Non-Arteritic Anterior Ischemic Optic Neuropathy with Vitreous Hemorrhage: a Case Report

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ABSTRACT

The present research reports an unusual case of a 69-year old man affected by unilateral non-arteritic anterior ischemic optic neuropathy (NA-AION) with vitreous hemorrhage. The patient visited with a complaint of sudden painless vision loss in the right eye for five days. His best- corrected visual acuity was 1/20 for the right eye. Assessment of the fundus revealed mild or moderate vitreous hemorrhage with an obscured view of the posterior pole in the right eye. Visual acuity improved to 4/20 in the right eye eight days later. Fundus examination showed marked disc swelling and peripapillary flame-shaped hemorrhage in the right eye. Optical coherence tomography (OCT) demonstrated optic nerve edema and subretinal fluid extending to the macula. The patient was diagnosed with presumed NA-AION with vitreous hemorrhage in the right eye. Furthermore, he had a spontaneous improvement in visual acuity without any treatment. This is the first-ever case report describing a vitreous hemorrhage in a patient with NA-AION. Keywords: non-arteritic anterior ischemic neuropathy, optic nerve edema, vitreous hemorrhage, optical coherence tomography.

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INTRODUCTION

Non-arteritic anterior ischemic optic neuropathy (NA-AION) is the most common form of ischemic optic neuropathy. Their pathogenesis, clinical features, and management have been controversial and confusing. It generally affects people over 50 years of age and there is no gender predisposition. NA-AION presents with loss of vision occurring over hours to days, often described as blurring or dimness in the area of the field loss. Hayreh et al noted that vision loss was often reported upon awakening, and some authors believe that the etiology for NA-AION is nocturnal hypotension.[1] Vision loss from NA-AION is variable but is typically less severe than the loss from arteritic anterior ischemic optic neuropathy (A-AION).

Initial visual acuity was 20/20 in 33%, better than 20/40 in 51%, and 20/200 or worse in 21%. This indicates that the presence of normal visual acuity does not rule out NA-AION. A relative afferent pupillary defect will be present as long as the contralateral eye is normal. Visual field defects following a nerve fiber layer distribution are typical visual field findings, with inferior altitudinal and arcuate defects being the most common. The main clinical finding on ophthalmic evaluation at the onset of visual loss is optic disc edema which resolves spontaneously in eight weeks, resulting in the generalized or sectoral pallor of the optic disc. The disc edema may or may not be accompanied by peripapillary hemorrhages. Subretinal fluid is an unusual finding.[2,3,4]

In this report, we present an unusual case of a 69-year-old man affected by unilateral NA-AION with vitreous hemorrhage.

CASE REPORT

A 69 - year-old man presented with a complaint of sudden painless vision loss in the right eye for five days. Additionally, he reported that he started observing a large floater or blurred spot in his vision. His symptoms were constant in nature, the appearance and location of a remain unchanged when he looked in different directions. Additionally, he denied any past ocular surgery or trauma. He denied symptoms such as jaw claudication, scalp tenderness, malaise, fever and weight loss. His ocular history included age related macular degeneration treated

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with anti- vascular endothelial growth factor injections. The patient's last ocular examination was two weeks ago. His best-corrected visual acuity was 8/20 for the right eye and 16/20 for the left eye. The right eye had wet age-related macular degeneration without evidence of retinal fluid accumulation. His past medical history included hypertension. His medications included aspirin 100mg/day and benidipin hydrochloride 4mg/day.

On the present examination, a right afferent pupillary defect was present. The patient's best- corrected visual acuity was 1/20 for the right eye and 16/20 for the left eye. Anterior segment examination in both eyes was unremarkable. The intraocular pressure by applanation tonometry was 14 mmHg in the right eye and 15 mmHg in the left eye. The fundus of each eye was examined after pharmaceutical mydriasis with 0.5% tropicamide and 10% phenylephrine hydrochloride ophthalmic solutions. Assessment of the fundus revealed mild or moderate vitreous hemorrhage with an obscured view of the posterior pole in the right eye. (Figure 1a) The patient had no evidence of a retinal tear or detachment on 360 degrees scleral depressed examination. The ophthalmoscopy examination of the left eye showed no relevant changes. Fluorescence angiography revealed the masking of fluorescence due to intravitreal hemorrhage around the optic disc in the right eye. (Figure 1b) OCT (Optical coherence tomography) demonstrated peripapillary hemorrhage. (Figure 1c-d)

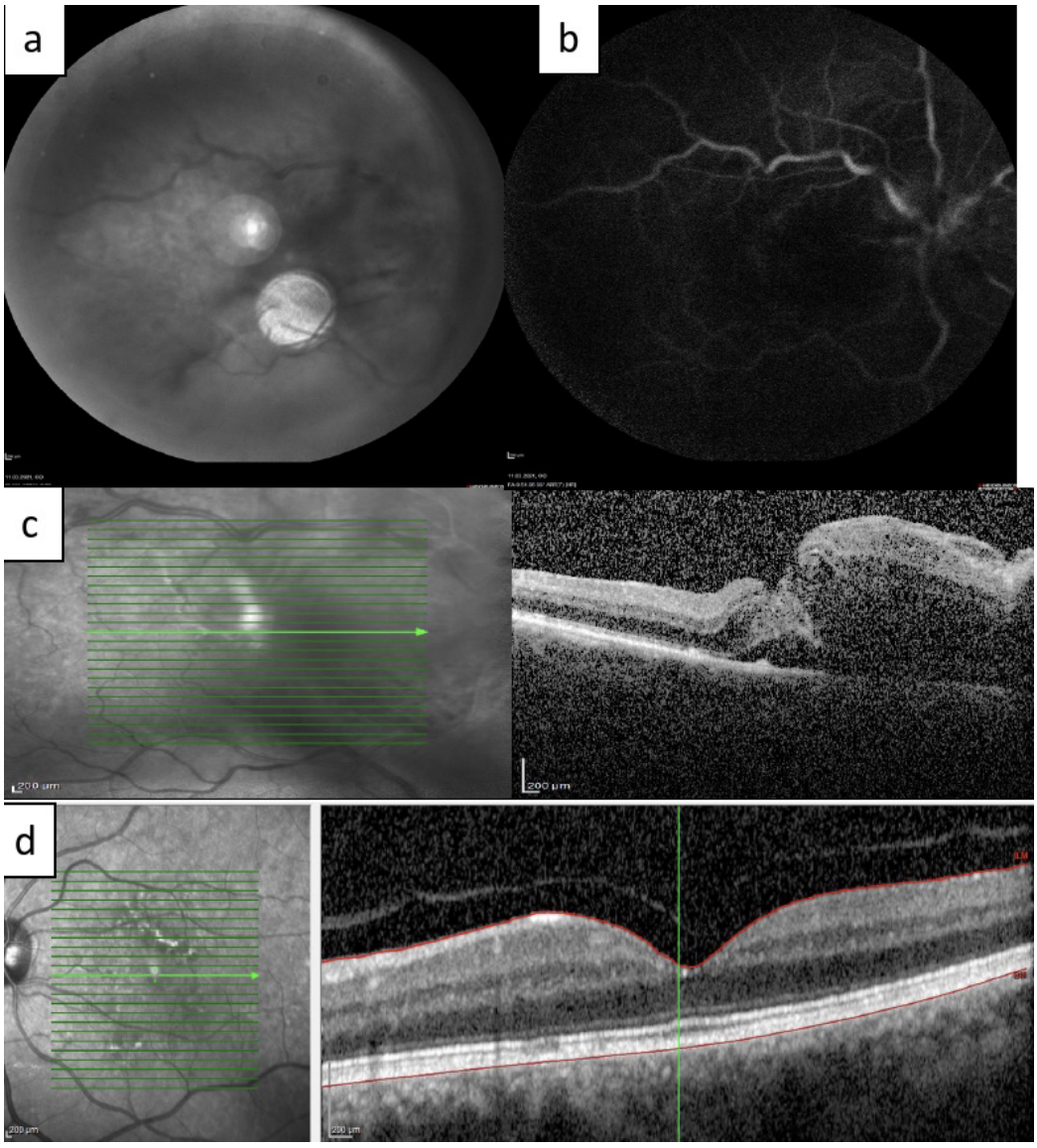

Figure 1: *(a) Red free fundus photography of the right eye at the initial visit shows vitreous hemorrhage (b) Fluorescence angiography at the initial visit shows the masking of fluorescence due to intravitreal hemorrhage around the optic disc in the right eye (c) Macular OCT at the initial visit shows peripapillary hemorrhage in the right eye (d) Macular OCT at the initial visit shows normal foveal depression in the left eye.*

The laboratory tests including complete blood counting, erythrocyte sedimentation rate and C-reactive protein were normal. Magnetic resonance imaging (MRI) brain/ orbits and magnetic resonance venography were unremarkable. Carotid doppler sonography was normal. Conservative measures such as head of a bed elevation are recommended to the patient. The visual acuity improved to 4/20 in the right eye eight days later. Color vision was 1/21 for the right eye and 21/21 for the left eye with ishihara pseudoisochromatic plates. Fundus examination showed marked disc swelling and peripapillary flame-shaped hemorrhage in the right eye. The periphery of the right eye was unremarkable 360 degrees. (Figure 2a-b) OCT demonstrated optic nerve edema and subretinal fluid extending to the macula in the

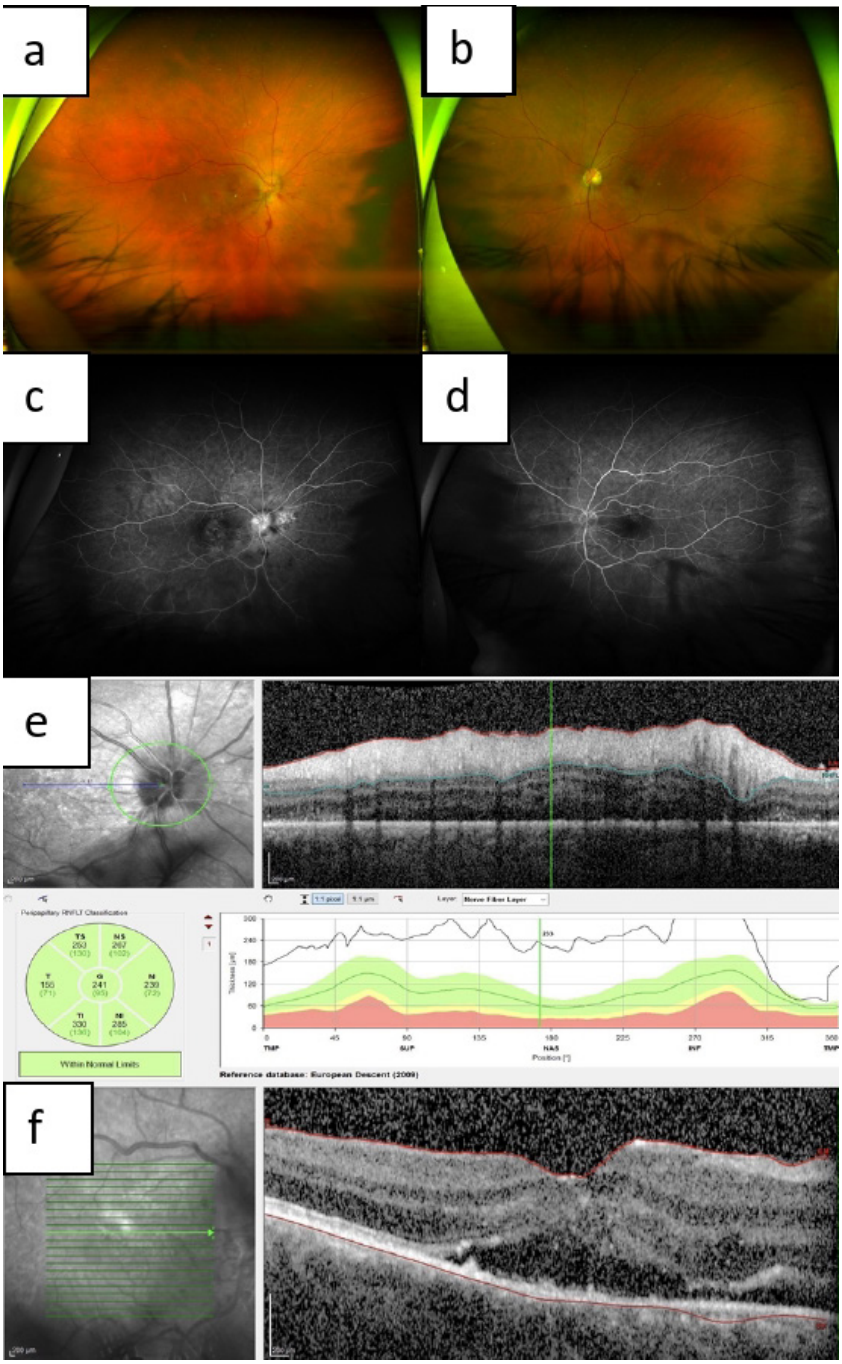

Figure 2: ***(a)*** *Color fundus fotography shows marked disc swelling and peripapillary flame-shaped hemorrhage in the right eye (b) and normal optic disc appearence in tne left eye (c)Fluorescence angiography shows hyperfluorescence of the disc at the late stage in the right eye (d) and normal optic disc appearence in the left eye (e) optic nerve OCT shows optic disc edema in the right eye (f) Macular OCT shows subretinal fluid extending to the macula in the right eye.*

right eye. (Figure 2e-f) Fluorescence angiography revealed hyperfluorescence of the disc at the late stage. (Figure 2c-d) In Humphrey visual field testing, there was an inferior altitudinal defect in the right eye. The left eye visual field showed an inferior rim artifact. (Figure 3a-b) Infectious (VDRL, HIV, hepatitis B-C, borrelia, bartonella) and ato-immune serology (antinuclear antibodies(ANA), anti-nuclear cytoplasmic antibodies(ANCA) anti-double-stranded DNA antibodies (anti-ds DNA) were all normal. The patient was diagnosed with presumed NA-AION with vitreous hemorrhage in the right eye. Brimonidine tartrate 0.2% twice daily for the right eye was initiated for neuro-protection. He was instructed to take his blood pressure medication in the morning if possible in order to reduce nocturnal hypoperfusion. He was advised to continue using anti-aggregant agent. İntravitreal injection for subretinal fluid was not considered because there was no profound vision loss.

At follow-up examination, at the end of the second month, the visual acuity improved to 8/20 in the right eye. Color vision and visual field defect did not improve in the right eye. The optic nerve edema and subretinal fluid resolved and the disc was slightly pale. (Figure 3c-d)

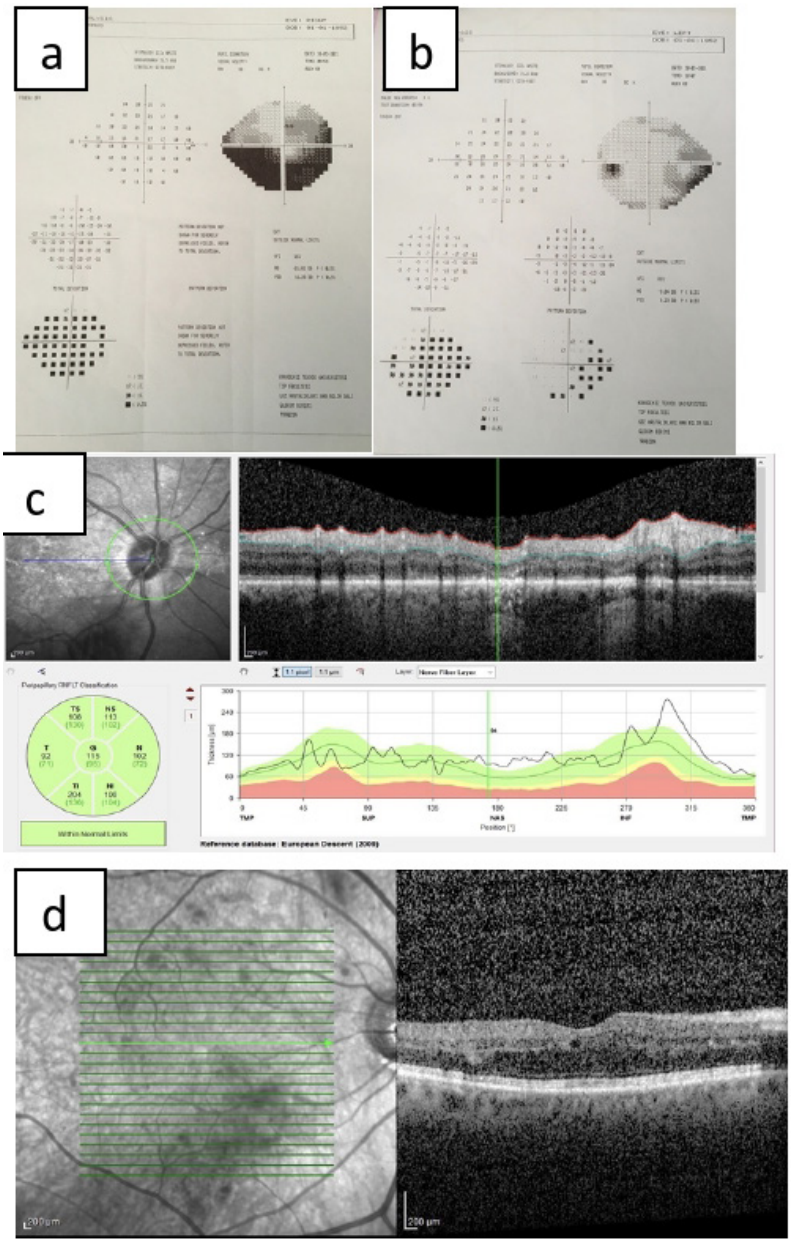

Figure 3: *(a) Humphrey visual field testing, shows an inferior altitudinal defect in the right eye. (b) and an inferior rim artifact in the left eye (c) Optic nerve OCT shows resolution of disc edema in the right eye (d) Macular OCT shows resolution of subretinal fluid in the right eye.*

The possible differential diagnoses of this patient included anterior ischemic optic neuropathy (AION), compressive optic neuropathy, hypertensive retinopathy, nonischemic central retinal vein occlusion and optic neuritis. Therefore, AION must be ruled out urgently. The patient in this report denied symptoms including scalp tenderness and jaw claudication, malaise ,fever and blood tests(ESR, CRP) were normal. Radiologic graphics excluded the diagnosis of compressive optic neuropathy. This patient's systemic history was positive for hypertension; however, it was not highly elevated at the time of his examination. Fundus examination did not show hemorrhages or cotton wool spots extending into the peripheral retina. Central retinal vein occlusion is a cause of unilateral, acute vision loss and hemorrhages are generally seen beyond the peripapillary area. However, in this case, hemorrhages were seen in the peripapillary area.

Our patient's age, gender and exam findings, such as normal extraocular eye movements excluded the diagnosis of optic neuritis.

Vitreous hemorrhage may develop secondary to posterior vitreous detachment (PVD). A hemorrhagic PVD strongly suggests there may be a retinal tear or detachment. However, our patient had no evidence of a retinal tear or detachment on 360 degrees scleral depressed examination.

CONCLUSION

Pathogenetically, NA-AION is of two types: the most common is caused by transient nonperfusion or hypoperfusion of the optic nerve head (ONH) circulation; the second, and rare one, is due to embolism to the arteries/ arterioles feeding the ONH. Common systemic risk factors for hypoperfusion of the ONH include arterial hypertension, diabetes mellitus, nocturnal arterial hypotension, cardiovascular disorders and hyperlipidemia. Ocular risk factors include absent or small cup in the optic disc, optic disc drusen, raised intraocular pressure and marked optic disc edema. Taking blood pressure medication at night can lead to nocturnal hypotension and thus hypoperfusion of the optic nerve. Systemic phosphodiesterase 5-inhibitors [5] and amiodarone[6] have also been linked to NA-AION, although the link is controversial. Embolic occlusion is a rare cause of NA-AION. Compared to the hypotensive type of NA-AION, the extent of ONH damage in this type is usually massive, severe, and permanent.

Unfortunately, there is no widely accepted treatment for acute NA-AION.

Fortunately, 40 percent of patients experience spontaneous improvement in visual acuity without treatment. According to Hayreh, improvement in visual acuity with systemic corticosteroids was demonstrated by reducing optic nerve head edema by reducing capillary permeability.[7] However, due to the lack of randomization in this study, it is not widely accepted. Mega-dose intravenous corticosteroid treatment was also proposed as a treatment for NA-AION.[8] The results were disappointing in that IV corticosteroids for NA-AION improved neither the visual acuity nor the visual field of NA-AION patients compared to untreated patients. Aspirin treatment failed because NA-AION is a hypotensive disorder, not a thromboembolic disorder. However, some reports showed that aspirin reduced the incidence of NA-AION in the fellow eye.[9]

The present case was 69 years old and had hypertension that could be considered as a predisposing factor for the development of acute NA- AION. Our case had a spontaneous improvement in visual acuity without treatment. Vitreous hemorrhage is an unusual finding with NA-AION. To the best our knowledge, this is the first-ever case report describing a vitreous hemorrhage in a patient with NA-AION. He reported that the appearance and location of the floater remain unchanged when he looked in different directions. Assessment of the fundus revealed mild or moderate vitreous hemorrhage with an obscured view of ONH in the right eye. We thought the vitreous hemorrhage was caused by the optic nerve. Although the exact pathogenesis of NA-AION remains unproven, it appears to be a multifactorial disease. It is presumed to be due to a transient disruption in the circulation of the ONH leading to hypoperfusion and ischemia. A transient ischemic event may cause ischemic swelling of the axons, leading to compression of capillaries in a restricted optic disc space. This "vicious cycle" of increased swelling causes more compression of the capillaries then induces further ischemia ultimately resulting in ischemic infarction of the ONH. Acutely, mechanical compression can damage venules allowing extravasation of blood into the vitreous. However, the exact cause of the vitreous hemorrhage has yet to be determined.

Subretinal fluid is also an unusual presentation in NA-AION not described as a part of the natural history of NA-AION. Hedges et al. reported that subretinal fluid occurs in about 15% of patients with NA-AION.[10] In our patient, fluorescence angiography did not show accumulation of dye in the macular region, indicating the fluid did not arise from the retinal vessels or directly from the choroid. Because of this, subretinal fluid may have escaped from the peripapillary choroid into the subretinal space and track into the macula. Subretinal fluid may contribute to some visual loss associated with NA-AION, and that resolution of the fluid may account for a portion of the visual improvement that often occurs with NA-AION.

Our main aim in presenting this case is that NA-AION may be accompanied by vitreous hemorrhage. Vitreous hemorrhage was cleared rapidly within eight days and typical clinical findings of NA-AION could be observed. In addition, our patient had subretinal fluid, another unusual finding. Another important practical implication is that routine use of macular OCT may be recommended for the detection of subretinal fluid in patients with NA-AION.

Declaration of conflicting interest

There is no conflict of interest

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Informed consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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