Primary Care of Secondary Glaucomas

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DR. TAYLOR KISER
DR. REENA LEPINE

Angle Closure Glaucoma

Mechanism

Obstruction of the trabecular meshwork by the iris impairs aqueous outflow causing elevated IOP

- Primary Causes
  - Pupillary Block
  - Plateau Iris
- Secondary Causes
  - Contracting membranes
  - Space Occupying Lesions
  - Inflammatory precipitates

Spectrum of Angle Closure

Primary angle closure suspect
- Considered precursor to PAC and PACG
- It has been estimated that 22% of eyes with PACS progress to PAC/PACG over 5-10 years

Primary angle closure glaucoma
- Glaucomatous optic nerve
- Visual field defects

Onset

- Acute
  - Sudden and severe symptoms
- Intermittent
  - Repeated “sub-clinical” attacks
- Chronic
  - May be asymptomatic until advanced visual field loss develops

Risk Factors

Shallow central anterior chamber depth
- Short axial length
- Thinner/anteriorly positioned lens
- Female
- Increasing age
- Race
  - Inuit
- Eastern Asians
- Family history of AG

Primary Causes

- Pupillary Block
- Plateau Iris
- Contracting membranes
- Space Occupying Lesions
- Inflammatory precipitates
Tools for diagnosis

- Tonometry
- Pupil evaluation
- Angle evaluation
  - Van Herick technique
  - Gonioscopy
  - Anterior segment OCT
  - Ultrasound Biomicroscopy (UBM)
  - EyeCam

Gonioscopy in ACG

- Occludable angle (per Shields)
  - No trabecular meshwork in 180 degrees or more
- Gonioscopy technique
- Indentation Gonioscopy
  - Appositional vs. Synechial
  - Previous apposition?
- Factors influencing gonioscopy findings
  - Inadvertent pressure
  - Lighting conditions

Anterior segment OCT

- Excellent supplement to gonioscopy
- Provides a more objective measurement
- Good for documentation and for evaluation of angle evolution over time
- Two types of scans
  - Low Resolution vs. High Resolution

Low Resolution AS-OCT

- Objective measurements
  - Angle opening distance (AOD 500, AOD 750)
  - Trabecular iris angle (TIA)
  - Trabecular iris space area (TISA)
  - Trabecular iris contact length (TICL)

High Resolution AS-OCT

- Objective measurements
  - Angle opening distance (AOD 500, AOD 750)
  - Trabecular iris angle (TIA)
  - Trabecular iris space area (TISA)
  - Trabecular iris contact length (TICL)

Precipitating Factors

- Mydriasis
  - Dim illumination
- Emotional stress
- Medications
Treatment
Primary angle closure suspect
Acute Angle Closure
Intermittent Angle Closure
Chronic Angle Closure

Pseudoexfoliation Syndrome
Pseudoexfoliation Glaucoma

Introduction
History
* First described in 1917 in Finland

Pseudoexfoliation Syndrome (PEX)
* Condition characterized by abnormal production and deposition of fibrillar extracellular material within the eye

Pseudoexfoliation Glaucoma (PXG)
* PEX with secondary open angle glaucoma

Pseudoexfoliation Syndrome
Systemic condition
* Heart, lung, kidney, gall bladder, liver, cerebral meninges
* Associated with cardiovascular and cerebrovascular diseases
* Systemic PEX has never been diagnosed without intraocular signs

Pseudoexfoliation Syndrome
Demographics
Females > Males
* Males more common to develop PXG
Incidence increases with age
* Rarely found: <50 year
* Highest prevalence: >70 years
Common in Scandinavia
In the US: 5-15% prevalence rate

Pseudoexfoliation Syndrome
Pathogenesis
Not fully understood

Aging epithelial cells
* Abnormalities of the basement membrane metabolism
* Production and accumulation of abnormal fibrillar extracellular material
### Pseudoexfoliation Syndrome

#### Ocular Manifestations
- **Produced:**
  - Equatorial lens capsule
  - Iris
  - Ciliary body
  - Trabecular meshwork

- **Deposited:**
  - Anterior lens capsule
  - Zonules
  - Ciliary body
  - Iris
  - Trabecular meshwork
  - Anterior vitreous face

#### Clinical Features
**Lens**
- Anterior capsule PEX material accumulation
- Central disk (1mm to 2.5 mm in diameter)
- Clear Middle Zone
- Due to contact between the iris and lens during pupil function
- Granular peripheral zone
- Observed in dilation

**Ciliary Body/Zonules**
- PEX material accumulation

**Complications**
- Cataract formation
- Phacodonesis
- Lens subluxation
- Angle-closure glaucoma

### Clinical Findings
**Iris**
- Pupillary margin deposits
- Peripapillary and iris sphincter atrophy
- Transillumination defects (“Moth-eaten”)
- Stromal deposits

**Complications**
- Pigment dispersion
- Iris rigidity
- Poor mydriasis
- Posterior synechiae

### Cataract Surgery Complications
**Surgical complications**
- Corneal endotheliopathy
- Poor mydriasis
- Zonular instability
- Lens subluxation

**Postoperative complications**
- Increase inflammation
- IOP elevation
- Late IOL decentration/prolapse

### Clinical Findings
**Anterior Chamber**
- Mild flare (pseudouveitis)
- Due to iris blood-aqueous barrier breakdown

**Cornea**
- Atypical corneal guttae
- PEX material on the endothelium
- Diffuse pigment deposition
- May form a Krukenberg spindle

**Complication**
- Endothelial decompensation
### Pseudoexfoliation Syndrome

#### Clinical Findings

**Gonioscopy**
- Pigment deposition
- Most marked inferiorly
- Patchy distribution
- Sampaolesi line
- Increased pigmentation has been correlated with increased IOP
- PEX material deposition
  - “Dandruff-like”

**Complication**
- IOP elevation
- OHT or Open Angle Glaucoma

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### Mechanism of IOP Increase

**Open-Angle Mechanism**
- Increase outflow resistance of the trabecular meshwork
- Melanin and PEX material blockage
- Disorganization of the canal structure
- Rapid pigment release
- IOP spikes

**Closed-angle Mechanism**
- Pupillary block
  - Posterior synechiae, iris rigidity, zonular weakness

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### Pseudoexfoliation Glaucoma Risk

**Cumulative Risk**
- 5% at 5 years
- 15% at 10 years

**High Risk**
- Unilateral PXG and PEX in the fellow eye
  - 50% at 5 years

**Low Risk**
- Unilateral PXG and no clinical PEX in the fellow eye

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### Pseudoexfoliation Glaucoma

**Usually diagnosed in the 7th decade**
- Higher mean IOP
- More advanced visual field defects

**Unilateral, open-angle glaucoma**
- Most common form of secondary open-angle glaucoma

**More aggressive compared to POAG**
- More treatment failures
- Higher incidence of progression
- More advanced visual field defects

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### Pseudoexfoliation Glaucoma

**IOP Measurements**
- Compared to POAG
  - Higher mean IOP levels
  - Marked IOP spikes
  - May occur after pupil dilation
  - May peak 2 to 3 hours after dilation
  - Takes 10 to 15 hours to return to normal IOP levels
### Pseudoexfoliation Glaucoma

**IOP Measurement**
- Compared to POAG
  - Greater diurnal fluctuations
  - May exhibit a diurnal range greater than 15 mmHg
  - 45% of PXG patients have IOP peaks outside of office hours

**Pseudoexfoliation Glaucoma Management**
- Increase difficulty to manage compared to POAG
  - Higher rates of surgical treatment
    - 87.8% of PXF patients needed a trabeculectomy

- First line therapy
  - Medical
  - Laser

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### Pseudoexfoliation Glaucoma Management

**Medical**
- Same as POAG
  - Initial treatment: Prostaglandins
  - Combination therapy usually needed
  - Pilocarpine is not recommended

**Laser Trabeculoplasty**
- Argon Laser Trabeculoplasty (ALT)
  - Greater percentage decrease in IOP than POAG patients
  - Due to trabecular hyperpigmentation
  - Within 3 years, nearly 50% of patients are back to baseline IOPs
- Selective Laser Trabeculoplasty (SLT)
  - Effective in IOP reduction
  - Effects may not last as long compared to POAG
- Complication
  - Postlaser IOP spike

### Uveitic Glaucoma

- Trabeculectomy
- Surgical outcomes not statistically different compared to POAG
- Cataract/Trabeculectomy
Prevalence

- 2 million people worldwide
- 10% of those patients will be blinded by it
- Retinal damage
- Secondary Glaucoma
- 20-40% of uveitis patients develop glaucoma

Demographics

- No race, sex, or age predilection for those that develop glaucoma
- Dependent on type of uveitis
  - Fuchs' heterochromic iridocyclitis
  - Posner Schlossman syndrome
  - Herpetic uveitis
  - Juvenile idiopathic arthritis
- Chronic>Acute
- Children>Adults

Pathogenesis of Uveitic Glaucoma

- Blockage of TM
  - Inflammatory cells, proteins, debris, or fibrin
- Posterior synechiae
- Peripheral anterior synechiae
- Ciliary body inflammation
- In chronic cases, trabecular meshwork scarring may occur

Relation to systemic health

- An underlying etiology is found in ~40% of patients
  - Infectious
    - Herpetic keratouveitis
    - Sarcoidosis
    - Congenital Rubella
    - AIDS
    - Hansen disease (Lepromatous Leprosy)
    - Disseminated meningococcal infection
    - Hemmorhagic fever with renal syndrome
    - Listeria monocytogenes
  - Inflammatory
    - Juvenile idiopathic arthritis
    - Ankylosing spondylitis
    - Reiter's syndrome
    - Bechet's disease
    - Rheumatoid arthritis
    - Systemic Lupus Erythematosus
  - Idiopathic

Symptoms

- Photophobia
- Redness
- Brow Ache
- Ocular pain
- Blurred vision
- Colored haloes
- Nausea

Signs

- Cell
- Flare
- Keratic precipitates
- Band keratopathy (with chronicity)
- Epithelial dendrites
- Stromal scarring
- Iris atrophy
- Cystoid macular edema
- Band keratopathy
- Cataract
- Iris nodules
- Posterior Synechiae
- Angle/Iris neovascularization
- Optic nerve damage
- Cystoid macular edema
- Retinitis
- Perivascular sheathing
- Choroidal infiltration
Medical Management of Uveitic Glaucoma

1. Treatment of the underlying systemic disease
2. Control of inflammation
3. Control of IOP
   - IOP lowering medications
   - Topical: CAI's, beta-blockers, alpha-agonists, and prostaglandins
   - Oral: CAI's and Hyperosmotics

Surgical Management

Required in approximately 40% of adult patients and 60% of adolescent patients.

Laser Iridotomy:
- High safety profile
- ~50% in first 20 days
- Surgical iridectomy is the next step.

Laser trabeculoplasty:
- ALT
  - No change
  - Introduces more inflammation than SLT
- SLT
  - 19.8% decreased of IOP after 1 year
  - No increase in flare up of inflammation

Trabeculectomy

Glaucoma drainage device implantation

Cyclodestructive procedures:
- Transscular diode laser cyclophotocoagulation (TDCP) is relatively safe for refractory glaucoma
  - Adults
  - Higher success rate in adults
- Compared to GDD it has similar success rate but higher rate of visually threatening complications
- Children
  - Lower success rate
  - Retreatment often needed
  - Lower rate of visually threatening complications

Posner-Schlossman Syndrome

Introduction

Glaucematoocyclitic Crisis or PSS

History
- First described in 1948 by Posner and Schlossman

Characteristics
- Recurrent attacks
- Mild, nongranulomatous uveitis
- Markedly increased IOP during attacks
- Acute attacks resolve spontaneously within hours to weeks

Epidemiology

Unilateral
- 50% have a bilateral involvement at different times

Young adults
- 20 to 50 years old
- Males > Females
Etiology
Unknown Cause
Infectious and Non-infectious Theories

Infectious Etiology Theories
Cytomegalovirus (CMV)
Helicobacter pylori
Varicella Zoster Virus (VZV)
Herpes Simplex Virus (HSV)

Non-infectious Etiology Theories
Autonomic Dysregulation
Ciliary Vascular Abnormalities
Genetic Factor
* HLA-Bw54

Possible Mechanism of IOP Elevation
During attacks: Prostaglandin levels increase
* Trabeculitis
* Prostaglandins mediate inflammation within the trabecular meshwork
* Aqueous Production Elevation
* Prostaglandin E increases aqueous production

Posner-Schlossman Syndrome
Clinical Findings
Symptoms
* Unilateral blurred vision
* Mild discomfort/pain
* Halos/Rainbows around lights
* History of prior events

Visual Acuity
* Mild decrease
* Can be severe

Pupils
* Slightly dilated
* Sluggish
### Posner-Schlossman Syndrome

#### Clinical Findings

**Conjunctiva**
- Mild injection

**Cornea**
- Microcystic edema
- Inferior, fine, and white keratic precipitates

**Anterior Chamber**
- Mild cells and flare

**Iris**
- Heterochromia or atrophy

**Tonometry**
- Increased IOP
- Greater than 30 mmHg
- Usually between 40 to 60 mmHg
- IOP elevation usually precedes the anterior chamber reaction

**Gonioscopy**
- Open angle without synechiae
- May observe keratic precipitates

**Posterior Segment**
- Possible vasculitis and snowbanking

**Optic Nerve Head**
- Healthy rim tissue
- ONH studies during an attack
- Decrease volume/area to the rim
- Increase volume/area to the cup
- Decrease to the RNFL area
- Blood flow reduction
- Neuroretinal rim
- Peripapillary nasal and temporal sectors

### Open-Angle Glaucoma

#### Diagnosis Risk

Glaucoma diagnosis: 45% of PSS patients

Over time: Develop ONH damage and visual field defects

Greatest risk factor
- Duration of PSS
- Risk of glaucoma after 10 or more years with PSS is 2.8 times higher than PSS patients diagnosed less than 10 years
Acute Attack Management

**Goal:** Control Inflammation and IOP Elevation

**Medical**
- Anti-inflammatory
  - Topical Corticosteroid
- Anti-inflammatory
  - Topical/Oral Non-steroidal anti-inflammatory (NSAID)
  - Steroid-response patients
  - Topical NSAID
  - Oral NSAID
  - Indomethacin
  - Prostaglandin antagonist

**Antiglaucoma**
- In-office treatment
  - Systemic carbonic anhydrase inhibitor
  - Acetazolamide 250mg
  - Apraclonidine
  - 1% shown to reduce IOP by 50.3% four hours after instillation

**Medical**
- Antiglaucoma
  - Topical Beta-Blockers
  - Timolol 0.5%
  - Topical Alpha-Adrenergic Agonist
  - Brimonidine 0.1% to 0.2%
  - Topical Carbonic Anhydrase Inhibitors
  - Dorzolamide 2%

**Cycloplegic agent**
- Homatropine 5%

**Procedure**
- Anterior chamber paracentesis
- IOP is considered dangerously high

**Follow-Up**
- Start: Every few days
  - IOP is elevated
- Then: Weekly
  - Attack has resolved
  - Steroids are tapered
**Between Attack Management**

- Anti-inflammatory and antiglaucoma medication
  - Not necessary
  - No frequency of attack reduction

- Self medicate
  - Well informed patients
  - Topical NSAID and antiglaucoma drops

- Follow-Up
  - Monitored as if they were diagnosed with POAG

**Antiviral Therapy Studies**

- Studies testing:
  - Systemic ganciclovir
  - Topical ganciclovir
  - Intravitreal ganciclovir
  - Intraocular ganciclovir

**Results**

- Systemic therapy: 91% responded to treatment
- Topical therapy: 64% responded to treatment
- Recurrence Rate
  - Topical therapy: 57%
  - Systemic therapy: 80%

**Surgical Management**

- Used with uncontrolled IOP

- Trabeculectomy with antimetabolites
  - Successful in preventing IOP spikes during attacks
  - May not require antiglaucoma drops after surgery
  - Filtering bleb may filter out some inflammatory cells

**Introduction**

- Traumatic glaucoma occurs when visual field loss and glaucomatous optic neuropathy occur secondary to elevated IOP after ocular trauma
Types of Trauma

- Blunt ocular trauma
- Penetrating Injury
- Open vs. closed globe
- Retained foreign body
- Chemical or Thermal Burns
- Radiation exposure

Demographics

- 85% Males
- 75% Younger than 30 years old
- Sporting and domestic injury accounted for almost 2/3 of these injuries
- Ball games most common
- Boxing is extremely high risk
- Other causes
  - industrial accidents
  - malicious acts
  - air bag inflation

Pathophysiology

When blunt trauma occurs, a change in the shape of the globe occurs from compression

- Anterior/posterior shortening
- Equatorial elongation

The lens-iris diaphragm is forced posteriorly
Their attachments are moving outward, producing a shearing force.

- [Video 1](http://www.youtube.com/watch?v=XjwO9InuFJk)
- [Video 2](http://www.youtube.com/watch?v=aGsntCznhcY)

Findings associated with trauma

- Pupil damage
- Angle recession
- Iridodialysis
- Cylodialysis
- Iridoschisis
- Cataract
- Trabecular Meshwork tear
- Hyphema
- Corneal edema
- Iritis
- Chorioretinal trauma
- Dislocated lens

Angle recession glaucoma

4-9% of those with more than 180 degrees of angle recession will eventually develop a chronic glaucoma called angle recession glaucoma:

- Misnomer
- Variable onset

Risk of development and POAG

Clinical exam

- History
- Tonometry
- Gonioscopy
  - Broad ciliary band
  - Localized depression of the iris
  - Torn iris processes
  - Abnormally white scleral spur
- Health exam
- Optic nerve assessment
### Medical Management of ARG

- Medications that reduce aqueous production (preferred)
  - Beta-blockers
  - Carbonic anhydrase inhibitors
  - Alpha-2 Agonists
- Prostaglandin analogs
  - Increase uveoscleral outflow
  - Bypass trabecular meshwork damage
- Miotics
  - Should be avoided!

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### Surgical management of ARG

- Laser trabeculoplasty may be attempted
  - Does not have a high success rate
- ND:YAG laser trabeculopuncture
  - Variable success
- Filtration surgery
  - Lower success rate than with POAG
  - Antimetabolites improve success
- Cyclodestructive procedure

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### Ghost cell glaucoma

- Occurs after vitreous hemorrhage
- Onset 1-3 weeks post-injury
- Degenerated red blood cells (ghost cells) obstruct aqueous outflow
- Khaki colored cells in AC
- “Candy Stripe” sign

**Treatment:**
- Medical therapy helps but often is insufficient
- Surgical therapy involves vitrectomy and anterior chamber lavage

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### Hemolytic Glaucoma

- Similar to “Ghost Cell Glaucoma”
- Occurs several days to weeks after an intraocular hemorrhage
- Trabecular meshwork occluded!
  - Hemoglobin filled macrophages
  - Free hemoglobin
  - Remnants of lysed red blood cells
  - RBCs stain in AC
- Reddish-brown hue to TM on gonioscopy

**Treatment:**
- Often self-limiting, medical therapy should be attempted first!
  - Anterior chamber lavage and possibly vitrectomy may be indicated

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### Hemosiderotic Glaucoma

- Rare condition
- Associated with prolonged history of intraocular bleeding
- As blood components break down they may produce toxic granules of inorganic iron
- Sclerosis and obliteration of intratrabecular spaces may occur!

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### Lens related glaucoma

- Pupillary block with subsequent angle closure:
  - Injury to the zonules may cause an anterior displacement of the lens
- Phacomorphic
  - Disruption of the Lens capsule during trauma may cause rapid swelling of the lens with angle closure
- Lens particle glaucoma
  - Disruption of lens capsule during trauma releases particles into the AC clogging the TM
  - A fine glistening may be seen in the angle
  - Chronic inflammation
Wrap up for traumatic glaucoma

Acute management
- Initial injury repair or treatment
- IOP monitoring and control
- Inflammation

Chronic management
- Varies based on initial insult
- Angle Recession
- Long term IOP monitoring
- Patient education

Pigment Dispersion Syndrome

Pigmentary Glaucoma

Introduction

History
- First pigmentary glaucoma case described in 1940

Pigment Dispersion Syndrome (PDS)
- Condition characterized by dispersion of iris pigment throughout the eye

Pigmentary Ocular Hypertension (POH)
- PDS with elevated IOP and no glaucomatous optic neuropathy

Pigmentary Glaucoma (PG)
- PDS with glaucomatous optic neuropathy

PDS/PD Demographics

<table>
<thead>
<tr>
<th>Pigment Dispersion Syndrome</th>
<th>Pigmentary Glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young (20-40 years)</td>
<td>Young (30-50 years)</td>
</tr>
<tr>
<td>Myopic</td>
<td>Myopic</td>
</tr>
<tr>
<td>Women = Men</td>
<td>Men (78-93% are males)</td>
</tr>
<tr>
<td>Caucasian</td>
<td>2.5% prevalence in the US</td>
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PDS Pathogenesis

Possibly genetic
- Autosomal dominant

Reverse-Pupillary Block
- Higher pressure in the AC than the PC
- Posterior bowing of the peripheral iris
- Friction between the posterior iris and zonular bundles
- Initiated by accommodation, blinking, eye movements, exercise

Pigment Dispersion Syndrome

Clinical Features

Cornea
- Krukenberg Spindle
- Pigment deposits on the corneal endothelium
- Vertical spindle-shaped pattern
- Caused by aqueous convection currents
- More common in women
Pigment Dispersion Syndrome
Clinical Features

Cornea
- Endotheliopathy
- Pleomorphism (abnormal shape)
- Polymegathism (abnormal size)
- Normal endothelial cell counts and corneal thickness

Anterior Chamber
- Depth
  - Deeper compared to POAG patients
  - Male AC depth > Female AC depth
- Pigment Showers
  - Circulating AC pigment
  - Mistaken for uveitic inflammatory cells

Iris
- Transillumination Defects
  - Presents in 80% of cases
  - More obvious in light colored eyes
  - Mid-peripheral iris
  - Spoke-like pattern
- Partial loss of pupillary frill
- Pigmentation
  - Pigment deposits on the anterior surface
  - Asymmetric cases
  - Darker iris: More affected

Pupils
- Anisocoria
  - Larger pupil observed in the eye with the greater iris transillumination defects
  - Mechanical irritation of smooth muscle cells

Gonioscopy
- Open angle
- Increased pigmentation
  - Trabecular meshwork
  - Schwalbe's line
- More prominent inferiorly
- Backward-bowing of the iris
- Greater number of iris processes
Pigment Dispersion Syndrome
Clinical Features

- Lens and Zonules
  - Pigment deposition
  - Anterior capsule
  - Posterior capsule
  - Scheie's stripe: Pigment accumulation at the insertion of the zonules into the posterior lens capsule

- Posterior Segment
  - Lattice degeneration (20-33%)
  - Retinal breaks (12%)
  - Retinal detachments (5.5-6.6%)

Mechanism of IOP Increase

- Reduced aqueous outflow due to pigment obstruction
- Destruction of the trabecular meshwork
  - Due to loss of the trabecular meshwork cells

Risk of Developing Pigmentary Glaucoma

- 10% at 5 years
- 15% at 15 years
- Mean age of diagnosis: 42 years
- Most significant risk factor: IOP >21 mmHg

Risk Factors for Developing Pigmentary Glaucoma

- Glaucoma Family History
  - 4.21% of PDS patients
  - 26-48% of PG patients
  - Family members do not all have PG

- Gender
  - Men > Women
  - 78-93% of cases are men
  - Men: 34-46 years
  - Women: 43-53 years
  - More aggressive in men
Risk Factors for Developing Pigmentary Glaucoma

**Refraction**
- 38-100% of PDS patients are myopic (> -1.00D)
- Degree of myopia is greater
  - More myopic eyes tend to have a deeper AC allowing more contact between the iris and the zonules

**Krukenberg Spindle**
- More common in PG eyes
- Develop in eyes with a greater degree of pigment dispersion

**Initial IOP**
- Most important factor for developing PG
- Risk increase if IOP at initial diagnosis is >21 mmHg

**Pupil Dilation**
- Increase IOP by inducing a pigment shower
- Following 10% phenylephrine use: No greater than a 2 mmHg elevation observed

**Exercise**
- Increases the posterior iris concavity
- Induces pigment dispersion
- Prevention
  - Laser iridotomy
  - Pilocarpine
- No studies have shown a ≥5 mmHg IOP increase

**Accommodation**
- Induces anterior lens movement
- Decreasing the AC depth thus increasing the AC pressure
- Results in posterior bowing of the iris
### Risk Factors for Developing Pigmentary Glaucoma

**Blinking**
- Pumps aqueous from the posterior chamber to the anterior chamber
- Increases anterior chamber pressure
- Results in posterior bowing of the iris

### Pigmentary Glaucoma

**Symptoms**
- Majority is asymptomatic
- Headaches and blurred vision after exercise
- Haloes around lights

### Pigmentary Glaucoma Clinical Findings

**IOP**
- Mean of 29 mmHg at diagnosis
- 25% of PG patients had an IOP >31 mmHg at diagnosis
- 12.5% of PG patients had an IOP >39 mmHg at diagnosis
- In a 25 year review, IOP ranged 24-56 mmHg at diagnosis

**Visual Field Defects**
- 28-44% progression in 11-17 years

**Optic Disc Cupping**
- No difference compared to POAG

**Degree of Pigmentation**
- In asymmetric cases
  - Severe eye will have a greater degree of pigment dispersion
  - Degree of trabecular meshwork pigmentation correlates with severity

**Filtration Bleb**
- Pigmentation has been observed within the filtering bleb
- Unknown if this affects the trabeculectomy function

**Retinal Detachment**
- Slightly higher (7.6-10%) compared to PDS
Pigmentary Glaucoma Burn-Out Phase

Occurs with advancing age
Reduction in pigment dispersion and IOP normalization
* Observed over a 10 year period
Inferior angle pigmentation clears before the superior angle

Management of PDS/PG

Depends on the Disease State

* Inactive pigment dispersion with stable IOP
  * PDS patients and burn-out PG
* Active pigment dispersion with stable IOP
  * PDS and PG patients with sufficient aqueous outflow facility

Medical Therapy
* Pilocarpine
  * Prevents
* Pupil dilation
  * Inhibits exercise-induced IOP elevation
  * Reverses iris bowing
* Side effects
  * Increase risk of retinal detachment
  * Accommodative spasm
  * Cataract formation

Pigmentary Glaucoma Management

Medical Therapy
* Beta-Blockers
* Carbonic Anhydrase Inhibitors
* Alpha-Adrenergic Agonists
* Prostaglandins
  * Uveoscleral outflow
  * No pigment dispersion increase

Theories
* Increasing axial length of the lens
* Age-related miosis
* Absent of pigment left in the posterior pigment epithelium
* Ciliary body shut down
* Accommodation

Management of PDS/PG

Depends on the Disease State

* Active pigment dispersion with progressive glaucoma and elevated IOP
  * PG patients
* Inactive pigment dispersion with progressive glaucoma and normal/elevated IOP
  * PG patients with poor aqueous outflow facility
Pigmentary Glaucoma Management

Laser Therapy
- Laser Trabeculoplasty
  - Argon Laser Trabeculoplasty
    - Effective due to greater energy absorption by the pigmented TM
    - More effective in younger patients
    - Effect diminishes with time: Success rate of 45% at 6 years
  - Selective Laser Trabeculoplasty

Laser Iridotomy
- Flattens the iris, reversing posterior iris bowing
- More effective in patients <40 years old
- Useful only in patients with active stages

Trabeculectomy
- Most effective treatment for PG compared to medical therapy
- Higher percentage require surgery compared to POAG
- Men tend to require it sooner