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Anterior Vitreous Migration after 0.01cc Intravitreal Injection

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Introduction

Intravitreal injection of anti-vascular endothelial growth factor (anti-VEGF) agents, including bevacizumab, ranibizumab, aflibercept, and faricimab, among others, have transformed retinal disease management, improving outcomes for several retinal conditions associated with macular edema and choroidal neovascularization.[1] In cases refractory to standard dosing, dose escalation has been explored. While dose, and thus volume, escalation may also enhance efficacy in some non-responders, the practice may also be associated with a higher risk of untoward outcomes in select cases.[2–5]

Transient intraocular pressure (IOP) elevation is a known short-term finding following intravitreal injection, especially when larger volumes are introduced into the vitreous cavity. [4,6] The sudden IOP increase generates mechanical forces capable of displacing the vitreous body into the anterior chamber, especially in eyes with zonular weakness.[7] This event can precipitate additional consequences, including intraocular lens dislocation, corneal decompensation, secondary glaucoma, and retinal breaks, among others.[7,8,9] Despite these potential consequences, descriptive reports remain sparse in the literature.[8]

We present a case of vitreous prolapse into the anterior chamber following intravitreal double-dose aflibercept (Eylea, 4 mg in 0.10cc) injection.

Case Report

A 74-year-old male with a history of grid-pattern laser photocoagulation for perfused macular edema due to branch retinal vein occlusion and history of secondary choroidal neovascularization in the left eye was receiving monthly intravitreal injections of standard-dose (2mg in 0.05cc) aflibercept. The left eye was also noted to have a posterior vitreous detachment and was pseudophakic with an intact posterior capsule. There was no history of ocular trauma or surgical complication, including posterior capsular tear, floppy iris, or zonulopathy.

Following an incomplete response to standard-dose aflibercept, double-dose aflibercept (4mg in 0.1cc) was initiated with expected post-injection IOP spikes after each one. These IOP elevations ranged from mid-30s to low 50s mmHg and were managed with topical apraclonidine. After his eighth double-dose injection, the patient experienced an acute IOP elevation to 54 mmHg that was reduced to 32 mmHg with topical apraclonidine.

The following day, the patient noted decreased vision and photophobia in the left eye. Corrected visual acuity in the left eye declined from 20/30 to 20/100. IOP was 17 mmHg. Slit-lamp examination revealed the posterior chamber IOL with intact posterior capsule subluxed superotemporally while displaced slightly posteriorly. Prolapsed vitreous was noted in the anterior chamber along the iris at 6 and 12 o'clock and seen engaging the IOL from 6-10 o'clock (Figure 1A). There was no evidence of corneal edema, and the anterior chamber was deep without signs of inflammation or hypopyon. Dilated fundus examination did not show any retinal breaks and revealed stable macular findings. OCT revealed a stable subfoveal drusenoid pigment epithelial detachment with patchy ellipsoid layer loss and juxtafoveal intraretinal hyperreflective material without macular edema (Figure 1B).

Double-dose aflibercept was discontinued and monthly intravitreal faricimab initiated.

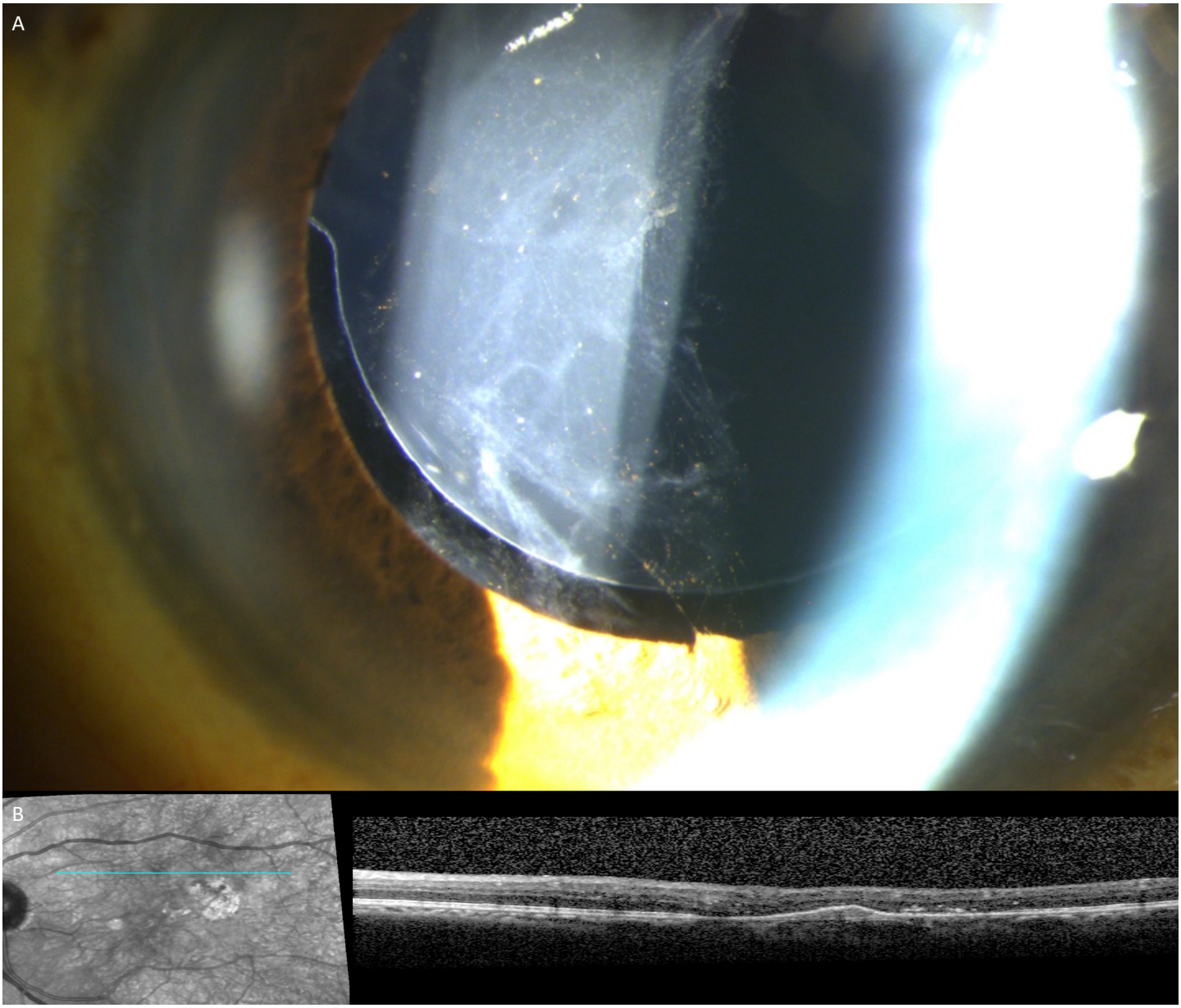

Figure 1. A. Narrow beam slit lamp microscopy demonstrated vitreous strands with suspected pigmented cells anterior to the intraocular lens and adhered to the iris. B. OCT demonstrated subfoveal drusenoid pigment epithelial detachment with patchy ellipsoid layer loss and juxtafoveal intraretinal hyperreflective material without significant macular edema.

Discussion

Transient IOP elevations are a well-established, short-term consequence following intravitreal injection.[4] The rapid increase in IOP generates mechanical forces that can displace the vitreous, particularly in eyes with pre-existing structural vulnerabilities.[7] In this case, repeated intravitreal injections resulting in transient IOP spikes likely weakened zonules, creating a pathway for future vitreous migration anteriorly. The observed IOL displacement, despite an intact posterior capsule, supports the hypothesis that compromised zonular integrity enabled vitreous migration under mechanical strain following an acute pressure spike.

The combination of acutely increased IOP and weakened zonules provides a plausible mechanism consistent with previously reported cases. Haddock et al. described vitreous prolapse through a scleroconjunctival defect following multiple intravitreal injections targeting the same region over time, underscoring the role of cumulative mechanical stress in weakening tissue.[7] Similarly, Degenring et al. reported vitreous migration into the anterior chamber in pseudophakic eyes with known posterior capsular defects after intravitreal injection of 20mg of triamcinolone acetonide in 0.20cc, highlighting the role of pre-existing structural weakness in facilitating vitreous migration into unwanted locations.[8]

Vitreous prolapse can contribute to untoward outcomes. Adhesion to the corneal endothelium may lead to corneal decompensation, while zonular loss can allow vitreous to displace the lens. Traction on the iris may lead to pupil irregularities, while obstruction of the trabecular meshwork can result in secondary glaucoma. Moreover, photopsia, macular edema, and tractional retinal detachment may be induced by Vitreous Tug Syndrome.[10] These complications underscore the need for tailored management based on severity, ranging from close monitoring if deemed safe to surgical intervention such as laser or surgical vitreolysis, anterior or pars plana vitrectomy, and/or IOL exchange, among others.[10,11]

While the incidence of vitreous prolapse into the anterior chamber is rare, preventative measures can reduce its risk. These include precise injection techniques such as avoiding injections too close (or far) from the limbus, using a smaller gauge needle, preferably 30G, and injecting the volume at a slower rate.[12] Studies suggest that pretreatment with aqueous suppressants can mitigate acute IOP spikes[4] and possibly mechanical displacement of vitreous. Considering the patient's history of post-injection IOP elevations and prior successful use of apraclonidine, such pre-treatment would have been a reasonable approach. Despite the frequent occurrence of IOP spikes after intravitreal injection, there remains no standardized pre-injection protocol to prevent acute IOP elevation.

The introduction of newer anti-VEGF agents such as faricimab and aflibercept high dose has transformed the management landscape. Faricimab, a bispecific antibody targeting both VEGF and angiopoietin-2, may offer enhanced efficacy with reduced dosing frequency in a 0.05cc volume.[13] Aflibercept high dose delivers 8 mg of aflibercept in 0.07cc, a slightly higher volume compared to the standard 0.05cc volume. While studies note this slight volume difference between standard aflibercept and aflibercept high dose, the resulting IOP spikes were reported to be statistically insignificant. This suggests that aflibercept high dose may deliver a more efficacious therapeutic load without increasing the risk of clinically significant short-term IOP elevation.[14] These newer agents reduce the need for double-dose volumes, which may in turn minimize the likelihood of acute IOP elevation in some eyes.

Conclusion

Vitreous prolapse following intravitreal injection may occur. Identifying risk factors, optimizing injection technique, and managing perioperative IOP may help decrease the risk of vitreous prolapse.

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Statement of Ethics

This case report adheres to patient confidentiality and ethical principles in accordance with the guidelines of the Declaration of Helsinki and relevant local regulations. Consent was obtained from the patient for the publication of this case report.

Conflict of Interest Statement

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Authorship

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