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Vestibular Neuronitis: Diagnosis,ManagementAnd Treatment

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Abstract*: Vestibular neuronitis (VN) is a common cause of sudden onset of vertigo. It results in hearing loss or ringing in the ears. Many cases are caused by reactivation of herpes simplex virus that affects the vestibular ganglion, and vestibular nerve. Certain neuro-toxicants have negative impact and aminoglycosides can cause vestibular toxicity.VN affects 3.5 per 100, 000, and age distribution between 40 to 50 years, with no gander difference. Symptoms of VN and labyrinths include dizziness or vertigo, disequilibrium, and nausea. Rotary vertigo lasting several days weeks, then slowly recedes with time. Most commonly unilateral, bilateral cases of vestibular neuritis have beendescribed. Chronic Anxiety Is A Common Side Effect Of Labyrinthitis. Diagnosis Can Usually Be Made Clinically; MRIor CT scanning may demonstrate tumors, hemorrhage or ischemic stroke. MRI is more sensitive than CT scanning but is not a pragmatic option. Management includes symptomatic supportive care during the acute phase. The efficacy of topical and systemic steroids requires further validation.*

Keywords*: Vestibulsar neuronitis, Neuritis, Vertigo, and Management*

I. Introduction

Labyrinthitis also known as otitis externa.Vestibular neuronitis and vestibular neuritis is a problem of the inner ear. It results in vertigo and also possible hearing loss or ringing in the ears. It can occur as a single attack or, a series of attack, or a persistent condition that diminishes over three or six weeks. It may be associated with nausea, vomiting. Vestibular neuritis may also be associated with eye nystagmus [1]. Neuronitis is the inflammation of the vestibular nerve but the etiology is thought to be a vestibular neuropathy. A theory gaining support is that a significant proportion of cases are caused by a reactivation of herpes simplex virus that affects the vestibular ganglion, vestibular nerve, labyrinth, or a combination of these[3,4].Vestibular neuritis affects approximately 3.5 per 100,000 people per year. It affects both adults and children, the typical age of onset is between 30 to 60 years, and the age distribution is between 40 to 50 years. There is no significant gander difference [5-7].The labyrinth and vestibular apparatus provides for maintenance of equilibrium and awareness of position. It senses the position of the eyes and head and reflexly controls tone in the limbs and body. Certain neuro-toxicants such as ethanol and organic solvents can have a specific impact on this system [8].Postural sway was impaired among workers exposed to formaldehyde [9].Aminoglycosides can cause vestibular toxicity, the target of drug-related vestibular toxicity is the type 1 hair cell of the summit of the ampullary cristae[10].Manifestations are usually abrupt, unsteadiness, nausea and vomiting, feeling of room being rotating, moving the head aggravates the symptoms, and distinguish between true rotational vertigo and giddiness, faintness or weakness[9,10].Diagnosis can usually be made clinically, imaging studies(MRI or CT scanning) may demonstrate tumors, hemorrhage or ischemic stroke[11].Treatment with drugs is neither necessary nor possible. The effect of glucocorticoids has been studied, but they have not been found to significantly affect long-term outcome [12].The paper reviews the current literature, diagnosis and management of vestibular neuronitis.

II. Etiology

The cause of this disease is unknown but neurotropic viruses have been implicated as the causative agent. The most commonly identified virus is herpes simplex virus (HSV-1), which is thought to exist in a latent form in human vestibular ganglion [13].The cause of this condition is not fully understood, and in fact many different viruses may be capable of infecting vestibular nerve. Some people will report having an upper respiratory infection (common cold) or flu prior to the onset of the symptoms of vestibularneuronitis, others will have no viral symptoms prior to the vertigo attack.[14].Acute localized ischemia of these structures also may been important cause. Especially in children, vestibular neuritis may be preceded by symptoms of a common cold. However, the causative mechanism remains uncertain [15].

Schuknecht and Kitamura also proposed a viral origin based on studies of human temporal bones obtained from patients with a known history of vestibular neuronitis, although the histological findings in temporal bones obtained from such patients vary somewhat according to the literature [16].A common finding is a diffuse lymphocytic infiltration with areas of gliosis in the vestibular nerve. The number of fibers in the vestibular nerve is also diminished on the affected side and there are marked signs of degeneration in the remaining fibers[17,18].This type of degeneration sometimes also involves Scrap's ganglion. In some cases the pathological findings include changes in the vestibular sensory epithelia with loss of sensory hair cells and other degenerative changes such as collapse of the ampullary walls over the neuroepithelium and loss of nerve fibers below the sensory epithelium [17].Sometimes the vestibular end organs are also infiltrated by lymphocytes, including the presence of an inflammatory response. Viralinclusions, however, have not been identified in the vestibular neurons according to current knowledge [18],nor were any signs of the vasculitis seen in the vestibular end organs or the vestibular nerve. These findings refer to patients with a single acute attack of the vestibular neuronitis but the histopathological appearance of vestibular nerve specimen as well as of the sensory end organs, are similar in cases with documented recurrent vestibular neuronitis[19].

Some researchers contend that vestibular system is a set of sensory inputs consisting of three semicircular canals, sensing changes in rotational motions, and the otoliths, sensing changing in linear motion. The brain combines visual cues with sensory input from the vestibular system to determine adjustments required to retain balance .The vestibular system also relays information on head movement to the eye muscle, forming the vestibuloocular reflex to retain continuous visual focus during motion [15].

In labyrinthitis,it is thought that generally viruses cause the infection, but rarely labyrinthitis can be the result of a bacterial middle ear infection.While there are several different definitions of the vestibular neuritis in the literature, with variable amounts of vertigo and hearing symptoms. The definition of Silvoniemi(1988) who stated that the syndrome of the vestibular neuritis is confined to the vestibular system. In vestibular neuritis, by definition, hearing is unaffected. In labyrinthitis, hearing may be reduced or distorted in tandem with vertigo[20].These definitions are flawed-they depend on clinical findings and imply anatomic localization that may not always be true.Recently evidence has been put forth that some patients with the clinical syndrome of "vestibularneuritis",anatomically may have lesions in the labyrinth(Murofushi*et al*,2003)[21].Although anatomic data is rarely available, if diagnostic technology improves in the future, there is a need to change the definition of "vestibular neuritis"[22].

II. Pathogenesis

Vestibular neuronitis is a common disorder of unknown origin. In spite of certain histological evidence, there is still a lack of uncontestable evidence that the condition is caused by inflammatory changes in the vestibular nerve. A good review of this condition and histopathological findings in affected patients is given by Nadol[17].The issue of ototxicity became a clinical reality after the introduction of streptomycin in mid-1940s.Hinshaw and Feldman reported on severe inner ear disturbances that occur after treatment with streptomycin[23].Causse*et al* could also show structural damage in the vestibular apparatus[24].Ototoxic drugs include: aminoglycosides, loopdiuretics,cytotoxic drugs,salicylate,qunine,anderythromycin. All these drugs proved to be more or less ototoxic and until the development of gentamicin that this new drug proved to be reliable and safe when properly monitored[25].A review of aminoglycoside effects on the inner ear by Bagger-Sjoback[26,SB,5] includes:

(a)accumulation in inner ear fluids and tissues
(b)action on cell or subcellular level
(c)cochleotoxic and /or vestibulartoxic effects and
(d) Often permanent ear damage.

The pathological findings in the vestibular end organs caused by aminoglycosides ototoxicity often start with a derangement of the sensory hairs resulting in hair fusion creating deformed or so called 'giant hairs'[27].As the damage progresses ,the cell bodies change in shape and structure and are often filled with vacuoles and degenerating mitochondria.As a result of these severe intracellular changes, the sensory hair cells subsequently die and disintegrate leaving a 'scar' in the epithelium[28].

Pathologic study of a single patient documented findings compatible with an isolated viral infection of Scarp's ganglion(the vestibular ganglion).There was a loss of hair cells"epithelization" of the utricular maculae and semicircular canal cristae on the deafferentedside, and reduced synapanic density in the ipsilateral vestibular nucleus[18].In spite of the limited pathology that would suggest involvement of the entire vestibular nerve, there is reasonable evidence that vestibular neuritis often spares of the vestibular nerve, the interior-division)e.g.Fetter and Dichgans,1996;Goebel *et al*,2001)[29,30] as well as spare the superior division(Aw *et al*,2001)[31] although not all agree(Lu *et al*,2003){32].Because of the inferior division supplies the posterior semicircular canal and saccule ,even a "complete" loss on vestibular testing(associated with a superior canal lesion)may be associated with some retained canal function.Similarly,an inferior division vestibular neuritis might be associated with a normal ENG test but an abnormal VEMP(vestibular-evoked myogenic potential)

test.Furthermore, it is common to have another dizziness syndrome,BPPV(benign paroxysmal positioning vertigo),follow vestibular neuritis. Presumably this happens because the utricle is damaged (supplied by the superior vestibular nerve), and deposit loose otoconia into preserved posterior canal (supplied by the interior vestibular nerve)[22]

III. Clinical Presentation

The symptoms of both vestibular neuritis and labyrinths typically include dizziness or vertigo, disequilibrium or imbalance, and nausea acutely, dizziness is constant. After a few days, symptoms are often precipitated by sudden movements. A sudden turn of head is the most common "problem"motion.While patients with these, disorders can be sensitive to head position, it is nor generally related to the side of the head which is down(as in BPPV).but whether the patient is lying down or sitting up[22].

The patients usually present with a single episode of episode of rotatory vertigo lasting for several days or weeks, which then slowly recedes over time. A smaller group of patients, however, may present with similar symptoms but in a relapsing mode. These episodes are usually less intense and also shorter than initial acute single attack. Whether these two conditions represent two conditions or whether they represent two different entities is not yet known.Although, most commonly unilateral bilateral cases of vestibular neuronitis have been described in the literature [19].

The typical clinical case of vestibular neuronitis is an adult patient who suddenly experiences quite violet rotatory vertigo. The patients are usually able to describe the exact moment when the vertigo occurred and the feeling of disequilibrium or dizziness is often so pronounced that patient cannot remain upright, but has to stay in in a supine position. There is also a typical absence of auditory symptoms such as hearing loss or tinnitus as well as other neurological symptoms and signs. The vertigo or feeling of disequilibrium slowly recedes over the following days and the patient is usually able to sustain him or her within a week. Normally the vertigo gradually reduces over a few weeks, but it may take up to several months before the patient is totally free of sense of disequilibrium. Some patients experience a postural vertigo due to unilateral sudden partial loss of vestibular function [33].

Chronic anxiety is a common side effect of labyrinthitis which can produce tremors, heart palpitations, panic attacks, decreolization, and depression. Three models have been proposed to explain the relationship between vestibular dysfunction and panic disorder[34].

(a) Psychosomatic model:vestibular dysfunction that occurs as a result of anxiety.
(b) Somatopsychicmodel: panic disorder triggered by misinterpreted internal stimuli(e.g.,stimuli from vestibular dysfunction),that are interpreted as signifying physical danger. Heightened sensitivity to vascular sensations leads to increased anxiety and, through conditioning, drives the development of panic.
(c) Network alarm theory: panic that involves noradregenic,serotonergic,andother connected neurol systems. According to this theory, panic can be triggered by stimuli that set off a false alarm via a afferents to the locus cerlueus,which then triggers the neuronal network. The network is thought to mediate anxiety and includes limbic,midbrain, and prefrontal areas. Vestibular dysfunction in the setting of increased locus ceruleus sensitivity may be potential trigger.

In Summary:(a)vestibular neuronitis has sudden onset(b) only the vestibular system is affected(c) there is severe rotatory vertigo at onset and,(d) a gradual recovery with time[19].

IV. Diagnosis

Investigations

Magnetic resonance imaging (MRI) with emphasis on the identification of both infarction and hemorrhage in the brain stem and cerebellum, is obtained in patients with risk factors for stroke,with additional neurological abnormalities, and who do not show improvement within 48 hours .Alternately ,computed tomography(CT) scanning with thin cuts to evaluate the brainstem cerebellum, and fourth ventricle may be obtained[35].In most patients, vestibular testing shows a complete or reduced caloric response in the injured ear. The caloric response eventually normalizes in 42 % of patients. VEMP responses are attenuated or reduced (35]. Diagnosis can usually be made clinically and blood tests are usually unhelpful .MRI or CT scanning may demonstrate tumors,hemorrhage or ischemic stroke or demyelination. Imaging tends to be reserved for cases where more sinister pathology is suspected. With respect tostroke, MRI is much more sensitive than CT scanning but is not often a pragmatic option[11].

Differential Diagnosis

The diagnosis of vestibular neuronitis is based on the constellation of symptoms and signs, however, they may be mimicked by other disorders as well. The need for further evaluation is necessary only if there is a concern for a central cause of acute vertigo or if the acute vertigo does not substantially improve in 48

hours[35].The primary central cause for acute vertigo lasting days is a brainstem or cerebellar stroke. In most cases, there are other neurological findings:diplopia,dysmetria,dysarthria,motor and sensory deficits, abnormalreflexes, the inability to walk without falling, and a central nystagmus is not affected by visual fixation and may change directions with changes in gaze. A purely vertical or purely torsional nystagmus is highly suggestive of a central disorder. In the event of an isolated inferior cerebellar stroke, the presentation may be indistinguishable with vestibular neurinitis.Also,25% of patients with risk factors for stroke who present with vertigo, nystagmus, and postural instability have had an inferior cerebellar stroke.Therefore ,patients with significant risk factors for stroke should have an imaging study if they present with these symptoms[35,36].

In Summary[37,38].(i)benign positional vertigo(ii)labyrinthis(iii)Meniiere's disease(iv)stroke,especially posterior inferior cerebellar artery syndrome(the central signs not always present so stroke should be considered in any patient with cardiovascular risk factors)[3].(v)multiple sclerosis(vi)subarachnoid hemorrhage(vii) tumors of the brain or acoustic neuroma and(viii) cervical spondylosis.

V. Management And Treatment

The primary management includes symptomatic and supportive care during the acute phase of illness.The patients are given vestibular suppressants and antiemetic to control the vertigo, nausea and vomiting. These medicine are withdrawn as soon as possible to avoid interfering with the central vestibular compensation [35].Frequently used management includes[39].

1. Assurance to be given to the patient that symptoms usually settle within a few weeks, even with no treatment. Alcohol .tiredness and recurrent illness can aggravate symptoms.
2. Advice on safety risks-driving, hazards at work (eg. heavy machinery, ladders) and falls at home during attacks.
3. Bed rest may be necessary if symptoms are severe but early activity should be encouraged to promote vestibular compensation.
4. Patient with marked vertigo with vomiting,an anti-emetic is useful and it may suppress the vertigo too.Nausea will retard gastric emptying and injection may be required.
5. Vestubular suppressant such as prochlorperazine (buccal or intramuscular if vomiting is severe) may be beneficial but should be stopped within few days of onset, as prolonged use may impeded the process of central vestibular compensation. This may be self- administered medication as necessary rather than regularly after first three.
6. Buccal prochlorperazine may be appropriate if the patient has recurrent attacks and needs self-administered medication.
7. Promethazine and domperidone may be effective.
8. Antiviral drugs and steroids are not recommended.
9. A Cochrane review found moderate evidence that vestibular rehabilitation (exercises to promote central nervous system compensation was effective in reducing dizziness in vestibular neuritis in the medium term)[40].

Fishman and colleagues in series of four randomized trials comparing corticosteroids with placebo, no treatment or other active treatment for adults diagnosed with idiopathic acute vestibular dysfunction, concluded that there is currently insufficient evidence to support the administration of corticosteroids to patients with idiopathic vestibular dysfunction [12].Jeong and associates contend that symptomatic medication is indicated only the acute phase to relieve the vertigo and nausea/vomiting. Vestibular rehabilitation hastens the recovery. The efficacy of topical and systemic steroids requires further validation [4].

VI. Prognosis

Patient will probably be unable to work for one or two weeks, and may be left with minor sensitivity to head motion which will persist for several years, and may reduce ability to perform athletic activities such a racquetball, volleyball and similar activities. After the acute phase is over, for a moderate deficit, falls are no more likely than in persons of your age without vestibular deficit[41].Persons in certain occupations, such as pilots, may have long term impact[42,].Patient may have problems with thinking. Even in persons who are well compensated, sensory integration seems to require more attention in persons with vestibular lesions than normal subjects [43].

In patients with recurrent vestibular neuritis-benign recurrent vertigo(BRV),fortunately, in great majority of cases (at least 95%) vestibular neuritis it is a one-time experience.Rarely(5%) the syndrome is recurrent, coming back at least once, and sometimes year after year. When it is recurrent, the symptom complex often goes under other names. These include benign paroxysmal vertigo in children[44].,benign recurrent vertigo[45,46] or vestibular Meniere's syndrome[47].Many authors attribute this syndrome to migraine

associated vertigo. There is often familiar pattern [48],and it may instead be an entity by itself[49] but lacking any clear diagnostic findings that distinguish it from recurrent vestibular neuritis or acephalagic migraine[22].
In most patients, symptoms resolve within few weeks, with or without symptomatic treatment[39].Another study found a persistence of dizziness related to anxiety in one third of patients one year after the initial episode[50].

VII. Conclusion

Vestibular neuronitis a common disorder of unknown originIt has a sudden onset; only the vestibular system is affected, with severe rotatory vertigo and gradual recovery with time. In some patients dizziness is related to anxiety.

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