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Clinical challenges

Vision loss with bending over

Michele D. Lee, BA[a], *Jeffrey G. Odel, MD*[a,*], *Danielle S. Rudich, MD*[a], *Robert Ritch, MD*[b], *Mark L. Moster, MD*[c,d]

[a] *Department of Ophthalmology, Columbia University, New York, New York, USA*
[b] *Einhorn Clinical Research Center, New York Eye and Ear Infirmary of Mount Sinai School of Medicine, New York, New York, USA*
[c] *Department of Neuro-Ophthalmology, Wills Eye Hospital, Philadelphia, Pennsylvania, USA*
[d] *Departments of Neurology and Ophthalmology, Thomas Jefferson University School of Medicine, Philadelphia, Pennsylvania, USA*

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(In keeping with the format of a clinical pathologic conference, the abstract and key words appear at the end of the article.)

1. Case report

A 66-year-old African American man with diabetes, hypertension, and hyperlipidemia had a 7-day history of transient episodic visual loss OD. He had advanced glaucoma and had undergone a trabeculectomy OD 5 years, and OS 2 years, prior to presentation. Despite low intraocular pressures (IOP), he continued to experience peripheral visual field loss. Three months previously he was found to have elevated IOP OD and received an injection of bevacizumab for vascularization of the filtering bleb, but his IOP was not lowered. At the time of presentation, he was taking dorzolamide/timolol drops twice a day and travoprost drops daily in both eyes.

The day before his vision loss began, he underwent unsuccessful bleb needling OD for scarring and elevated IOP. That evening, after opening his eyes following drop administration, he noticed a "cloud" in his vision and reported mild discomfort OD. He noticed afterward that he had immediate and transient painless loss of vision when he bent down, with recovery of his vision after tilting his head back for 20–30 minutes. This change in his vision was prominent on awakening. He described the change in his vision akin to a "steam room" with static gray steam that would last for 20–30 minutes with gradual clearing.

His medications included metformin, losartan, and rosuvastatin. He denied any systemic symptoms. There was no recent trauma or increased life stressors and no history of a stroke, transient ischemic attack, or other cardiovascular event. He denied any redness in his eyes during episodes of vision loss.

Given the pattern of his visual loss and the cardiovascular risk factors, he was referred for neuro-ophthalmic evaluation. His visual acuity was 20/20 OU. He was unable to see any AO-HRR color plates OU, unchanged from previous examinations. There was no nystagmus or motility abnormality. Pupils were

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* Corresponding author: Jeffrey G. Odel, MD, Columbia University Harkness Eye Institute, 635 West 165th Street, New York, NY 10032, USA.
E-mail addresses: Odel1@aol.com, jgo1@columbia.edu (J.G. Odel).

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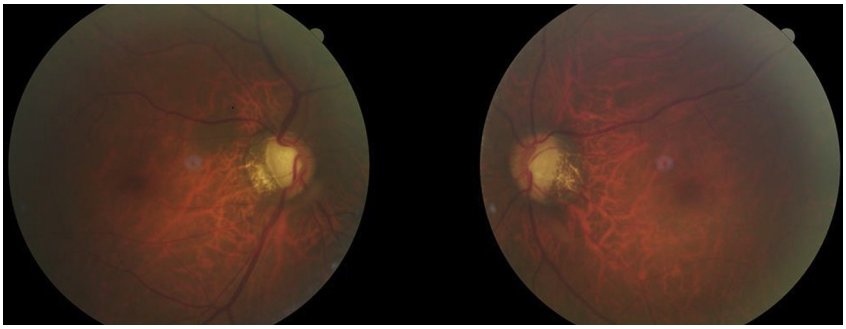

Fig. 1 – Fundus photographs. Bilateral disk cupping and pallor, with a slightly engorged vein OD superior to the optic disk.

round, with no relative afferent pupillary defect. Slit-lamp examination was notable for a non-leaking cystic bleb, mild conjunctival injection, and trace cell OD. Intraocular pressure was 14 mm Hg OD and 8 mm Hg OS, consistent with IOP from his recent examinations. There was glaucomatous cupping in both eyes (Fig. 1). There was a slightly engorged vein superior to the optic disk OD.

At this point, what are your considerations and how would you proceed?

1.1. *Comments by Mark Moster, MD*

In this 66-year-old man with risk factors of diabetes mellitus, hypertension, and hyperlipidemia presenting with transient monocular visual loss (TMVL) the leading diagnosis would normally be a transient ischemic attack (TIA). The postural nature of the TMVL and the onset on the day of needling the bleb, however, suggest a different etiology.

In most patients of this age with TMVL, my first recommendation is to rule out giant cell arteritis (GCA) and obtain erythrocyte sedimentation rate, C-reactive protein, and platelet count. I doubt GCA in this patient as he has no systemic symptoms, the episodes are longer than usual for GCA, and GCA is uncommon in African Americans. Nonetheless, because of the high-risk nature of GCA, these labs should be checked.

In a patient who is seen within days of a TIA, my workup would include noninvasive carotid imaging, echocardiography, blood work for lipids and hemoglobin A1C, and magnetic resonance imaging with diffusion-weighted imaging looking for evidence of asymptomatic infarct, which would increase the risk of impending stroke.

Intermittent angle closure is a consideration, but not likely without pain and no indication of a narrow angle on examination.

Two features of the history in this patient suggest an alternative diagnosis. These include the postural nature of the TMVL and the onset on the day of needling the bleb. This might suggest an intermittent hyphema, and I would try to reproduce the symptoms by having the patient bend over and re-examine his anterior chamber looking for red blood cells.

2. Case report (continued)

Although there was concern for a vascular etiology of the amaurosis fugax, the description of his vision loss and the positional aspect was unusual and warranted further investigation. He was re-examined after inducing his visual symptoms by asking him to bend down. Fundus photography taken at the onset of visual loss gave only a hazy view of the posterior pole, with slow clearing as seen with an image acquired over 20 minutes (Fig. 2). During this attack, there was no corneal edema, and his IOP remained in the low teens in both eyes.

What is your differential diagnosis now and what additional tests would you perform?

3. Comments (continued)

3.1. *Comments by Dr. Moster*

With a postural induced change, hazy view of the fundus, and subsequent clearing I would worry about an intermittent hyphema. If he were pseudophakic, I would be concerned about UGH (uveitis, glaucoma, hyphema) syndrome. My next step would be to try to induce the symptoms by having him bend over and obtain a good anterior segment examination, including gonioscopy, looking for a leaking vessel.

4. Case report (concluded)

The image series led us toward an ocular media etiology, and we re-examined him during an episode of visual loss. During this episode, his IOP was 13 mm Hg, there was 4+ dispersed red blood cells in the anterior chamber OD, and a thin layer of red blood cells was smeared across his cornea. Gonioscopy revealed a small hyphema in the inferior angle OD, with no actively bleeding vessels or other clear source of the hyphema except a tiny capillary vessel seen within the bleb (Fig. 3).

Four days later, the patient reported no change in his visual symptoms and slit-lamp examination was unchanged from initial presentation except for a subjectively darker hyphema OD. Three weeks after his symptoms began, he reported that his symptoms had resolved, and there was no capillary vessel or hyphema and a paucity of cell visualized on exam.