

# Secondary Acute Angle-Closure Attack: Diagnosis and Management

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## ABSTRACT

Patients may come with acute angle-closure attack that mimics primary disease, but actually there was a hidden cause. Mostly, fellow eye with wide open angle might be a clue for secondary cause. However, an area with high prevalence of narrow angle makes it harder to notice while fellow eye has narrow angle too. The most important thing in order to approach acute angle-closure attack is to know a possible underlying cause that should be looked for. This article will simplify various pathologies that could result in secondary acute angle-closure attack. Clinical presentation, special investigation and treatment were all summarized.

**Keywords:** Secondary acute angle-closure attack; secondary acute attack; diagnosis; management (Siriraj Med J 2018;70: 91-94)

## INTRODUCTION

The definition of acute angle-closure attack is at least 2 of the following symptoms: ocular pain, nausea or vomiting, and a history of intermittent blurring of vision with halos; and at least 3 of the following signs: IOP greater than 21 mm Hg, conjunctival injection, corneal epithelial edema, shallower chamber in the presence of occlusion and mid-dilated nonreactive pupil. If patients with acute angle closure have glaucomatous optic neuropathy, it is defined as acute angle closure glaucoma. Acute angle closure can be classified into 2 categories due to presenting of identifiable causes. A term 'Primary' represents acute angle closure with no identifiable cause and 'Secondary' belongs to another category that had definite causes. To the best of my knowledge, there is no review article for secondary angle closure in an acute setting. This article will be the first to review diagnosis and management of secondary acute angle closure.

### Primary vs. secondary

When patients come with acute angle closure, is there any clue to tell us that this is secondary? The

answer is yes because there are several histories and physical examinations that could lead you to identify possible causes. First, if the fellow eye has wide open angle that acute angle-closure attack could be secondary. Second, bilateral involvement is an alert of underlying systemic causes. There are many conditions that could result acute angle-closure attack and they have their own characteristics. Therefore, it would be easy to approach patients according to the possible pathologies.

## Pathologies

### Spontaneous suprachoroidal hemorrhage

Suprachoroidal hemorrhage can happen either as a complication of intraocular surgery or spontaneously. Spontaneous cases can present with acute angle-closure attack in outpatient setting and it should be differentiated from primary acute angle-closure attack. Although, the condition is rare, it could end up with devastating result.

Any pathology that could cause massive suprachoroidal hemorrhage, also results in abrupt lens-iris diaphragm movement forward due to annular hemorrhagic ring.<sup>4</sup> Suspicious predisposing factors of

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spontaneous suprachoroidal hemorrhage are ocular pathologies (such as age related macular degeneration (AMD), polypoidal choroidal vasculopathy (PCV)<sup>1-3</sup>, arteriovenous malformation), anticoagulants usage, blood dyscrasia<sup>22</sup>, or systemic hypertension.<sup>3</sup> Patients should be asked for history of those factors. Patients usually present with very poor visual acuity compared to primary acute angle-closure attack. As the result from review of literature on PCV/AMD/other related cases complicated by secondary ACG, there were 9 patients (8 patients had no PL visual acuity, 1 patient with hand motion).<sup>2</sup> Mechanism of angle closure can be complicated as it might not result from one mechanism. Pupillary and non-pupillary mechanism could play their role; thus, iris bombe or uniformly shallowed anterior chamber could be found. Ultrasound might benefit in suspicious secondary acute angle-closure attack cases since it is not invasive and easily accessible. Management of such cases are quite difficult and no consensus due to nature of the conditions which are severe and rare. Aim of treatment may be to reduce ocular pain rather than good visual acuity. Anti-glaucoma medications, laser peripheral iridotomy, laser iridoplasty, sclerotomy drainage, or diode laser cyclodestructive procedure have been used, but the results were poor. Pesin SR et al., documented in the review of 9 patients with acute angle-closure attack which resulted from spontaneous suprachoroidal hemorrhage that, most patients end up with phthisis bulbi<sup>2</sup> and one case was enucleated as an attempt to control pain.<sup>4</sup>

### Ciliochoroidal effusion

Any pathology that can cause ciliochoroidal effusion can result in anterior ciliary body rotation and lens-iris diaphragm movement forward. Ciliochoroidal effusion can be caused by medications, ocular inflammation, venous congestion, systemic diseases and unknown etiologies. Uniformly shallow anterior chamber without iris bombe is an important sign. Treatment of an underlying cause is important. Completely different from primary angle closure, It should be highlighted that laser peripheral iridotomy does not work and topical miotics should be avoided due to mechanism of angle closure that is caused from anterior rotation of ciliary body not pupillary block.

### Medication-induced ciliochoroidal effusion

Topiramate, Zonisamide, Chlorthalidone, Acetazolamide, Hydrochlorothiazide, Methazolamide, and Indapamide are sulfonamide derivative drugs that are used to treat various conditions such as seizure,

migraine, systemic hypertension, microbial infection, reduce intraocular pressure and they have been reported to cause acute angle closure glaucoma.<sup>6-9</sup> The underlying pathogenesis is unknown, with increased lens thickness due to changing in osmotic state of crystalline lens and forward crystalline lens displacement as the earlier theory that tried to explain underlying pathogenesis.<sup>17</sup> After that, there were evidences from several case reports that acute angle closure could be induced by sulfonamide derivatives which could happen in pseudophakic eye 6 and Craig et al.,<sup>18</sup> showed that lens thickness only accounts for 9 to 16% of anterior chamber shallowing. Therefore, they support idiosyncratic inflammation of uveal tissue that causes ciliary body swelling and anterior rotation. The nature of idiosyncratic reactions is unpredictable and does not depend on dosage, so it is hard to study a definite biochemical mechanism. Inflammation of uveal tissue, especially in anterior part, and ciliary processes, result in anterior rotation of ciliary body and move the lens-iris diaphragm forward. Moreover, it causes zonular relaxation which results in crystalline lens thickening, thus a patient may suffer from myopic shift. Clinically, most of the cases are bilateral 20 and the risk of developing angle closure glaucoma is within the first month after drug administration.<sup>19</sup> Acute bilateral blurred vision from acute myopic shift might be the first clinical complaint. The patient may less frequently present with angle closure symptoms. IOP measurement, gonioscopy along with a temporal relationship between initiating or increasing the dose of a sulfonamide derivative and onset of symptoms will help in making diagnosis. B-scan ultrasound can document ciliochoroidal effusion. Subtle ciliochoroidal effusion could be detected by ultrasound biomicroscope. Discontinuing of an offending medication is the mainstay treatment of such cases. Topical anti-glaucoma medications are used for controlling intraocular pressure. Prostaglandin derivatives should be avoided because ciliochoroidal effusion may be mediated by prostaglandin pathway. Topical miotics are contraindicated since they can cause ciliary spasm and worsen symptoms.<sup>20</sup> On the other hand topical cycloplegics can be used to combat myopic shift.<sup>21</sup> Topical steroid is recommended in cases with anterior chamber inflammation. In severe cases, systemic steroid and intravenous mannitol are worth trying. The symptom usually improves within hours to days. Since a mechanism is not a pupillary block, laser iridotomy could not help in relieving angle closure. Laser iridoplasty, choroidal drainage and trabeculectomy should be reserved for medical recalcitrant cases.<sup>8</sup>

### Myelofibrosis, leukemia, lymphoma

Albert et al., reported bilateral acute angle closure glaucoma complicating primary myelofibrosis. Moreover, there are documented cases of leukemia and lymphoma presenting with acute angle closure.<sup>23-25</sup> The mechanism is uveal effusion that may result from cellular choroidal infiltration or paraneoplastic syndrome.<sup>23</sup> It can be unilateral or bilateral involvement, but one should be alerted for systemic conditions when dealing with bilateral acute angle closure. Treatment of the underlying causes with immunomodulation or chemotherapy along with controlling intraocular pressure are recommended.

### Posterior scleritis

Posterior scleritis can cause annular ciliochoroidal effusion which leads to acute angle closure.<sup>26-29</sup> Treatment of posterior scleritis with systemic steroid should be considered. For controlling intraocular pressure, management should follow that of acute angle closure from ciliochoroidal effusion.

### Others systemic diseases

Systemic lupus erythematosus choroidopathy<sup>30</sup>, acquired immune deficiency syndrome<sup>32-39</sup>, uveal effusion syndrome<sup>40-41</sup>, Vogt-Koyanagi-Harada syndrome (Harada disease)<sup>42-44</sup>, septic condition of Korean hemorrhagic fever<sup>45</sup>, and campylobacter jejuni infection<sup>31</sup> have been reported to be associated with ciliochoroidal effusion.

### Lens-induced

Subluxated crystalline lens can occur in patients who have the following risk factors which can cause zonular weakness such as trauma, pseudoexfoliation syndrome, connective tissue disease<sup>49</sup>, retinitis pigmentosa, or post vitreoretinal surgery.<sup>47</sup> For intraocular pressure regulation, cataract surgery with intraocular lens implantation is an effective procedure.<sup>48</sup>

### Aqueous misdirection syndrome

This form of glaucoma can occur post-surgically or spontaneously. Clinical features are raised intraocular pressure, uniformly shallow anterior chamber and patent peripheral iridotomy. The pathophysiology is unknown. Patients with primary angle-closure glaucoma who underwent surgery especially glaucoma drainage surgery were recommended to follow up closely during the early post-operative period. There are medications, laser and surgical management, so these should be considered according to lens status and severity.<sup>46</sup>

### Miscellaneous

There are several rare conditions that have been documented in case reports that can result in acute angle closure. Seongyong Jeong et al., reported a case with acute angle closure after intravitreal bevacizumab injection<sup>50</sup>; thus, there were several possible causes. First, pupil dilation before procedure may precipitate acute attack. Second, retinal vein occlusion might be associated with narrow angle after intravitreal injection had increase pressure from posterior of the eye, so it could result in angle closure. Le Du B et al., reported acute angle closure attack secondary to BrightOcular® cosmetic iris implant.<sup>51</sup> Snake bite has been documented as a cause of acute angle closure, and although it is rare, it should be kept in mind because it needs timely recognition and management. Proposed mechanism is capillary leakage syndrome, which results in ciliochoroidal effusion.<sup>52</sup>

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