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Medication induced transient myopic shift

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CASE STUDY

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ABSTRACT

A 30-year-old female presented two days post myomectomy with blurred vision due to myopic shift. She was found to have a shallow anterior chamber with normal intra-ocular pressures and no uveal effusion. This was thought to be medication induced and after suspending celecoxib her vision improved back to baseline.

Key Words
Myopic shift, ophthalmic side effects, refraction, COX-2

Implications for Practice:

1. What is known about this subject?
COX-2 inhibitors can cause blurry vision with no clear mechanism. Medication induced myopia is a side effect of sulfonamides insulin and cold and flu medication.

2. What new information is offered in this case study?
COX-2 inhibitors may be similar to sulfonamides which cause myopic shift of the lens iris diaphragm, swelling of the lens and spasm of the ciliary muscles.

3. What are the implications for research, policy, or practice?
Anaesthetists and ophthalmologists need to be aware of this potential side effect and known that by simply ceasing COX-2 inhibitors this condition improves.

Background

Myopic shift due to medications is rare and can often be misdiagnosed and mistreated. This paper highlights the potential mechanisms for myopic shift induced by COX-2 inhibitors and demonstrates that reversal of this pathology can be achieved by cessation of the medication which is the culprit. It aims to highlight to clinicians the importance of taking a thorough medication history in considering iatrogenic causes of acute refractory changes within the eye.

Case details

A 30-year-old female was reviewed by ophthalmology on day two post laparotomy and myomectomy with bilateral blurry vision which started following the operation. Her intraoperative medications included sevoflurane, midazolam, propofol, fentanyl, vecuronium, cephazolin, dexamethasone, oxycodone, paracetamol, metaraminol, parecoxib, ondansetron, glycopyrrolate and neostigmine. Past medical history included: uterine fibroids requiring a previous myomectomy and hypothyroidism due to Hashimoto's thyroiditis for which she was taking 50 micrograms of thyroxine daily. After her previous myomectomy she did not suffer any post-operative visual disturbances although was given similar medications other than COX-2 inhibitors. Her baseline refraction was mild myopia (-0.25). Post-operatively, the patient was given regular celecoxib, cefazolin and paracetamol with oxycodone and ondansetron available PRN.

On initial review, she was found to be myopic (-5 and -4.5 dioptres in her right and left eyes respectively) with best corrected visual acuity of 6/5 in both eyes. She had a shallow anterior chamber (right eye 2.77mm and left eye 2.76mm with axial lengths of right eye 22.79mm and left eye 22.68mm respectively) (Table 1). On gonioscopy, her angle was narrow but open[a]. Her intraocular pressures

[a]Right eye findings: nasal quadrant pigmented trabecular meshwork, temporal quadrant Schwalbe's line, superior quadrant pigmented trabecular meshwork, inferior quadrant ciliary body.

were normal bilaterally. Autorefraction was -6.00 +1.50 (right eye) and -4.75+ 0.50 (left eye). B-scan ultrasonography demonstrated normal ciliary body size and position with no uveal effusion. Images of her anterior segment OCT demonstrated an open but shallow anterior chamber (Image 2). Her pupils were dilated with Cyclopentolate 1% which led to a mild improvement of her angles and refraction after only 30 minutes (-3 dioptres in her right eye and -2.5 dioptres in the left eye). IOL Master initially demonstrated a shallow anterior segment and thick lens likely due to lens iris diaphragm anterior shift. Repeat measurements post dilation demonstrated posterior movement of the lens-iris diaphragm towards the normal anatomical position, increasing zonular tension and resulting in subtle lens thinning (Figure 1). Ciliary body spasm may an explain this small improvement with use of a cycloplegic; however, does not fully account for the patient's myopic shift as this did not lead to improvement back to baseline.

Her myopia persisted on day three post op (Table 1) and it was at this stage, after a review of the literature and seeing no lasting improvement from cyclopentolate, that celecoxib and cyclopentolate were ceased. This was done as it was thought she had medication induced myopic shift from celecoxib which seemed theoretically reasonable based upon the reading of the literature and lasting anterior displacement of the lens iris diaphragm with lens thickening. The patients' vision and myopic shift drastically improved over the next two days after ceasing celecoxib and her vision soon returned to baseline, supporting this theory.

Discussion

Medication induce myopia is an uncommon side effect of many medications and has been documented to occur with medications such as sulphonamides (e.g., acetazolamide, topiramate, indapamide and sulphasalazine), cold and flu medication and insulin.[1,2] Often medication induced myopia is associated with choroidal detachment, maculopathy and retinal folds.[2]

There are three proposed pathophysiologic mechanisms by which this occurs.[2] The first is through lenticular swelling due to water imbibition leading to increased refractory power of the lens.[2] The second, is by swelling and rotation of the ciliary body leading to increased lens curvature, forward displacement and rotation of the lens iris diaphragm.[1,3] The third mechanism is sustained contraction or spasms of the ciliary muscles leading to decreased tension on the zonular fibres increasing refractory power of the lens.[2] All three may have been demonstrated in this case although it's difficult to determine if lenticular swelling was the cause of lens thickening or if it was primarily due to ciliary body swelling and relaxation of the zonular fibres.

There are approximately five cases of blurry vision documented from cyclo-oxygenase 2 inhibitors (COX-2 inhibitors) in the literature.[4] However, using National Registry of Drug-Induced Ocular Side Effects as well as case reports from the National Registry, the Food and Drug Administration and the World Health Organisation blurry vision due to COX-2 inhibitors was reported in 569 patients, 232 patients of which had a positive de-challenge.[4] Parecoxib associated cases were not included in this review.[4] The primary cause of blurry vision was thought to be caused by altered retinal blood flow due to inhibition of prostaglandins and mediation of synthesis of prostacyclin's.[4,5] It is also considered that COX-2 inhibitors may cause myopic shift through the same mechanism as sulphonamides as almost all COX-2 inhibitors contain a sulphonamide group or have a metabolite that has a sulphonamide group.[4]

Conclusion

From this case report we cannot confirm that celecoxib is the culprit of the patient's myopic shift. However, we believe that this is likely to be the case based upon the large number of reported cases of blurry vision from COX-2 inhibitors as well as the similarities in the physiological changes seen in this case and other cases of medication induced myopic shift. This case report demonstrates that myopic shift and anterior displacement of the lens iris diaphragm may be the primary cause of blurry vision caused by COX-2. It is possible that parecoxib caused the initial insult that led to myopic shift that continued to progress due to Celecoxib. It also highlights the importance of taking a thorough medication history when assessing patients with vision loss and that rapid recovery often occurs after removing the offending medication.

Left eye findings: nasal quadrant pigmented trabecular meshwork, temporal quadrant Schwalbe's line, superior quadrant pigmented trabecular meshwork, inferior quadrant pigmented trabecular meshwork).

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PEER REVIEW

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CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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PATIENT CONSENT

The authors, Kingston, E; Darian-Smith, E; Brothers, A; Bank, A declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.

Table 1: Post-operative measurements

		VA	Refraction	Anterior chamber depth (IOL master in mm)	Axial length (IOL master in mm)	Lens Thickness (mm)
Day 2 post operation (pre-cycloplegic)	RE	6/36	-5.00 → VA 6/5 (subjective refraction)	2.77	22.79	4.03
	LE	6/60	-4.50 → VA 6/5 (subjective refraction)	2.76	22.68	4.01
Day 2 (post cycloplegic)	RE	-	-3.00 +1.25	2.98	22.81	3.90
	LE	-	-2.50 +0.50	2.96	22.66	3.87
Day 3 post operation (morning)	RE	6/36	-5.50/+0.75 x 78 (autorefraction)	2.96	22.79	3.98
	LE	6/36	-4.75/ +1.00 X 91 (autorefraction)	2.94	22.65	3.96
Day 3 post operation (9 hours after last Celecoxib)	RE	6/36	-5.75/ +1.00 x 72 (autorefraction)	2.98	22.77	4.00
	LE	6/24	-5.25/ +0.75x71 (autorefraction)	2.98	22.64	3.95
Day 5 post operation (2 days after ceasing Celecoxib)	RE	6/5+4	-1.25 / +1.00 x90 → VA 6/4-1 (subjective refraction)	3.25	22.77	3.77
	LE	6/6-3	-1.00/ +0.50 x50 → VA 6/4-1 (subjective refraction)	3.21	22.64	3.76

Figure 1: Graphs of IOL master measurements over duration of admission