American Academy of Optometry: Case Report 3

A Case of Prolonged Visual Field Depression in Migraine with Aura

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Abstract:

Patients with a migraine headache may show visual field defects during and shortly after the migraine. These defects may be the result of transient ischemia and re-perfusion resulting in oxidative stress to the optic nerve much like the proposed pathogenesis for normal tension glaucoma, which has been linked to migraine sufferers. It is therefore prudent to inquire about migraine history during routine eye exams and to run visual fields and RNFL OCTs on migraine patients to monitor for and address any progressive nerve damage.

Keywords:

Migraine with Aura, Migraine, Headache, Visual Field Loss, International Headache Society, Normal Tension Glaucoma

Introduction

Headaches are commonly experienced by over 90% of individuals at least once during their lifetime.[1] A migraine is a common primary headache disorder characterized by unilateral, pulsating pain of moderate or severe intensity, aggravated by light, sound, and/or routine physical activity.[2] The International Headache Society identifies two major subtypes, migraine with aura and migraine without aura. Eye care professionals frequently evaluate headache complaints, especially when the pain reaches the orbit.[3] Although local eye diseases such as iritis, scleritis, angle-closure glaucoma, as well as uncorrected refractive error can present with a primary complaint of a headache, most headaches are not secondary to ocular causes.[3] Therefore, in addition to a complete ophthalmologic examination, a neurological screening of cranial nerves, mental status, gross motor function, balance and reflexes should be evaluated to rule out more ominous intracranial processes.[1,3]

Case Report

A 28-year-old African-American male complaining of headache and left visual field loss in both eyes was referred to optometry from the family health clinic on January 4, 2017. The patient reported his symptoms began on January 2, 2017, while driving. He initially noticed "heat waves" appearing on the lower left side of his vision. He pulled over on the side of the road hoping the symptoms would go away. Half hour later, he experienced a severe headache behind his left eye that eventually settled on the right, posterior side of his head. The headache was described as constant, pulsating, 8/10 pain, worse when sitting still, lying down, and in bright conditions. The patient also reported severe dizziness and nausea when he tried to lie down to sleep the past two nights. The patient did not recall any triggers that could have induced the symptoms. The patient denied recent head trauma, and reported no changes in diet, medication, or stress level. The patient reported the visual disturbances had persisted throughout the past two days, but had improved slightly at the time of the visit. The patient denied prior occurrences and personal and family history of migraine headaches.

The patient's medical history was significant for enlarged heart with a weak left ventricle, systemic hypertension, allergic rhinitis, anxiety and chronic posttraumatic stress disorder (PTSD). He had a prescription for 5mg amlodipine besylate, 1 tab a day, but reported he had not been taking any medications aside from Excedrin and unspecified multivitamins and supplements. The patient has also been in counseling for anxiety since November 2015. Patient reported being at his baseline level of anxiety that day.

Review of systems was unremarkable except for the complaints of headache and visual disturbances. The patient did not have fevers, chills, and denies recent weight gain or loss. The patient's vitals taken at the family health clinic were unremarkable: the blood pressure measured 132/88 mmHg at 08:13AM, heart rate was 63 beats per minute, respiratory rate was 12 breaths per minute, body temperature was 98.2ºF, blood oxygen level was 98%. Neurological screening of cranial nerves II through XII were unremarkable bilaterally. Motor, coordination, balance,

reflexes, gait and stance were all normal. The patient was diagnosed with Headache, and given 2 intramuscular injections of 30mg/mL ketorolac (Toradol) at 08:57AM at the family health clinic. The patient was also given a prescription for 8mg Ondansetron (Zofran ODT) for nausea and vomiting, and a prescription for 800mg ibuprofen (Motrin) for pain, as needed. He was then sent to optometry for further vision evaluation.

The patient presented to optometry clinic wearing habitual spectacle correction, OD: -1.75 DS, OS: -1.00-1.00x110, and seeing 20/20-1 OD and 20/20 OS. Intraocular pressures measured 15 mmHg OD and OS at 09:32 AM with a non-contact tonometer (NCT). Extraocular motilities were normal in both eyes without restrictions, diplopia, or pain with eye movement. Pupillary testing was normal without afferent pupillary defect. Slit lamp examinations of the anterior segments were unremarkable for both eyes. Undilated view of the posterior pole was unremarkable OU (**Figure 1)**.

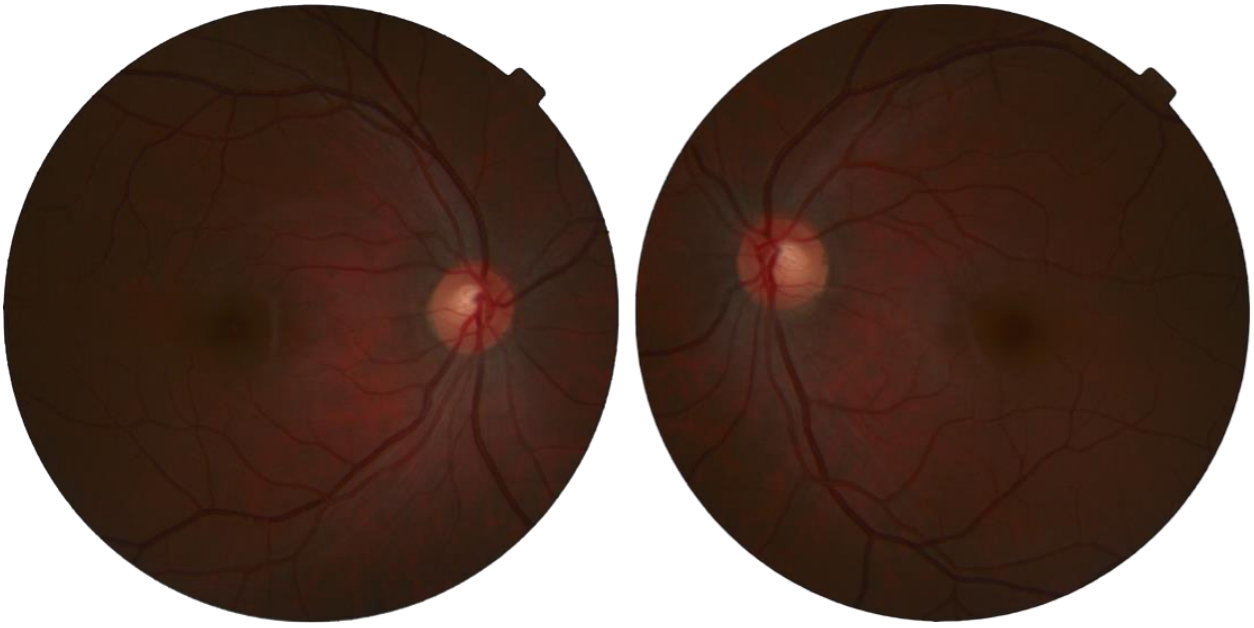

Figure 1 – Fundus photographs of the right and left posterior pole taken at the follow-up visit on 06 Jan 2017. Fundus appearances did not change from initial presentation on 04 Jan 2017.

The patient was then assessed on the Zeiss Humphrey Field Analyzer Model 750i, and completed a Central 24-2 SITA-Standard Threshold Test for each eye. The patient completed the field for the right eye in 6 minutes and 38 seconds, with 0/16 fixation losses, 7% false positive error, 20% false negative error, mean deviation of -2.94 decibels, and pattern standard deviation of 3.89 decibels. The right eye revealed a small superior nasal step just beyond 20 degrees of fixation and some points of depression scattered almost in an arcuate pattern, superiorly (**Figure 2)**. The patient completed the field for the left eye in 7 minutes and 24 seconds, with 2/16 fixation losses, 2% false positive error, 7% false negative error, mean deviation of -6.78 decibels, and pattern standard deviation of 7.07 decibels. The left eye revealed a significant infratemporal wedge defect that seemed to respect both horizontal and vertical midlines (**Figure 3**). There were

also scattered depressions throughout the remaining three quadrants of the left eye. Due to the limits of this test, the peripheral extents of the defects remained unknown.

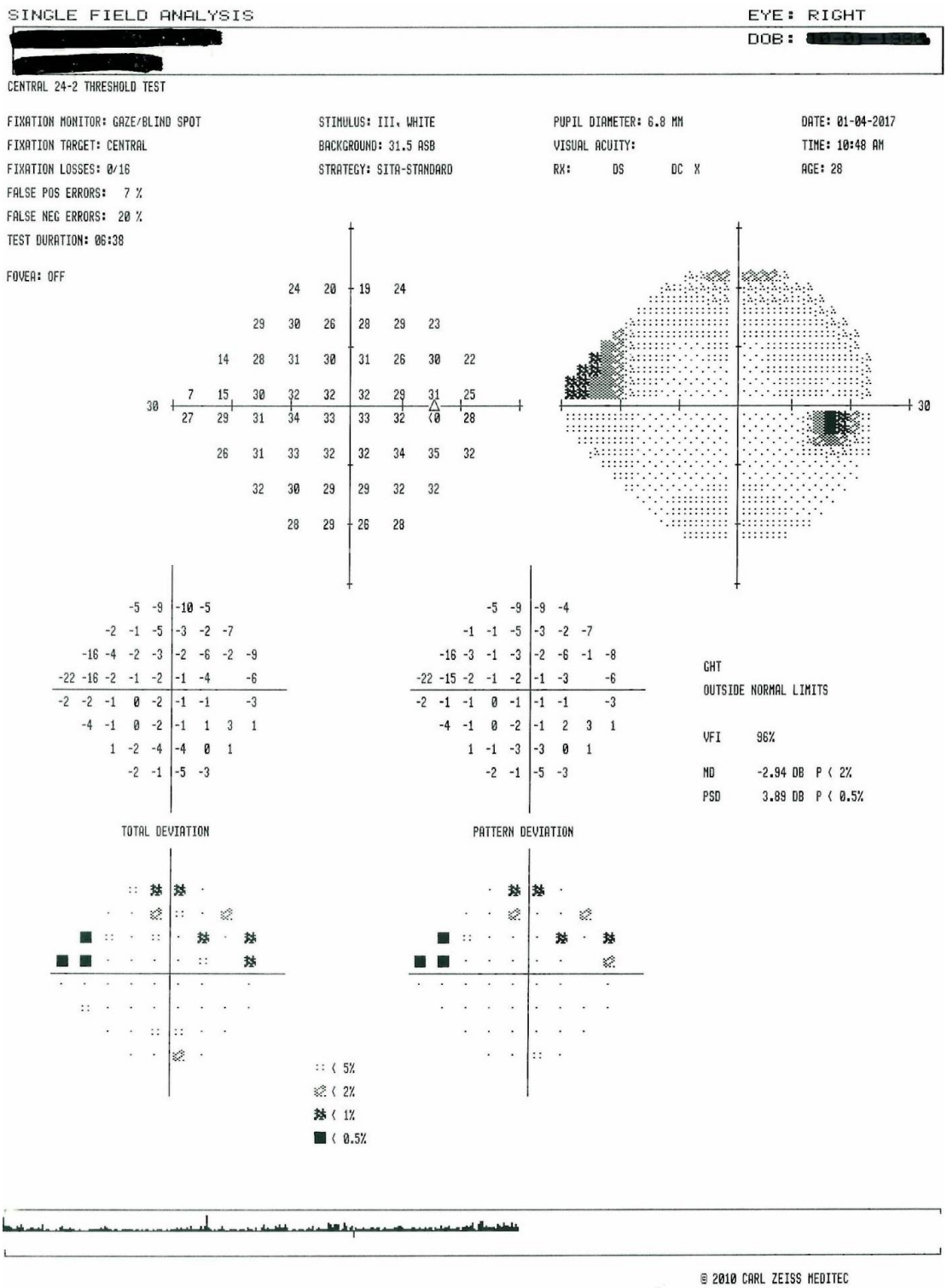

Figure 2 – HVF Central 24-2 SITA-standard for the right eye on the third day of migraine-like symptoms.

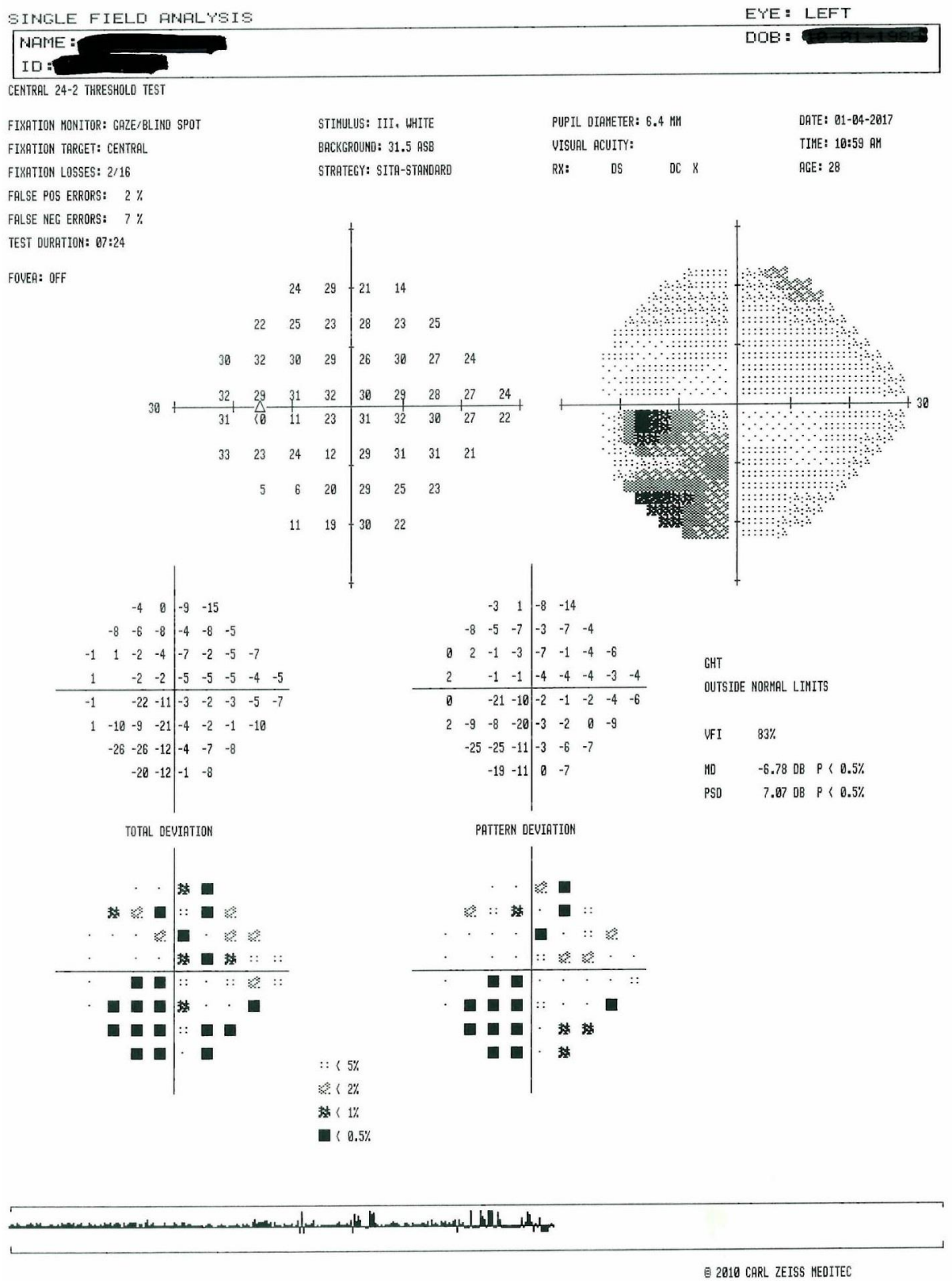

Figure 3 – HVF Central 24-2 SITA-standard for the left eye on the third day of migraine-like symptoms.

Differential diagnoses considered at this point include:

Normal Tension Glaucoma
Retrobulbar Neuritis
Nonarteritic Ischemic Optic Neuropathy
Retrochiasmal Lesions
Transient Ischemic Attack
Bilateral Branch Artery or Vein Occlusions
Migraine with Aura

- Normal Tension Glaucoma (NTG) is a disease of the optic nerve where progressive damage occurs despite normal or mildly elevated IOP. Drance hemorrhage, a flame-shaped hemorrhage on the optic disk indicating an ischemic event, is present in 40% of NTG patients. Risk factors for NTG include enlarged physiologic cupping, poor vascular autoregulation, arteriosclerotic vascular disease, systemic hypotension, carotid artery disease, arrhythmias, diabetes, primary vascular dysregulation (PVD), Alzheimer's disease, and migraine.[4]
- Retrobulbar Neuritis (ON) is an inflammatory demyelination of the optic nerve behind the lamina cribrosa. The optic disc may appear normal for up to 4 weeks from the initial attack. Multiple sclerosis is the most common cause, while Lyme and syphilis are common infectious causes. Eye pain is present in 63% of cases. Visual field defect is generally an overall depression in the affected eye, but peripheral only defects have been reported.[4,5]
- Nonarteritic Ischemic Optic Neuropathy (NAION) is a stroke of the optic nerve head that occurs in 46-65 year olds, in males more than females. Risk factors include hypertension, diabetes, peripheral vascular disease, hyperlipidemia and small cup-to-disc ratio. Amaurosis fugax is common and typically lasts 2-3 minutes, rarely up to 30 minutes. The optic nerve typically appears hyperemic, edematous, and splinter hemorrhages could be present. 42% present with visual acuity worse than 20/100. Visual field defects can vary, but inferior altitudinal or arcuate scotoma is common.[4]
- Retrochiasmal Lesions affecting the right optic tract, lateral geniculate body or anterior optic radiations can result in incongruous homonymous hemianopia.[4] These lesions can be caused by, but not limited to stroke, trauma, tumor, and demyelination.[6] Even pituitary macroadenoma has been shown to result in incongruous homonymous hemiananopsia.[7]
- Transient Ischemic attack typically affect older patients at risk for stroke. Symptoms include numbness, visual loss, paralysis, and ataxia. Typical duration is less than one hour and recurrent spells can occur over days, weeks or months. Headaches typically present at the time of the TIA.[8]
- Branch Artery Occlusion (BRAO) is an infarction of an area of retina supplied by the arterial branch due to an embolic event. The affected retina may gradually become whitish due to ischemia. A permanent visual field defect corresponding to the area of damage results from the ischemic event.[4]
- Branch Retinal Vein Occlusion (BRVO) is an obstruction of outflow anterior to the cribriform plate, usually at sites of AV crossings. This obstruction causes a build up of

intravascular pressure resulting in retinal hemorrhages and cotton wool spots. Risk factors for BRVO include cardiovascular disease, systemic hypertension, diabetes and open-angle glaucoma.[4] Blurred vision can occur in the visual field corresponding to the affect retinal area.[9]

- Migraine with Aura, as defined by International Headache Society (IHS), is a primary headache disorder characterized by reversible visual, sensory, and/or speech symptoms for an hour followed by a headache. Complicated migraines have been reported to include attacks lasting greater than 72 hours or auras persisting for more than 1 week.[2]

The patient was dilated at 11:28AM with one drop of 0.5% Proparacaine hydrochloride ophthalmic solution, followed by one drop of 1% Tropicamide ophthalmic solution, and one drop of 2.5% Phenylephrine hydrochloride in each eye. The dilated fundus exams were unremarkable OU. There were no retinal or vascular abnormalities observed, and both eyes had healthy foveal reflexes. The optic nerves appeared healthy in both eyes, with no signs of pallor, edema, or hemorrhaging. The cup to disc ratio for the right optic nerve was 0.50x0.50, and for the left optic nerve was 0.55x0.55, both following the ISNT rule.

In the absence of retinal findings on dilated fundus examination and the presence of positive visual field defects OU, optical coherence tomography (OCT) of the optic nerve and of the macula were ordered to make sure subclinical signs were not missed. The retinal nerve fiber layer (RNFL) analysis of the optic nerve were unremarkable OU, except for a borderline suspicion of a superonasal wedge thinning in the right eye (**Figure 4**). The ganglion cell analysis OU were unremarkable (**Figure 5**).

The appearance of the optic nerves on fundus examination and on the OCT did not correlate with the bilateral defects found on visual field testing. The absence of hemorrhages, cotton wool spots, retinal pallor, and other retinovascular abnormalities ruled out bilateral branch retinal artery and branch retinal vein occlusions from the list of differential diagnoses. The optic nerves did not show notching, inferior thinning, RNFL dropout, Drance hemorrhage, or other signs indicative of glaucoma. The optic nerves also did not appear crowded, edematous, or hyperemic, and the visual field loss did not appear typical of NAION, which are typically altitudinal. The evidence for retrochiasmal lesions was low due to the lack of optic nerve head pallor, the incongruity of the field defects, and the absence of other focal neurological signs and symptoms. The persistence of visual symptoms and headache in the past 48 hours was also atypical for a transient ischemic attack. The suspicion for retrobulbar optic neuritis was also low based on patient's reported symptoms and denial of pain during eye movements. The history of "heat waves" in the peripheral visual field and the onset of acute headache after 30 minutes, were more consistent with the characteristics of migraine with aura. Thus, the bilateral peripheral visual field depressions were attributed to residual effects of the migraine event and expected to improve over the next 7 to 10 days.[10]

Since the patient was reporting a noticeable relief after the injection of 60 milligrams of Toradol, no additional testing and treatments were recommended. The patient was diagnosed with 1.5.2

probable migraine with aura, since he failed to meet the ICHD-3 criteria of having at least two attacks.[2] The patient was advised to avoid bright lights, loud sounds, strong odors, and foods with monosodium glutamate and/or nitrates.[11] The patient was also advised to continue the management of headache symptoms with the family health provider, and to return to optometry in two days for a repeat of the visual field test.

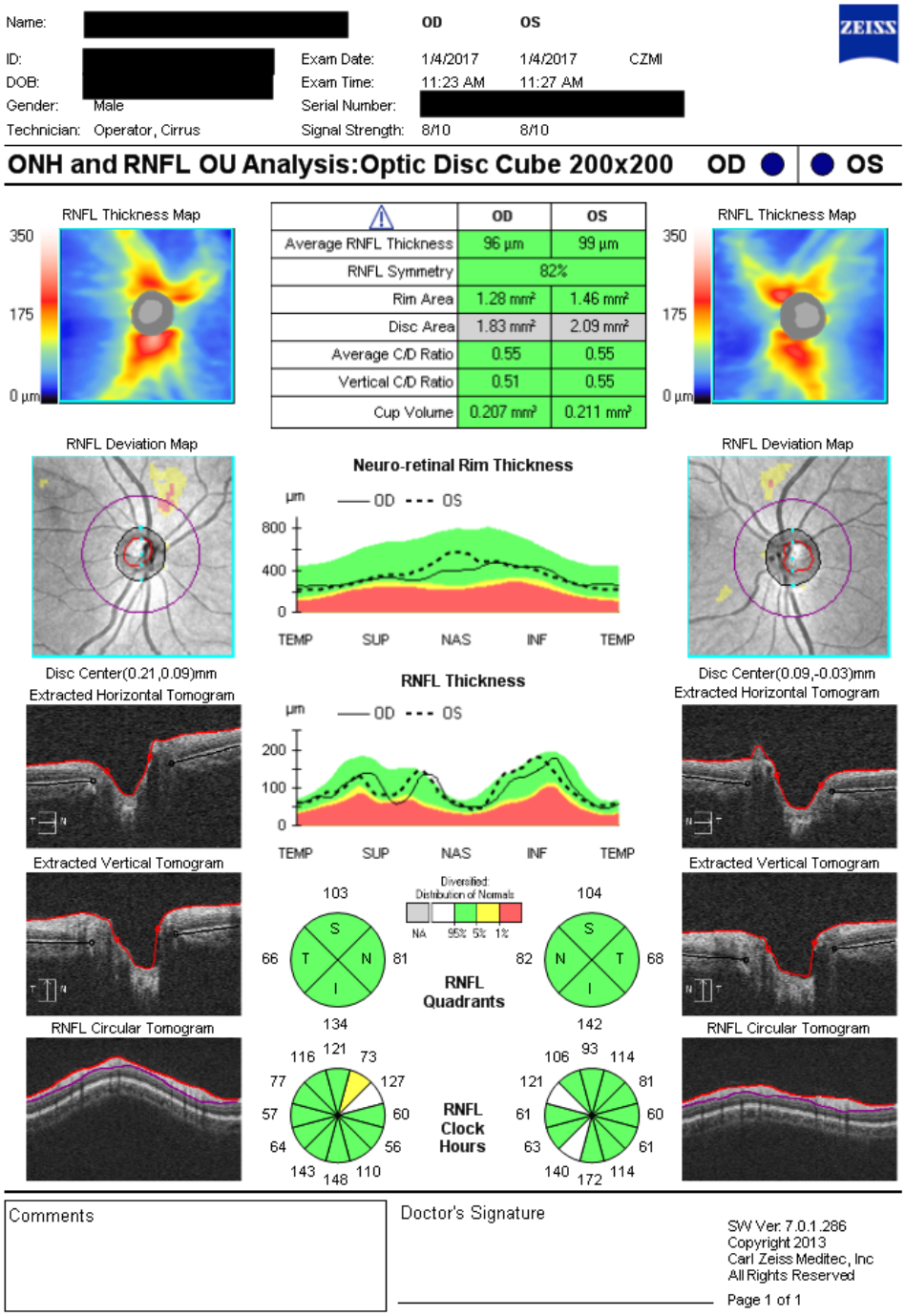

Figure 4 – RNFL OCT of optic nerves showing a borderline wedge defect at 1 o'clock hour of right optic nerve.

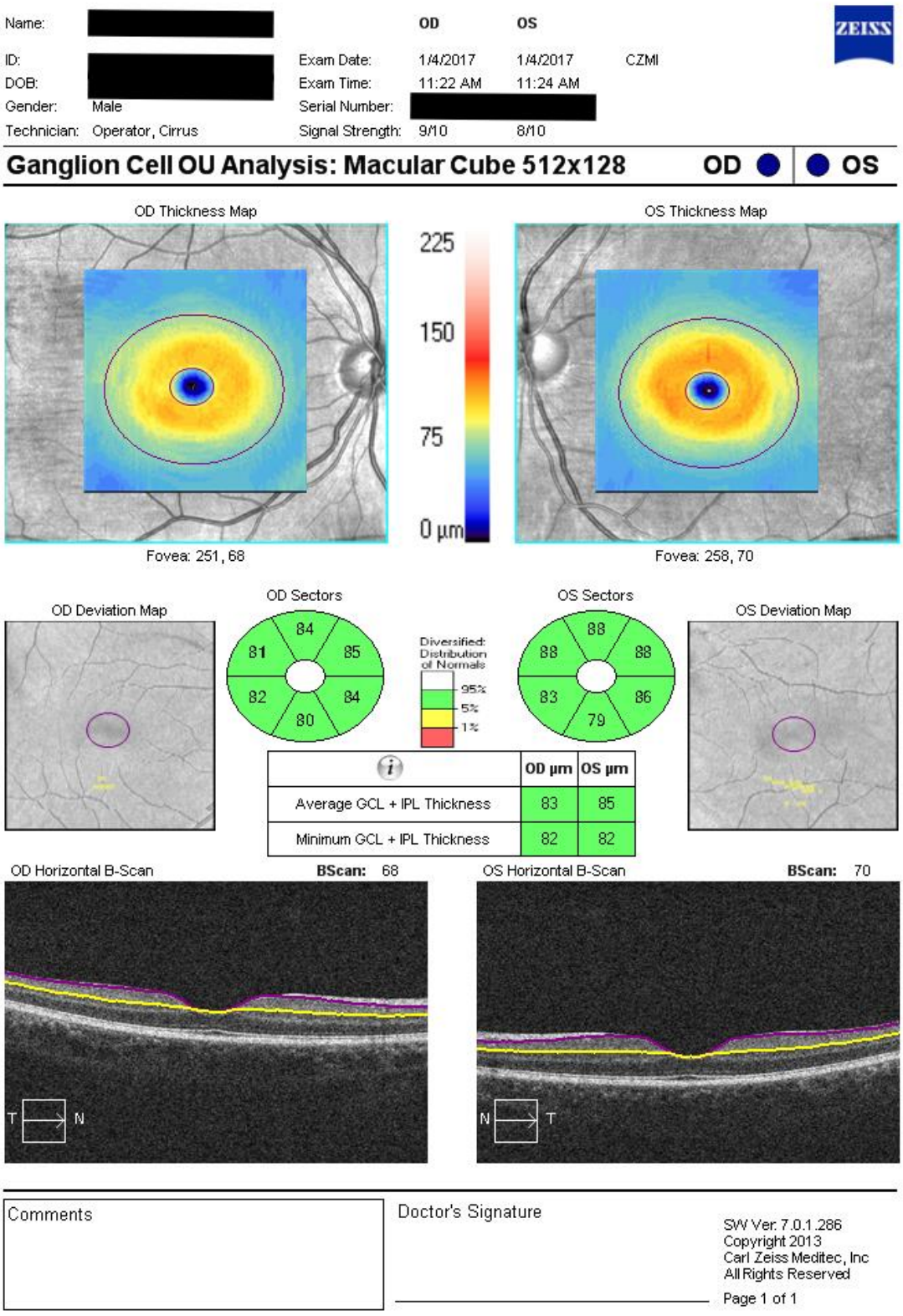

Figure 5 – Ganglion Cell Analysis (GCA) OCT over the maculae were unremarkable in both eyes.

Follow-Up #1

The patient returned to clinic on Jan 6, 2017 for follow up. The patient reported feeling a lot better. He took Zofran and ibuprofen yesterday for prophylaxis, although he did not like to take medication, as he preferred more natural remedies. The patient reported the visual disturbances had gone away and was asymptomatic. The patient entered with 20/20 acuity in each eye wearing his habitual glasses. His eye pressure measured 15mmHg OD, 14mmHg OS at 09:20AM with the NCT. Extraocular motilities were smooth, accurate, full and extensive, without pain or diplopia. Pupils testing revealed equal, round pupils in dim and bright, reactive to light, and without afferent pupillary defect in either eye. The patient also passed color vision with 6 out of 6 correct on the HRR pseudoisochromatic plate test #4 in each eye. Anterior segment was unremarkable in each eye. The posterior pole through undilated 78D lens was also normal with 0.50x0.50 cup to disc measurements for each nerve and no signs of edema, pallor, or hemorrhage (**Figure 1**).

The Central 24-2 SITA-Standard Threshold Test was repeated on each eye. The patient completed the field for the right eye in 5 minutes and 36 seconds, with 1/15 fixation losses, 2% false positive error, 0% false negative error, mean deviation of -3.56 decibels, and pattern standard deviation of 7.35 decibels. The right eye revealed a progression of the superior nasal step expanding 10 degrees towards fixation; however, all the points of depression in the other quadrants had improved (**Figure 6)**. The patient completed the field for the left eye in 5 minutes and 51 seconds, with 0/14 fixation losses, 0% false positive error, 0% false negative error, mean deviation of -1.85 decibels, and pattern standard deviation of 2.74 decibels. The left eye revealed an overall improvement from two days ago (**Figure 7**). There were still some scattered points of depressed sensitivity throughout the supranasal and infratemporal quadrants of the left eye. Again, due to the same limits of this test, the peripheral extents of the defects remained unknown.

Based on the mixed results of the visual field tests, the RNFL and GCA OCT scans were repeated. The results of the RNFL OCT were stable to the original scan on Jan 4, 2017 (**Figure 8)**. Likewise, the ganglion cell analysis OCT returned similarly stable results compared to the prior scans (**Figure 9**). As with most visual field findings, patients are typically insensitive to decreases in peripheral vision. However, in the setting of significant improvements in other parts of the visual fields and the unremarkable fundus examination, this progression of the right nasal defect was attributed to the patient's course of migraine with aura. Clinical decision to continue monitoring was made. The patient was advised to self-monitor for any changes in his vision, monocularly. A one-week follow-up appointment was made to re-evaluate his vision and visual field. The patient was advised to return sooner if vision and headache symptoms worsened.

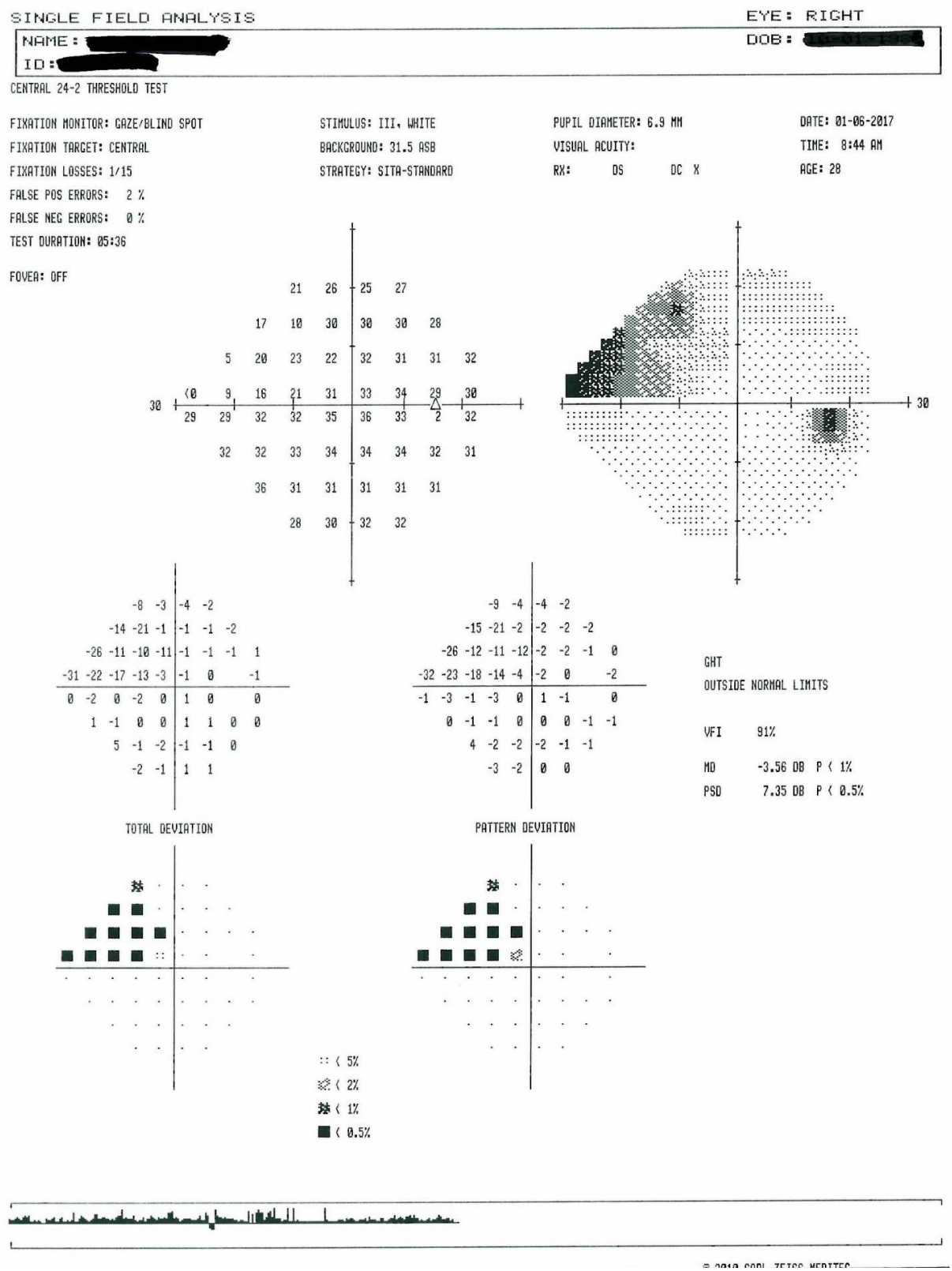

Figure 6 – HVF Central 24-2 SITA-standard for the right eye on the fifth day of the initial migraine attack. The field showed a worsening of the superonasal step as it expanded towards fixation.

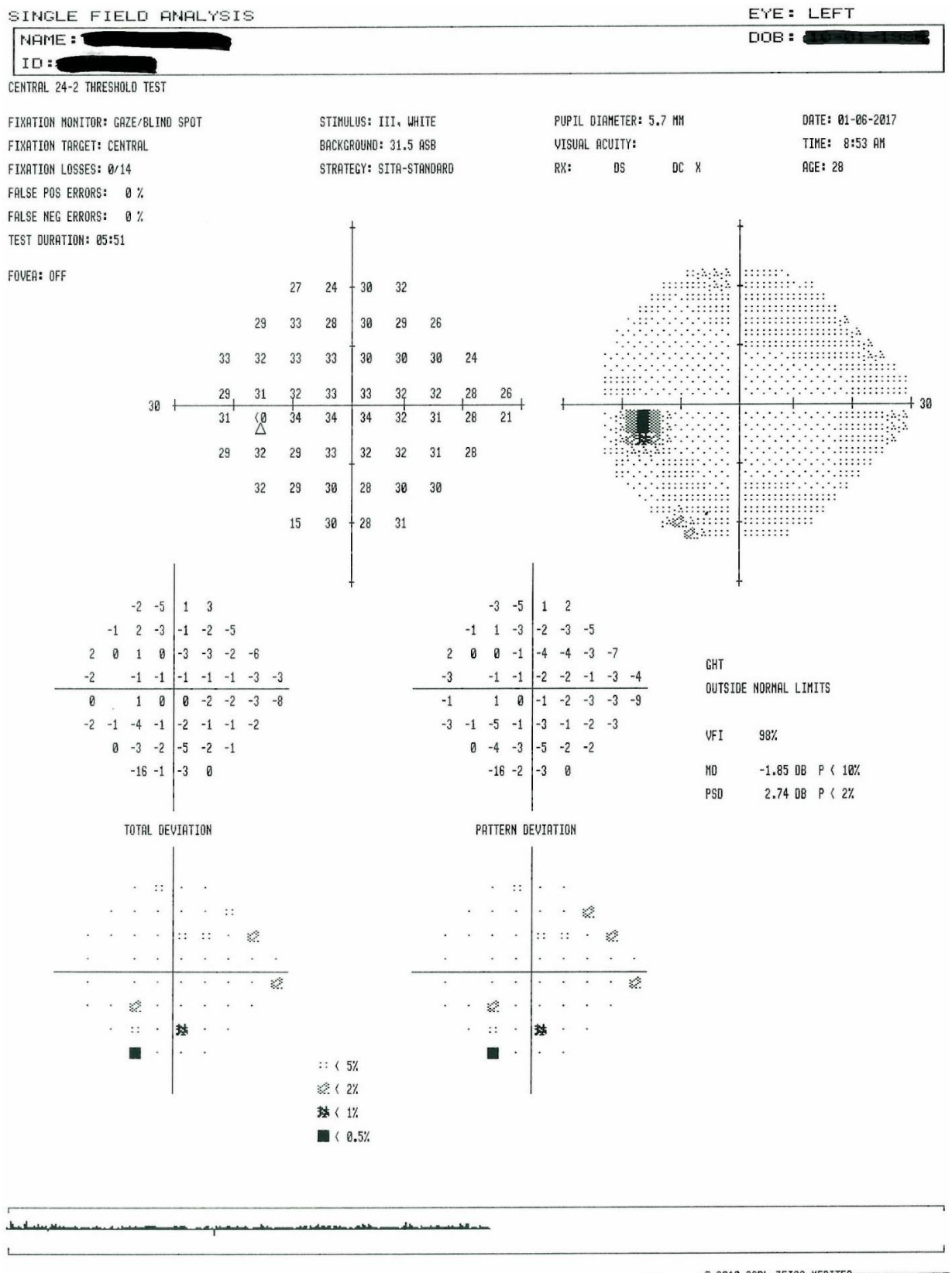

Figure 7 – HVF Central 24-2 SITA-standard for the left eye on the fifth day of the initial migraine attack showing marked improvement of the inferotemporal defects from 04 Jan 2017.

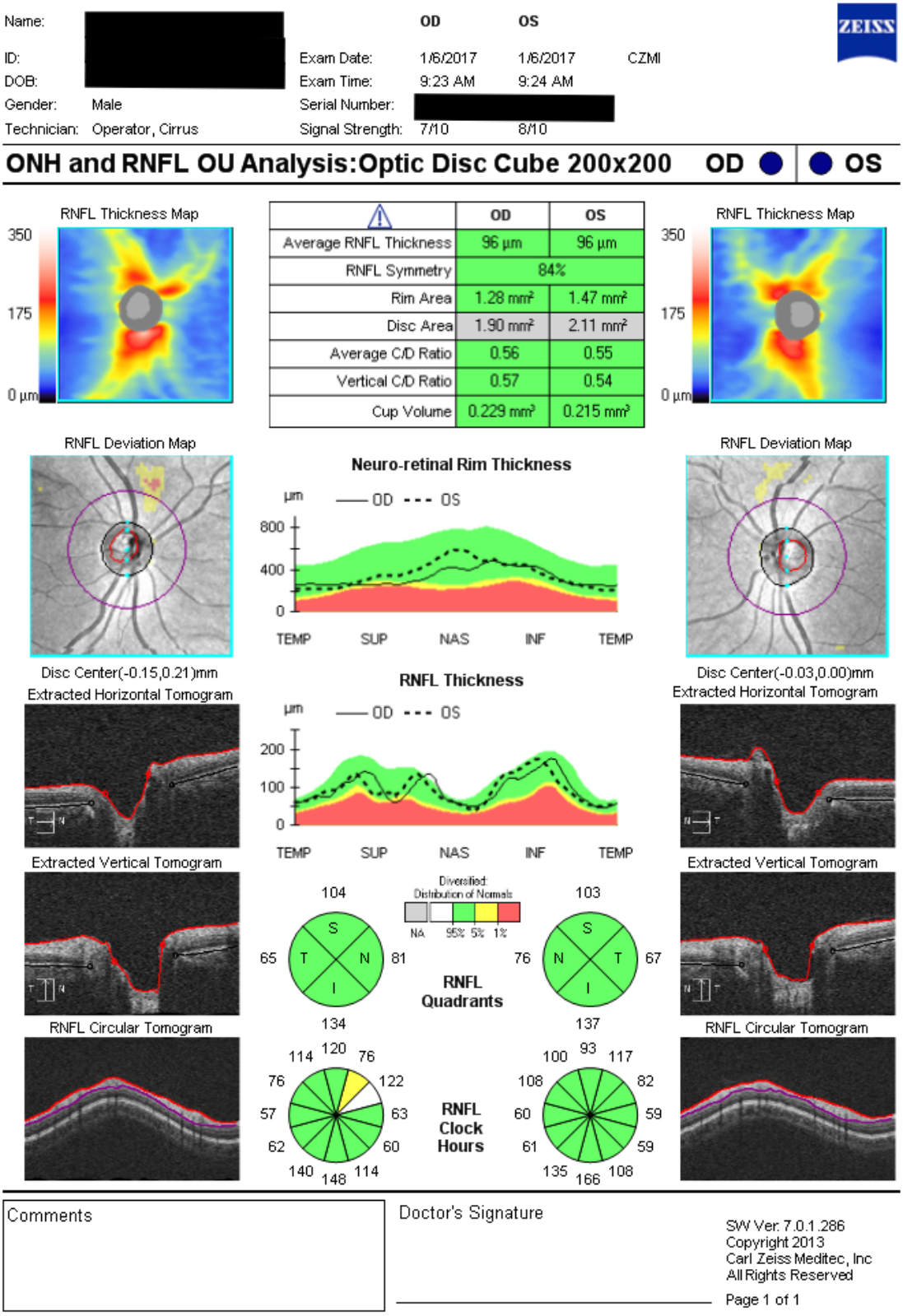

Figure 8 – RNFL OCT of optic nerves showing a borderline wedge defect at 1 o'clock hour of right optic nerve, relatively stable with 3µm of thickening compared to OCT on 04 Jan 2017.

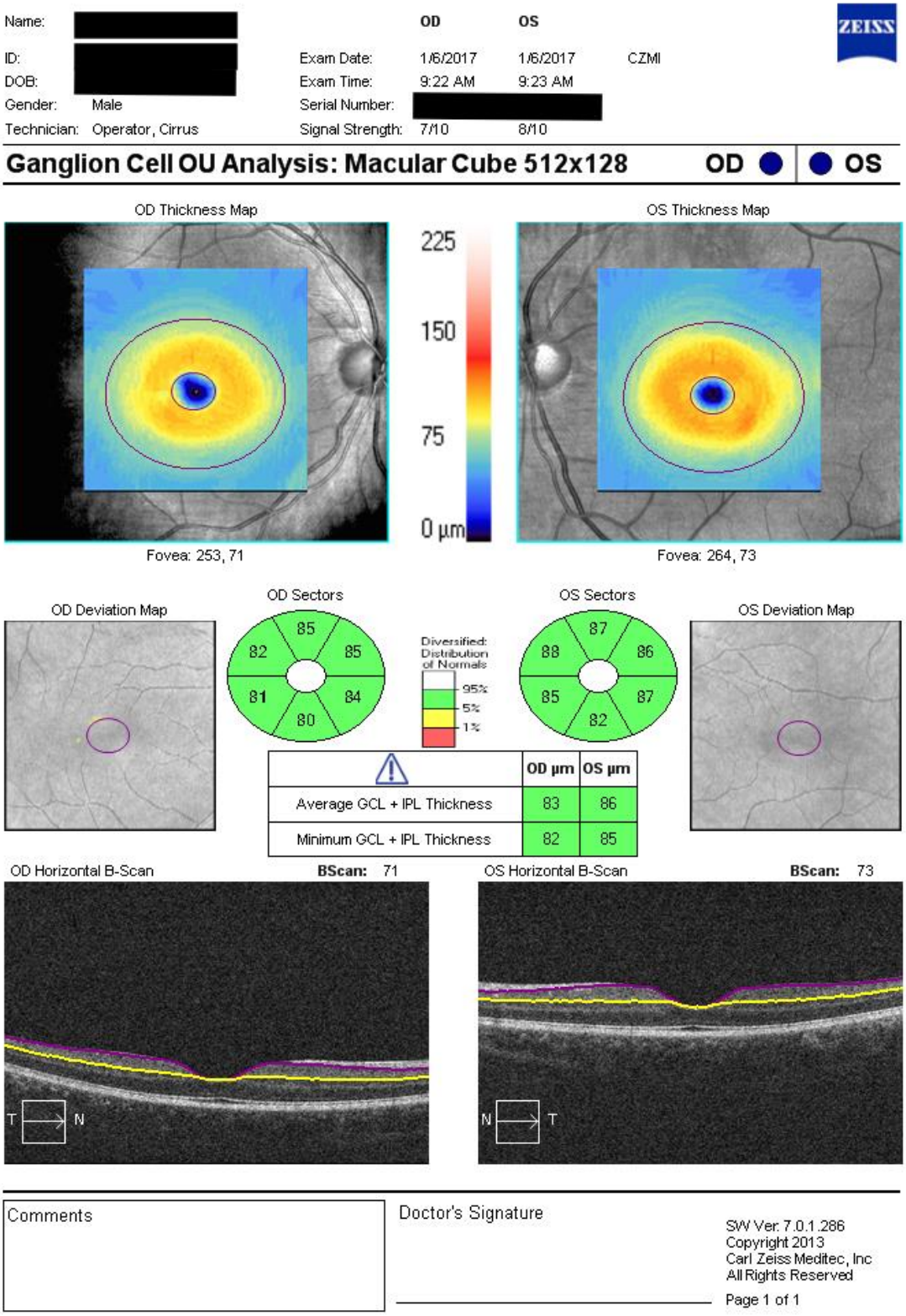

Figure 9 – GCA OCT scan over the maculae OU showing normal ganglion cell layer thickness, stable to 04 Jan 2017 scan.

Follow-Up #2

The patient returned to clinic on January 13, 2017 for re-evaluation. The patient was seen by Dr. Swanson, while Dr. Zhang was on a temporary duty assignment. The patient reported his headaches and visual symptoms were gone, and did not have any other concerns. The patient entered with 20/20 vision in each eye with his habitual glasses. His IOP measured 16mmHg in the right eye, 13mmHg in the left eye, at 07:53AM. Pupils and extraocular motilities testing were unremarkable in both eyes. Anterior segment and undilated posterior segment examination were also unremarkable and stable to prior findings.

The Central 24-2 SITA-Standard Threshold Test was repeated on each eye. The patient completed the field for the right eye in 5 minutes and 08 seconds, with 1/14 fixation losses, 0% false positive error, 2% false negative error, mean deviation of -1.81 decibels, and pattern standard deviation of 1.90 decibels. The right eye showed a few points of depressed sensitivity, not repeatable from prior tests, and overall significant improvement in all quadrants (**Figure 10)**. The patient completed the field for the left eye in 5 minutes and 04 seconds, with 2/13 fixation losses, 0% false positive error, 0% false negative error, mean deviation of -1.50 decibels, and pattern standard deviation of 1.37 decibels. The left eye also improved significantly overall, revealing an essentially clean field (**Figure 11**). RNFL OCT was repeated and revealed a suspicious thinning in the superotemporal wedge of retinal nerve fiber layer in the right eye and a superior thinning of RNFL in the left eye (**Figure 12**). Ganglion cell analysis scan was not performed at that visit. The areas of thinning did not correlate with the visual field findings that day. The patient was advised to continue monitoring for changes in vision and to return to the clinic in 4 weeks for repeat of the visual field in the right eye only and RNFL OCT in both eyes. However, the patient did not keep his one-month appointment, and did not make another follow-up appointment.

Record Review

The patient eventually presented to the local emergency room on April 24, 2017 with complaints of intermittent tingling and twitching on the right side of the face for the past one week. Occasionally, he would experience a surge on the right side of this head and brain, followed by pain in the neck, and a right eye twitch. The patient denied headache, trauma, focal deficit, diplopia, ataxia, and weakness. Patient also denied chest pain, shortness of breath, abdominal pain, nausea, vomiting, and diarrhea. The patient denied family history of aneurysms. Physical examination including neurological screening was unremarkable. A computerized tomography CT scan of the head was ordered without contrast, and the findings were negative. The patient was discharged in stable condition with diagnosis of episodic, unspecified headache, and ordered to follow up with his primary care provider at the medical treatment facility on base.

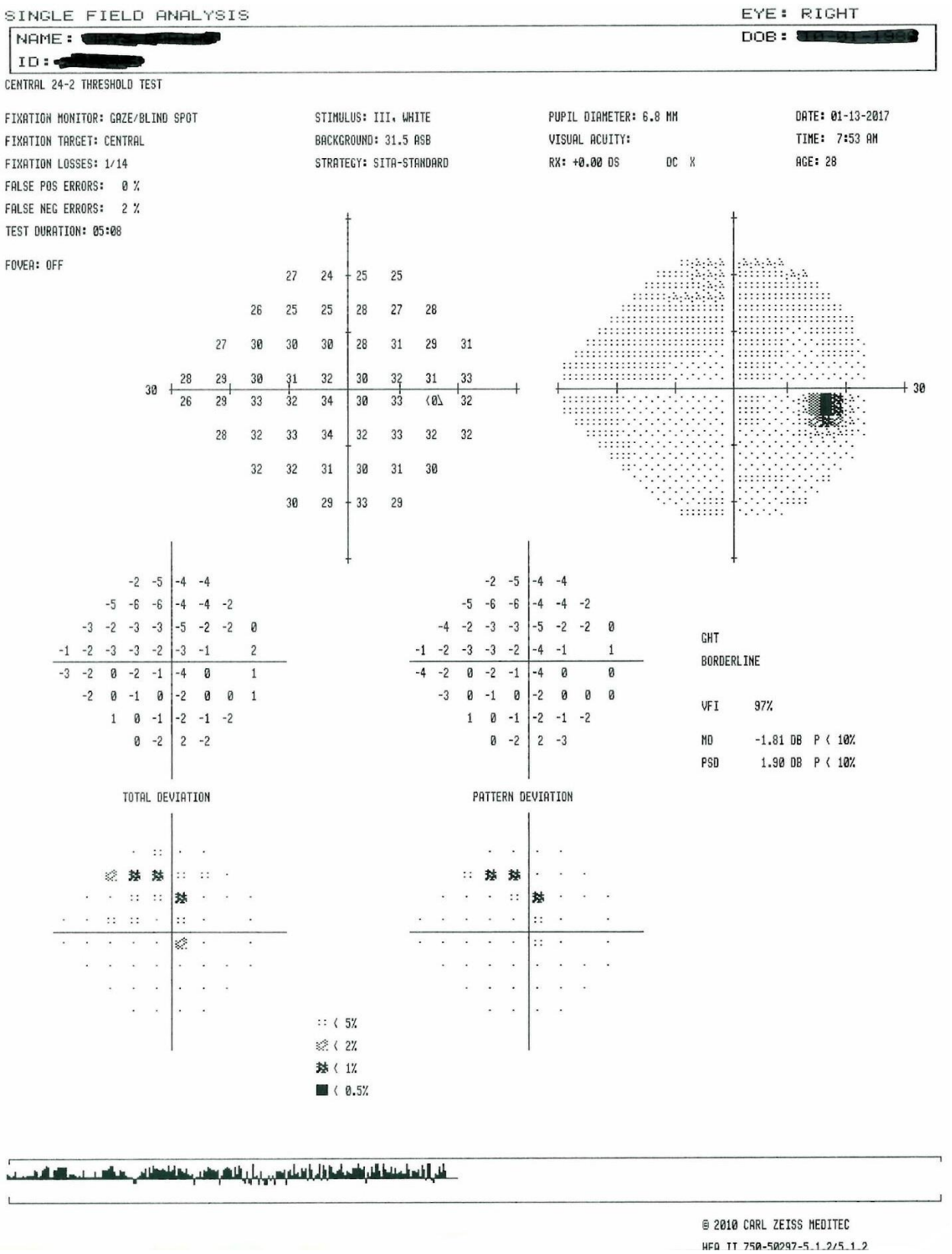

Figure 10 – HVF Central 24-2 SITA-standard for the right eye on the twelfth day since the initial migraine attack showing overall improvement of the field.

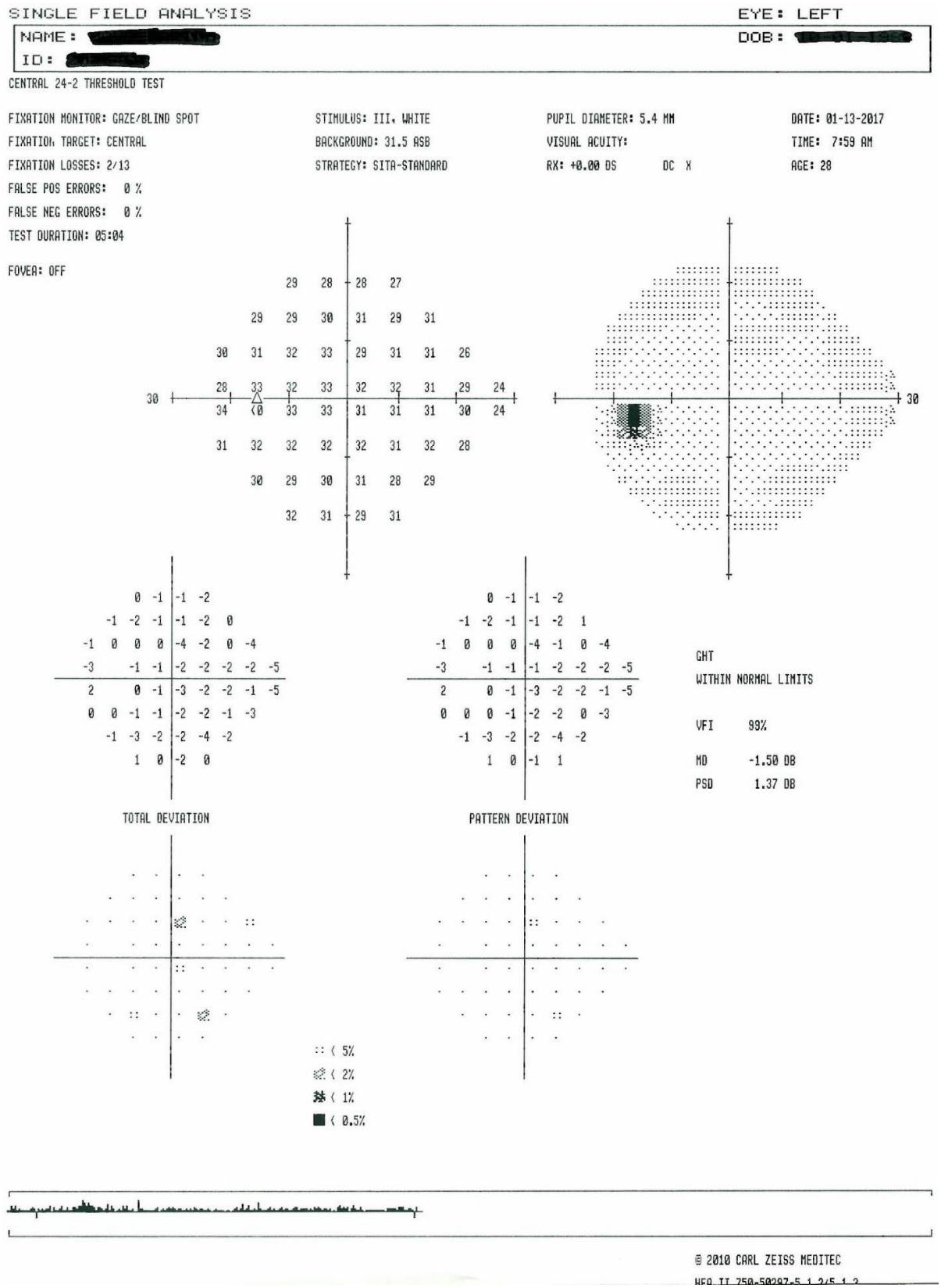

Figure 11 – HVF Central 24-2 SITA-standard for the left eye on the twelfth day since the initial migraine attack showing stable improvements over the previous 2 fields.

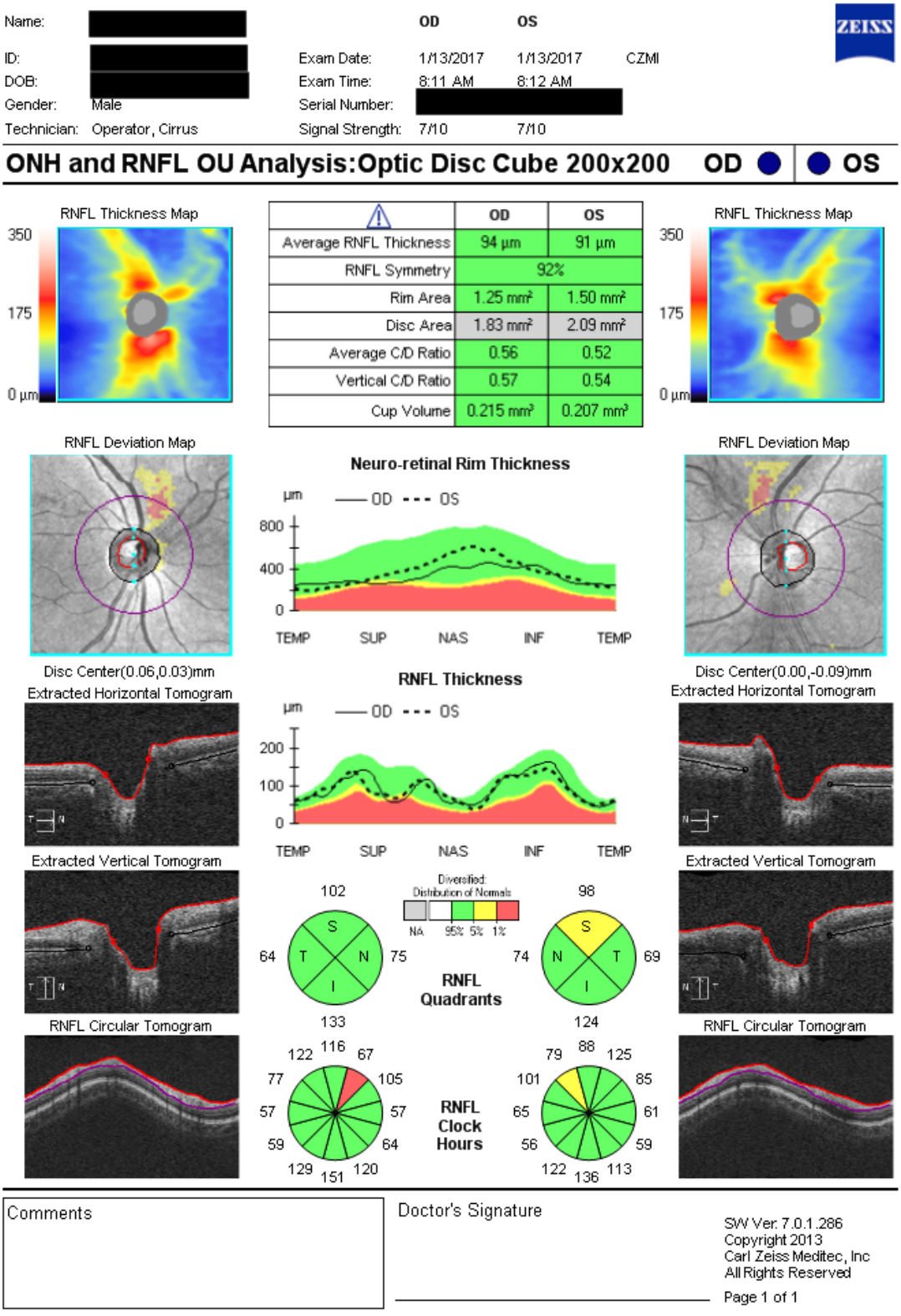

Figure 12 – RNFL OCT of optic nerves showing a suspicious wedge defect at 1 o'clock hour of right optic nerve, with 6 µm of thinning from 04 Jan 2017 scan. The left RNFL also showed 6 µm of thinning in the superior quadrant compared to 04 Jan 2017.

The patient followed up at the family health clinic on May 1, 2017. The patient reported four total, significant episodes of deep pulsatile sensation behind the right eye followed by involuntary closure of right eyelid, right-sided face twitch, neck weakness, and arm trembling after 15 minutes. The patient reported resting and hydration relieved the symptoms. The patient reported stress from recent failed in vitro fertilization (IVF) attempt may be a possible trigger. The patient was again diagnosed with headache, an MRI scan of the head was ordered, and the patient was referred to neurology for further evaluation.

The MRI scan with and without gadolinium on May 9, 2017 revealed a couple of tiny nonspecific high T2/FLAIR weighted signal intensity foci in the posterior left periventricular white matter. Acute mass, midline shift, intracranial hemorrhage, hydrocephalus and abnormal focal enhancements were ruled out. Intracranial intravascular flow were grossly normal. No acute intracranial pathology was identified.

The neurology visit on May 10, 2017 identified right frontoparietal, pulsating headache with associated right lid droop for one minute followed by trembling and decreased visual-spatial orientation. Neurological workup was unremarkable. In the setting of negative findings on the MRI with and without gadolinium, the neurologist's diagnosis was primary headache. A CT angiogram of the intracranial vessels was ordered, and the results were pending. The neurologist suspected the CTA findings to be normal, and thus did not schedule a follow up appointment while awaiting CTA results.

Discussion

Migraine is a recurrent headache disorder affecting 18% of women and 6% of men each year.[1] Approximately 90% of migraine sufferers report some disability associated with the disease, and many lose several days of work each year due to the condition.[1] In addition, migraine sufferers also show a higher incidence of stroke, normal tension glaucoma, and affective disorders such as depression and anxiety.[1]

The International Headache Society created an International Classification of Headache Disorders, currently in its third edition, beta form, ICHD-3beta, for the purpose of classifying headaches for epidemiologic and clinical trial studies.[2] According to ICHD-3beta, a diagnosis of 1.1 migraine without aura must include at least five (5) attacks of the following characteristics:

1. The headache attacks last 4 to 72 hours (untreated)
2. The headache must have at least 2 of the following properties:
    a. unilateral
    b. pulsating
    c. moderate or severe pain
    d. aggravated by routine physical activity
3. During headache, one of the following symptoms must also be present:
    a. Nausea, vomiting, or both
    b. Photophobia or phonophobia

Also according to ICHD-3beta, a diagnosis of 1.2 migraine with aura must include at least two (2) attacks of the following characteristics:

1. One or more fully reversible aura symptoms affecting the following area:
    a. Visual
    b. Sensory
    c. Speech and/or Language
    d. Motor
    e. Brainstem
    f. Retinal
2. At leaset 2 of the following four characteristics:
    a. At least one aura symptom spreads gradually over 5 minutes or more, and/or two or more symptoms occurring in succession
    b. Each individual aura symptom lasts 5-60 minutes
    c. At least one aura symptom is unilateral
    d. The aura is accompanied, or followed within 60 minutes, by headache.

For all migraine diagnoses, other ICHD-3beta diagnoses and transient ischemic attack must be ruled out. If any of the symptoms fall outside the range set by the strict diagnostic criteria, the diagnosis of 1.5.1 probable migraine without aura or 1.5.2 probable migraine with aura should be used instead.[2]

In migraine with aura sufferers, 99% experience some form of visual aura, 31% experience somatosensory auras such as tingling or numbness, 18% experience dysphasia, 6% experience motor weakness.[12] Visual auras have been classically described as scintillations of C-shaped scotomas with jagged edges, known as teichopsia for its fort-like appearance. These scintillations, occurring in 41.8% of aura sufferers,[12] often appear to enlarge and/or march across the visual field. Other common visual auras have been described as general visual blur (54.1%), as if looking through a film or water; flashes and sparkles of light (47.5%); shimmering or appearance of heatwaves; and rotation of objects.[12] Less common visual auras may include metamorphopsia, macropsia, micropsia, palinopsia, cerebral polyopia, dyschromatopsia, and prosopagnosia.[1,13] Typical auras (1.2.1 Migraine with typical aura) last between 5 and 60 minutes.[2]

In some cases, the migraine symptoms can fall outside the range in the diagnostic criteria for typical migraine with or without aura. In these cases, the additional complication should be coded separately from the primary migraine subtype diagnosis.[2] Such is the case in this study. At the initial presentation, the patient did not have any prior history of similar occurrences, and thus failed the criterion of having at least two migraine with aura attacks. Additionally, the visual field defect had persisted beyond the 60 minute range for the typical migraine with aura diagnosis. Based on record review of subsequent migraine-like episodes and neuroimaging clear of cerebral infarction, the patient likely had a persistent aura without infarction, although the persistence of aura symptoms for at least 1 week cannot be confidently supported.

The pathophysiology of migraine with aura is not completely undertood.[1,12,14,15] The most accepted explanation to date links migraines with a phenomenon known as the cortical spreading depression (CSD) originally discovered by Leão in 1944.[1,8,12] In CSD, the disturbance of cell membrane function is transmitted to nearby cells, and results in a self-propagating wave of neuronal and glial depolarization across the cerebral cortex at a rate of 2-3mm per minute.[1,8]

Studies linking migraines to defects in chromosomes 1 and 19, coding the function of calcium and potassium channels, suggest channelopathy as a potential cause for the neuronal hyperexcitability seen in CSD.[1] This hyperexcitability is presumed to activate parts of the cortex and the trigeminovascular system.[8] The cortical activation has been proposed to be the origin of aura, while the trigeminovascular activation has been proposed to cause a neurogenic inflammation that results in sensitization of afferent neurons and the perception of pain in migraine headaches.[8]

Prolonged and persistent aura symptoms are rare but have been reported.[1,8,13] Most cases are that of bilateral, positive visual phenomenon of either formed (e.g. pinwheels, concentric circles) or unformed (e.g. visual snow, sparkles) visual hallucinations.[1,13] In this case report, the patient experienced a prolonged positive visual aura ("heat waves") for three days followed by a persistent decrease in peripheral visual sensitivity for at least seven days. The finding of peripheral vision loss is consistent with the findings in a study by Drummond and Anderson, in which the kinetic perimetry of 20 migraine with aura subjects showed decreased peripheral fields a day after an attack with subsequent resolution of field defects between 7 and 10 days.[10] The visual aura could be explained by the cortical spreading depression hypothesis.[1,8] However, the prolonged peripheral visual field deficit does not fit patterns of higher cortical involvement.[15]

McKendrick et al proposed a precortical involvement, hypothesizing that peripheral vasospasm around the optic nerve and surrounding choroid could be involved in the migrainous visual field changes.[15] Numerous studies have linked migraine to the progression of normal tension glaucoma and occurrence of Drance hemorrhages, suggesting impaired microvascular autoregulation and resulting ischemia as a common vascular etiology for both NTG and migraine.[5,15,16,17] In studies on the treatment of normal tension glaucoma, calcium channel blockers have shown to significantly reduce the rate of optic nerve and visual field damage.[16,17] Interestingly, calcium channel blockers have also been widely used for migraine prevention, yet evidence supporting its efficacy have been deemed weak and conflicting.[18] In addition, tolerance seem to develop with calcium channel blockers after 8 weeks of treatment for headaches.[18] Nonetheless, antihypertensive medications, specifically beta blockers, ACE inhibitors, ARBs, and thiazides, have been shown to reduce the prevalence of headaches compared to placebo.[18] Based on these findings, the patient may experience added benefit of reducing migraine occurrences and risk of glaucomatous nerve damage with improved management of systemic hypertension.

Conclusion

Patients with complaints of headaches are often referred to eye care providers for co-management, especially when visual symptoms are present. In such cases, in addition to taking a careful history to rule out more dire etiologies, a visual field test should be ordered to confirm the absence of highly localizing processes. It is not unexpected for migraine patients to show some peripheral, incongruous visual field defects within 7 days of the migraine attack. These field defects can worsen before they improve. With the high incidence of normal tension glaucoma in patients with migraine headaches, visual field testing should be assessed on all suspected migraine cases. If the field results are completely normal, they can serve as a baseline for future routine evaluations. At the same time, history of migraine headaches should be elicited

during all routine eye exams, so that migraine patients are regularly evaluated on the OCT and the visual field for signs of glaucomatous change. This will ensure prompt treatment when progression becomes evident.

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