Case Report

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A giant in disguise: giant cell arteritis presenting with optic nerve edema without the usual symptoms – a case report

Abstract

Background: Giant cell arteritis (GCA) is a medical emergency that most often presents with headaches, jaw claudication, scalp tenderness, and systemic symptoms. Atypical or minimally symptomatic presentations can delay diagnosis and place vision at risk. Prompt recognition, laboratory evaluation, and early systemic corticosteroid therapy are essential to prevent irreversible vision loss.

Case report: A 74-year-old white male presented with one day of a brown hue, light flashes, eye pain, color vision change, and visual hallucinations in the left eye. He denied temporal headache and jaw claudication. Visual acuity was 20/20 in both eyes with no afferent pupillary defect, but the left optic nerve showed chalky, pale edema from 07:00-13:00 clock hour confirmed by optical coherence tomography. Non-arteritic anterior ischemic optic neuropathy was the leading differential, but because of the risk of impending vision loss he was sent for same-day laboratory testing. Erythrocyte sedimentation rate (73, [ref 0-15mm/hr]) and C-reactive protein (122, [ref <5mg/L]) were markedly elevated. He was admitted, received three days of intravenous corticosteroids, and a temporal artery biopsy performed four days after presentation was positive for GCA. He was transitioned to oral prednisone 60 mg daily. At two-week follow-up, the optic nerve edema had resolved, inflammatory markers had normalized, visual acuity remained 20/20, and a residual inferior arcuate defect persisted in the left eye.

Conclusion: GCA can be presented without the classic symptoms. Any edematous optic nerve in a patient over 50 years of age warrants investigation for GCA, and elevated inflammatory markers should prompt initiation of intravenous corticosteroids while awaiting temporal artery biopsy.

Keywords: giant cell arteritis, temporal arteritis, optic nerve edema, arteritic anterior ischemic optic neuropathy, erythrocyte sedimentation rate, C-reactive protein, corticosteroids

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Emma A Horstkamp, Virginia O Bice, Chad E Gosnell, Len V Koh
Mann-Grandstaff VA Medical Center, Spokane WA 99205, USA

Correspondence: Len V Koh OD PhD, Mann-Grandstaff VA Medical Center Eye Clinic Building 30, 4815 N Assembly Street, Spokane WA 99205, USA, Tel 509-901-0212

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Introduction

Giant cell arteritis (GCA), also known as temporal arteritis, is a granulomatous large-vessel vasculitis and a medical emergency that requires prompt referral and treatment to preserve vision. It is thought to occur when T cells migrate into the vessel wall and drive release of inflammatory cytokines in the vascular adventitia.[1] Age is the greatest risk factor, and people older than 50 years are almost exclusively affected. Women are about two and a half times more likely to develop GCA than men.[1]

Common symptoms include vision loss, jaw claudication, loss of appetite, malaise or fever, indigestion, scalp tenderness, temple pain, and headache. Less common manifestations reported in the literature include scalp necrosis, tongue claudication, tongue necrosis, amaurosis fugax, limb claudication, and blood pressure inequality between the arms.[2,3] Because GCA can present with any number of symptoms, patients who lack the classic features can be misdiagnosed, as illustrated by reports of GCA masquerading as migraine.[2]

Diagnosis relies on a positive temporal artery biopsy following elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) values, interpreted within the framework of the 2022 American College of Rheumatology/EULAR classification criteria.[4] Intravenous glucocorticoids are first-line therapy and are followed by long-term oral glucocorticoids.[1] We report a case in which GCA presented as unilateral optic nerve edema in the absence of temporal headache or jaw claudication.

Case report

A 74-year-old white male presented to the eye clinic with a one-day history of a brown hue over the left side of his vision, light flashes in the left eye, left eye pain, color vision changes in the left eye, and visual hallucinations in the left eye. He denied temporal headache and jaw claudication. Also, he had no other symptoms of scalp tenderness, malaise, fever, weight or appetite loss.

His past ocular history was significant for chronic iritis and mild non-proliferative diabetic retinopathy. His past medical history was significant for atrial fibrillation, benign prostatic hyperplasia, hypertension, type 2 diabetes mellitus, immune thrombocytopenia, morbid obesity, obstructive sleep apnea, subdural hematoma, and shingles on the face without ophthalmic manifestation. His medications included apixaban, carvedilol, empagliflozin, fluocinolone, hydrochlorothiazide, lisinopril, metformin, rosuvastatin, and tamsulosin.

At the initial visit, uncorrected visual acuity was 20/20-2 OD and 20/20-1 OS. Pupils were equal, round, and reactive to light with no afferent pupillary defect. Extraocular motilities and confrontation fields were full in both eyes. Intraocular pressures were 12 mmHg in each eye. Ishihara color vision testing was equal and normal in both

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eyes despite the patient's subjective color complaint. Blood pressure was 101/69 mmHg in the right arm, seated.

Dilated fundus examination of the right optic nerve showed no edema. The left optic nerve showed edema from 0700 to 0200 that was chalky and pale, with no hemorrhages (Figure 1). The cup-to-disc ratios were 0.30 in the right eye and 0.15 in the left eye. Optic nerve head radial and retinal nerve fiber layer (RNFL) optical coherence tomography (OCT) confirmed edema from 0700 to 0200 in the left eye (Figure 2).

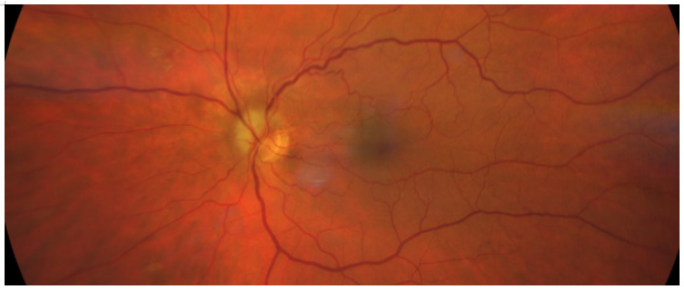


Figure 1 Left optic nerve edema at initial presentation.

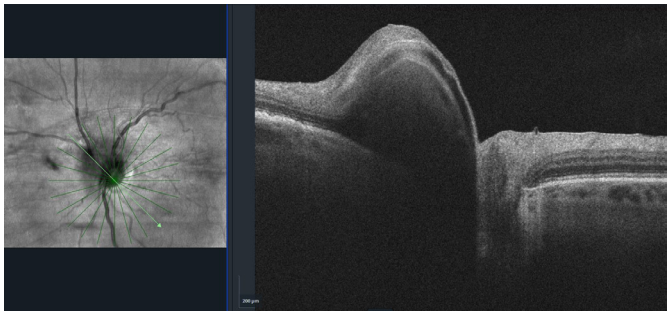


Figure 2 Superior-nasal to inferior-temporal high-definition radial optic nerve head OCT showing edema.

At this presentation, nonarteritic anterior ischemic optic neuropathy (NAION) was the leading differential diagnosis, since the patient was minimally symptomatic for arteritic anterior ischemic optic neuropathy (AAION). Given the risk of vision loss, however, he was sent to urgent care for same-day laboratory testing. ESR was 73 and CRP was 122, both markedly elevated. Computed tomography (CT) and CT angiography showed no acute intracranial abnormality, chronic occlusion of the left middle cerebral artery, moderate stenosis of both cavernous internal carotid arteries, and no significant stenosis of the posterior circulation.

The patient was admitted for a temporal arteritis workup and received intravenous corticosteroids for three days. He was referred for temporal artery biopsy within two weeks; the biopsy was performed four days after initial presentation. The final pathologic diagnosis of the biopsy was artery with mild chronic inflammation and focal disruption of the internal elastic lamina to include rare giant cells, consistent with temporal arteritis. Treatment was changed to oral prednisone 60 mg once daily, to be continued indefinitely.

At the two-week follow-up, visual acuity was 20/20 in both eyes. Pupils remained without an afferent pupillary defect, extraocular motilities were full, and intraocular pressures were 11 mmHg in the right eye and 10 mmHg in the left eye. Confrontation fields were full in the right eye with inferior constriction in the left eye. The left optic nerve edema had resolved (Figure 3), although a disc hemorrhage was noted superiorly and the superior-nasal margin remained slightly indistinct. RNFL OCT showed no edema in the right eye and RNFL thicknesses of 168 µm superiorly and 99 µm nasally in the left eye (Figure 5) (Figure 4). The 30-2 Humphrey visual field was clean in the right eye and showed an inferior arcuate defect in the left eye (Figure 6) (Figure 7); reliability was low in the right eye because of excessive fixation losses. ESR had fallen to 4 and CRP to 1.08. The patient established care with a rheumatologist for long-term management (Table 1).

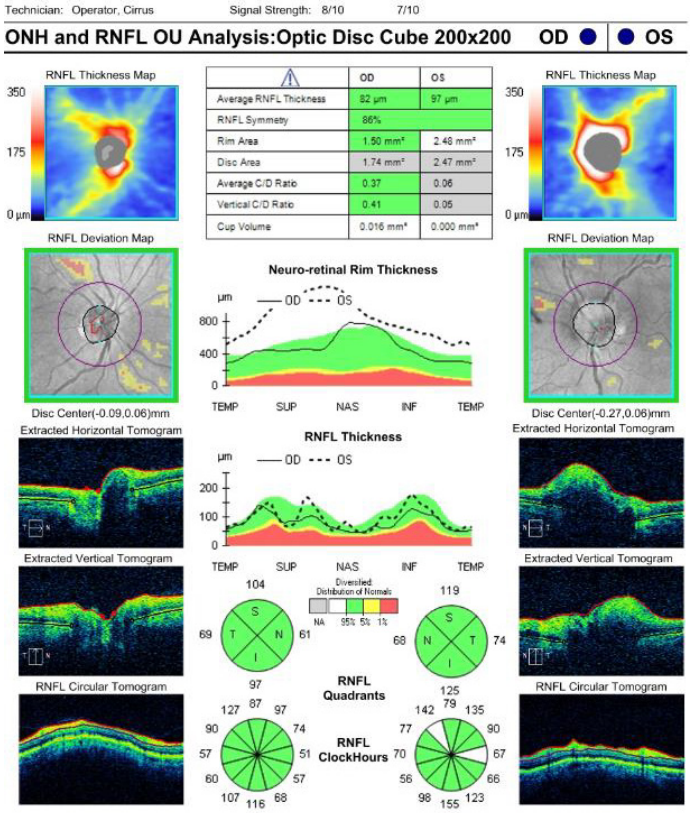


Figure 3 Resolved left optic nerve edema at the two-week follow-up.

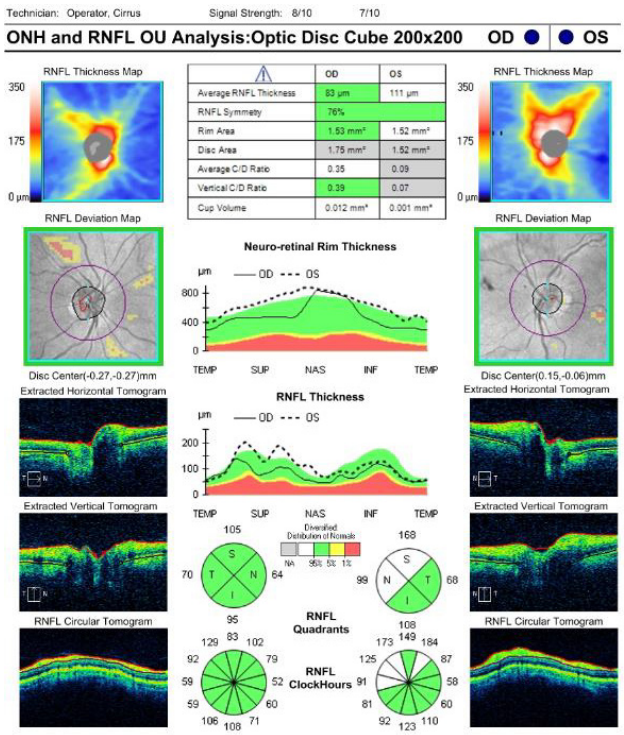


Figure 4 Optic nerve head RNFL OCT at the initial visit.

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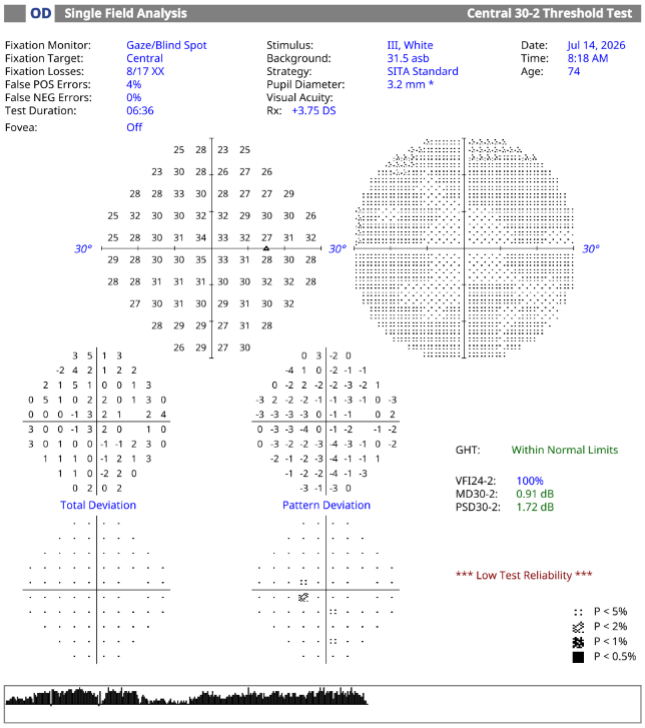


Figure 5 Optic nerve head RNFL OCT at follow-up.

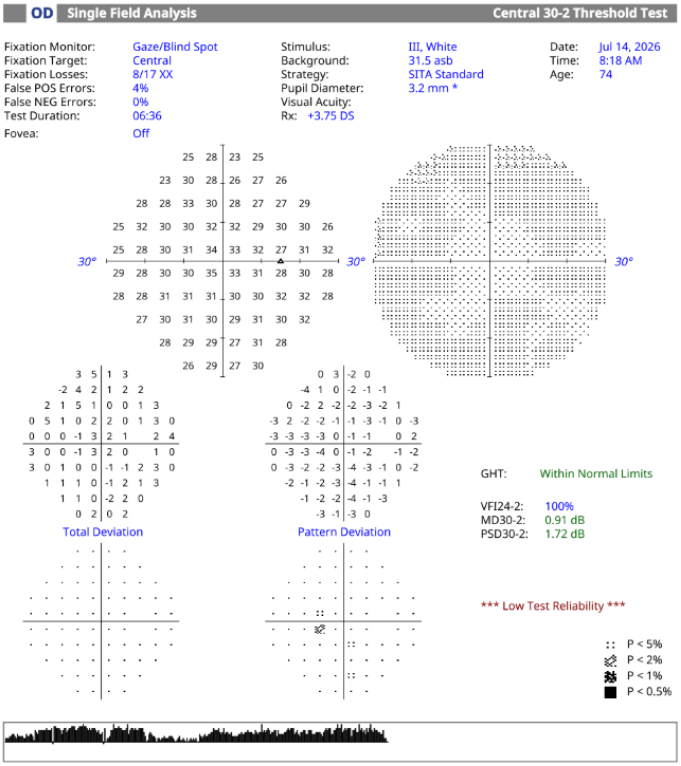


Figure 6 30-2 Humphrey visual field of the right eye at follow-up; low test reliability due to excessive fixation losses, otherwise clean.

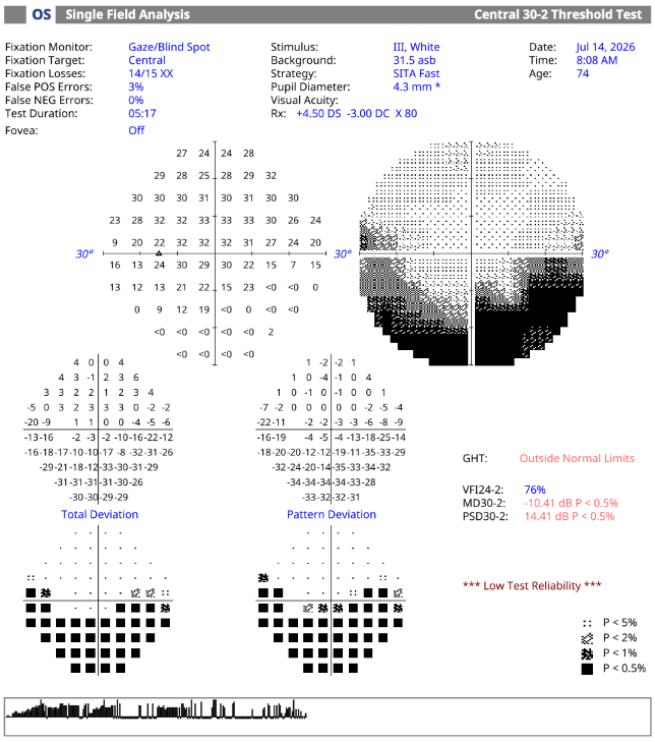


Figure 7 30-2 Humphrey visual field of the left eye at follow-up; low test reliability, with an inferior arcuate defect.

Table 1 summarizes the findings at presentation and follow-up

Parameter	Initial visit	Two-week follow-up
Visual acuity	OD 20/20-2; OS 20/20-1	OD 20/20; OS 20/20
Pupils	PERRL, no APD	PERRL, no APD
Confrontation fields	Full OU	Full OD; inferior constriction OS
IOP (mmHg)	12-Dec	11-Oct
Left optic nerve	Edema 0700-0200, chalky and pale, no hemorrhage	No edema; superior disc hemorrhage, slightly indistinct superior-nasal margin
RNFL OCT (OS)	Edema 0700-0200	168 µm superior, 99 µm nasal
30-2 visual field	Not performed	OD clean; OS inferior arcuate defect
ESR	73 [ref. 0-15mm/hr]	4
CRP	122 [< 5 mg/L]	1.08

Summary of clinical findings and laboratory values at initial presentation and at the two-week follow-up. APD, afferent pupillary defect; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; IOP, intraocular pressure; OCT, optical coherence tomography; RNFL, retinal nerve fiber layer.

Discussion

GCA is a rare medical emergency, and good visual outcomes depend on prompt referral and treatment.[1] Our patient illustrates how GCA can present with ocular findings alone. He had no temporal headache and no jaw claudication, and his visual acuity, pupillary responses, and color vision testing were normal. The only objective abnormality was a pale, chalky edematous left optic nerve, together with unusual visual phenomena including light flashes and visual

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hallucinations. Other symptoms of GCA described in the literature, such as scalp necrosis, tongue claudication and necrosis, amaurosis fugax, limb claudication, and blood pressure inequality, were not present, which underscores that the disease can present with any number of symptoms.[2,3]

The leading differential diagnoses were AAION and NAION. Others considered were orbital optic nerve tumor, optic neuritis, diabetic papillitis, hypertensive optic neuropathy, and posterior uveitis. This patient carried several vascular risk factors common to NAION, including hypertension, diabetes, obstructive sleep apnea, atrial fibrillation, and chronic occlusive cerebrovascular disease, and he was minimally symptomatic for AAION. These factors made NAION a reasonable initial impression and might have led to a less urgent workup. We feel it was the combination of an edematous optic nerve and age over 50 years that justified same-day laboratory testing, which revealed markedly elevated ESR and CRP values and changed management.

Because delay in treatment risks permanent vision loss, elevated inflammatory markers should prompt a temporal arteritis workup with intravenous corticosteroid administration while awaiting biopsy.[1,4] Our patient received three days of intravenous steroids, and the biopsy performed four days after presentation remained positive. This supports the practice of starting treatment before tissue confirmation. He was then transitioned to oral prednisone 60 mg daily, in keeping with the standard approach of intravenous glucocorticoids followed by long-term oral glucocorticoids.[1] ESR and CRP dropped significantly within two weeks, the optic nerve edema resolved, and visual acuity remained 20/20. A residual inferior arcuate defect in the left eye was nonetheless present, a reminder that visual field loss may persist despite prompt therapy. Long-term management was coordinated with rheumatology.

Conclusion

GCA can present with any number of symptoms, including optic nerve edema in the absence of temporal headache and jaw claudication. Eye care providers should investigate any edematous optic nerve in persons above 50 years of age, even without classic temporal arteritis symptoms. Elevated ESR and CRP values should prompt a temporal arteritis workup with intravenous steroid administration while waiting for a temporal artery biopsy result, as prompt recognition and treatment are central to preserving vision.

Acknowledgement

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Conflicts of interest

The authors declare that they have no known competing financial interests or personal relationships that appeared to influence the work reported in this study.

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