positional vertigo	Multiple sclerosis	Orthostatic hypotension
Vestibular neuronitis	Trauma	Severe anemia
Labyrinthitis	Vertebrobasilar stroke Vertebrobasilar insufficiency	Aortic stenosis
Vestibular schwannoma	Tumors	Exogenous intoxications
Perilymphatic fistula		Drugs and alcohol Psychogenic vertigo
Superior semicircular canal dehiscence syndrome		
Trauma		
Herpes zoster oticus (Ramsay Hunt syndrome)		

ETIOLOGY AND DIAGNOSTIC TESTS

Classification of vertigo can be based either on chronological criteria (acute, recurrent or chronic vertigo) or on topographical criteria (peripheral or central vertigo). Peripheral vertigos result from disorders of the inner ear or the eight cranial nerve, while central vertigos result from disorders of the central nervous system. The most common causes of vertigo are summarized in Table **1**.

Some clinical features may help the clinician in the differential diagnosis. Nystagmus represents the first clinical sign to be investigated. In peripheral vertigo, nystagmus is horizontal and rotational and diminishes when the patient focuses the gaze [7], unlike the central vertigo in which nystagmus is vertical, horizontal or rotational and does not fade when the patient focuses the gaze [8]. The duration of the attacks is also of paramount importance. Ménière's disease causes vertigo attacks lasting hours, while vertigo that develops in a period of hours and slowly resolves over a period of days or weeks is more probably due to vestibular neuronitis and labyrinthitis. The episodes of vestibular migraine and vertebrobasilar insufficiency typically last minutes. Uncompensated peripheral vestibular lesion and benign paroxysmal positional vertigo (BPPV) are associated with a duration of seconds and with head movements. The most useful diagnostic tests to correctly differentate the different forms of vertigo are Dix-Hallpike maneuver (Figure **1**), head impulse test (HIT) (Figure **2**) and electronystagmography. HIT detects unilateral or bilateral vestibular hypofunction through the vestibulo-ocular reflex response [9]. The negative HIT associated with a nystagmus that changes direction is very suggestive of central lesion. Electronystagmography is used to record nystagmus and is based on the corneal retinal potential measurement using electrodes that record the changing voltages that result from eye movements during different tests [10]. The standard electronystagmography evaluation consists of oculomotor evaluation, positional testing and caloric test. The electronystagmography is helpful in determining whether a vertigo is central or peripheral in origin. The Dix-Hallpike maneuver is useful for the diagnosis of BPPV [11], albeit it should be remembered that the sensitivity of this test for BPPV is 79% and the specificity is 75% [12].

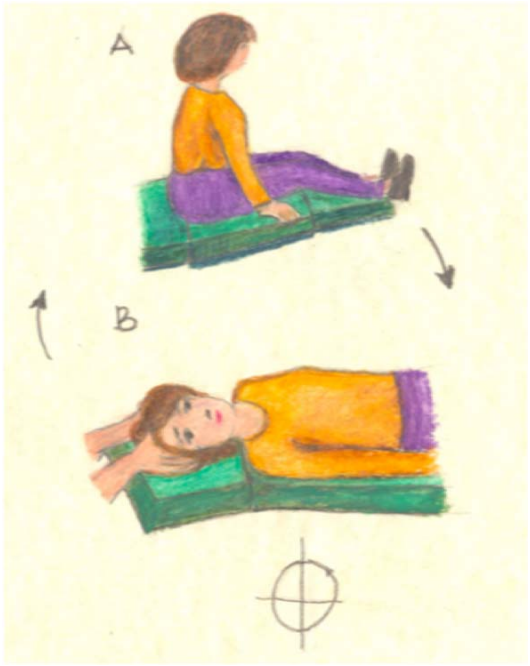

Figure 1: This figure shows the Dix-Hallpike test for benign paroxysmal positional vertigo (BPPV). Patient's position passes from sitting to supine, with head extended 20 degrees backward and turned 45 degrees laterally. When in supine position, patient's eyes are observed for 30 seconds looking for nystagmus. If the maneuver fails to evoke nystagmus, the patient is brought back to sitting position and, after 30 seconds, the maneuver is repeated, testing the other side.

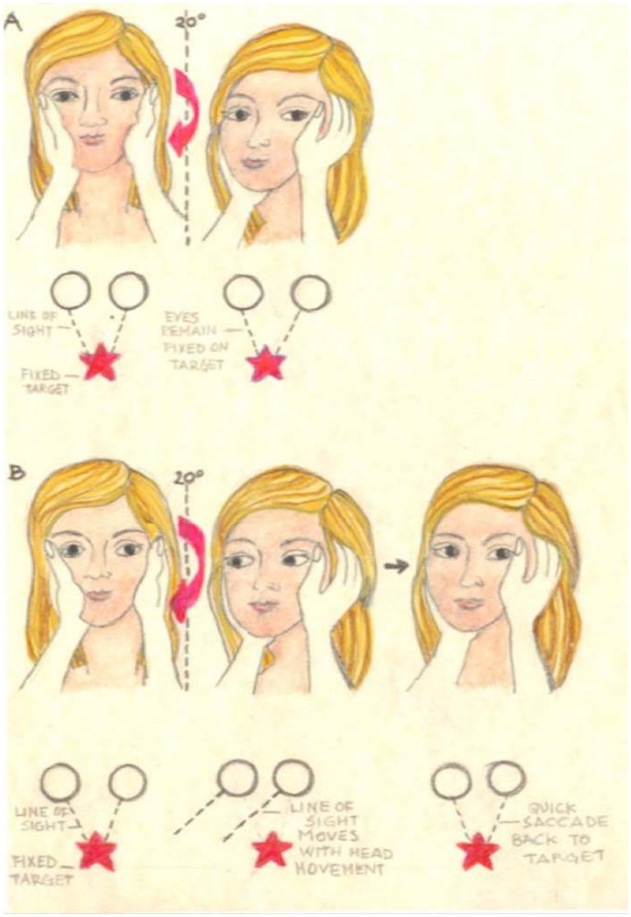

Figure 2: The head impulse test (HIT) is used to evaluate the vestibular function. It tests the vestibule-ocular reflex and can help to differentiate if the vertigo is peripheral or central in origin. With the patient sitting, the clinician instructs him to fix his gaze on the examiner's nose. The examiner holds the patient's head from the front and then rapidly turns the head to about 20° off the midline while watching the patient's eyes looking for any corrective movements. (**A**) Normally the eyes stay locked on the target (e.g. the nose of the examiner). (**B**) The test is considered positive when a corrective saccade is visible. It should be remarked that this test is usually positive in patients with vertigo due to a peripheral lesion, while it is negative if the lesion is central in origin. In the latter case this happens because the primary vestibule-ocular reflex pathway bypasses the cerebellum.

COMMON PERIPHERAL VESTIBULAR DISORDERS

Benign Paroxysmal Positional Vertigo

BPPV represents the most common cause of vertigo [13]. It affects patients in the fourth and fifth decades of life and women are more often affected than men [14]. It is assumed that BPPV is caused by the movement of free-floating small crystals of calcium carbonate, called "canaliths" localized in the semicircular canals (commonly the posterior semicircular canal) that create the sensation of vertigo through an asymmetrical activation of vestibular hair cells [15]. Tipically patients experience transient episodes of intense rotatory vertigo when they rapidly change their head position. These episodes last for seconds. The vertigo is not associated with tinnitus or hearing loss. It can be associated with nausea, but the episode of vertigo is too brief to evoke vomiting [16]. Attacks of BPPV are generally separated by remissions. The Dix-Hallpike manoeuvre is useful to confirm the diagnosis and to localise the affected side and should be performed to diagnose BPPV (level of evidence C) [17-19]. Epley or canalith repositioning manoeuvre (Figure **3**) is a safe and effective way to treat BPPV (level of evidence B) [20-23] and represents the first line of treatment. Vestibular rehabilitation is also an effective treatment for BPPV

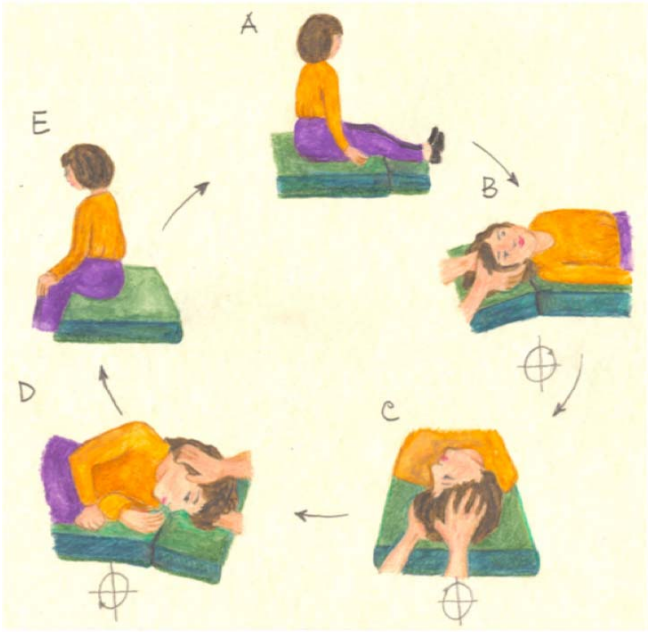

Figure 3: (**A**) The Epley manuever starts with the patient sitting upright with the legs extended and the head rotated 45 degrees towards the affected side. (**B**) The clinician tilts the patient backward to a horizontal position with the head in an about 30 degree neck extension with the affected ear facing the ground. The clinician observes the patient's eyes looking for the possible occurrence of "primary stage" nystagmus. The patient usually lies in this supine position for 1-2 minutes. (**C**) While maintaining the 30 degree neck extension, the clinician turns patient's head of 90 degrees to the other side. At the end of this step the unaffected ear faces the ground. The patient lies in this position for additional 1-2 minutes. (**D**) The clinician makes the patient roll on the shoulder, keeping patient's head and neck in a fixed position relative to the body. At the end of this step, the patient is looking downwards at a 45 degree angle. The clinician observes the patient's eyes again, looking for the possible occurrence of "secondary stage" nystagmus that should beat in the same direction as the primary stage nystagmus. The patient remains in this position for 1-2 minutes. (**E**) Finally, the patient slowly returns to a sitting position, while maintaining the 45 degree rotation of the head. The patient maintains the sitting position for 30 seconds. The Epley manuever may be repeated up to three times.

(level of evidence B). Patients that do not respond to canalith repositioning manoeuvres may be candidates for singular neurectomy or posterior semicircular canal occlusion [14].

Meniere's Disease

Meniere's syndrome is defined as a condition of endolymphatic hydrops of known etiology, while the term Meniere's disease (MD) refers to an idiopathic condition of endolymphatic hydrops. The worldwide incidence of MD is about 12 out of every 1.000 people [24] and it is most common between fourth and sixth decades, although it may affect also younger people [25]. MD is characterized by recurrent attacks of vertigo often with nausea and vomiting, hearing loss, tinnitus, and aural fullness. Such episodes may occur in clusters and typically last minutes to hours. Symptoms of MD are variable and patients may refer a predominance of either cochlear (hearing loss and tinnitus) or vestibular (vertigo) disturbances. MD course is progressive but fluctuating and vertigo usually resolves in the interictal phases, while hearing progressively deteriorates [14]. Initially, the hearing loss typically involves the low frequencies and generally becomes worse with each attack. Over time, the hearing loss progresses to involve the higher frequencies and finally leads to a severe hypoacusia up to bilateral anacusia. The diagnosis of MD is based on the clinical history and instrumental investigations. Audiometry shows a sensorineural hearing loss that is usually unilateral, sometimes bilateral. In the long-lasting forms of MD a transmission component of hearing loss can be detected. In the early phase of the disease, during the acute attack a spontaneous nystagmus may be present, while in the interictal phases there are no nystagmus or static and dynamic balance disorders. In the later phases of the disease, it is possibile to elicite a latent nystagmus, and to detect static and dynamic balance disorders at posturography and vestibular hyporeflexia at caloric test.

Treatments of the acute attack include vestibular sedatives and anti-emetic drugs. Prophylaxis strategies most commonly used are low salt diet (less than 1 to 2 g of sodium per day), diuretics (e.g. hydrochlorothiazide/triamterene) [26] and vasodilators (betahistine) [27], although there are no large-scale randomized controlled trials to support these therapies [28]. Surgical treatment is considered when the patient fails to respond to medical therapies. Intra-tympanic gentamycin [29], endolymphatic sac surgery, vestibular neurectomy and labyrinthectomy are all possible options.

Vestibular Neuronitis

Vestibular neuronitis (VN) is a common condition characterized by the inflammation of the vestibular nerve. Although a viral infection is believed to be the most likely cause of the disease, no specific virus has been identified except in the case of Ramsay-Hunt syndrome, in which the pathogen is Varicella Zoster Virus [30]. The association of VN with a recent or concurrent upper respiratory tract infection is well known [11]. Patients affected by VN experience the acute onset of initially severe vertigo, nausea and vomiting, in the absence of hearing loss or tinnitus. The vertigo then gradually diminishes over a period of days or weeks [31]. During the acute phase, the patient may not be able to stand upright. Nystagmus is generally present and can be horizontal or rotatory and always directed away from the side of the inflamed vestibular nerve. It should be remembered that sometimes the inflammatory process can be bilateral, with the involvement of both vestibular nerves. A minority of patients may experience recurrences with vertiginous episodes following rapid head movement for years after onset [32]. Fitzgerald-Hallpike caloric test may reveal caloric weakness on the affected side, while audiometric evaluation is normal. In Ramsay-Hunt syndrome, the patient may experience also peripheral facial nerve palsy, ipsilateral sensorineural hearing loss and cutaneous and mucosal rashes (herpes facialis and herpes auricularis). The specific treatment of the patients affected by VN is controversial, largely because of the uncertainty regarding the etiology of the disease. Methylprednisolone has been associated with a significant improvement of the vestibular function [33], whereas no added benefit has been demonstrated with the use of antivirals. Despite their widespread use in Ramsay Hunt syndrome, if antiviral agents have a beneficial effect on outcomes in this condition is still a matter of debate.

Labyrinthitis

The term “labyrinthitis” means an inflammation of the membranous labyrinth, affecting both the vestibular and cochlear end organs. Like vestibular neuronitis, the onset of labyrinthitis is often preceded by an upper respiratory tract infection. Viral etiology is much more common in adults, while bacterial labyrinthitis is less frequent. Bacterial labyrinthitis [34] resulting from meningitic infection is more common in children less than 2 years of age and tipically cause bilateral symptoms, while otitis media [35], head injury and cholesteatoma generally cause unilateral symptoms

and are more common in adults. Fever and altered inflammatory markers are suggestive of infection. Vertigo presents suddenly and is associated with hearing loss. Audiometric evaluation reveals a sensorineural hearing loss or mixed hearing loss, if middle ear is involved. Anti-emetic drugs may be of some utility in the acute phase, but antibiotic therapy should be promptly administered and should aim to eradicate the underlying infection and to ensure supportive care. In cases of mastoiditis, surgical drainage of middle ear effusions may be necessary.

Vestibular Schwannoma

Vestibular schwannoma (VS) is a benign, usually slow-growing tumor that originates from the Schwann cells lining the vestibular portion of the eight cranial nerve. It is unilateral in about 95% of the cases but bilateral cases are possible and are associated with type II neurofibromatosis. In a small percentage of cases, VS arises from the cochlear branch [36]. Although generally considered benign neoplasms with low lethality, if VSs are associated with type II neurofibromatosis, they tend to be more aggressive. Clinical presentation of VSs is characterized by signs of both cochlear and vestibular involvement, due to compression of the eight cranial nerve. Patients may experience asymmetric hearing loss and tinnitus, vertigo and disequilibrium. The peculiar position of the tumor at the level of the cerebellopontine angle can result in facial or trigeminal nerve compression, causing facial weakness and numbness, respectively. In advanced stages the VS can compress also the brainstem and cerebellum. This event can be life-threatening, causing death by compression of respiratory centers of the brainstem [37]. Head MRI scan is very important for the diagnosis and audiogram is also useful to evaluate the hearing loss. After the radiological evaluation, a balance has to be made between surgical options, radiation therapy, and clinical and radiological observation with serial MRI. The size of the tumor, the symptoms reported by the patient at the time of clinical assessment, comorbidities and the age of the patient represent the main factors to be considered for therapeutic decision. Detailed descriptions of the various treatments can be found in more specific reviews [38-40].

Other Peripheral Causes of Vertigo

Among the other peripheral causes of vertigo we cannot forget perilymphatic fistula (PLF). PLF consists in an abnormal communication between the perilymphatic space and the middle ear. The most common cause of PLF is head trauma. Other causes are stapedectomy, explosive blast, barotrauma, and physical exertion. Patients generally experience episodic attacks of vertigo associated with fluctuating hearing loss, but symptoms are widely variable. Fistula test is useful and is positive if nystagmus is elicited after applying a positive pressure to the ear through pneumatic otoscopy. If conservative treatment with laxatives and bed rest is not efficacious then surgical exploration is needed and the fistula is repaired.

Another peripheral cause of vertigo is the superior semicircular canal dehiscence syndrome, first described in 1998 by Minor *et al*. [41]. This syndrome is caused by a dehiscence of the part of the temporal bone overlying the superior semicircular canal. This may act as a third window allowing transfer of sound and pressure into the vestibular system. Clinical presentation consists in hyperacusis to bone-conducted sounds and conductive hearing loss with normal acoustic reflexes [42], vertigo and vertical-torsional eye movements induced by loud sounds or factors that change middle ear or intracranial pressure [43], and Tullio phenomenon that refers to sound-induced vertigo, nystagmus and oscillopsia [44]. In fact these patients present rotational and vertical nystagmus after intense sound stimuli. Conductive hearing loss can be documented with tonal audiometry and the diagnosis is confirmed with high resolution temporal bone CT scan. Surgery represents the treatment of first choice and the two most used techniques are the total plugging of the superior canal and the reconstruction of the temporal bone with fibrin glue.

Exostoses and osteomas are benign bony lesions of the internal auditory canal that can significantly narrow the lumen compressing the seventh and eighth cranial nerves causing symptoms of acoustic-facial bundle involvement [45]. These rare tumors should be considered in the differential diagnosis of vestibular disorders. Finally paraneoplastic neurological syndromes should be considered in the diagnostic workup of patients with subacute hearing loss and vertigo, since the eighth cranial nerve may be involved, particulary in anti-Hu paraneoplastic syndrome [46].

COMMON CENTRAL VESTIBULAR DISORDERS

Migrainous Vertigo

Migrainous vertigo is an increasingly recognized cause of episodic vertigo [47] and represents the most common cause of central vertigo. The diagnosis of

definite migrainous vertigo is based on the following criteria [48]: (1) episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusionary self or object motion, positional vertigo, head motion intolerance); (2) migraine according to the International Headache Society criteria [49]; (3) at least one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras; and (4) other causes ruled out by appropriate investigations. Patients with migrainous vertigo usually refer spontaneous or positional vertigo lasting hours to days. Typically the patient with migrainous vertigo reports a recent increase in his/her headache frequency and presents headache and vertigo not necessarily at the same time. Moreover, the vertigo may present atypical characteristics of migrainous aura: it may last from seconds up to 72 hours rather than the usual 5-60 minutes and it may occur before, during or after the cefalalgic phase. Migrainous vertigo is a diagnosis of exclusion. In such cases a careful anamnesis may reveal a personal or family history of migraine, trigger elements (for example, foods or alcohol) and other associated aura features. Tone audiograms exclude a sensory neural hearing loss. Pathological spontaneous and positional nystagmus is present in 70% of patients during acute migrainous vertigo [47]. Patients with headache and vertigo of new onset have to be admitted because cerebrovascular events can mimic migrainous vertigo and so acute neuroimaging may be required on first presentation. The treatment of migrainous vertigo is with standard antimigraine prophylactic agents. Triptans are useful in the acute attacks, but they are more efficient if taken early after the onset of symptoms. Nonsteroidal anti-inflammatory drugs and analgesics remain useful for acute treatment [50].

Vertebrobasilar Stroke

The differentiation between peripheral vertigo and central vertigo is mandatory in the case of a verebrobasilar stroke as patients with stroke may require urgent intervention. Vertigo as a manifestation of a stroke is not an infrequent symptom [51]. In most cases, vertigo of cerebrovascular origin presents in association with other neurological symptoms or signs, such as gait instability, limb ataxia, dysarthria, hemiparesis, deafness, loss of consciousness and dysphagia, but sometimes vertigo may be the sole manifestation of a such potentially serious condition as cerebellar infarction or impending basilar occlusion [52].

In particular, isolated vertigo can occur in posterior inferior cerebellar artery territory infarction [51], in the stenosis of the caudal or middle portion of the basilar artery (presumably close to the anterior inferior cerebellar artery origin) or in the case of a widespread slow vertebrobasilar flow on magnetic resonance angiography [53, 54].

There are some “red flags” that suggest a central origin of the vertigo (Table **2**).

Table 2: Here are Summarized the “Red Flags” that Suggest a Central Origin of the Vertigo

“Red flags” for central vertigo
Any central neurological symptoms or signs associated with vertigo
New type of headache (especially occipital)
Acute onset of deafness
Vertical nystagmus

Immediate brain imaging is indicated in suspected verebrobasilar stroke (Figure **4**).

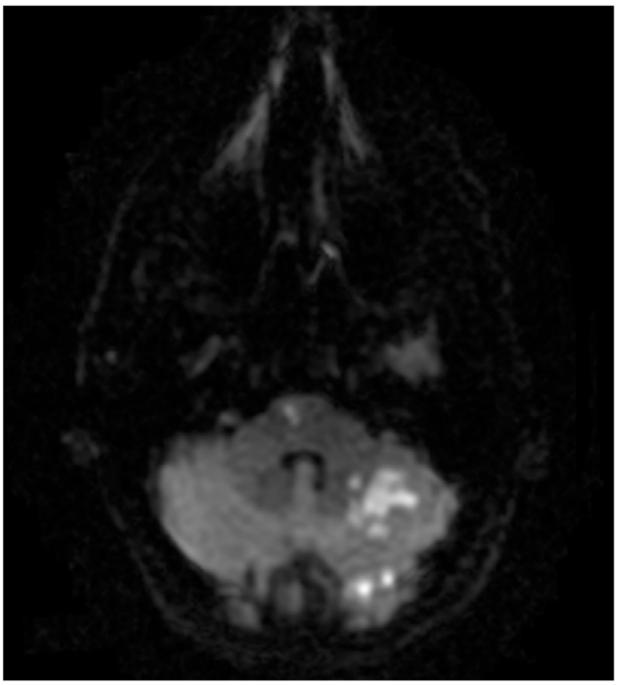

Figure 4: Diffusion-weighted magnetic resonance imaging showing cerebellar infarction in a patient with vertigo.

Vertebrobasilar Insufficiency

The term vertebrobasilar insufficiency (VBI) denotes a condition of recurrent symptomatic ischemia in regions irrigated by the posterior circulation [55] Common symptoms of VBI are visual disturbances, including hallucinations [56], transient crossed motor or

sensory deficits (eg, right facial and left hemibody numbness) [57], hemiataxia, diplopia, Horner's syndrome, vertigo and hemiparesis, due to brainstem dysfunction.

Vertigo is the most common manifestation of brainstem ischemia. It is usually associated with nausea, vomiting and nystagmus [58], but can be also the sole manifestation or the prominent symptom of VBI. In such cases, VBI may mimic peripheral vestibular disorders such as BPPV, labyrinthitis and vestibular neuritis. It is mandatory to distinguish VBI due to external compression of the vertebral artery from VBI due to intrinsic vascular disease (atherosclerosis or dissection) in order to promptly correctly treat the patient.

Multiple Sclerosis

Multiple sclerosis represents an uncommon cause of acute vestibular syndrome. Demyelinating lesions may be localized in the intra-pontine eighth nerve fascicle, medulla, inferior, middle and superior cerebellar peduncles, posterior pontine tegmentum and midbrain. Vertigo can be the first manifestation of demyelinating diseases and is associated with central oculomotor signs [59]. Brain and spinal cord MRI is useful to detect the presence of active demyelinating lesions showing contrast enhancement. In such cases, intravenous therapy with corticosteroids is mandatory and these patients should be referred to a neurologist for definitive diagnosis, treatment and follow up.

CONCLUSION

Given the number of different causes, the diagnosis and treatment of vertigo is challenging because of the large number of potential underlying conditions. It is extremely important to perform a complete physical and neurological examination in order to determine whether the origin of the vertigo is central or peripheral and correctly choose the appropriate diagnostic tests. Making a correct diagnosis is the key step to prescribe the proper treatment which can dramatically improve the quality of life of these patients.

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