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Case Report

A rare case of retinal migraine

Shrishti Samanta[1]*, Sanjay Kumar[2], Keshav K. Sinha[1], Rani I. Sinha[1]

[1]Department of Pharmacology, Patna Medical College, Patna, Bihar, India
[2]Department of Neurology, Patna Medical College, Patna, Bihar, India

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***Correspondence:**
Dr. Shrishti Samanta,
Email: samantashrishti@gmail.com

ABSTRACT

Retinal migraine (ophthalmic migraine, ocular migraine, anterior visual pathway migraine) is a rarely encountered medical condition, but with an interesting and complex clinical presentation. Retinal migraine is defined as repeated attacks of monocular visual disturbance including scintillations, scotoma or blindness associated with migraine headache. they can occur at the same time as migraine, headache and are sometimes seen in a patient with a prior history of migraine. They occur because of hypoperfusion of either the eye or the optic nerve. This is a different entity to typical migraine with aura, which involves the cerebral cortex and is associated with binocular visual phenomena. Here I am reporting a case of retinal migraine in a 22 years old female patient from the OPD of Department of Neurology, Patna medical College and Hospital, Patna.

Keywords: Hypoperfusion, Retinal migraine, Scintillations, Scotoma

INTRODUCTION

Retinal migraine was introduced by Galezowski in 1882, The term "ophthalmic megrim" is used to describe a permanent loss of vision associated with headache.[1] The official term "retinal migraine" was introduced in 1970 by Carroll and the authors have used it to describe a multitude of monocular visual symptoms, including events without the associated headache and those resulting in persistent visual loss.[2] The evolution of terminology accepts retinal migraine, ophthalmic migraine, ocular migraine, and anterior visual pathway migraine as the same notion, but all of these should not be confused with ophthalmoplegic migraine, which is manifest by ophthalmoplegia and unilateral headache.[3] A retinal migraine is a rare disorder. Most commonly patients present in their 20s with retinal migraine but can be found between 7 years to 40 years of age. 29% of retinal migraine patients have a history of migraine attack and 50% have a family history of migraine headache.[4] Pathophysiology of migraine still remains unclear but release chemical factors such as substance P, nitrous oxide, calcitonin gene related peptides leading to plasma extravasation, neurogenic inflammation, vasodilation can be a possible mechanism.[5]

Photophobia in the migraine may start by hypersensitive excitation effect on light sensitive thalamic neurons. Photophobia is aggravated by light intensity, dependence, when the eyes of the patients are exposed to different wavelength of visible light. Funduscope and fluorescence angiogram expose a delayed killing or occlusion of central retinal artery and its branches with either normal ciliary circulation or irregular/discontinuous coral defects and capillary leakage flux.[6] Symptoms when aura is present: flashing, sparkling and twinkling of lights (scintillation). Non-aura: blind spot, a partial loss of vision, temporary blindness, scotoma. Retinal migraine attack begins with mono blind spot, a partial loss of vision, temporary blindness, scotoma. Retinal migraine attack begins with monocular visual symptoms, afterwards when relaxation

time of blood vessel is manifested, blood flow resumes and sight returns.

Table 1: Must have at least two attacks and fulfill criteria from Column A and Column B.

Column A	Column B
Aura with fully reversible monocular positive and/or negative visual phenomena confirmed during attack by at least one of the following	Two or more of the following
Clinical visual field examination. Patient's drawing of a monocular field defect	Aura spreads gradually over 5 or more minutes Aura symptoms last 5-60 minutes Headache occurs during or within 60 minutes of aura

CASE REPORT

A 22 years old female patient visit OPD department of neurology medical College, and hospital Patna with the complaints of haemicranial headache associated with blurred vision, with negative symptoms (for 30 mins) vomiting, photophobia, and phonophobia. She reported headache episode of one attack in every three months, which persist for 3 to 4 days. She got her last attack with monocular aura 1.5 years back. She also added as during her headache days her daily life activities got hampered with visual analog score (VAS) of 9. She has documented precipitating factor as tea, coffee and chocolate. She has got positive family history of her father and grandmother.

Further investigation further were done with MRI with MODSA sequence, Fundoscopy, complete blood count (CBC), liver function test (LFT), kidney function test (KFT), lipid profile, thyroid function test, serum electrolytes with magnesium levels.

The patient was advised tablet propranolol 40 mg at bed time daily, tablet flunarizine 5 mg at bedtime, tablet naxdom 500 when required. Patient was asked to start medication and review with reports and after 4 weeks. Patient came with reports after 3 days and were found normal.

MR angiogram done on TOF and 3DMIP reconstruction shows normal circle of will is and its major vascular branches. No significant narrowing or focal aneurysm. No abnormal enhancement noted on post contrast study. Fundoscopy was normal in non-attack days.

Patients visited to OPD after 3 weeks with a headache associated with blurred vision in right eye. No vomiting with VAS of 6. Fundoscopy was done during the attack constriction of retinal vessels was seen

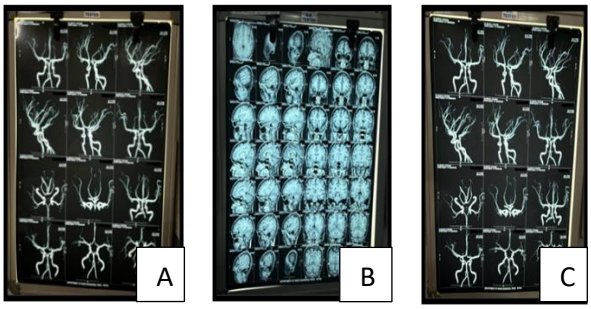

Figure 1 (A-C): MRI with MODSA sequence images.

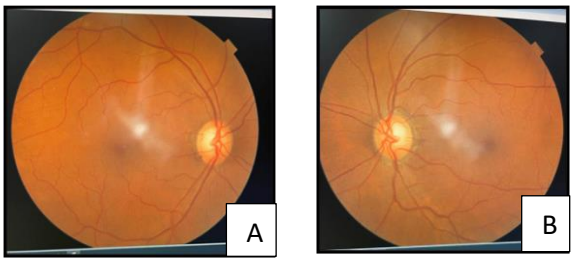

Figure 2 (A and B): Fundoscopy image of Right and left eye on non-attack days.

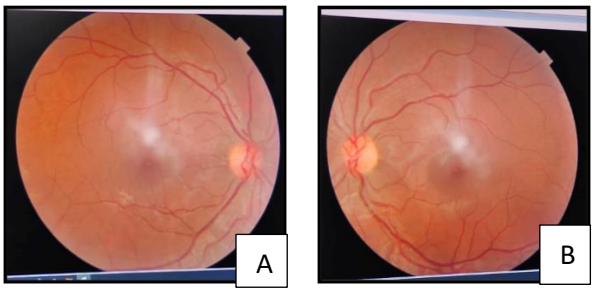

Figure 3 (A and B): Fundoscopy image of Right and Left eye during attack showing constriction of vessels.

Patient was symptomatically relieved. Patient was advised to continue medication. Patient was asked to come for follow up every 2 months. Patient visited after 2 months with 4 headache episodes with no associated symptoms and blurred vision with VAS of 6. After 4th month the headache episodes were decreased to 1 with no associated symptoms with VAS of 3. After 6th month of medication, she has not experienced the attack.

DISCUSSION

Retinal migraine occurs when blood vessels of eyes suddenly narrow, restricting the blood flow. It can occur due to stress, caffeine, alcohol, dehydration, and genetic factors. Retinal migraine patients present with wide range of symptoms such as flashing of lights and scintillating scotoma while the negative symtoms are black shaded white areas of different sizes of scotomata.[8,9] In the present case negative symptoms were present.

Cortical neuronal hyperactivity followed by spreading depression is considered to be the mechanism of typical migraine aura and persistence of this phenomenon is the most likely cause of prolonged aura without infarction.[10] Every migraineurs should keep a headache diary to know the causes and headache episodes. In the above case patient experienced migraine with monocular aura for 30 minutes fulfilling the ICHD-3 criteria of retinal migraine. But there have been patients reported where migraine and monocular aura duration is more than 60 minutes. James et al reported a case of 36 years old male with the history of migraine accompanied with monocular visual defects for upto 2 days in either eye with headache.[11] Mee et al reported a 34-year-old female migraineur without a conventional visual aura who presented with a persistent monocular visual field defect of at least 1 month related to attack of migraine.[12] Here, patient has got positive family history. Her grandmother experienced same symptoms when she was of her age. She has also documented headache episode after intake of caffeine. Patient showed significant improvement with tablet propranolol and tablet flunarizine.

CONCLUSION

Retinal migraine is a rare, but increasingly recognized clinical condition. It is a cause of transient, monocular, vision, loss, and uncommon, during prolong periods of hypoxia, it can lead to permanent loss of vision. It has been seen in previous researches, and in this case, also that renal migraine response well to the prophylactic treatment. But it is also important to attempt to change the lifestyle factors that precipitate this condition.

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