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A Case of Drastic Improvement in a 43 Year Old Male with Severe Refractory Incapacitating Vertigo

Gauri Billa[1*] and Karan Thakkar[2]

[1]Urban Health Center, NMMC, Koparkhairane, India.
[2]Urban Health Center, NMMC, Ghansoli, India.

Authors' contributions

This work was carried out in collaboration between both the authors. Both the authors were involved in diagnosis and treatment of the case and preparation of this case report.

Case Study

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ABSTRACT

Introduction: Vertigo is a common and distressing complaint seen in primary care. It can be due to central or peripheral causes, which should be determined right at the outset. In some cases, the vertigo is transient and easily manageable while in others it can be more troublesome and difficult to treat.
Case: A 43 year old male presented with episodes of severe vertigo and vomiting progressively increasing since six months. Despite multiple consultations, complete work up and treatment with the standard therapy for suppressing vertigo, his symptoms persisted. On history and examination, it seemed to be a case of Ménière's Disease and we started him on specific therapy. The patient showed marked improvement within a week and continued to do so at the end of three months despite stopping all treatment.
Conclusion: Identification of the cause of vertigo, multidisciplinary management and specific treatment, rather than just vestibular suppressants, are paramount in managing a case of vertigo.

Keywords: Dizziness; imbalance; Ménière's disease; benign positional paroxysmal vertigo; vestibular neuritis; labrynthitis.

**Corresponding author: Email: dr.karanthakkar@outlook.com;*

1. INTRODUCTION

Vertigo has been defined as '*An erroneous perception of self- or object-motion or an unpleasant distortion of static gravitational orientation that is a result of a mismatch between vestibular, visual, and somatosensory systems*' [1]. It is a common complaint seen in approximately 20-30% individuals attending outpatient clinics [1–3]. It can be mild and transient or severe and incapacitating.

The various causes of vertigo are shown in Table 1 [1,3–6]. It is very important to differentiate between central and peripheral causes of vertigo. In cases of central vertigo, the perceived spinning can be in any direction, visual fixation is not suppressed, vertigo and/or nystagmus are not fatigable and the patients usually have severe instability and additional neurological features [6]. Peripheral vertigo is the opposite and has the following features: the perceived spinning is horizontal and never vertical and they commonly have additional otological features [6]. Although vasodilators like Betahistine and vestibular suppressants like Cinnarizine, etc., are used widely to treat vertigo, identification of the cause and specific treatment are paramount.

Table 1. Various causes of vertigo

Type	Cause
Central	Vertibro-basilar insufficiency
	Ischemia in the cerebellar artery distribution
	Cerebellopontine angle tumours/ vascular lesions
	Multiple Sclerosis
	Generalised Anxiety disorder
Peripheral	Benign Paroxysmal Positional Vertigo
	Ménière's Disease (Idiopathic Endolymphatic Hydrops)
	Vestibular Neuritis
	Labrynthitis
	Migranous vertigo
	Perilymphatic fistula
	Superior canal dehiscence
	Otitis media
	Chronic otomastoiditis
	Cholesteatoma
	Toxin/ drug induced: (Alcohol, Aminoglycosides, Salicylates, Frusemide etc.)

2. CASE PRESENTATION

A 43 year old male and his wife walked in our clinic and we could see that he was holding his head straight and, as stiff as a stick. His chief complaints were bouts of severe vertigo accompanied by vomiting since the last six months. His vertigo was precipitated by head or body turning to either side and lasted for about 30 minutes to an hour. He had obtained several consultations till now.

During one of his consultations, he was diagnosed as a case of vestibular migraine and treated with Tab Flunarizine 10mg at bedtime for one month. He had also been referred to an ENT specialist who didn't find any abnormality, but had not documented the performance and results of the Dix-Hallpike test or any other tests, and also didn't conduct an Audiometric

testing. When he came to us, the patient was already on Tab Betahistine 16mg three times a day, Tab Cinnarizine 30mg two times a day, mouth dissolvable Ondansetron 4mg on an 'as and when' basis and B complex vitamins.

Since the last month and a half, his symptoms had become more frequent and severe despite treatment. He was severely incapacitated and couldn't even leave his house for the fear of falling. On enquiry, he also gave a history of a buzzing sound and fullness in his left ear and a fluctuating hearing loss. He gave no history of earache, ear discharge, headaches, aura, photophobia, lightheadedness, blackouts, unsteadiness, weakness, fever, any focal neurological complaints or intake of any ototoxic drugs. There was no history of antecedent cold/ flu, head trauma, surgery or any major life stressor in the recent past. Review of his lab reports, CT-scan and MRI didn't reveal any abnormalities except a raised erythrocyte sedimentation rate (ESR) of 45mm/hr at the end of the first hour.

When we tried to examine his vestibular system and the vestibulo-ocular reflex by performing a Dix-Hallpike manoeuvre and the Head impulse test, respectively, he vomited and we had to stop the test. After a while, when he was feeling better, we conducted his general and neurological examination but didn't find any abnormality. Also, there was no evidence of orthostatic hypotension. His symptoms seemed to fit the description of Ménière's Disease (MD) and we decided to treat him for the same. We didn't conduct an electrocochleography (ECOG) because it has been reported that there is poor correlation between clinical progress and ECOG findings [7].

We gave him an intramuscular injection of Dexamethasone 8mg and started him on Tab Prednisolone 20mg once a day in the morning for 10 days, Tab Acetazolamide 250mg twice a day and a salt restricted diet. When he came to our office after a week, the marked improvement in his symptoms was clearly evident from the way he could freely walk and move his neck. He had not experienced even a single bout of vomiting during that week. We continued Acetazolamide at the same dose, reduced Prednisolone to 10mg once a day and Betahistine to 16mg twice a day, and stopped Cinnarizine. After seven days, we stopped Betahistine and reduced Prednisolone to 10mg every alternate day, and then stopped Prednisolone after seven more days. We continued him on Acetazolamide 250mg once a day for a month and stopped that too. It has been three months after stopping his medication and he has experienced only a single episode of mild transient vertigo without nausea. He has reported that his hearing also seems to be better and the buzzing sound in his years has disappeared.

3. DISCUSSION

The most important thing to do when one encounters a case of vertigo is to differentiate central and peripheral vertigo otherwise the consequences can be disastrous. In case of our patient, his history, absence of neurological signs and symptoms, presence of otological symptoms and a normal MRI and CT scan ruled out a central cause. From amongst the various causes of vertigo, in our opinion, MD was the best fit, as per the criteria described by the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) Committee on Hearing and Equilibrium diagnostic guidelines (1995) [8]:

1. Multiple episodes of vertigo lasting for more than 20 minutes.
2. Associated with fluctuating hearing loss (although, not Audiometrically documented in our case).

3. Associated with a low pitched tinnitus, described by our patient as a buzzing sound, and ear fullness.

The clinical features of other common causes of vertigo are given in Table 2. Although, classically, vertigo induced by head movement is caused by Benign paroxysmal positional vertigo (BPPV), it can be seen with other causes also. The reason being that in any kind of central or peripheral vertigo, head movement will accentuate the baseline asymmetry in vestibular function leading to vertigo [5]. Besides, in BPPV the episode of vertigo is usually transient and not associated with nausea or vomiting, hearing and balance function tests are normal, they don't respond very well to medication and the treatment of choice is otolith repositioning manoeuvres like Epley's manoeuvre [5,9,10].

Table 2. Clinical features of various common causes of peripheral vertigo

Disorder	Duration of an episode	Auditory Sx	Chronicity	Associated Sx	Triggers
Vestibular neuritis	Days	None	Single event	Nausea, imbalance	None
BPPV	Seconds	None	Daily with clusters lasting days or weeks	Imbalance	Head movement in vertical or rotational plane
Ménière's Disease	Hours	Hearing loss, tinnitus, fullness	Episodic	Nausea, imbalance	High-salt, stress, spontaneous
Labyrinthitis	Days	Hearing loss	Single event	Nausea, imbalance	None
Posterior fossa ischemia	Days (TIAs are usually in minutes)	Sudden deafness	Stuttering onset or single event	Central ocular abnormalities	None
Migrainous vertigo	Minutes-days, usually hours	Occasional tinnitus, fullness. Rarely hearing loss	Episodic	Photophobia, phonophobia, headache, aura	Typical migraine triggers
Perilymphatic fistula	Seconds	Sudden, fluctuating, or progressive hearing loss, ear fullness, tinnitus	Multiple times a day	Imbalance	Sound and pressure
Superior canal dehiscence	Seconds	Bone > air conduction, tinnitus, fullness	Multiple times a day	Autophony*, oscillopsia, imbalance	Sound and pressure

*Sx, symptoms; TIA, Transient ischemic attack. * Hearing one's own body sounds like eye movements, breathing, stepping, and so on*

Patients with vestibular neuritis experience severe persistent vertigo, nausea and vomiting. These cases respond very well to steroids. Although audiometric analysis was not conducted on the patient, the patient reported an episodic decrease in hearing. In cases of vestibular neuritis, hearing is usually normal and they don't have a tendency to become chronic as in this case [5,11]. The pathophysiology of MD is still not very clear [6,9,12,13]. It has been said that autoimmune phenomenon may be responsible for as much as 30% of the cases of MD and that early treatment with steroids may even limit the damage to the inner ear [4,12].

Diagnosis and management of MD is a complicated affair and there is a lot of heterogeneity in the treatment protocols used [14,15]. Good quality, robust clinical trials in MD are lacking and there have been conflicting reports about the utility of Betahistine, diuretics like Acetazolamide and salt restriction in the management of MD [14,16–20]. Nonetheless, they are commonly used in the management of MD. In our patient, the vestibular suppressant Cinnarizine was used for about six months when he came to us. It has been recommended that vestibular suppressants be used only, on an 'as and when needed' basis in acute vertigo because long term use can can hinder central compensation and prolong the recovery period [5].

The final resort in non-responders is ablative procedures like intratympanic aminoglycosides or surgery. The response rate for these procedures is around 40-50% and they may lead to permanent sensorineural hearing loss due to damage to the hair cells [14,21]. Intratympanic injections of steroids have also been used in non-responders and the response rate seen is around 30-50% [14]. Intratympanic injections are an invasive modality and require expert skill and training. Hence, they may not be feasible at the primary care level.

4. CONCLUSION

- Differentiation of central and peripheral vertigo should be the first step in evaluation of a patient with vertigo.
- This should be followed by a diagnosis of the specific cause of vertigo and its treatment.
- Vestibular suppressants should be used only on an 'as and when' basis, and not for a long time because it will delay recovery.
- In patients with severe and or refractory vertigo due to MD, a trial of systemic steroids should be given before starting any invasive or ablative procedures. Since autoimmunity plays an important role in the pathogenesis of MD, early steroids may help prevent damage and hasten recovery.

CONSENT

There is no data in the manuscript which reveals the identity of the patient in any way.

All authors declare that 'written informed consent was obtained from the patient for publication of this case report.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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