Lower lid entropion secondary to treatment with alpha-1a receptor antagonist: a case report

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Abstract

Introduction: The use of alpha-1a receptor antagonists (tamsulosin) is widely accepted in the treatment of benign prostatic hypertrophy (BPH). It has previously been implicated as a causative agent in intra-operative floppy iris syndrome due to its effects on the smooth muscle. We report a case of lower lid entropion that may be related to a patient commencing treatment with tamsulosin.

Case presentation: A 74-year-old Caucasian man was started on alpha 1-a receptor antagonist (Tamsulosin) treatment for benign prostatic hypertrophy. Eight days later, he presented to the ophthalmology unit with a right lower lid entropion which was successfully treated surgically with a Weiss procedure.

Conclusion: We report a case of lower lid entropion that may be secondary to the recent use of an alpha-1a blocker (tamsulosin). This can be explained by considering the effect of autonomic blockade on alpha-1 receptors in the Muller’s muscle on a patient that may already have an anatomical predisposition to entropion formation due to a further reduction in muscle tone.
However, due to its effect on alpha-1a adrenergic receptors in iridal smooth muscle, it has also been documented to cause the intra-operative floppy iris syndrome (IFIS) [2]. It is believed that tamsulosin blocks the iris dilator muscle and this constant receptor blockade results in semi-permanent loss of muscle tone leading to a flaccid and floppy iris. However, no significant relationship has been found between the duration of tamsulosin intake and severity of IFIS [3].

The Muller’s muscle is a smooth muscle that lies just deep to the orbital septum in both upper and lower eye lids. Its primary function is to assist in the retraction of both lids and is primarily innervated by alpha-2 adrenergic receptors although recent studies have also shown the presence of alpha-1 receptors [4]. This is of clinical significance in Horner’s syndrome where the interruption of sympathetic supply to the muscle can result in ptosis and an elevation of the lower lid by as much as 1 mm [5]. Although not reported to be of benefit in entropion management, apraclonidine has shown improvement in lid function in Horner’s syndrome. Apraclonidine is a weak alpha-1 agonist and a potent alpha 2 agonist. In Horner’s syndrome, there is upregulation of alpha 1 receptors leading to denervation hypersensitivity. This, in turn, causes the observed lid retraction with apraclonidine [6,7]. Building on this knowledge and given the recent use of tamsulosin in our patient, we hypothesize that there was an alpha-1 blockade on the Muller’s muscle in the right lower lid. This led to increased lower lid laxity, which may have been predisposed to the development of the entropion soon after commencing tamsulosin.

Conclusion
We report a case of lower lid entropion that may be secondary to the recent use of an alpha-1a blocker (tamsulosin). This can be explained by considering the effect of autonomic blockade on alpha-1 receptors in the Muller’s muscle on a patient that may already have an anatomical predisposition to entropion formation due to a further reduction in muscle tone.

Consent
Written and informed consent was obtained from our patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Authors’ contributions
SW and PS clinically diagnosed and managed our patient including the surgical intervention needed. Both authors were involved in writing the manuscript and approved the final version for submission.

Competing interests
The authors declare that they have no competing interests.

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