A Dissertation On

A CLINICAL STUDY ON THE AETIOPATHOGENESIS OF GLAUCOMA ASSOCIATED WITH TRAUMA

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CERTIFICATE

This is to certify that this dissertation titled "A CLINICAL STUDY ON THE AETIOPATHOGENESIS OF GLAUCOMA ASSOCIATED WITH TRAUMA" submitted by **Dr. T.G. UMAMAHESWARI** appearing for Part II M.S Branch III (OPHTHALMOLOGY) degree examination in March 2008 is a bonafide record of work done by her under our direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu, Dr. M.G.R. Medical University, CHENNAI, TAMILNADU.

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CONTENTS

A CLINICAL STUDY ON THE AETIOPATHOGENESIS OF GLAUCOMA ASSOCIATED WITH TRAUMA

INTRODUCTION

Definition of Glaucoma

Glaucoma is a chronic, progressive optic neuropathy caused by a group of ocular conditions which lead to damage of the optic nerve with loss of visual function. The most common risk factor known is raised intraocular pressure.

Traumatic glaucomas are secondary open or closed angle glaucomas which represent a very hetrogenous group of entities due to a variety of pathogenetic mechanisms which increase the intraocular pressure in the early or late phase after trauma (blunt or penetrating injury, acid or alkali burns, thermal injury, radiation damage).

Hyphema, lens associated mechanisms, inflammation and angle recession are the most common causes of traumatic glaucoma after blunt trauma.

Secondary angle closure glaucoma due to Peripheral anterior synechiae is the most common pathogenetic mechanism leading to glaucoma in patients with penetrating eye injuries and chemical burns.

So understanding the pathogenesis of intraocular pressure elevation is critical in selecting the appropriate management of glaucoma associated with ocular trauma.

CLASSIFICATION OF GLAUCOMA

There are several systems by which the glaucomas can be classified. The two most commonly used are based on

1. **Etiology** - the underlying disorder that leads to an alteration in the aqueous humor dynamics.

2. **Mechanism** - the specific alteration in the anterior chamber angle that leads to a rise in the intraocular pressure.

1. CLASSIFICATION BASED ON ETIOLOGY

(a) **PRIMARY:** Confined to the anterior chamber angle or conventional outflow pathway, with no apparent contribution from other ocular or systemic disorder.

(b) **SECONDARY:** Partial understanding of the underlying predisposing ocular or systemic events.

2. CLASSIFICATION BASED ON MECHANISM

(a) OPEN ANGLE GLAUCOMA: Those in which the anterior chamber angle structures i.e. the trabecular mesh work, ciliary body band are visible by Gonioscopy. The elements obstructing the aqueous outflow may be located on the anterior chamber side of the trabecular mesh work (pretrabecular) within the trabeculum (trabecular) or distal to the mesh work in the Schlemm's canal or further along the aqueous drainage system (post trabecular mechanism).

(b) ANGLE CLOSURE GLAUCOMA: This includes situations in which the peripheral iris is in opposition with the trabecular meshwork or the peripheral cornea. The peripheral iris may either be pulled (anterior mechanism) or pushed (posterior mechanism) into this position.

(c) DEVELOPMENTAL ANOMALIES OF THE ANTERIOR CHAMBER ANGLE

AQUEOUS HUMOR OUTFLOW AND ANATOMY OF ANTERIOR CHAMBER ANGLE STRUCTURES

AQUEOUS HUMOR OUTFLOW

The ciliary epithelium produces the aqueous humor which enters the posterior chamber, passes through the pupil into the anterior chamber and leaves the eye by two main routes:

(a) Conventional or canalicular system – accounts for 83% to 90% of aqueous outflow. It consists of the following:

(1) Trabecular meshwork

(2) Schlemm's Canal

(3) intrascleral or collector channels and

(4) episcleral and conjunctival veins

(b) **Unconventional or uveoscleral and uveo-vortex system –** accounts for 5 – 15% of aqueous outflow.

ANATOMY OF THE ANGLE STRUCTURES

1. **Scleral spur** - On gonioscopy it appears as thin prominent white line that is the posterior portion of the scleral sulcus. It may be obscured from view by dense uveal meshwork. It represents the location for attachment of the corneoscleral meshwork anteriorly and the ciliary body (longitudinal muscle) posteriorly.

2. **Trabecular Meshwork** – It is a sieve like structure in the angle of the anterior chamber which converts the scleral sulcus into a circular channel called schlemm's canal and is divided into three portions.

(a) **Uveal meshwork** - It is the innermost portion arranged in rope – like trabeculae that extend from iris root to the schwalbe's line. The intertrabecular spaces are relatively large and offer little resistance to the passage of aqueous (25 – 75

openings).

(b) **Corneoscleral mesh work** – It forms the middle portion which extends from the scleral spur to Schwalbe's line and consists of trabecular sheets perforated by elliptical openings (5 – 50

(c) **Juxta Canalicular tissue –** It is the outermost portion which consists of connective tissue lined on either side by endothelium. Outer layer comprises the innerwall of Schlemm's Canal, while inner layer in continuous with the remainder of the trabecular endothelium. It offers the major proportion of resistance to aqueous outflow.

Gonioscopically, the trabecular meshwork consists of the following two parts:

(1) The anterior nonfiltering, nonpigmented part lies posterior to the schwalbe's line.

(2) The posterior, filtering pigmented part lies adjacent to the scleral spur.

3. Schlemm's Canal

It is an endothelial – lined. circular channel that runs circumferentially around the globe with a diameter of 190 – 370

4. Collector Channels: They arise from the outer wall of the Schlemm's canal and drain into intrascleral, episcleral and conjunctival venous plexus.

5. Schwalbe's Line – Located at the termination of Descemet's membrane. It consists of a circumferential ring of collagenous fibres and basement membrane material. It marks the forward limit of the anterior chamber angle structures and serves as the anterior attachment site for the trabecular mesh work.

MAJOR HISTORICAL LANDMARKS

1882	Priestley Smith	First noted glaucoma following blunt trauma.
1890	Heinz	Described Heinz bodies
1892	Collins	Pathologic description of Angle recession
1915	Clegg	Secondary open angle glaucoma following siderosis due to IOFB
1926	Mayon	
1947	Lowenstein and Foster	
1919	Fuchs	First described Ghost cells
1960	Vannas	Hemosiderotic glaucoma in eyes with intraocular hemorrhage
1962	Wolf and Zimmerman	Correlated traumatic glaucoma and angle recession.
1963	Fenton and Zimmerman	Described hemolytic glaucoma
1963	Rodman	Described traumatic glaucoma and its various causes
1976	Campbell, Simmons and Grant	Ghost cell glaucoma

AETIOPATHOGENESIS OF GLAUCOMA ASSOCIATED WITH TRAUMA

Glaucoma secondary to trauma can be caused by

(1) Contusion injuries

(2) Penetrating injuries

(3) Chemical injuries

(4) Thermal burns

(5) Electric shock and

(6) Radiation damage

Traumatic Glaucoma can be either open or closed angle and can occur in the early or late post traumatic period.

(1) MECHANISM OF RAISED INTRAOCULAR PRESSURE (IOP) IN CONTUSION INJURIES

- Trauma to the trabecular meshwork
- Inflammation
- Hyphema
- Lens swelling with pupillary block

- Lens subluxation with pupillary block
- Angle recession
- Ghost cell glaucoma and hemolytic glaucoma
- Peripheral anterior synechiae following trabecular tear
- Posterior synechiae with pupillary block
- Vitreous filling the anterior chamber
- Uveal effusion and angle closure (rarely)
- Neovascular glaucoma

(2) MECHANISMS OF RAISED IOP IN PENETRATING INJURIES

Penetrating injuries can induce elevated IOP through various mechanisms. These include

- Flat anterior chamber with formation of peripheral anterior synechiae
- Inflammation
- Intraocular hemorrhage including hyphema and ghost cell glaucoma
- Lens swelling with pupillary block

- Lens subluxation with pupillary block
- Lens-particle glaucoma
- Vitreous filling anterior chamber
- Phaco anaphylaxis
- Posterior synechiae with pupillary block
- Epithelial downgrowth
- Fibrous ingrowth
- Retained intraocular foreign body

a. Organic - Inflammatory glaucoma

b. Iron - Siderosis

c. Copper - Chalcosis

(3) MECHANISM OF RAISED IOP FOLLOWING CHEMICAL BURNS

Ocular chemical injuries, especially with alkalis and to a lesser extent with acids, frequently have devastating consequences for vision and the integrity of the globe. Intraocular pressure alteration occurs in a relatively characteristic pattern of an initially elevated pressure, followed by a period of hypotony, and ultimately elevated pressure in the intermediate to late phase of the disease.

(a) Early Phase

Possible mechanisms of the initial intraocular pressure elevation include anterior segment shrinkage and increased uveal blood flow, which may be prostaglandin-mediated. Anterior chamber inflammation, with hypopyon in more severe cases, may develop and contribute to the pressure rise.

Management

Treatment for ocular hypertension in the early phase is limited to

2 – adrenergic agonists, carbonic, anhydrase inhibitors, and hyperosmotics.

However, because reepithelialization of the ocular surface may be impaired by topical medications, systemic medications may be preferred.

Miotics are relatively contraindicated, as they may aggravate anterior segment inflammation.

(b) Intermediate Phase

The intermediate phase (weeks to months) is characterized by repair, scarring and ongoing inflammation. The causes of raised intraocular pressure (IOP) include irreversible trabecular meshwork damage, peripheral anterior synechiae formation from inflammation and pupillary block by posterior synechiae.

Management

Inflammation should be treated with oral corticosteroids. Topical corticosteroids are relatively contraindicated because of the risk of stromal lysis. If glaucoma is caused by pupillary block, medical management including vigorous cycloplegic/mydriatic therapy should be attempted. If medical treatment fails, however, laser iridotomy should be performed. If the cornea is not sufficiently clear to allow laser iridotomy, an incisional iridectomy should be performed. If the lens causes the elevation of intraocular pressure, cataract extraction should be considered.

(c) Late Phase

Elevation of intraocular pressure in the late phase of ehcmical injury is usually caused by trabecular damage and peripheral anterior synechiae formation.

Patients often present with scarred corneas, making intraocular pressure, optic disc, and visual field evaluation problematic. The pressure may be measured best with a Tono-Pen or pneumotonometer. (When the ocular injury is unilateral, these devices should be calibrated against a Goldmann applanation tonometer in the fellow eye.

Management

Medical therapy may successfully moderate intraocular pressure, but filtering surgery may be required. Extensive conjunctival scarring may make conventional filtering surgery impossible, in which case glaucoma shunting procedures (or alternatively cyclodestructive procedures) should be considered.

4. THERMAL BURNS

Rarely produce rise in IOP by orbital congestion and massive

periorbital swelling.

5. ELECTRICAL INJURY

Transient intraocular pressure elevation has been reported after accidental and therapeutic electrical injuries such as electroshock therapy and cardioversion.

The pressure spike may be associated with loss of iris pigment epithelium, which is evident on slit-lamp retroillumination. Venous dilation and contraction of the extraocular muscles also may be involved in the pathogenesis of the intraocular pressure alteration.

Treatment is seldom required because the pressure elevation is typically transient.

6. RADIATION INJURY

When radiotherapy is used to treat intracranial, periocular, or intraocular neoplasms, elevated intraocular pressure may result. Possible pathogenic mechanisms include:

(1) elevated episcleral venous pressure related to generalized telangiectasia of the conjunctiva after anterior segment

irradiation,

(2) neovascular glaucoma caused by radiation-induced iris and anterior chamber angle neovascularization, and

(3) hemolytic changes associated with intraocular hemorrhage.

Because of the severity of the underlying ocular disease and radiation damage in many of these cases the visual prognosis is poor, even with medical and surgical management of the glaucoma.

A. CAUSES OF OPEN ANGLE GLAUCOMA FOLLOWING TRAUMA

Early causes	**Late Causes**
1. Hyphema	1. Angle Recession
2. Inflammation	2. Ghost cell glaucoma
3. Lens particle glaucoma	3. Hemolytic glaucoma
	4. Hemosiderotic glaucoma

B. CAUSES OF ANGLE CLOSURE GLAUCOMA FOLLOWING TRAUMA

Early Causes

1. Lens Subluxation with pupillary block

2. Lens Dislocation with pupillary block

3. Intumescent Lens

4. Flat AC (anterior chamber) leading to Peripheral Anterior Synechiae

5. Uveal Effusion

I. EARLY CAUSES OF POST TRAUMATIC GLAUCOMA

1. HYPHEMA - "Blood in AC"

Blunt trauma to the eye can be associated with anterior segment injuries, including hyphema, iris sphincter tear, iridodialysis, cyclodialysis, trabecular tear, inflammation and zonular rupture and lens subluxation.

PATHOPHYSIOLOGY IN BLUNT INJURY

Blunt injury caused by object small enough or sufficiently deformable to fit inside the rim of the orbit in order to strike the globe

and indent the anterior surface of the eye. This causes stretching of the limbal tissue, equatorial scleral expansion, posterior and peripheral movement of the aqueous, posterior displacement of the lens-iris diaphragm, and acute rise in IOP.

All these cause tearing of the tissues near AC angle. 90 percent of hyphema is from tear in the anterior face of the ciliary body.

The usual source of bleeding is a tear in the anterior face of the ciliary body between the longitudinal and circular ciliary muscle fibers which leads to disruption of branches of major arterial circle causing bleeding into the AC (also from recurrent choroidal arteries or ciliary body veins).

In small percentage of cases the bleeding is from ruptured iris vessels, cyclodialysis or iridodialysis. As the IOP rises bleeding diminishes and a clot forms. Clot lysis and retraction occur 1 to 2 days after the injury and maximal incidence of rebleeding from the injured vessels occurs at this time. The rebleed is often more severe than the initial episode and can lead to a total hyphema, also known as "black ball" or "eight ball" hyphema. Unlike the typical initial bleeding episode, total hypema is initially associated with extreme pain, nausea

and other symptoms related to acute glaucoma.

PATHOPHYSIOLOGY IN PENETRATING INJURY

Direct damage to the vessels or rarely due to sudden drop in IOP.

COURSE OF HYPHEMA

Bleeding stops by IOP tamponade, vascular spasm, formation of fibrin or platelet clot. Maximum integrity of the clot occurs between 4-7 days. Main pathway out of anterior chamber is trabecular mesh-work for degraded products. Incidence of secondary hemorrhage or rebleeding is 3.8 to 38 percent and mostly occurs in 2 to 5 days. Nearly all occur before the 7^{th} day.

Pathology of rebleeding is fibrinolysis and clot retraction or bleeding from fragile new capillaries.

CLASSIFICATION OF TRAUMATIC HYPHEMA

According to

Type of Hemorrhage

Primary

Secondary

Continuous

Volume (microscopic) – No layered blood, only circulating RBC.

Grade I	-	< 1/3 of anterior chamber
Grade II	-	1/3 – 1/2 of anterior chamber
Grade III	-	> 1/2 of anterior chamber
Grade IV	-	total

Duration

Acute	-	1-7 days
Subacute	-	7-14 days
Chronic	-	14 days

Character

Liquid	-	Red
Clotted	-	Brown or black or mixed
Organised	-	Tan, grey or white

MANAGEMENT

Medical Management

Conservative treatment of elevated IOP – drugs that reduce aqueous formation resulting in sufficient lowering of the pressure so as to allow gradual resorption of hyphema.

Improvement is signalled by the appearance of a mixture of brighter blood and the aqueous near the upper limbus.

If no further rebleeding occurs, total resorption of the residual blood usually occurs in 5 to 7 days.

1. Pad and bandage to protect from further injury.

2. Topical cycloplegics to relieve ciliary spasm, prevent posterior synechiae and to view the posterior segment. Topical steroids to reduce significant anterior chamber inflammation.

3. Topical

–blockers, and systemic carbonic anhydrase inhibitors to reduce the IOP.

4. In acute raise in IOP, IV mannitol or oral glycerol should be given. Monitor the patient every 12 hours. IOP should be lowered to a level of 24 mm Hg. Acetazolamide and epinephrine are contraindicated in sickle cell hemoglobinopathies.

5. Aspirin and other nonsteroidal antiinflammatory drugs

(NSAIDs) are avoided.

6. Aminocaproic acid or Tranexamic acid: Oral antifibrinolytic agent dose-50 mg/kg body weight every 4 hours for 5 days upto 30 gm per day.

7. Intracameral tissue plasminogen activator.

Surgical Management

Indications

1. The presence of microscopic corneal bloodstaining

2. Total hyphema with an intraocular pressure of 50 mm Hg or more for five days or of 35 mm Hg for seven days.

3. Total hyphema that does not resolve below 50 percent by six days when associated with an intraocular pressure of 25 mm Hg or more since it is more likely to result in corneal bloodstaining.

4. Hyphema that remains unresolved for nine days because it probably will result in the formation of anterior synechiae.

Methods

1. Paracentesis is done to let out the blood. If needed AC wash is done in restricted cases.

2. Clot expression – entire clot does not need to be removed, as only the circulating red blood cells obstruct the outflow pathways

3. Automated hyphemectomy

4. Ultrasoic emulsification

5. Trabeculectomy.

COMPLICATIONS OF HYPHEMA

(1) Rebleeding

If rebleeding occurs, it may be wise to try to find the source of the continuing or recurrent hemorrhage.

Secondary hemorrhages may be anticipated between the second and seventh days after trauma.

One-third of all secondary hemorrhages progress to total hyphema, and the rate of recovery to normal under these circumstances is only 36 percent.

(2) Blood Staining of Cornea

Glaucoma following a traumatic hyphema can increase the chance of blood-staining; however, it should be understood that bloodstaining can occur in the presence of a normal tension if

(1) hyphema persists for a long while,

(2) the endothelium is damaged, or

(3) there is a high concentration of blood in the anterior chamber.

Blood staining of cornea will usually clear, although it may take months.

(3) Posterior Synechiae Formation

The formation of posterior synechiae is a complication that may be noted in patients with traumatic hyphema.

This problem is caused by prolonged iritis and/or by organization of the clot in the anterior chamber.

However, the tendency for synechiae to occur is greater in patients who have undergone surgery for the hyphema.

2. INFLAMMATION

Inflammation by trauma can produce glaucoma through a variety of mechanisms including:

- Increased viscosity of aqueous humor
- Obstruction of the trabecular meshwork by inflammatory cells and debris
- Swelling and dysfunction of the trabecular meshwork.
- Liberation of active substances such as prostaglandins and substance P.

In acute inflammation the IOP is low due to reduction in aqueous production, but later the production normalizes but the outflow facility remains low thereby increasing the IOP.

Treatment - Topical cycloplegics and steroids, usually helps to resolve the inflammation and decrease the IOP. Systemic steroids

employed with possible adverse side effects must be accepted in severe cases. Rarely a retinal detachment related to traumatic retinal dialysis causes inflammatory changes in the anterior segment leading on to increased IOP after several months. Though rare it is an important consideration in differential diagnosis since surgical treatment can cure this rare type of glaucoma.

3. LENS INDUCED GLAUCOMAS

(a) Lens Dislocation or Subluxation

Dislocation or subluxation of the crystalline lens most commonly occurs with blunt trauma. Disruption of the zonules allows the lens to move anteriorly or posteriorly. Depending on the position of the lens and vitreous status, pupillary block may occur. Pupillary block may lead to angle closure with a precipitous increase in IOP. Creeping angle closure may develop in a more sub-acute or chronic form, leading to late onset development of glaucoma. Rarely, complete dislocation of the lens may allow vitreous to come forward to fill the anterior chamber and lead to vitreous block glaucoma.

Treatment

Treatment depends on the type of glaucoma presenting.

Careful examination is critical to determine the presence or absence of pupillary block. If pupillary block is present, a laser iridotomy or surgical iridectomy is indicated.

Lensectomy may be required for visual reasons and for recurrent episodes of pupillary block.

The technique of lensectomy will depend on the degree of subluxation.

Surgical removal may involve a planned extracapsular extraction or phacoemulsification approach, or pars plana lensectomy with vitrectomy.

(b) Lens Swelling

A blunt trauma can cause traumatic cataract and sudden swelling of the lens causing pupillary block. A sudden increase in intraocular content may also lead to glaucoma. Treatment is removal of the lens.

(c) Lens Particle Glaucoma

Disruption of the lens capsule by penetrating trauma liberates lens material which can obstruct the trabecular meshwork. The resulting glaucoma depends on the amount of lens materials liberated, the inflammatory response of the eye and the ability of the trabecular mesh work to clear the foreign matter.

If tension is not relieved, surgical intervention should be considered.

4. FLAT ANTERIOR CHAMBER LEADING TO FORMATION OF PERIPHERAL ANTERIOR SYNECHIAE

In case of penetrating injury if the wound closure is delayed and if the anterior chamber is not formed, peripheral anterior synechiae forms leading to angle closure glaucoma. This is also seen in case of poor wound closure by a poor surgical technique.

5. UVEAL EFFUSION

Uveal effusion may lead to angle-closure glaucoma in small eyes. Also a post traumatic uveal inflammation causes obstruction to outflow

by inflammatory cells, debris, protein. Topical steroids will settle the problem.

II. LATE CAUSES OF POST TRAUMATIC GLAUCOMA

1. ANGLE RECESSION

Pathogenesis

At the moment of impact aqueous is forced laterally and posteriorly against the iris and angle. These hydrodynamic forces cause a tear between the longitudinal and the connecting circular and oblique muscle. This may disrupt branches of anterior or posterior ciliary arteries resulting in bleeding into the anterior chamber.

Eyes with unusual wide angle may be more prone to develop angle recession than eyes with narrow angle.

In years following trauma the inner circular radial muscle may atrophy. On microscopic examination the trabecular meshwork undergoes degenerative changes with resultant scarring. There is fibrosis and obliteration of the intertrabecular spaces and Schlemm's canal. This

leads to decreased outflow facility.

Damage to the ciliary muscle disrupts the tension exerted on the trabecular meshwork and may reduce the functional capacity of the meshwork.

CLASSIFICATION OF ANGLE RECESSION

(i) Shallow

Shallow angle recession consists of a separation of the iris processes from the meshwork such that the ciliary body band and scleral spur are more visible compared to the fellow eye. Here no actual traumatic cleft in the ciliary body occurs.

(ii) Moderate Tear

A cleft appears in the ciliary body corresponding to the separation of the longitudinal and circular muscle fibers.

(iii) Deep Tear

A fissure extends deeper into the ciliary body. Moderate and deep tears can be visualized with anterior segment ultrasound biomicroscopy.

Patients with moderate to severe tears often proceed to sealing of these clefts with formation of peripheral anterior synechiae and fibrosis in the angle, obscuring the evidence of angle recession later.

Eyes with less than 180° recession are unlikely to develop late glaucoma. But this is not a rule. Even a smaller recession may give rise to glaucoma.

Increase in IOP occurs after 6 weeks and upto 8 percent of eyes with 180º or more of recession may eventually develop glaucoma.

In patient with angle recession glaucoma lifetime risk of developing primary open-angle glaucoma (POAG) in the fellow non-traumatized eye may be as high as 50 percent.

Mechanism

The mechanism for glaucoma in angle recession depends on the timing of the IOP elevation after injury. Early IOP increase is due to trabecular inflammation and the presence of circulating blood and

inflammatory products. This often resolves within weeks to months, and the patient may enter a "honeymoon period", with normalization of IOP. The ophthalmologist must be aware that this period of normal IOP can be short lived; such eyes deserve close follow-up, at least every 6 months.

Late angle recession glaucoma is secondary to the formation of a "glass membrane" over the trabecular meshwork, thought to be an extension of Descemet's membrane.

CLINICAL FEATURES

- **Anterior chamber** - deep
- **Evidence of injuries** – Iris sphincter tear, iris atrophy, iridoschisis, iridodonesis, heterochromic iridis, traumatic mydriasis, anterior subcapsular cataract, vossius ring, phacodonesis, etc.
- **Gonioscopy** – Asymmetry of the angle of both eyes or in different portions of the angle in the involved eye. Broad ciliary body band seen with retro-displacement of the iris root, including the

ciliary processes and circular ciliary muscle.

TREATMENT

Initial therapy is medical – effective in eyes with small degrees of angle recession. Surgical management is difficult.

Laser trabeculoplasty – limited success for short term.

Nd: YAG laser trabeculopuncture is an additional modality for selected cases.

Trabeculectomy bleb fibrosis in more common. So antifibrotic agents are coupled with the conventional surgery.

In failed cases – implant surgery can be done.

2. GHOST CELL GLAUCOMA

Pathophysiology

There are three important steps in the pathophysiology of ghost cell glaucoma:

(1) erythrocyte degeneration within the vitreous (regardless of the site of origin of the blood within the vitreous),

(2) a port of communication between the vitreous and anterior segment through a disruption of the anterior hyaloid face, and

(3) trabecular obstruction by ghost cells because of their unusual rheologic characteristics.

After the erythrocytes reach the vitreous cavity, they undergo morphologic, calorimetric, rheologic changes. The RBCs degenerate from red, biconcave, pliable cells to tan or khaki colored, spherical, and hollow, less pliable "Ghost cells". Within one to three weeks, these changes occur and they remain in the vitreous cavity for months.

The intracellular hemoglobin is lost presumably through leaky membranes into the extracellular vitreous space. During the conversion

the hemoglobin that remains within the cell, denatures and forms clumps called Heinz bodies, which adhere to the inner surface of the plasma membrane.

The extracellular hemoglobin forms clumps or large accumulations that tend to adhere to vitreous strands. In contrast, the ghost cells do not adhere to each other or to the strands and are free to move anteriorly.

The anterior hyaloid face serves as a natural boundary for the products of the hemorrhage. In trauma, disruption in the anterior hyaloid face, allows the ghost cells to come into the anterior chamber, which in turn obstruct the trabecular meshwork causing glaucoma (the ghost cells are less pliable and cannot pass through human trabecular meshwork with ease.)

Clinical Features

Patient usually presents with a history of trauma 2 to 3 weeks earlier, with poor vision and pain, in the presence of an uninflammed or a mildly inflammed eye. Occasionally an 8-ball hyphema is complicated by ghost cell glaucoma in association with vitreous hemorrhage. Conjunctiva is generally white, unless the IOP is very high. Cornea may be normal or edematous or may have collections of khaki-colored cells at its back. Anterior chamber – the aqueous humor is typically filled with a multitude of tiny, tan colored cells, often circulating slowly. If fresh blood also co-exists, a double layer precipitate of light, khaki on top of red is present called "candy-stripe" sign which is pathognomonic of ghost cell glaucoma.

Gonioscopically angle is wide open with discolored trabecular meshwork due to the presence of fine layer of khaki-colored cells. Sometimes a pseudohypopyon is seen in the inferior angle.

Differential Diagnosis

Neovascular glaucoma, Hemosiderosis.

Management

Vitreous examination shows khaki-hue of degenerated hemorrhage and aqueous tap examined under magnification shows Heinz bodies. In milder cases, standard medical therapy including β–blockers and carbonic anhydrase inhibitors will suffice to lower the IOP. In few cases, repeated irrigation of anterior chamber to remove the ghost cells and in turn to lower the IOP is required. If the vitreous hemorrhage is large, vitrectomy with special attention to remove all ghost cells including those at the base, otherwise vitrectomy itself will further increase the IOP. Since ghost cell glaucoma is not inflammatory, topical steroids are ineffective. Ghost cell glaucoma is typically transient. As there is no permanent damage to the trabecular meshwork, prognosis is fairly good.

3. HEMOLYTIC GLAUCOMA

Hemolytic glaucoma is an open-angle glaucoma that occurs within days to weeks after a large intraocular hemorrhage.

The elevated intraocular pressure is associated with hemolytic debris, including hemoglobin-filled macrophages, which obstruct the trabecular meshwork.

Reddish-brown blood cells are evident in the aqueous humor with slit-lamp examination. Gonioscopy reveals an open angle without neovascularization, but the trabecular meshwork is covered with reddish-brown pigment, especially inferiorly.

The diagnosis is confirmed by cytologic examination of the aqueous, which characteristically shows macrophages containing golden-brown pigment rather than ghost cells.

Mechanism

The mechanism of intraocular pressure elevation is an obstruction of the trabecular meshwork by macrophages laden with pigment, erythrocytes, and debris. An ultrastructural study has demonstrated degenerative changes in trabecular endothelial cells, which also had phagocytized blood.

Management

Hemolytic glaucoma is typically a self-limited condition that responds to medical management with β–adrenergic antagonists, α_2-adrenergic agonists, carbonic anhydrase inhibitors, and hyperosmotic agents. Recalcitrant cases may require surgical intervention, such as

anterior chamber washout.

4. HEMOSIDEROTIC GLAUCOMA

Hemosiderotic glaucoma is a rare condition associated with long-standing intraocular hemorrhage, the exact mechanism of which is unclear. Hemoglobin released from degenerated erythrocytes is phagocytized by endothelial cells of the trabecular meshwork. The iron liberated by the hemoglobin may cause siderosis of the trabecular meshwork, eventually resulting in decreased aqueous outflow.

5. RETAINED INTRAOCULAR FOREIGN BODY (IOFB)

The increased IOP in patients with intraocular foreign body may be due to:

(a) a consequence of direct trauma to ocular structures

(b) secondary to the effects of blunt trauma on the trabecular meshwork

(c) as a late sequela of a retained metallic foreign body.

Presence of a tear in the lens capsule with sealed corneal wound,

with or without phacogenic glaucoma, presence of an iris hole or transilluminaton defect in the iris, direct visualization of foreign body by slit lamp examination or ophthalmoscopy are few clinical features of retained IOFB. Appropriate radiographic studies, ultrasonography, computerized axial tomography and at times by using pediatric dental X-ray cassette lodged lengthwise in the medial canthus may confirm the clinical suspicion.

The immediate increase in IOP may be due to

- Direct and indirect effect of injury
- Phacogenic (Lens induced)
- Flattening of AC with inflammation leading to peripheral anterior synechiae.

Siderosis

Late increase in IOP may be due to iron deposition in the trabecular meshwork of eyes with siderosis – leading to decrease in aqueous outflow and secondary glaucoma.

Uniocular glaucoma after injury, heterochromia, mydriasis, rust

colored stain to posterior corneal surface, multiple rust colored anterior subcapsular lens deposits are the classical clinical signs of siderosis bulbi.

Chalcosis

Copper also may be oxidized within the eye, with tissue damage that is nearly as severe as that encountered with ferrous foreign bodies. glaucoma is apparently less frequently associated with chalcosis than with siderosis, although the retinal damage caused by copper may cause visual field defects that mimic glaucomatous changes.

6. POSTERIOR SYNECHIAE WITH PUPILLARY BLOCK

Following chronic inflammation due to injury or retained foreign body, dense posterior synechiae forms, leading to seclusio pupillae, iris bombe and peripheral anterior synechiae resulting in secondary angle-closure glaucoma.

7. EPITHELIAL INGROWTH

Lacerating trauma to the eye may lead to a fistula that develops

from the external surface of the eye to the anterior chamber. This provides a conduit for epithelium to enter the anterior segment. This is an uncommon problem in the setting of microsurgical repair, but must be searched for in eyes that develop glaucoma at a time remote from the initial lacerating injury.

The characteristic slit lamp appearance of a scalloped endothelial membrane is diagnostic for epithelial downgrowth. Argon laser application to the involved iris makes a white spot. These two findings confirm the diagnosis, although histopathologic confirmation via iris biopsy may be required in certain cases.

Treatment involves excision of all involved iris and explantation of the intraocular lens, if present. Cryotherapy is applied to the involved ciliary body and cornea. Visual prognosis is guarded in these cases. Tube-shunt procedures may also be effective.

8. FIBROVASCULAR DOWNGROWTH

This is due to difficult surgery, incarceration of material in the wound, poor wound closure and chronic inflammation.

Fibrous membrane located adjacent to the wound extends on the

posterior corneal surface and extend over the angle, iris and vitreous seen as a thick enveloping membrane.

In the angle fibrovascular tissue contracts to form peripheral anterior synechiae.

Mechanism

- Pretrabecular block by membrane
- Peripheral anterior synechiae

9. NEOVASCULAR GLAUCOMA

This type of glaucoma following injury is rare and is usually due to chronic inflammation.

10. RHEGMATOGENOUS RETINAL DETACHMENT

Rhegmatogenous retinal detachment is most commonly associated with ocular hypotension.

However, 5 to 10% of the patients may develop elevated IOP (Schwartz's Syndrome).

The mechanism for increased IOP is most likely due to direct

photoreceptor obstruction of aqueous outflow in the anterior segment.

Schwartz's syndrome can be difficult to diagnose, and commonly occurs in the setting of a shallow, peripheral retinal detachment with the appearance of an anterior chamber inflammatory reaction.

The "cells" in the anterior chamber may be photoreceptor outer segments. It is not unusual for Schwartz's syndrome to be misdiagnosed as inflammatory glaucoma.

It is of particular importance to establish the correct diagnosis of Schwartz's syndrome, as it is a curable entity, with a treatment different from that for inflammatory glaucoma.

Repair of the retinal detachment most commonly results in a prompt and permanent return of IOP to normal.

11. LATE CLOSURE OF A CYCLODIALYSIS CLEFT

A separation of ciliary body from the scleral spur result in a cyclodialysis cleft. This may occur following blunt trauma. This can result in temporary or permanent hypotony. Goldmann has postulated that a reduction in the normal flow of aqueous across the trabecular

meshwork results in a reduced permeability of the meshwork to aqueous outflow. This may account for the marked, acute IOP elevation that can occur following closure of cyclodialysis clefts. The IOP increase tends to be severe and is associated with significant pain.

Treatment – involves use of aqueous suppressants, oral or intravenous hyperosmotics as well as alpha-agonists. Most cases can be managed medically through the short period of time that the IOP is elevated. Miotics are avoided because they can lead to recurrence of the cleft.

PART – II

A CLINICAL STUDY ON THE AETIOPATHOGENESIS OF GLAUCOMA ASSOCIATED WITH TRAUMA

AIM OF THE STUDY

1. To analyse the types of glaucoma associated with trauma.

2. To study the causative mechanism of glaucoma associated with trauma.

MATERIALS AND METHODS

100 patients with glaucoma following trauma who attended the Regional Institute of Ophthalmology, Government Ophthalmic Hospital, Chennai, during the period of June 2005 to October 2007 were taken for the study.

INCLUSION CRITERIA

All patients with raised intraocular pressure following trauma were included in the study. Cases with all types of injuries causing raised IOP were included.

EXCLUSION CRITERIA

1. Patients with pre-existing glaucoma were excluded from the study.

2. Patients who had other pre-existing ocular diseases such as those with anterior segment infection, inflammation were also excluded from the study.

EVALUATION OF THE PATIENT

A thorough ocular examination was done for all the patients which consisted of

(1) A detailed history taking was done which included

(i) Eye affected - Right / Left

(ii) Nature of Injury - Penetrating injury
Blunt injury
Chemical burns
Thermal burns

(iii) Nature of object which caused the injury -

Fist/ball/stone/metal/wood/glass/others

(iv) Time interval between injury and admission to the hospital –

immediate or late

(v) Any pre-existing ocular diseases or surgeries

(2) Examination of the eye injured

External injuries like lid oedena, contusion or tear were looked for and a thorough examination of the anterior segment was done with slit lamp.

The following signs of injury were looked for:

(i) Conjunctiva - Congestion, subconjunctival hemorrhage, chemosis and tear.

(ii) Cornea - Oedema, foreignbody, keratic precipitates, tear, tissue incarceration

iii. Anterior Chamber - Depth - Normal, Shallow, deep or irregular

Clarity - Whether clear, turbid, or with flare or cells,

presence of hypopyon, hyphema, foreign body, vitreous or lens matter.

(iv) Iris - Normal pattern, hole, iridodialysis, anterior or posterior synechiae, incarceration in the wound, new vessels.

(v) Pupil - Normal, miotic, mydriatic, whether reacting to light, sphincter tear.

(vi) Lens - Clear, cataractous, subluxation, dislocation, foreign body, pigments

(vii) Vitreous - Hemorrhage, incarceration in wound

A complete glaucoma work up was done for all the cases which included.

(a) Vision - Right eye and left eye

(b) Tension - Measured by Goldmann applanation (mm Hg) tonometer and noncontact tonometer in selective cases.

(c) Gonioscopy- with Goldman single mirror indirect goniolens.

(d) Visual Field- tested using Automated Perimetry, in cases where it was possible.

(e) Fundus examination- Depending on the degree of visibility, Direct ophthalmoscopy, slit lamp examination with 90 D lens and indirect ophthalmoscopy were done in the patients and the following were looked for –

- Cup disc ratio
- Retinal detachment
- Presence of vitreous hemorrhage
- Presence of any foreign body

All the cases were investigated with

(1) X-ray orbit - to look for foreign body

(2) Ultrasonography - Done for posterior segment evaluation

Ultrasound Biomicroscopy - for selective cases suspected to have angle recession.

ANALYSIS AND DISCUSSION

Age Distribution

Age in years	No. of cases	%
0 – 10	8	8
11 – 20	15	15
21 – 30	28	28
31 – 40	19	19
41 – 50	17	17
51 and above	13	13

On analysing the age distribution, there is a preponderance of traumatic glaucoma in the less than 40 years age group. This is in accordance with other studies which also show a similar distribution.

Sex Distribution

Sex	No. of cases	%
Male	82	82
Female	18	18

Sex distribution showed a preponderance in males which is in accordance with other studies. This reflects their work carrying a greater risk to occupational hazards. In females the commonest cause of injury was found to be domestic and agricultural injuries.

Laterality

Side	No. of cases	%
RE	62	62
LE	38	38

In this study, right eye is involved more than left eye. No cases had involvement of both eyes.

Place Distribution

Area	No. of cases	%
Urban	74	74
Rural	26	26

Place distribution showed that people in the urban area are more affected than in the rural side. This could be because of the study being conducted in a tertiary eye-care centre in the city. Also the most common cause of injury in the urban sector was industrial accidents In the rural population the commonest causes were domestic and agricultural injuries.

Time interval between Injury and admission

Time	No. of cases	%
Immediate (< 1 wk)	70	70
Late (>1 wk)	30	30

Majority of patients presented early after injury. The reason for this being defective vision and severe pain. Among this group, patients with hyphema and lens associated injuries presented in the immediate post traumatic period.

Few patients, inspite of poor vision came late as there was not much symptoms immediately.

Mode of Injury

Mode	No. of cases	%
Industrial	29	29
Agricultural	11	11
Domestic	15	15
Sports	8	8
Assault	10	10
RTA	10	10
Cracker	11	11
Quarry	6	6

Industrial accidents were more common than others, as the main population of the study were from urban area. This is followed by

Domestic and agricultural injuries.

The next common mode of injuries were assault, RTA and crackers.

Trauma due to sports and quarry work are the other modes of injuries.

Type of Injury

Type	No. of cases	%
Blunt	78	78
Penetrating	20	20
Chemical	2	2

Blunt injury is the major type of injury in this study followed by penetrating injury. This may be due to the careless handling of blunt objects without knowing that they are also as dangerous as sharp objects.

Chemical injuries producing rise in intraocular pressure were less commonly seen.

Agent Causing Injury

Agent	No. of cases	%
Metal	30	30
Wood	25	25
Stone	24	24
Fist	7	7
Ball	6	6
Chemical (Alkali)	2	2
Glass	1	1
Tyre	1	1
Others	4	4

Metal is the commonest agent causing injury mainly in industrial accidents.

This is followed by wood which is the agent involved in domestic and agricultural injuries. Wood injuries include stick, thorn, wood pieces etc.

Injury with stone is mainly seen in trauma due to crackers, quarry work and RTA.

Ball is the main agent among sports injuries, as their impact on the globe is more due to their convex surface.

Fist was the common agent in medicolegal cases.

Type of Glaucoma

Type of Glaucoma	No. of cases	%
Open angle glaucoma	49	49
Angle closure glaucoma	51	51

Number of patients with open angle and angle closure glaucoma were almost equal.

The Causative mechanisms of open angle glaucoma

Mechanism	No. of cases	%
Hyphema	25	25
Inflammation	15	15
Angle recession	6	6
Lens Particle	3	3

The commonest cause of open angle glaucoma following trauma is hyphema. These patients present in the immediate post traumatic period due to defective vision.

This is followed by traumatic glaucoma due to inflammation.

Angle Recession and lens particle glaucoma are other less common causes.

The causative mechanisms of angle closure glaucoma

Mechanism	No. of cases	%
Peripheral anterior synechiae	13	13
Anterior dislocation of lens with pupillary block	13	13
Intumescent lens	15	15
Subluxated lens with pupillary block	9	9
Neovascular glaucoma	1	1

Majority of cases with angle closure were contributed by lens induced glaucomas.

They include anteriorly dislocated and subluxated lens causing pupillary block and intumescent lens. All these cases presented early due to severe pain.

The next common cause was formation of peripheral anterior synechiae. This was due to inflammation following blunt trauma.

In penetrating injury, the cause for synechial closure was a shallow anterior chamber.

There was one case of post traumatic neovascular glaucoma which presented very late after the injury. It was due to chronic inflammation. This case was associated with retinal detachment.

B – Scan

Findings	No. of cases	%
Normal	75	75
Only vitreous hemorrhage (VH)	16	16
VH with Retinal Detachment (RD)	2	2
VH with IOFB	2	2
RD	3	3
RD with posterior vitreous detachment	1	1
Dislocated Lens with VH with RD	1	1

25 cases in this study had important posterior segment findings which was detected by B–scan. The commonest problem was vitreous hemorrhage followed by retinal detachment which accounted for the very poor vision in the early post traumatic period.

UBM (Ultrasound Biomicroscopy)

Angle recession was confirmed by UBM in 6 patients in whom it was suspected. All the six cases presented late after injury ranging from 4 weeks to 8 weeks.

Angle Recession

Among the 6 cases of angle recession, 3 cases had recession involving 180º (2 Quadrants), 2 cases had recession with slightly more than 180º and one case had a 270º recession.

In all the six cases, the reason for angle recession was blunt injury by various agents like stone, ball, fist and wood.

SUMMARY

Of the 100 cases taken for the study 78 cases were due to blunt trauma, 20 cases were due to penetrating injuries and 2 were caused by chemical injury.

70% cases were under the less than 40 years age group. This is in accordance with other studies which also show a similar preponderance. It is because of the reason that this group is actively involved in their occupation they are engaged.

Sex distribution showed that 82% were males reflecting their work carrying a greater risk to occupational hazards.

Involvement of right eye is more (62%) than the left eye (38%) in this study. There was no bilateral involvement.

Place distribution showed that people from urban area were mostly affected (74%). The reason for this regional variation could be due to the fact that this study was conducted in a tertiary eye care city hospital.

About 70% patients presented in the immediate post traumatic

period. This could be because of defective vision and severe pain. 30% of them came late as the symptoms were very less in the early post traumatic period.

Industrial accidents were the commonest mode of injury (29%). Because, the majority of cases were from urban area. This was followed by domestic (15%) and agricultural injuries (11%) The other modes of injuries were assault (10%), RTA (10%), Cracker (11%), Sports (8%) and Quarry work (6%).

Blunt injury constituted 78%. This could be due to careless handling of blunt objects by people with less awareness that even blunt objects could be as hazardous as sharp materials. Penetrating injuries accounted for 20% and chemical injury was least common constituting (2%).

Metal is the commonest agent causing injury (30%) mainly in industrial accidents. In Industrial works metal pieces are driven with a tremendous force, which on impinging on the eye produce severe ocular damage.

This is followed by wood (25%) which is the common agent in domestic and agricultural injuries. They include stick, thorn, wood piece

etc. Injury with stone (24%) is mainly seen in trauma due to RTA, cracker and quarry injuries.

Ball is the main agent in sports injuries (5%). Chemical injuries were least common (2%) and were caused by alkali.

Number of patients with open angle (49%) and angle closure glaucoma (51%) were almost equal.

Among the open angle glaucomas, that caused by hyphema contributed around 25%. These patients present early due to defective vision. This is followed by inflammation (15%).

Less common causes are angle recession (6%) and lens particle glaucoma (3%). Angle recession glaucomas occur only if the recession is about 180º or more.

In the post traumatic angle closure glaucomas, Lens induced glaucomas contributed to (37%). Of these pupillary block due to anterior dislocation was around (13%), intumescent lens (15%) and subluxated lens (9%).

The next common cause was due to peripheral anterior synechiae (13%). This was due to inflammation in blunt trauma. In penetrating

injury it was due to a shallow anterior chamber resulting in synechial closure.

There was one case of neovascular glaucoma which was due to chronic inflammation following trauma.

B-scan was useful in evaluating the posterior segment status in these eyes. 75% of eyes had a normal posterior segment. In the rest of the cases there were important posterior segment findings like vitreous hemorrhage (20%), retinal detachment (7%). The other associated findings in a few cases were IOFB, PVD and lens dislocation. Thus ultrasonography is very important not only in the management point of view but also for deciding about prognosis.

UBM confirmed angle recession in 6 cases were it was suspected.

On gonioscopy, all the 6 cases had angle recession involving 180º are more.

- 3 cases with 180º involvement
- 2 cases with recession involving slightly more than 180º
- 1 case with 270º recession.

Blunt injury with fist, wood, stone and ball following assault, RTA, bursting crackers and sports were the causes in these 6 cases.

CONCLUSION

Injury to the eye leads to a chain of various complications, of which glaucoma is the most dreadful one. Visual prognosis of ocular trauma depends on several factors.

More emphasis should be laid out on preventive measures by educating the population regarding eye injuries and the importance of protective measures.

Using protective goggles by people working in industries and while bursting crackers must be stressed upon.

Examination and treatment should be carried out immediately following an eye injury to prevent visual loss.

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PROFORMA

1. Name : OP / IP No:

2. Age : Glaucoma Clinic No.

3. Sex : Male/Female Date:

4. Address:

5. Affected Eye:

6. Time internal between injury and admission :

7. Mode of injury:

8. Type of injury : Penetrating / Blunt / Chemical /Thermal

9. Agent causing injury: Stone / Metal / Ball / Glass / Fist Chemicals / Others

10. Ocular Examination

i. Vision :

ii. Conjunctiva :

iii. Cornea :

iv. Anterior chamber : Depth Clarity

v. Iris :

vi. Pupil :

vii. Lens :

viii. Tension (mm Hg) : By applanation / By non contact tonometer

ix. Gonioscopy :

x. Field :

xi. Fundus :

xii. X-ray orbit :

xiii. B – Scan :

xiv. UBM :

xv. Diagnosis (Type of Glaucoma) :

MASTER CHART

Sl. No	Name	Age	Sex	Address Rural/ Urban	Eye Affected R/L	Time internal between injury & admission Imm / Late	Mode of Injury	Type of injury	Agent causing Injury	Vision		Tension mm Hg		Gonio scopy in AE	Fundus in AE	Fields in AE	B. Scan in AE
										RE	LE	RE	LE				
1.	Kumar	43	M	R	L	I	DO	B	W	6/12	PL+	12	30	NP	HV	NR	N
2.	Mohammed Hussain	22	M	U	L	I	CR	B	STO	6/6	6/24	17.5	29	O	N	N	N
3.	Kumar	25	M	R	L	I	IN	P	M	6/9	½/60	17	42	NP	HV	NR	VH/IOF B
4.	Anand Babu	13	M	R	R	I	DO	B	W	PL+	6/6	42	17	NP	NV	NR	N
5.	Rajendran	27	M	R	R	I	IN	B	M	CFCF	6/6	42	17	NP	NV	NR	N
6.	Govindammal	50	F	R	L	L	DO	B	M	6/6	PL+	14	54	NP	NV	NR	N
7.	Suryaprakash	6	M	U	L	L	CR	B	M	6/6	3/60	42	14	NP	HV	N	N
8.	Azhagesan	37	M	U	L	I	CR	B	STO	6/6	3/60	12	34	NP	HV	N	N
9.	Muthukumaran	63	M	R	L	L	AS	B	STO	6/12	1/60	17	24	PAS	HV	NR	N
10.	Deenadayalam	27	M	U	L	L	RTA	B	M	6/36	N	17	29	PAS	RD	NR	RD
11.	Pushparaj	54	M	R	R	I	DO	B	W	½/60	6/6	38	12	NP	NV	NR	VH
12.	Ranu	37	M	U	R	I	IN	B	W	PL+	6/6	46	14	NP	NV	NR	N
13.	Koteeswari	12	F	R	R	L	DO	P	W	HM	6/6	42	12	NP	NV	NR	N
14.	Ramachandran	27	M	U	R	L	IN	B	M	6/24	6/6	29	17	O	HV	N	N
15.	Selvakumar	18	M	U	R	I	CR	B	STO	PL+	6/6	42	14	NP	NV	NR	N
16.	Jalal	24	M	U	R	L	IN	B	M	6/6	HM	12	42	NP	NV	NR	N
17.	Thomas	60	M	U	R	I	Q	B	STO	CFCF	6/36	34	14	NP	HV	NR	N
18.	Palani	42	M	U	R	I	AS	B	M	PL+	6/9	46	14	NP	NV	NR	VH
19.	Kumaran	27	M	U	R	I	CR	P	STO	½/60	6/6	42	17	NP	NV	NR	VH
20.	Sudarsan	20	M	U	R	L	SP	B	B	6/36	6/6	54	20	AR	CD.9	TF	N
21.	Murugan	35	M	R	L	L	AG	B	STI	6/6	5/60	14	34	PAS	N	N	N
22.	Mahesh	22	M	U	R	I	RTA	P	M	PL+	6/6	30	14	O	HV	NR	VH
23.	Pongothai	50	F	U	R	L	DO	B	W	HM	6/18	34	14	NP	NV	NR	N
24.	Nagaraj	25	M	R	L	L	SP	B	STO	6/36	6/6	24	12	PAS	N	N	N
25.	Ilanchezhiyan	24	M	U	R	L	RTA	P	STO	PL+	6/6	29	14	PAS	CD.9	NR	N
26.	Jayalakshmi	25	F	R	L	L	DO	P	W	6/6	HM	14	29	C	CD.9	NR	N
27.	Subbulaxmi	26	F	U	R	L	DO	B	F	6/36	6/6	38	14	AR	RD	N	RD
28.	Nagappa	53	M	U	L	I	AS	B	F	6/60	PL+	17	59	NP	NV	NR	-
29.	Alagarasani	45	M	U	R	L	RTA	B	STO	6/36	6/9	42	14	AR	CD.5	N	N
30.	Johnson	51	M	U	R	I	RTA	B	M	HM	6/6	39	17	NP	NV	NR	N
31.	Emmanel	8	M	U	L	L	SP	B	B	6/9	CFCF	14	54	NP	NV	NR	N
32.	Murugesan	50	M	U	L	I	AS	B	F	6/24	1/60	17	42	NP	HV	NR	N
33.	Nagendran	10	M	R	L	I	CR	B	STO	6/6	HM	12	54	NP	NV	NR	N
34.	Prabaharan	20	M	U	R	I	SP	B	B	2/60	6/6	38	17	NP	HV	NR	N
35.	Sudarsan	35	M	R	R	L	AS	B	F	HM+	6/6	24	14	AR	CD.9	NR	N
36.	Arunkumar	6	M	U	R	I	SP	B	B	6/36	6/6	29	12	O	N	N	N
37.	Sivaraj	28	M	U	R	I	Q	B	STO	PL+	6/6	52	17	NP	NV	NR	N

38.	Sethu	24	M	U	L	I	IN	B	M	6/6	½/60	42	14	NP	NV	NR	N	
39.	Parthiban	7	M	U	R	I	CR	B	STO	PL+	6/6	59	17	NP	NV	NR	N	
40.	Jaiendran	11	M	U	R	I	SP	B	B	1/60	6/6	42	14	NP	NV	NR	N	
41.	Nandagopal	47	M	U	R	L	AS	B	W	5/60	6/9	34	14	AR	N	N	N	
42.	Sarathkumar	7	M	U	R	I	CR	P	STO	PL+	6/6	42	17	NP	NV	NR	VH	
43.	Vijay	27	M	U	R	I	IN	B	M	PL+	6/6	38	14	NP	NV	NR	N	
44.	Arul	12	M	U	L	I	SP	B	W	6/6	CFCF	17	34	NP	NV	NR	N	
45.	Sasikumar	29	M	U	R	I	Q	B	STO	PL+	6/6	42	17	NP	NV	NR	VH/ IOFB	
46.	Rajasekar	12	M	U	L	I	DO	B	W	6/6	6/60	28	14	O	N	N	N	
47.	Raju	45	M	U	L	I	IN	B	T	6/9	PL+	17	69	C	NV	NR	VH	
48.	Ghouse Basha	35	M	R	L	I	AG	B	W	6/36	HM	17	49	C	NV	NR	DL/ VH/RD	
49.	Thangavel	45	M	U	R	I	Q	B	STO	CFCF	6/18	39	17	NP	NV	NR	N	
50.	Amudha	28	F	U	R	I	DO	B	E	6/36	6/6	28	14	O	N	N	N	
51.	Kathiresan	37	M	U	L	I	IN	B	M	6/6	6/60	17	34	O	N	N	N	
52.	Palanivel	20	M	U	R	I	CR	B	STO	CFCF	6/6	42	17	NP	NV	NR	N	
53.	Velan	34	M	U	R	L	IN	C	A	4/60	6/6	38	14	PAS	HV	N	N	
54.	Jeeva	16	F	R	R	L	AG	B	STI	NOPL	6/6	34	16	C	NV	NR	VH	
55.	Suresh	27	M	U	R	I	IN	B	M	½/60	6/6	28	14	NP	NV	NR	N	
56.	Anandhi	20	M	U	R	I	DO	B	W	1/60	6/6	30	16	NP	HZ	NR	N	
57.	Ravikumar	27	M	R	R	I	IN	B	M	CFCF	6/6	40	14	NP	NV	NR	N	
58.	Muniraj	35	M	U	L	L	RTA	B	M	6/36	NOPL	14	34	PAS	RD	NR	RD	
59.	Satish	32	M	U	R	I	IN	B	M	PL+	6/9	38	16	NP	NV	NR	VH	
60.	Ambalam	41	M	U	R	I	AG	B	STI	6/60	6/12	28	14	O	N	N	N	
61.	Mahivanan	28	M	U	R	I	IN	P	M	HM+	6/6	42	16	NP	NV	NR	N	
62.	Kumaresan	24	M	U	R	I	IN	P	M	CFCF	6/6	38	14	NP	NV	NR	VH	
63.	Dhanapal	47	M	U	R	I	RTA	B	STO	½/60	6/12	40	16	NP	NV	NR	N	
64.	Veeralaximi	18	F	R	L	L	AG	P	W	6/6	PL+	14	52	NP	NV	NR	VH	
65.	Rajagopal	32	M	U	L	L	AS	B	F	6/9	6/60	12	28	C	N	N	N	
66.	Saroja	42	F	U	L	I	DO	B	STI	6/6	6/36	16	34	O	N	N	N	
67.	Kathirvel	54	M	U	R	I	RTA	B	STO	6/60	6/9	28	14	O	HV	N	N	
68.	Ambrose	47	M	U	L	I	IN	P	M	6/12	HM	16	38	C	NV	NR	VH	
69.	Janakisam	39	M	U	R	I	IN	B	M	PL+	6/6	40	14	NP	NV	NR	RD	
70.	Asokan	53	M	U	L	I	RTA	B	M	6/60	6/9	28	14	C	N	N	N	
71.	Prema	37	F	U	L	I	DO	B	H	6/6	6/36	14	24	O	N	N	N	
72.	Margaret	50	F	U	R	I	DO	B	W	6/60	6/12	34	12	O	N	N	N	
73.	Appasamy	39	M	R	R	I	AG	P	W	HM+	6/6	42	16	NP	HV	NR	RD/ PVD	
74.	Jebaraj	44	M	U	R	I	AS	B	F	PL+	6/9	38	14	NP	HV	NR	N	
75.	Shanmugam	35	M	U	R	I	IN	P	M	PL+	6/6	54	16	NP	NV	NR	N	
76.	Ethiappan	61	M	R	R	L	AG	B	W	4/60	6/36	34	14	PAS	N	N	N	
77.	Arokiaraj	42	M	U	R	I	IN	B	M	HM+	6/18	40	16	NP	NV	NR	N	
78.	Kani	35	M	U	L	I	IN	B	M	6/6	6/60	14	34	O	HV	N	N	
79.	Pappammal	62	F	R	R	L	AG	P	STI	PL+	6/24	50	14	NP	NV	NR	N	
80.	Selvi	25	F	R	L	L	AS	B	F	6/6	PL+	16	54	NP	NV	NR	VH	

81.	Sukumar	28	M	U	L	I	IN	B	M	6/6	2/60	14	28	O	HV	N	N	
82.	Alagappan	37	M	U	R	I	IN	B	M	5/60	6/6	24	12	O	N	N	N	
83.	Chinnamma	62	F	R	R	I	AG	P	STI	PL+	6/36	60	14	NP	NV	NR	N	
84.	Nathan	32	M	U	R	I	IN	B	M	HM+	6/6	38	16	NP	NV	NR	VH	
85.	Savarimuthu	40	M	U	R	L	RTA	B	M	PL+	6/12	42	14	NP	NV	NR	VH	
86.	Kannan	16	M	U	R	I	CR	B	STO	6/60	6/6	28	14	O	N	N	N	
87.	Muniandi	45	M	U	L	I	Q	B	STO	6/9	4/60	16	24	C	N	N	N	
88.	Pappammal	58	F	R	R	L	AG	P	TH	PL+	6/18	30	16	O	NV	NR	N	
89.	Kumar	26	M	U	R	I	IN	P	G	HM+	6/6	54	14	NP	NV	NR	N	
90.	Samikannu	29	M	R	L	I	AS	B	W	6/6	CFCF	16	38	NP	NV	NR	N	
91.	Papanivel	37	M	U	L	I	IN	B	M	6/6	6/60	16	24	O	N	N	N	
92.	Bakkiam	35	F	U	L	I	Q	B	STO	6/6	HM+	14	58	NP	NV	NR	VH	
93.	Arivasan	14	M	U	R	I	SP	B	B	3/60	6/6	30	14	C	HV	NR	N	
94.	Munusamy	56	M	R	R	L	AG	B	W	½/60	2/60	16	34	C	N	N	N	
95.	Kesavan	24	M	U	L	I	IN	B	M	6/6	2/60	14	52	C	NV	NR	N	
96.	Anbuselvi	24	F	R	R	I	AG	P	TH	PL+	6/6	14	52	C	NV	NR	N	
97.	Velmurugan	33	M	U	L	I	IN	B	M	6/6	6/70	16	28	O	N	N	N	
98.	Hari	10	M	U	R	I	CR	P	STO	PL+	6/6	54	16	C	NV	NR	VH	
99.	Chellakumar	27	M	U	R	I	IN	C	A	6/60	6/6	28	14	O	N	N	N	
100	Neela	60	F	U	L	L	DO	B	E	6/36	3/60	44	14	PAS	NV	NR	N	

KEY TO MASTER CHART

Address	:	U	-	Urban
		R	-	Rural
Time interval between injury & Admission	:	I	-	Immediate
		L	-	Late
Mode of Injury	:	AS	-	Assault
		AG	-	Agricultural
		CR	-	Cracker
		DO	-	Domestic
		IN	-	Industrial
		SP	-	Sports
		Q	-	Quarry
		RTA	-	Road traffic accident
Type of Injury	:	B	-	Blunt Injury
		P	-	Penetrating injury
		C	-	Chemical injury
Agent Causing Injury	:	A	-	Alkali

		B	-	Ball
		E	-	Elbow
		F	-	Fist
		G	-	Glass
		H	-	Hand
		M	-	Metal
		STO	-	Stone
		T	-	Tyre
		TH	-	Thorn
		W	-	Wood
		STI	-	Stick
Gonioscopy	:	O	-	Open
		C	-	Closed
		NP	-	Not possible
		PAS	-	Peripheral Anterior Synchiae
		AR	-	Angle Recession
Fundus	:	N	-	Normal
		NV	-	No view
		HV	-	Hazy View

		RD	-	Retinal detachment
		CD	-	Cup disc ratio
Field	:	N	-	Normal
		NR	-	Not Recordable
		TF	-	Tubular Field
B-Scan	:	N	-	Normal
		VH	-	Vitreous hemorrhage
		RD	-	Retinal detachment
		PVD	-	Posterior Vitreous detachment
		IOFB	-	Intraocular Foreign Body
		DL	-	Dislocated lens

UBM (Ultrasound biomicroscopy)

		AR	-	Angle recession
Type of Glaucoma :		O	-	Open
		C	-	Closed

Mechanisms Causing Glaucoma

AR - Angle Recession

DL - Dislocated Lens

IL - Intumescent Lens

IN - Inflammation

HY - Hyphema

NV - Neovascularization

PAS - Peripheral anterior synechiae

SL - Subluxated Lens

LIST OF FEW SURGERIES PERFORMED

Sl. No.	Name	Age	Sex	OP/IP No.	Diagnosis	Surgery
1.	Natarajan	54	M	400006	LE-Mature Cataract	LE-ECCE/PCIOL
2.	Haniffa	58	M	405274	RE – Immature Cataract	RE–ECCE/PCIOL
3.	Ramanujam	60	F	492513	LE-Nuclear Cataract	LE-SICS/PCIOL
4.	Subramani	72	M	490121	LE-Immature Cataract	LE-SICS/PCIOL
5.	Mani	52	M	389781	RE-Secondary Angle Closure Glaucoma	RE – Combined surgery
6.	Neelaveni	64	F	376594	RE-PACG	RE – AG Surgery
7.	Chandra	37	F	62567	RE-Pterygium	RE-Excision/ Autograft
8.	Thulasi	18	F	389901	RE-Conjunctival tear	RE-Conjunctival Suturing
9.	Ezhumalai	28	M	400971	RE-Rupture globe	RE-Corneoscleral suturing
10.	Palani	60	M	403584	LE-Panophthalmitis	LE-Evisceration
11.	Prema	27	F	397230	LE-Pterygium	LE-Pterygium excision/ autograft
12.	Santhanam	28	M	27251	LE-Lower Lid chalazion	LE-Incision and curettage
13.	Sampath	72	M	394473	LE-Chronic Dacryocystitis	LE-DCT
14.	Velammal	43	F	48724	LE-Chronic Dacryocystitis	LE-DCT
15.	Govindhan	60	M	402662	RE-Nuclear Cataract	RE-ECCE/PCIOL
16.	Jeyammal	42	F	402814	RE-PCC	RE-ECCE/PCIOL
17.	Periyasamy	58	M	497721	LE-Immature Cataract	LE-SICS/PCIOL
18.	Kesavan	53	M	498237	LE-Immature Cataract	LE-SICS/PCIOL
19.	Kavikannan	75	M	403416	LE – Fungal Corneal ulcer with stromal abscess	LE – TKP 8mm over 7.5 mm
20.	Jagadeeswari	32	F	28549	RE-Chronic Dacryocystitis	RE-DCR
21.	Pushpa	48	F	41108	RE-Chronic Dacryocystitis	RE-DCR
22.	Mohan	52	M	752018	RE-Lowerlid granuloma	Excision
23.	Chinnasamy	65	M	36860	LE-Chronic Dacryocystitis	LE-DCT