ANGLE CLOSURE GLAUCOMA AND SECONDARY GLAUCOMAS

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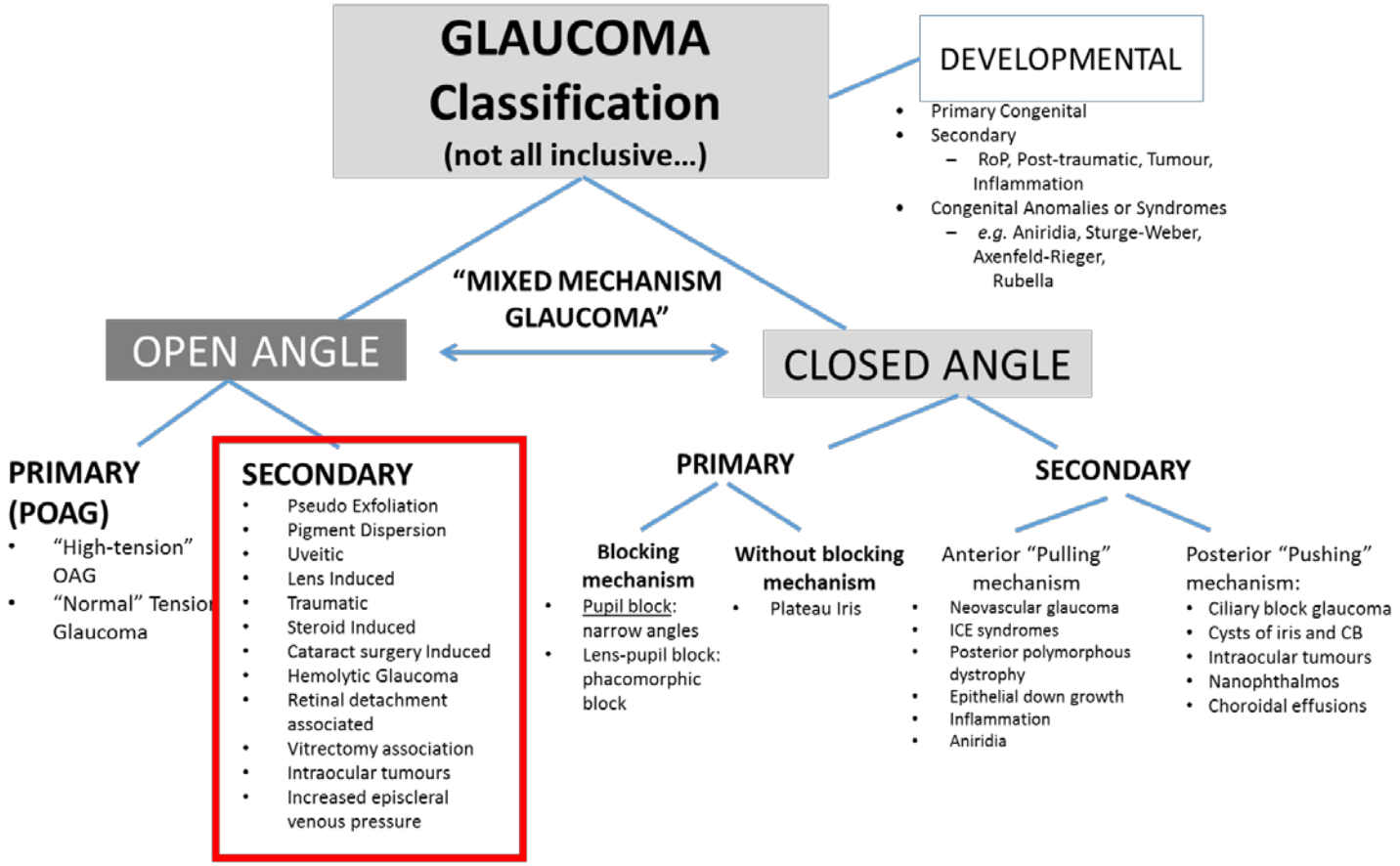


SECONDARY OPEN ANGLE GLAUCOMA

Secondary open angle glaucoma (OAG) conditions are associated with ocular or systemic abnormalities that cause an elevated intraocular pressure (IOP).

The most common secondary OAG conditions are as follows:

- Pigmentary dispersion
- Pseudo-exfoliation
- Angle recession
- Steroid-induced
- Inflammatory-induced

EVALUATION OF SECONDARY OAG

Careful evaluation of the eye is the key to diagnosing secondary OAG.

- Eye Health Examination:
  - Cornea
    - Endothelial abnormalities
  - Anterior chamber
    - Cells and flare
    - Depth
  - Iris

        - Transillumination defects
    - Crystalline lens
        - Pigment/material on surface
    - Optic nerve head
        - Glaucomatous damage
- Auxiliary testing using a range of instruments:
    - Tonometry
    - Gonioscopy

PIGMENTARY DISPERSION

PIGMENTARY DISPERSION SYNDROME (PDS)

Pigmentary dispersion syndrome is characterized by the liberation of pigment from the iris pigment epithelium, usually from the rubbing of the pigment epithelium on the lens zonules.

The pigment is carried by aqueous humor convection currents and deposits on a range of tissues in the anterior segment:

- Corneal endothelium
- Lens surface
- Trabecular meshwork (TM)

The pigment granules accumulate in the trabecular meshwork, which then may lead to progressive trabecular dysfunction and elevation of IOP.

PIGMENTARY GLAUCOMA (PG)

Pigmentary glaucoma (PG) is caused by the chronic elevation of IOP from the presence of pigmentary dispersion syndrome.

Elevation of IOP, due to the build-up of pigment in the trabecular meshwork, leads to the development of glaucomatous optic neuropathy.

- Glaucoma develops in 30-50 % of individuals i.e. converts from PDS to PG

PIGMENTARY DISPERSION SYNDROME PATHOPHYSIOLOGY

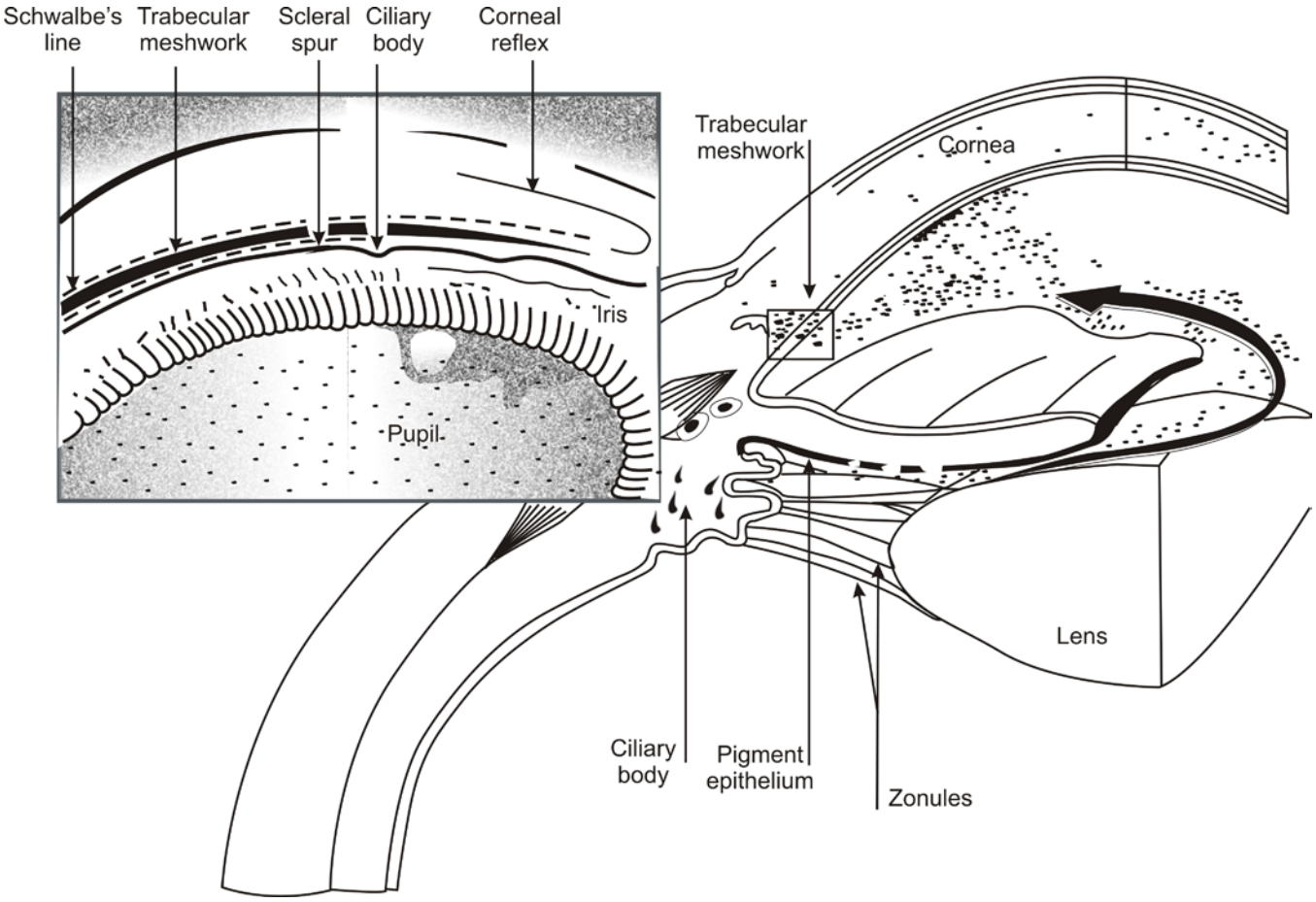


Figure 12.1: *Anterior chamber angle anatomy and pigment dispersion*

Mechanical contact between the concave posterior iris surface and the anterior zonules is responsible for the release of pigment granules that may deposit on structures throughout the anterior segment. There is greater pigment liberation in eyes with more pronounced concavity to iris.

In the trabecular meshwork, pigment accumulates on the trabecular endothelium and obstructs the inter-trabecular spaces - this leads to trabecular endothelial damage and meshwork dysfunction which results in elevated IOP in susceptible individuals (around 30% of cases).

CLINICAL FEATURES OF PIGMENT DISPERSION

- Early onset (age 20-40 years)
- Bilateral but asymmetric
- Size and density of Krukenberg spindle does not correlate with damage to the TM
- Density of pigment in TM plays a role in elevation of IOP
- Occurs more commonly in males and in myopic individuals
- Associated with lattice degeneration (twice as common) and retinal detachment (2-3%)

CLINICAL TRIAD FOR PDS

- Krukenberg spindle
- Mid-peripheral iris trans-illumination defects
- Densely pigmented trabecular meshwork (seen with gonioscopy)

The concept of reverse pupillary block is used to explain the development of iris concavity in PDS. The main features are as follows:

- Increase in pressure of the anterior portion of the anterior chamber in comparison to the posterior portion
- Pressure gradient forces iris against the lens creating a 'flap valve' and a concave configuration
- Pigment release is increased during pupil mydriasis and exercise – may lead to elevated IOP
    - Iris becomes more concave during exercise
    - Symptoms of increased IOP may develop following exercise
        - Haloes and glare around light
        - Nausea
- Pigment liberation lessens with time. PDS may reverse and PG may stop progressing
    - Pigment starts to release from endothelium and trabecular meshwork
    - Occurs 10% of the time
    - May explain non-progressive NTG in the elderly

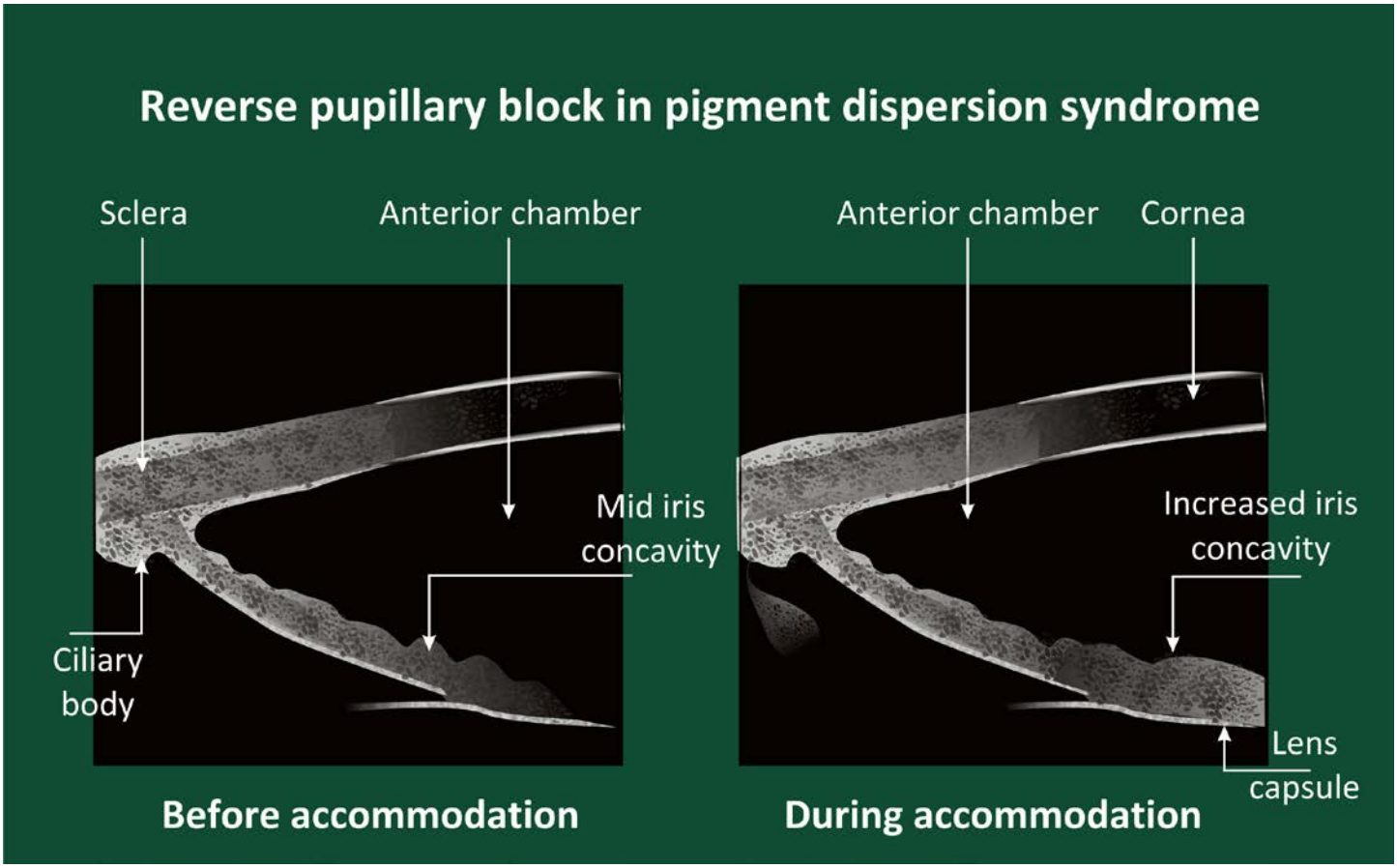


Figure 12.2: *Reverse pupil block*

GONIOSCOPY IN PDS

Figure 12.3: *Gonioscopic view of the anterior chamber angle*

Three things to note on gonioscopy as seen in Figure 12.3 are:

1) Dense pigmentation in the trabecular meshwork
2) Concave Iris configuration
3) Pigmentation in Schwalbe's line in a superior angle

PIGMENT DISPERSION GLAUCOMA

Less than half of individuals with PDS will develop ocular hypertension, not all of these individuals will develop glaucoma however it is important to monitor for its development. Some of the main features of PG are as follows:

- Development of glaucoma is pressure dependent – IOP tends to be high (usually above 29 mm Hg)
- IOP difficult to manage but there is a tendency for the condition to "burn out" with increasing age
- Progressive glaucomatous optic neuropathy

MANAGEMENT OF PG

The management of PG is essentially similar to POAG.

- Peripheral iridotomy (Figure 12.4) may have a role early in the disease process – it will change the configuration of the iris so that the iris flattens and no longer rubs on the zonules
- When IOP is harder to control – argon laser trabeculoplasty may be required

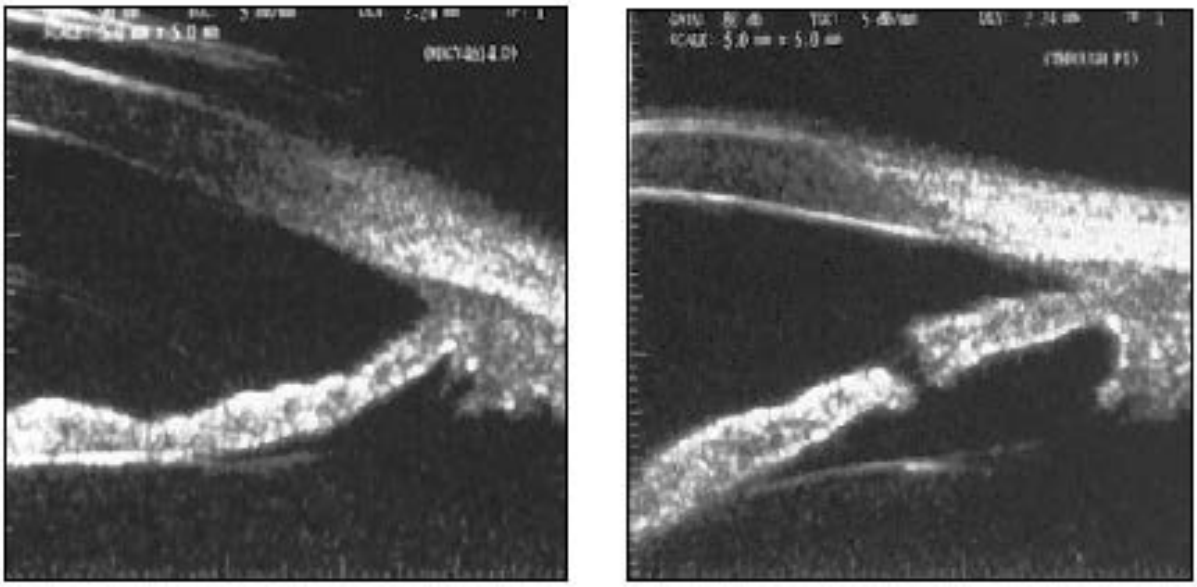


Figure 12.4: *Peripheral iridotomy following laser*

EXFOLIATION SYNDROME/EXFOLIATION GLAUCOMA

Exfoliative glaucoma (classically known as pseudo-exfoliative glaucoma) is caused from the production of a polymorphous fibrin glycoprotein from epithelial cells throughout the body. In the eye, this material is produced from ciliary body epithelial cells which then accumulates on the anterior lens surface with a dandruff-like appearance.

This material causes friction between the pupillary border pigment epithelium and lens capsule causing the release of pigment cells from the iris, which then accumulates in the TM potentially causing elevated IOP and optic nerve damage.

PATHOPHYSIOLOGY OF EXFOLIATION SYNDROME

The pathophysiological basis of exfoliation syndrome is as follows:

- Production of abnormal fibrillar extra cellular matrix (ECM) within the anterior chamber
- Deposit of ECM material on anterior chamber structures. Clinically, it is seen on the:
    - Anterior lens capsule
    - Cornea
    - Iris surface
    - Trabecular meshwork – usually too difficult to be seen clinically
- Accumulation of material on the pupillary margin
    - Increased apposition with the iris
    - Subsequent erosion of iris pigment as pupil dilates and constricts
        - “double ring” pattern seen clinically
- The condition is considered a systemic disease because of the development and deposit of material in other parts of the body– no evidence that it increases morbidity or that it causes pathology in other parts of the body

CLINICAL FEATURES OF EXFOLIATION SYNDROME

The main clinical features of exfoliation syndrome are as follows:

- Relatively same age group as POAG
    - Increased incidence > 65 years

- Can occur in any nationality
    - Used to be considered most prevalent in Caucasian and Scandinavian
- Exfoliation syndrome alone more common in women
    - Combination of exfoliation syndrome and glaucoma occurs the same in both women and men
- Unilateral first then becomes bilateral (usually asymmetric)
- May be associated with a higher risk of acute cerebro-vascular diseases and chronic cerebral conditions like Alzheimer's disease

HALLMARK SIGN OF EXFOLIATION SYNDROME

The main sign of exfoliation syndrome is a build-up of material on the anterior surface of the crystalline lens (Figure 12.5).

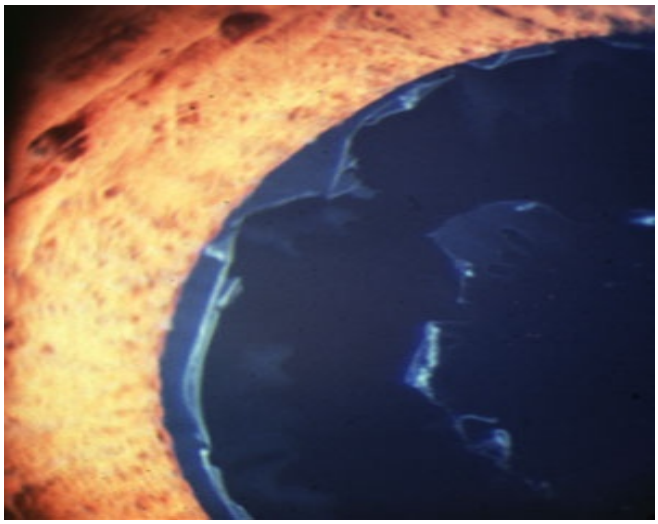


Figure 12.5: *Exfoliation material on the crystalline lens*

Characteristic features of the exfoliation syndrome includes the following:

- Classic pattern in the anterior lens surface
- Central translucent disc surrounded by a clear zone, which in turn is surrounded by a granular grey-white ring with scalloped edges
- Best observed after pupil dilation
- Dandruff like flakes may be deposited on the corneal endothelium, trabecular meshwork, anterior and posterior iris, pupillary margin, zonules and ciliary process, anterior hyaloid phase in aphakic eyes
- Peri-pupillary margin develops a moth-eaten pattern that is observed with transillumination
- Pigment becomes released from the iris and is deposited in the TM, anterior iris and corneal endothelium
- The angle pigmentation is moderate to heavy and often patchy in distribution
- Sampaolesi's line (pigment on Schwalbe's line)
- A shower of pigment may be seen upon dilation of the iris

GONIOSCOPY FINDING IN EXFOLIATION SYNDROME

The main observations (Figure 12.6) with a gonioscopy lens in exfoliation syndrome include the following:

- Open angle
- Clumpy TM pigment
- Sampaolesi's line
- Moderate to dense TM pigmentation

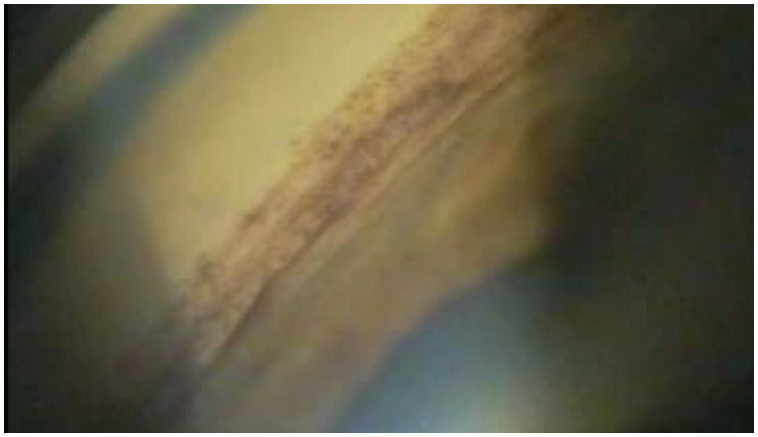


Figure 12.6: *View of the anterior chamber angle using a gonio lens*

COMPLICATIONS OF EXFOLIATION SYNDROME

The main complications in exfoliation syndrome are as follows:

- Secondary open angle glaucoma development
  - Management similar to POAG
  - After cataract extraction, signs will be harder to see thus increasing the importance of gonioscopy evaluation
- Lens zonule weakness
  - Makes cataract extraction more difficult
- Cataract formation

EXFOLIATION GLAUCOMA

Exfoliation glaucoma is the most common identifiable cause of glaucoma worldwide. Some of the main features of exfoliation glaucoma include the following:

- The number of corneal endothelial cells is reduced and central corneal thickness is also greater in eyes with exfoliation glaucoma, perhaps reflecting early corneal dysfunction
- The higher IOP observed with exfoliation glaucoma can lead to more rapid ONH damage and VF loss
  - Serial photographs and automated visual fields are more appropriate than IOP measurements for managing this condition. The patient may have progression yet manifest normal IOP in the office if the measurement coincides with a trough in the diurnal variation of the IOP
  - Wider fluctuations in IOP occur throughout the day than do so in patients with POAG
- It is an aggressive condition with an unpredictable course

MANAGEMENT OF EXFOLIATION GLAUCOMA

The main management strategy in exfoliation glaucoma is similar to POAG**.** The condition responds less to medical therapy and often requires laser or surgical management.

GLAUCOMA RELATED TO TRAUMA

Trauma to the eye, such as blunt or penetrating injuries, or chemical burns, can cause a number of anatomical changes resulting in glaucoma. Traumatic glaucoma is most often unilateral and can be divided into early and late (post-injury period) onset.

EARLY ONSET

Hyphema, or bleeding in the anterior chamber, may occur at the time of trauma, with or without angle recession. Acute IOP elevation may occur from blood in the angle, and later, from the accumulation of depigmented erythrocytes in the angle. There is usually a secondary inflammation as well.

There is a risk of rebleeding in the first 5 days after the formation of the hyphema, so, bed rest, cycloplegia, and topical steroid should be considered as part of the treatment.
Aminocaproic acid used orally has been suggested to reduce the risk of a rebleed by inhibiting clot dissolution, but the risks are great for thrombic emboli elsewhere in the body.

LATE ONSET

The natural crystalline lens can be subluxated causing anterior chamber closure. Angle recession commonly results from closed eye trauma, due to a split of the anterior angle structures at the level of the scleral spur. These angles appear to be very deep with gonioscopy, but are non-functional due to tissue changes.

Angle recession is often missed because gonioscopy is not routinely done on patients following trauma. If there is a 90-degree or greater angle recession, the risk of glaucoma increases dramatically. These eyes should be followed throughout the patient's life since glaucoma may develop years after the initial trauma.

ANGLE RECESSION GLAUCOMA

The main features of angle recession glaucoma are as follows:

- 4-10% of angle recessions >180° will develop glaucoma years later (angle recession glaucoma)
    - The initial trauma to the TM stimulates proliferative/ degenerative changes in the trabecular tissue leading to obstruction of aqueous outflow
    - Extension of endothelial layer with Descemet's-like membrane from the cornea over the AC angle
    - Majority of eyes that will develop glaucoma, have pre-disposition to reduced aqueous outflow in the fellow eye
- 30-50% will develop glaucoma in the fellow eye indicating that the eyes that develop glaucoma from angle recession may be prone to glaucoma development

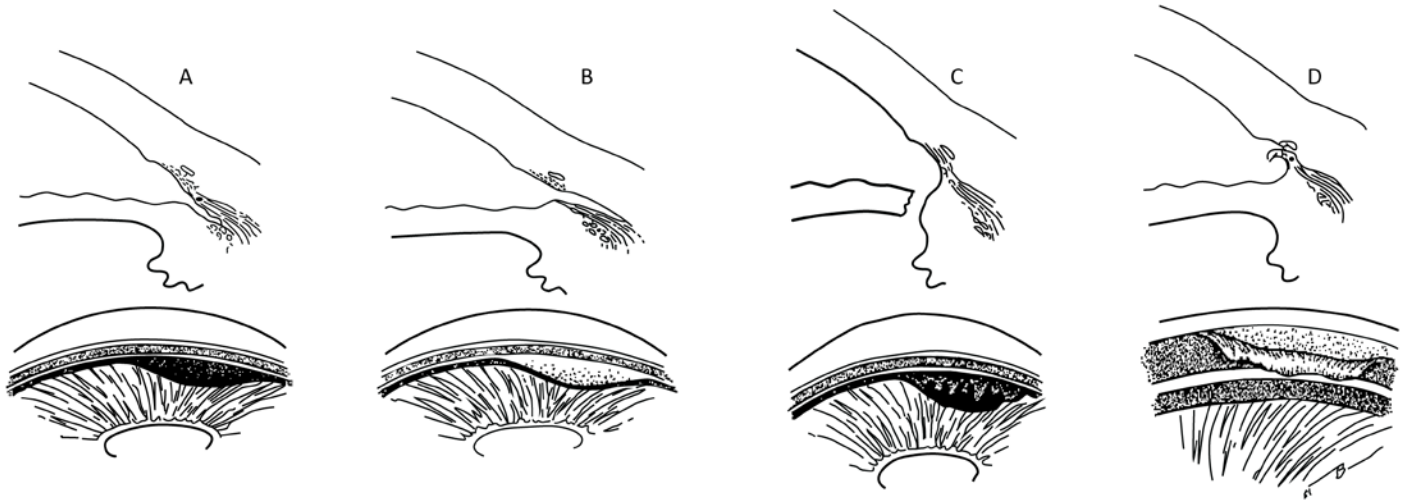


Figure 12.7: *Damage to the eye due to trauma*

Trauma can cause significant damage to the eye. The following observations are shown in Figure 12.7:

A) Angle recession – tear between longitudinal and circular ciliary body muscles
B) Cyclodialysis – separation of CB from scleral spur with widening of supra-choroidal space
C) Iridodialysis – tear at iris root
D) Trabecular damage – tear in the TM, flap of TM (very rare, difficult to observe)

Management of Trauma-induced Glaucoma

The main management strategies for glaucoma induced by ocular trauma include the following:

- Prostaglandins are thought to be the most effective medication because they enhance uveal/scleral outflow of aqueous humor
- Miotics and argon laser trabeculoplasty (ALT) have little value
- Medical management is more difficult and to achieve more effective results the eye may require filtration surgery

CORTICOSTEROID-INDUCED GLAUCOMA

The main features of steroid-induced glaucoma include the following:

- Prolonged use of topical corticosteroids may cause elevation of IOP and a clinical picture similar to POAG
- 5-6% of normal eyes develop marked IOP rises after 4-6 weeks use of topical high potency steroids
    - Prevalence increases with potency, frequency and duration of topical steroid
- Mechanism of action is still not fully understood but believed to involve alterations in the outflow of aqueous
    - Patients with POAG more likely to be steroid responders
        - Patients with family history of glaucoma may be at more risk of developing glaucoma when they have a steroid response
- Most of the steroid response is from ophthalmic corticosteroids
- Can also develop from:
    - Glucocorticoid creams, lotions and ointments applied to the face or eyelid
    - Nasal steroid spray
- IOP rise will usually decrease on cessation of steroid use
- Important to ask about prior steroid use in patients with suspicious glaucomatous optic neuropathy and normal IOPs

PRIMARY ANGLE CLOSURE GLAUCOMA (PACG)

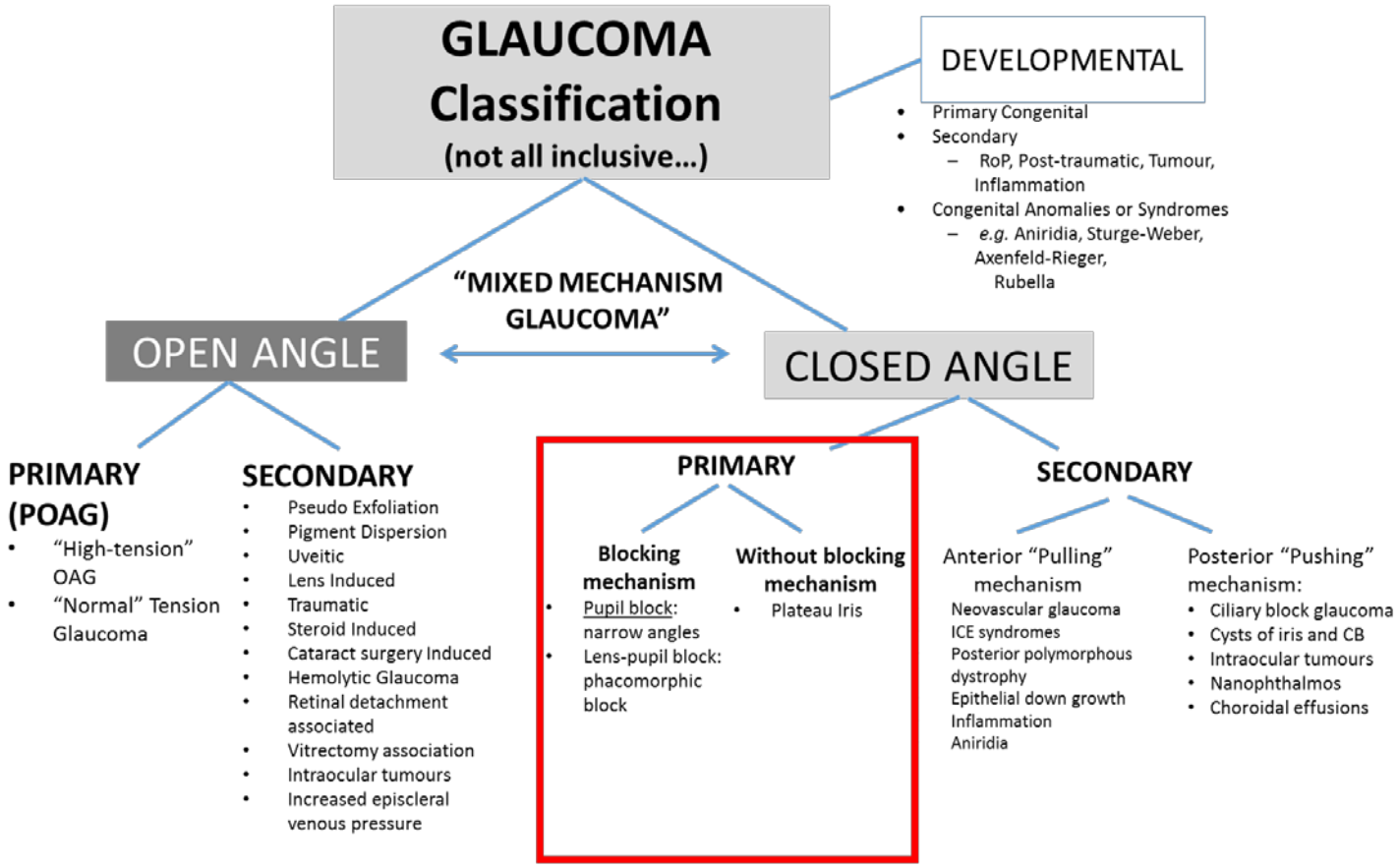


Figure 12.8: *Classification of glaucoma*

The following nomenclature is important in order to correctly classify cases of angle closure.

- **Primary Angle Closure Suspect**
  - Pigmented trabecular meshwork blocked < 180 degrees
  - No peripheral anterior synechiae (PAS), no increase in IOP, no damage to optic nerve, no visual field defects

- **Primary Angle Closure**
  - Pigmented trabecular meshwork is blocked by iris > 180 degrees
  - May have peripheral anterior synechiae (seen on gonioscopy)
  - Elevated IOP
  - No optic nerve damage or visual field defects

- **Primary Angle Closure Glaucoma**
  - Pigmented trabecular meshwork blocked > 180 degrees
  - May have PAS
  - Elevated IOP
  - Development of glaucomatous optic neuropathy and visual field loss

In angle closure glaucoma there is appositional or synechial closure of the angle so that aqueous humor cannot drain from the eye. The closure results in elevated IOP that leads to signs of glaucoma such as optic nerve damage and visual field loss. When the angle closure is sudden, the IOP elevation is significant and causes symptoms including pain, nausea, headache, red eye and tearing.

MECHANISMS OF ANGLE CLOSURE

Pupillary block is the most common reason for a blockage of the anterior chamber angle. The main characteristics of pupillary block are as follows:

- Resistance to trans-pupillary flow from posterior chamber to anterior chamber is increased by irido-lenticular contact
- Relative pressure gradient between the two chambers causes iris to bow forward and occlude the TM
    - Iris bombe
- Does not affect the depth of the central anterior chamber
- In individuals prone to pupillary block it is more likely to be induced when there is:
    - Emotional upset
    - Dim illumination
    - Pharmacological involvement

HOW DOES PUPILLARY BLOCK HAPPEN?

A) Relative pupil block
- From a predisposing anatomic basis
- Usually a narrow anterior chamber
- Iris normally sits on anterior surface of the crystalline lens
    - In narrow angles or another predisposing circumstance the contact force increases

B) Iris Bombe
- Iris-lens apposition increases resistance so much that the peripheral iris is forced forward (Figure 12.9)

C) Irido-corneal contact
- Forward motion of the iris occludes the trabecular meshwork, inhibiting aqueous drainage
- Causes appositional closure followed by peripheral anterior synechiae

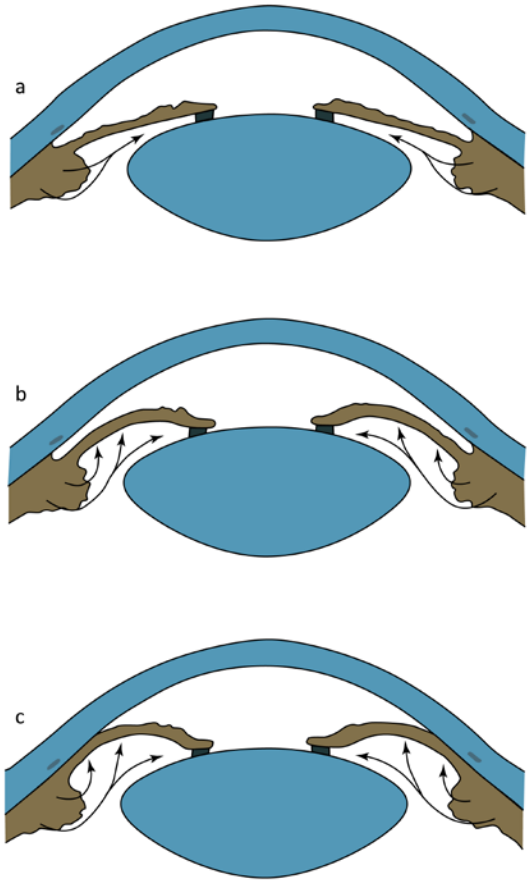


Figure 12.9: *Mechanism of iris bombe*

MANAGEMENT OF ANGLE CLOSURE FROM PUPILLARY BLOCK

LASER PERIPHERAL IRIDOTOMY

Obstruction of flow of aqueous humor from posterior chamber to anterior chamber occurs during a relative or absolute pupillary block. The blockage interferes with the normal physiologic pathway for aqueous flow and induces a pathologic pressure gradient between the posterior and anterior chambers which results in billowing of the peripheral iris and obstruction of the trabecular meshwork. A laser is used to create a full thickness hole in the iris that provides an alternate pathway for fluid to flow.

The main features of a peripheral iridotomy are as follows:

- Provides an alternate pathway for aqueous to flow
- Normalizes the pressure gradient between the anterior and posterior chambers
- Iris flattens, releasing the angle blockage

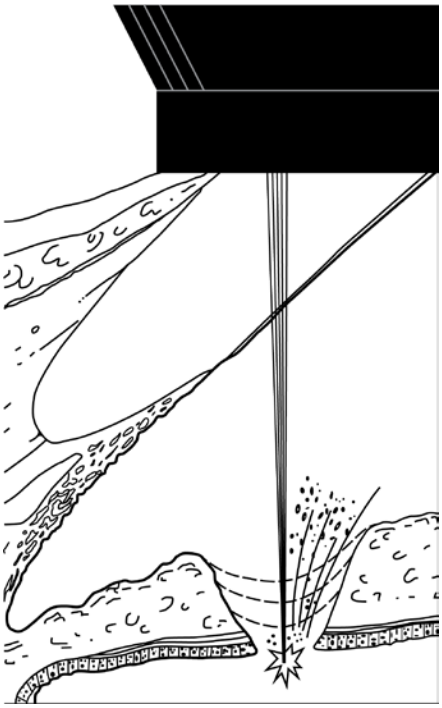


Figure 12.10: *Use of a laser to produce a peripheral iridotomy*

PERIPHERAL IRIDOTOMY PROCEDURE

The main characteristics of the procedure are as follows:

- A spot between 11 and 1 o'clock at peripheral iris is determined
- An iris crypt is localized
- Laser is used to create a full thickness hole at the selected location
- Usually done using Nd:YAG laser, however if that is not available an argon laser may be used

Figure 12.11: *Peripheral iridotomy hole at 12 o'clock*

PUPILLARY BLOCK GLAUCOMA

Pupillary block glaucoma is divided into two categories as described below:

- **Acute**
  - Rapid and large increase in IOP from a sudden blockage of the anterior chamber angle
- **Sub-acute (Intermittent)**
  - May be asymptomatic or have symptoms of the acute form but less severe
  - Episodes resolve over the course of days to weeks and resolve spontaneously
  - Peripheral anterior synechiae may be noted, usually in the superior quadrant
  - Normal function of IOP between attacks (therefore, IOP may be normal when measured clinically)

ANGLE CLOSURE GLAUCOMA

The most common form of angle closure glaucoma is the chronic type, which accounts for 80% of cases. The remaining 20% of cases are the acute type.

The main characteristics of angle closure glaucoma are as follows:

- Asymptomatic in many cases
- Diagnosis made with gonioscopic evaluation
- Chronically elevated with IOP, does not respond well to ocular hypotensive drops
- Significant patchy or total peripheral anterior synechiae
- Usually have optic nerve head and visual field changes
- Develops in a slow manner with on-going evolution of the synechiae

RISK FACTORS FOR ANGLE CLOSURE GLAUCOMA

The main risk factors for developing angle closure glaucoma are as follows:

- **Race**
  - Most prevalent amongst East Asians and Inuits
- **Gender**
  - Much more common in females (2-4x)
- **Age**
  - Risk increases with each decade after 40 years
  - Increased thickening of the lens, and its forward movement with age
- **Refraction**
  - Hyperopia
    - Regardless of race
  - If patient has significant myopia: look for secondary mechanisms
- **Family history**
  - Correlation with first degree relatives
  - 6 x more likely in East Asian population
  - 3.5 x more likely in the Inuit population

The following ocular anatomy characteristics are important risk factors for angle closure glaucoma:

- Small ("crowded") anterior segments
- Short axial lengths
- Thick crystalline lens
- Increased curvature of the crystalline lens
- Small corneal diameter

Medications that are potential risk factors for angle closure glaucoma include the following:

- Acetazolamide
- Hydrochlorothiazide
- Trimethoprim-sulfamethoxazole
- Indapamide
- Promethazine
- Spironolactone
- Isosorbide dinitrate
- Bromocriptine
- Tetracycline
- Corticosteroids
- Penicillamine
- Quinine
- Metronidazole
- Isotretinoin
- Aspirin
- Topiramate (Topamax**)
- Viagra

It is important to be able to clarify that risk of glaucoma in those taking these medications is only greater in individuals with an ocular anatomy that puts them at high risk for angle closure

ACUTE PACG CLINICAL SYMPTOMS

The symptoms associated with PACG may develop very quickly and include the following:

- Pain
- Red eye
- Photophobia
- Decreased or blurred vision
- Haloes around lights
- Headache
- Nausea
- Vomiting

TYPICAL ACUTE PACG CLINICAL SIGNS

The signs associated with PACG include the following:

- Decreased visual acuity
- High IOP
  - Between 40 and 90 mm Hg
- Circum-limbal injection
- Mid-dilated, non-reactive pupil
- Corneal edema
- Anterior chamber inflammation (cells and flare)
- May see iris bombe

- Closed angle is apparent on gonioscopy
- Peripheral anterior synechiae on gonioscopy

TYPICAL SUB-ACUTE/CHRONIC ACG SIGNS

The sub-acute and chronic signs associated with PACG include the following:

- Episodic blurred vision
- Complaints of haloes
- Narrow angles
- May have increased IOP
- Usually PAS superiorly
- Sectoral iris atrophy
- Glaukomflecken
- Optic nerve cupping
- Nerve fiber layer defects
- Visual field defects

GOAL OF TREATMENT FOR PACG

The main goal in treating PACG is to change the anatomy of the anterior chamber angle. Lowering of the IOP is only one part of the treatment process.

The initial goals of treatment include the following:

1. Decrease the IOP and break the pupillary block component
2. Perform definitive treatment of laser peripheral iridotomy (LPI) or surgical iridectomy (usually the latter)
3. Evaluate the fellow eye for prophylactic LPI treatment and perform if there is any indication the eye is also at risk

ACUTE PACG MANAGEMENT

The management strategy for acute PACG includes the following:

- Relieve corneal edema using oral glycerin
- Force aqueous into the anterior chamber angle, thus forcing the angle to open. This is achieved by using corneal indentation with e.g. a 4-mirror gonioscopy lens
- Use of a Beta-Blocker (1 drop or no more than 2 drops)
    - Check cardiovascular status: Blood pressure and pulse
- Use Pilocarpine 2%
    - If IOP < 40 mm Hg
    - IOP > 40 mm Hg has been theorized to be ischemic
    - Make sure diagnosis is correct first
    - Do not use concentrations greater than 2%
- Iopidine or Alphagan x 2 drops
- Pred Forte q15 minutes
- Diamox 500 mg (2 x 250 mg tabs)
- Osmoglyn cocktail
    - Isosorbide if patient is diabetic
- Laser PI is always indicated

SUGGESTED MANAGEMENT PROTOCOL

An appropriate management protocol is as follows:

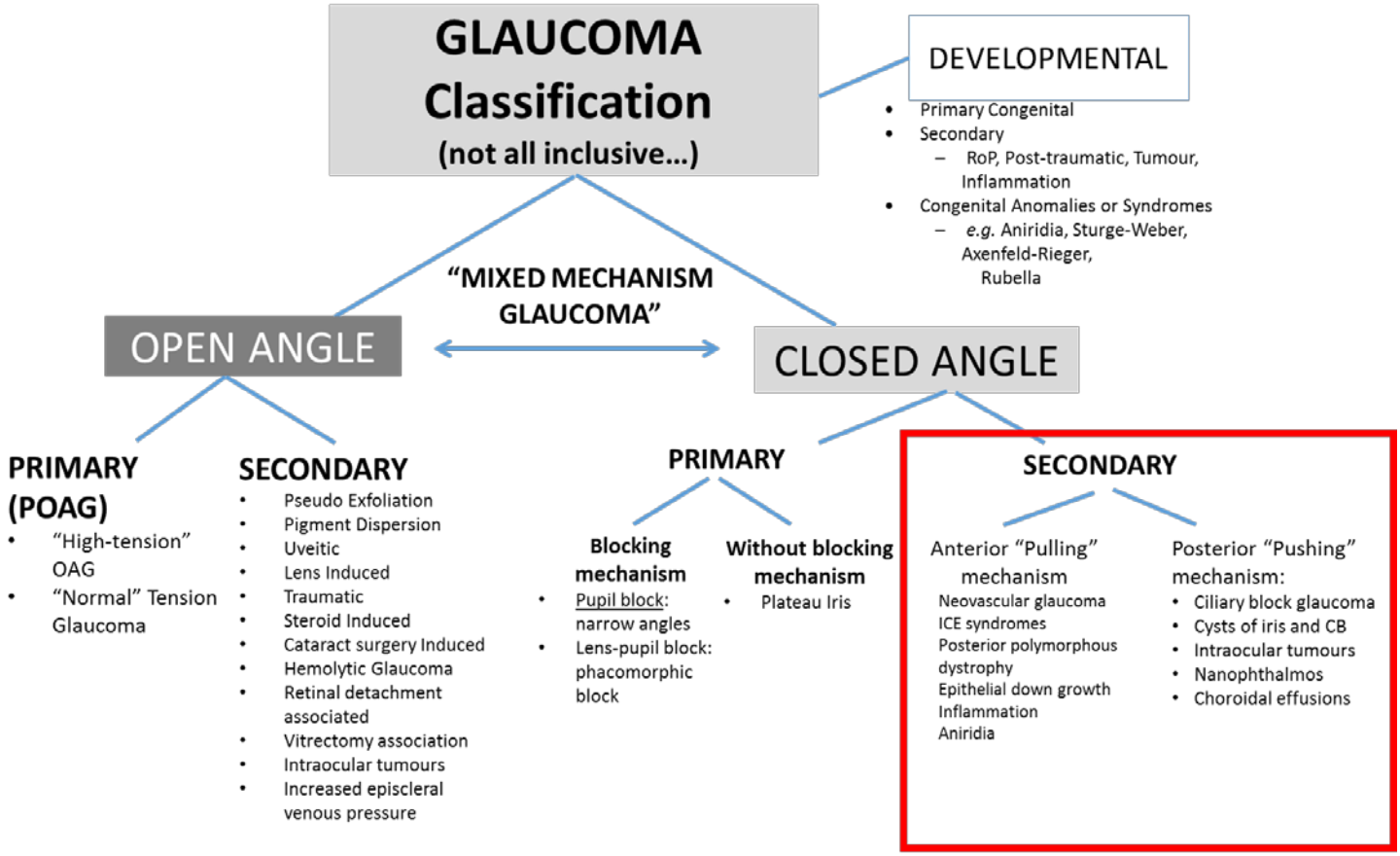
- Patient should immediately receive all topical medications listed above
- Recheck IOP after 30 min
  - If high: repeat all topical medications (maximum of 2 drops of topical beta-blocker should be given)
- IOP after 1 hour:
  - If high: use oral hyperosmotic medication
- IOP after 4-6 hours
  - If high: emergency LPI or surgery should be attempted

PLATEAU IRIS CONFIGURATION

A plateau iris is a rare condition and the diagnosis can be made using a gonioscopy lens. The features of a plateau iris include the following:

- Peripheral iris occludes the angle in patients with an atypical iris configuration (anteriorly positioned ciliary body forces peripheral iris into angle)
- Normal central anterior chamber depth
- Flat iris plane from pupil to peripheral iris
  - On gonioscopy will see a characteristic flat iris with a prominent roll nearby the insertion of the iris
  - Upon compression gonioscopy – iris does not fall backward, instead it bunches into the angle
- Plateau angle closure will **NOT be** relieved with LPIs
  - Therefore, it is necessary to do gonio on patients who have had an LPI in the past to make sure they don't have plateau iris
  - These patients will go on to develop complete PAS of the angle and angle closure glaucoma

SECONDARY ANGLE CLOSURE GLAUCOMA



In this classification an identifiable ophthalmic condition is present alongside the onset of angle closure i.e. the angle closure is secondary to the condition. Such a scenario is capable of producing severe elevations of intraocular pressure and marked loss of vision.

There are two fundamental mechanisms involved and these are:

- Anterior pulling mechanism
- Posterior pushing mechanism

ANTERIOR PULLING MECHANISM

The mechanism of action in this case is due to the peripheral iris being pulled forward onto the trabecular meshwork by the contraction of a membrane, inflammatory exudate, or fibrous band. The membrane, band, or inflammatory material contracts and a PAS forms and the angle subsequently is zippered shut.

Examples of the condition include:

- Neovascular glaucoma
  - Ocular vascular occlusive diseases
    - Diabetic retinopathy
    - Vein occlusions
- ICE syndrome

THE CLINICO-PATHOLOGIC STAGES OF NEOVASCULAR GLAUCOMA

Disease processes causing chronic hypoxia to the anterior segment can result in neo-vascularization of the iris at the pupillary border and also in the anterior chamber angle resulting in fibrosis and zippering shut of the angle. When this occurs, a very recalcitrant type of glaucoma follows.

The most common conditions causing this type of glaucoma are ischemic central retinal vein occlusion, diabetes and ocular ischemic syndrome which results from internal carotid artery stenosis. Gonioscopy is required to confirm the diagnosis, and treatment includes retinal laser, treating the underlying condition, and probable surgery such as shunt surgery.

The development of neo-vascular glaucoma is divided into the following stages:

A) The pre-glaucoma stage (rubeosis iridis)

- New vessels on the iris, iris and angle or angle alone

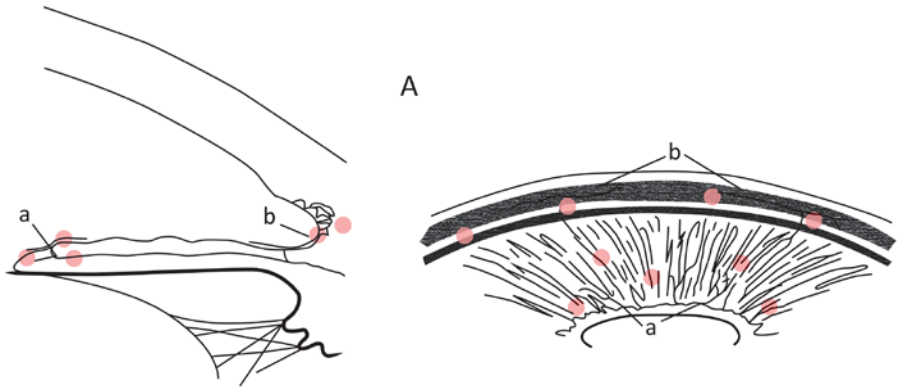


Figure 12.12: *Development of new vessels*

B) The open-angle glaucoma stage

- Increase in new vessels and the development of a fibro-vascular membrane on the iris and angle
- The growth of the fibro-vascular tissue initially decrease aqueous outflow

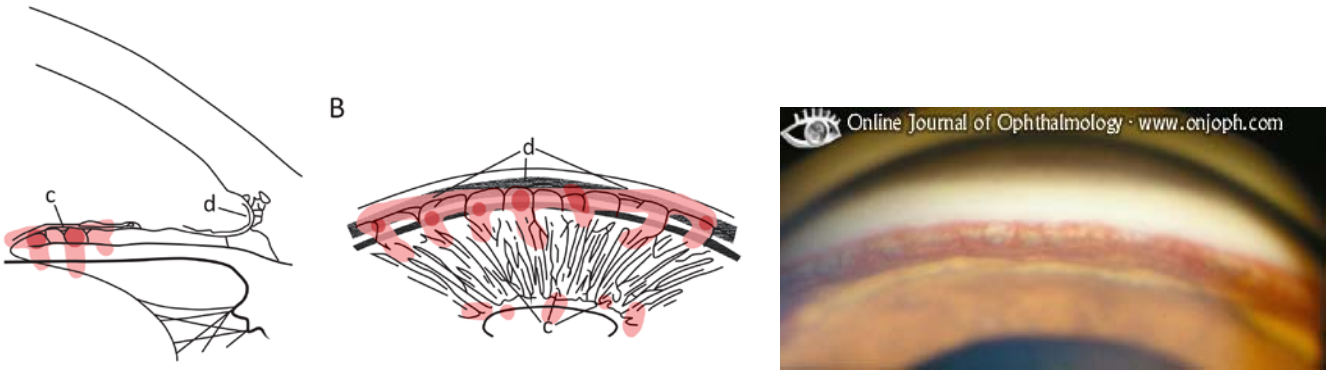


Figure 12.13: *Development of a fibro-vascular membrane*

C) Angle closure glaucoma

- Myo-fibroblasts proliferate and contract thereby pulling the angle closed
- Corneal endothelial proliferation over the angle can also occur

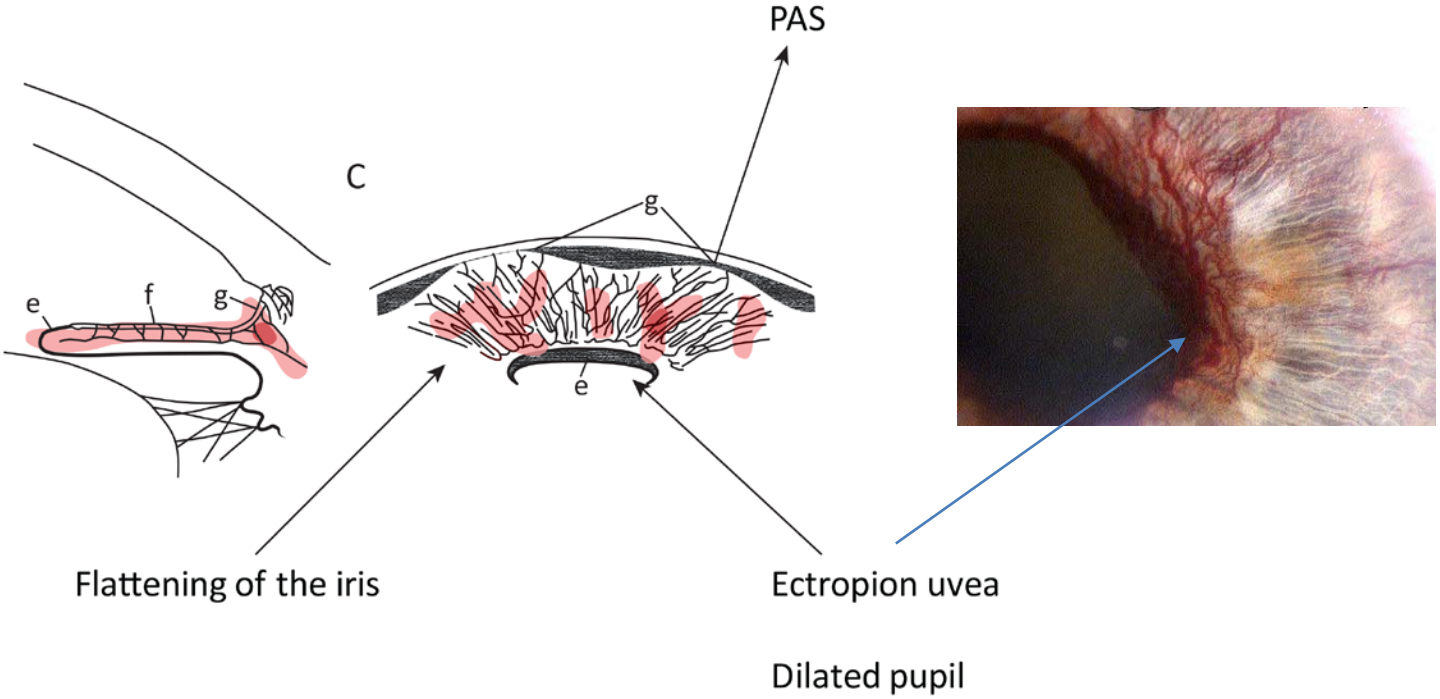


Figure 12.14: *Severe neovascular glaucoma*

POSTERIOR PUSHING MECHANISM

In this mechanism the peripheral iris becomes displaced forward by the lens, vitreous or ciliary body. The lens-iris diaphragm is moved sufficiently forward to close the angle. Often, there is an associated swelling and anterior rotation of the ciliary body thereby further closing the angle.

- Ciliary body swells, rotates forward about its attachment at the scleral spur and shortens diameter of ciliary ring
- Causes reduction of tension on zonules and lens moves forward

Reasons for Posterior Pushing Mechanism

The main causes of the posterior pushing mechanism include the following:

- Idiopathic e.g. uveal effusion syndrome
- Inflammatory
  - Uveitis
    - Sympathetic ophthalmia
    - Vogt-Koyanagi-Harada Syndrome
    - Pars planitis
  - Scleritis
    - Herpes Zoster
    - Collagen vascular disorders
    - AIDS
  - Infected scleral buckle
  - Thyroid disease
- Increased choroidal vascular pressure

    - Venous obstruction
        - Nanophthalmos
        - Hypoplastic vortex veins
        - Scleral buckling procedure
        - PRP
    - Arterial communication
        - Nanophthalmos
        - Hypoplastic vortex veins
        - Scleral buckling procedure
        - PRP
- Hypotony
    - Glaucoma surgery
    - Cataract surgery
- Infiltrative involvement
    - Tumours
        - Melanoma
        - Metastatic disease
        - Leukemia
        - Lymphoma
    - Lymphocytic hyperplasia

MANAGEMENT OF POSTERIOR PUSHING

The appropriate management involves the immediate referral to manage the underlying cause of the condition. Obtaining the best outcome usually requires referral to glaucoma or retinal specialist.