Migrainous Etiology of a Unilateral Nasal Hemianopia Visual Field Defect

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ABSTRACT

Patients who suffer from migraine headaches frequently visit an optometrist's office with a chief complaint of headache or visual aura symptoms. It is less well known that migraine patients can experience a transient loss of their visual field prior to or during a migraine attack. These visual field losses are measurable with optometric visual field testing, and have been reported to manifest as various types of defects. An important component of visual field loss with a strictly migrainous etiology is the complete reversibility of the loss.

A 53-year-old female patient presented to an optometrist's office with a chief complaint of acute migraine. Visual field testing revealed a unilateral nasal hemianopia. The patient was sent for an urgent CT scan to rule out an intracranial pathology such as aneurysm, malignancy, or ischemic event. All imaging was negative, and visual field testing after resolution of the migraine episode was essentially clear in both eyes. This case report introduces the current theories regarding the pathophysiology of migraine headache components, and explains why it is important for optometrists to understand them. It also emphasizes the critical role that optometrists can play in the management of patients suffering from migraines.

KEY WORDS:
migraine headache, visual fields, aura, scotoma, ischemia

INTRODUCTION

Migraines are a highly prevalent form of primary headache, affecting an estimated 15% of the general population.[1,2] They disproportionately affect females at a ratio of 3:1.[2] Migraines can be differentiated from common tension-type headaches through a thorough investigation of associated symptoms. Migraine headaches will often worsen with routine physical activity and be accompanied by nausea and/or moderate to severe photophobia and phonophobia.[1]

Migraines can present in two major forms: with and without an aura. Aura is defined as symptoms of repeated perceptible attacks of the central nervous system that are fully reversible.[1] Visual aura, the most common form of aura, can include positive visual disturbances such as the perception of zigzag lines in the bilateral temporal vision or negative visual disturbances in the form of scotomas.[1] These aura symptoms are often prodromal and precede the onset of a migraine. It is currently being debated whether the visual field loss that can occur during migraines should be classified as visual aura, or whether it should be its own separate entity due to a suspected difference in pathophysiology between the two.[2-6] In either case, these visual disturbances, positive or negative, often lead a migraine patient to an optometrist's chair.

Reported visual field defects from migraines have been extensively documented and include nasal steps, arcuate defects, altitudinal defects, constricted fields, homonymous defects, and total field loss.[4,5,7,8] It is important for optometrists to conduct a thorough investigation of all visual field loss to ensure no underlying pathology, as migrainous visual field loss can closely mimic symptoms of other, more concerning, conditions.[9,10] Furthermore, closer follow-up management of patients with migrainous visual field loss is indicated, as migraine recurrence can actually increase the risk of concerning complications including retinal artery occlusions, cerebral infarctions, and glaucoma.[9-11] This paper reports a case where a unilateral nasal hemianopia visual field defect in a middle-aged female, almost perfectly respecting the vertical midline, was completely reversible and existed with no underlying pathological cause; it was eventually diagnosed as a migrainous etiology. The role optometrists play in the management of patients who regularly suffer from migraine episodes is also discussed.

CASE REPORT

A 53-year-old female patient presented with chief concerns of a severe constant headache, moderate to severe photophobia and phonophobia, and a sore neck, with an onset of one month previously and persistently worsening. The headache pain was reported to be throbbing and located behind her eyes. The patient reported no visual disturbances apart from photophobia. The patient reported mild symptom relief with hair pulling. This was the patient's fourth presentation to the clinic for these concerns, and during the previous three examinations, no ocular cause had been found for the reported symptoms. A case history revealed a self-reported history of iritis, but no signs of ocular inflammation had been present in any of the previous examinations. The patient had been counselled at all previous appointments to return to the clinic or go to the emergency room should her symptoms change or worsen. The patient's current medications were quetiapine fumarate, sertraline HCl, amoxicillin trihydrate, and an inhaler. There was no reported history of previous ocular trauma, and no pertinent underlying medical conditions such as hypertension or diabetes were known.

Entering unaided visual acuities were 20/300 (6/90) in the right eye (OD) and 20/30 (6/9) in the left eye (OS). For comparison, at the initial visit, visual acuities had been 20/300 (6/90) OD and 20/60 (6/18) OS, and pinhole visual acuities had been 20/40 (6/12) OD and 20/25- (6/7.5) OS. Auto-refraction results at the exam in discussion were +0.25/-5.75x006 OD and -0.25/-2.00x170 OS. Cover test revealed orthophoric ocular alignment. Extraocular motilities could not be assessed as the patient reported extreme dizziness with eye movement. Pupils were equally round and reactive to light, and no RAPD was present. No ptosis or lid retraction was present. Intraocular pressures were 18 mmHg OD and 16 mmHg OS by iCare tonometry. Slit lamp examination was difficult due to the patient's extreme photophobia, but revealed deep and quiet anterior chambers OU and anterior chamber angles open by Van Herick assessment OU. Fundus examination revealed healthy optic nerve heads OU with no presenting edema.

Humphrey visual field testing by a 30-2 SITA Fast method was remarkable for a unilateral right nasal hemianopia with acceptable reliability (Figure 1). The patient was tentatively diagnosed with a suspected neurological abnormality of unknown origin resulting in this visual field defect. Differentials of the neurological abnormality included an aneurysm, neoplasm, or ischemic event affecting the right temporal fibres of the optic chiasm. The patient was sent to the local emergency department for a same-day urgent CT scan of the head and orbits to rule out intracranial pathology. The CT scan revealed no acute pathology. The patient was given metoclopramide, a fluid bolus, and acetaminophen for the migraine symptoms.

The patient returned two weeks later for a full eye examination. Case history revealed that symptoms were much improved. Best corrected visual acuities were 20/25+ (6/7.5+) OD and 20/20 (6/6) OS. The pupils, intraocular pressures, anterior segment ocular health, and posterior segment ocular health were all within normal limits. Repeat visual field testing with acceptable reliability was essentially clear OU (Figure 2). The patient was diagnosed with a transient visual field defect of migrainous etiology, and no further follow-up was conducted.

DISCUSSION

Patients experiencing migraines may frequently present at an optometrist's clinic to seek relief from the visual symptoms that can accompany a migraine. The most common type of aura, or reversible central nervous system symptoms that precede a migraine, is visual aura.[1,3,12] Visual auras start in the central visual field and extend peripherally, almost always respecting the vertical midline and existing symmetrically in both eyes.[3] These aura symptoms typically last between 5 and 30 minutes before vision is restored.[3] Positive visual disturbances have a zigzag or serrated appearance scintillating at the edge of the aura, and can appear either in colour or black and white.[3] Visual aura can also present as negative visual disturbances, or scotomas, that appear in a similar fashion as the positive

visual aura.[3] These negative aura symptoms are separate from the transient visual loss that can occur during a migraine attack and can cause measurable visual field defects, which may be described as either a graying of vision or a complete loss of vision in a portion of the visual field.[3] While the pathophysiologies of the three components of a migraine (the headache itself, the preceding visual aura, and the concurrent visual field loss) are still being debated, current theories suggest that they each have a different mechanism, all of which are important to understand for the optometric management of patients with migraines.

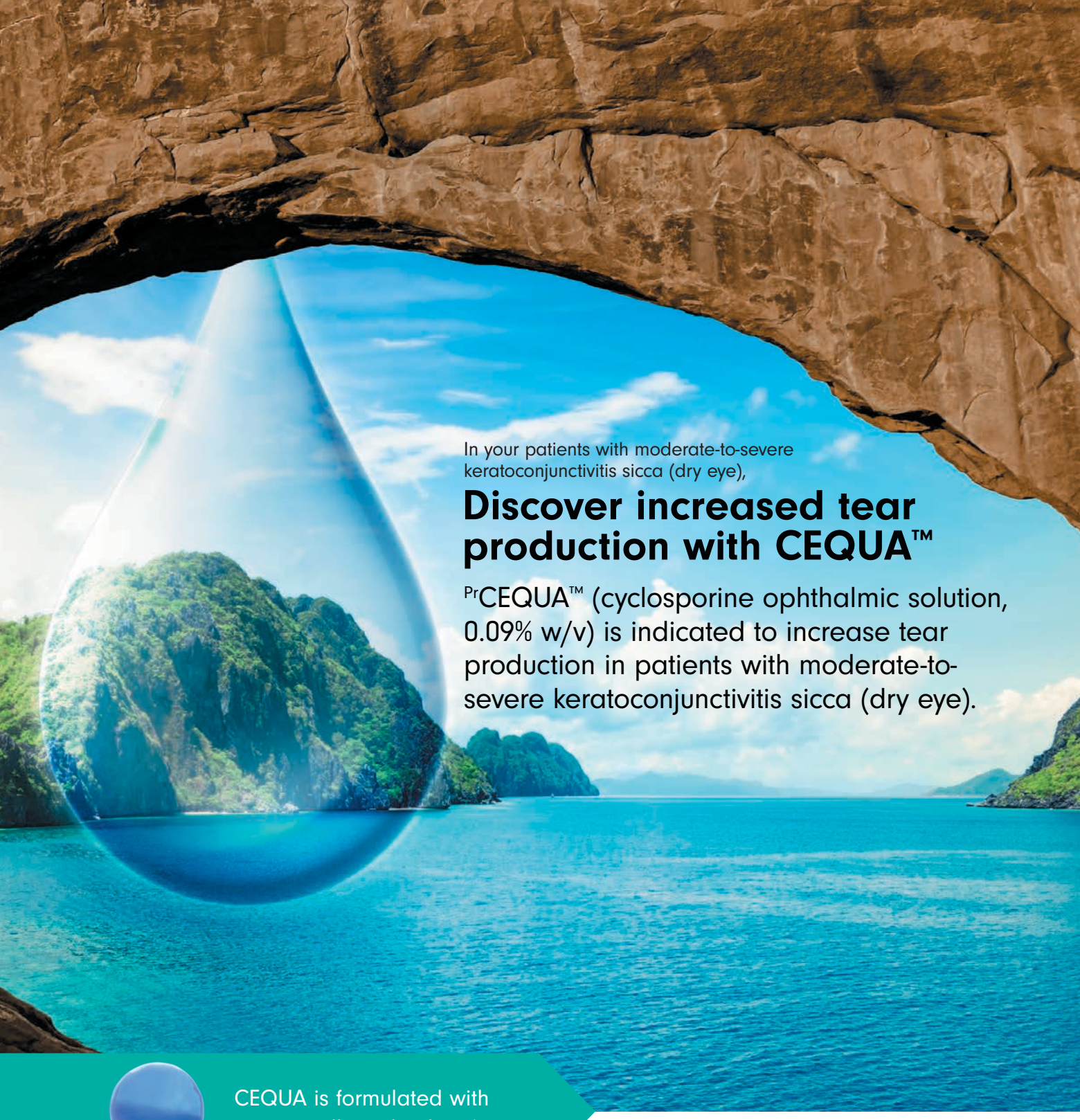
Different theories have been proposed to explain the pathogenesis of migraine headaches. It is currently accepted that trigeminovascular neurons, whose afferent fibres innervate the meninges and its vessels, are activated first.[2] The mechanisms of this activation are debatable; some reports conjecture a peripheral activation from structures such as extracranial arteries, some claim that there is a dysfunction in the brainstem neurons, and others theorize that "migraine triggers" play a role in this activation.[2,13-16] These migraine triggers include anxiety, stress, fatigue, dehydration, certain odors, certain foods, alcohol, smoking, glare, certain neck movements, and certain phases of a patient's menstrual cycle.[17] It is now argued that these triggers are self-proclaimed and exist only as an artifact of recall bias, as studies that sought to induce migraines in patients using their self-perceived triggers revealed a low success rate.[2,15,18] Through whichever means activation occurs, vasoactive peptides are released once these neurons are activated, and this induces local inflammatory responses.[2] Second-order neurons in the brainstem and third-order neurons in the thalamus are activated, and finally nociceptive impulses travel to the somatosensory and cortical areas that are involved in pain perception.[2]

The pathogenesis of visual aura in a migraine is thought to occur through a different mechanism than the headache itself. This is evidenced in neuroimaging of patients who experience migraine with aura, which demonstrates certain hemodynamic changes that are not present in patients who experience migraine without aura[2]. The current theory regarding the physiology of migrainous visual aura is that it arises through "cortical spreading depression" (CSD), a wave of mechanical or chemical neuron depolarization that self-propagates slowly across the cerebral cortex, producing hyperexcitation followed by suppression and resulting in cerebral hypoperfusion.[2,3] This is further supported by the fact that the cortical regions that are suspected to be involved in migraine aura, particularly the motion-detecting dorsal stream and the line orientation selective primary visual cortex, correspond with the hypoperfusion and CSD seen in fMRI studies in animal models.[2]

The reversible, transient visual field defects that present in some migraine patients also have a debated etiology. Older hypotheses theorized a pathophysiology similar to that of visual aura, where changes in cerebral perfusion and neuron depolarization played a major role.[6] However, the minority of case presentations of migrainous visual field loss are consistent with a cerebral locus, as few are homonymous or bitemporal.[6] Unilateral visual field deficits are more commonly reported, suggesting a pre-cerebral locus as the underlying etiology of field loss.[6] It has been suggested that subtle vascular anomalies in the pre-cortical peripheral vasculature may be present in these patients, leading to episodes of mild transient ocular ischemia and loss of the visual field.[6]

A few important points and differentials should be discussed in terms of defining the present patient's case. Because migraine aura is thought to have a cortical locus that would result in bilateral visual disturbances, migraine visual field loss is believed to have a pre-cerebral locus that could result in unilateral visual effects. Thus, the solely monocular visual field defect in the present case is not thought to be a result of migraine aura, but rather a result of a temporary migrainous infarct of the peripheral vasculature.[2,6]

Another differential diagnosis would be a so-called "retinal migraine". While the term retinal migraine is commonly used by optometrists interchangeably with the term visual aura, these are two very different conditions. Retinal migraines are repeated attacks of fully reversible visual disturbances occurring monocularly during a migraine headache, and are very rare.[1] Other causes of monocular visual disturbances and transient vision loss should be thoroughly investigated prior to making a diagnosis of retinal migraine.[1] While the disturbances in retinal migraine can include positive scintillations or negative scotomas, the patient in this case was not aware of any visual disturbances and the visual field loss was only made evident after Humphrey Visual Field testing; because of this and the fact that her field defect was vertical, we concluded that the visual field loss was a result of her migraine rather than an episode of retinal migraine. Finally, the well-accepted typical migraine definition applies to symptoms that last 4-72 hours.[1] Because the present patient had symptoms for nearly a month, it may be more appropriate to define her diagnosis as status migrainosus, which is a migraine complication and is defined as a headache attack that is both debilitating and lasts for more than 72 hours.[1] Status migrainosus can be caused by medication overuse. Thus, a further case history into the patient's alcohol and substance use should have been conducted.[1] Chronic migraines



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are defined as migraine headaches that are present on 15 or more days per month, for more than three months.[1] Though further follow-up for at least two more months is required before making this diagnosis, it is possible that the present patient may eventually be diagnosed as chronic migraine.

The role of optometrists in migraine management can be separated into three critical areas: proper in-office testing and counselling, prompt referral for additional systemic management, and appropriate follow-up management of the increased risk of ischemic complications.[11,19,20] A thorough in-office ocular examination with appropriate referrals must be performed to rule out other causes of the visual symptoms. Christakis et al.[9] presented a 60-year-old male with a strong history of migraines; migrainous right inferior visual field loss was diagnosed instead with a right superior hemiretinal artery occlusion. The authors proposed that the multiple recurrences of consistent right inferior migrainous visual field loss suggested a vulnerability to occlusion by vasospasm of the superior retinal vasculature of the right eye, and that the numerous migraine episodes themselves created vasospasm episodes leading to the arterial occlusion.[9] Similarly, Bylund et al.[10] presented a 49-year-old male with a significant history of migraines; migrainous left inferior homonymous visual field loss was diagnosed with a cerebral infarction of the right occipital lobe. He presented to the emergency department only after his visual field loss did not resolve as quickly as usual during one of his migraines, resulting in delayed diagnosis of his stroke.[10] The authors suggested that his multiple migraines and thus multiple ischemic episodes may have contributed to his stroke.[10]

An additional concern for optometrists is the similarity of the symptoms of visual aura and retinal detachment. Proper counselling of all migraine patients on how to differentiate these two conditions is critical, as both the treatment and urgency of the conditions vary greatly. Furthermore, if there is any concern for an intracranial pathology such as aneurysms, malignancies, or acute ischemic events as the cause of a patient's symptoms, prompt referral for neuro-imaging should be undertaken by the optometrist prior to making a working diagnosis of migraine.

Optometrists should also feel an obligation to refer these migraine patients to their general physician for further and continued migraine management, including acute or preventative pharmacological therapy.[2] Preventative therapy is doubly important as it aids in the prevention of patient discomfort and in prevention of the ocular ischemia that has been suggested to occur during migraine episodes.

The importance of consistent optometric follow-up management of migraine patients is evidenced in the increased risk of ischemic conditions seen in these patients, including the retinal occlusions and cerebral infarctions

discussed in the cases above.[9,10] More prevalent and concerning is the increased risk of normal-tension glaucoma (NTG) in these patients.[11,19,20] Migraine episodes are a known risk factor for NTG, as they cause a temporary decrease in ocular blood flow and temporary episodes of ischemia.[11] This is further evidenced in the disproportionate number of females affected by both migraine episodes and NTG[11]. The current pathophysiology theories discussed above highlight that ischemic events appear to occur more often in patients who experience visual field loss with their migraines. This risk assessment justifies the close monitoring of patients with migraines, especially those who also experience visual field loss, with glaucoma evaluations. It also emphasizes the role that preventative migraine therapy can play in reducing ischemic events and lowering the risk of NTG.

Though relatively complete management was undertaken in the present patient's case with thorough optometric and neuro-imaging investigation through a CT scan, there are important shortcomings in this patient's management that must be addressed. Firstly, a more thorough case history regarding the headache itself should always be conducted, including specific questions on the location of the headache, the pain severity, whether the onset was sudden or gradual, the timing of the onset, whether the headache changed over time, any triggering or relieving factors, and whether aura symptoms are present. Secondly, there should have been an increased focus on pertinent negatives such as temporal artery tenderness, limitations of eye movement, eyelid ptosis, and lid retraction to rule out the conditions of giant cell arteritis, orbital apex or pituitary tumours, a partial third nerve palsy or Horner's syndrome from an intracranial tumour, and compressive optic neuropathy from thyroid eye disease, respectively. Colour vision testing is pertinent in these cases to rule out optic neuritis, and should have been conducted at the initial examination and at all subsequent examinations. Specific questions regarding the patient's lifestyle, such as the patient's blood pressure history, smoking history, alcohol and substance use, diet, and sleep history, should have been included in the case history of all appointments. This patient was assessed in a remote, rural practice where access to further, more complex investigations was limited. Thus, the decision was made to keep the patient under optometric care after her negative CT scan. However, in an ideal situation, the patient should also have been referred for an ultrasonic duplex Doppler exam of the carotid artery and a neuro-ophthalmologist appointment with an MRI scan. The carotid artery Doppler would rule out calcification of the right internal carotid artery, a possible cause of a unilateral nasal hemianopia visual field defect through ischemia of the right temporal fibers of the optic chiasm.[21,22] An MRI scan with contrast of the optic chiasm is essential in cases of hemianopia visual field defects even when a CT scan is negative, as the concern is a compressive lesion of the optic chiasm, and pituitary-region tumours can be missed by CT scans.[23]

This discussion also demonstrates that the present patient's optometric management is far from over. It is recommended that optometrists follow-up their migraine patients closely, watching for consistent visual field loss that could suggest vulnerable areas of the eye or visual pathway, referring for further testing when a migraine symptom is not consistent with past episodes, and ensuring proper preventative treatment to decrease the ocular ischemic episodes brought on by migraine episodes. It is important to ensure that patients who suffer from migraines do not "slip through the cracks," as the implications of incorrectly assuming a mere migraine episode can be devastating to the patient's vision and overall health. ●

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Figure 1: *30-2 SITA Fast Humphrey visual field test results in a 53-year-old woman during an acute migraine attack, revealing a unilateral right nasal hemianopia.*

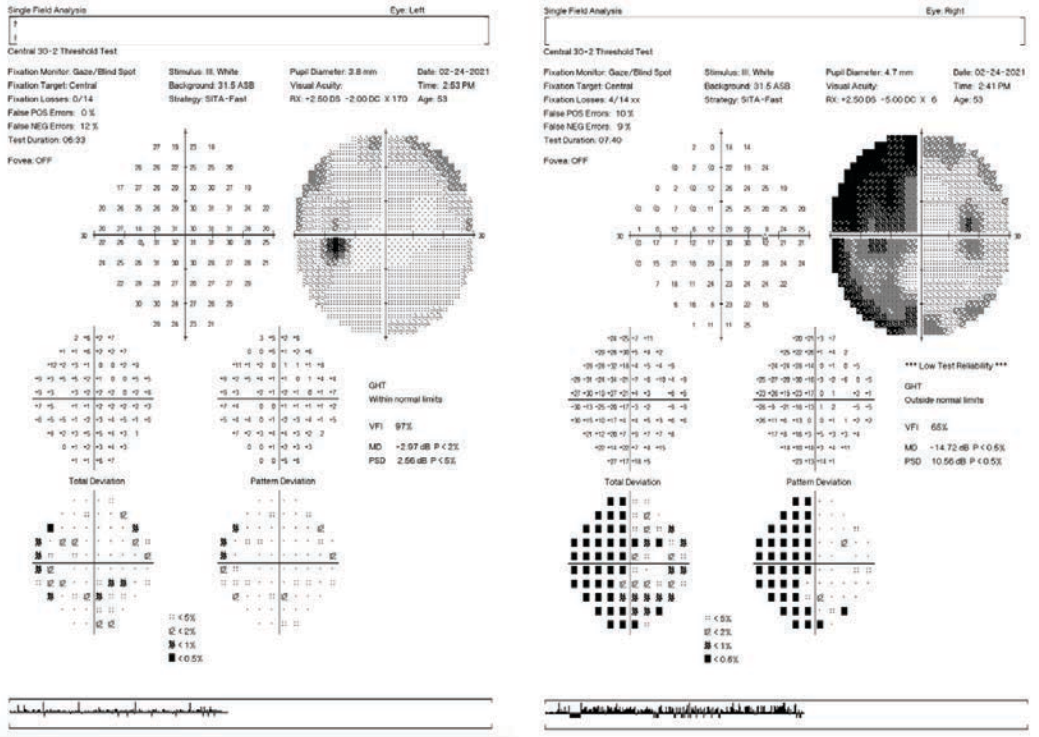
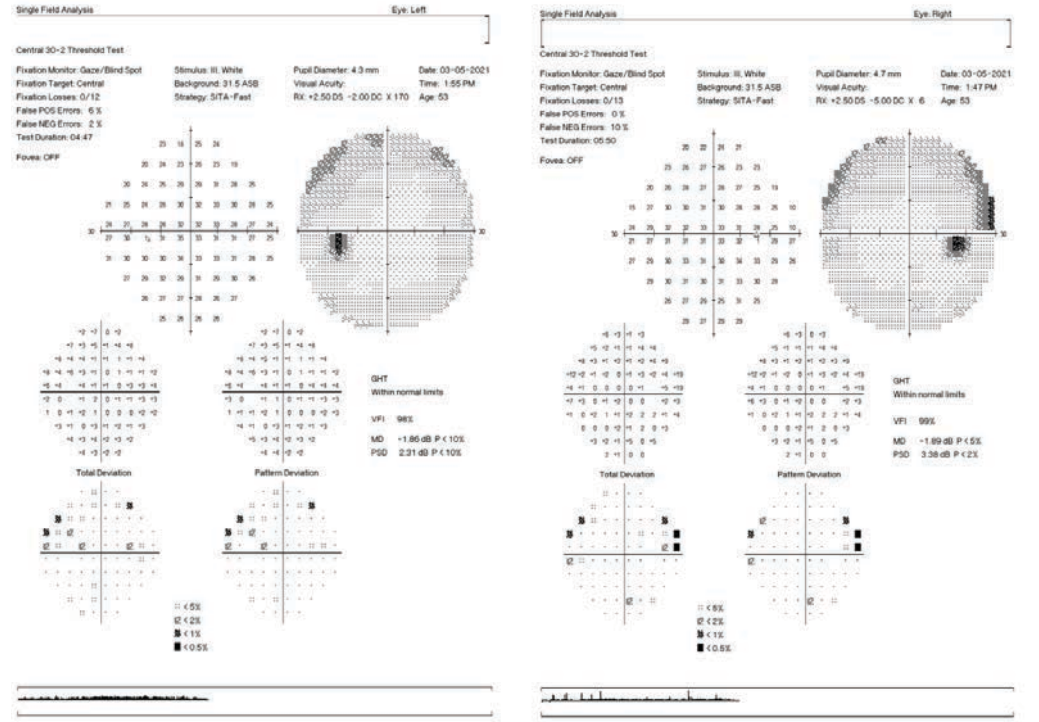


Figure 2: *30-2 SITA Fast Humphrey visual field test results in a 53-year-old woman two weeks after an acute migraine attack, revealing the disappearance of the previously present unilateral right nasal hemianopia and essentially clear bilateral visual fields.*



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