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Short Note

Vestibular Neuritis

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DEFINITION

Vestibular neuritis (VN) is the second most common cause of peripheral vestibular vertigo. Other names have been used for this entity due to the ambiguity respecting the physiopathology, including vestibular neuronitis (involvement of the vestibular ganglion), labyrinthitis (when is associated with infection or inflammation of the labyrinth), and acute peripheral vestibulopathy (when there is no specific pathophysiology) [1]. VN is now considered the more accurate term when hearing is preserved.

EPIDEMIOLOGY

In recent studies an incidence of VN has been estimated in 11.7 to 15.5 per 100,000 per year [2]. Affects in most cases adults between 30 and 60 years of age, with no demonstrated differences between sexes. In the pediatric population with vertigo VN has been reported between 1 to 5% of cases [3]. Brodsky in 2015 found a prevalence of 3.6% of vestibular neuritis in a group of 301 pediatric patients evaluated in the vestibular clinic at Boston Children´s Hospital [4]. Epidemic incidences in certain periods of the year have been found (spring and summer) and approximately 50% of cases have relationship with certain respiratory infections. Bilateral disease was reported in 3.7% in a group study of 108 patients with bilateral vestibulopathy [5].

PATHOPHYSIOLOGY

Its etiology remains uncertain; the viral infection theory describes a reactivation of herpes simplex virus type 1 or an inflammation of the eighth cranial nerve caused by a neurotropic virus causing a dysfunction of the superior division of the vestibular nerve in most cases [6]. This might be secondary to the longer and narrower bony canal traversed by the superior nerve making it more susceptible to compressive swelling [7]. According to recent studies authors have found vascular risk factors in patients with VN, supporting a vascular etiology [2]. Other factors such as the increase of acute phase reactants like plasma fibrinogen, tumor necrosis factor alfa, cell adhesion molecules, and C-reactive protein associate VN with a vascular process [8]. In a retrospective study by Uffer et al. analyzed the vestibular lesion patterns in 25 patients with VN to determine the pathophysiology; they found that the lesion patterns varied significantly. 76% of the cases did not support the neuritis hypothesis since an innervation pattern was absent, 24% had a lesion pattern that either definitely (16%) or probably (8%) supported the Neuritis Hypothesis. This study concluded to reconsider the treatment for VN as the cause may be inside the vestibular labyrinth, therefore an intratympanic steroid injection might be a more effective management [9].

SYMPTOMS

VN presents with acute vertigo that develops over minutes to hours, associated with nausea, vomiting, and imbalance. It presents with horizontal spontaneous nystagmus with a fast-phase component toward the unaffected ear. VN is not associated with hearing alterations or with any focal neurologic complaints [10]. Generally dizziness lasts days, with gradual improvement. Vestibular imbalance can persists during months to years, and presents as persisting dizziness and oscillopsia during rapid head and body movements. The majority of cases are of a single occurrence; fewer than 5% will experience recurrent symptoms [11].

DIAGNOSIS

The caloric test is the gold standards for identifying the loss of vestibular function, enables the function of each labyrinth, and differentiates peripheral from central vertigo. During the acute stage of VN the caloric test are invariably abnormal, in some cases they might be normal, the abnormal findings is usually maintained for more than 1 year [12]. A Follow-up evaluation should be considered when the findings of the initial caloric test are normal [13]. The Video Head Impulse Test (vHIT) assesses the same reflex as the caloric test by using a video assisted examination of the impulsive maneuver. This test complements the caloric test [14]. Head-thrust testing in the canal plane helps to localize the pathology to the inferior or superior vestibular nerve. Vestibular- evoked myogenic potential (VEMP) testing is rarely useful. In a retrospective case-control study investigated the significance of neutrophil to lymphocyte ratio (NLR) and platelet to lymphocyte ratio (PLR) in VN. The mean NLR and PLR values of the patient group were significantly higher than those of the control group supporting the vascular theory [15]. Imaging studies such as MRI should be reserved for anyone who presents with an acute vestibular syndrome with high vascular risk factors. Neuroimaging has revealed in recent studies that during the acute phase of VN induces perfusion and metabolic changes in the vestibular cortex, with alterations in different sensory modules as a result of acute plasticity in the central nervous system [16].

Differential diagnosis

Includes acute vertigo of central origin (cerebellar or brainstem infarct or hemorrhage), labyrinthine ischemia, which

presents with hearing loss from associated cochlear ischemia. Migraine associated vertigo, Meniere's disease and retrocochlear lesion.

TREATMENT

The treatment has been supportive and symptomatic for vertigo and for the vegetative symptoms. To control the severe vertigo, nausea, and vomiting, vestibular suppressants and antiemetics are often used and should be limited to the first several days. Methylprednisolone should be started within 3 days of onset of symptom and has been beneficial in improving the caloric test response [17]. Antivirals recently have been reported as possible treatments, however according to placebo controlled double-blind studies valacyclovir was not shown to have any affect and should be reserved for patients though to have Ramsay hunt syndrome [18,19]. In the pediatric population the rate of spontaneous resolution is reported to be high among children younger than 15 years old compared with patients older than 15 years old. Further study is needed to determine the efficacy of oral steroids in the pediatric population [4]. If the etiology is unsure, a combination of systemic and intratympanic steroid treatment may be adequate. Vestibular rehabilitation is helpful in promoting soon recovery.

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