Finasteride Induced Clinical Ocular Toxicity
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PURPOSE
Finasteride, a 5-alpha reductase inhibitor (5-ARI), received FDA approval for treatment of prostate disease in 1992 and androgenic alopecia in 1997. The mechanism of action involves inhibition of the steroid Type II 5-reductase, which facilitates the conversion of testosterone to 5-dihydrotestosterone (DHT) within epithelial membranes and the central nervous system. The active nonsteroidal products of the 5-reductase pathway function in many physiological systems and such inhibition results in many adverse effects (Fig. 1).1,2

Clinical studies have reported irreversible side effects associated with finasteride use, which include, permanent loss of libido, erectile dysfunction, depression, and altered mental status. Finasteride use has also been associated with ocular side effects such as blurred vision, conjunctival injection, eyelid edema, and Meibomian gland dysfunction.3

We seek to further investigate the potential for occult ocular toxicity associated with finasteride and 5-ARI use. We performed a retrospective review of all patients in our clinic, who reported use of finasteride, to assess whether there was evidence of damage to the visual pathway.

METHODS AND MATERIALS
Investigation of 5-ARI induced ocular toxicity was prompted by the presentation of idiopathic atypical cystoid macular edema (CME) in two patients who had been prescribed finasteride. Both patients failed to respond to all CME treatments and showed additional signs of retinal and optic nerve dysfunction. Using our electronic health records (AthenaHealth), we identified 28 patients with history of finasteride use. A retrospective chart review was performed to identify anomalies on macular optical coherence tomography (OCT), visual electrophysiology tests (visual evoked potential [VEP] and electroretinography [ERG]), and microperimetry (MP) to determine whether finasteride use may be a clinically significant factor.

RESULTS
Out of 58 eyes of 29 patients known to have taken finasteride, there was evidence of visual dysfunction in 56 eyes of 28 patients. Of those 56 eyes, 35 eyes of 20 patients had persistently abnormal OCT macula studies. In the 16 eyes of 8 patients found to have normal OCTs, 12 eyes of 6 patients demonstrated optic nerve and retinal damage per visual evoked potential (VEP) and/or electroretinogram (ERG). 36 eyes of 21 patients were also found to have deficits in their MP. A total of 38 eyes of 16 patients had macular edema with 11 eyes of 6 patients having CME.

CONCLUSION
Our findings confirm retinal and optic nerve damage in addition to OCT changes in patients who had been on finasteride. The data indicates finasteride use may be strongly associated with abnormal retinal function along with an atypical refractory cystic edema. We recognize our small sample study is prone to selection bias since clinical patients are likely to have visual dysfunction. Nevertheless, there is significant justification for baseline testing in individuals prescribed 5-ARI drugs to proactively reduce risk of visual loss. Further studies will be required to understand the mechanism resulting in possible finasteride induced neurotoxicity to correlate known neuropsychiatric symptoms to our ocular findings. Prescribing physicians should be aware of the adverse side effects of 5-ARI drugs and exercise extreme caution prior to initiating therapy.

REFERENCES