

## IMPORTANCE OF DEMOGRAPHIC RISK FACTORS FOR PRIMARY ANGLE CLOSURE

Neacsu Alina Mihaela  
County Emergency Hospital, Braila, Romania

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**Correspondence to:** Alina Mihaela Neacsu, MD  
County Emergency Hospital, Braila, Romania  
2 Buzaului Road, Braila  
Phone: 0239 692 222, E-mail: alinamihaela.neacsu@yahoo.com

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### Abstract

According to the Guidelines of the European Glaucoma Society (fourth edition), the family history in the closing angle is an important factor that makes the family screening vital in these families.

It is present in the clinical case in which two twin patients in different circumstances show the same symptoms of angle closure.

**Keywords:** family history, angle closure glaucoma

Open-angle glaucoma (OAG) and angle-closure glaucoma (ACG) are the second leading cause of blindness worldwide. ACG affects 16 million people, and almost 4 million are bilaterally blind. Although three times more people have OAG than ACG worldwide, the greater morbidity of ACG means that the absolute number blind is similar to that of OAG.

Angle-closure and angle-closure glaucoma result from disturbed physiological mechanisms and anatomical measures with a genetic influence but the future testing can exploit knowledge of these factors [8,9].

At this moment, we recognize the following as Demographic risks factors for Primary Angle Closure (PAC):

- Older Age
- Female
- Asian and Eskimoan Race
- Family History – vital/ first degree

relatives may have a 1 in 4 risk of PAC disease but a robust evidence for significant increased risk does not exist yet [2].

Family History of Angle Closure Disease risk was especially high in mothers and siblings. In patients with FHG, knowledge of genetic disposition of the glaucomas, may have led to an earlier diagnosis [5].

The prevalence of narrow angles was 2.2% and twice as high in women. In two subjects (0.03%), an attack of AACG developed in one eye after diagnostic mydriasis [4,5].

Angle-closure and ACG are more common in eyes with shorter axial length (AL), shallower anterior chamber (AC), and a relatively larger lens. Eye size is in part genetically determined-twin studies support a genetic influence of the disease. Many genes were found to be associated with ACG, but no specific gene has yet been identified [9].

Anatomic risk factors for ACG do not adequately explain why many people with small eyes and narrow angles never develop the disease.

Static measurements ignore the internal structures of the eye change from moment to

moment but physiological risk factors are at least as important as the anatomic ones [8,9].

Dynamic Features of the Eye in combination with anatomical structures (small eyes –genetic factors), which contribute to Angle-Closure are the following: high resistance in the iris-lens channel, iris volume retention on pupil dilation, high choroidal expansion - small eyes with thick sclera, plateau configuration of the iris, dilatator insertion (anterior positioned process ciliary) [8,9].

## Case report

A 34-year-old woman presented in the Ophthalmology Emergency Room for: decreased vision Both Eyes (BE), blurred vision BE, redness BE, pain BE, headache, nausea/ vomiting.

The history of the present illness begun the last day the patient was examined from an ophthalmological point of view and she was dilated with Tropicamide 1% for fundus examination. The patient did not have a medical history and, in the ocular history, we did not find any ocular disease. However, the patient underwent many ophthalmological consultations and every time she was dilated, she presented severe headaches. Also, in dark, she had ocular pains.

Regarding the personal history, the patient had Hyperopia Right Eye (RE) + 1,75/ Left Eye (LE) + 3,75.

### Clinical examination

Visual Acuity (VA)	Intraocular Pressure (IOP)
RE 10/100	50 mm Hg
LE 10/100	68 mm Hg

### Biomicroscopy

RE/LE conjunctival hyperemia with ciliary injection, epithelial corneal oedema, pupil oval, asymmetric, poorly reactive, mild-dilated (LE >RE), central anterior chamber depth – relatively normal, peripheral AC depth grade 0 Van Herick.

### Gonioscopy

RE I evaluation

- iridotrabecular contact for 360°
- no visible angle structures
- the iris contour is flat – mild convex

After indentation the iris contour slightly changes.

LE I evaluation was not possible - high corneal oedema

II day in the morning - the iris contour is flat – mild convex

A thicker iris with anterior insertion - ciliary band not seen

After indentation the iris contour slightly changes

### Fundoscopy Examination

BE – normal color and shape of papilla, vertical report, normal neuroretinal rim, without alpha and beta para papillary atrophy, macula with red reflex, normal arteries and veins.

In this case, Diagnosis is RE/ LE Acute Angle Closure (AAC) with plateau iris configuration, Hyperopia.

### Differential Diagnosis

#### 1. AAC mechanism of angle closure

- Pupillary block (the iris is very convex, forward-ballooned iris (iris bombe), in contact with the anterior wall of angle; after indentation, the angle becomes wider; the mild iris is convex UBM [1,2].

- Anomaly on the level of the lens and posterior of the lens.

#### 2. Secondary angle closure

Neovascular glaucoma, Inflammation resulting in both PAS and posterior synechiae, which can result in a secluded pupil with iris bombé, Iridocorneal endothelial syndrome, Uveal effusion associated with systemic medications (e.g. topiramate, sulfonamides, phenothiazines, anticholinergics), Suprachoroidal effusions, Malignant glaucoma, Iris or ciliary body mass lesions or cysts, Other secondary causes of pupil block (e.g. aphakia without an iridectomy, phakic intraocular lens [IOL], anterior chamber IOL, silicone oil), History of blunt or penetrating trauma, Axenfeld-Rieger syndrome, Epithelial downgrowth [1,2].

### PLAN for Treatment

#### 1. Medical treatment:

- IOP lowering drops,
- systemic hyperosmotics medications

#### 2. Surgical treatment RE/ LE

Laser Iridotomy

#### 3. Clinical assessment

- Argon Laser Peripheral Iridoplasty
- Surgical Iridectomy

On the first day, the patient received the following:

1. Medical treatment

- IOP lowering drops systemic

\*Pilocarpine 2% 1 drop for three times in first hour, afterwards 1 drop/ hour

\*Brimonidine 0,2% 1 drop twice per day

- Hyperosmotics medications

\*Mannitol 20% 250 ml x2/ day

\*Acetazolamide 250 mg x2/ day

- Topical Nonsteroid drug

\*Pranoprofen 0,1% 1 drop x4/ day

On the second day, a surgical treatment was performed. Neodymium YAG Laser Iridotomy was performed. The pretreatment measures were the following:

\* pilocarpine 2%, brimonidine 0,2%, topical anesthesia oxibuprocaine 4%

The Laser Settings were the following:

- Pulses per burst: 1-3
- Power: 2 mj
- Spot Size: 50 µm spot size
- superior quadrant

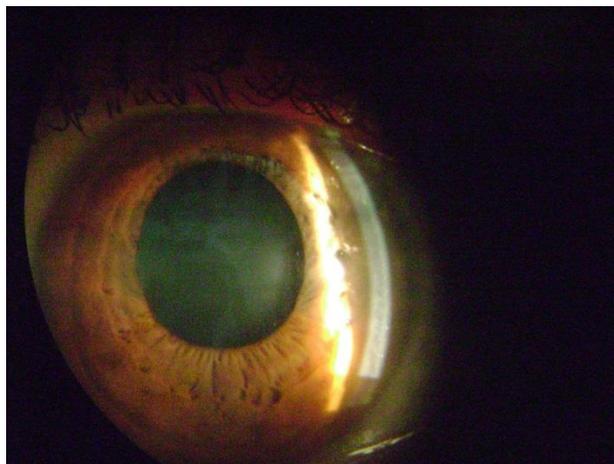


Fig. 1 LE after Iridotomy

The next day the patient had the following results: VA RE 100/ 100, IOP 12 mm Hg, LE 100/ 100(ps), mild mydriasis, iris atrophy temporal quadrant, IOP 14 mm Hg.

2 days later, RE was IOP 14 mm Hg and LE IOP 15 mm Hg.

At the next evaluation, IOP was normal RE and LE.

But, what was very interesting in this case was that the patient had a twin sister who

had the same problems: pain and headache in dark. The twin sister came to consultation and the clinical examination was the following:

VA RE 100/ 100, IOP 12mm Hg, CCT 546 µm

LE 100/ 100, IOP 13 mm Hg, CCT 551 µm

**Table 1.** Biomicroscopy Anterior Chamber (AC) with normal central depth, peripheral AC Grade 2 Von Herick

Biometry	RE/ AAC	LE/ AAC	RE	LE
Axial diameter	21,9	21,69↓	22,21	22,46
AC	2,64	2,43↓	2,63	2,73
Lens	4,25	4,25	4,17	3,99
Vitreous	15,01	15,01	15,41	15,75
CCT	546	551	552	558
C/ D v	0,3	0,3	0,3	0,3

The diagnosis for the Twin Sister was the following:

BE Primary Angle-Closure Suspect (Occludable Angle)

In this case, the Therapeutic Plan was the following:

1. Laser YAG peripheral Iridotomy BE.
2. Clinical Assessment Visual Acuity, Visual Field, Intraocular pressure and Optic Nerve.

After the laser treatment, the patient's evolution was good.

## Conclusion

The clinicians previously thought that ACG only had the pupillary block mechanism and that other entities had their own unique mechanisms (plateau iris, malignant glaucoma, and nanophthalmos).

What remained important was the clinical examination: AC depth to biomicroscopy and gonioscopy. Moreover, it was needless to ask about the Family Medical History.

These other mechanisms were dominant in some entities, but they contributed, such as

physiological risk factors for primary ACG. Research in the multiple dynamic features of ACG might explain its risk factors and lead to a better diagnosis [1-3].

The mechanisms behind ACG remain mysterious, but research has begun to reveal some clues. Women might be more prone to choroidal expansion, or Asians might have different iris fluid exchange [3-5].

These hypotheses should be tested in longitudinal studies of individuals at risk for ACG [6,7].

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