



Case Study: Arteritic Anterior Ischemic Optic Neuropathy

Submitted By: **Todd J. Purkiss, MD, PhD** – Seeing Patients in Louisville, Elizabethtown, and Frankfort

CASE PRESENTATION

A 55-year-old white male presented with progressive blurred vision and visual field loss in the left eye for one week. His medical history included type 2 diabetes and hypertension, both well controlled with medication.

EXAMINATION AND COURSE

Best corrected visual acuity measured 20/25 in the right eye and 20/100 in the left. Intra-ocular pressures were normal, and no afferent pupillary defect was detected. Confrontation fields were full, though testing of the left eye showed mild delay and uncertainty.

Dilated fundus exam revealed left optic disc edema with flame hemorrhages (**Figure 1**). The right eye was normal, and no diabetic retinopathy was seen. OCT showed normal retinal nerve fiber layer (RNFL) thickness of 104 microns OD and significant thickening of 395 microns OS (**Figure 2**).

The differential diagnosis for unilateral optic nerve swelling included diabetic papillopathy versus anterior ischemic optic neuropathy (AION). Given the absence of other diabetic eye disease, papillopathy was unlikely. AION exists in two forms: non-arteritic, commonly linked to vascular risk factors, and arteritic, associated with giant cell arteritis (GCA). GCA can present with headache, scalp tenderness, jaw pain, or weight loss. The patient reported recent significant weight loss without other classic symptoms. His only diabetes medication was metformin, not typically associated with weight loss.

Screening labs for GCA—erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and platelets—were obtained. ESR was elevated at 80 mm/hr (normal <20) and CRP at 13.5 mg/L (normal <8), while platelets were normal at 283,000. Though nonspecific, the elevated ESR/CRP raised strong suspicion for GCA. Because untreated GCA can cause bilateral blindness and stroke, high-dose oral prednisone (80 mg) was initiated, and temporal artery biopsy was recommended.

The first surgeon who evaluated him suspected non-arteritic AION, stopped prednisone, and deferred biopsy. On follow-up, left optic nerve swelling had improved and vision was 20/80, but the right eye now showed disc edema and decreased vision to 20/50 (**Figures 3 & 4**). Prednisone was resumed, and biopsy was performed by a second surgeon, confirming GCA.

At his most recent visit, vision measured 20/25 OD and 20/50 OS. Edema had largely resolved, though optic nerve pallor OS suggested permanent damage (**Figures 5 & 6**). Because of steroid-induced hyperglycemia, he will transition to a steroid-sparing immunosuppressant under rheumatology care. Long-term therapy, likely at least a year, will be required.

DISCUSSION

Giant cell arteritis is the most common primary systemic vasculitis in adults, affecting an estimated 160,000 Americans annually. Onset typically occurs after age 50, with a median presentation age of 75. Women are more frequently affected, though men may also present with vision-threatening disease.

The cause of GCA is not fully understood but appears to involve a maladaptive autoimmune response leading to granulomatous inflammation of medium- and large-sized arteries. Ocular involvement is common, but systemic risks include myocardial infarction and stroke.

Prompt recognition and treatment are critical, as visual loss can rapidly progress to both eyes and become permanent. High-dose corticosteroids remain first-line therapy, though relapses may occur during tapering. Transition to steroid-sparing immunosuppressants (such as methotrexate or tocilizumab) is often needed to minimize systemic steroid side effects.

This case highlights the importance of early suspicion for GCA in patients presenting with unilateral optic nerve edema, especially when inflammatory markers are elevated and weight loss or systemic symptoms are present. Timely initiation of steroids before biopsy confirmation can be vision-saving.

Figure 1

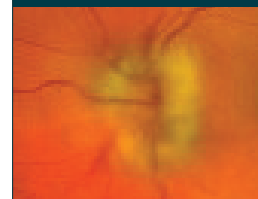


Figure 2

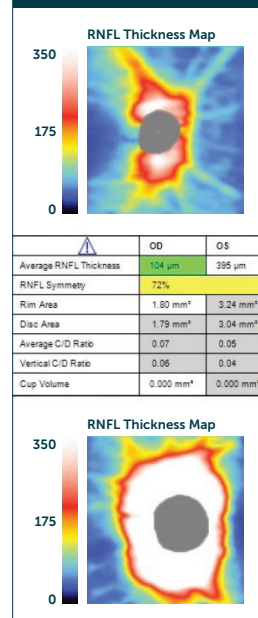


Figure 3



Figure 4

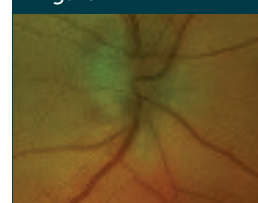


Figure 5

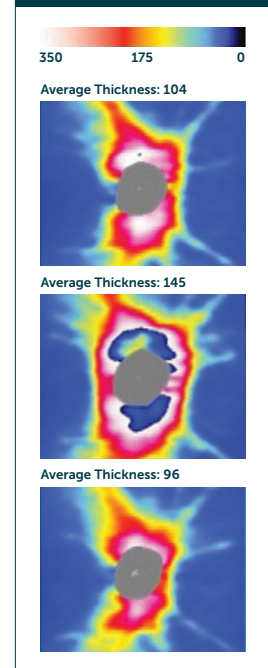
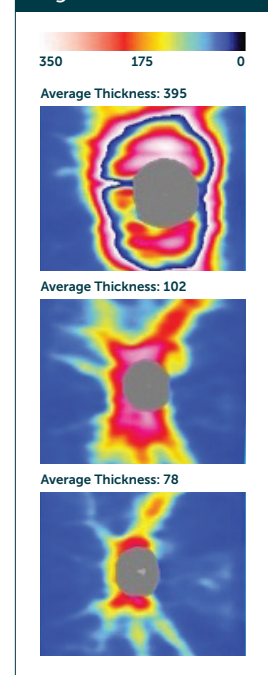


Figure 6





Case Study: A Case of Retinal Detachment with Retinoschisis

Submitted By: **Thomas W. Stone, MD** – Seeing Patients in Louisville, Lexington, and Jeffersonville, IN | Contributing Author: **Paras Vora, MD**

Figure 1



Figure 1: Widefield pseudocolor fundus photo demonstrating a bullous retinal detachment with retinoschisis in the same quadrant.

Figure 2

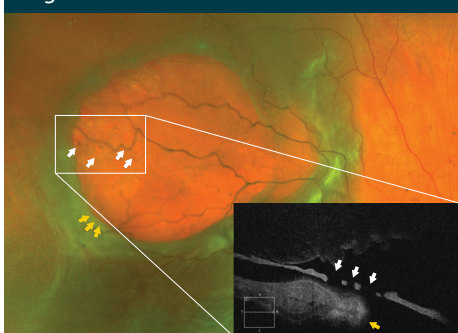


Figure 2: Focused on the area of retinoschisis. The white arrows indicate small inner retinal holes, and the yellow arrows highlight the rolled edge of a large outer retinal break. The posterior hyaloid, which is elevated in this patient, allows liquefied vitreous to pass through the inner and outer retinal holes, leading to retinal detachment (as shown in the fundus photo).

CASE PRESENTATION

A healthy 65-year-old male was referred for symptomatic flashes and floaters in the right eye that began one week prior. He has history significant for mild cataract and, otherwise, an unremarkable medical history. There is no family history of eye problems. On presentation, he noted he developed visual field loss three days prior, which prompted his visit to the eye doctor.

Visual acuity on presentation was 20/25 in the right eye, and 20/20 in the left eye. He had normal intraocular pressures. Visual field on confrontation was notable for an inferonasal field defect of the right eye.

On anterior examination, he was noted to have a nuclear sclerotic cataract, as well as pigment in his anterior vitreous.

Funduscopy examination revealed media opacity with significant pigment. Attention was drawn to a superotemporal bullous area of elevated retina, corresponding to the patient's visual field defect (**Figure 1**). Corrugations were noted along the inferior aspect of the elevated area, concerning for a rhegmatogenous retinal detachment; curiously, no retinal tear was discovered on scleral depression. An area of retinoschisis was noted in the same quadrant.

Zooming in on the superotemporal quadrant, we found multiple inner retinal holes in the area of retinoschisis, surrounded by a large outer retinal hole (**Figure 2**). The OCT camera was steered to this quadrant, and a line scan was obtained demonstrating an elevated posterior hyaloid over the schisis, with multiple inner retinal holes (white arrows) and the edge of a large outer retinal hole (yellow arrow).

An OCT of the macula revealed subretinal fluid encroaching on the superotemporal macula (**Figure 3**).

DIAGNOSIS

The patient was diagnosed with a schisis involving retinal detachment. It can be challenging to differentiate between retinoschisis, chronic rhegmatogenous detachment, and retinoschisis with rhegmatogenous detachment, and management varies depending on this diagnosis.

Retinoschisis refers to the splitting of the neurosensory retina. Typically, in the degenerative or senile form, these can be found incidentally. Inner retinal holes are usually of no consequence; however, the presence of both outer and inner retinal holes can lead to a retinal detachment. While the funduscopy exam in our case had corrugations consistent with rhegmatogenous detachment, and the patient was symptomatic due to the progressive scotoma, the use of OCT imaging proved indispensable in confirming the diagnosis.

Isolated retinoschisis without retinal breaks or progression can be observed; patients are usually asymptomatic when these are discovered. Progressive schisis with the presence of outer retinal breaks or associated retinal detachment require surgical treatment, especially if symptomatic. Patients with combined retinoschisis and retinal detachment tend to have similar outcomes as those with standard rhegmatogenous retinal detachment alone, although identifying inner retinal holes can be challenging, as scleral depression is not as effective at identifying inner breaks preoperatively. In our case, the patient underwent pneumatic retinopexy to stabilize the macula and subsequently underwent scleral buckle with pars plana vitrectomy. The patient's retina remains attached with a long-acting gas tamponade.

Figure 3

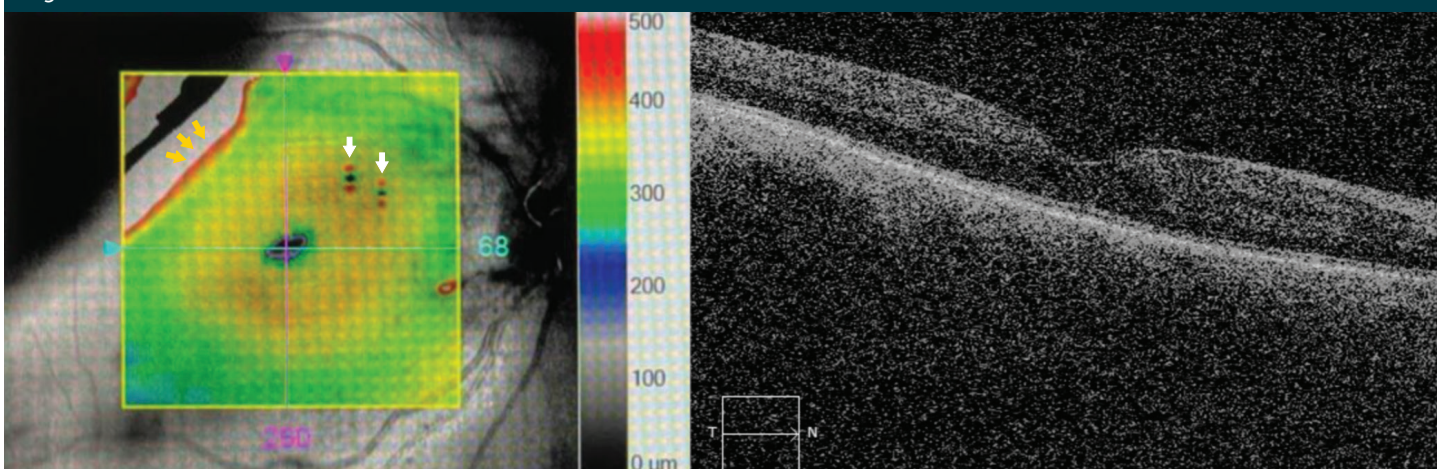


Figure 3: Focused on the area of retinoschisis. The white arrows indicate small inner retinal holes, while the yellow arrows highlight the rolled edge of a large outer retinal break. The posterior hyaloid, which is elevated in this patient, allows liquefied vitreous to pass through both the inner and outer retinal holes—contributing to the resulting retinal detachment (as shown in the OCT image).



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— John W. Kitchens, MD

Meet Our Fellows

Retina Associates of Kentucky is proud to partner with the University of Kentucky to offer a joint fellowship program, providing excellent post-residency fellowships in diseases and surgeries of the retina.



Paras Vora, MD

Paras Vora, MD, graduated from Washington University in St. Louis in 2015 with a Bachelor of Science in biomedical engineering and computer science. Dr. Vora then relocated to Lexington, Kentucky, obtaining his medical degree in 2020 from the University of Kentucky College of Medicine. Dr. Vora continued his education at the University of Kentucky, completing his residency in the Department of Ophthalmology and Visual Sciences. He was recognized with the Mark Gross Research Award and the Development and Innovation Award during this time. Dr. Vora has also been the recipient of the Outstanding Leadership and Community Service Award from the University of Kentucky.



Nicholas Fowler, MD

Nicholas Fowler, MD, received his undergraduate degree from Ohio Wesleyan University and completed his medical degree and ophthalmology residency at the University of Kentucky, where he served as co-chief resident. Outside of ophthalmology, he enjoys running, playing basketball, and spending time with his family.



Matthew Pfannenstiel, MD

Matthew Pfannenstiel, MD, grew up in Lawrence, Kansas, graduating with a Bachelor of Science in behavioral neuroscience from the University of Kansas in 2017. He then went on to earn his medical degree from the University of Kansas School of Medicine in Kansas City in 2021. He remained in Kansas City to complete an internship in internal medicine and his residency in ophthalmology at the University of Kansas Medical Center. During his residency, he was recognized with the Nellie V. Warren Rising Star Award and was selected by the faculty to serve as chief resident during his final year. He has presented his research on vitreoretinal surgery at several national conferences, including AAO, ASRS, and ARVO. Outside of ophthalmology, he enjoys playing guitar in a 1960s and 1970s cover band, golfing, and cheering on the Kansas Jayhawks and Kansas City Chiefs.

A Message of Gratitude To our valued colleagues in the eye care community, we thank you for your support and entrusting us with the care of your patients. We look forward to our continued collaboration!

Come Visit Us!

If you would like to observe any of our physicians in clinic or the operating room, please contact Crystal Powell, Practice Liaison, at 859.208.1589 or cpowell@retinaky.com. We look forward to hosting you!

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OFFICE EXPANSION AND RENOVATION

We officially welcomed our first patient to the newly renovated and expanded 4th-floor suite of our Lexington office on October 20, 2025. The upgraded space provides a more comfortable and efficient environment for both patients and staff, reflecting our continued growth and commitment to exceptional care.

OUR PHYSICIANS

Thomas W. Stone, MD
John W. Kitchens, MD
Todd J. Purkiss, MD, PhD
Belinda Shirkey, MD
Blake Isernhagen, MD
Jack Hollins, MD
Miquel Busquets, MD
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