

Clinical & Refractive Optometry is pleased to present this continuing education (CE) article by Dr. Samuel J. Multack and Dr. Richard F. Multack entitled **A Case of Cognitive Error Bias in Ophthalmology**. In order to obtain a 1-hour Council of Optometric Practitioner Education (COPE) approved CE credit, please refer to page 183 for complete instructions.

A Case of Cognitive Error Bias in Ophthalmology

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ABSTRACT

A 50-year-old African American female presented for evaluation of a sty on her OD lower eyelid and was subsequently found to have OHTN and a bitemporal hemianopsia. The patient had a negative work-up for a pituitary tumor and was treated for her OHTN in light of a clear nasal visual field defect on the follow-up study. The patient was evaluated by neuro-ophthalmologists and was subsequently sent to a retina specialist who diagnosed retinitis pigmentosa sans pigmentosa with incomplete penitance initially involving one to two quadrants in each eye.

CASE REPORT

A 50-year-old African American female presented to the ophthalmology clinic for evaluation of a sty. The patient was subsequently found to have ocular hypertension 26/24 (-2 pachymetry); optic nerve and GDx were normal. Visual field testing showed a bitemporal hemianopsia. Work-up for a pituitary lesion affecting the optic chiasm showed a partially empty sella and minimal sinus disease. Over a period of time, the visual field showed arcuate progression toward the nasal field OU, implying a glaucoma component. The patient was treated for her ocular hypertension with a prostaglandin analog but continued to progress. She was sent to a neuro-ophthalmologist after pituitary disease was ruled out as a cause of the bitemporal field changes and because the nasal arcuate field loss with progression was inconsistent with the optic nerve appearance and GDx findings.

HISTORY

The patient presented to the clinic with a complaint of swelling of her OD lower eyelid for a 2-week period. The lesion first appeared around December 2005 but her symptoms had worsened. She complained of visual difficulty with near tasks and she stated she had to “strain in one particular location to see things”. She had floaters in both eyes for the past year that were unchanged.

Previous History

The patient was a contact lens wearer and had a color deficiency in both eyes which was diagnosed 10 years ago, as well as posterior vitreous detachments in both eyes. She had a history of hypertension and dyslipidemia. She was taking Olmesartan (Benicar[®], Daiichi Sankyo) and Simvastatin (Zocor[®], Merck Frosst). Her family history included glaucoma in both of her parents, hypertension, and diabetes mellitus.

VISUAL EXAMINATION

Visual acuity in her right eye was 6/9 (20/30) and 6/12+2 (20/40) in her left eye with no improvement with pinhole. She was able to identify 1/11 Ishihara plates in both eyes. Confrontational fields were contracted bitemporally. Extraocular muscles were full. Pupils were normal with no afferent pupillary defect. Applanation tonometry revealed 26/24 at 16:45 pm.

Slit-lamp examination revealed trace injection of her conjunctiva in both eyes and mild nuclear sclerotic cataracts in both eyes. Vitreous floaters were present in both eyes. Dilated fundus exam revealed essentially normal appearing optic nerves with normal contour, slight paleness of the temporal side of both nerves, and normal capillary content. Each nerve measured .4/.4. The artery to vein ratio was 2:3, veins were normal, and the arteries were mildly attenuated in the lower arcuate branches in both eyes (Fig. 1A, B).

INVESTIGATION

GDx was performed and showed normal TSNIT scores, and NFI of 12/11, with mild thinning on the nasal side of both eyes (Fig. 2). Humphrey visual field testing 24-2

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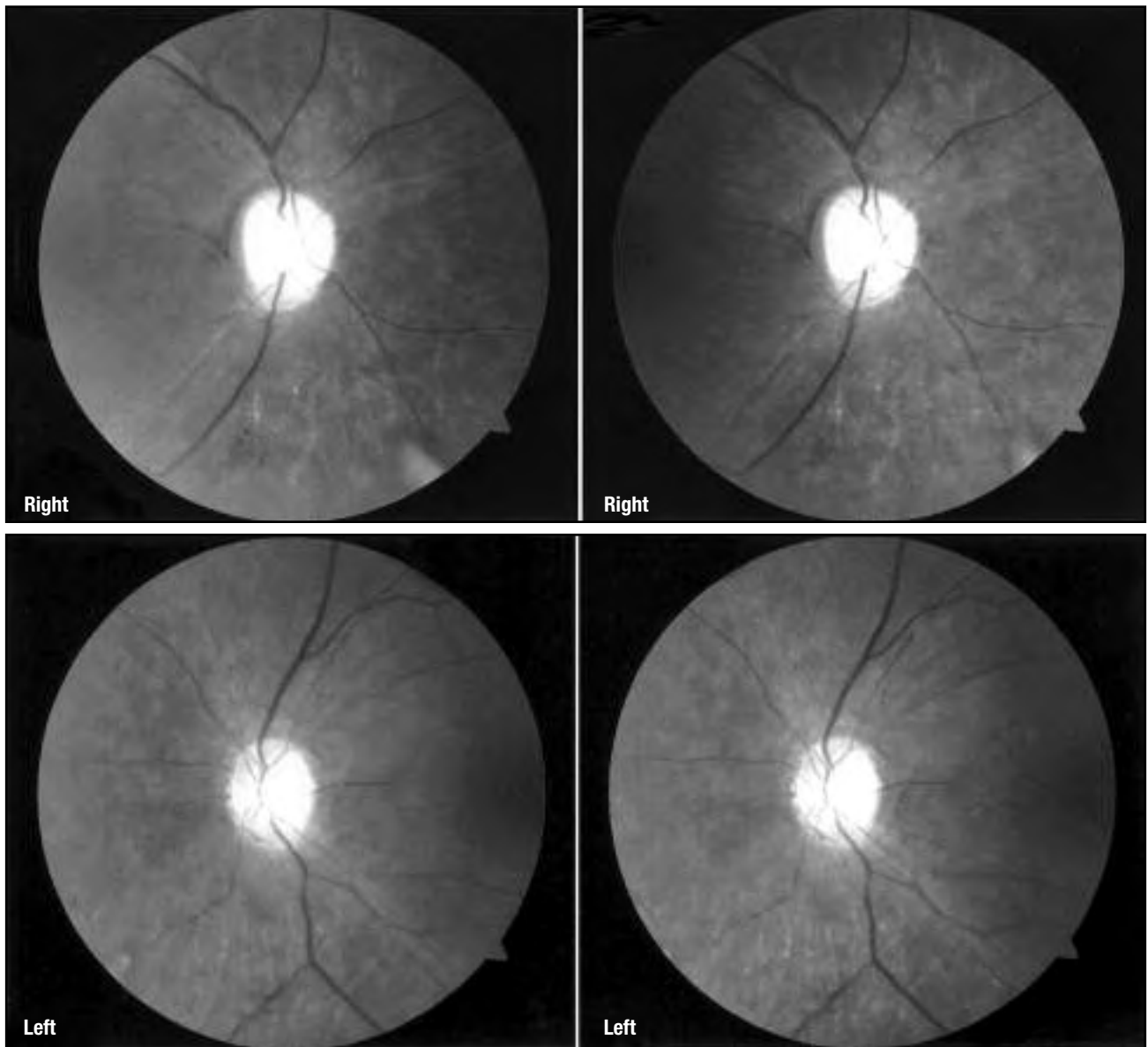


Fig. 1 Photographs of the left and right optic nerve demonstrating slight paleness of the temporal side of both nerves.

revealed a bitemporal hemianopsia that respected the mid line (Fig. 3A, B). CT scan performed on the GE light-speed ultra scanner using 1.25 mm sections revealed a partial empty sella and minimal ethmoid sinus disease.

The patient was sent to neurology for evaluation of the field loss to rule out an atypical stroke or demyelinating disease. MRI was subsequently ordered using a 0.3 tesla open MRI scanner with contrast, revealing a negative pre- and post-infusion MRI of the brain with a partial empty sella due to flattened pituitary gland along the floor of the sella turcica without intrinsic abnormality.¹

TREATMENT

The patient was treated for her ocular hypertension on presentation with a prostaglandin analog.¹ With modest improvement in her IOPs on subsequent visits, she was placed on an alpha agonist and beta blocker twice a day. Visual field testing was repeated and showed progression in the superior arcuate area of both eyes; the patient was sent to a neuro-ophthalmologist (Fig. 4A, B). Neuro-ophthalmology evaluated the patient and requested she see a retina specialist to rule out any retinal cause for her field loss. The patient was subsequently diagnosed with retinitis pigmentosa sans pigmentosa.

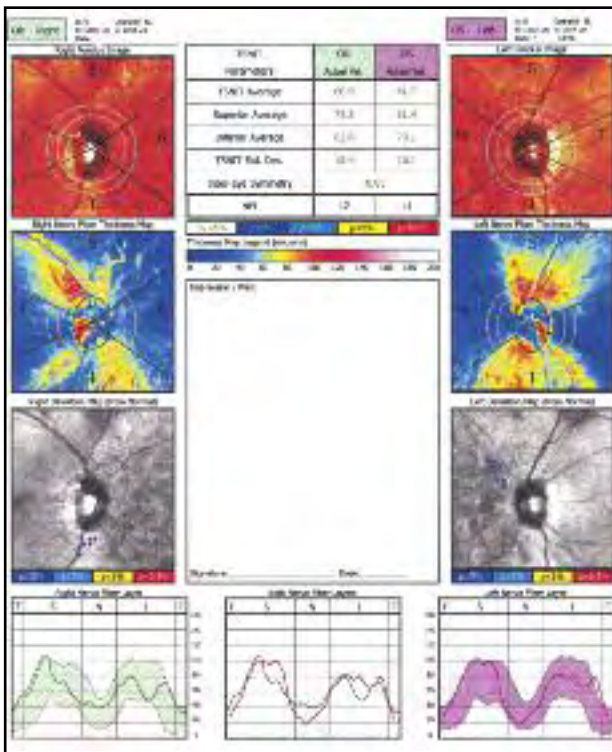


Fig. 2 GDX documenting mild thinning on the nasal side of both eyes.

Currently, the patient is enrolled in a retinitis pigmentosa clinic. She remains on her eye medicines to treat her ocular hypertension with IOPs on her last visit 14/15 at 08:15 am. Best corrected visual acuity is 6/9 (20/30) OU.

DISCUSSION

The purpose of this Case Report is to highlight the cognitive bias error that was present when the patient first presented. The diagnosis of glaucoma was focused on, with the patient presenting with IOPs in the high twenties, first degree relatives with glaucoma, and decreased confrontational fields OU. The diagnosis of glaucoma was inconsistent with the appearance of the optic nerves² and normal GDX. Once a pituitary tumor was ruled out, the visual field defects were easily confused with glaucomatous damage.³

Reducing diagnostic errors is a goal in all branches of medicine. There are three major categories of diagnostic errors in medicine that have been described. No fault errors, system errors and, lastly, cognitive errors.⁴ No fault errors occur when the disease is silent, presents atypically, or mimics something more common. System errors play a role when diagnosis is delayed or missed because of latent

imperfections in the health care system. These errors can be reduced by system improvements, but can never be eliminated because these improvements lag behind and degrade over time, and each new fix creates the opportunity for novel errors.

Cognitive errors reflect misdiagnosis from faulty data collection or interpretation, flawed reasoning, or incomplete knowledge. The limitations of human processing and the inherent biases in using heuristics guarantee that these errors will persist. Opportunities exist, however, for improving the cognitive aspect of diagnosis by adopting system-level changes (e.g., second opinions, decision-support systems, enhanced access to specialists) and by training designed to improve cognition and cognitive awareness.⁵ There are more than thirty-five described cognitive dispositions to respond (dispositions to respond to particular situations in predictable ways) that may lead to diagnostic errors as described by Croskerry.⁶ In this particular case, at least four “CDRs” were present that initially led us to the incorrect diagnosis.

Anchoring: This is the tendency to perceptually look into salient features in the patient’s initial presentation too early in the diagnostic process, and failure to adjust this initial impression in the light of later information. This cognitive disposition may be severely compounded by confirmation bias.⁶

Ascertainment bias: This occurs when a physician’s thinking is shaped by prior expectations.⁶ The bitemporal hemianopsia is driven into our minds on board exams as an indication of a pituitary tumor. This expectation took us on the initial path of CNS imaging.

Availability: The disposition to judge things as being more likely, or frequently occurring, if they readily come to mind.⁶ In our institution, glaucoma is more prominent than retinitis pigmentosa and thus a more available diagnosis.

Confirmation bias: The tendency to look for confirming evidence to support a diagnosis, rather than look for disconfirming evidence to refute it, despite the latter often being more persuasive and definitive.⁶ In our case, the evaluation of the optic nerve, GDX and VFT did not correlate with the diagnosis of glaucoma. With a normal CT scan, MRI, normal neurology and endocrinology examination, glaucoma was a likely diagnosis. Initially, the diagnosis was not refuted. Eventually, we decided to look for other opinions and testing to refute our biases and reached the correct diagnosis, albeit late in the process.

This case reiterates the importance of cognitive errors in the diagnosis of disease and the essential nature

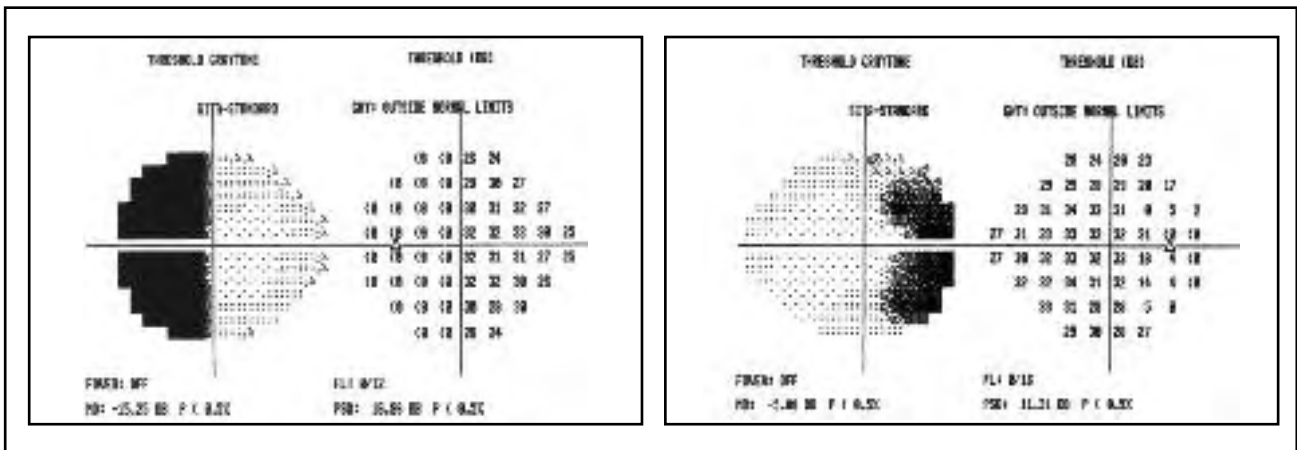


Fig. 3 Humphrey 24-2 visual fields showing a bitemporal hemianopsia.

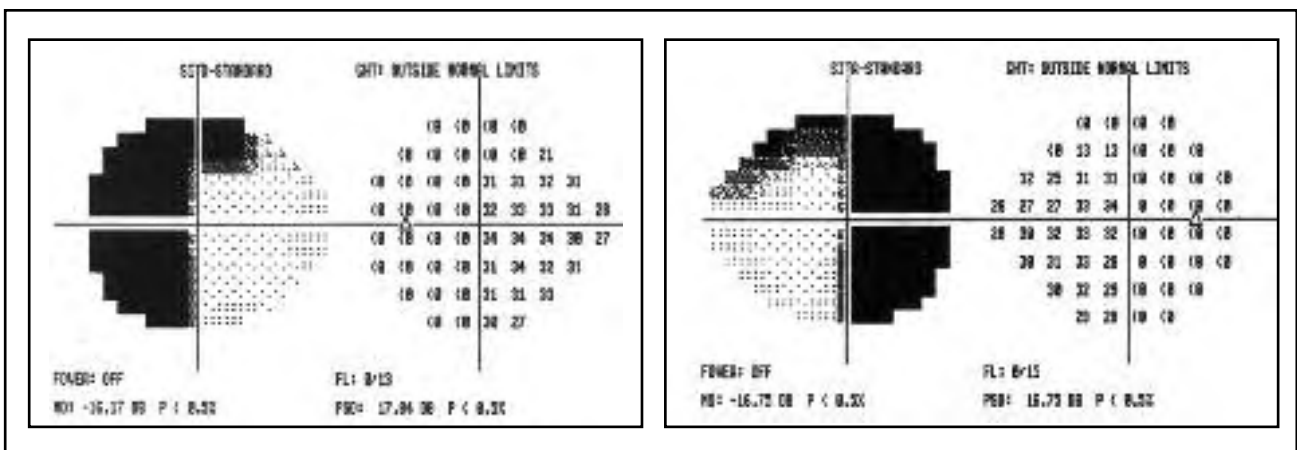


Fig. 4 Repeat of Humphrey visual fields showing progression in the superior arcuate area of both eyes.

of cognitive decision-making as a key part of medical education. It underscores the need for further exploration to reduce cognitive diagnostic errors as related to all fields of medicine. □

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