SOME ASPECTS OF VERTIGO : A REVIEW

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Vertigo is an aberrant sensation of movement of oneself or the surrounding which results in true or sensory vertigo. Affection of the vestibular sub-system, starting from the peripheral labyrinth to the cerebral cortex (temporal lobe and post-central gyrus) leads to dizziness. Nystagmus is the most important manifestation of vestibular disease[1].

History taking is the fundamental factor in the diagnosis of vertigo. At the end of history taking one can usually make a clinical diagnosis of a condition causing vertigo.

Onset of attacks

History of attacks, their frequency and recurrences or paroxysms should be noted as Meniere's disease induces extremely severe paroxysmal attacks in groups within a period of weeks or months followed by freedom from vertigo for months or years, while bilateral vestibular destruction due to ototoxic affection gives rise to symptomatology of unsteady gait and in acoustic neuroma, one observes baffling vestibular symptoms at different stages of the disease . Thus whether attack is abrupt or progressive is one of predominent determining factor in the clinical picture.

Unsteadiness is a loss of equilibrium in relation to one's environment often discribed by the patient as falling or swaying sensation.

Associated Symptoms

Cochlear Symptoms- deafness and its type, whether low frequency or high frequency, permanent or fluctuating in nature, tinnitus, sensation of stuffy ear, earache should be noted :

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Visual disorders - Imbalance in visual perception related to kinetosis can be experienced as a spectator of projection on a wide screen. Clinically the blurred vision resulting from recently operated cataract or fusion disorders including badly corrected refraction. Vertigo of visual origin has got three characteristics: it diminishes on closure of eyes, not accompanied by nystagmus and all factors which interfere with its compensation i.e. intake of sedatives can aggravate its manifestation (vomiting).

Past History

An episode of acute otitis media, meningitis or viral disease like influenza. A recent head injury often can cause vertigo; it could be due to labyrinthine disturbance or fracture of petrous bone with facial paralysis, Cophosis or otorrhagia. True Meniere's disease can eventually follow labyrinthine disturbance. In the case of head injury with skull base lesions multiple cranial nerve neuropathies can occur.

Precipitating or aggravating factors:-

Peripheral Paroxysmal Vertigo cases are aggravated by changes in head Posture . Blowing of the nose or cough which could provoke a Labyrinthine fistula.Hyper-extension or hyper-rotation could cause vertebrobasilar insufficiency causing an attack of vertigo resulting in a fall, tendency of nausea or vomiting. These symptoms indicate peripheral vertigo.

Drug intake, mostly seen with aminoglycosides, salicylates, diuretics and ear drops especially cold ear drops can lead to vertigo. Complications of ear surgery can also lead to vertigo.

History of atherosclerosis, diabetes, hypertension cardiovascular and neurological diseases should be enquired into. Family

history of Neurofibromatosis, Meniere's disease or otosclerosis needs to be documented. Alcohal intake is also of importance as a cause of dizziness.

Traumatic labyrinthine fenestration

Barotraumatism, blast, diving accidents or even a slap can be followed by repeated attacks of vertigo.

A lesser degree of vertigo could be due to deficiency in tubal function which is likely to induce attacks of Meniere's disease. Air travel and habitation in mountains can also lead to vertigo.

Vestibular Signs

Nystagmus is the chief sign which is an involuntary, rhythmic, oscillation of the eye balls, affecting both eyes in two phases: a rapid phase which is clinically visible, therefore indicating the direction of nystagmus; a slow phase of vestibular origin. Nystagmus is investigated by vestibulo-occular function tests. It is simple and very important to check nystagmus with magnifying glasses (+20 diopters), because peripheral nystagmus may be abolished by gaze fixation. Nystagmus can be variable in intensity or degree. The number of jerks per 30 seconds is a good criterion for evaluation. Nystagmus is abolished by visual fixation and should be studied at the patient's bedside with discreet lighting (pocket lamp), whereby the use of magnifying or illuminating lenses is dispensed with. The direction of the nystagmus shows which side of the ear is presumably affected, and this makes it possible to define whether the nystagmus is irritative (directed to the diseased side), or destructive (directed to the healthy side).

Evaluation at the time of the nystagmus is an important factor: progressive abolition usually indicates compensation: inversion, in general, signifies transition from the irritative to the destructive stage; cyclic alternance is encountered in certain affections (Meniere's disease). Besides the labyrinthine receptor, other factors also contribute to regulation of vision, particularly vision itself, proprioceptive sensitivity of extrinsic muscles of the eyeball.

Lateralised segmental corporeal deviations revealed by vestibulospinal function test - *Finger test* for deviation of the index finger during extended arm test. *Romberg test* with eyes closed which shows up a tendency to fall sideways. *A blind walk test* is for zig-zag deviation in gaze. E.N.T. examination including otoscopy is fundamental requirement specially looking for serous otitis, acute otitis media or cholesteatoma.

Neurological Examination

Puretone & speech audiometry - In Miniere's disease on pure tone audiometry, low tone fluctuating sensorineural hearing loss accompanied by diplacusis, recruitment and impaired speech discrimination but in eustachian tube dysfunction loss is conductive and no diplacusis or recruitment or impairment of speech discrimination present while in cerebellopontine angle tumour there is unilateral sensorineural loss with marked loss of speech discrimination and decay.

Following tests need to be done according to clinical suspicion: Impedance audiometry, BERA, Electrocochleography, Electro-nystagmography (ENG)

Radiology - May be needed to show a fracture or tumour of the base of skull. Brain tumours or extensive cholesteatoma or acoustic neuroma cases need to be further evaluated by C.T. scan & M.R.I. (with contrast)

Differential Diagnosis

Middle ear affections -

A *cholesteatoma* - Suspected by chronic fetid otorrhea with upper quadrant (attic) perforation of tympanic membrane with flakes coming out on aural toilet and with mixed type of hearing loss. Surgery is compulsory; an emergency operation may even be needed.

Fistulas of semicircular canal, oval window or round window can be caused by the disease. Leakage of perilymph can produce episodic

vertigo, vertigo begins abruptly after rapid pressure changes in middle ear. The patient often complains of constant feeling of imbalance or unsteadiness, high frequency unilateral hearing loss is the differentiating feature on audiometry. A positive fistula test with pneumatic speculum is accurate in 24% cases[2].

Fistula testing with impedance audiometry and Electronystagmography can be positive with limited accuracy[3].

Bacterial labyrinthitis - It can be a complication of any type of otitis media.

Otospongiosis (Otosclerosis) Diagnosed when there is conductive hearing loss with intact tympanic membrane, reduced compliance (less than 0.5cc) and absent acoustic reflex on impedance. There is unsteadiness, nystagmus , nausea or vomiting are usually not present.

Post opereative complication of ossiculoplasty, stapedectomy and mastoid surgery can manifest as vertigo[4,5].

Positional Vertigo

It is diagnosed by violent vertigo associated with nystagmus, ataxia and nausea or vomiting. The posture is characteristic as vertigo is induced by turning the head in a particular position. If the head is placed in the same position a few moments later the vertigo or symptoms are diminished or absent[6].

Vertigo always lasts for less than thirty seconds. Most of the time precipitating posture corresponds to one of the following three positions (a) Lying down with the cheek on the pillow and the head turned to the Right or left (b) turning over while lying down in bed (c) Sudden inclination of the head laterally when in a seated posture[7,8].

The vertigo can be reproduced by asking the patient to mimic the precipitating posture or by Dix and Hallpike manoeuvre as follows:- the patient being seated on a hard bed is made to lie down abruptly on his back with his head turned laterally on one ear.

Non-fatigable positional vertigo may be due to mid line cerebellar lesion. Positional vertigo usually lacks hearing impairment and it is a self limiting disease.

If the patient is motivated, patient can accustom himself by assuming the precipitating posture every three hours until there is remission of attack for over two days.

Post Traumatic Vertigo

Associated with head injury or due to surgical assault is easy to diagnose from the history.

Vestibular Neuronitis

Clinically manifested by violent vertigo associated with nystagmus lasting for days or even weeks, aggravated by head movements. Disease is sudden and unilateral. Cochlear signs are absent, Viral cause is suspected, Caloric test confirms it by unilateral absent response.

Eustachian Tube Occlusion

Alteration in intratympanic pressure can result in momentary vertigo, more pronounced in mountaineers, divers and after a flight, hearing loss is conductive, no diplacusis, no recruitment or impairment of speech discrimination, on impedance, pressure is negative and compliance is significantly reduced but acoustic reflex is usually present until associated with severe sensorineural deafness or adhesive otitis media.

Meniere's disease

The four cardinal symptoms, episodic vertigo, fluctuating hearing impairment, aural fullness and roaring type of tinnitus are diagnostic of Meniere's disease. Vertigo is not related to posture, always accompanied by nausea, vomiting, nystagmus, ataxia and vagal symptoms may be associated with it. Attacks of vertigo occur in groups at any time within a period of weeks or months followed by complete relief for months or even years[9].

On caloric testing response is diminished on the affected side in most of the cases, rotatory chair test can suggest peripheral or central cause of vertigo.

On Audiometry low frequency sensorineural deafness with distortion and diplacusis are

characterstic, recruitment of loudness is usually present. Short increment sensitivity index is high.

There is improvement in speech discrimination as the attack subsides or on giving glycerol.

On Electrocochleography summating Potential as well as summating potential to action potential ratio is prolonged[10].

Cerebellopontine angle tumour

Most often schwannoma of vestibular nerve is the cause in this space.Disease is progressively destructive hence vestibular symptoms are discrete usually presents as progressive unilateral sensorineural deafness with poor speech discrimination, dizziness and headache, recruitment to loud sound is absent[11]. Caloric test is usually absent. Decay test is positive and on impedance acoustic reflex decay of more than 50% is pathognomonic of acoustic neuroma. B.E.R.A. is diagnostic.

Cervical Spondylosis

A clinical syndrome resulting from degeneration of the intervertebral disc and consequent pressure on the cervical nerve roots or cervical spinal cord. The protruding intervertebral disc and osteophytes produce two clinical syndromes depending on their effects: (1) spinal cord pressure or myelopathy, and (2) spinal nerve root pressure or radiculopathy.

Presents with symptoms of pain in muscles of cervical spine, giddiness. Spontaneous nystagmus is absent. When a normal neck is moved the vertebral and basilar arteries have a considerable excursion. If the fibrosis associated with spondylosis sufficiently restricts this excursion, the patient on twisting his neck , may feel dizzy or even black out and fall. Angiography demonstrates the lesion. On autopsy the spinal cord showed indentation at the levels of intervertebral discs, and impingement on the vertebral arteries laterally was also present due to the malalignment of the spine[11].

Caloric shows usually hyper active response. Oblique radiographs of cervical spine will show the appearances of osteophytes and bony lips impinging on the intervertebral foramina. Kopfschuttel nystagmus (on shaking the head sideways around a vertical axis for 20 times) was observed a significant number of cases of cervical spondylosis[1].

Sudden Labyrinthine deafness

Occurs mostly in young and middle aged with sudden onset of tinnitus and severe deafness followed by vertigo, distortion, diplacusis, recruitment and impaired speech discrimination[12].

Occlusion of the internal auditory artery or of its branches by spasm, embolus or thrombus may be the cause. A viral etiology of Mumps, Influenza, Ramsay Hunt syndrome or Herpes Zoster may be the cause; it can be confirmed by seroconversion tests.

Other Causes

In *cerebral ischaemia* there is feeling of dizziness but true sense of vertigo is lacking. In *central vestibular lesion* of brainstem or cerebellum, an equilibrium continues to be disturbed, Nystagmus, nausea and vomiting, hearing impairment are usually not present.

Hypoglycemia provokes a lipothymic state, can create a confusion of vertigo, history of specific time and blood sugar levels clinches the diagnosis.

Orthostatic vertigo results by abrupt fall in cerebral pressure; it occurs during vertical body movement, *Subclavicular stealing* results from vascular insufficiency in the vertibrobasilar area, the attack occur during physical activity of the upper limbs. Hypothyroidism, anaemia, leukaemia, hypertension should also be looked for.

Phobias

Aero-phobia - Morbid fear of air, airborne influences or bad air.

Agora-phobia - Phobia to a variety of situations or situational phobia. The patient feels intense fear to places away from home, or in crowd or in closed places.

Vertebrobasilar insufficiency

Patient usually presents with transient numbness and tingling of face especially around the perioral region. When the neck is hyper-extended or twisted extensively results in frank sensation of vertebro-basilar insufficiency leading to labyrinthine ischaemia. It can be recorded by electro nystagmography[5].

Medical Management

Labyrinthine sedatives

Phenothiazine - Suppress central vestibular pathways

- Prochlorperazine 10mg x TDS
- Triflu promazine HCl 5-10 mg TDS.

Antihistaminics - acts as end organ suppressant

- Promethazine HCl - 25 mg x BD
- Dimenhydrinate - 50 mg x TDS

Benzodiazepins - Diazepam

Anxiolytic and anti-depressants modify the subjective response to vertigo by decreasing resting activity in vestibular nuclei reticular facilitatory system and affecting the brain amine levels by interfering with their uptake into binding sites. Diazepam 2-5 mg 8 hourly and Amitryptaline 10 mg x TDS.

Diazepam is the best labyrinthine sedative, next is dimenhydrinate 150 mg in three divided doses and promethazine 25 mg x BD.

Any patient complaining of dizziness should not be indiscriminately treated with Meclazine, Prochlorperazine, Diazepam etc. as a blanket cover, since this practice is not only helpful in many cases; but on the contrarily, it does harm to many in the form of delaying the recalibration process and at times perpetuating the symptoms.

Cinnarizine - It has labyrynthine sedative action and it also depresses vestibular reflexes induced by caloric stimulation of the labyrinth. Cinnarizine has also demonstrated calcium channel blocking activity[13]. Both cinnarizine and its difluorinated derivative flunarizine inhibit calcium - induced contraction of depolarized arteries by reducing calcium transfer to depolarized vascular smooth muscle. Thus, its antivasoconstrictor effect is accounted for by its calcium entry blocking activity. It is well absorbed orally and has a half-life ranging from 3 to 24 hours. Cinnarizine 75 mg. b.d., for seven days has demonstrated satisfactory vertigo suppression in patients with well defined peripheral or central vestibular disorders. Particularly favorable results are seen in patients with sudden peripheral vestibular deficit from vertigo of circulatory origin and from post-traumatic vertigo. Results were less conclusive in patients with Meniere's disease or central neurologic disorders (multiple sclerosis or heredito - degenerative diseases). In trials, the superiority of long term treatment with cinnarizine has not been demonstrated conclusively over placebo due to spontaneous improvement occuring in that group[14].

Pyridoxin acts as GABA mediated inhibition 100 mg in two divided dose.

Anticholinergic drugs - Inhibit central and peripheral cholinergic pathways thus blocking parasympathetic system and depression of smooth muscle activity, and of cerebral and medullary centre.

- Atropine - 0.5 mg I.M.I.
- Propantheline - 15 mg x BD
- Glycopyrrolate - 0.2 mg twice a day by intramuscular injection

Adrenergic - It acts by depressing the Chemoreceptor trigger zone and vomiting centre, activates reticulo vestibular pathways and depresses the vestibular nuclei - dextro amphetamine 10mg x BD, Ephedrine 25mg x TDS is the dose.

Severe Vertigo - Droperidol is a neuroleptic agent and quite effective in controlling acute vertigo.

Low Molecular weight - Dextran, I.V. is quite effective.

Vasodilators

Intravenous histamine - Histamine Diphosphate 2.75 mg in 250 ml of Normal saline infusion

40 drops per minute.
Carbogen inhalation - 5% Carbon dioxide and 95% oxygen mixture is given for 20 min. 2-3 times a day for 10-15 days or till the vertigo lasts.

Cyclandelate 400 mg twice a day or 200 mg 4 times a day. Papaverine 5 mg/Kg body weight for 7 days.

Kinesitherapy

Optokinetic training or yogic exercises in their way help in achieving better arousal hence relief in vertigo[15-19].

Salt Restriction and Salt substitute, Diuretics (Decreases the intralabyrinthine pressure) food supplements for potassium.

Corticosteroids - decreases labyrinthine oedema and swelling due to virus infection, Prednisolone 40-60 mg, in single daily dose.

Allergic Managements

- for middle ear affection
- food allergies

Smoking and stress - Vasoconstrictive nature of nicotine probably effects the microvasculature of the endolymhatic sac and decreases the absorption of labyrinthine fluid.

Acetazolamide and Methozolamide, used in glaucoma are of value in controlling vertigo but all patients can not tolerate it.

Streptomycin Therapy

Streptomycin causes audiologic and vestibular dysfunction[20]. 1gm x BD x 10days gives a good response in decreasing the attack of chronic vertigo, Caloric test and audiometry are mandatory during the regime.

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