American Journal of Ophthalmology Case Reports 23 (2021) 101152

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

Case report

Multimodal etiology of drug induced angle closure with topical glaucoma therapy

Aditya Mehta [a,*], Andrew Lewis [b]

[a] William Beaumont Army Medical Center, El Paso, TX, USA
[b] Wilford Hall Ambulatory Surgical Center, San Antonio, TX, USA

ARTICLE INFO

Keywords:
Drug-induced glaucoma
Acute angle closure
Choroidal effusions
Brimonidine
Uveitis

ABSTRACT

Purpose: We present a case of acute onset of bilateral choroidal effusions leading to angle closure glaucoma attributed to multiple mechanism of actions causing ciliary body and aqueous flow disruption in the setting of topical glaucoma therapy with latanoprost, brimonidine 0.2%, and Brinzolamide 0.1%.

Observation: The patient presented with ocular hypertension in the setting of bilateral choroidal effusions, leading to angle closure without pupillary block. After cessation of the glaucoma drops and starting steroids and cycloplegics, the patient's symptoms resolved.

Conclusions and importance: This case report highlights the various physiological mechanisms of action that can induce angle closure glaucoma from commonly used topical medications for glaucoma treatment. Thus, a keen awareness is warranted of this idiosyncratic reaction in order to avoid morbidity and long term vision loss.

1. Introduction

Drug induced bilateral angle closure is a well reported phenomenon, especially with sulphamate-substituted compounds, such as topiramate, hydrochlorothiazide, and acetazolamide .[1] Topical brinzolamide, a carbonic anhydrase inhibitor, brimonidine, alpha 2 adrenergic receptor agonist, and latanaprost, a prostaglandin analog, are routinely used to for the treatment of glaucoma. This case presents a patient with bilateral angle closure, who improves rapidly after cessation of all glaucoma drop therapy. We seek to identify various mechanisms of action of each glaucoma drug taken by our patient that may have lead and compounded to the development of angle closure.

2. Case presentation

A 78 year old female presented to the ophthalmology clinic with acute vision loss and headache. Her past ocular history was significant for pseudophakia OU, dry macular degeneration and open angle glaucoma, for which she had chronically used latanaprost 0.005% QHS OU, and recently prescribed Simbrinza™ (Brimonidine 0.1%/Brinzolamide 0.1%) BID OU. Her medical history was significant for COPD. On examination, her visual acuity was 20/400 OD and 20/200 OS. Pupils were equally reactive and without a relative afferent pupillary defect. Her IOP was 24 OD and 23 OS. Slit lamp exam showed a quiet anterior chamber but shallow peripherally. Indentation gonioscopy revealed closed angles OU. UBM demonstrated irido-corneal apposition in the setting of 360° choroidal effusions OU (Fig. 1). A manifest refraction revealed a 3 diopter myopic shift. The diagnosis of bilateral secondary acute angle closure secondary to choroidal effusions from brinzolamide was made. As a result, Simbrinza™ was discontinued and brimonidine 0.15% TID OU was added to control her IOP, as well as topical homatropine BID OU.

The following day the patient's VA had worsened to CF OD and 20/400 OS although IOP improved to 9 OD and 7 OS. There was now 2+ anterior chamber cell OU. The choroidal effusions had enlarged and were now extending over the temporal macula OU (see Fig. 2). Brimonidine and latanoprost were discontinued and the patient was prescribed prednisolone 1% QID OU in addition to her cycloplegia. The patient continued to show improvement over five days as the choroidal effusions resolved and her vision returned to baseline. Examination revealed a deep and quiet anterior chamber with angles open to scleral spur 360° OU. Her latanoprost was restarted and the patient maintained an adequate IOP.

3. Discussion

Our patient presented with acute angle closure, without pupillary

* Corresponding author. William Beaumont Army Medical Center, 5005 N Piedras, St El Paso, TX, 79911, USA.
E-mail address: adimehta115@gmail.com (A. Mehta).

https://doi.org/10.1016/j.ajoc.2021.101152
Received 14 May 2020; Received in revised form 9 April 2021; Accepted 14 June 2021
Available online 17 June 2021

Abbreviations

IOP	Intraocular Pressure
OU	Oculus Uterque - Both eyes
QHS	Quaque hora somni - Every night
COPD	Chronic Obstructive Pulmonary Disease
OD	Oculus Dexter
OS	Oculus Sinister
UBM	Ultrasound biomicroscopy
Va	Visual Acuity
CF	Count Fingers
QID	Four times a day
TID	Three times a day
BID	Two times a day
VKH	Vogt-Koyanagi-Harada Disease

block, in the setting of large choroidal effusions after recently starting a combination of brinzolamide and brimonidine. The differential diagnosis for bilateral choroidal effusions include melanoma, metastasis, hypertensive choroidopathy, chronic hypotony, VKH, aqueous misdirection, posterior scleritis, or posterior uveitis. However, none of these conditions were present in our patient.

We initially hypothesized the choroidal effusions were due to brinzolamide, as it is a carbonic anhydrase inhibitor that shares the same sulfamate moiety [2], as topiramate and acetazolamide, and therefore, has the potential to cause this idiosyncratic reaction. However, the patient had worsening choroidal effusions and subsequently developed anterior uveitis when brimonidine was continued alone. Nevertheless, brinzolamide may still have a residual effect on this idiosyncratic reaction since it had only been stopped for 24 hours, given it has a longer washout period.

Drug-induced uveitis due to brimonidine has been well established but choroidal effusions have not been reported [3,4] Previous reports have reported other non-sulfamate moiety containing drugs causing secondary bilateral angle closure [1,5,6] Multiple hypothesis have been postulated as to the cause of this idiosyncratic reaction including dysregulation of aqueous flow and inflammation in the ciliary body [7], which could have been potentiated by the uveitis caused by brimonidine in our patient. Interestingly, the IOP dropped significantly prior to the resolution of choroidal effusions, thought likely due to ciliary body shutdown in the presence of uveitis.

Lastly, the concomitant use of latanoprost in our patient may also have compounded to the rapid enlargement of choroidal effusions. Latanoprost increases aqueous flow into uveo-scleral outflow pathway and could disrupt blood-aqueous barrier in pseudophakic eyes .[8] Previous case report, have shown topical latanoprost as a culprit for choroidal effusions .[9]

Ultimately, the drugs causing this reaction lead to a similar clinical course - the development of choroidal effusions with forward rotation of ciliary processes-iris-lens complex, leading to angle closure without pupillary block and acute myopic shift.

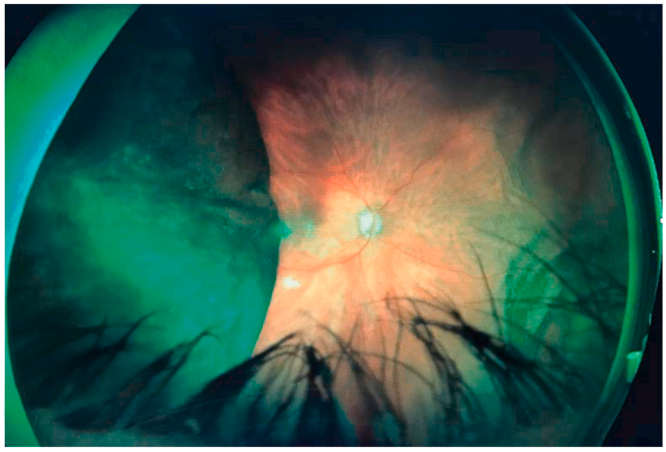

Fig. 2. Optos fundus photo OD demonstrating temporal choroidal effusion extending into the macula and peripheral nasal choroidal effusions.

The discontinuation of all glaucoma drops, with the addition of prednisolone and a cycloplegic, led to the rapid resolution of the anterior chamber cell and choroidal effusions. The use of brimonidine in our patient met 7 out of 7 criteria established by Naranjo et al. in order to determine causality of a drug to the reported adverse event .[10] However, a re-challenge with Simbrinza, brimonide or brinzolamide alone was not performed, in order to avoid further morbidity to our patient. This case report highlights multiple mechanisms by which routine glaucoma drops (latanoprost, brinzolamide, and brimonidine) may compound and lead to drug induced bilateral acute angle closure. Physicians must be highly aware of this idiosyncratic reaction and stop all inciting medications to promptly treat the patient.

Patient consent

This report does not contain any personal identifying information.

Funding

No funding or grant support

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

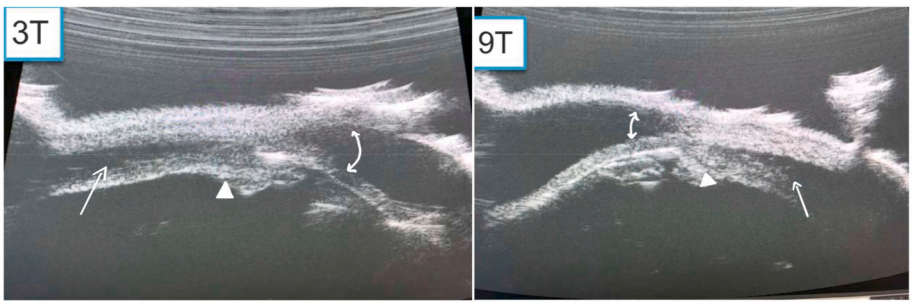

Fig. 1. UBM OD, demonstrating irido-corneal apposition in the setting of suprachoroidal fluid (white arrows) with resultant anterior rotation of ciliary processes and closed angle (arrowhead). The curved arrows indicate a shallow peripheral anterior chamber.

Declaration of competing interest

None of the authors have any financial disclosures.

Acknowledgements

None."

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