

Unilateral Progressive Glaucoma at Low IOPs

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CASE PRESENTATION

A 64-year-old male physician presented in October 2008 with progressive visual field damage in his right eye, the IOP of which never exceeded the midteens. Despite undergoing surgery on the eye with the Trabectome (NeoMedix Corporation, Tustin, CA) in June 2008, he continued to demonstrate a progressive visual field decline and was subsequently started on latanoprost and timolol/dorzolamide eye drops by his ophthalmologist.

The patient's past ocular history included bilateral LASIK in 2002. His IOP prior to laser refractive surgery was 17 mm Hg on two separate occasions in 1991 and 1994. The patient's past medical history was significant for the irradiation of enlarged tonsils at age 5, acoustic neuroma at age 28, and thyroid carcinoma at age 30. In addition, he had hypertension controlled on medications and exercise-induced asthma for which he required short courses of prednisone.

On examination, the patient's BCVA was 20/25 OD and 20/20 OS. His IOP measured 7 mm Hg OD and 11 mm Hg OS with Goldmann applanation tonometry, and the central corneal thickness was 472 μ m OD and 537 μ m OS. His color vision was normal bilaterally. Gonioscopy showed open angles up to the scleral spur in both eyes, with scarring in the nasal angle of the right eye from the Trabectome procedure. The slit-lamp examination revealed moderate hyperemia and minimal cataract in both eyes. The right optic nerve had advanced glaucomatous damage with a loss of the superior and inferior rim, whereas the left optic nerve was relatively normal in appearance with a well-preserved neuroretinal rim. Neither eye exhibited optic nerve pallor. Standard automated perimetry revealed a superior hemifield defect splitting fixation in the patient's right eye and a normal

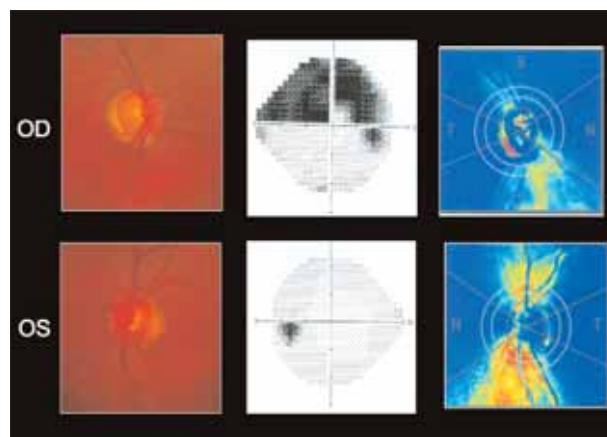


Figure 1. Advanced glaucomatous damage of the right optic nerve with corresponding damage of the visual field. Note the severe attenuation of the retinal nerve fiber layer on the GDx VCC scan.

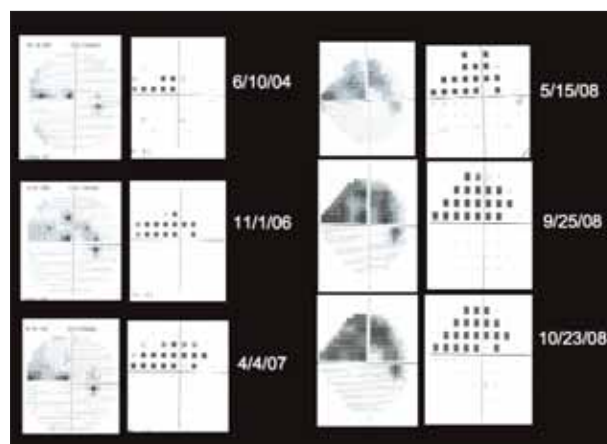


Figure 2. The superior visual field defect in the patient's right eye became progressively worse between 2004 and 2008.

visual field in his left eye. Testing with the GDx VCC (Carl Zeiss Meditec, Inc., Dublin, CA) confirmed severe thinning of the retinal nerve fiber layer in the right eye (Figure 1). A review of the patient's prior visual field records (2004 to 2008) showed rapid progressive worsening of the superior defect in his right eye (Figure 2).

"Despite the high likelihood that this patient has glaucoma, the asymmetric structural and functional damage is sufficiently atypical to warrant further investigation."

A modified diurnal test was obtained with IOP measurements every 2 hours while the patient was in a seated position from 8:00 AM to 2:30 PM. The peak and trough IOPs were 9 and 5 mm Hg OD and 12 and 9 mm Hg OS, respectively. After a detailed discussion with the patient of the prognosis and the risks and benefits of therapy versus no therapy, a decision was made to monitor the patient with serial visual fields and to start him on memantine 10 mg/day as an off-label use in the hope that the drug would slow visual field progression.

Comments on the Clinical Presentation

RNW: Is this glaucoma?

DG: This patient has a classic arcuate nerve fiber bundle visual field defect with an absence of neuroretinal rim pallor and excavation consistent with the visual field defect. Atypically, the optic neuropathy is unilateral with relatively rapid progression at a very low IOP; we assume it is low, although he had LASIK in the past, which might artifactually lower Goldmann applanation tonometry readings. Overall, however, I think this clinical picture is consistent with glaucoma.

RNW: Do you think his acoustic neuroma has anything to do with it?

DG: I am unaware of a syndrome associated with thyroid carcinoma, acoustic neuroma, and unilateral optic neuropathy. It is unlikely to be radiation-induced optic neuropathy, given the absence of optic disc pallor or other accompanying retinal changes from radiation.

RNW: The patient has asymmetric IOP between his two eyes. Do you think that the difference in IOP can

be accounted for by the difference in central corneal thickness between the two eyes and that it is not necessarily the result of the Trabectome procedure?

JL: Certainly, the thinner cornea in the patient's right eye suggests that the actual IOP is higher than what is being measured. He is relatively young and progressing at a relatively rapid pace. Despite the high likelihood that this patient has glaucoma, the asymmetric structural and functional damage is sufficiently atypical to warrant further investigation.

DG: The pathology appears to be localized to the structures of the prechiasmal visual pathway, since the left nerve and left visual field are normal. If a neuro-imaging study is performed, I would suggest an MRI examination of the orbit and brain, including the chiasm.

RNW: Is the patient so atypical that you should perform imaging?

DG: The clinical picture in this case is entirely consistent with glaucoma, and the literature suggests that neuro-imaging in such patients will not uncover a compressive visual pathway lesion.¹ This patient has a classic nerve fiber bundle visual field defect and does not have features suggestive of orbital compression (eg, proptosis, motility disturbance, retinal venous congestion, optic disc edema or pallor, or a decrease in central vision).

JL: As previously pointed out, the unilateral, extremely asymmetric presentation in this young person is atypical. I would retake a careful history with a particular focus on the use of steroids, remote ocular trauma, prior uveitis, yoga postures, sleep position, and so on. This step is important before committing the patient to a filtering procedure.

Comments on Management

RNW: What are the therapeutic options for this patient?

JL: The options include continued surveillance or surgical intervention. There is clear evidence of progression over time in spite of therapy, and probably the only way to eliminate the IOP component is to perform filtering surgery. I would discuss the risks, benefits, and alternatives to surgery with the patient to learn if he wants to be exposed to the risk of trabeculectomy in his right eye, when the IOP is already very low and a fur-

ther decrease may not benefit him, because the disease could be independent of pressure.

RNW: Are there any other surgical approaches that you would consider?

JL: No.

RNW: What would be your target pressure?

JL: Exactly what it is now, single digits.

RNW: You mean less than 5 mm Hg.

JL: I would aim for a target in the single digits.

DG: Look at the flatness of this diurnal curve. At least during the diurnal period, there is not much IOP fluctuation that one could blunt by performing a filtering operation. We can assume that the IOP may be higher at night, but I would hesitate to proceed with trabeculectomy in a myope with progression at single-digit IOP levels. The risk/benefit ratio is unfavorable given the high risk of hypotony maculopathy, induced astigmatism from a large bleb, and bleb dysesthesia.

RNW: You think the potential benefits of a trabeculectomy would not outweigh the risks. What would you do?

DG: I would offer the patient two therapeutic options. One choice, as you indicated, is memantine as an off-label use in the hope that the drug will slow or prevent further visual field progression.²⁻⁵ The second option is a tube shunt procedure with or without cataract removal, which is associated with a reduced risk of hypotony compared with trabeculectomy.⁶ I might consider a smaller-sized shunt such as a 250-mm² Baerveldt (Abbott Medical Optics Inc., Santa Ana, CA) or an Ahmed Glaucoma Valve (New World Medical, Inc., Rancho Cucamonga, CA). I think a drainage device would be safer than a trabeculectomy.

RNW: Are you able to achieve IOPs in the 4- to 8-mm Hg range with a tube shunt procedure?

DG: I agree that, in a typical patient with an IOP in the mid-20s or higher, it is unlikely that one could achieve an IOP of 4 to 8 mm Hg, even with adjunctive medications, following a tube shunt procedure. In this particular patient, however, even a 20% reduction in IOP might suffice.

Note that there is also a third option, which, as mentioned earlier, is to do nothing. The visual field loss in this individual is completely localized to one hemifield. Some patients are predisposed to develop damage that is entirely localized to one visual hemifield, and they will never develop visual deficits in the opposing hemifield with continued observation.

RNW: Would you make any modifications to the trabeculectomy?

JL: I would use mitomycin C and close the scleral flap with tight sutures. I would check the patient's IOP 2 and 6 hours after surgery to avoid a potential elevation that could result in a loss of fixation (snuff), and I would monitor him closely. I would delay suture lysis for as long as possible.

RNW: Would you try memantine or any other unproven therapy in this patient?

JL: Memantine is not approved by the FDA as a treatment for glaucoma. I would consider its off-label use, however, for this desperate patient who is progressing with IOPs in a very low range. I would also try oral acetazolamide for at least a week before filtering surgery to see if it lowered the IOP and if he could tolerate it.

RNW: Have you seen patients who have had progressive glaucoma after undergoing LASIK?

JL: My colleagues and I studied patients before and after LASIK to see if there were any change in the topography of the optic nerve or the thickness of the retinal nerve fiber layer. There was not.^{7,8} The other question is, does LASIK accelerate the glaucomatous damage to an eye with preexisting glaucoma? My experience is that most of these patients probably had significant undiagnosed glaucoma before the LASIK procedure. I suspect that was the case here; the process was ongoing beforehand. It is an interesting theoretical question.

CONCLUSION

Although the clinical presentation in this case is atypical, the appearance of the optic nerve and visual field defects are consistent with glaucoma. Imaging to exclude causes of optic neuropathy other than glaucoma is reasonable, given the unilateral presentation of rapidly progressive visual field damage. Although reducing the IOP might slow the patient's visual field progression, safely decreasing the pressure in an eye with a single-digit IOP is extremely challenging. □

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