Background: Antidepressants have been associated with acute angle closure glaucoma; however, the pathophysiology of this relationship remains unclear. The following case discusses the development of an acute angle closure attack in a patient treated for depression with Paroxetine.

I. Case History: A 55 y/o male reported to the clinic Aug 2011 with symptoms of blurry vision in his left eye upon wakening at 3am. The patient did not have a history of trauma and denied associated symptoms of pain or light sensitivity. Patient has been followed closely with the ophthalmology department for a conjunctival nevus that has remained stable for the past 2 years. Patient is currently taking Insulin, Amlodipine, Propranolol, Lisinopril, Crestor, Paroxetine, Duloxetine, and Lorazepam for control of Type II DM, Hypertension, Hyperlipidemia and Anxiety. Other pertinent medication information, it was discovered that the patient’s dosage of Paroxetine had recently been increased from 60 mg to 80mg 2 weeks prior to visit in eye clinic.

II. Pertinent Findings: Best-corrected visual acuity was 20/20 OD and 20/50 with pinhole to 20/30 OS. Ocular examination revealed 4+ Descemet’s folds and central stromal haze OS with an intraocular pressure of 16 OD and 14 OS. Pachymetry indicated a central cornea thickness of 575 in the right eye and 710 in the left eye. The patient was noted to have shallow anterior chambers in both eyes. Gonioscopy was attempted, exhibiting a narrow angle, open to ATM OD and appeared open but narrow OS despite the difficulty of evaluating the left eye due to the significant corneal edema.

III. Differential Diagnosis: In the management of a patient with an acute onset of blurry vision secondary to unilateral corneal edema with stromal haze, differential diagnoses included Acute Angle Closure Glaucoma, Intermittent Angle Closure Glaucoma, Chandler’s Syndrome, and Fuch’s Endothelial Dystrophy

IV. Diagnosis and Discussion: The patient was diagnosed with sub-acute angle closure glaucoma. Intermittent (subacute) angle closure occurs in susceptible patients with an anatomical narrow angle. Intermittent angle closure is characterized by rapid closure of the angle and sudden increase in IOP, which resolves spontaneously once the angle reopens. A dilated pupil or forward movement of the iris/lens diaphragm, ultimately causing pupillary block, can trigger angle closure attacks. The attack usually lasts 1-2 hours and is broken by physiological miosis such as exposure to bright sunlight or sleeping. After the attack the eye will appear white and quiet and the IOP is normal.
There have been several reports implicating Selective Serotonin Reuptake Inhibitors (SSRIs) such as Paroxetine and Fluoxetine as the causative agents in patients with acute angle closure based on their anticholinergic and/or serotoninergic properties; however, the exact mechanism remains unclear.

V. Treatment/Management: The patient was not treated with medical therapy and was instructed to f/u the next day with ophthalmology for LPI evaluation. Patient presented the next day showing a reduction in corneal edema with a decrease in central cornea thickness to 670. There was a 2 line improvement in visual acuity and IOP measured 13 OD and 12 OS. Prophylactic LPI was initiated in both eyes and the patient was instructed to return to clinic in 1-week for a post LPI evaluation. At the 1-week f/u, the patient's vision had recovered to 20/20 and there was no evidence of residual corneal edema. It should be noted, the patient was not encouraged to discontinue or change his current Paroxetine therapy before consulting with his PCP.

Current literature relating SSRI’s to acute angle closure suggests a slow serotoninergic effect on the iris/ciliary body, evidenced by a delay of 2 weeks after increasing the dosage before the initial attack. According to a 2008 article in Current Neuropharmacology, there have been 6 reported cases of angle closure attack induced by Paroxetine. A small number of reports relate acute angle closure with use of Citalopram and Fluvoxetine; although, a greater number of attacks were reported with Paroxetine as compared to other SSRI’s. This article further details the role of mydriasis as a leading factor in the onset acute angle closure attack.

VI. Conclusion: The number of patients in the clinical population diagnosed with depression and anxiety continues to grow making antidepressants one of the most frequently prescribed medications amongst general practitioners. With the number of people taking antidepressants increasing, there has been a rise in reports linking angle closure attacks with the use of SSRIs. There have been a limited number of animal studies with evidence of serotonin receptors located in the iris and ciliary body and through stimulation of these receptors, mydriasis and increased IOP will occur. Antidepressants have the potential to cause angle closure glaucoma especially in individuals susceptible to this type of event such as those with a hyperopic refractive error or narrow anatomical angles. Patient’s started on these drugs should be made aware of the ocular risks associated with these medications along with associated symptoms.
Bibliography


