



When Tensions Run High: Managing Ocular Hypertension in a Child with Optic Nerve Hypoplasia

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INTRODUCTION

Limited research exists regarding diagnosing and treating ocular hypertension in a pediatric patient. Diagnosis is largely based on clinical findings as there is currently no normative OCT database for patients under the age of 18. Treatment typically includes topical medications, which may be lifelong and carry financial and emotional burdens. This poster highlights the management strategy of an 11-year old with monocular status secondary to optic nerve hypoplasia.

CASE HISTORY

An 11-year old female was referred from an outside provider for evaluation of ocular hypertension. She had been using Latanoprost 0.005% QHS OU for one year as prescribed by her referring doctor. Her Tmax was 25mmHg OD and 24mmHg OS with normal corneal thicknesses of 547 μ m OD and 543 μ m OS. She has no family history of glaucoma. She has light perception vision OS since birth secondary to optic nerve hypoplasia and has worn glasses for low myopia OD since she was 5 years old. She was born full term without complications and all other history was unremarkable.

CLINICAL FINDINGS

Externals

	OD	OS
VA cc	20/25-	LP @ 2ft
CVF	FTFC	FT transilluminator @ 2ft
EOMs	FROM	FROM
Pupils	PERRL	PERRL (+)APD
Cover Test		22PD CLET

Ocular Health

	OD	OS
IOP	16mmHg c Latanoprost 0.005% (measured with Goldmann)	16mmHg c Latanoprost 0.005% (measured with Goldmann)
Gonio	Open to CB 360	Open to CB 360
ONH	Pink, healthy, distinct 360 C/D 0.3	Hypoplastic, double ring sign C/D <0.1

Additional Testing

	OD	OS
OCT GCC and Macula	Normal	RNFL thinning Lack of foveal dip Decreased ganglion cell number
Visual Field	Non-glaucomatous defects	Non-glaucomatous defects glaucomatous defects
MRI w/ and w/o contrast	No brain abnormality, optic pathway glioma, or septo-optic dysplasia	No brain abnormality, optic pathway glioma, or septo-optic dysplasia
VEP	Normal	Severely decreased p-wave amplitudes consistent with LP vision

FIGURE 1
OD normal, OS hypoplastic optic nerve head with double ring sign, C/D <0.1

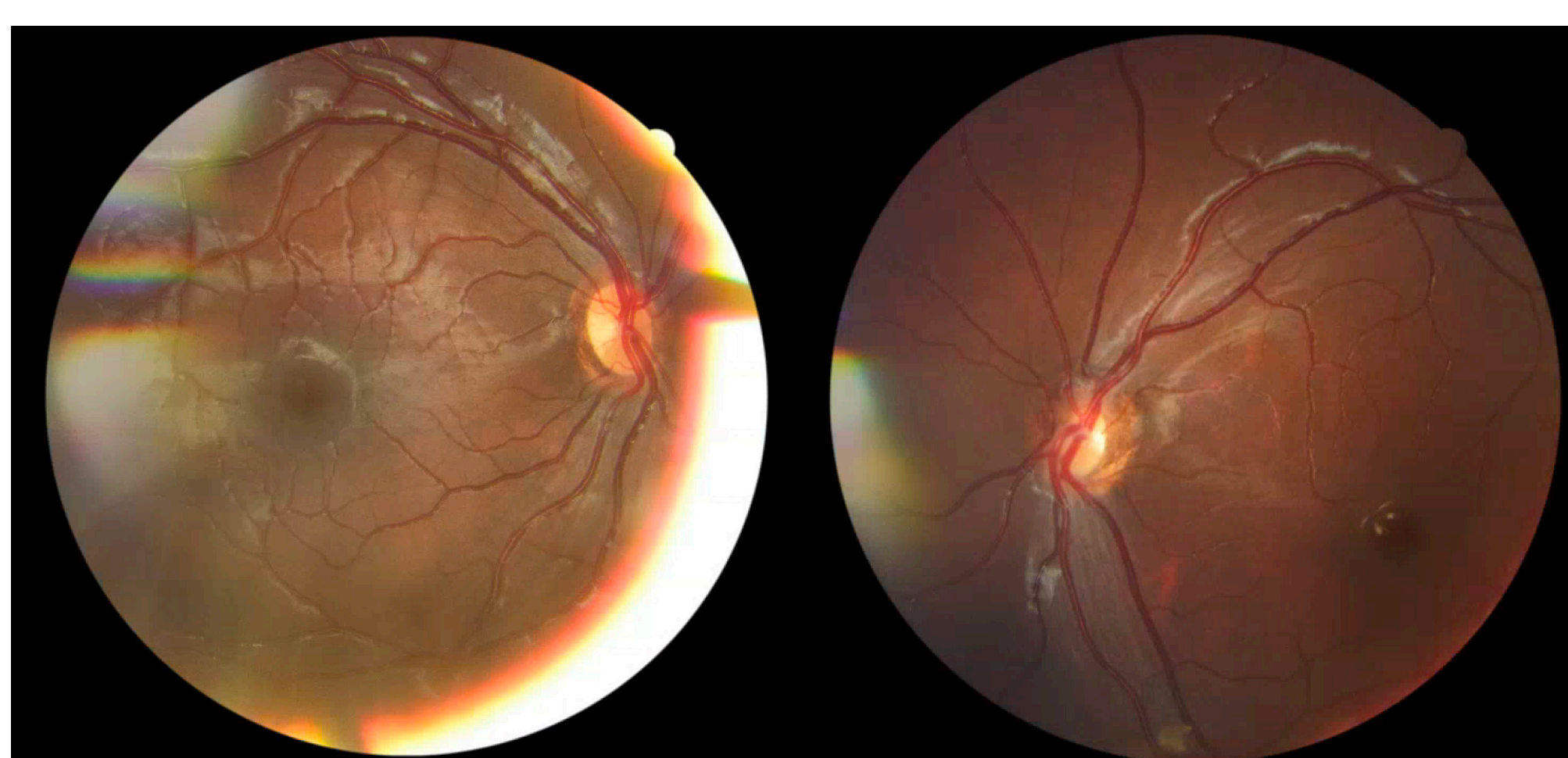
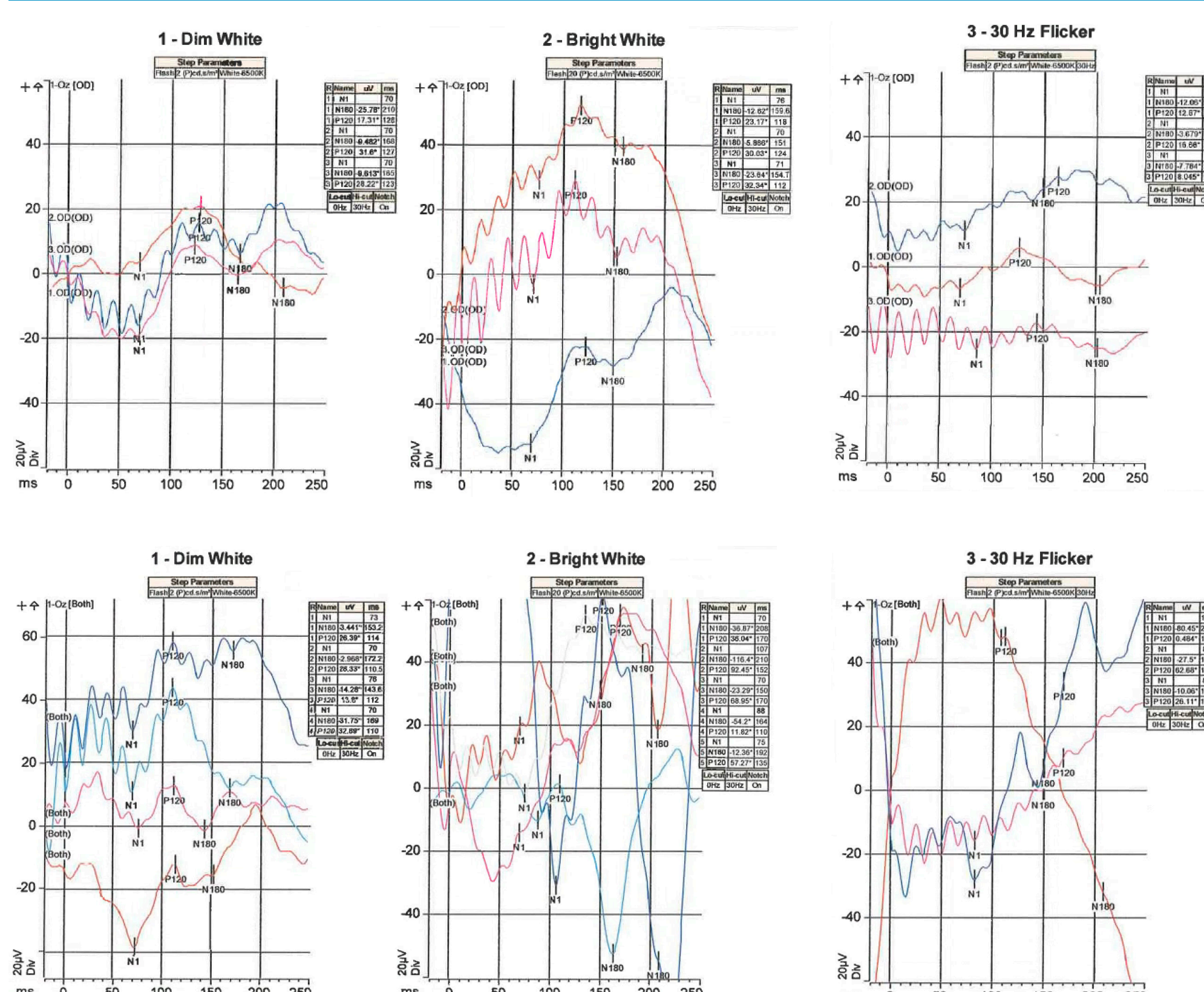


FIGURE 2
VEP showing severely decreased p-wave amplitudes consistent with LP vision OS. Normal OD.



DIAGNOSIS

- Given the patient's history of consistently high IOPs, RNFL thinning and decreased ganglion cell count, they were diagnosed with pediatric ocular hypertension and started on Latanoprost 0.005% QHS OU.
- Given the clinical findings of a small optic nerve with a double ring sign, (+)APD, sensory strabismus and light perception VA, the patient was diagnosed with optic nerve hypoplasia OS. Their MRI and endocrine workup ruled out septo-optic dysplasia.

DISCUSSION

Pediatric Ocular Hypertension

Risk Factors	Family history of glaucoma, large C/D ratios, consistently high IOPs, thin central corneal thicknesses, greater pattern standard deviation, myopia
Clinical Signs	RNFL thinning, visual field defects, optic nerve head cupping, optic nerve atrophy
Diagnostic Tests	Goldmann tonometry, central corneal thickness measurements, visual fields, OCT

- In determining when to treat ocular hypertension in a pediatric patient, it is important to consider the risk factors and probability of visual impairment. The decision to treat is individualized and one must consider the cost, side effects, and quality of life as treatment will likely be lifelong.
- Treatment targets IOP reduction, and so topical medication is the first line of treatment. Target pressures are lower for children compared to adults due to the longer life expectancy and severe nature of pediatric glaucoma. For extremely high IOPs, surgical intervention may be necessary.

Optic Nerve Hypoplasia

Risk Factors	Young maternal age, premature birth, maternal diabetes, maternal use of alcohol or drugs, sporadic
Clinical Signs	Small optic disc, double ring sign, vascular tortuosity, thinning of RNFL and ganglion cell layer, (+)APD, sensory nystagmus and strabismus. Vision can range from normal to light perception.
Diagnostic Tests	MRI and endocrine workup

- An important differential diagnosis to consider is septo-optic dysplasia, which presents with CNS abnormalities (thinning of the optic chiasm, absent septum pellucidum, agenesis of corpus callosum) and endocrine abnormalities (growth hormone deficiency, hypothyroidism, hypercortisolism) and typically runs in families. An MRI and endocrine workup is indicated to rule out septo-optic dysplasia.

TREATMENT AND MANAGEMENT

- The patient had a good reduction of IOP with Latanoprost 0.005% QHS OU. Given their history of an IOP spike when they self-discontinued the drops, the patient and their mother were motivated to continue using the drop. The patient will continue to be managed every 6 months for IOP checks.
- The pros of treatment outweigh its cons in this case given the patient's monocular status. In our more conservative approach, we reduce the risk of any possible glaucomatous damage early on, thus preserving as much vision for the patient as possible.
- The cons of treatment include emotional and financial burdens of having to pay for a medicated eye drop with possible lifelong use.
- Due to their monocular status, the patient was also prescribed full time wear of specs for protection and referred to low vision services.

CONCLUSION

- The decision to treat ocular hypertension in a pediatric patient is individualized. Factors to consider are consistently elevated IOPs, family history of glaucoma, optic nerve head cupping, RNFL thinning, and high probability of visual impairment. In this case, the patient's monocular status rendered more conservative management.
- Topical medications are typically the first line of treatment. Extremely high IOPs may require surgical intervention.
- When diagnosing optic nerve hypoplasia, it is important to rule out septo-optic dysplasia with an MRI and endocrine workup.

REFERENCES

Available upon request

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