CASE REPORT

IMPORTANCE OF EARLY DETECTION OF ACUTE PRIMARY ANGLE CLOSURE GLAUCOMA: A CASE REPORT

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ABSTRACT

Background: Acute Angle Closure Glaucoma is an ocular emergency that caused by a rapid increase in intraocular pressure due to outflow obstruction of aqueous humor. This condition can result visual losses that is irreversible so that early detection with the right diagnosis by general practitioner or ophthalmologist is vital to minimize the risk of permanent damage. We report the case of a patient who gained significant improvement after glaucoma surgery immediately after diagnosed with acute angle closure glaucoma in the ER.

Case Presentation; A 53-year old female was presented with a chief complaint of throbbing pain and redness on both eyes (left eye (LE) at first, followed by right eye (RE)) for 3 days. She also had blurred vision, headache, and was unable to sleep due to the pain. Her presenting visual acuity (VA) was 20/80 in the RE and hand movements in the LE, with measured intraocular pressure (IOP) was 65 mmHg in the RE and 63 mmHg in the LE. After diagnosed with acute angle closure glaucoma and senile nuclear cataract on both eyes, a series of peripheral iridotomy laser followed with trabeculectomy was performed. Four weeks after the surgery, her VA is improved to 6/6 with corrected refraction and IOP is successfully lowered to 10mmHg on both eyes. Slit-lamp examination also showed an improvement of anterior chamber depth now that it is deep.

Conclusion: Although degeneration of optic nerve due to glaucoma is deemed irreversible, our patient showed a significant improvement in visual function following surgery. Early detection and early intervention with surgery might play an important role in acute closure angle glaucoma to prevent further optic nerves damage and deterioration of visual function.

Keywords: Acute Glaucoma, Angle Closure Glaucoma, Trabeculectomy

BACKGROUND

Glaucoma are a group of diseases characterized by progressive degeneration of optic nerve, with loss of retinal ganglion cells and progressive excavation of the optic disc, resulting loss of visual field.[1,2] Glaucoma is often defined by elevated intraocular pressure (IOP) that is later classified as open-angle (POAG) or closed-angle (PACG) and as primary or secondary. This condition is leading to optic neuropathy and vision loss if left untreated. [3,4]

Acute angle-closure glaucoma is an ocular emergency that caused by a rapid increase in intraocular pressure due to outflow obstruction of aqueous humor. Several factors lead to the obstruction in acute angle closure glaucoma, but the major predisposing factor is the structural anatomy of the anterior chamber, leading to a shallower angle between the iris and the cornea. Acute angle-closure glaucoma presents as a sudden onset of severe unilateral eye pain or a headache associated with blurred vision, rainbow-colored halos around bright lights, nausea, and vomiting. The physical exam will reveal a fixed midpoint pupil and a hazy or cloudy cornea with marked conjunctival injection.[5]

Glaucoma is one of the highest causes of irreversible blindness all over the world. The global prevalence of primary open angle glaucoma is 2.2%, with an estimated 57.5 million people are affected worldwide, while the prevalence of primary angle closure glaucoma is only 0.17% affecting individuals younger than 40 years, particularly East Asians.[5] However, primary angle closure glaucoma usually has more severe clinical manifestation with acute onset, and considered as an ocular emergency.It has been projected that approximately 76 million people suffer from POAG and PACG by 2020, and will increase to 111,8 million by 2040. While the prevalence of POAG is highest in Africa (4.20%; 95%. Crl, 2.08-7.35), PACG prevalence is highest in Asia (1.09%; 95% Crl, 0.43-2.32). From all people with PACG, 86% are in Asia, with approximately 48% in China, 23,9% in India, and 14,1% in Southeast Asia.[6,7]

The medical treatment for acute angle-closure glaucoma aims to decrease the intraocular pressure by blocking the production of aqueous humor, increasing the outflow of aqueous humor, and reducing the volume of the aqueous humor.[8,9,10] Definitive treatment is peripheral iridectomy after the acute episode subsides. Laser iridectomy is the treatment of choice. Surgical iridectomy is indicated when laser iridectomy cannot be accomplished. Iridectomy relieves the pupillary block as the pressure between the posterior and anterior chamber approaches zero by allowing the flow of aqueous humor through a different route. Iridectomy should be as peripheral as possible and covered by the eyelid to avoid monocular diplopia through this second hole in the pupil.[11]

However, there were several case reports which showed an improvement of visual function following surgical therapy.[12,13] Previous study also reported that optic nerve head morphology might undergo reversible changes due to the reduction of intraocular pressure.[14] Therefore, functional and structural improvement may be possible in some glaucomatous patients, although the underlying mechanisms still not fully understood.

As mentioned that the damage is irreversible, it is really important to detect glaucoma at an early stage so that advanced risk of visual impairment can be minimized.[15] While management of PAC depends on the type of clinical presentation, making the right diagnosis of PACS, PAC, or PACG is just as important.[16] The aim of this article is to report the case of a patient diagnosed with PACG at initial presentation that showed marked improvement in visual acuity following laser peripheral iridotomy and trabeculectomy.

CASE PRESENTATION

A 53-year old female was presented to our emergency room with a chief complaint of redness on both eyes for 3 days before admission. Redness began in the left eye and then followed by the right eye. Additional complaints were throbbing pain in both eyes, blurred vision, epiphora, tenderness around the eyes, headache, and was unable to sleep due to the pain. Another complaint like fever, nausea, or vomiting were not found.

History of past glaucoma, hypertension, and diabetic mellitus were denied. She had no history of previous ocular surgery or trauma and was not using any systemic medications, including corticosteroids. There was not any positive family history of glaucoma.

On ophthalmic examination, her presenting visual acuity was 6/24 in the right eye (RE) and hand movements in the left eye (LE). There was injection of both conjunctivae, corneal staining in the RE, while the cornea in the LE was clear. Slit-lamp examination showed corneal edema, shallow anterior chamber, and blurred lens on both eyes. Both pupils were mid-dilated and both irises was unresponsive. Intraocular pressure (IOP) was 65 mmHg in the RE and 63 mmHg in the LE measured by Non-Contact Tonometer. The optic nerve was difficult to visualize because of corneal edema and visual field testing was not done because of discomfort (Fig. 1). Vital sign and laboratory examination showed a normal result. A diagnosis of acute primary closure angle glaucoma was made based on the history and clinical presentation, and the patient was commenced on oral Acetazolamide (Glaucon) and Calcium Aspartate (Aspar K), and topical Brimonidine Tartrate (Alphagan), Brinzolamide 1% (Azopt), Timolol 0.5%, and Prednisolone Acetate (P-red) on both eyes.

Figure 1. Ophthalmic Examination on Right (a) and Left (b) Eye

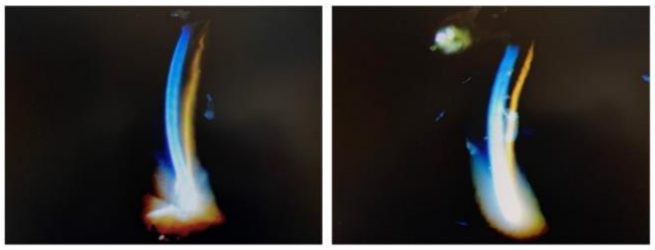

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Figure 2. Slit Lamp Examination on Right (a) and Left (b) Eye

Management of this patient with primary angle closure glaucoma was quite challenging. This patient was immediately treated with Laser Peripheral Iridectomy on the right eye followed with trabeculectomy 1 week later. Trabeculectomy was performed in both eyes on different time (left eye 2 weeks after right eye) using block anesthesia. Exposure of globe was performed using corneal-traction-suture, fornix-based technique was done, position of scleral flap and paracentesis using superonasal with punch sclerostomy. Iridectomy was performed, 3-4 stiches were used for conjunctival closure. Mitomycin C (MMC) was also given as antifibrotic agent to prevent excessive post-operative scarring. Examination evaluation revealed clearer vision with minimal pain in the right and left eye. Second laser surgery was then performed on the left eye 3 weeks after trabeculectomy of the left eye. There were no complications noted and at day one post-operative review.

Over the next week, IOP was gradually lowered through loosening of the releasable sutures and showed postoperative review was 16 mmHg on the right eye and 10 mmHg on the left eye. Surprisingly, the patient reported a gradual improvement and clearer in vision in both eyes since surgery. The reduction in IOP and improvement in visual acuity has subsequently been maintained. During this period, the patient also reported a subjective amelioration of vision.

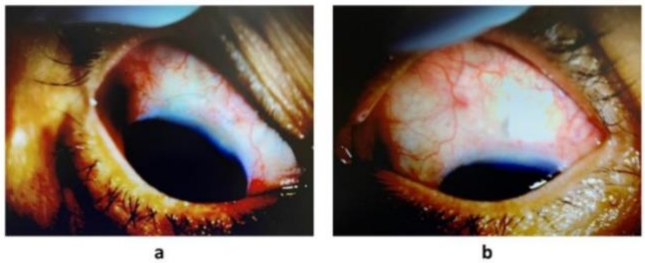

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Figure 3. Slit Lamp Examination on Right (a) and Left (b) Eye After Surgery

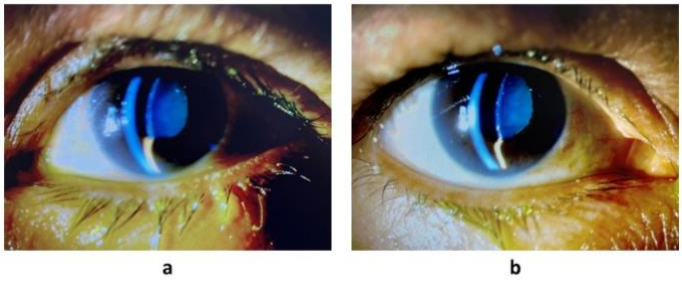

Figure 4. Slit Lamp Examination on Right (a) and Left (b) Eye After Surgery

DISCUSSION

Acute angle closure glaucoma is an emergency ophthalmic condition that occurs due to a sudden closure of the angle in the anterior chamber of the eye, causing an abrupt intraocular pressure (IOP) increase. Several factors lead to the obstruction in acute angle-closure glaucoma, but the major predisposing factor is the structural anatomy of the anterior chamber, leading to a shallower angle between the iris and the cornea.[17,18] Several risk factors have been implicated to be related in the presence of acute angle-closure glaucoma, classified into strong and weak risk factors.[19]

Strong risk factors including female gender, hyperopia, shallow eye having angle closure, also Inuit and Asian ethnicity. Related to gender, there is a 4 to 1 ratio of the incidence of angle-closure glaucoma in women versus men. The anterior chamber depth and volume are smaller in hyperopic eyes. Having smaller anterior segment dimensions is the main ocular risk factor for closure of the angle, with anterior chamber depth having the strongest correlation with angle closure and angle closure glaucoma. Anatomic factors of both eyes are virtually always similar. An untreated fellow eye has a 40% to 80% chance of developing an acute episode of angle closure over the next 5 to 10 years. In relation to ethnicity, highest rates of angle closure glaucoma are reported in Inuit and Asian populations. Angle-closure glaucoma is more common in Southeast Asians, Chinese, and Eskimos. It is uncommon in black populations. In whites, acute angle-closure glaucoma accounts for 6% of all glaucoma diagnoses.[20]

Weak risk factors of angle closure glaucoma are advanced age, family history, and the use of medications that induce angle narrowing. Acute ACG is most common between the ages of 55 and 65 years. The size of the lens increases progressively with age, thus crowding the region of the anterior chamber angle, making it shallower. Family history has been suggested as a risk factor because ocular anatomic features are inherited, but is not universally recognized. Several medications which could induce angle narrowing, such as anticholinergic topical pupil dilators (example: cyclopentolate or atropine) or systemic medication (example: sulfonamides,

topiramate, phenothiazines).[5,19]

Because acute angle-closure glaucoma is an ocular emergency; rapid diagnosis, immediate intervention, and referral are crucial and have profound effects on patient outcome and morbidity. The key diagnostic factors with common frequency include the presence of risk factors, elevated intraocular pressure, and several signs and symptoms which is present in the acute and subacute forms but not with the chronic form of angle closure. In this case, our patient had several risk factors which support the higher probability to be diagnosed as acute angle-closure glaucoma. Our patient was an Asian female, aged 53-year-old. Even the patient tends to have younger age than the common onset of angle-closure glaucoma attack, it is still close enough to the average age of the population which had higher risk for angle-closure glaucoma. Nevertheless, there were no history of acute angle-closure glaucoma in the family and the patient also do not consume any medication inducing angle narrowing.

An acute attack of angle-closure glaucoma is precipitated by pupillary dilatation, leading to increasing iris and lens contact increasing the pupillary block.[5] The increasing pupillary block leads to bulging of the iris, acutely closing the angle between the iris and cornea, thus obstructing the aqueous humor outflow tract. The intraocular pressure rises acutely, leading to symptomology. Acute angle-closure glaucoma presents as a sudden onset of severe unilateral aching eye or eyebrow pain; deep, dull, periocular headache associated with blurred vision; rainbow-colored halos around bright lights; nausea and vomiting. The physical exam will reveal a fixed midpoint pupil and a hazy or cloudy cornea with marked conjunctival injection (most prominent at the limbus). Patient also revealed the reduced acuity. One of the key diagnostic factors is the elevated intraocular pressure. In healthy eyes, intraocular pressure is generally 10 to 21 mmHg. Intraocular pressure will be elevated and can be as high as 60 to 80 mmHg in an acute attack. A mild amount of aqueous flare and cells may be seen. The optic nerve may also be swollen during an acute attack.[21,22]

Our patient in this case presented with complaints that were found to be in accordance with the clinical manifestation of acute angle-closure glaucoma. Patient complained redness in the left and right eye in the last 3 days before admission. Redness began in left eye and then to the right eye. She also complained epiphora, blurred vision, intense pain in both eyes, tenderness around the eyes, dizziness and caused sleep disturbance. Rapid and progressive headache, eye soreness, reduction in vision, and eye hyperemia are the symptoms that occur in all cases.[10] Moreover, the ophthalmic examination also supported the diagnosis of acute angle-closure glaucoma. Ophthalmic examination revealed the decrease of bilateral visual acuity, bilateral conjunctival hyperemia and corneal edema, symmetrical fixed mid-dilated pupil, and

shallow anterior chamber inflammation. There were also elevated intraocular pressure above 60 mmHg on both eyes. The optic nerve is difficult to visualize due to corneal edema.

The diagnostic criteria of acute angle-closure attacks including the presence of at least 2 of the following symptoms: (1) ocular or periocular pain; (2) nausea or vomiting; and (3) antecedent history of intermittent blurring of vision with haloes, plus presenting IOP > 21 mmHg, and the presence of at least 3 of the following signs: (1) conjunctival injection; (2) corneal epithelial edema; (3) mid-dilated unreactive pupil, and (4) shallow anterior chamber.[10,21] In this case, our patient had 2 symptoms (intense ocular pain and blurring of vision with halos); plus, elevated intraocular pressure (>60 mmHg); plus, 4 signs (conjunctival injection, corneal edema, mid-dilated pupil, and shallow anterior chamber). Thus, the diagnosis of acute angle-closure attacks is confirmed.

The immediate goal of treatment is to relieve the acute symptoms and decrease intraocular pressure, which is usually achieved with medical therapy. Oral or topical carbonic anhydrase inhibitors, topical beta-blockers, and topical alpha-2 adrenergic agonists lower IOP through suppression of aqueous humor production. Beta-blockers reduce intraocular pressure by around 20% to 30% within 1 hour of instillation. Alpha-agonists reduce intraocular pressure by around 26% within 2 hours post-dose. Carbonic anhydrase inhibitors, topical beta-blockers, or alpha-2 adrenergic agents may be used as first-line therapies either alone or more typically in combination.[8]

In patients where angle closure is thought to be secondary to pupillary block or plateau iris syndrome, cholinergic agents (such as pilocarpine). Evidence should be started after IOP decreases to < 40 mm Hg. Cholinergic agents can paradoxically result in shallowing of the anterior chamber and narrowing of the angle in eyes with angle closure secondary to lens-induced mechanism or aqueous misdirection (malignant glaucoma). They are therefore contraindicated in these cases. If these medical treatments are unsuccessful, hyperosmotic agents should be used. Hyperosmotic agents are also used initially when pressures are exceedingly high. Following resolution of the acute attack, definitive surgical treatment should be performed within 24 to 48 hours with the aim of achieving a persistently open angle.[8,10]

Definitive surgical management performed after resolution of acute attack is aimed at achieving a persistently open angle. Definitive treatment is peripheral iridectomy after the acute episode subsides. Laser iridectomy is the treatment of choice. Surgical iridectomy is indicated when laser iridectomy could not be accomplished. Iridectomy relieves the pupillary block as the pressure between the posterior and anterior chamber approaches zero by allowing the flow of aqueous humor through a different route. Iridectomy should be as peripheral as possible and

covered by the eyelid to avoid monocular diplopia through this second hole in the pupil.[11]

The initial medical therapy received by our patient was oral Acetazolamide (Glaucon) and Calcium Aspartate (Aspar K), and topical Brimonidine Tartrate (Alphagan), Brinzolamide 1% (Azopt), Timolol 0.5%, and Prednisolone Acetate (P-red) on both eyes. After achieving resolution of acute attack, the patient was immediately treated with Laser Peripheral Iridectomy on the right eye followed with trabeculectomy 1 week later. Trabeculectomy with MMC was performed in both eyes on different time (left eye 2 weeks after right eye) followed by second Laser Peripheral Iridectomy on the left eye 3 weeks after trabeculectomy of the left eye. Examination evaluation revealed clearer vision with minimal pain in the both eyes. There were no complications reported on our patient. The intraocular pressure after surgery on right and left eyes are 16 mm Hg and 10 mm Hg, respectively. The patient also reported a gradual improvement and clearer in vision in both eye since surgery. The prognosis depends on early detection and prompt treatment of acute closed-angle glaucoma.[5] Thus, early detection and prompt treatment are very crucial in the management of patient with acute closed-angle glaucoma.

CONCLUSION

In conclusion, although degeneration of optic nerve due to glaucoma is deemed irreversible, our patient showed a significant improvement in visual function following surgery. Early detection and early intervention with surgery might play an important role in acute closure angle glaucoma to prevent further optic nerves damage and deterioration of visual function. The improvement of visual function may occur in early detected acute angle closure glaucoma patient following surgical therapy. Trabeculectomy with or without laser surgery should be considered as first line treatment in patient with advanced disease.

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