

Do we really need to panic in all acute vision loss in ICU? Acute angle-closure glaucoma

Ali Akal,¹ Ahmet Kucuk,² Funda Yalcin,³ Saban Yalcin⁴

Abstract

Acute angle closure glaucoma is a sight-threatening situation characterized by a sudden and marked rise in intraocular pressure (IOP) due to obstruction of aqueous humour outflow. Many local (ocular drops, nasal and nebulized agents) and systemic drugs (e.g. atropine, adrenaline, ephedrine, some psychoactive and antiepileptic drugs) that are widely used in intensive care units have the potential to precipitate such an acute attack. In this case report, we describe progressive visual loss due to acute angle-closure glaucoma (AACG) in a 59 year old female patient followed in the ICU due to a massive pulmonary embolism.

Keywords: Acute angle closure glaucoma, Intraocular pressure, Intensive care unit, Visual loss.

Introduction

Visual loss in critical care patients is a point of concern that warrants thorough assessment.¹ Acute angle closure glaucoma (AACG) is a possible blinding situation represented by an unexpected and pronounced rise in intraocular pressure (IOP) due to obstruction of aqueous humour outflow. Numerous local (nasal and nebulized agents) and systemic drugs (e.g. atropine, adrenaline, ephedrine, etc.) that are widely used in intensive care units have the capacity to provoke such an acute attack.² The risk is increased in predisposed subjects, including certain ethnic groups, such as Asians, Canadians, and Eskimos; elderly and female gender with a shorter eyeball, larger crystalline lens, shallower anterior chamber, and a narrower drainage angle.³ In this case report, we describe progressive visual loss due to acute angle-closure glaucoma (AACG) in a 59 year old female patient followed in the ICU due to a massive pulmonary embolism.

Case Report

A 59 year old female patient was admitted to our emergency department in May, 2012, with chest pain and

¹Department of Ophthalmology, ^{2,4}Department of Anesthesiology and Reanimation, ³Department of Chest Disease, Harran University Medical Faculty, Sanliurfa, Turkey.

Correspondence: Saban Yalcin. Email: sabanyalcin@yahoo.com

cyanosis. Her heart rate was 127 beats per minute (bpm), blood pressure was 78/46 mm/Hg, and peripheral oxygen saturation was 89% with 10/L O₂ via a nonrebreather mask. Her medical history revealed a urological operation due to renal stones three weeks previously. Bedside echocardiography showed an enlarged right ventricle and electrocardiography displayed a new right bundle branch block that was suspicious for a massive pulmonary embolus. Computerized tomography confirmed the diagnosis of bilateral massive pulmonary emboli (PE) and she was transferred to the ICU. Infusions of 0.01 µg/kg/min epinephrine and with 10 µg/kg/min dopamine were started due to haemodynamic instability. Twenty-four hours after the epinephrine and dopamine infusion, visual loss in the right and left eyes with a bilateral frontal headache began and an ophthalmology consultation was requested. Intraocular pressure was measured as 44 mm Hg (right) and 53 mm Hg (left) with a Schiotz tonometer. Characteristics of AACG, including diffuse epithelial corneal oedema, shallow anterior chambers with iris convexity, and fixed, mid-dilated pupils were observed. The view of the retina was restricted due to corneal oedema. Gonioscopy was not tried in the critical care setting. Right and left eye acute angle closure glaucoma was diagnosed and all medications were rechecked, dopamine and adrenaline infusions and nebulized agents were stopped. Oral acetazolamide (250 mg) four times daily, topical pilocarpine (4%) four times a day, and timolol (0.5%) twice daily were started. Two days after this

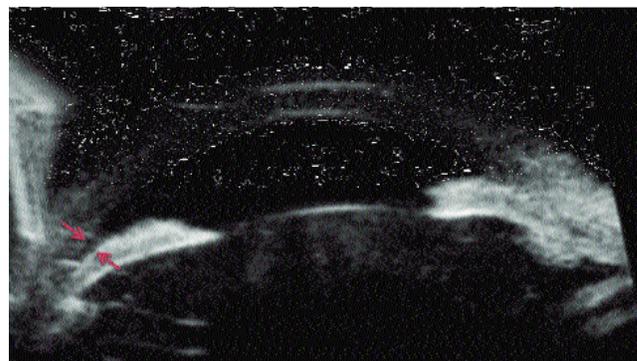


Figure: Left eye UBM image of 59 year-old female patient with bilateral primary angle closure glaucoma. UBM image shows nearly closed iridocorneal angle (arrows).

acute attack, the right and left eye intraocular pressures were in the normal range, and visual acuity had completely recovered. She was discharged home one month after admission and right and left eye laser peripheral iridotomy was performed 3 weeks after ICU discharge (Figure). A one year follow-up revealed no abnormalities.

Discussion

Ophthalmologic complications are often encountered in intensive care unit (ICU) patients, including metastatic endogenous endophthalmitis, ischaemic optic neuropathy, pupil abnormalities, vascular occlusions, rhino-orbital cerebral mucormycosis and acute angle closure glaucoma.⁴ Glaucoma is a potentially blinding disease caused by progressive and irreversible optic neuropathy.⁵ Studies have shown that a single AACG attack has been associated with permanent retinal nerve fiber layer loss, visual field defects, and optic disc pallor.⁶

Several classes of drugs, including topical mydriatics, anticholinergics, antidepressants, anticonvulsants, antihistaminics, sulphamate derivative adrenergics and sympathomimetic drugs have the capacity to actuate an acute attack of angle closure.⁷

Sympathomimetic drugs, including epinephrine, norepinephrine, dopamine, phenylephrine, and ephedrine are widely used in the ICU and are reported to cause AACG in several case reports. Phenylephrine drops that are used for ocular fundus examination might induce AACG in about 0.03% of nonselected patients.^{8,9} Systemic administration of ephedrine for flu and surgical anaesthesia or epinephrine to treat anaphylactic shock and ventricular fibrillation have been shown to cause AACG. Nasal ephedrine and naphazoline are reported to induce bilateral AACG in the acute management of epistaxis,² and β 2-adrenergic agents (salbutamol, albuterol, terbutaline) with anticholinergic drugs might also trigger transient angle closure.¹ Medications with indirect sympathomimetic activity, including amphetamines, some antidepressant agents (imipramine, monoamine oxidase inhibitors), and cocaine have been shown to induce AACG.^{2,10} In our case, epinephrine, dopamine and nebulized agents were used in the ICU due to haemodynamic instability and bronchoconstriction. All these drugs were stopped after the diagnosis and improvement in symptoms were resolved with the AACG treatment within the first 24 hours. Taking into consideration all the predisposed factors, such as female gender, age, and ethnicity, we think that medications might be the main factors that induced AACG in this patient.

Symptoms including ocular or periocular pain, nausea and/or vomiting, and an antecedent history of intermittent vision and/or haloes might be seen, and conjunctival injection, corneal epithelial oedema, mid-dilated unreactive pupil, and shallow anterior chamber might be observed in ophthalmology consultation. IOP might also be elevated.¹¹ However, in ICU settings, the characteristic signs and symptoms of AACG may be incorrectly ascribed to other causes, resulting in improper pharmacologic treatment, which may prolong the acute episode and postpone the exact diagnosis. Any patient presenting with signs or symptoms of AACG should be referred promptly to an ophthalmologist. Rapid diagnosis and appropriate treatment will facilitate prohibiting visual loss. In differential diagnosis, acute orbital compartment syndrome, conjunctivitis, corneal abrasion, laceration, ulceration, ulcerative keratitis endophthalmitis and vitreous haemorrhage should be kept in mind.

A standardized protocol of management involves intravenous acetazolamide (500mg), followed by oral acetazolamide (250mg) four times daily, topical pilocarpine (4%) four times a day, and timolol (0.5%) twice daily, as well as betamethasone or prednisolone acetate every 3 hours applied to the affected eye. If the IOP is not reduced after 2 hours of this standard treatment, other medications, such as intravenous mannitol, oral glycerol, topical latanoprost, or brimonidine can be added. Patients are reviewed at regular intervals for IOP control. Subjects are also examined for adverse effects of the medical therapy.¹¹

After starting conventional treatment of AACG, definitive surgical treatment, including laser peripheral iridotomy or argon laser peripheral iridoplasty should be done immediately, but these techniques must be delayed until haemodynamic stability of ICU patients is achieved. Also, sufficient corneal clarity is crucial for surgery to be performed safely.¹² In patients not suitable for immediate definitive surgery, a delay in aborting an acute attack carries a significant risk of developing glaucomatous optic neuropathy.¹³ We believe that prompt medical treatment is essential in patients who are not appropriate for immediate definitive surgery, especially those in the ICU with haemodynamic instability.

Conclusion

We believe that all individuals responsible for medication of patients in the ICU should be fully aware of the adverse effects of drugs. Nevertheless, all patients should have initial ophthalmic evaluation and ICU staff should be aware of eye problems, particularly in predisposed patients.

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