

DIZZCOVER VERTIGO UPDATE

VESTIBULAR MIGRAINE

VOL 2, 2019

CONTENTS

1. About the newsletter
2. Guest editorial
3. Issue in focus – Vestibular Migraine
4. Case review
5. Journal scan
6. Historical note – Robert Slater
7. Radiology quiz
8. Winners and answer for the previous radiology quiz

About the Newsletter

This newsletter is an outreach to the community of health professionals, who are interested in the scientific analysis and treatment of vertigo and balance disorders. It aims to provide a platform for education and sharing.

The current issue focuses on Vestibular Migraine.

We solicit feedback and would be happy to print relevant comments, case reports and journal reviews which will be duly acknowledged.

Kindly email them to
dizzcover@cyclopsmedtech.com or WhatsApp
to 9448480152.

GUEST EDITORIAL



Prosper Meniere, the French physician scientist was the first to realize the relation between Migraine and Vertigo, because he himself suffered severe Migraine. For decades there were ill-defined thoughts and hazy outlines to describe what was previously called Migraine -Anxiety Related Dizziness (MARD), Migraine-induced Vertigo or Migrainous Vertigo. In

the last few years, collaborative efforts of the International Headache Society and Barany Society have succeeded to reach a consensus on diagnostic criteria of Vestibular Migraine. Migraine is a neurological disease, considered a central vestibular disorder, yet it sometimes shows peripheral patterns of clinical findings. The real challenge for diagnosis of vestibular migraine is the fact that Migraine is very common, it can be present in association with other disorders such as Meniere's disease, BPPV, Epilepsy and the diversity of signs and patterns of nystagmus which could be found in patients having vestibular migraine. Vestibular migraine is still a diagnosis of exclusion, where audio-vestibular investigations as well as radiological studies should take place to exclude other peripheral and central vestibular disorders. In our vestibular clinic, it represents the second most common cause of vertigo after BPPV. Other clinical syndromes are closely related to vestibular migraine such as Paroxysmal Vertigo of childhood and motion sickness which are considered as precursors of vestibular migraine. Also, Mal De Debarquement syndrome is somehow appears to be related to Vestibular Migraine. On one hand, underestimation of the prevalence of Vestibular Migraine leads to unnecessary patients suffering for years. On the other hand, overdiagnosis without application of correct clinical criteria leads to missing other diagnoses. Right balancing is all what clinicians need while dealing with vestibular migraine.

Alfarghal Mohamad, MD, AuD
King Abdul Aziz Medical City-Jeddah

Issue in Focus: VESTIBULAR MIGRAINE

A. Introduction

Migraine is one of the common forms of primary headache. Each patient with migraine on this planet is a variant in itself. The term Vestibular Migraine (VM) is used for patients with past or current history of migraine, presenting with dizziness as the predominant symptom with or without headache.

B. Nomenclature

VM is a generally accepted term for this condition. The previous terms were migrainous vertigo, migraine-associated vertigo, migraine-associated dizziness, migraine-related vestibulopathy.

C. History

Despite the notes on association between episodic vertigo and migraine as early as 1873, VM remained a poorly defined entity until 1979, when Dr. Robert Slater a Neurologist described its features, using the term benign recurrent vertigo. The spectrum of VM however is still evolving and more clarity is being obtained through investigation and research.

D. Epidemiology

Migraine and vertigo are common neurologic complaints in the general population, each being more common in the presence of the other. The link therefore was initially suspected on the basis of epidemiology. VM can affect children as well adults. Females appear to be more affected than males.

E. Pathogenesis

The pathogenesis of VM is heterogenous and not yet completely understood. Various potential mechanisms have been proposed like stimulation of trigeminovestibular connections, migraine induced ischemia of inner ear, ion-channel defect and endolymphatic hydrops in migraine.

F. Clinical features

Vestibular symptoms commonly seen are spontaneous vertigo, induced by position, complex visual stimulus, head

motion, imbalance, dizziness, extreme sensitivity to motion. Typically, VM is spontaneous, positional or spontaneous transforming into positional.

The positional vertigo differs from BPPV, it persists for as long as the head position is maintained though the symptoms of vertigo and lasts for minutes to days at the most, unlike BPPV which may continue for weeks. Head motion intolerance is often associated, as is visual field movement provoking vertigo.

Migraine classically is a headache with or without aura though migraine related condition can be diagnosed without headache. The aura may be abnormal smells, lights or hallucinations. Vertigo is not an aura like symptom, as its duration is too long and may arise during or after the headache. Other auras may co-exist with vertigo.

Other symptoms which can occur are transient auditory symptoms like muffled hearing, ear pain and tinnitus, nausea, vomiting, prostration.

The history of vertigo is that it triggers specifically for food and emotional disturbances. Its description, duration, course and accompanying symptoms are the best way to decide whether the criterion described below are met. Childhood vertigo, food triggers, are often classic descriptors, but note that only a third of the vertigo patients that suffer from migraine have vestibular migraine as per strict criterion below. **Diagnostic Criterion for Vestibular Migraine (as per the International Headache Society and Barany society 2012)**

A. At least five episodes fulfilling criteria C and D

B. A current or past history of migraine

C. Vestibular symptoms of moderate or severe intensity, lasting between 5 minutes and 72 hours

D. At least half of the episodes are associated with at least one of the following three migrainous features



1. Headache with at least two of the following four characteristics:

- a) unilateral location
- b) pulsating quality
- c) moderate or severe intensity
- d) aggravation by routine physical activity

2. Photophobia and phonophobia

3. Visual aura

E. Not better accounted for by ICHD-3 diagnosis or by another vestibular disorder.

G. Differential diagnosis

The common differentials of VM are meniere's disease, BPPV, persistent postural phobic dizziness, brainstem ischemia, migraine with brainstem aura (Formerly Basilar migraine) and Vestibular paroxysmia.

H. Evaluation

There are no conclusive diagnostic tests. Vestibular testing usually reveals non-specific abnormalities.

I. Treatment

• Pharmacologic treatment

1. Treatment of acute attacks:

Vestibular suppressants can be used in attacks that last for more than 20-30 min.

Note: These are not treatments of migrainous headaches, but of vestibular disorders such as Meclizine, Dimenhydrinate, Droperidol, Promethazine, Prochlorperazine. Triptans are not routinely recommended unless the VM is accompanied by headache.

2. Preventive treatment:

- a. Avoidance of triggers
- b. Prophylaxis - indications for prophylaxis are severity, frequency and disabling nature of the attacks.

These, however, are more in line with the medication for classical migraine as suggested by the American Association of Neurology (AAN).

c. All the preventive drugs work reasonably well and should be selected based on the co-morbidities and concern for side effects. Various class of drugs which can be used for prevention are beta-blockers, tricyclic antidepressants, calcium channel blockers, some anti-epileptic drugs. The SNRI Venlafaxine can be used in VM with associated P3D.

d. Treatment of the co-morbid conditions like anxiety, P3D.

e. Physical therapy - It can be helpful in a subset of patients with interictal symptoms and comorbidities like P3D.

• Non-pharmacologic treatment

Lifestyle changes, stress reductions, diet etc.

J. Prognosis

The natural history of VM has not been well studied. Some studies show that in time the frequency of vestibular symptoms reduce and the cochlear symptoms increase.

K. Debatable and unresolved issues in VM

The debatable issues are the site of the lesion within the vestibular system in VM (Central vs peripheral), objective documentation of vestibular deficits in VM, differentiation between VM and Meniere's disease, best treatment for VM. The reasons behind the debatable issues are that the clinical features vary and overlap with other vestibular disorders, poor understanding of the pathophysiology, lack of specific tests and biomarkers, lack of standard nomenclature.



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MD, DM: Specialist
Neurologist, Aster clinic,
Dubai.

CASE REVIEW 1

History

A 31-year-old young female complained of episodic dizziness and persistent imbalance over the last 2 weeks. She had 2 such episodes of dizziness, since then each episode of dizziness lasted a few seconds or less than 2 minutes. These episodes were not related to change in head position or change in posture.

There was an associated mild nausea. There were no aural symptoms. Also, there was no difficulty in walking, speech or any indication of a visual disturbance. There was no h/o any drug intake. She revealed a past h/o positional vertigo 3 to 4 years ago. This would occur while lying down in bed and turning to either side. The duration of each episode was a few seconds. There was no related nausea or vomiting. There was a spontaneous resolution of symptoms in 2 weeks.

In addition, she had migraine since her school days. The headaches were unilateral, throbbing in nature. The patient couldn't tolerate bright light and loud sounds during the headaches. However, the dizziness/vertigo was not related in its timing to headaches. The headaches as well as dizziness were related to periods of stress and mental exertion.

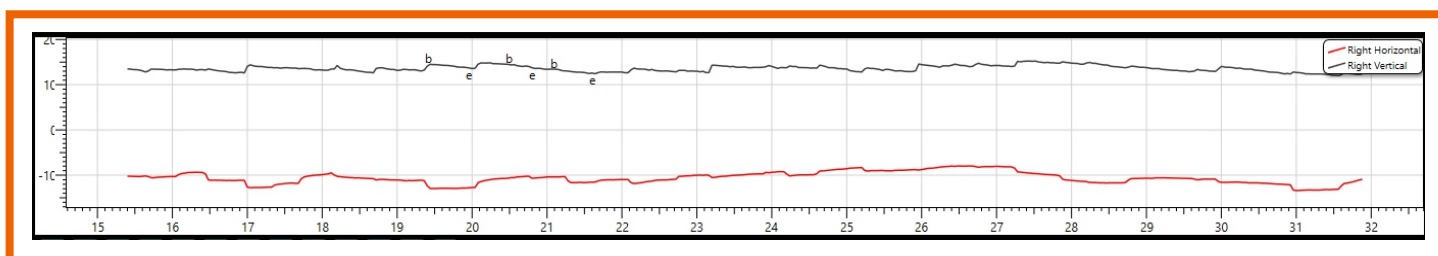
Clinical examination and Neuro-otology tests:

The ears were normal. Romberg's test and Unterberger's stepping test were normal. There was no gait abnormality. Pure tone audiometry revealed normal hearing in both ears.

Videonystagmography findings were as follows

1. A saccadic smooth pursuit
2. A low amplitude up-beating nystagmus on removal of fixation
3. A left beating horizontal nystagmus on gaze testing without fixation

An MRI of the brain was requested (in view of saccadic vertical smooth pursuit and up-beating nystagmus), which was reported as normal.



A provisional diagnosis of vestibular migraine was made. Acute treatment was not required, as the patient's symptoms were mild at the time of presentation. She was advised lifestyle modifications including avoidance of stress. Prophylactic Flunarizine was started in a dose of 5 mg. per day. The patient is asymptomatic since the last 1 month.

CASE REVIEW 2

History

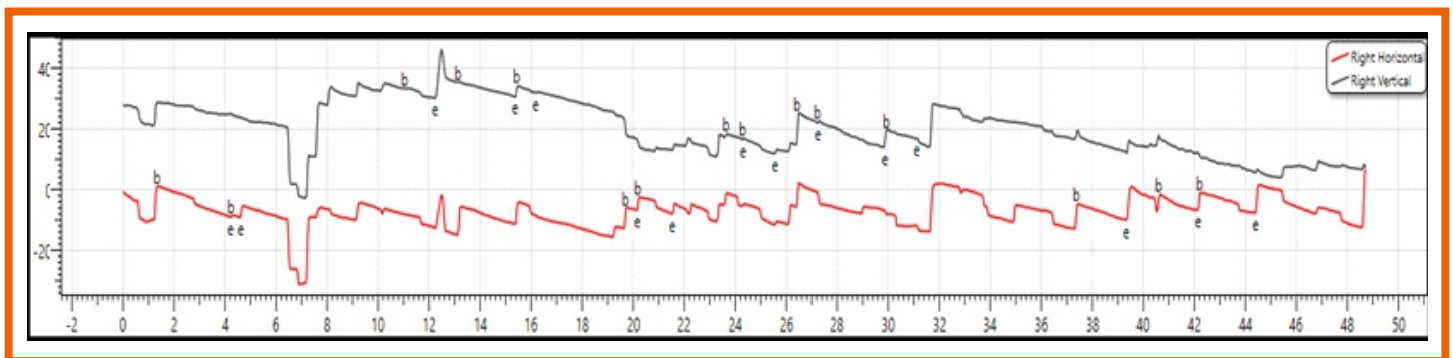
A 25 years young female patient, presented with a history of suffering from a severe rotatory vertigo, 3 weeks prior to coming to our clinic. The duration of the attack was 24 hours.

The vertigo worsened on head movement and while walking. She felt better at rest. She had associated symptoms of nausea, vomiting, sweating and photophobia. There was a bilateral ringing sensation in both the ears during the attack and a unilateral headache of a pulsating nature. There was no history of hearing loss or of fullness in the ears. She did not complain of any diplopia, dysarthria or numbness. She had similar milder attacks 4 times since then, interspersed with headaches. There was a strong past h/o recurrent headache with visual aura and photophobia. A relation to occurrence during menstrual cycle and times of stress could be drawn.

Clinical examination and Neuro-otology tests: The ears were normal. Romberg's test and Unterberger's stepping test were normal. There was no dysdiadochokinesia. There was no gait abnormality. Pure tone audiometry revealed normal hearing in both ears.

Videonystagmography findings:

- Smooth pursuit was saccadic.
- No spontaneous nystagmus with fixation.
- Spontaneous right beating horizontal nystagmus, and up-beating nystagmus, on removal of fixation, which did not enhance on high frequency head shaking.
- The head impulse test was normal. An MRI of the brain was requested (in view of saccadic smooth pursuit and up-beating nystagmus), which was reported as normal.



A provisional diagnosis of vestibular migraine was made. Acute treatment was not required, as the patient's symptoms were mild at the time of presentation. She was advised lifestyle modifications including avoidance of stress. Flunarizine given in a dose of 5 mg. per day



Dr. Avinash Bijlani,
ENT/ Otorhinolaryngology,
Madhaw Medical Centre.

Answers for the Radiology Quiz in the previous Newsletter Vol 1, 2018

1. What is the diagnosis in this patient?

Fistulous communication of the middle ear with the labyrinth, secondary to long standing cholesteatoma.

2. What is the site & side of the pathology?

From the HRCT it is clearly visible that there is a fistulous communication between the middle ear and the lateral semicircular canal (LSCC) on the right side. (www.cyclopsmedtech.com)

3. What is the management protocol for this patient?

Surgical exploration of the mastoid at the earliest to remove the underlying pathology and surgical repair of the fistula (using cartilage and bone pate is the author's preferred method, but others may have their own preferences). Using Oral Steroid pre and post-operative may help in reducing the labyrinthine inflammation thereby minimizing the chances of a sensorineural hearing loss.

4. Tips & Tricks for identifying Lateral Canal Fistula on Radiology

- Always requisition a High-Resolution CT Scan (sub 1 mm slices, to be precise). Since the LSCC is a mere centimeter in its entirety, missing out a fistula on a normal resolution CT scan is very common.

- Always ask for both axial and coronal cuts and if possible Multiplanar 3D Reformation.
- A dimpling of the compact labyrinthine bone over the LSCC should raise the index of suspicion for the presence of a fistula, especially in the background of the clinical symptoms and sign.
- The presence of a bony erosion over the LSCC on three consecutive slices on axial cuts is definite proof of a fistula.
- The fistulous communication could be bony and membranous. The presence of a circular hypointense shadow within the lumen of the LSCC usually indicates an air bubble with the canal, indicating more towards a fistula on the membranous canal as well. Chances of dizziness, vestibular signs and SNHL are more when the membranous canal is also compromise.

Winners for the quiz



Dr Chetana Naik
Consultant ENT surgeon
and Neurologist, Goa.



Dr Vinoth Manimaran
Senior Resident in
SRMC, Chennai.



Journal Scan

1) Is the Headache in Patients with Vestibular Migraine Attenuated by Vestibular Rehabilitation?

Sugaya N1, Arai M2, Goto F2.

Front Neurol. 2017 Apr 3;8:124.

Vestibular rehabilitation is the most effective treatment for dizziness due to vestibular dysfunction. This study aimed to compare the effects of vestibular rehabilitation on headache and other outcomes relating to dizziness, the psychological factors in patients with VM patients, patients with dizziness, tension-type headache and patients without headache.

METHODS: Our participants included 251 patients with dizziness comprising 28 patients with VM, 79 patients with tension-type headache, and 144 patients without headache. Participants were hospitalized for 5 days and taught to conduct a vestibular rehabilitation program. They were assessed using the Dizziness Handicap Inventory (DHI), Headache Impact Test (HIT-6), Hospital Anxiety and Depression Scale (HADS), and Somatosensory Catastrophizing Scale (SSCS) and underwent center of gravity fluctuation measurement as an objective dizziness severity index before, 1 month after, and 4 months after their hospitalization.

RESULTS: The VM and tension-type headache groups demonstrated a significant improvement in the HIT-6 score with improvement of the DHI, HADS, SSCS, and a part of the objective dizziness index that also shown in patients without headache following vestibular rehabilitation. The change in HIT-6 during rehabilitation in the VM group was positively correlated with changes in the DHI and anxiety in the HADS. Changes in the HIT-6 in tension-type headache group positively correlated with changes in anxiety and SSCS.

CONCLUSION: Vestibular rehabilitation contributed to improvement of headache both in patients with VM and patients with dizziness and tension-type headache, in addition to improvement of dizziness and psychological

factors. Improvement in dizziness following vestibular rehabilitation could be associated with the improvement of headache more prominently in VM compared with comorbid tension-type headache.

EDITORIAL COMMENTS: The study suggests that the pathophysiology of headache in Vestibular Migraine is similar to that which causes Vertigo. However we should note that the pharmacologic agents in the two conditions are not equally efficacious. Stricter application of the Guidelines of diagnosis will possibly help us in confirming these findings before accepting this as a treatment guideline.

2) Effects of Vestibular Rehabilitation in the Management of a Vestibular Migraine: A Review.

Alghadir AH1, Anwer S1.

Front Neurol. 2018 Jun 12;9:440.

Vestibular rehabilitation (VR) has been shown to be effective for many vestibular disorders.

This review focuses on the current evidence on the effects of physical therapy in the management of vestibular symptoms in individuals with a vestibular migraine (VM). The individuals with a history of a migraine tend to have a high incidence of vestibular symptoms with some or all of their headaches.

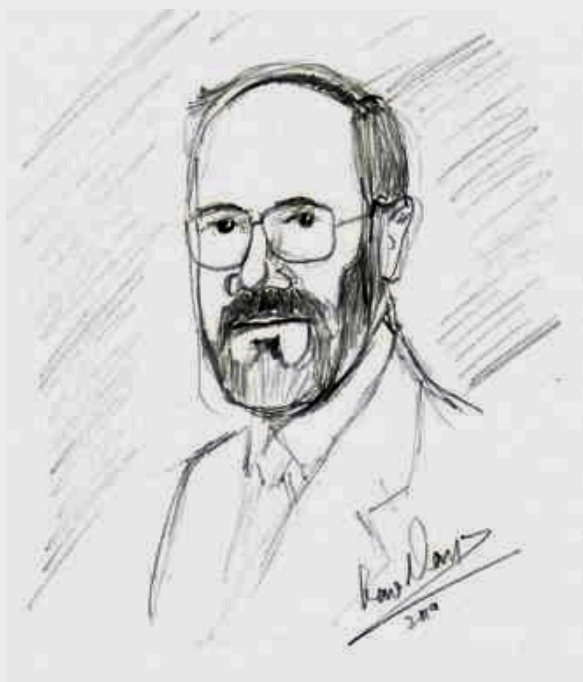
A total of six included studies investigated the effects of VR in the management of VM. The critical review form for quantitative studies was used to appraise quality assessment and risk of bias in the selected studies.

Previous studies validated the use of VR in the treatment of vestibular symptoms for individuals with a VM to include improved headache and migraine-related disability in patients with a VM. From the current evidence, it is difficult to provide conclusive evidence regarding the efficacy of VR to minimize vestibular symptoms in patients with VM. Therefore, more randomized controlled studies are required to make firm evidence on the effect of VR in reducing vestibular symptoms in patients with VM.

The future prospective, blinded, randomized controlled studies may help to isolate possible therapeutic effects of VR and other general effects.

EDITORIAL COMMENTS: This article reinforces the conclusion that the pathophysiology of Vertigo in Vestibular Migraine is more related to Vascular and autonomic dysfunction, than mere vestibular dysfunction.

HISTORICAL NOTE – Robert Slater



Benign recurrent vertigo

ROBERT SLATER

From the Division of Neurology, Delaware Country Hospital, and Department of Neurology, School of Medicine, University of Pennsylvania, USA

SUMMARY: Patients with recurrent vertigo in the absence of cochlear signs remain a diagnostic problem. The absence of a standard system of nomenclature further hampers the understanding of these disorders. The term benign recurrent vertigo is suggested as a useful term to characterise many of these patients. The disorder shares some of the features of migraine and seems likely to have a similar vasospastic aetiology.

RADIOLOGY QUIZ No. 2

A 65-year-old male patient with a pre-existing history of primary hypertension and dyslipidaemia, presented with a history of multiple attacks of very short-lasting rotatory vertigo and a feeling of falling back, for the last 18 months.

The vertigo spells were very short, lasting few seconds to a few minutes, the longest being vaguely described as probably 3-4 minutes. There were multiple attacks in a day, ranging from 8-10, with sometimes the patient losing count of the number of attacks throughout the day.

Most of the attacks were rotatory in nature, though the patient often had a feeling of falling back (retropulsion) and occasionally felt acutely dizzy for a short period when he suddenly turned his head to the right in the yaw axis e.g.: when called from behind by someone.

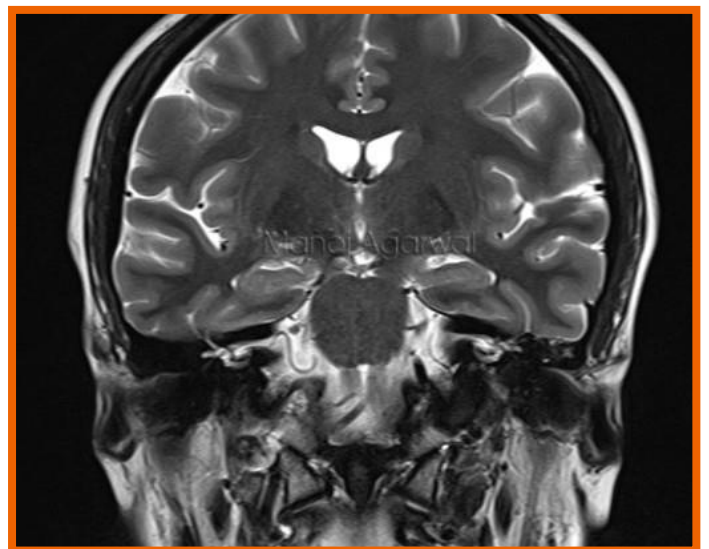
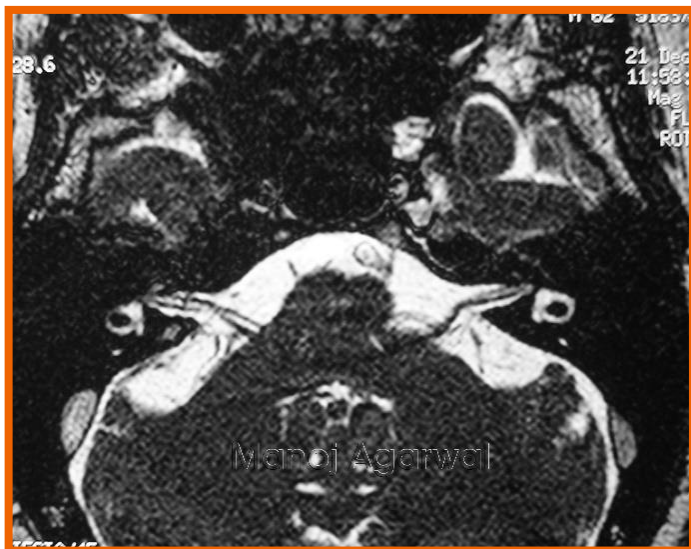
The patient also complained of a transitory right sided tinnitus, pulsatile in nature, while he felt dizzy, which was

not present otherwise. The tinnitus disappeared a while after the resolution of the episode of vertigo. The family members provided a history of the patient having some degree of a hearing problem, tending to watch the television louder and missing out some parts of a conversation, especially in a group. A specific history of the hearing worsening during the attack of vertigo was answered in the negative.

Clinical examination revealed a left beating nystagmus on hyperventilation. Rest of the neuro-otological examination was normal, though there was some evidence of myringosclerosis.

Pure tone audiometry showed evidence of a mild to moderate sloping sensorineural loss.

The patient underwent an MRI and the images are as below.





- 1) What is the diagnosis in this patient?
- 2) What is the site and side of the pathology?
- 3) What specific imaging modality/protocol would you requisition?
- 4) What criteria need to be fulfilled to diagnose this condition?
- 5) What is the management protocol for this patient?



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Consultant Otologist, Neurologist & Lateral Skull Base Surgeon, Kolkata.
Send answers to Email: dizzcover@cyclopsmedtech.com

The correct answers and names of the winners will be announced in the next issue.

Newsletter Editorial team

Dr Ravi Nayar

Dr Srinivas

Editorial Assistant

Ms Sanjana John

Invited contributors to this issue

Dr. Alfarghal Mohamad, Dr. Vishal Pawar, Dr. Avinash Bijlani and Dr. Manoj Agarwal.

Forthcoming issue on: **Role of VEMP In Vestibular Diagnosis**



Artist

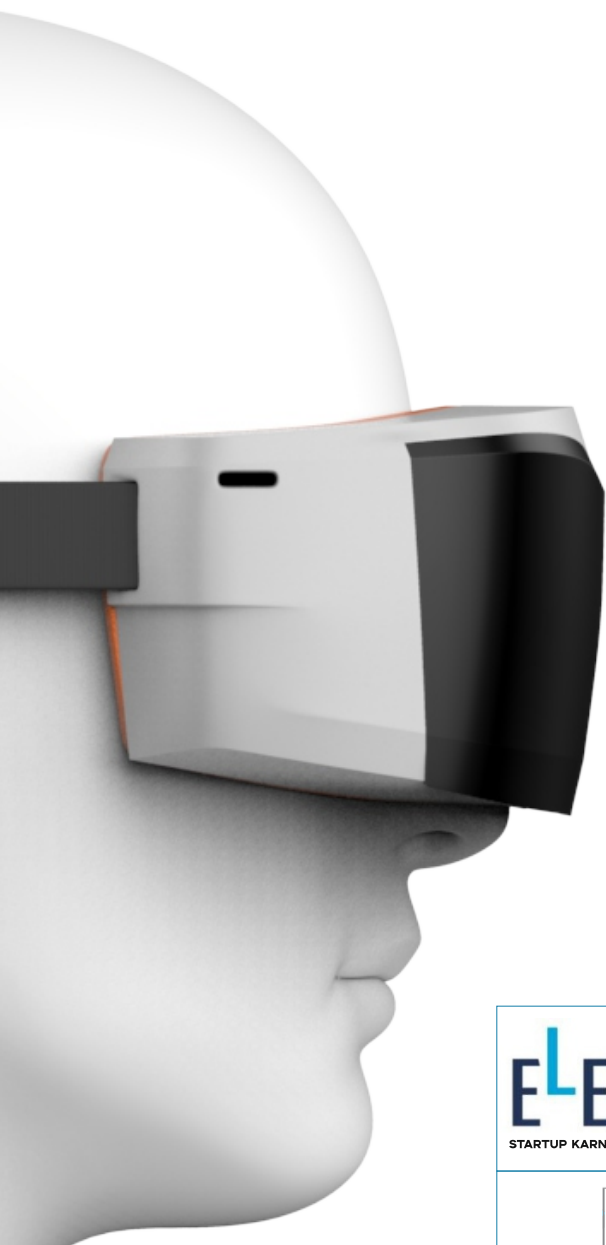
T M Sreenivasa Murthy

Year: December 2016. Acrylic on canvas. Measures 6*4 feet.

The painting was inspired by an award-winning photograph depicting the bull race “pacu jawi” from Indonesia, a race similar to the “kambala”, held in the South Kanara region of Karnataka.

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