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DIFFERENTIAL DIAGNOSIS OF VERTIGO

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Summary

Vertigo is the illusion of motion, usually rotational motion. Vertigo is among the most common symptoms causing patients to visit a physician, and as patients become older the incidence of vertigo increases. Vertigo can be caused by an inner ear disturbance – peripheral vertigo, by a central disturbance – central vertigo, by systemic diseases, or it can be psychogenic. The most common causes of vertigo are benign paroxysmal positional vertigo, acute vestibular neuronitis, Ménière's disease, migraine, anxiety disorders, vertebrobasilar ischemia and tumors of the pontocerebellar angle. It is important to distinct between peripheral and central vertigo in order to guide management decisions. In this article a differential diagnosis of vertigo is presented.

Key words: vertigo, peripheral vertigo, central vertigo, benign paroxysmal positional vertigo, acute vestibular neuronitis, Ménière's disease, migraine, vertebrobasilar ischemia, tumor of pontocerebellar angle

INTRODUCTION

Body position in space is controlled by the ocular, vestibular and somatosensory systems. The somatosensory system provides information from the skin, muscles and joints, the most important being the proprioceptive system located in the neck muscles and joints. Information from these three systems is processed in the brain stem, and,

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finally, integrated into the cortical perception system enabling postural reflexes that maintain the body position in space and conscious perception of spatial orientation [1].

A mismatch of this sensory information causes vertigo. Vertigo is defined as a hallucination of movement or erroneous perception of self or object motion. It is usually an unpleasant sensation due to the distortion of the static gravitational orientation perceived by the cortical spatial perceptual system, associated with difficulties in balance and gait. This erroneous perception of motion of person or environment may be linear or angular (rotatory). Central compensatory mechanisms enable deficiencies in one area to be overcome by other intact sensory systems [1,2].

Vertigo is a symptom that is perceived at higher cortical levels. Vertigo may be due to excessive physiological stimulation or pathological dysfunction. Symptoms that most often accompany vertigo include nausea, vomiting, nystagmus, and imbalance in standing and walking. Patients with vertigo may also complain of dizziness, lightheadedness, unsteadiness, imbalance, spinning, floating, and swaying. Gait imbalance or ataxia results from inappropriate or abnormal signals from the vestibulospinal system. Nausea and vomiting may occur from the activation of the chemoreceptor trigger zone in the medullar vomiting center. Nystagmus may be observed as a result of the dysfunction of the vestibulo-ocular system [1,2,3].

Vertigo is among the most common symptoms causing patients to visit a physician, almost as common as back pain and headache. The overall incidence of vertigo is 20-30% [4], reaching 50% in older patients and it is the most frequent symptom in patients older than 75 years [5]. Vertigo was the cause for 2% of consultations in general practice in the United Kingdom [6].

Vertigo can be caused by an inner ear disturbance – peripheral vertigo, by a central (brain) disturbance – central vertigo, by systemic diseases, or it can be psychogenic. Most authors take vestibular nuclei in the brainstem as the point differentiating peripheral (before the vestibular nuclei) and central vertigo (after the vestibular nuclei). As a result, central compensatory mechanisms, symptoms of peripheral labyrinth dysfunction, will eventually recover. Symptoms of central nervous dysfunction, although usually milder, tend to persist over time [1,2,7].

The most common causes of peripheral vertigo include benign paroxysmal positional vertigo (BPPV), vestibular neuronitis and Ménière's disease. The most common cause of central dizziness is migraine, frequently referred to as vestibular migraine or migraine-associated dizziness, while other central causes include vertebrobasilar insufficiency, cerebellar and brainstem lesions, acoustic tumors, and demyelination [2, 9].

Vertigo can be induced by physiological and pathological causes. In physiological vertigo the sense of disequilibrium is due to the physiological excess of visual, vestibular, or somatosensory signals which cannot be compensated by other systems. In path-



ological vertigo there is an abnormal sensory signal (from the sensors) or abnormal signal processing (by the central nervous system) [10,11].

Physiological vertigo

Physiological vertigo occurs in normal individuals when the brain is confronted with a mismatch among the three stabilizing sensory systems when the vestibular system is subjected to unfamiliar head movements to which it is un-adapted, such as in seasickness; unusual head/neck positions, like in extreme head extension (e.g. painting a ceiling); or following a spin. The intersensory mismatch also explains carsickness, height vertigo, and the visual vertigo most commonly experienced during motion picture chase scenes where the visual sensation of environmental movement is unaccompanied by concomitant vestibular and somatosensory movement information, or when inadequate spectacles are worn. Space sickness, a frequent transient effect of active head movement in the weightless zero-gravity environment, is another example of physiological vertigo [1,3,9,11,12].

Pathological vertigo

Pathological vertigo results from lesions of the visual, somatosensory, or vestibular systems. Visual pathological vertigo occurs due to a sudden onset of an extra-ocular muscle paresis usually accompanied with diplopia. Somatosensory vertigo, rare in isolation, is usually due to a peripheral neuropathy or myelopathy especially of dorsal columns that reduces the sensory input necessary for central compensation when there is a dysfunction of the vestibular or visual systems. In the past the most often cause was tabes dorsalis [2,3,10].

However, the most common cause of pathological vertigo is the vestibular dysfunction. Pathological vestibular vertigo can be due to either the peripheral labyrinth dysfunction, systemic derangement (such as metabolic, endocrine, or circulatory abnormalities), or central vestibular dysfunction. This type of vertigo is frequently accompanied by nausea, nystagmus, postural unsteadiness, and gait ataxia. Psychogenic vertigo results from hyperventilation in a patient with a known psychiatric disease who can complain of severe vertigo without the associated nystagmus or other physical findings. Severely incapacitating vertigo may be seen in anxiety attacks [2,5,9].

Sudden onset and vivid memory of vertiginous episodes are often due to an inner-ear disease, especially if a hearing loss, ear pressure, or tinnitus are also present. Gradual and ill-defined symptoms are most common in central nervous system (CNS), cardiac, and systemic diseases. The time course of vertigo is also important. Episodic true vertigo that lasts for seconds and is associated with head or body position changes is probably due to BPPV. Vertigo that lasts for hours or days is probably caused by Ménière's disease



or vestibular neuronitis. Patients with peripheral vertigo can usually ambulate during episodes and are consciously aware of their environment [3,5,7,8,13].

A sudden onset vertigo that lasts for minutes can be due to a brain or vascular disease, especially if cerebrovascular risk factors are present. Central vertigo secondary to brainstem or cerebellar ischemia is often associated with other brainstem characteristics, including diplopia, autonomic symptoms, nausea, dysarthria, dysphagia, or focal weakness. Patients with cerebellar disease are frequently unable to ambulate during acute episodes of vertigo. Dysdiadochokinesis and gait ataxia during episodes are more likely due to cerebellar diseases, especially in the elderly population. Sensory and motor symptoms and signs are usually associated with CNS diseases [10,11,13,14,15].

History

Patients should be thoroughly asked about the characteristics and pattern of the vertiginous sensation, the onset and duration of vertigo, factors intensifying vertigo, such as positional changes, or whether vertigo is worse with the eyes open or closed. Associated auditory symptoms such as tinnitus, ear fullness, pain, or hearing loss usually indicate peripheral vestibular dysfunction. Signs of central neurological dysfunction include diplopia, ataxia, dysphagia, dysphonia, or sensory and motor complaints. The history should include a review of systems (especially head trauma and/or ear diseases) and screening for anxiety and/or depression. A history of prescription medicines, over-the-counter medications, herbal medicines, and data about smoking, alcohol and illicit drugs intake can help to identify pharmacologically induced syndromes. A history of headaches, especially migraine headaches, can be associated with migraine-related dizziness. A previous viral illness, cold sores, or sensory changes in the cervical C2-C3 or trigeminal distributions usually indicate vestibular neuronitis or recurrent episodes of Ménière's disease [1,2,3,5].

Causes of vertigo

The most common systemic causes of vertigo include cardiac diseases, arterial hypertension and hypotension, hematological diseases (e.g. anemia, leukemia, lymphomas, polycythemia), hypoglycemia, hypoadrenalism, Cogan's syndrome (interstitial keratitis with vestibular symptoms) and cervical vertigo.

Psychogenic vertigo results most often from hyperventilation in a patient with a known psychiatric disease. A patient with psychogenic vertigo may have a subjective complaint of severe vertigo without the associated nystagmus or other physical findings. Severely incapacitating vertigo may be seen in anxiety attacks or in severe height vertigo (acrophobia). The treatment of psychogenic vertigo would be based on the underlying psychiatric diagnosis. Psychotherapy and desensitization procedures are of-



ten useful. A diagnosis of psychogenic vertigo presumes that no physical findings substantiate an organic cause for the vertigo symptoms [18].

The most common causes of peripheral vertigo

One of the most common peripheral vestibular syndromes is benign paroxysmal positional vertigo (BPPV), which may occur at any age. The characteristic history includes brief episodes of positionally induced vertigo, particularly in rapid changes in position such as getting out of bed. True vertigo or rotational sensation usually lasts less than one minute; however, a nonspecific dizziness, often described as a swimming sensation or disequilibrium, may last hours to days. BPPV has often been described as “self-limiting” because symptoms often subside or disappear within six months of the onset. Although BPPV usually remits spontaneously, one third of patients have recurrent symptoms for more than one year [19,20].

Acute unilateral labyrinth dysfunction (vestibular neuritis or neuronitis) manifests as an acute onset of severe vertigo with an associated positional imbalance, nausea, and nystagmus. This syndrome is different from benign paroxysmal vertigo because it has a much more prolonged course, is usually more severe, and is not positionally induced. Vestibular neuronitis often has viral etiology. In vestibular neuronitis, due to the reduced signal from the affected side, the nystagmus fast phase is directed away from the affected side. Three to five days after the onset of acute vertigo the patient will probably have spontaneous resolution of nausea and will be able to partially suppress nystagmus by fixation. Generally, within two to three weeks the vertigo ceases [21,22].

Ménière’s disease (endolymphatic hydrops) is a common cause of recurrent vertigo and auditory symptoms. Ménière’s disease is characterized by a fluctuating hearing loss in the low frequencies, a sensation of ear fullness or pressure and tinnitus, and prolonged vertigo reaching its maximum over minutes and resolving over hours with an associated postural imbalance and nausea. There is often a low tolerance for loud noises. During the vertigo attack, which usually lasts 30 to 60 minutes, a characteristic nystagmus is seen, with the fast phase away from the affected ear [23].

Toxic substances known to cause vertigo and auditory symptoms include alcohol, heavy metals and drugs. Aminoglycoside antibiotics, such as streptomycin and gentamicin, are known vestibular toxins, while neomycin and kanamycin are ototoxic. Other vestibulotoxic and ototoxic drugs include acetylsalicylic acid intoxication, chloroquine, furosemide, quinidine and quinine [24].

Central vertigo

Central causes of vertigo are less common than peripheral. Causes of vestibular vertigo include migraine, cerebrovascular diseases of the posterior cerebral circulation



including transient ischemia attack (TIA) (vertebrobasilar insufficiency), epilepsy, demyelinating disease of the posterior fossa, congenital malformations such as Arnold-Chiari malformation, subdural hematoma, fractures, cysts, arachnoiditis, syringobulbia, platibasia, neoplastic diseases, degenerative diseases of the posterior fossa, infectious diseases, toxic lesions, lesions of the temporal lobe, and supratentorial lesions compressing the brainstem [1,2,14,15].

Lesions of the vestibular nuclei and the vestibular portion of the cerebellum may cause vertigo, nystagmus, disequilibrium, and nausea. There are usually other signs of central nervous system dysfunction. Symptoms result from the involvement of the brain stem structures responsible for eye movement, speech, sensation of the face, extremities, and trunk, and motor control of the facial muscles and extremities. Presence of other neurological signs helps distinguish central from peripheral vertigo. Central vertigo tends to be less severe with fewer autonomic symptoms such as nausea and vomiting. It tends to persist over longer periods of time and occur in less sudden or severe attacks, except in the case of migraine or vascular disease [1,2,15,25].

Due to the dysfunction of brain stem compensating structures in central vertigo syndromes, vertigo, as well as nystagmus, may persist over considerable periods of time. Central nystagmus looks more severe than the patient's corresponding symptoms of vertigo or nausea. Postural changes tend to stimulate peripheral vertigo more than the central one. Peripheral vertigo tends to be reduced with fixation with the eyes open. Central vertigo tends to be worse with the eyes open, because of the conflict of visual and vestibular information. With the eyes closed, visual information is reduced, which reduces the visual vestibular conflict and reduces the sense of vertigo. Peripheral vertigo tends to fatigue with repeated head movements because of the intact brainstem compensation mechanisms. In central vertigo the vertigo does not fatigue or habituate with repeated movements, however it may vary on a day to day basis [2,25,26,27].

Cerebrovascular diseases

Vertebrobasilar insufficiency (TIA of the posterior cerebral circulation) denotes reversible episodes of focal ischemic neurological deficit that most often last 2-15 minutes causing transient neurological symptoms. Vertebral-artery disease can cause transient attacks of vertigo that are usually accompanied by other brain-stem or cerebellar symptoms [25].

The most common causes of vertebrobasilar ischemia are embolism, large-artery atherosclerosis causing arterial stenosis and occlusion, penetrating small-artery disease, artery dissection and subclavian steal syndrome (narrowing of the subclavian artery proximally to the vertebral artery origin when blood flows around through the left and right vertebral arteries, and posterior parts of the brain receive insufficient blood supply



causing neurological symptoms). Stenosis and occlusion most often occur at or near the origin of the vertebral artery [26,27].

Posterior-circulation ischemia rarely causes only one symptom but rather produces a collection of symptoms and signs. Symptoms and signs depend on the affected part of the brain: brain stem (medulla, pons, midbrain), cerebellum, and posterior parts of brain [26].

Dizziness, vertigo, headache, vomiting, double vision, loss and blurring of vision, ataxia, numbness and/or weakness, gait and limb ataxia, oculomotor palsies, and oropharyngeal dysfunction are frequent symptoms in patients with vertebrobasilar artery disease [25,26,27].

TIA can occur after a patient has been standing or in situations that reduce blood pressure or blood flow. These symptoms are related to ischemia of vestibulocerebellar structures in the medulla and cerebellum, most often consisting of dizziness, difficulty focusing visually, vertigo, loss of balance, and spells of decreased vision and ataxia [25, 26,27].

Patients with cerebellar infarcts often report dizziness, occasionally in conjunction with frank vertigo, blurred vision, difficulty walking, and vomiting. They often veer to one side and cannot sit upright or maintain an erect posture without a support. Patients may have hypotonia of the arm on the side of the infarct.

Ischemic infarcts can involve one posterior cerebral artery, which most often leads to a hemianopia of the contralateral visual field. Hemisensory symptoms may be present on the same side of the body and face as the hemianopia. Difficulty reading and naming colors often accompanies large infarcts of the left posterior cerebral artery, whereas neglect of the left visual field and disorientation to place may accompany infarcts of the right posterior cerebral artery [25,26,27].

Stenosis and occlusion of the basilar artery usually cause bilateral symptoms or crossed findings: ipsilateral symptoms of cranial nerves and contralateral symptoms of the trunk and limbs. Embolic infarction of the rostral midbrain and thalamus leads to a top-of-the-basilar syndrome characterized by somnolence and sometimes stupor, inability to make new memories, small, poorly reactive pupils and defective vertical gaze, and when severe, can cause the locked-in syndrome [25,27,28].

Wallenberg, or lateral medullary, syndrome occlusion of the vertebral or posterior cerebellar artery causes vertigo, nausea, vomiting, facial pain, ataxia, nystagmus, diplopiae, ipsilateral decreased pain and temperature in face, Horner's syndrome, limb ataxia, laryngeal and pharyngeal paralysis causing hoarseness, dysphagia and contralateral decreased pain and temperature sensations in the trunk and limbs [1,2,3,25].

Occlusion of the superior cerebellar artery causes vertigo, ipsilateral deafness, facial paresis and ipsilateral ataxia. Occlusion of the labyrinth artery causes infarction of the labyrinth with vertigo, deafness and nystagmus. [1,2,25].



Vertebral artery dissection

Vertebral artery dissection may be caused by a trauma of the cervical spine such as whiplash injury, fierce rotational movements of the head, manipulative therapy of the neck, hyperextension of the neck, degenerative spondylotic changes of the cervical spine, hereditary connective tissue disorders and genetic disorders, migraine, high serum homocysteine level, infection, and use of oral contraceptives. The vertebral artery is most mobile and thus most vulnerable to mechanical injury at C1 to C2 as it leaves the transverse foramen of the axis vertebra and suddenly turns to enter the intracranial cavity. Women are 2.5 times more frequently affected by extracranial vertebral dissections. Intracranial vertebral artery dissections are more common in men [25,28].

The cardinal symptom in patients with a vertebral artery dissection is pain, most often in the posterior part of the neck or occiput, spreading into the shoulder. Diffuse, mostly occipital, headache, dizziness, or diplopia also occur. Intracranial vertebral artery dissections cause medullary, cerebellar, and pontine ischemia and can cause subarachnoidal hemorrhage [28,29,30].

Tumors of the pontocerebellar angle

Tumors of the pontocerebellar angle are most often benign. In younger patients the most frequent is acoustic neuroma, while in older patients meningioma is more common.

The earliest symptoms of tumors of the pontocerebellar angle include unilateral sensorineural hearing loss/deafness, disturbed sense of balance and altered gait, vertigo with associated nausea and vomiting, and pressure in the ear, all of which can be attributed to the disruption of normal vestibulocochlear nerve function. Additionally, most patients reported tinnitus (most often a unilateral high-pitched ringing, sometimes a machinery-like roaring or hissing sound, like a steam kettle). Large tumors of the pontocerebellar angle may affect other local cranial nerves. Involvement of the facial nerve may lead to facial weakness and impairment of glandular secretions; involvement of the trigeminal nerve may lead to loss of taste and loss of sensation in the face and mouth, involvement of the glossopharyngeal and vagal nerves may lead to altered gag or swallowing reflexes. Even larger tumors may compress the adjacent brainstem and lead to increased intracranial pressure, with its associated symptoms, such as headache, vomiting, and altered consciousness [31,32].

Migraine and vertigo

Many patients with migraine have vertigo, between 26,5% - 42% of migraine patients experience vertigo. Almost one third of them have vertigo even without a head-

ache, while others experience vertigo during and after headache episodes. Patients having a migraine with aura have vertigo more often, probably because they can experience vertigo during aura [33,34,35]. On the other hand, 16-32% patients with vertigo have migraine [36,37]. These data suggest some connection between migraine and vertigo. Half of the patients with BPPV younger than 50 years fulfill diagnostic criteria for migraine [38,39]. Migraine is threefold more common in patients with BPPV than in the control group [40]. Therefore, BPPV could be a form of migraine without a headache, or a migraine aura that does not evolve into a migraine headache attack. The other hypothesis suggests that the inner ear could be damaged with migraine associated vasospasm leading to symptoms of BPPV [40].

In basilar migraine vertigo is one of the symptoms lasting from 5 minutes to 1 hour, accompanied with tinnitus, hearing loss, ataxia, dysarthria, visual symptoms, diplopia, paresthesias, paresis, and consciousness disorders followed by a migraine headache. Most patients with basilar migraine have positive family history [41].

Cervical vertigo

Proprioceptive information from neck muscles and joints assists in the coordination of the eyes, head and body. Therefore, disorders of the proprioceptive information could cause vertigo named cervical vertigo. Symptoms accompanying cervical vertigo include disorientation, instability, gait ataxia, and gaze abnormalities that tend to worsen with head movements. Cervical vertigo is more common in elderly patients probably due to degenerative changes of the cervical spine and atherosclerosis [42, 43].

However, some authors do not consider cervical vertigo a distinct form of vertigo because the mechanisms of cervical vertigo are not fully understood, and in differential diagnosis it is difficult to exclude other forms of vertigo [44].

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Sažetak

Diferencijalna dijagnoza vertiga

Vertigo je iluzija kretanja, najčešće rotacijskog kretanja. Vertigo je među najčešćim simptomima koji dovode bolesnike liječniku, a sa starenjem učestalost vertiga se povećava. Vertigo može biti uzrokovan poremećajima unutaršnjeg uha – periferni vertigo, centralnim poremećajima – centralni vertigo, sustavnim bolestima, a može biti i psihogeni. Najčešći uzroci vertiga su benigni paroksizmalni pozicioni vertigo, akutni vestibularni neuronitis, Ménièreova bolest, migrena, anksiozni poremećaji, vertebrobazilarna ishemijaa i tumori pontocerebelarnog kuta. Važno je razlikovati periferni od centralnog vertigo kako bi se mogle donijeti odluke o liječenju. U ovom članku prikazana je diferencijalna dijagnostika vertiga.

Ključne riječi: vertigo, periferni vertigo, centralni vertigo, benigni paroksizmalni pozicioni vertigo, akutni vestibularni neuronitis, Ménièreova bolest, migrena, vertebrobasilarna ishemija, tumor pontocerebelarnog kuta