# A CASE OF BILATERAL ACUTE ANGLE CLOSURE ATTACK WITH SOME UNUSUAL CLINICAL FEATURES

*Singh P, Rijal AP, Rizyal A, Karmacharya S*

Department of Ophthalmology, Nepal Medical College Teaching Hospital,
Attarkhel, Gokarneshwor-8, Kathmandu, Nepal

## ABSTRACT

Bilateral acute angle closure attack is a rare ocular emergency. Early diagnosis and immediate intervention can have a profound effect on patient's visual outcome and ocular morbidity. A 70 year old female presented with vomiting about 7 to 8 episodes along with sudden diminution of vision in both eyes for last 3 days. Initially she was examined by physician where all the routine blood tests and upper GI endoscopy was advised which failed to reveal the cause. She was then referred to our department for ophthalmic evaluation. On ocular examination she was diagnosed as a case of bilateral acute angle closure attack with some unusual clinical features. After treatment with hyper osmotic agent, anti glaucoma drugs and Nd: Yag peripheral laser iridotomy, intraocular pressure came down to normal limits and the patient regained good vision.

## KEYWORDS

Bilateral angle closure attack, intraocular pressure, Laser Peripheral Iridotomy

## CORRESPONDING AUTHOR

Dr Pranisha Singh,
Assistant Professor,
Department of Ophthalmology,
Nepal Medical College Teaching Hospital,
Attarkhel, Gokarneshwor-8, Kathmandu, Nepal
Email: prani_s@hotmail.com
ORCID ID: 0000-0001-8459-6515

## INTRODUCTION

Acute angle closure attack is an ophthalmic emergency that progresses to blindness if untreated. Most attacks of acute angle-closure are unilateral. Acute Angle Closure (AAC) attack presents with a sudden onset of pain or aching on the side of the affected eye. This pain is accompanied by diminution of vision or colored haloes around lights and ocular congestion. Ocular examination shows markedly raised intraocular pressure (IOP), corneal edema, shallow anterior chamber, mid-dilated and sluggishly reactive pupil, closed angle and peripheral anterior synechiae on gonioscopy. Since most cases of AAC are unilateral, we have reported a case of bilateral AAC with some unusual clinical features. This case responded well to treatment.

## CASE REPORT

A 70 year old female presented with vomiting about 7-8 episodes, along with sudden diminution of vision in both eyes (BE) for the last 3 days. She had no history of headache, ocular pain and colored haloes. She gave no history of similar symptoms in the past. There was no history of wearing glasses or taking any ocular or systemic drug other than antihypertensive and anti diabetic drugs for the last 8 years. Initially she was examined by physician where all the routine blood tests and upper GI endoscopy were advised but failed to reveal the cause. She was then referred to our department for ophthalmic evaluation. On examination, her blood pressure was 110/70mmHg, pulse was 84/min, respiratory rate was 24/min and temperature was 97.2˚F. Systemic examinations were within normal limits. On ocular examination her visual acuity was finger counting 1ft in BE which was not improved with pinhole. There was no circumciliary congestion of conjunctiva, no corneal epithelial edema but few Descemets membrane folds were noted in BE (Fig. 1). Anterior chamber depth was shallow (Van Herick's Grade I) with occasional cells bilaterally (Fig. 2). Iris looked normal with no atrophic patches. Both pupils were mid-dilated around 4-5mm and non reactive (Fig. 1). Nuclear sclerosis grade I was noted with no glaucomfleckons in both lens (Fig. 1). Fundus examination showed hyperemic disc in BE. Cup Disc Ratio was 0.3:1 with intact neuroretinal rim bilaterally. Intraocular pressures (IOP) by applanation tonometer was 50mmHg and 46mmHg in right and left eye respectively. The patient was admitted and treated with infusion 20% mannitol intravenous 200ml over 45 minutes stat, Tab acetazolamide 250mg one tab four times per day, pilocarpine eyedrops 2% one drop four times per day, combination of timolol 0.5% and brimonidine eyedrops 0.15% one drop two times per day, combination of ofloxacin 0.3% and dexamethasone 0.1% eyedrops one drop four times per day. After two hours of treatment her vision had improved to 6/60 bilaterally and IOP came down to 22 mmHg and 20 mmHg in right and left eye respectively. On the next day of admission, patient was very comfortable with no specific complains. She regained her vision to 6/18 in BE. Her IOP was 10 mmHg bilaterally. Gonioscopy was done which revealed closed angle in BE with no peripheral anterior synechiae. Nd:Yag Laser Peripheral Iridotomy (LPI) was done at superior temporal region in right eye (RE) and superior nasal region in left eye (LE). Tab acetazomalide and pilocarpine eyedrops were stopped; other eye drops were continued and patient was advised to follow up after 7 days. On the 7$^{th}$ day, her best corrected visual acuity (BCVA) was 6/12 in BE and IOP was 10 mmHg bilaterally.

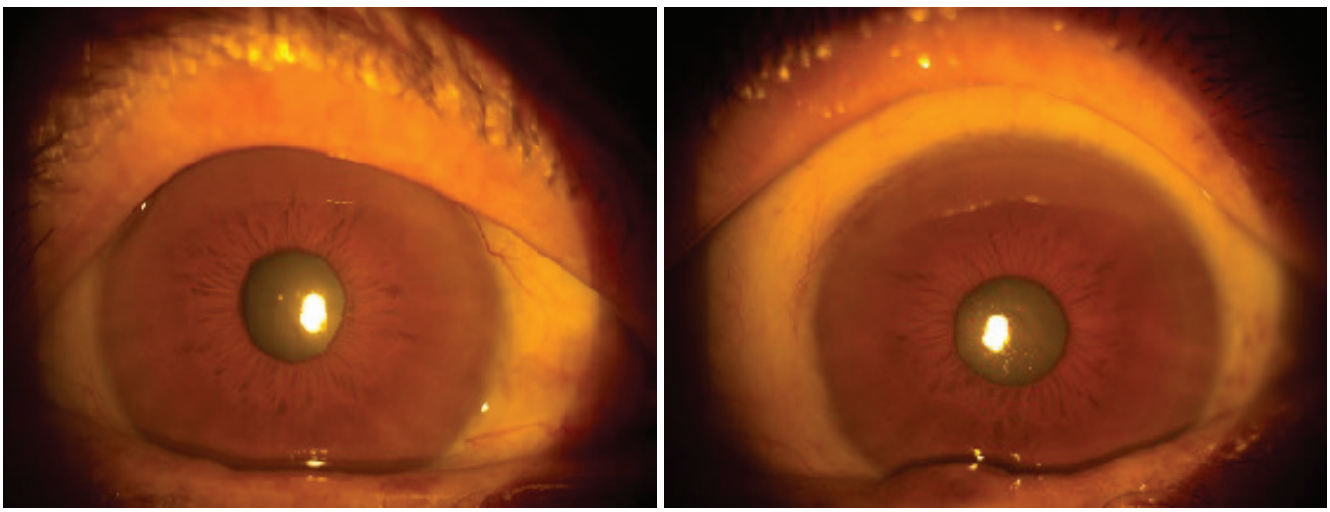

**Fig. 1:** BE showing no circumciliary congestionof conjunctiva, clear cornea, mid-dilated pupil and nuclear sclerosis grade I.

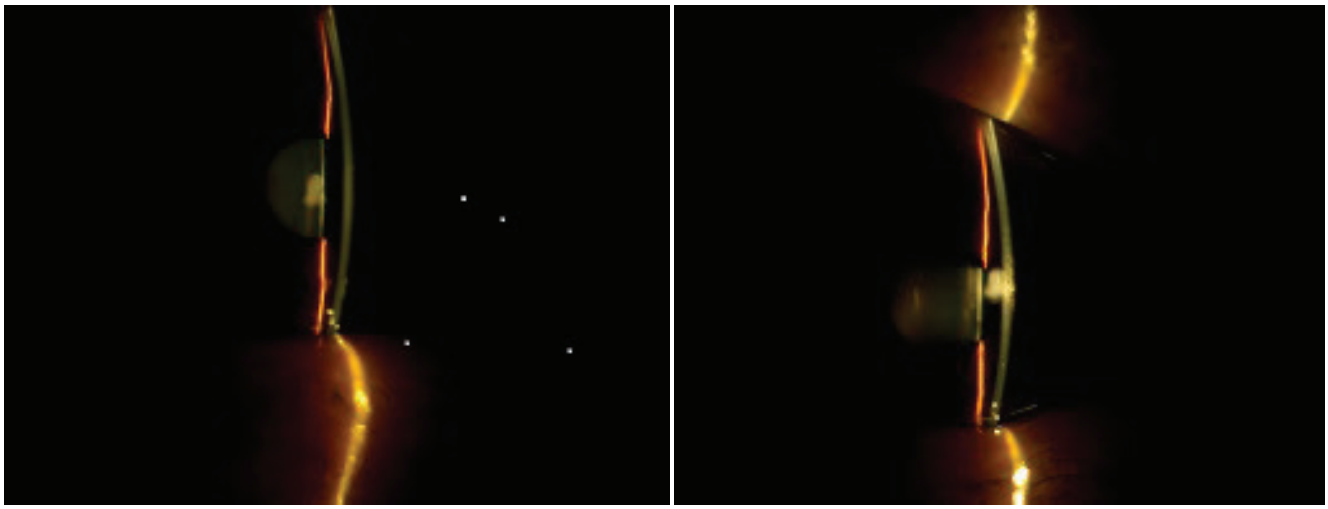

**Fig. 2:** BE showing shallow anterior chamber depth (Van Herick's grade I).

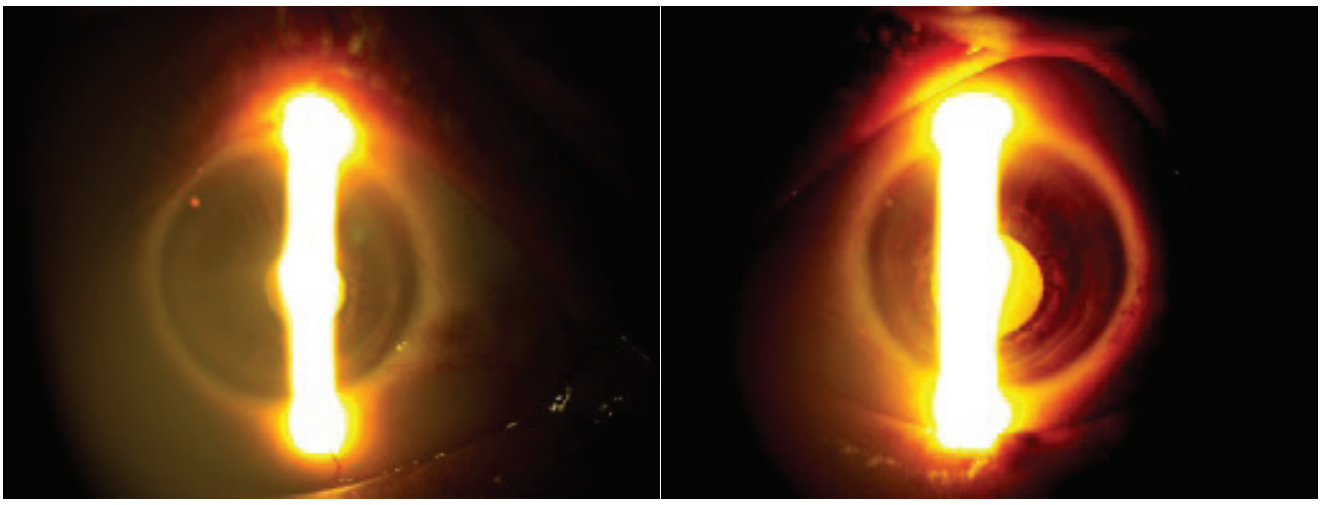

**Fig. 3:** BE showing patent LPI in superior temporal and superior nasal region of RE and LE.

LPI was patent in BE (Fig. 3) and all the eye drops were discontinued. Patient was seen after two months of attack, where her BCVA was 6/6 in both eyes and IOP was stabilized to 12 and 10 mmHg in RE and LE respectively. The Aaxial length was 21.63mm in RE and 21.59mm in LE. Though the axial lengths were short she was not hyperopic, but she had a refractive error of -0.75Dsp in RE and no error in LE. Her central corneal thickness in RE was 512µm and in LE was 510µm respectively.

## DISCUSSIONS

Acute angle closure is an ophthalmic emergency that progresses to blindness if untreated. Most attacks of angle-closure are unilateral. However, 5-10% of the attacks may affect both eyes simultaneously.[1]

Drug induced bilateral ACC is a well known entity reported to be precipitated by drugs like typical antipsychotics, serotonin-specific reuptake inhibitors, tricyclic antidepressants, antihistamines, adrenergic agonists, anticholinergics, sulpha based drugs, anticoagulants etc.[2] In our case there was no history of taking any such systemic drugs but also she had bilateral attack. She was only on antihypertensive and anti diabetic drugs for the last 8 years.

Bilateral acute primary angle closure attack is a rare condition. Lai *et al*[3] reported that in his study 98% of acute angle closure attack was presented with pain but none of the cases was bilateral. But here we present a case of bilateral AAC attack without eye pain.

Idiopathic bilateral simultaneous attack is a very rare entity. Saunders[4] reported a series of 41 patients who presented with acute angle closure and only one had simultaneous bilateral symptoms of unknown cause. In our case also patient had no obvious cause for bilateral involvement.

Acute angle closure is an urgent, dramatic symptomatic event accompanied by blurred vision, painful red eye, headache, nausea, and vomiting. It is diagnosed by high IOP, corneal edema, shallow anterior chamber depth, and a closed angle on gonioscopy.[5] In this case patient had some unusual clinical features like severe vomiting along with sudden diminution of vision bilaterally without a history of headache and painful red eyes; absence of clinical signs like circumciliary congestion of conjunctiva, corneal epithelial edema in spite of high IOP.

Hunter *et al*[6] reported a case of simultaneous bilateral angle closure occurred in a patient with giant cell arteritis. Our patient did not have this condition. A case of bilateral angle closure glaucoma was reported after treatment with inhaled bronchodilator by Drimitry V *et al*.[7]

Face-down positioning poses a risk of AAC in predisposed patients. Several previous case reports identified prolonged prone or face-down positioning as a cause of bilateral AACG. Singer MS[8] *et al* reported a case of bilateral ACC after face down spine surgery. Another case of bilateral ACC was reported by Chong LE as a complication of face-down positioning after macular hole surgery.[9] The prone position induces forward movement of the lens and enhances the effect of relative pupillary block in predisposed patients.[10] In our case there was no history of any ocular or spine surgery.

Acute angle closure can easily masquerade a systemic illness and these patients may present not only with painful eye with reduced vision but also with systemic symptoms and the diagnosis can easily be missed.[11,12] Therefore ophthalmologists and physicians should be aware that ACC can present bilaterally and the clinical features may be unusual.

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