Acute angle closure glaucoma as a rare complication of anticoagulant therapy

Abstract: Acute angle closure glaucoma secondary to suprachoroidal hemorrhage is a rare, but devastating complication of anticoagulant therapy. Subretinal neovascularization is often the source of bleeding. Clinicians should understand this risk in patients with wet ARMD.

I. Case History

Patient demographics:
- 80 year-old white male

Chief complaint:
- intense pain and loss of vision OD for four days
- seen 5 days prior in the ophthalmology clinic for a comprehensive eye exam

Ocular History:
- Wet ARMD OS with PED
- Status post 4 intra-vitreal Lucentis injections OS
- Submacular heme OD with history of wet ARMD
- Grade 2+ nuclear sclerotic cataracts OU and early PSC OD

Medical history:
- atrial fibrillation
- benign prostatic hypertrophy
- coronary artery disease
- hyperlipidemia
- hypertension

Current medications:
- lorazepam 0.5 mg
- tamulosin 0.4 mg
- warfarin 5 mg
- furosemide 20 mg
- aspirin 81 mg
- simvastatin 80 mg
- metoprolol succinate 200 mg

Laboratory studies:
- INR: 5.26 (Most recent lab result from 2 weeks prior to onset of symptoms)

II. Pertinent findings

- Best-corrected visual acuity was: OD NLP, reduced from CF at 4 feet 5 days prior, OS 20/70 with no improvement on pinhole
Entrance tests revealed full extraocular motility OU, poor pupillary reaction to light OD, and round reactive pupil OS
- Slit lamp examination OD showed grade 3 conjunctival injection with chemosis, hazy cornea, and iris bombe
- Angle assessment: Van Herrick grade 0 OD, open OS
- Gonioscopy OD with 4-mirror: no visible structures
- Anterior segment exam was unremarkable OS
- Applanation tonometry measured: OD 60 mmHG, OS 14 mmHG
- B-scan ultrasound revealed hyperechoic material, consistent with a massive choroidal hemorrhage, and no apparent retinal detachment.

III. Differential diagnosis

For Angle Closure Glaucoma:

1. Pupillary block
2. Intraorbital tumor or mass
   Yanoff found that approximately 3% of patients with posterior uveal melanoma can present with acute or chronic angle closure glaucoma secondary to a spontaneous subretinal or intravitreal hemorrhage.
3. Chronic myelocytic leukemia (CML)
   Kozlowski et al reported that alteration of vessel walls by leukemic cells or faulty blood clotting mechanisms in CML may have contributed to a case of massive subretinal hemorrhage with angle closure.
4. Hemorrhagic choroidal or retinal detachment
5. Phacomorphic acute angle closure

IV. Diagnosis and Discussion

This patient was diagnosed with an acute angle closure glaucoma attack resulting from a suprachoroidal hemorrhage incited by wet macular degeneration and anticoagulant therapy. Patients with choroidal neovascular membrane or subretinal hemorrhage are at increased risk for bleeding while on anticoagulant therapy. When the subretinal or choroidal hemorrhage occurs, it can lead to a detachment with the accumulation of serous or serosanguinous material in the suprachoroidal space. This causes posterior pressure on the lens-iris diaphragm precipitating the angle closure glaucoma attack. This most likely occurred in our patient who had a history of wet ARMD OD with a pre-existing submacular hemorrhage.
Anticoagulants are used in the prevention and treatment of thromboembolic disorders such as deep vein thrombosis, atrial fibrillation, and cerebrovascular accident. By inhibiting thrombosis, patients are at increased risk for hemorrhagic complications. Ocular complications are generally rare in patients taking anticoagulants. The most common ocular finding is subconjunctival hemorrhage. At the other end of the spectrum is spontaneous hemorrhagic retinal or choroidal detachment causing secondary angle closure glaucoma. This medical emergency oftentimes has a poor visual prognosis resulting in a blind, extremely painful eye with elevated intraocular pressure. Other risk factors for spontaneous suprachoroidal hemorrhage include hypertension, advanced age, and subretinal neovascularization or bleeding.

V. Treatment and Management

Initial treatment included immediate efforts to reduce intraocular pressure with instillation of topical medications such as a beta blocker (eg Timolol 0.5%), alpha-2 agonist (eg Aproclonidine 1%), pilocarpine 1%, topical carbonic anhydrase inhibitor (eg Dorzolamide), and oral carbonic anhydrase inhibitor (eg Diamox 500 mg p.o). The patient was referred immediately to Ophthalmology for surgical care to lower intraocular pressure.

The patient was prescribed Diamox 500 mg bid SR, Pred Forte QID OD, Cosopt BID OD and Alphagan TID OD. He was scheduled for laser peripheral iridotomy the next day. Two days s/p LPI OD the IOP was 32 OD, LPI was not patent, and no structures were visible on gonioscopy. After a second LPI procedure OD, poor response to anti-glaucoma medication, and subjective reports of persisting pain, patient was advised on enucleation vs. cyclocryoablation. The patient preferred keeping his eye and underwent cyclocryoablation. At one month post-op the patient’s subjective reports of intense pain OD have diminished almost entirely, with an applanation tonometry measurement of 28 mmHg OD.

Other treatments of suprachoroidal or subretinal hemorrhage include external sclerotomy with drainage of blood or serosanguinous material.

VI. Bibliography


**VII. Conclusion**

Although this complication is rare, with the increasing number of patients on anticoagulant therapy it is important for both practitioners to be aware of the association. Therefore, early diagnosis and treatment can improve the patient’s prognosis.

It is essential to consult with the patient’s primary care physician and/or cardiologist to determine if lowering anticoagulant medication levels is warranted. Our patient possesses several of the characteristics for suprachoroidal hemorrhage such as advanced age, hypertension, and ARMD which puts him at increased risk for complications with his other eye.