# Glaucoma

Dr R.Hari Babu Professor& Head Department of Pharmacy Practice Chebrolu Hanumaiah Institute of Pharmaceutical Sciences



#### Introduction

- Glaucoma are ocular disorder characterized by changes in the optic nerve head (optic disc) and by loss of visual sensitivity and field.
- It may be generally defined as those conditions in which the intraocular pressure (IOP) is too high for the normal functioning of the optic nerve head.

## Classification of glaucoma

Primary glaucoma
Open angle
Angle closure

Secondary glaucoma Open angle Angle closure

Congenital glaucoma

## ETIOLOGY

The factors that determine the level of IOP are

- > The rate of aqueous humour production
- The resistance encountered in the outflow channels fine balance between these is necessary to keep the pressure within the eye in the range of 16-21mmHg.

# Rate of aqueous humour production

Production of aqueous humour occurs in the ciliary epithelium by two mechanisms:

Secretion due to an active metabolic process

Ultrafilteration influenced by the level of blood pressure in the ciliary capillaries

## **OUT FLOW OF AQUOUS HUMOR**

- 80% through the trabecular meshwork into the canal of schelemm and into the venous circulation via the aqueous veins.
- 20% through the clilary body into the suprachoroidal space to be drained into the ciliary body ,choroid sclera via the venous circulation (uveosclera pathway)

# PRIMARY OPEN - ANGLE GLAUCOMA

- Referred to as chronic simple glaucoma
- Associated with a relative obstruction to aqueous outflow through the trabecular meshwork
- Is a chronic progressive disease of insidious onset
- Affecting both the eyes .

#### **PREVELANCE**

- It is most common type of glaucoma and affects approximately 1in 200 of the population over the age of 40 years.
- POAG is responsible for about 20% of all cases of blindness in the UK
- Affects both sexes equally
- Frequently an inherited condition; with approximately 10% of first degree relative of POAG suffers eventually developing the disease.

#### PATHOPHYSIOLOGY-POAG

In POAG increased resistance within the drainage channels causes the rise in IOP

Resistance to aqueous outflow lies in the dense juxtacanalicular trabecular meshwork or the endothelium lining wall of schlemm's canal

Additional contributing factor include increased suseptibility of the optic nerve to ischemia, reduced or dysregulated blood flow excitotoxicity autoimmune reaction and other abnormal physiological processes.



# PRIMARY ANGLE CLOSURE GLAUCOMA

Is a condition in which closure of the angle by peripheral iris results in a reduction in aqueous outflow

The disease affects 1 in 1000 adults over the age of 40 years and occurs in four times as many females as males

Angle Closure Glaucoma: The angle formed by the cornea and the iris narrows, preventing the aqueous humor from draining out of the eye. This can lead to a rapid increase in intraocular pressure. Source: The Mayo Clinic (www.mayoclinic.com)

C Mayo Foundation for Medical Education and Research. All rights reserve

## Pathophysiology-PACG

In PACG the rise in IOP is caused by a decreased outflow of aqueous humor, due to closure of the chamber angle by the peripheral iris

# SECONDARY OPEN ANGLE GLAUCOMA

Decreased outflow of aqueous results from other CAUSES

Psedoexfoliative glaucoma Pigmentary glaucoma Topical Corticosteroids Phacolytic glaucoma

#### PAEDIATRIC GLAUCOMA CAN BE CONGENITAL OR DEVELOP DURING INFANCY OR LATER CHILDHOOD.

#### **CLINICAL PRESENTATION**

- Open -angle glaucoma is slowly progressive and is usually asymptomatic until the onset of substantial visual field loss.
- Visual field defects include

>

General peripheral visual field constriction Isolated scotomas or blind spots Nasal step Enlargement of blind spot Arc like scotomas Reduced contrast sensitivity Reduced peripheral acuity Altered colour vision

In closed angle glaucoma, patients typically experience intermittent prodromal symptoms

- Blurred vision
- Hazy vision with halos around lights
- Headache

Acute episodes produce symptoms associated with a cloudy. Edematous corna ocular pain nausea vomiting and abdominal pain and diaphoresis

#### Diagnosis of Glaucoma

- 1. Intraocular pressure (IOP) and its measurement. (tonometry)
- > 2. Optic disc examination.
- 3 Visual Field examination (perimetry)

TREATMENT OF OCULAR HYPERTENSION AND OPEN ANGLE GLAUCOMA

- Treatment is indicated for ccular hypertension if the patient has a significant risk factors
- ► IOP greator than 25mmHg
- vertical cup disc ratio greator than 0.5
- central corneal thickness less than 555µm.
- family history of glaucoma
- sever myopia, and presence of only one eye.
- Treatment is indicated for all patients with elevated IOP and characteristic optic disc changes or visual field defects

## **B** adrenergic blocking agents

Reduces aqueous production by ciliary body

Betaxolol 0.5% 1 drop bid
0.25% 1 drop bid

Cartelol 1% 1 drop bid

Levobunolol 0.25, 0.5%

Metipranolol 0.3%

Timolol 0.25, 0.5%

1 drop bid

1 drop bid

1 drop qd or bid

Non specific adrenergic agonist increases aqueous humor outflow

Epinephrine0.5%1 drop bidDipivefrin0.1%1 drop bid

<u>Alpha adrenergic agonist</u>
Reduces aqueous humor production
Increase uveoscleral outflow

Apraclonidine Brimonidine

0.5% 1 drop tid0.15% 1 drop bid

#### Prostaglandin analogues

increased aqueous uveoscleral outflow and to a lesser extent , trabecular outflow

| Latanoprost<br>1 drop q h s | 0.0005% | 1 drop q h s bimatoprost | 0.035 |
|-----------------------------|---------|--------------------------|-------|
| Travoprost                  | 0.004%  | 1 drop q h s             |       |

| Carbonic anhydras                     | <u>e inhibit</u> | ors              |               |  |  |  |
|---------------------------------------|------------------|------------------|---------------|--|--|--|
| reduces aqueous p                     | roductio         | n by cilia       | ry body       |  |  |  |
| Brinzolamide<br>Drozolamide           | 1%<br>2%         | bid or<br>bid or | r tid<br>tid  |  |  |  |
| Systemic carbonic anhydrase inhibitor |                  |                  |               |  |  |  |
| Acetazolamide<br>day                  | 125 to           | o 250 mg         | 2to 4 times a |  |  |  |
| Dichlorphenamide<br>day               | 25to 5           | 0 mg             | 1to 3 times a |  |  |  |
| Methazolamide<br>day                  | 25 to            | 50 mg            | 2 to 3 tmes a |  |  |  |

#### Cholinergic agonist

- increased aqueous humor outflow through trabecular
- Carbachol 0.75, 1.5,2.25, 3% 1 drop bid or tid
- pilocarpine 0.25, 0.5, 1, 2, 4, 6, 8, 10% 1 drop bid or qid.

# TREATMENT OF CLOSED ANGLE GLAUCOMA

Requires rapid reduction of IOP.

Iredectomy is the definitive treatment, which produces a hole in the iris that permits aqueous flow to move directly from posterior to anterior chamber

Drug therapy of an acute attack typically consist of an osmotic agent and secretory inhibitors ( eg. Beta blockers , alpha 2 agonist latanoprost, or CAI)with or without pilocarpine

# TREATMENT OF CLOSED ANGLE GLAUCOMA

- Osmotic agent are used because they rapidly decrease IOP
- Eg: GLYCERINE 1to 2 g /kg orally

Mannitol 1to 2g/kg iv

Topical corticosteroids can be used to reduce ocular inflammation

- Argon laser trabeculoplasty
- This treatment involves the application of laser energy (usually argon green) to the trabecular meshwork, thereby improving the rate of outflow of aqueous humour.



(www.glaucoma-association.com)

Surgical Procedures for Glaucoma

Trabeculectomy ( with or without antimetabolites

> Cataracts may progress rapidly after trabeculectomy.

