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Case Report

A case of bilateral acute angle closure attack with some unusual features

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ABSTRACT

A 45 year old male presented with sudden severe pain, redness and marked diminution of vision in both eyes along with corneal oedema, pigmented KPs and raised IOP. After treatment with hyper osmotic agents, IOP came down and cornea became clear. Anterior chamber was shallow bilaterally with ring synechia. Gonioscopy revealed peripheral anterior synechiae in left eye and occludable angle in the right eye. Fundus examination was within normal limit. The patient was treated with antiglaucoma medications and steroid. Trabeculectomy was done in left eye with laser PI in the fellow eye. IOP came down within normal limits on subsequent visits with residual iris sphincter damage.

Keywords: Bilateral angle closure attack, Pigmented kp, Bilateral uveitis

INTRODUCTION

Most attacks of angle-closure glaucoma are unilateral. However, 5-10% of the attacks may affect both eyes simultaneously.[1] It presents with a sudden onset of pain or aching on the side of the affected eye. This pain is accompanied by blurred vision or coloured haloes around lights and ocular congestion.

Examination shows markedly raised IOP, corneal oedema, Shallow anterior chamber, Mid-dilated and sluggishly reactive pupil, Closed angle and peripheral anterior synechiae on gonioscopy. Cells and flare are present in anterior chamber but signs of anterior uveitis like pigmented KPs and ring synaechia are not commonly seen.

On the other hand uveitic glaucoma is usually a unilateral condition which gives signs of uveitis with increase intraocular pressure. Unilateral glaucoma should raise suspicion of an inflammatory cause.[2] Bilateral uveitic glaucoma is rare and on first attack primary angle closure usually would not leave such signs like sphincter damage or ring synechia. Our case presented with acute onset of eye pain along with an IOP of 50.6mm of Hg in right eye and 43.4mm of Hg in left eye. There were signs of bilateral uveitis without any history of previous attacks.

When corneal edema subsided, fundus showed normal disc, but there was sphincter damage, along with fully dilated pupil, glaukomflecken and ring synechia. Gonioscopy revealed occludable angle in both eyes (all quadrants in right eye and two quadrants in left eye) and peripheral anterior synechiae in two quadrants in left eye. Therefore we think this is a unique case and hence we are presenting it.

CASE REPORT

One male patient of 45 years security guard by occupation was brought to Eye O.P.D. with sudden onset of severe pain, redness and marked diminution of vision in right eye for the last three days. Similar symptoms were also present in the left eye since last two days. These symptoms started at 2 am in the morning during his night duty and were associated with severe headache,

vomiting, coloured halos and intolerance to light in right eye.

The other eye was affected with same symptoms on the following night. There was no history of similar attacks in past. There was no history of use of any topical or any systemic medications.

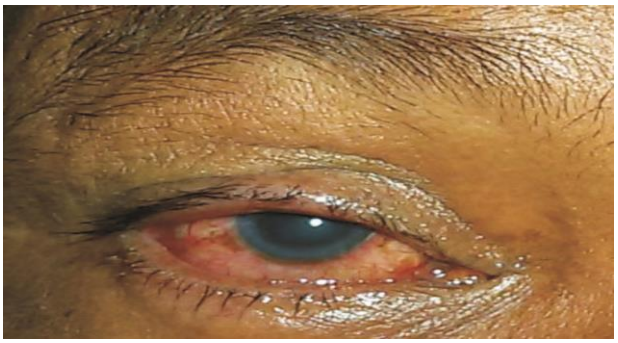

Figure 1: Right eye of the patient.

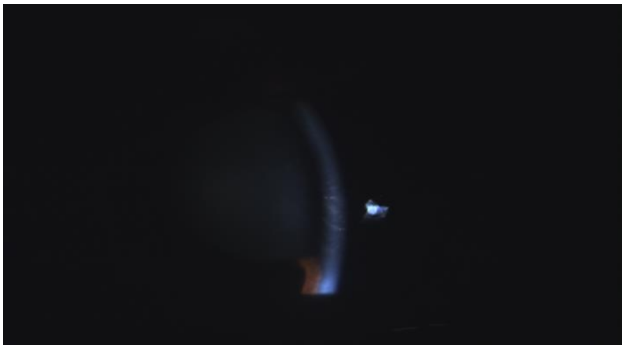

Figure 2: Slit lamp photograph of right eye.

Examinations revealed that in both eyes, conjunctiva was congested (Both superficial and deep congestion); cornea was hazy and oedematous (Figure 1 and Figure 3); descemet's folds were seen along with granular pigmented KPs (Figure 2 and Figure 4).

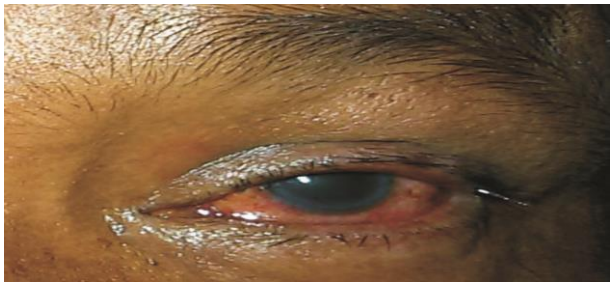

Figure 3: Left eye of the patient.

Corneal sensation was impaired in both eyes; anterior chamber was shallow; (Van-Herick's grading – Grade 0). Pupils were mid-dilated, round and not reacting to light: iris was just visible through hazy cornea; fundus was not visible. Visual acuity in both eyes was hand movements close to face. IOP in right eye was 50.6mm of Hg and in left eye 43.4mm of Hg.

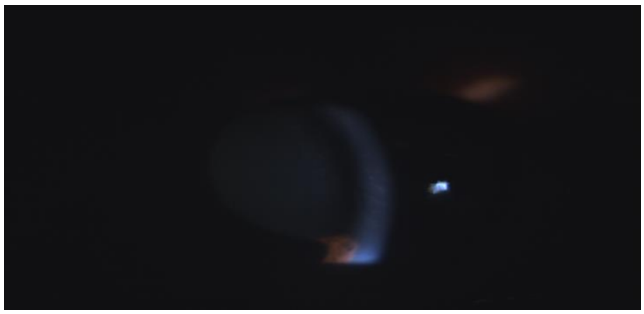

Figure 4: Slit lamp photograph of right eye.

Systemic examinations were nothing contributory. The patient was admitted and kept under medical treatment immediately-with mannitol 20% 200ml iv infusion over 30 to 60 mins. Along with tab. acetazolamide 250mg one tab twice daily after meal for 5 days, E/D dorzolamide + timolol maleate (0.5%) one drop 2 times daily in B/E to continue, E/D prednisolone acetate one drop 4 times daily B/E and tab. aceclofenac one tab after meal SOS.

After 24 Hours with aforesaid treatment, his intraocular tension came down, apprecia¬bly. Congestion and redness of the conjunctiva were less; corneal oedema subsided and both eyes revealed ring synechia and glauckomflecken. Treatment was continued for two days more and Gonioscopy was done on next day. Gonioscopy findings were (Figure 5 and 6).

According to Shaffer:

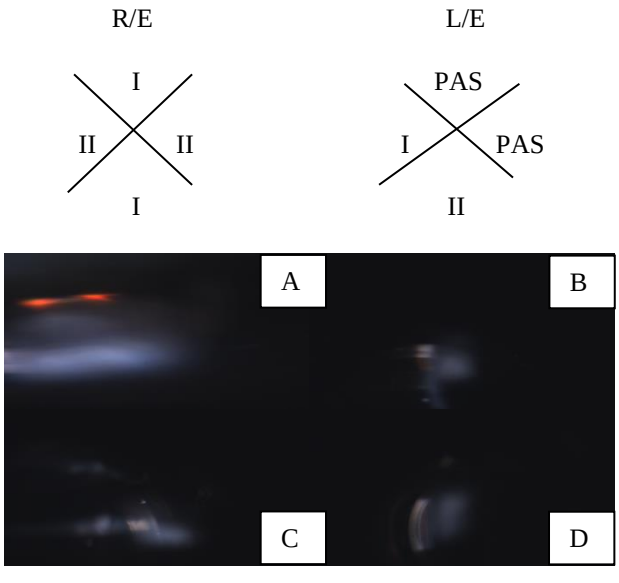

Figure 5: Gonioscopy photograph of right eye.

Trabeculectomy L/E and Laser PI R/E was done. At the time of discharge, in both eyes inflammation was subsided along with well-formed anterior chamber and

good subconjunctival bleb. Visual acuity in right eye was 6/18 and in left eye was 6/24.

The patient came for follow up after 15 days and his IOP was found to lie within normal range, well-formed anterior chamber and good sub-conjunctival bleb with no decrease in visual acuity.

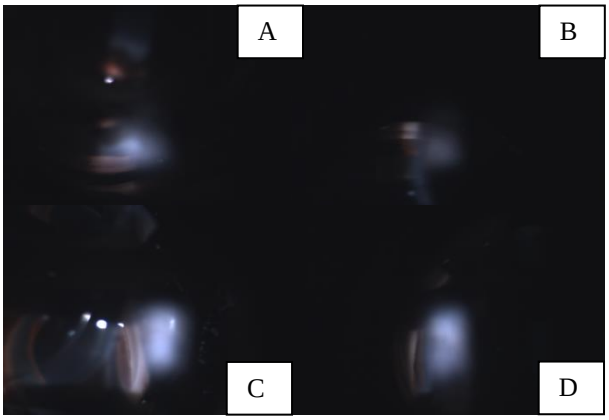

Figure 6: Gonioscopy photograph of left eye.

DISCUSSION

Acute primary angle closure (APAC) is a distinctive form of clinical disease, with a constellation of presenting signs and symptoms requiring urgent intervention, as well as preventive measures for the fellow eye**.** Visual acuity is usually 6/60 to HM. The IOP is usually very high (50-100 mmHg) with conjunctival hyperaemia, violaceous circumcorneal injection and corneal epithelial oedema. The AC is shallow, and aqueous flare is usually present. An unreactive mid-dilated vertically oval pupil is seen.

The fellow eye typically shows an occludable angle; if not present, secondary causes should be considered. In resolved acute primary angle closure attack- early findings are- low IOP (ciliary body shutdown and effect of intensive treatment), folds in Descemet membrane if IOP has reduced rapidly, optic nerve head congestion and choroidal folds.

Bilateral Acute primary angle closure attack is rare. Dr. JSM Lai et al reported that in his study 98% of acute angle closure attack was presented with pain but none of the cases were bilateral.[3] But in this case patient presented with bilateral severe eye pain and coloured halos. Visual acuity in both eyes was hand movements close to face. High IOP was in both the eyes. Other signs like conjunctival hyperaemia, violaceous circumcorneal injection and corneal epithelial oedema, shallow AC, unreactive mid-dilated vertically oval pupil, occludable angle were also present bilaterally.

Late findings of APAC are- iris atrophy with a spiral-like configuration, glaukomflecken (white foci of necrosis in the superficial lens) and other forms of cataract, and irregular pupil due to iris sphincter/dilator damage and posterior synechiae; the optic nerve may be normal or exhibit varying signs of damage, including pallor and/or cupping.[4] But in this case glaukomflecken, iris sphincter damage were present although there was no history of past attacks.

In present case there were also bilateral ring synechia, with pigmented Kps which are not common in APAC and point towards uveitis but bilateral acute anterior uveitis and uveitic glaucoma is rare. Study conducted by Anitha et al shows only 5% of cases of acute anterior uveitis are bilateral and most of the patients presented with pain in the eyes.[5] Rathinam SR et al shows 15% of cases of acute anterior uveitis were bilateral at presentation.[6]

Presenting symptoms of acute anterior uveitis include pain, redness, photophobia, and blurred vision**.** Slit lamp examination will reveal anterior chamber cells and flare. Ciliary flush, keratic precipitates. Elevated pressure can be seen during the acute phase of the disease, particularly with viral anterior uveitis, Possner Schlossman syndrome and uveitic glaucoma. An open angle can be blocked by inflammatory cells and fibrin, decreasing aqueous outflow. Over time, peripheral anterior synechiae formation can lead to complete angle closure and iris bombe.

Previous episodes of anterior uveitis will lead to sequele of anterior chamber inflammation that can be seen on slit lamp examination. These findings include old, pigmented or crenated keratic precipitates, peripheral anterior synechiae, posterior synechia or remnants of broken posterior synechiae on the anterior lens capsule, pupillary membrane or occlusio pupillae. But in this case ring synechia, with pigmented Kps were present without any history of past attacks.

Unilateral attacks of mild non-granulomatous anterior uveitis with elevation of IOP are the hallmark of Posner Schlossman syndrome (PSS).[7] Symptoms may include unilateral blurred vision and pain. Clinical examination reveals mild anterior chamber reaction and small to medium sized non-granulomatous keratic precipitates, corneal edema. IOP is usually 40-60 mmHg.[8]

CONCLUSION

So clearly this case of bilateral acute angle closure attack has some unusual clinical signs like bilateral ring synechia, with pigmented Kps which mimics uveitis.

The symptoms in this case and signs like presence of peripheral anterior synechia, occludable angle, iris sphincter damage and glaukomflecken more points towards acute angle closure attack and does not match completely with clinical features of either acute anterior uveitis or acute primary angle closure or Posner Schlossman syndrome. So we think this is an unique case presenting a diagnostic dilemma.

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