



WHEN TROCHLEAR NERVE PALSY GOES UNDETECTED



A patient case highlights the importance of comprehensive testing when narrowing in on an accurate diagnosis and management plan.

BY CHRISTOPHER J. BORGMAN, OD, FAAO

A 62-year-old female presented for continuation of her glaucoma care. She had been managed by an outside doctor, but wanted to switch to our clinic due to its proximity to her home. She reported good compliance with her glaucoma drops, denied any side effects with the drops, and reported stable vision OU. Her medical history was positive for hypertension and depression. Family ocular and medical histories

were unremarkable. Her medications included travoprost (Travatan Z, Novartis), amlodipine, mirtazapine, and sertraline. She denied any allergies to medications. Her social history was also unremarkable.

CASE FINDINGS

The patient's BCVA was 20/20 OU with moderate hyperopic spectacle correction. Her pupils were equal, round, reactive to light, and without relative afferent pupil defect.

Confrontation visual fields were full to counting fingers OU. Extraocular motilities appeared full OU, without obvious restriction. Cover testing showed a small hyperdeviation OS of low amplitude. Single Maddox rod testing with Modified Thorington card confirmed a small 1 prism diopter left hyperphoria. Parks-Bielschowsky three-step testing with single Maddox rod testing showed worsening of the left hyperdeviation in right gaze

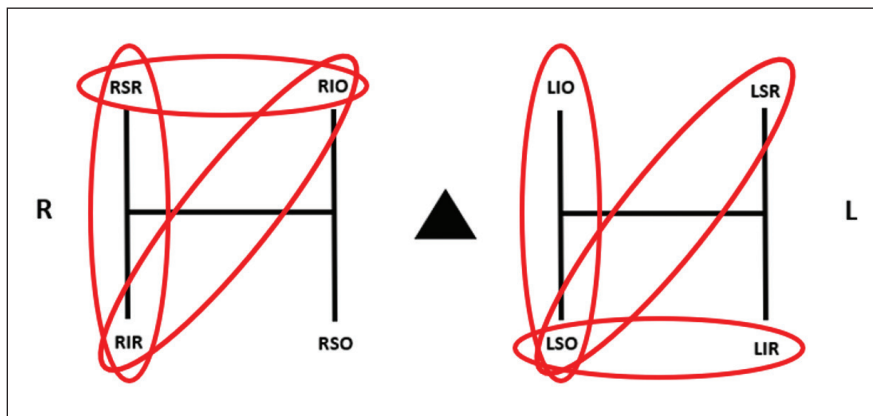


Figure 1. Results of the patient's Parks-Bielschowsky three-step test show left hyperdeviation in primary gaze, worse in right gaze, and left head tilt. Note the left superior oblique muscle is circled three times, suggesting weakness of the muscle and resultant CN IV paresis.

and left head tilt, suggesting left superior oblique weakness (Figure 1). Double Maddox rod testing showed subjective increased excyclotorsion of 3° OS and no torsion OD. Inspection of that same day's retinal nerve fiber layer analysis on OCT (Spectralis, Heidelberg Engineering) also revealed increased objective excyclotorsion OS and normal torsion OD (Figure 2). The patient's IOP was 13 mm Hg OD and 15 mm Hg OS with Goldman

applanation tonometry. Aside from her glaucomatous optic nerves, the remainder of her ocular examination was normal OU.

Based on these clinical findings, the patient was diagnosed with a left trochlear (CN IV) paresis of unknown duration. She was entirely asymptomatic, denying symptoms of diplopia, eyestrain, stroke, etc. She also denied neuroimaging of her head/brain. She was told to continue the travoprost at bedtime OU and

to return to the clinic in 3 months for glaucoma follow-up. I requested records from her previous eye doctor to review tests and notes and evaluate whether the CN IV weakness was acute or chronic in nature.

Upon receiving the records from the patient's previous doctor a few days later, I compared several OCT scans (incidentally, that doctor also used Spectralis OCT for her glaucoma management) dating back at least 3 years, and confirmed the increased excyclotorsion OS (Figure 3). Her left hyperdeviation had indeed been chronic for at least 3 years, maybe longer. Of note, there was no mention in the previous doctor's notes of CN IV paresis OS or left hyperdeviation, and I could not find any documentation of a cover test performed by that outside doctor. Given the excyclotorsion was obviously there 3 years prior and there was no evidence of prior cover testing, I speculated that the patient's CN IV paresis was likely present, but missed by the previous doctor.

DISCUSSION

The trochlear nerve innervates the superior oblique muscle in the orbit, and trochlear nerve palsy is the most common cause of acquired vertical diplopia.^{1,2} Each trochlear nerve's respective nucleus is located in the inferior dorsal midbrain at the level of the inferior colliculi on the contralateral side.^{3,4} The trochlear nerve is long and thin, making it particularly susceptible to trauma.² Under normal conditions in primary gaze, the superior oblique has a primary action of incyclotorsion, secondary action of depression, and tertiary action of abduction.⁵ So, in cases of superior oblique weakness (eg, CN IV palsies), the involved eye is abnormally excyclorotated and elevated.⁵ CN IV palsies are most commonly caused by trauma and microvascular or congenital etiologies.^{2,5} Full recovery in 3 months

AT A GLANCE

- ▶ The included case is of a woman who presented for continuation of glaucoma care, but ended up with a diagnosis of longstanding chronic CN IV palsy/paresis OS that had been missed by her previous provider.
- ▶ Trochlear nerve palsy is the most common cause of acquired vertical diplopia.
- ▶ The patient was completely asymptomatic and declined intervention and workup unless something worsened in the future. For now, her CN IV paresis is being monitored and her glaucoma managed at regular follow-up intervals.
- ▶ Objectively assessing and evaluating ocular torsion, especially in cases of vertical diplopia or vertical deviation, holds significant value for providers and patients alike.

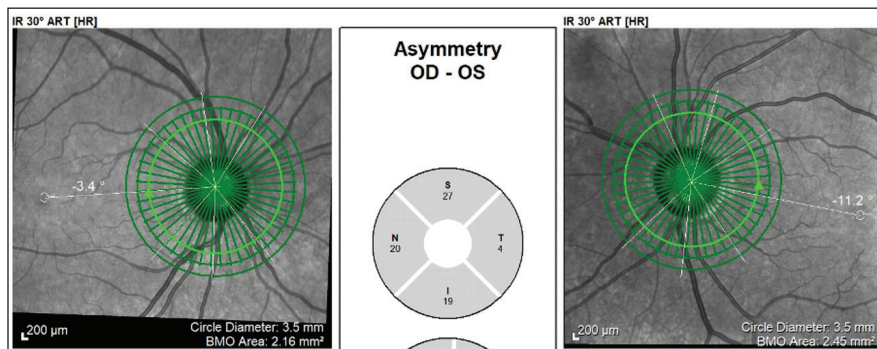


Figure 2. Retinal nerve fiber layer OCT scan of the patient at her first visit shows a normal amount of ocular torsion OD of 3.4° and an increased amount of exocyclotorsion OS of 11.2°. ⁶⁻⁸

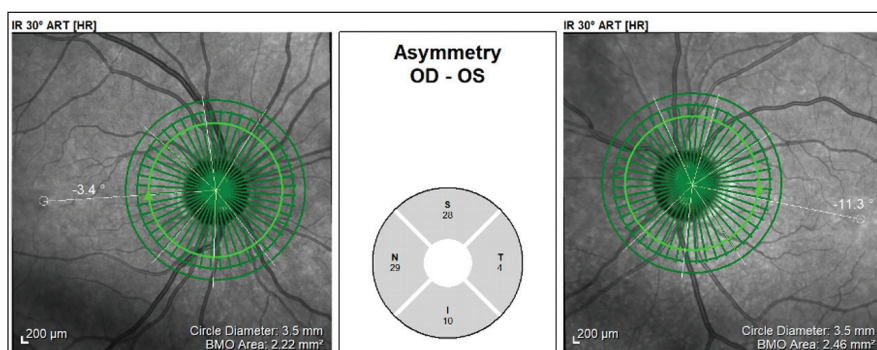


Figure 3. Retinal nerve fiber layer OCT scan of the patient from 3 years earlier shows similar degrees of ocular torsion during this 3-year span in each eye (3.4° OD and 11.3° OS), suggesting a longstanding chronic CN IV palsy/paresis OS.

has been reported in about 79% of patients with acute CN IV palsies.²

In unilateral CN IV palsies, the hyperdeviation typically worsens with contralateral gaze and ipsilateral head tilt, which is the typical pattern noted on the Parks-Bielschowsky three-step test.¹ The vertical hyperdeviation can be measured subjectively (ie, with single Maddox rod testing) and/or objectively (ie, with a cover test with prism neutralization).^{1,2} The exocyclotorsion can be measured subjectively (ie, with double Maddox rod testing) and/or objectively (ie, with OCT or fundus photography).⁶⁻⁸ There is a large range of normal exocyclotorsion values in normal patients tested with the Spectralis (-3.8° to -9.4°, average -6.6°).⁶

A second study also looking at

normal ocular torsion values with Spectralis OCT showed a broader range of normal torsion, but similar average torsion (+2.5° to -17.5°, average -6.7°).⁷

A third, more recent study compared torsion in healthy controls versus CN IV palsies with Spectralis OCT, and found that torsion in CN IV palsies ranged from -7.7° to -14.9°, with an average of -11.3°. ⁸ This same study also found that normal ranges of ocular torsion were -4.3 to -8.5°, with an average of -6.4°. ⁸

Treatment of CN IV palsies typically includes one or more of the following options: monitor, occlusion, prism correction, vision therapy, and/or surgical correction.⁵

CASE CONCLUSIONS

I discussed the patient's hyperdeviation and management

options at length with her. However, once she learned the deviation was longstanding for at least 3 years and was otherwise completely asymptomatic, she declined any further intervention and workup, including CT/MRI, unless something worsened in the future. For now, we are simply monitoring her CN IV paresis and managing her glaucoma at regular follow-up intervals.

This case highlights the importance of performing cover testing frequently and documenting it appropriately in our records, as well as having keen observation skills when interpreting patients' glaucoma testing, as there can be clues of other ocular and systemic pathologies on these tests that may be unrelated to the reason the tests were run initially. Sometimes clues, such as abnormal torsion, are easily seen on objective testing, including OCT and fundus photography, when we're working with a low threshold. Objectively assessing and evaluating ocular torsion, especially in cases of vertical diplopia or vertical deviation, holds significant value for providers and patients alike. ■

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