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Case Report

Episodic Angle Closure after Visian™ Implantable Collamer Lens Implantation in a Patient Using Adderall®

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Implantable collamer lens · Angle closure · Corneal refractive surgery · Adderall · Case report

Abstract

Introduction: Amphetamine-based medications such as Adderall®, used for the treatment of attention deficit-hyperactivity disorder (ADHD), may theoretically elicit angle closure through their adrenergic mechanisms. The relationship between the use of implantable collamer lenses (ICLs) and angle closure has been extensively investigated based on appropriate vault and lens sizing and postoperative changes in the anterior chamber angle (ACA) and corneal morphology. This case reflects a synergistic impact from both Adderall® use and ICL implantation for the proposed mechanism of angle closure. ***Case Presentation:*** A 36-year-old myopic female with ADHD controlled with Adderall® underwent toric ICL implantation in the right eye after undergoing preoperative laser peripheral iridotomy. Shortly after, the patient developed episodic angle closure in the right eye, with episodes mainly occurring after taking an additional dose of Adderall® in a dimly lit environment. The patient later had an ICL exchange with a smaller sized EVO+ toric ICL in the right eye and remained asymptomatic after. ***Conclusion:*** Additive mechanisms from both the ICL and Adderall® were present in our patient. The ICL caused crowding of the ACA through a pseudophacomorphic mechanism, and the Adderall® caused increased iridotrabecular contact secondary to pharmacologic mydriasis. This resulted in episodic angle closure with subsequent spikes in the intraocular pressure. There are no current reports or studies in the current literature describing the combined mechanisms

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of ICL implantation and Adderall® use in the potential development of angle closure. Further studies may be done to assess interactions of such medications in patients after ICL implantation.

Introduction

The implantable collamer lens (ICL) is a posterior chamber phakic IOL that is used to correct refractive error and can be used as an alternative to corneal refractive surgery [1]. Although uncommon, complications such as corneal endothelial loss, pigment dispersion, anterior subcapsular cataract, ocular hypertension, angle-closure glaucoma, and retinal detachment have been reported [1].

One parameter that is highly associated with successful postoperative outcomes is an appropriate vault (i.e., the distance between the anterior crystalline lens surface and the posterior ICL surface) secondary to appropriate ICL sizing, which can be measured through various diagnostic methods [2]. The optimal vault is between 250 and 750 μm or 0.5–1.5 times the corneal thickness [3], with vaults exceeding 750 μm being associated with angle-closure glaucoma and ocular hypertension [2].

Independent of ICL implantation, there are medications that may solely themselves put patients at risk for developing angle closure. Certain adrenergic, anticholinergic, antidepressant, antipsychotic, and sulfa-based agents may increase the risk of angle closure through pharmacologic mydriasis and subsequent narrowing of the anterior chamber angle (ACA). Adderall®, an amphetamine-based medication used for the treatment of attention deficit-hyperactivity disorder (ADHD), may potentially elicit this risk through its pharmacological mechanisms [4].

We present a case of episodic angle closure in a patient who underwent ICL implantation in both eyes and was concurrently using Adderall®. To the best of our knowledge, there are only studies and reports in the current literature that separately describe the association of ICL with angle closure [2] and the association of amphetamine use with angle closure [5]. We aim to uncover the potential additive mechanisms from both ICL and Adderall® use for our patient. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary file (for all online suppl. material, see https://doi.org/10.1159/000540080).

Case Presentation

A 36-year-old myopic female (−9.00 D right eye, −10.00 D left eye) with a past medical history of ADHD managed with 30 mg of Adderall® every morning, gastroesophageal reflux disease, and migraines presented for evaluation of candidacy for ICL implantation. Presurgical VuMAX ultrasound biomicroscopy (Sonomed Escalon, New Hyde Park, NY) measured a sulcus-to-sulcus of 11.35 mm, ACA of 43° (nasal)/38.6° (temporal), crystalline lens rise of 0.77 mm, and an aqueous depth of 3.0 mm for the right eye (Fig. 1). Further measurements of anterior segment biometric parameters including white-to-white (WTW) were performed with various diagnostic devices (Table 1). The decision was made after informed consent to proceed with ICL implantation in one eye at a time, starting with the right eye. ICL size selection was made based on optimized WTW,

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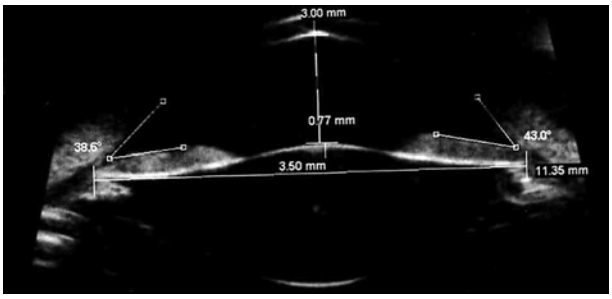

Fig. 1. UBM displaying presurgical STS, ACA, crystalline lens rise, and AQD of the right eye. UBM, ultrasound biomicroscopy; STS, sulcus-to-sulcus; AQD, aqueous depth.

Rocamora, Rivera, and Parkhurst nomograms. Two weeks prior to her surgery, the patient underwent 2 separate laser peripheral iridotomies (PIs) to account for the lack of a central aquaport in the selected ICL.

A −11.5 diopter 12.6 mm Visian™ toric ICL (STAAR Surgical, Monrovia, CA, USA) was implanted at a 180° meridian in the right eye through a 3.2-mm temporal corneal incision under topical anesthesia. Immediate postoperative vault was 359 μm. Intraocular pressure (IOP) measured via Tonopen was 14 mm Hg in the right eye. On postoperative day one, vault was 959 μm, uncorrected distance visual acuity (UDVA) was 20/20 + 2, and IOP via Goldmann was 20 mm Hg for the right eye. Slit lamp exam showed mild temporal corneal edema at the surgical incision site.

On postoperative day two, the patient presented with mydriasis and decreased vision in the right eye, with associated nausea and headache. UDVA and IOP measured via Goldmann were 20/40 + 2 and 47 mm Hg, respectively. The slit lamp exam showed corneal edema, a shallow anterior chamber, anterior displacement of the peripheral iris with ACA narrowing, and corneal haze. ACAs of the right eye measured via anterior segment optical coherence tomography were 17.19° (nasal) and 18.15° (temporal) (Fig. 2). Timolol maleate and brimonidine tartrate eye drops were administered to the right eye, the patient's existing PIs were enlarged, and a third additional PI was created at the 12 o'clock position to manage the new-onset acute angle closure. The patient's IOP subsequently normalized, and her UDVA improved to 20/16-1 over the next several days.

During the following month, the patient experienced recurrent episodes of eye pain with associated headache, "pressure" sensation, and blurred vision in the right eye. The patient reported that her symptoms would present approximately 1 h after taking an additional dose of Adderall®. Symptoms were temporarily relieved by inducing miosis with a light. UDVA in the right eye was 20/20. IOPs ranged from 15 mm Hg to 17 mm Hg. Vaults in the right eye at her appointments during this time ranged from 750 to 904 μm. PIs were patent and open. The patient was managed with timolol and brimonidine eye drops three times daily, and prednisolone eye drops twice daily. The patient was instructed to discontinue the Adderall®, which led to a temporary resolution of her symptoms for 1 month. The patient returned to the clinic 9 weeks after the initial surgery with a similar presentation, stating that she had restarted her Adderall® 1 day prior due to strenuous school and work demands. Once again, she noticed her symptoms started about 1 h after taking the medication. Due to the patient's persistent and necessitated use of Adderall®, a scheduled ICL exchange with the smaller −11.0 diopter 12.1 mm EVO+ Visian™ toric ICL (STAAR Surgical, Monrovia, CA, USA), which featured a central aquaport, was performed the following week. Postoperative ACA and ACD in the right eye were 33.5° (nasal)/34.6° (temporal) and 2.44 mm, respectively (Fig. 3). The patient achieved a favorable postoperative visual outcome and has remained asymptomatic to this date despite her continued usage of Adderall®.

Table 1. Anterior segment biometric parameters

	WTW, mm	ACA, °	CCT, µm	AQD, mm
NIDEK OPD-Scan III	11.81	–	–	–
OCULUS Pentacam® Topography	11.80	40.8	550	2.92
ZIEMER Galilei G4 Topographer	11.98	33.9	543	2.99
Haag-Streit Lenstar LS900 Optical Biometer	11.80	–	547	2.81

AQD, aqueous depth; CCT, central corneal thickness.

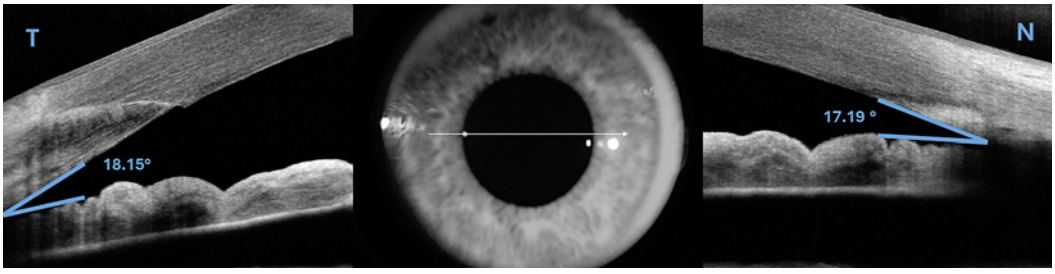

Fig. 2. AS-OCT image displaying nasal (right) and temporal (left) ACAs of the right eye after initial ICL implantation. AS-OCT, anterior segment optical coherence tomography.

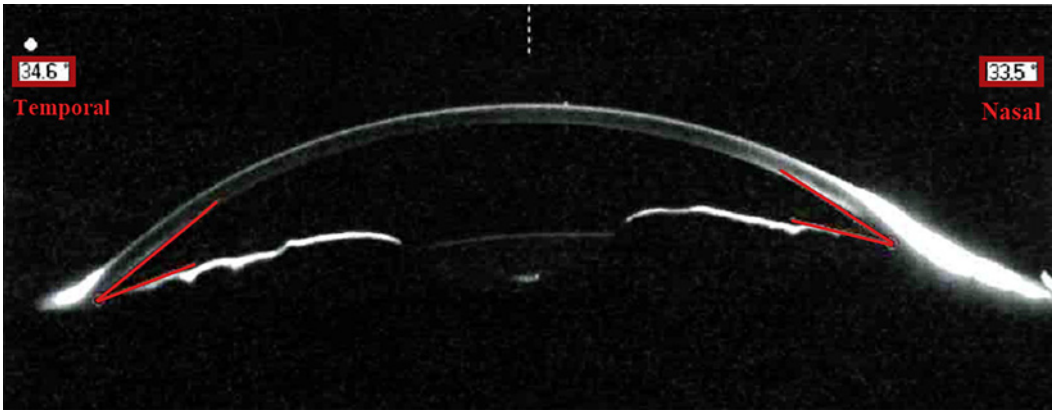

Fig. 3. Pentacam image displaying nasal (right) and temporal (left) ACAs of the right eye after ICL exchange.

Discussion

As mentioned earlier, postoperative vault is an important indicator for postoperative complications after ICL implantation. With vaults >750 µm, patients are at increased risk for elevated IOP and angle-closure glaucoma [2]. Some proposed mechanisms of elevated IOP and angle closure after ICL include pupillary block, non-pupillary block, aqueous misdirection, pigment dispersion, and steroid-induced [6, 7]. After ICL placement, our patient initially presented with mydriasis and anterior displacement of the peripheral iris with subsequent ACA narrowing in the right eye, leading to the preliminary diagnosis of angle closure due to a pupillary block. Despite enlarging existing PIs and creating a third PI to account for the lack of a central aquaport in her original ICL, the patient continued to be episodically symptomatic, with relatively increased IOPs compared to presurgical measurements. Additionally, there

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was absence of iris pigment in the trabecular meshwork on exam or anterior rotation of the ciliary bodies on ultrasound biomicroscopy, eliminating the possibilities of pigment dispersion or aqueous misdirection, respectively, as etiologies. Therefore, it was concluded that the cause of the patient's angle closure was a pseudophacomorphic mechanism, a type of non-pupillary block due to the increased volume in the posterior chamber from the ICL [8]. Despite this conclusion, the patient's presurgical WTW, sulcus-to-sulcus, ACA, and aqueous depth were all accurately measured, and the correct ICL size was chosen at the initial consultation visit based on nomogramic data. Given that the patient's symptoms mainly arose shortly after taking an additional Adderall® dose, we must consider the ophthalmic effects of this medication and how it may interact with ICL patients.

Adderall® is a combination medication consisting of racemic amphetamine and dextroamphetamine used for the treatment of ADHD [9]. Although the mechanism of action is not fully understood, the proposed theory is that Adderall® increases dopamine, serotonin, and norepinephrine concentrations in the synaptic cleft via metabolism inhibition and exocytosis of presynaptic vesicles [10]. Although Adderall® is beneficial for controlling the symptoms of ADHD patients, systemic effects of this medication must not be ignored [11]. Regarding ophthalmic manifestations, Adderall® and other ADHD stimulants may cause dry eye, mydriasis, accommodation loss, and blurred vision through activation of the sympathetic nervous system [12]. Pharmacologic mydriasis has been shown to increase iridotrabecular contact and crowding of the ACA, raising the risk for angle closure [4]. While the existing literature does not contain any instances detailing the association between Adderall® and angle closure, there have been reported cases with other amphetamine derivatives [5, 13]. A pediatric ADHD patient taking lisdexamfetamine developed bilateral acute angle closure, with resolution of symptoms after discontinuation of the medication [5]. Another rare case described a 39-year-old patient who developed bilateral angle closure shortly after 3,4-methylenedioxymethamphetamine use with subsequent spontaneous resolution [13]. It is evident that through their sympathomimetic effects, amphetamine derivatives have the potential to increase the risk for the development of acute angle closure.

The temporal association between the patient taking an additional Adderall® dose and the subsequent episodic angle closure and the resolution of symptoms after discontinuation of the medication are significant findings for our case. Even after creating a new PI to further combat the lack of a central aquaport in her original ICL, the patient continued to have recurrent episodes shortly after taking her Adderall®. Given the patient's negative past ocular history, we speculate that synergistic mechanisms from both the ICL implantation and Adderall® use led to the patient's episodic angle closure. After ICL implantation, studies have shown that there is a statistically significant decrease of the ACA by a mean value of 15° in patients with anatomically normal angles [14, 15]. With the pharmacologic mechanisms of Adderall® mentioned before, it is likely that the pharmacologic mydriasis from this medication amplified the patient's already crowded ACA and induced her angle closure and IOP spike. The patient subsequently had complete symptomatic resolution following ICL explantation and implantation of the smaller EVO+ ICL.

After a conducive search of the current literature, there are no current reports or studies describing the potential effects of Adderall® or any other amphetamine derivatives on ICL patients. This case is the first to our knowledge to describe the synergistic mechanisms of ICL implantation and Adderall® use behind the cause of episodic angle closure in ICL patients. Our findings demonstrate that clinicians should consider the use of the newer EVO/EVO+ ICLs for patients with confounding factors that may increase their risk for angle closure. Further studies need to be performed to investigate the potential need of a lower vault threshold, EVO+ ICL utilization, or switching to non-stimulant medications for patients who are on Adderall® or other amphetamine-based derivatives.

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Statement of Ethics

No ethics approval was required for this report in accordance with national guidelines. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest Statement

None of the authors have any conflicts of interest to declare.

Funding Sources

The authors have no funding to declare.

Author Contributions

Dr. Majid Moshirfar, Dr. Kayvon Moin, Muhammed Jaafar, and Kenneth Han reviewed the patient's medical record, formulated the original draft, and revised intellectual content. Soroush Omidvarnia and Dr. Phillip C. Hoopes revised the manuscript and intellectual content.

Data Availability Statement

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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