"PREVALENCE OF OCULAR TRAUMA IN KLES HOSPITAL – A ONE YEAR CROSS SECTIONAL STUDY"

By

REG. NO. BK0113001

Dissertation

Submitted to the KLE University, Belagavi, Karnataka.

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY
IN
OPHTHALMOLOGY

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELAGAVI, KARNATAKA.

APRIL 2016

KLE UNIVERSITY, BELAGAVI,
KARNATAKA.

Endorsement by the Head Of Department, Principal/ Head of the Institution

This is to certify that the dissertation entitled **"Prevalence of Ocular Trauma in KLES HOSPITAL – A one year cross sectional study"** is a bonafide research work done by **REG. NO. BK0113001.**

Seal & Signature of the HOD

Dr. (Mrs.) Rekha B. K **M.S, DOMS , PhD**
Professor & Head,
Department of Ophthalmology,
J. N. Medical College,
Belagavi – 590010.
Karnataka, India.

Date :

Place: Belagavi

Seal & Signature of the Principal

Dr. (Mrs.) N. S. Mahantashetti **MD (Paed)**
Principal,
J. N. Medical College,
Nehru Nagar,
Belagavi - 590010.
Karnataka, India.

Date :

Place: Belagavi

LIST OF ABBREVIATIONS USED

CT	-	Computed Tomography
IOFB	-	Intraocular foreign body
IOP	-	Intraocular pressure
MRI	-	Magnetic Resonance Imaging
RAPD	-	Relative Afferent Pupillary Defect
RPE	-	Retinal Pigment Epithelium
RTA	-	Road Traffic Accident
TON	-	Traumatic Optic Neuropathy
VA	-	Visual acuity

ABSTRACT

Background and Objectives

Although the eyes represent only 0.1% of the total body surface, most of the information reaches humans through vision. *Ocular trauma* causes a great socio-economic impact. Those affected by eye injury often have to face loss of career opportunities, major lifestyle changes and occasionally permanent disfigurement. In addition to the physical and psychological trauma of eye injuries to the individual, the direct and indirect financial costs to the society are enormous. Prevention of ocular injuries should form the basis of management for which collection of data is a must. Seeing the seriousness and enormity of ocular trauma, the present study is being undertaken to know the prevalence, cause of injury, clinical and demographic profile of ocular trauma patients. Aims are:-

1. To find the prevalence of ocular trauma in patients attending our hospital
2. To find the clinical profile of ocular trauma patients and the cause of trauma in them.

Methodology

The present one year cross sectional study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore hospital and Medical Research Centre, Belagavi on patients presenting with ocular trauma to the casualty and Ophthalmology OPD of the hospital during the period between 1st January 2014 and 31st December 2014. The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi. All cases of injury to the eyeball, optic nerve, orbit, upper and lower lids, eyebrows and the lacrimal system were

included and examined in a systematic manner. Investigations like CT scan, X ray and B scan were carried out where ever required. The conditions were treated medically or surgically, as was the need.

Results

A total of 2308 patients presented to the casualty and the Ophthalmology OPD, out of these, 217 patients had ocular trauma. Hence the prevalence rate of ocular trauma came out to be 9.40%.

Maximum patients (27.19%) were in the age group of 21-30 years and minimum patients (0.92%) belonged to the age group of 71-80 years.

182 (83.87%) patients were males and 35 (16.13%) patients were females. Male:Female ratio was 5.2:1.

Most common cause of ocular trauma was RTA (66.36%), followed by occupational injury (6.91%) and fall from height (6.91%). Foreign bodies entering the eye accounted for 6% of injuries. Other causes which were responsible for 1-2% of injuries included assault, vegetative matter, stick, knife, firecracker, bull horn, glass, ball, sports related, stone chip and blast injury.

Time of the day was divided into Morning (5 AM – 12 PM), Afternoon (12 PM – 4 PM), Evening (4 PM – 8 PM) and Night (8 PM – 5 AM). In our study, 92 (42.40%) patients were injured in the night followed by 53 (24.42%) at afternoon, 40 (18.43%) in the evening and 32 (14.75%) in the morning.

Right eye was involved in 106 (48.85%) patients followed by left eye in 78 (35.94%). In 33 (15.21%) patients, both eyes were involved.

Clinically good vision was considered as vision of counting fingers at a distance of more than 3 meters when recorded in the absence of a visual acuity chart in the casualty. 145 eyes (58%) had clinically good vision whereas no perception of light was present in 5 eyes (2%).

190 (87.56%) patients presented to the casualty of the hospital whereas 27 (12.44%) presented to the Ophthalmology OPD.

Patients presented with orbital fractures (12.90%), eyelid findings (84.79%), conjunctival involvement (71.43%), corneal findings (15.67%), anterior chamber findings (12.44%), pupillary findings (24.88%), iris involvement (5.07%) and lens findings (6.45%).

Out of 217 ocular trauma patients, 138 (63.59%) patients underwent medical treatment and the rest 79 (36.41%) patients underwent surgical treatment.

Conclusion

Most common cause of ocular trauma was found to be Road Traffic Accidents (RTA), seen most commonly amongst males in the age group of 21-30 years. Males are commonly injured in RTA which is probably related to both exposure and risk taking behavior. Several human and environmental risk factors were found to be associated with increased risk of RTA. Apart from RTA, occupational hazard was the next major cause of ocular trauma. Certain laws and legislations and their strict enforcement can curb these two major causes of ocular trauma.

Keywords

Ocular trauma, Prevalence, Road Traffic Accident

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

LIST OF PHOTOGRAPHS

INTRODUCTION

Although the eyes represent only 0.1% of the total body surface, most of the information reaches humans through vision. *Ocular trauma* causes a great socio-economic impact. Those affected by eye injury often have to face loss of career opportunities, major lifestyle changes and occasionally permanent disfigurement.

Population based epidemiological studies in Andhra Pradesh and Tamil Nadu have found prevalence rates of ocular trauma as 2-10 %. In the United States alone, there are approximately 2.5 million cases of eye injuries each year and 40,000 – 60,000 cases of trauma related visual impairment annually. In addition to the physical and psychological trauma of eye injuries to the individual, the direct and indirect financial costs to the society are enormous. In USA, the total cost of disabling and non-disabling eye injuries comes to billions of dollars. The annual incidence rate of hospitalization for eye injuries per 1 lakh population/year is 5-16% worldwide. Blindness resulting from trauma has prevalence rates of 0.6-0.8%. The use of eye protective devices in India is very low.

The prophylactic measures like implementation of seat belt laws have reduced the incidence of eye injuries by 47-65%. The eye injury patterns may be different in different geographical areas.

Similar type of data as available in the United States on ocular injuries cannot be found in this part of the country; hence the purpose of the study is to collect similar type of data.

Prevention of ocular injuries should form the basis of management for which collection of data is a must. Seeing the seriousness and enormity of ocular trauma, the present study is being undertaken to know the prevalence, cause of injury, clinical and demographic profile of ocular trauma patients presenting in KLE hospital, Belagavi from this region so that strategies can be planned for the prevention and management of ocular trauma in a better way.

AIMS AND OBJECTIVES

- To find the prevalence of ocular trauma in patients attending our hospital.
- To find the clinical profile of ocular trauma patients and the cause of trauma in them.

REVIEW OF LITERATURE

The history of ocular injuries began, when one primitive man fought with another, first walked through the undergrowth of the forest, or first chipped a piece of flint to make his primitive tool. However, in modern times, the scope of ocular injury has been widened to include occupational injury, industrial injury, domestic injury, accidents, as also vehicular accidents.

The TARN[1] (Trauma Audit Research Network) collected data about 2,08,007 patients between 1989 and 2004 of whom 39,073 had major trauma, and among them 905 were recorded as having eye injuries. Out of whom 31% had corneal injury, 12.9% had conjunctival injury, 13.2% had optic nerve injuries & 5.9% had injury to the retina.

In a study conducted at Christian Medical College, Vellore between July 2004 and January 2005 comprising of 379 cases coming to their emergency services found out that ocular trauma showed a gradual decline with age with maximum number of patients in the 0-10 years age group, there was a bimodal peak in the months of January and November, 71% of the cases were male patients and work related trauma accounted for 22.4% of the cases.[2]

Shtewi M El et al[3] in a 2 year study, from Oct 1993 to Sept 1995, of road traffic accidents in patients attending, or referred to Tripoli Eye hospital, Libya found that the percentage of road traffic accidents, resulting in ocular trauma is 12.5%.

The Beaver Dam Eye Study[4], a population based cross sectional study to find out the cumulative lifetime prevalence and 5-year incidence of self reported history of ocular trauma, having 4926 cases showed that the lifetime prevalence was 19.8% and 5-year incidence was 1.6%, ocular trauma in both eyes was reported in 15% of prevalent cases and in 8% of the incident cases, males had four times the prevalence of females.

The Aravind Comprehensive Eye Survey[5], a population based cross sectional study of 5150 persons showed blunt injuries were the major cause of trauma in this population, the most common setting where ocular trauma occurred was during agricultural labor, 74.2% of those who reported ocular trauma sought treatment from an eye specialist and 20.6% from a traditional healer, the age adjusted prevalence for blindness in any eye caused by trauma was 0.8%.

The Andhra Pradesh Eye Disease Study[6], a population based cross sectional study pointed out that the prevalence of history of eye injury in the study population was 7.5%, men were more likely to have an eye injury than women, injury with vegetable matter such as a thorn, branch of a tree, plant secretion etc. was the major cause of trauma in this population, majority of the eye injuries occurred at the workplace followed by home, majority of those affected did not wear eye protection at the time of trauma and it was responsible for unilateral blindness in 39 subjects.

A study about orbital fractures in a tertiary health care center by Sumana J Kamath et al[7], out of the 35 patients, 32 (91.42%) were males and 3 (8.5%) were females. Most common age was between 20-40 years accounting for 13 cases (48%).

Soong TK, Koh A, Subrayan V and Loo AV[8] studied 546 patients presenting with ocular trauma to their hospital. Among eye injury cases, 481 patients (88.1%) were male, with a male-to-female ratio of 7.4:1. Of the patients, 412 (75.5%) were Malaysian while the remaining 134 (24.5%) were of non-Malaysian nationality. The average age was 31.5 years (range 1-81 years). A total of 238 injured eyes (43.6%) were work-related. The common sources of eye trauma include the use of high-powered tools (30.8%), motor vehicle accident (23.1%), and domestic accidents (17.7%). Only six patients (2.5%) reported to having used *eye protective device* at time of their work-related injuries.

Mustafa Serinken et al[9] conducted a studied on 817 patients who got admitted to the emergency department after suffering work related injury. 778 subjects were male (95.3%), mean age of the patients was 28.1±6.5 (range 15-54), maximum patients were in the age group of 25 and 34 years. Most workers were working in the metal and machinery industries. Most common cause of injuries was exposure to welding light (n=219), followed by drilling/cutting injuries (n=172). Lack of protective measures ranked the highest among the workplace related causes (n=207).

ANATOMY OF THE EYE

The eye is a delicate sense organ that is surrounded by specialized structures and protected by the bony orbit, soft tissue, and eyelids. The globe itself is composed of three primary layers or “coats”: the sclera, the uvea, and the retina. Anteriorly, the cornea covers the central area of the eye and the conjunctiva covers the sclera. The iris, the ciliary body, and the choroid constitute the uvea. The crystalline lens separates the anterior and posterior chambers from the vitreous body. The optic nerve transmits images from the retina to the brain.

Clinical Anatomy of the orbit

The orbit is a four-sided conical structure with its base directed forwards and apex projecting medially towards the optic foramen. The base or the orbital rim is outlined by thick strong bone: the supra-orbital arch of the Frontal bone above, the Zygoma and Maxilla inferiorly, the Zygoma laterally and the frontal process of the Maxilla medially. The walls of the orbit consist of relatively thin bone. The orbital volume is about 30 ml and the orbital depth is approximately 4.5 cm. Consequently slight change in the bony orbit will be reflected on soft tissue and globe position.

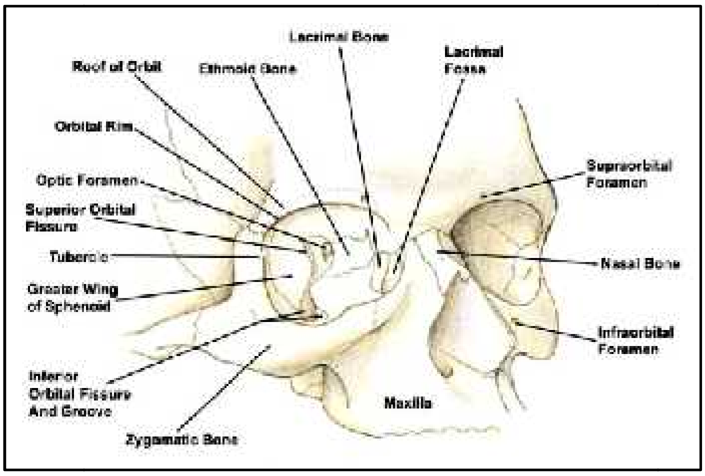


Figure-1: Bony orbit

Orbital Bones contributing to each wall

Roof

- Frontal bone
- Lesser wing of the Sphenoid bone

Medial wall

- Frontal process of the Maxillary bone
- Lacrimal bone
- Ethmoid bone
- Body of the Sphenoid bone

Floor

- Maxillary bone
- Zygomatic bone
- Palatine bone

Lateral Wall

- Zygomatic bone
- Greater wing of Sphenoid bone

Eye lids

The eyelids protect the surface of the eye and also contain glands that contribute components to the tear film. The upper eyelid can be divided into two lamellae:

Anterior: Skin, orbicularis.

Posterior: Tarsus, levator aponeurosis, Muller's muscle, palpebral conjunctiva.

Conjunctiva

The conjunctiva is a clear, vascular, mucous membrane composed of non-keratinized epithelium with goblet cells and underlying loose stromal tissue.

Conjunctiva is divided into:

i. **The Bulbar conjunctiva –** covers the anterior sclera and is loosely adherent except at its attachment to the corneo-scleral junction, where it fuses with Tenon's capsule.

ii. **The Palpebral (or Tarsal) conjunctiva** covers the inner surface of the eyelids, where it is firmly attached to the tarsal plates.

iii. **The Fornices** (Superior fornix and inferior fornix) are blind pouches where the conjunctiva reflects upon itself between the bulbar and palpebral surfaces.

Sclera

The sclera is the tough, avascular, outer fibrous layer of the eye that forms a protective coating. It is composed of dense collagen fibrils that are not highly organized. The sclera is covered by fascia and conjunctiva anteriorly and fat posteriorly.

Uvea

The uvea refers to the pigmented layer of the eye and is made up of three distinct structures: the iris, the ciliary body, and the choroid.

The iris divides the anterior chamber and the posterior chamber. The ciliary body is the 6 mm portion of uvea between the iris and choroid, and is attached to the sclera at the scleral spur. It is composed of two zones, the anterior 2 mm pars plicata, which contains the ciliary muscles, vessels and processes and the posterior 4 mm pars plana.

The choroid is the tissue between the sclera and the retina, and is attached to the sclera at the optic nerve and scleral spur. This highly vascular tissue supplies nutrition to the RPE and outer retinal layers.

Lens

The crystalline lens, located between the posterior chamber and the vitreous cavity, separates the anterior and posterior segments of the eye. The lens grows by elongation and transformation of the epithelial cells into lens fibers.

Vitreous

The vitreous humour is a viscous, gel like substance that fills the posterior segment of the eye between the lens and the retina. The vitreous is composed mainly of water, but also contains collagen fibers, mucopolysaccharides and hyaluronic acid.

Retina

The retina is the delicate transparent light sensing inner layer of the eye that functions like film in a camera. Light travels through the retina to the photoreceptor in the outermost layer. The rod and cone photoreceptor cells convert the light into neural signals that pass back through the retina to the ganglion cells whose axons form the optic nerve.

Optic nerve

The optic nerve is essentially a cable of many wires that transmits images from the eye to the brain where they can be interpreted. The nerve contains approximately 1.2 million axons formed from the retinal ganglion cells in the retina and appears as a yellow – orange circle, nasal to the fovea.

NEW STANDARDIZED CLASSIFICATION OF OCULAR TRAUMA TERMINOLOGY (Birmingham Eye Trauma Terminology - BETT)[10]

Term		**Definition**
Eye wall	:	Sclera and cornea
Closed-globe injury	:	The eye wall does not have a full thickness wound (eye wall not opened).
Open globe injury	:	The eye wall does have a full thickness wound.
Rupture	:	Full thickness eye wall wound caused by a blunt object. The impact results in a momentary increase of IOP and an inside-out mechanism.
Laceration	:	Full thickness wound of the eye wall usually caused by a sharp object. The wound occurs at the impact site by an outside-in mechanism.
Penetrating injury	:	Single laceration of the eye wall, usually caused by a sharp object.
Intra-ocular foreign body: (IOFB)		Retained foreign object(s) causing entrance laceration(s).
Perforating injury	:	Two full thickness lacerations (entrance and exit) of eye wall usually caused by sharp objects.

OCULAR MANIFESTATIONS OF TRAUMA

Eyelids

Injury to the eye lid may be divided into Blunt or Penetrating trauma.

Blunt Trauma

Ecchymosis and edema are the most common presenting signs of blunt trauma. A haematoma (black eye) is most commonly a result of blunt injury to the eyelid or forehead and is generally innocuous.

Penetrating Trauma

These can be classified as:

i. Laceration not involving eye lid margin

ii. Laceration involving the eye lid margin

Superficial eyelid laceration involving just the skin and orbicularis muscle usually require only suturing of the skin alone. The presence of orbital fat in the wound means that the orbital septum has been breached. Any foreign bodies should be searched for meticulously before these deeper eyelid lacerations are repaired.

Copious irrigation lavage washes away any contaminated material in the wound. Levator exploration should be carried out if orbital fat prolapse is noted in the upper eyelid wound.

Trauma involving the canthal soft tissue

Trauma to the medial or lateral canthal areas is usually the result of horizontal traction on the eyelid, causing eye lid avulsion at its weakest point, the medial or lateral canthal tendon.

Lacerations in the medial canthal area demand evaluation of the lacrimal drainage apparatus, which is always involved in an avulsion injury. Canalicular involvement is usually confirmed by inspection and gentle probing. Trivial medial canthal injuries result in canalicular lacerations.

Contusion and concussion injury

Concussion occurs due to sudden acceleration or deceleration imparted by impact of a blunt force sustained by a blow from a large object or in a collision or fall.

Concussion indicates that there is no disorganization of tissue and the changes are reversible. Contusion injury indicates there is disorganization of tissues, like bruising, perivascular haemorrhage etc, with intact surface integument.

As a general rule, either the anterior segment of the eye in front of the iris-lens diaphragm, or the posterior half is preferentially affected.

Mechanism of concussion injury

When a force impinges upon the cornea, this tissue is thrust inwards and may even be forced against the lens and iris, the wave of aqueous pushes these structures backwards, the compression wave rebounds from the back of the eye and they are thrust forward again. They may thus be severely traumatized.

At the same time, there is a horizontal wave of pressure striking the retina and choroid as well as the angle of the anterior chamber, which may do considerable damage

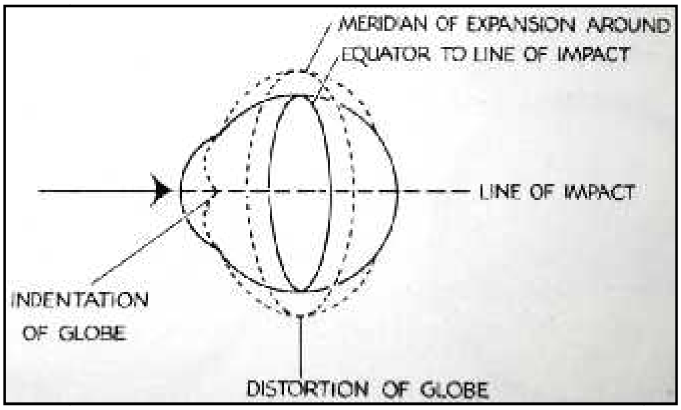


Fig-2: Effect of a Concussion on the globe.

Ophthalmic effects of contusion or concussion injury[11]

Ocular tissue involved	Clinical manifestations
Orbit	• Fracture • Orbital hematoma • Carotid – cavernous fistula
Eye lids	• Hematoma • Laceration
Conjunctiva	• Subconjunctival haemorrhage
Anterior Uvea	• Hyphaema • Tears of the iris sphincter and iridodialysis • Angle recession and cyclodialysis
Lens	• Rosette cataract • Subluxation of the lens • Rupture of the anterior or posterior capsule
Sclera	• Rupture, commonly at the limbus or behind the insertion of the recti
Vitreous	• Haemorrhage
Choroid	• Choroidal rupture • Suprachoroidal haemorrhage
Retina	• Retinal or subretinal haemorrhage • Retinal oedema, commotio retinae • Retinal dialysis • Macular oedema or hole
Optic nerve	• Optic nerve avulsion • Hemorrhage of the optic nerve sheath

Orbital fractures

Orbital trauma can damage both the facial bones and the adjacent soft tissue. Fractures may be associated with injuries to orbital contents, intracranial structures, and paranasal sinuses.

Zygomatic fractures

Zygomatico – maxillary complex fractures are called tripod fractures, although the Zygoma is usually fractured at its articulations with the adjacent bones (Lateral orbital rim, Zygomatic arch, and lateral wall of the maxillary sinus). The zygomatico – maxillary complex fracture involves the orbital floor to varying degrees.

Orbital Apex Fractures

Orbital Apex fractures usually occur in association with other fractures of the face, orbit, or skull and may involve the optic canal, superior orbital fissure, and structures that pass through them. Possible associated complications include damage to the optic nerves with decreased visual acuity, cerebrospinal fluid leaks, and carotid cavernous sinus fistula.

Orbital roof fractures

Orbital roof fractures are usually caused by blunt trauma and are more common in young children who do not yet have pneumatized frontal sinus. Frontal trauma in older patients tends to be absorbed by the frontal sinus, which acts as a crumple zone, preventing extension along the orbital roof. Complications include intracranial injuries, cerebrospinal fluid rhinorrhea, pneumocephalus, subperiosteal hematoma, ptosis, and extraocular muscle imbalance[12].

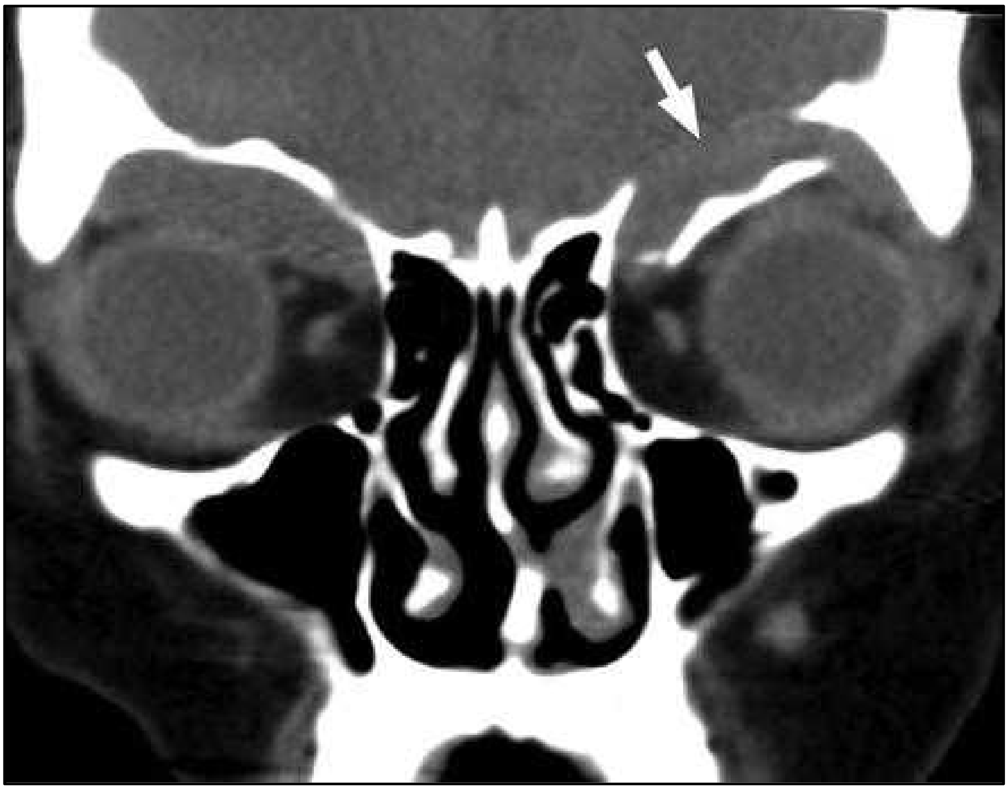


Figure-3: Orbital roof fracture in an adult. Coronal non enhanced CT image shows an isolated blow-in fracture of the left orbital roof (arrow).

MEDIAL ORBITAL FRACTURES

Direct (Naso-orbital-ethmoidal) fractures

These fractures result from the force striking solid surfaces. These fractures commonly involve the frontal process of maxilla, the lacrimal bone, and the ethmoid bones along the medial wall of the orbit. Patients characteristically have a depressed bridge of the nose and traumatic telecanthus.

Indirect (blowout) fractures

These are frequently extensions of blow out fractures of the orbital floor. Isolated blowout fractures of the medial orbital wall also may occur. Emphysema of the eyelids and orbit is commonly associated with fractures of the medial orbital wall. The risk of enophthalmos is greatest when both the floor and the medial wall are fractured and displaced[13].

Orbital floor fractures

Direct fractures of the orbital floor can extend from fracture of the inferior orbital rim. Indirect fractures of the orbital floor are not associated with fractures of the inferior orbital rim.

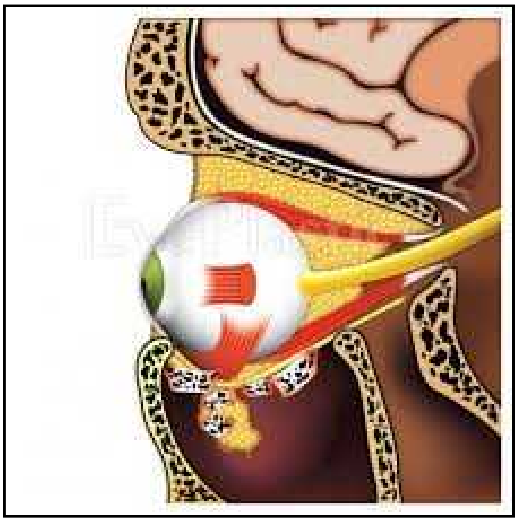


Figure-4: Orbital floor fracture

Past theory held that blow out fractures resulted from sudden increase in intraorbital pressure resulting from the application of force by a non penetrating object, usually smaller in diameter than the orbital entrance. According to this theory, there is compression of contents of the orbit posteriorly towards the apex of the orbit and the orbital bones break at their weakest point, usually the postero medial part of the floor in the maxillary bone. The orbital contents may be entrapped or may prolapse through the fracture in the maxillary sinus. A more recent theory, however, is that the striking object causes a compressive force at the inferior rim, which leads directly to buckling of the orbital floor. The amount of increased orbital pressure determines whether or not the orbital contents are pushed inferiorly through the fracture into the maxillary antrum[14, 15].

The patient may present with

1. Eyelid signs: Ecchymosis and edema of the eyelids, but external signs of injury can be absent.
2. Diplopia with limitation on upgaze, downgaze, or both.
3. Enophthalmos of the globe and ptosis.
4. Hypo aesthesia in the distribution of the infra orbital nerve.
5. Emphysema of the orbit and eyelids.

Conjunctiva

A blunt injury may produce subconjunctival haemorrhages, chemosis or lacerations of the conjunctiva. But in mild injury, no damage is done.

Sub-conjunctival Haemorrhage

Following an injury, the blood vessels of the conjunctiva are torn, giving rise to bleeding into the sub conjunctival tissues. The haemorrhage may vary in size, from petechiae to a large extravasation, sometimes producing a "bag of blood" appearance protruding over the lid margin.

The haemorrhage frequently occurs in the bulbar conjunctiva, and rarely in the palpebral area, because of the anatomical predisposition. A subconjunctival haemorrhage frequently conceals a scleral rupture.

Chemosis

There is an accumulation of transudate in the sub-conjunctival tissue. The conjunctiva then bulges, and may even fold over the cornea, or protrude between the lids.

Conjunctival laceration

The conjunctiva is very thin and gets lacerated following a direct impact. This is rarely extensive, as conjunctiva is freely mobile.

Cornea

Corneal Abrasion

Corneal abrasion involves a break in the continuity of the epithelium, which stains with fluorescein. If it is present in the pupillary area, vision may be grossly impaired. This is an exquisitely painful condition.

Blunt Trauma

Although blunt injury of insufficient force to rupture the globe can cause severe intraocular damage, its effects on the cornea are usually transient. A direct, focal, concussive blow causes mechanical deformation injury to the surrounding endothelium, evident as a ring of corneal edema[16].

Such localized contusion resolves with recovery of the injured cells or their replacement by adjacent endothelium. In more severe corneal contusion, ruptures of Descemet's membrane with concomitant breach of the endothelial barrier result in acute hydrops with massive stromal edema.

Although the decrease in vision and the acute appearance of corneal edema may be capable of healing such defects by sliding over the area of retracted Descemet's membrane and restoring the normal endothelial pump and barrier to maintain corneal deturgescence.

Specular microscopy of the corneal endothelium after blunt trauma may reveal a significant decrease in the endothelial cell population and increased cellular pleomorphism compared to the fellow eye.

Corneal rupture is an uncommon consequence of blunt trauma and typically occurs only in eyes predisposed by a corneal stromal thinning disorder, such as extreme Terrien's marginal degeneration, or by prior surgery such as keratoplasty[17] or radial keratotomy.

Traumatic Hyphaema

An accumulation of blood in the anterior chamber is known as hyphaema. Trauma producing bleeding into the anterior chamber of the eye is common. Hyphaema can be primary, secondary and recurrent.

Primary Hyphaema

It occurs at the time of the injury. The bleeding is self – limiting, irrespective of whether it occurs from a small vessel or a large vessel.

This is because

1. Equilibrium is maintained between the vascular and the intraocular pressure.

2. Once a vessel of the iris ruptures, there is immediate contraction of its wall. This is also the reason why there is no bleeding following an iridectomy. If the haemorrhage is large, it suggests that a larger size vessel near the root of the iris or, in the ciliary body is torn.

Secondary Hyphaema

Following an injury at the onset, there may not be any haemorrhage. But the haemorrhage occurs on the 2nd to 5th day. Secondary haemorrhages usually produce small hyphaemas. Incidence of secondary haemorrhage is variable: 5% to 30%[18]. Secondary haemorrhage is more common when the amount of blood initially is large[19].

Recurrent Hyphaema

Rarely, haemorrhage into the anterior chamber recurs for weeks or months. Recurrent hyphaema following a blunt injury to the eye may at times be associated with a more severe prognosis than occurring from the initial trauma.

The complications which can develop are: higher risk of secondary glaucoma, corneal blood staining, poor visual activity.

The presence of fresh blood in the anterior chamber, or an increase in the amount of blood in the anterior chamber is considered indicative of a recurrent bleeding. The risk factors which are associated with the development of recurrent haemorrhage are not well defined and the exact mechanism is not known[20].

It is hypothesized, that, once the initial vasospasm is relieved or after fibrinolysis occurs, the platelets can no longer adhere to the vessel wall or cannot aggregate without the persistence of the tissue pressure found elsewhere in the solid tissue of the body. The initial clot which is formed is expressed into the low – resistance anterior chamber producing further haemorrhage[20]

Grading of Traumatic Hyphaema[21]

Grade	Size of hyphaema (fraction of anterior chamber filled with blood)
I	< 1/3
II	1/3 – ½
III	½ - near total
IV	Total (Eight –ball)

Associated ocular findings

Angle recession, a separation between the longitudinal and circular muscle fibres of the ciliary muscle[22], is the most important associated anterior segment finding in traumatic hyphaema.

The amount of angle recession does not necessarily correlate with the size of the hyphaema or degree of acute IOP elevation. The incidence of angle recession is variably reported as 30% to 85%[23, 24, 25].

More extensive recession is associated with a higher incidence of late onset glaucoma, and it is estimated that approximately 6% to 10% of hyphaema patients will develop angle recession glaucoma[26].

A cyclodialysis cleft with separation of the scleral spur from its ciliary body attachments occurs less commonly and may cause post injury hypotony.

Traumatic iritis invariably accompanies hyphaema. Furthermore, pigment liberation may result in endothelial pigment dusting and increases trabecular meshwork pigmentation. Iris atrophy occurs less commonly, and a Vossius ring, signifying compression of the pupillary margin on the anterior lens capsule, may develop.

Guidelines for surgical intervention in Traumatic Hyphaema[27]

To prevent optic atrophy, surgical invention should be done

- Before IOP averages >50mm Hg for 5 days
- Before IOP averages >35mm Hg for 7 days

To prevent corneal blood staining, surgical invention should be done

- Before IOP averages >25mm Hg for 6 days
- If there is any indication of early blood staining

To prevent peripheral anterior synechiae, surgical invention
should be done

- Before a total hyphaema persists for 5 days
- Before a diffuse hyphaema involving most of the anterior chamber angle persists for 9 days.

In hyphaema patients with sickle cell haemoglobinopathies, surgical invention should be done

- If IOP averages ≥ 25mm Hg for 24 hours
- If IOP has repeated transient elevations > 30mm Hg.

Traumatic Mydriasis and Miosis

Blunt trauma to the globe might result in traumatic mydriasis or, less commonly, miosis. Traumatic mydriasis is frequently associated with iris sphincter tear that can permanently change the shape of the pupil. Miosis tends to be associated with anterior chamber inflammation. Pupillary reactivity may be sluggish in both situations.

Traumatic Iritis

Traumatic iritis is often associated with diminished vision and perilimbal conjunctival injection. The anterior chamber reaction can be surprisingly minimal but is usually present if carefully sought.

Iridodialysis and Cyclodialysis

Iridodialysis

Blunt trauma may cause traumatic separation of the iris root from the ciliary body. Frequently, anterior segment haemorrhage ensues, and the iridodialysis may not be recognized until hyphaema has cleared. A small iridodialysis requires no treatment. A large dialysis may cause polycoria and monocular diplopia, necessitating early surgical repair before the tissue becomes fibrotic.

Cyclodialysis

Traumatic cyclodialysis is characterized by a separation of the ciliary body from its attachment at the scleral spur, resulting in a cleft. Gonioscopically, this cleft can be seen at the junction of the scleral spur and the ciliary body band. Sclera may be visible through the detached tissue. A cyclodialysis cleft can cause increased uveoscleral outflow and aqueous hypo secretion, leading to chronic hypotony and macular edema.

Glaucoma Associated with blunt injury to the Angle

Glaucoma can occur either early or late following blunt injury and with or without angle recession. The great majority of these glaucomas are associated with recession, with or without hyphaema. These glaucomas can be classified as

i. Acute glaucoma with Hyphaema
ii. Acute glaucoma without Hyphaema
iii. Chronic glaucoma with recession (Angle recession glaucoma)
iv. Chronic glaucoma without recession.

i. Acute glaucoma with Hyphaema

This glaucoma is due to trabecular meshwork obstruction by RBCs, platelets fibrin, and due to direct damage to the outflow channels.

ii. Acute Glaucoma without Hyphaema

Immediately following blunt injury, the intraocular pressure may often be slightly depressed[28, 29].

This may be due to decreased aqueous production by the traumatized and inflamed ciliary body or due to increased outflow either through the conventional pathway or the uveoscleral pathway[30].

Occasionally, however, the pressure may be immediately elevated in association with the presence of inflammatory cells in the anterior chamber, which transiently obstruct the intertrabecular spaces. In addition, this elevation is usually associated with severe angle recession, although it is rare to have recession without haemorrhage. The increased resistance to outflow, in association with significant recession, is presumed to be due to associated trabecular meshwork disruption.

Acute, severe, and often permanent glaucoma will rarely ensue without persistent inflammation and without visible angle recession[31].

iii. Chronic Glaucoma with Angle Recession

Almost all patients who develop hyphaema following blunt injury have some degree of either recession or direct trabecular meshwork damage[32, 33].

If hyphaema recovers without complication and the pressure normalizes, the patient should be examined gonioscopically approximately 6 weeks following the initial injury. If more than 180^0 of the angle is recessed, there is upto 10% chance of subsequent development of chronic glaucoma[34-37].

Glaucoma that develops years later is believed to be due to direct trabecular meshwork damage at the time of injury with subsequent scarring[38].

iv. Chronic glaucoma without recession

Raised IOP can occur in the absence of angle recession due to post inflammatory pupil seclusion due to posterior synechiae and peripheral anterior synechiae.

Ghost cell glaucoma can also occur following 1 to 3 weeks of injury where fresh blood cells in vitreous convert to less pliable khaki colored ghost cells which block the trabecular meshwork and elevate the IOP.

Lens Injuries

Lens injury is a major complication of blunt ocular trauma. Concussion cataracts may occur as either early or late sequelae of the injury. Concussion cataracts are not invariably progressive; some remain stable and do not require surgery.

Pathophysiology

The pathogenesis of lens changes following blunt ocular trauma has been suggested by Wolter[39] and by Weidenthal and Schepens[40].

These authors have descried "Coup" and "Contrecoup" injuries, along with equatorial expansion of the globe due to trauma, or the pathophysiological mechanisms responsible for ocular damage.

"Coup" refers to direct damage resulting in abrasion or laceration; "contrecoup" denotes injury occurring along a plane of trajectory, causing damage at a distal site as a result of shock waves.

Contusion cataract formation, for instance, may result from contrecoup damage following a blow to the orbital area. Shock waves pass through the eye, possibly rupturing the anterior or posterior lens capsule with subsequent lens opacification.

The equatorial expansion theory suggests that blunt trauma applied to the globe in an anterior or posterior direction causes shortening of that meridian, with concomitant equatorial stretching. The equatorial expansion may cause rupture in the lens capsule or equator, resulting in lens opacification.

Zonular disruption during a sudden increase in an equatorial meridian causes incomplete and asymmetric lens support, with resultant dislocation or subluxation.

Traumatic rosette shaped opacity

A rosette shaped opacity is also known as a stellate cataract, filiform cataract, or radiating sub-capsular cataract. They occur following blunt or perforating injuries. These opacities were initially described by Dyer in 1986. There are two types of rosette shaped opacities.

i. Early rosette
ii. Late rosette

i. Early rosette

These opacities may be evident within a few hours after the injury, or may take weeks or a few months to develop. Following an injury to the lens capsule (anterior or posterior), fluid droplets accumulate between the radiating lens fibers in the subcapsular region appearing as feathery parallel rays, which run away from the suture lines, and appear to branch out from the axial region towards the equator, giving a rosette appearance. The feathery parallel rays around a central suture give the appearance of “Petal” and many such petals give the appearance of a “rosette”. They have a uniform thickness.

ii. Late rosette opacities

These are noticed by the examining ophthalmologist some years following an injury. It may be that the patient has forgotten the injury as it may have been trivial or when minimal amount of damage is produced in the subcapsular region, the damage becomes clinically apparent at a later date. These opacities lie deep in the cortex, or in the adult nucleus. The overlying lens fibres are clear.

Vitreous

Changes in the vitreous body after a concussion of any severity are invariable.

Vitreous Haemorrhage

Vitreous haemorrhage may develop as a result of blunt trauma, causing minimal to severe visual loss. The bleeding occurs as a result of damage to ciliary body, retinal, or choroidal blood vessels. Vitreous haemorrhage from blunt trauma may be associated with retinal tear, and indirect ophthalmoscopy with scleral depression should be performed with great care in attempting to identify retinal abnormalities.

Vitreous haemorrhage often is limited immediately after injury, with the initial fundus examination allowing best visualization of retinal details. Subsequent diffusion of the haemorrhage or further bleeding may compromise later examinations. If the surgeon suspects an occult scleral rupture, scleral depression should be deferred.

In general, patients with a non penetrating ocular injury and a vitreous haemorrhage should be observed for several months. In case of minimal diffuse haemorrhage, bed rest with the head of the bed elevated should be suggested to allow settling of the blood with gravity and permit indirect ophthalmoscopy. However, if the blood does not settle, the patient should be followed every 4-6 weeks with ultrasonography repeated to confirm retinal attachment. If retinal detachment is seen or suspected on ultrasonography, pars plana vitrectomy should be performed; otherwise, vitrectomy usually should be deferred for 3-6 months to permit spontaneous resorption of the haemorrhage.

Vitreous Base Avulsion

Trauma to the globe may disinsert the vitreous base from the peripheral retina and pars plana. Avulsion of the vitreous base results in minimal ocular symptoms, although the patient may complain of floaters associated with a partial vitreous detachment. Vitreous base avulsion alone does not have to be treated, but examination must be performed for associated traumatic ocular damage such as retinal dialysis, giant retinal tear, angle recession, or subluxated crystalline lens.

Retina

Several posterior pole retinopathies may result for blunt injury.

Commotio retinae

Commotio retinae (Berlin's oedema), first described by Berlin in 1873, is characterized by a transient, gray white opacification at the level of the deep sensory retina occurring after blunt trauma. This opacification may be confined to the macula or may involve extensive areas of the peripheral retina.

The reasons for this retinal opacification are disputed, with some attributing it to extracellular oedema[41], however, experimental and histopathological studies have shown that disruption of the photoreceptor cells' outer segment and damage to the retinal pigment epithelium accounts for the retinal whitening[42-44].

Vision can be markedly decreased with commotio retinae but vision most often improves as the swelling resolves over a 3-4 week period. However, the macula can develop an atrophic appearance with granular hyperpigmentation associated with decreased vision. In addition, the cystoid areas can coalesce to form a long cyst, which can lead to macular hole formation.

Choroidal rupture

Choroidal ruptures, first described by von Graefe in 1854[45], are tears of the choroid, Bruch's membrane, and RPE resulting from non penetrating ocular trauma and are usually associated acutely with subretinal or Sub RPE – haemorrhage.

Direct choroidal rupture occurs anteriorly at the site of impact and are oriented parallel to the ora serrata; indirect choroidal ruptures occur posteriorly, away from the site of impact, and are generally crescent shaped with orientation concentric to the optic disc[46].

The mechanism of choroidal rupture is thought to be primarily mechanical, although vascular damage may play a role[46, 47]. The sclera is somehow protected from mechanical compression and from sudden hyper expansion of the globe by its tensile strength, as the retina, is protected by its elasticity.

However, Bruch's membrane lacks both strength and elasticity and thus is most likely to rupture. Patients with angioid streaks are particularly vulnerable to choroidal rupture because of increased fragility of Bruch's membrane.

A major cause of poor vision resulting from choroidal rupture, other than extension of the rupture into the fovea is late development of subretinal neovascularization[48, 49, 50].

Traumatic Retinal tears and detachment

Retinal tears resulting from trauma are usually the result of damage to the retina at the vitreous base, although some tears may result from either direct blunt damage or indirect contrecoup injuries.

Blunt trauma also can cause retinal breaks by transmission of the force to the vitreous, leading to acute severe vitreo-retinal traction. Rapid displacement of the vitreous can tear the retina in various ways, including retinal dialysis with or without avulsion of the vitreous base, operculated retinal tear, macular hole and horseshoe-shaped retinal tears at the posterior margin of the vitreous base, at the edge of a meridional fold, or at the equator[51, 52].

Pathogenesis

The initiating event in the pathogenesis of retinal detachment resulting from blunt trauma is the forceful anteroposterior compression of the globe, which causes a lateral expansion of the equatorial area and tractional forces on the vitreous base.

For a retinal detachment to occur after blunt trauma, a combination of two pathological changes must be present. First, retinal breaks may occur because of direct contusion or subsequent tissue necrosis as a result of vitreo-retinal traction following lateral equatorial expansion of the globe. Second, traumatic syneresis of the vitreous gel overlying the retinal break may occur either immediately or months after blunt trauma. This liquefied vitreous may then dissect under the retinal break and cause a retinal detachment.

Blunt trauma accounts for 70% to 86% of traumatic retinal detachments and characteristically occurs in young males[53-56].

Tasman[57] reported 52 consecutive patients with ocular contusion examined within the first 3 weeks after injury and followed prospectively for 2 years. Retinal dialysis was found to be a common feature and was diagnosed within 3 weeks of injury in nine patients.

Other Retinal Breaks

Cooling[58] described small, radial, slit like retinal tears located in the paravascular area between the posterior pole and the equator after contusion injuries. These tears occur at areas of strong perivascular vitreoretinal adhesion after forceful traumatic separation of the vitreous from the retina and may be associated with vitreous haemorrhage.

Traumatic Macular Holes

Most traumatic macular holes result from closed – globe contusion injuries. Possible mechanism for traumatic macular hole includes post contusion necrosis, subfoveal haemorrhage, and acute vitreoretinal traction as a result of a contrecoup injury.

Margherias and Schepens[59] reported 10 (1.3%) macular breaks in 758 eyes with traumatic retinal detachments.

A traumatic macular hole rarely leads to retinal detachment.

Traumatic Optic Neuropathy[60]

It refers to acute optic nerve injury secondary to trauma. Patients present with abnormal visual acuity, an ipsilateral relative afferent pupillary defect, impairment of color vision, and maybe a visual field defect. Traumatic Optic Neuropathy (TON) occurs in about 1.6% cases of head trauma cases and in 2.5% of maxillofacial trauma and midface fractures. The optic nerve axons may be damaged either directly or indirectly and the visual loss may be partial or complete. An indirect injury to the optic nerve typically occurs from the transmission of forces to the optic canal from blunt head trauma. This is in contrast to direct TON, which results from an anatomical disruption of the optic nerve fibers from penetrating orbital trauma, bone fragments within the optic canal, or nerve sheath hematomas.

The pathophysiology of TON is thought to be multifactorial, and some researchers have also postulated a primary and secondary mechanism of injury. In indirect TON cases, the injury to the axons is thought to be induced by shearing forces that are transmitted to the fibers or to the vascular supply of the nerve. Studies have shown that forces applied to the frontal bone and malar eminences are transferred and concentrated in the area near the optic canal. The tight adherence of the optic nerve's dural sheath to the periosteum within the optic canal is also thought to contribute to this segment of the nerve being extremely susceptible to the deformative stresses of the skull bones. Such injury leads to ischemic injury to the retinal ganglion cells within the optic canal.

A secondary mechanism can result in optic nerve swelling after the occurrence of acute injury. The optic nerve swelling can exacerbate retinal ganglion cell degeneration by further compromising the vascular blood supply, either through a rise in intraluminal pressure or reactive vasospasm. These secondary mechanisms, in theory, form the rationale for optic canal decompression via medical (i.e, steroids) or surgical means (i.e, bony decompression). Finally, the intracranial segment of the optic nerve may be damaged by forces delivered to the axons by the shifting of the brain following head trauma. With this mechanism, the nerve fibers may be injured against the falciform dural fold or through a shearing force where the nerve becomes fixed as it enters the intracranial opening of the optic foramen.

CT scan and MRI are an important part of the assessment when TON is suspected. In the post-trauma setting, CT scanning is the preferred modality for demonstrating the presence of an optic canal fracture, a displaced bony fragment impinging upon the optic nerve, a metallic foreign body in the orbit, orbital emphysema, or an optic nerve sheath hematoma. A brain and orbit MRI may be useful in certain settings to delineate the extent of hemorrhage involving the neurovascular structures at the orbital apex or to rule out inflammatory or infiltrative causes for an optic neuropathy. The vast majority of patients with TON suffer an indirect injury to the optic nerve within the optic canal, and neuroimaging studies typically demonstrate no abnormalities of the anterior visual pathways, although a fracture in the region of the optic canal may be seen.

The main treatment options for traumatic optic neuropathy (TON) include systemic corticosteroids and surgical optic nerve decompression, either alone or in combination. The current knowledge base on the use of steroids for TON is based on small retrospective studies, anecdotal reports, and extrapolation from national traumatic brain and spinal cord injury studies. Steroid therapy for TON can be categorized as follows: moderate dose (60-100mg of oral prednisolone), high dose (1 gram of intravenous methyl prednisolone/day), or mega dose (30 mg/kg loading dose of intravenous methyl prednisone, followed by 5.4 mg/kg/hr for 24 hours).

In 1990, Bracken and colleagues published their findings on the use of mega dose corticosteroid therapy in the National Acute Spinal Cord Injury Study 2 (NASCIS 2). The NASCIS 2 was a multicenter clinical trial that evaluated patients with acute spinal cord injury treated with placebo, methyl prednisolone, or naloxone. The study showed that methyl prednisolone (30 mg/kg loading dose, followed by 5.4 mg/kg/h for 24 hours) started within 8 hours of injury was associated with a significant improvement in both motor and sensory function compared with patients treated with a placebo. In 2005, the results of the Corticosteroid Randomization After Significant Head Injury (CRASH) trial raised concerns regarding the use of mega dose steroids (same dose as given in the NASCIS 2 study) in traumatic brain injury. This study was the largest randomized study that evaluated steroids in patients with traumatic brain injury and was stopped early due to the significantly increased risk of death in patients that received mega dose steroids at their 6-month follow-up when compared with the placebo group.

Based on the current evidence, a therapeutic role for corticosteroids in the management of TON is unsubstantiated. If steroids are considered for TON, they should not be used in cases with concomitant traumatic brain injury or in patients that present 8 hours or more after initial injury. Whether clinicians should use mega dose rather than lower doses of steroids for selected cases of TON is also not clearly defined by the literature. The NASCIS studies used mega dose steroids in their protocol to demonstrate a beneficial effect in a subset of their patients, but the CRASH study identified several serious complications associated with their use in the trauma setting.

If there is no visual improvement seen or if the patient presents more than 8 hours following injury, surgical decompression of the orbit is indicated. Surgical decompression is thought to help reduce optic nerve compression and subsequent vascular compromise that may occur as a result of the indirect injury. Additionally, surgery has been postulated to remove bone fragments that may be impinging on the optic nerve within the optic canal. The variety of surgical approaches used in optic nerve decompression include intracranial, extracranial, orbital, transethmoidal, endonasal, and sublabial approaches, and the selection of the technique tends to be based on the surgeon's training, background, and experience.

Review of the available literature provides insufficient evidence to conclude that corticosteroid therapy and/or optic canal surgery provides a therapeutic benefit over observation alone in patients with TON. Hence, the optimal treatment for TON remains a debate among physicians.

Optic nerve Avulsion

Optic nerve avulsion is an uncommon yet visually devastating event that usually occurs after non-penetrating ocular trauma, typically when an object intrudes between the globe and the wall of the orbit and displaces the eye. The avulsion injuries may occur as an isolated lesion.

The optic nerve is forcibly disinserted from the retina, choroid and vitreous, and the lamina cribrosa is retracted from the scleral rim. Both complete and partial avulsions have been described.

Complete avulsion results in a blind eye with a fixed and dilated pupil.

Sudden extreme rotation of the globe may be the mechanism in some cases of optic nerve avulsion[61]. Other possible mechanisms include a sudden marked rise of intraocular pressure that forces the nerve out of the scleral canal or a sudden displacement of the globe anteriorly.

There is no effective medical or surgical treatment for this condition.

Sclera

Ruptures of the sclera:

Ruptures are due to a direct impact on the globe by a slowly moving blunt force, which is sufficiently powerful to burst it. There are two types of contusion ruptures based on the mechanism. They are:

1. Direct ruptures.
2. Indirect ruptures.

Direct scleral ruptures:

It is seen over the anterior part of the sclera and is more common when the sclera is thin due to an old inflammation, high myopia, staphyloma or buphthalmos.

Mechanism:

When the force hits the eye with sufficient intensity, the site of impact is suddenly indented and ruptures. This is of two types:

1. Complete direct rupture.
2. Incomplete direct rupture.

Complete direct rupture: When all the layers of the sclera are ruptured.

Incomplete direct rupture: When only part of the scleral thickness is ruptured.

It is common for the inner layers of the sclera to rupture. It is clinically difficult to prove whether a scleral rupture is complete or incomplete. Incomplete ruptures commonly occur in the region of the canal of Schlemm and usually follows a minor trauma at the limbus from light bodies e.g. a small piece of metal, pencil, etc.

Indirect ruptures

They occur following a severe blunt trauma to the globe. They are usually associated with grave intraocular damage and the complications are severe.

The rupture always occurs away from the site of impact.

They are of two types:

1. Complete indirect ruptures.
2. Incomplete indirect ruptures.

Complete indirect ruptures:

They are relatively common. This is more common in old patients because of the loss of resilience of the sclera as age increases, or in a diseased sclera e.g. staphyloma, buphthalmos and high myopia. In a diseased sclera, a less severe injury can produce a rupture.

Indirect incomplete ruptures:

They are rare. The inner layers of the sclera undergo a rupture. According to the pathological anatomy, there are two types of ruptures:

- Typical ruptures.
- Atypical ruptures.

Typical ruptures:

They start internally in the extreme periphery of the anterior chamber, traverse the trabecular meshwork and the canal of Schlemm and run through the sclera, posteriorly and obliquely reaching the surface of the globe 3-4mm behind the cornea. Commonly, some amount of intraocular tissue is incarcerated in the rupture.

Atypical ruptures:

They start internally as mentioned above and after traversing the canal of Schlemm, they run in an obliquely forward direction, to appear externally at the limbus, or on the cornea. They are usually 2-4mm in length. They have a good prognosis, partly because of their small size and partly because the prolapse of the intraocular structures is confined to the iris.

Clinical features of a ruptured globe:

There is pain, excessive lacrimation and loss of vision.

1. Cardinal sign-softness and collapse of the globe.
2. Lids-swollen, bruised and often it is difficult to open the eye.
3. Proptosis with orbital hemorrhage.
4. The conjunctiva may be torn or may be intact. Often, a massive sub conjunctival haemorrhage obscures the scleral rupture.
5. Once the subconjunctival hemorrhage is absorbed, the scleral rupture is visible.

There is a gape in the sclera, with eversion of its edges. The uveal tissue is seen to bulge into the gape as a black tissue.

In a complete rupture if the conjunctiva is also torn, the contents of the globe are extruded outside.

In a less severe trauma, the following features are seen:

1. The iris may be intact.
2. The lens may be dislocated. If it gets incarcerated in the rupture, it may prevent extensive prolapse of the intraocular tissues. Very rarely, the lens may remain in its normal anatomical position.

Prognosis:

The prognosis in general is poor.

1. The best prognosis is in small limbal ruptures in which the ciliary body is not involved. The final vision is good.
2. Prognosis is also good in cases where not much damage has occurred to the ocular structures.
3. The prognosis is very poor in cases with severe trauma. The vision is severely impaired due to vitreous opacities and proliferation of fibrous tissue in the posterior segment of the globe.

Sympathetic ophthalmia has been known to occur rarely.

METHODOLOGY

The present study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi on ocular trauma patients presenting to the Ophthalmology OPD and the casualty of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study design

One year cross sectional study.

Study period

The present study was conducted from 1st January 2014 to 31st December 2014.

Method of collection of data

Source of Data

All the patients attending the Ophthalmology OPD and casualty with ocular trauma at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Sample size

All patients presenting to the Ophthalmology OPD and casualty with ocular trauma from 1st January 2014 to 31st December 2014.

Selection criteria

Inclusion Criteria

All cases of injury to the eyeball, optic nerve, orbit, upper and lower lids, eyebrows and the lacrimal system.

Exclusion Criteria

- Patients having ocular manifestations in head injury cases.
- Facial injury patients without ocular manifestations.
- Polytrauma cases without ocular manifestations.
- Comatose patients having ocular manifestations.
- Ocular trauma patients unwilling to participate for the study.

Procedure

After finding the suitability as per inclusion and exclusion criteria patients were selected for the study and briefed about the nature of the study and written informed consent was obtained. Detailed history of trauma was obtained under the following headings:

- Cause of injury
- Mechanism of the injury
- Place of injury
- Time of injury
- Unilateral/Bilateral
- Any treatment taken before reaching the hospital

History was followed by ocular examination that included visual acuity testing for distance using counting finger method in the casualty and using Green's visual acuity chart in the OPD, checking extraocular movements, examination under diffuse light using a torch followed by anterior segment examination with a slit lamp, a dilated fundoscopy(wherever possible) using topical Tropicamide and Phenylephrine combination (Itrop plus) dilating drops and intraocular pressure measurement (wherever possible) using Schiotz indentation tonometer. Flourescein staining of the cornea was done in cases where corneal abrasion was suspected. B scan of the orbit was done in penetrating trauma cases, intraocular foreign body cases and cases where the posterior segment details were not clear due to opaque media. CT scan was done in cases of ocular trauma having head trauma also.

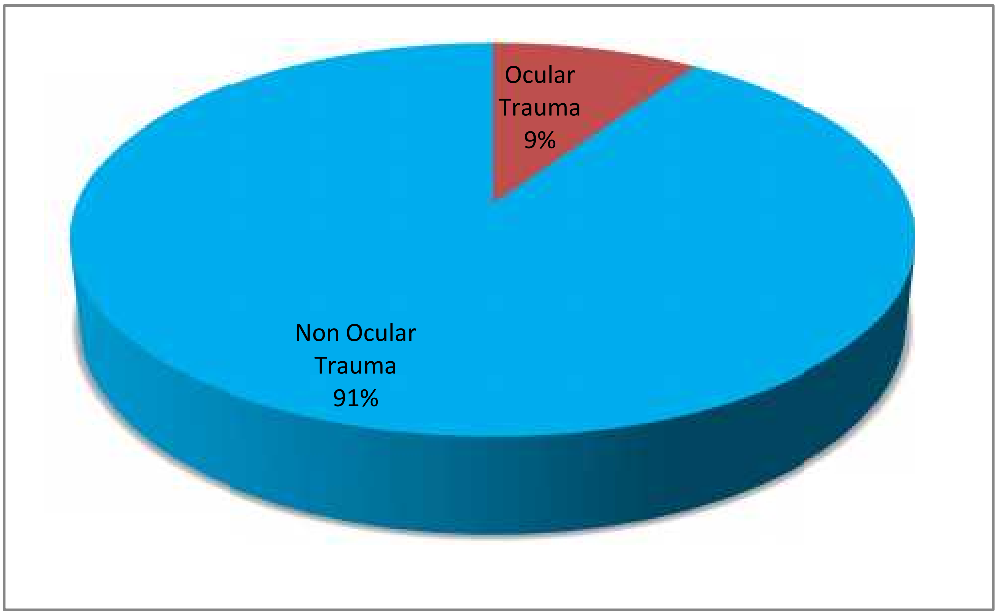
RESULTS

The present study was conducted on 2308 trauma patients who presented to the casualty and the Ophthalmology OPD of KLES Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Table 1 – Prevalence of Ocular Trauma

Cases	Number of cases	Percentage
Ocular Trauma	217	9.40
Non Ocular Trauma	2091	90.6
Total	2308	100

Chart 1 – Prevalence of Ocular trauma

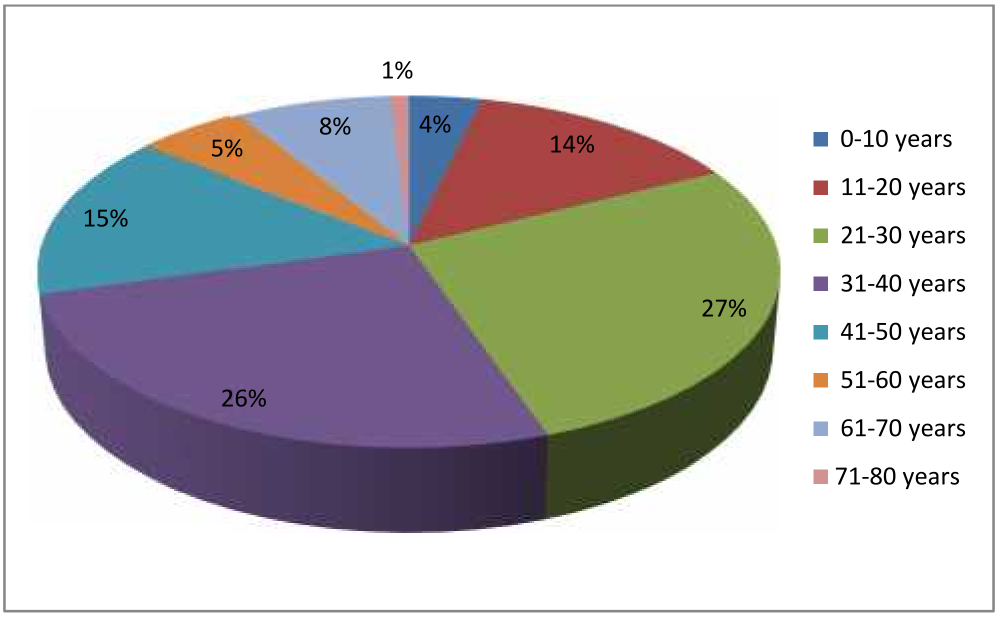


Out of the 2308 patients, 217 patients presented with ocular trauma, resulting in prevalence rate of trauma as 9.40%.

Table 2 – Age wise distribution of patients

Age group (in years)	Number of patients	Percentage
0 - 10	8	3.69
11 – 20	30	13.84
21 – 30	59	27.19
31 – 40	56	25.81
41 – 50	33	15.21
51 – 60	12	5.52
61 – 70	17	7.82
71 – 80	2	0.92
Total	217	100

Chart 2 - Age wise distribution of patients



The mean age of patients was 34.47±19.49 years. Maximum patients (27.19%) were in the age group of 21-30 years and minimum patients (0.92%) belonged to the age group of 71-80 years. This young age group is most susceptible as they are people who have acquired new driving licenses, don’t have much driving experience and hence cause RTA’s.

Table 3 – Sex distribution

Sex	Number of patients	Percentage
Male	182	83.87
Female	35	16.13
Total	217	100

Chart 3 - Sex distribution

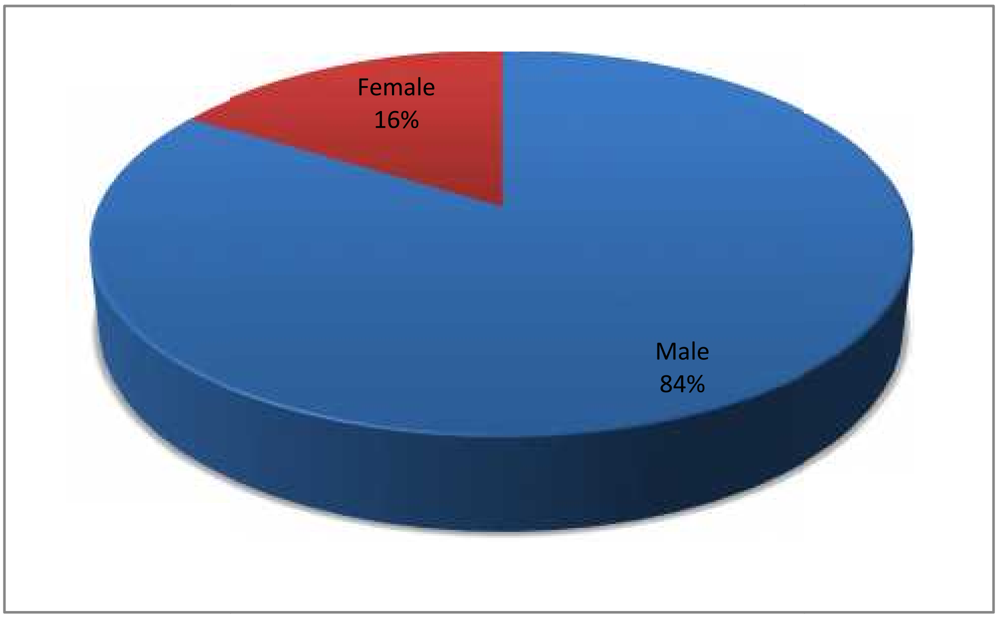
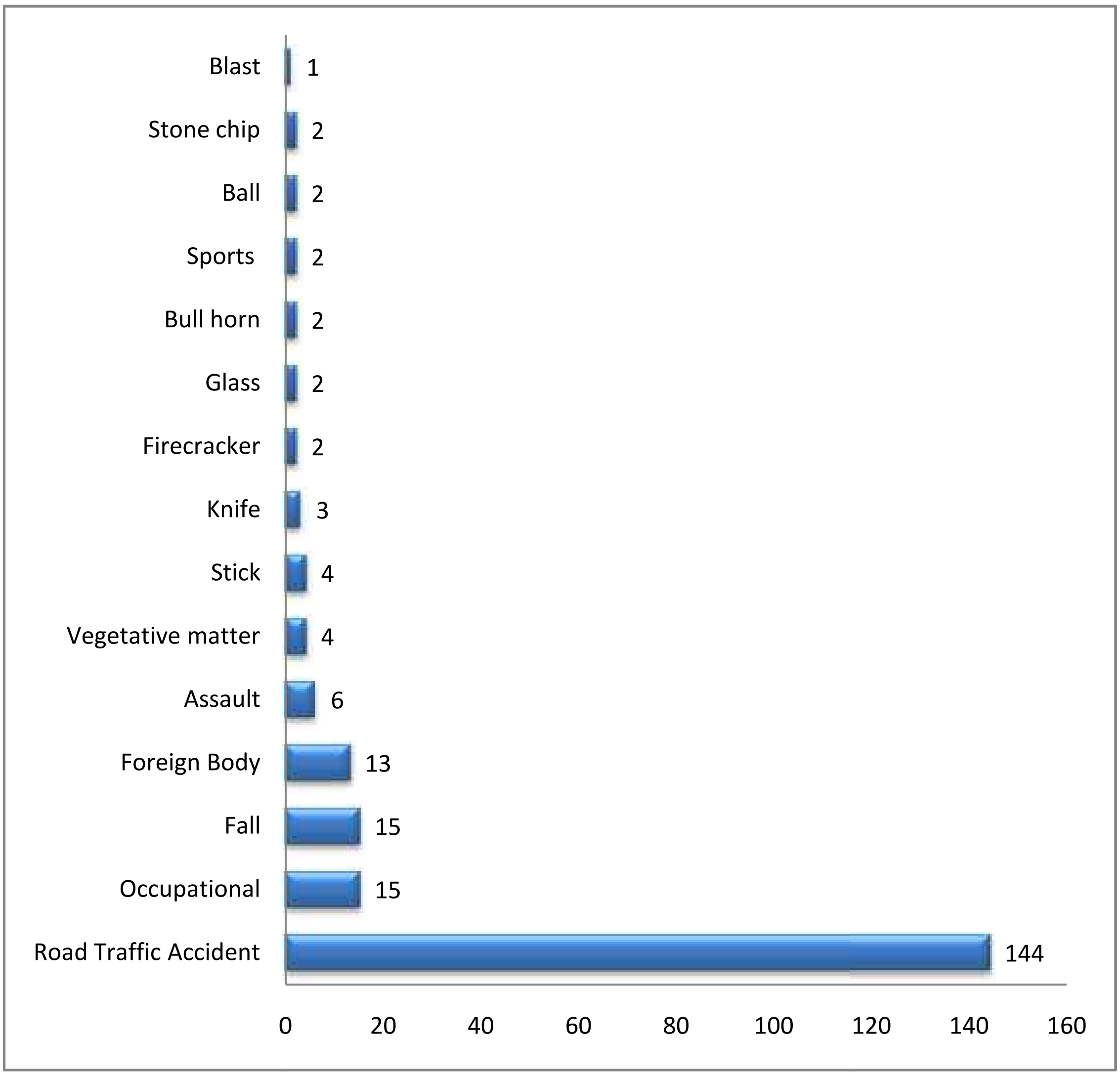


Table 3 shows 182 (83.87%) patients were males and 35 (16.13%) patients were females. Male:Female ratio was 5.2:1. Males are affected more because of more outdoor activity as compared to females and also more risk taking behavior.

Table 4 – Etiology

Etiology	Number of patients	Percentage
Road Traffic Accident	144	66.36
Occupational	15	6.91
Fall	15	6.91
Foreign Body	13	5.99
Assault	6	2.76
Vegetative matter	4	1.84
Stick	4	1.84
Knife	3	1.38
Firecracker	2	0.92
Glass	2	0.92
Bull horn	2	0.92
Sports	2	0.92
Ball	2	0.92
Stone chip	2	0.92
Blast	1	0.46
Total	217	100

Chart 4 – Etiology

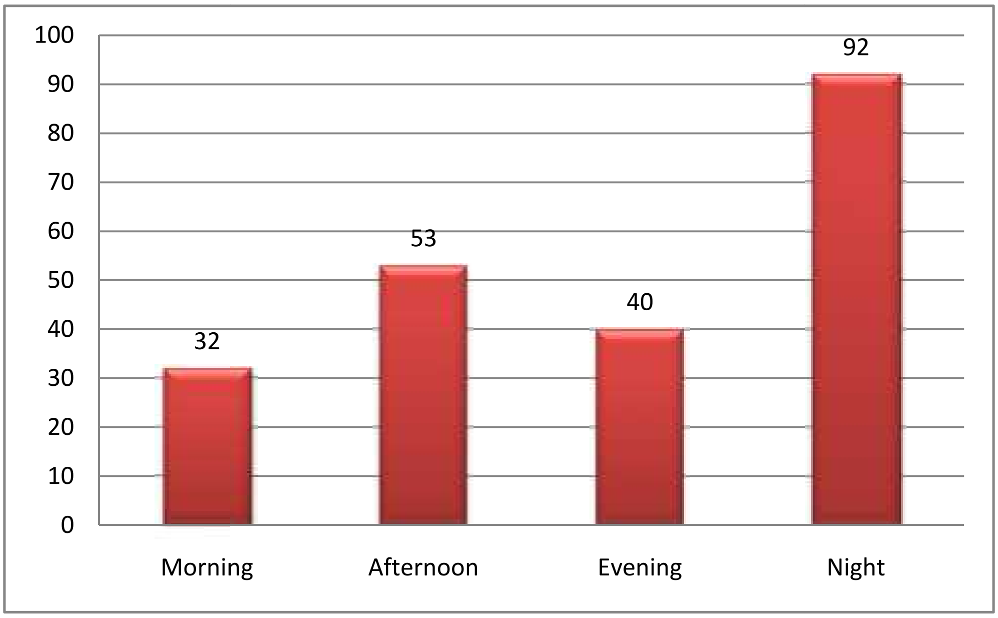


Most common cause of ocular trauma was RTA (66.36%), followed by occupational injury (6.91%) and fall from height (6.91%). Foreign bodies entering the eye accounted for 6% of injuries. Other causes which were responsible for 1-2% of injuries included assault, vegetative matter, stick, knife, firecracker, bull horn, glass, ball, sports related, stone chip and blast injury. Ocular trauma due to RTA was mostly because the two wheeler riders were not wearing a helmet while riding. Reason for RTA being the most common can be attributed to drunk driving, not obeying traffic rules and less driving experience amongst young drivers.

Table 5 – Time of Injury

Time of the day	Number of patients	Percentage
Morning	32	14.75
Afternoon	53	24.42
Evening	40	18.43
Night	92	42.40
Total	217	100

Chart 5 – Time of Injury

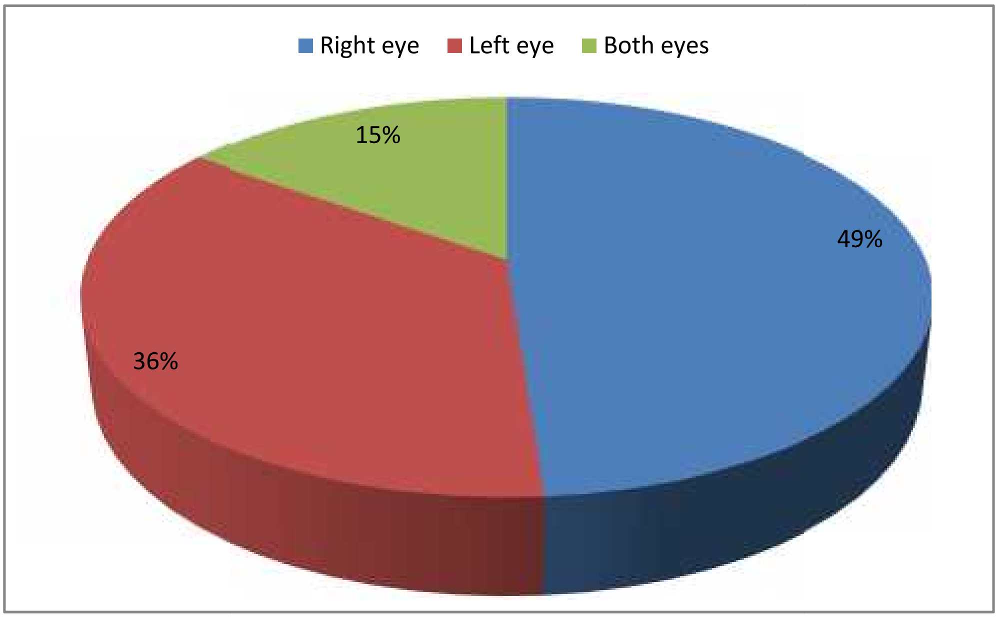


Time of the day was divided into Morning (5 AM – 12 PM), Afternoon (12 PM – 4 PM), Evening (4 PM – 8 PM) and Night (8 PM – 5 AM). In our study, 92 (42.40%) patients were injured in the night followed by 53 (24.42%) at afternoon, 40 (18.43%) in the evening and 32 (14.75%) in the morning.

Table 6 – Eye affected

Eye affected	Number of patients	Percentage
Right eye	106	48.85
Left eye	78	35.94
Both eyes	33	15.21
Total	217	100

Chart 6 – Eye affected

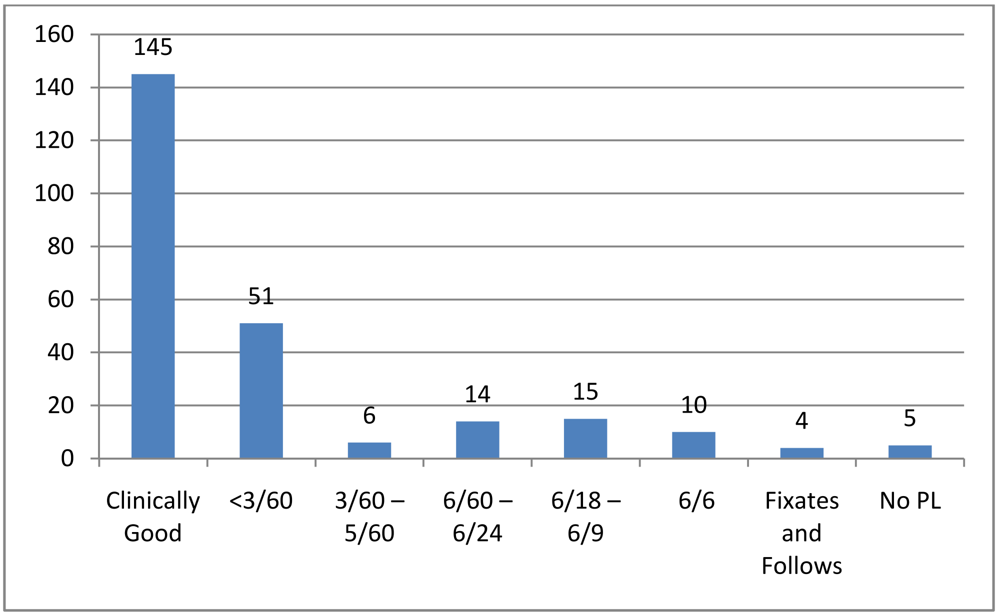


Right eye was involved in 106 (48.85%) patients followed by left eye in 78 (35.94%). In 33 (15.21%) patients, both eyes were involved.

Table 7 – Visual Acuity at Presentation

Vision	Number of eyes	Percentage
Clinically Good	145	58
<3/60	51	20.4
3/60 – 5/60	6	2.4
6/60 – 6/24	14	5.6
6/18 – 6/9	15	6
6/6	10	4
Fixates and Follows	4	1.6
No PL	5	2
Total	250	100

Chart 7 – Visual Acuity at Presentation

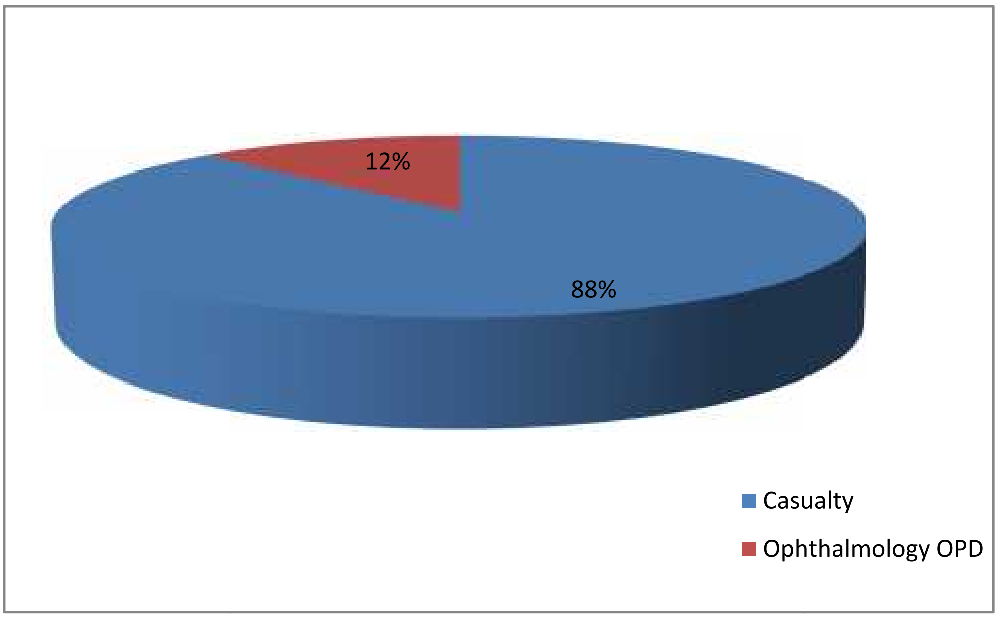


Clinically good vision was considered as vision of counting fingers at a distance of more than 3 meters when recorded in the absence of a visual acuity chart in the casualty. 145 eyes (58%) had clinically good vision whereas no perception of light was present in 5 eyes (2%).

Table 8 – Place of presentation

Place	Number of patients	Percentage
Casualty	190	87.56
Ophthalmology OPD	27	12.44
Total	217	100

Chart 8 – Place of presentation

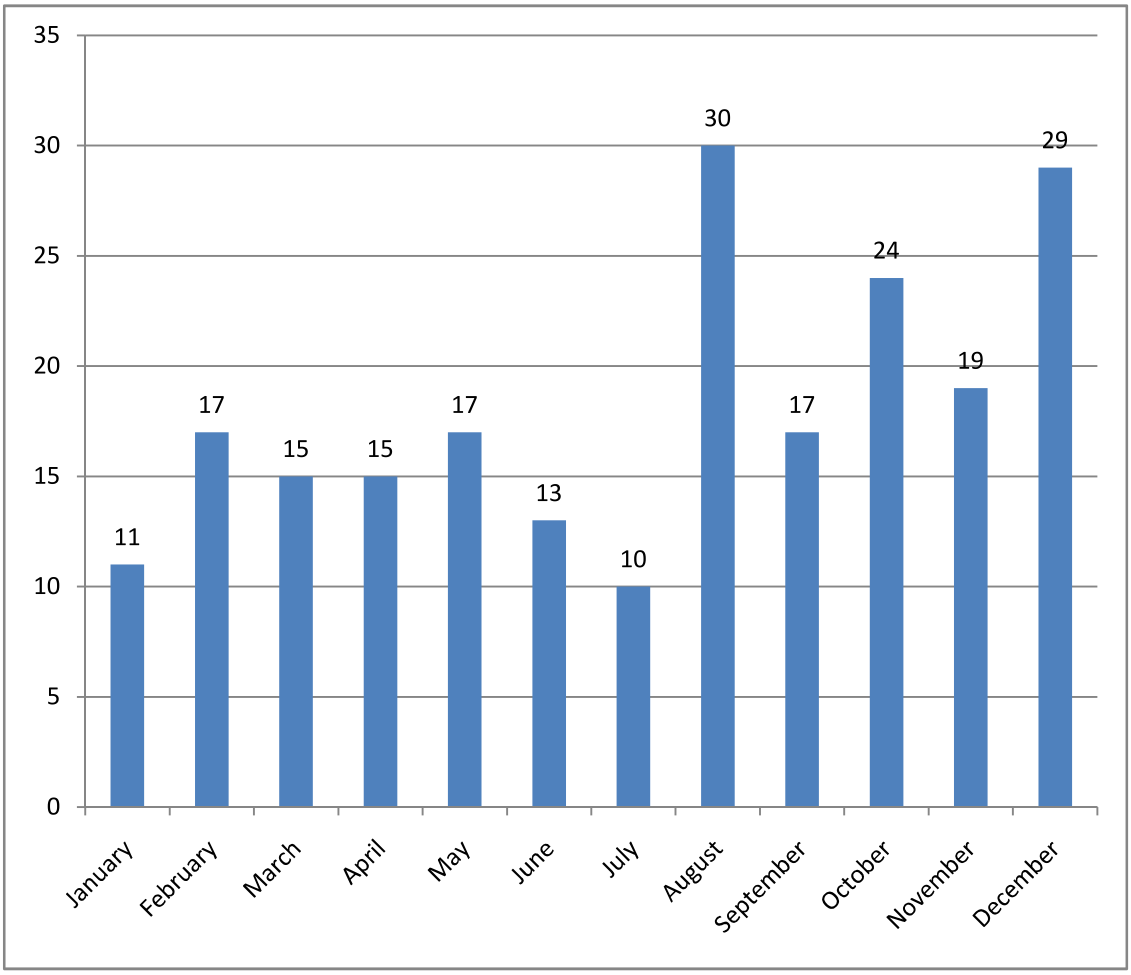


190 (87.56%) patients presented to the casualty of the hospital whereas 27 (12.44%) presented to the Ophthalmology OPD. Since most of the cases were due to RTA, they presented as emergency cases to the hospital casualty.

Table 9 – Month of trauma

Month	Number of patients	Percentage
January	11	5.07
February	17	7.83
March	15	6.91
April	15	6.91
May	17	7.83
June	13	5.99
July	10	4.61
August	30	13.82
September	17	7.83
October	24	11.21
November	19	8.76
December	29	13.36
Total	217	100

Chart 9 – Month of trauma

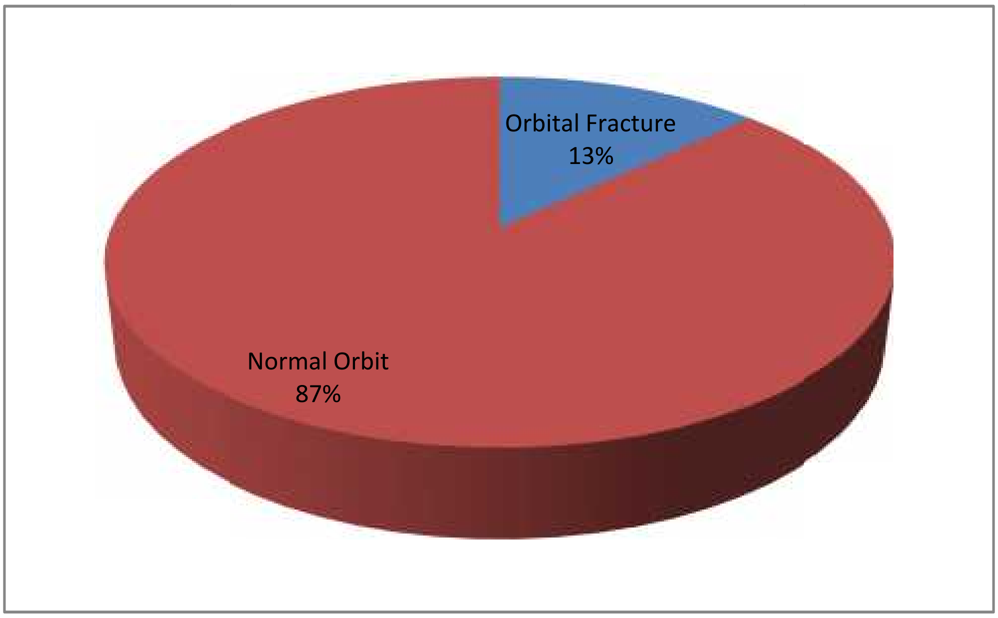


Maximum incidence of ocular trauma was seen in the months of August (13.82%) and December (13.36%) and minimum incidence was seen in the month of July (4.61%). More cases are seen in August possibly due to rainy season at that time, hence more chances of RTA.

Table 10 – Orbital Fractures

State of Orbit	Number of patients	Percentage
Orbital Fractures	28	12.90
Normal Orbit	189	87.10
Total	217	100

Chart 10 – Orbital Fractures

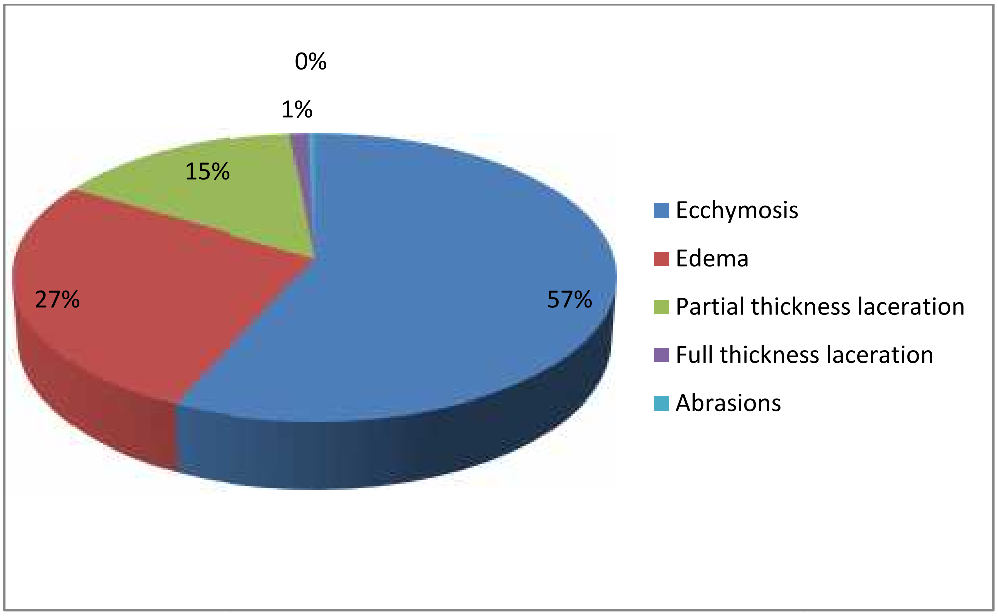


Out of 217 ocular trauma patients, 28 (12.90%) patients had orbital fractures.

Table 11 – Eyelid findings

Findings	Number of eyes	Percentage
Ecchymosis	142	57.79
Edema	73	27.14
Partial thickness laceration	41	15.24
Full thickness laceration	3	1.12
Abrasions	10	0.37
Total	269	100

Chart 11 – Eyelid findings

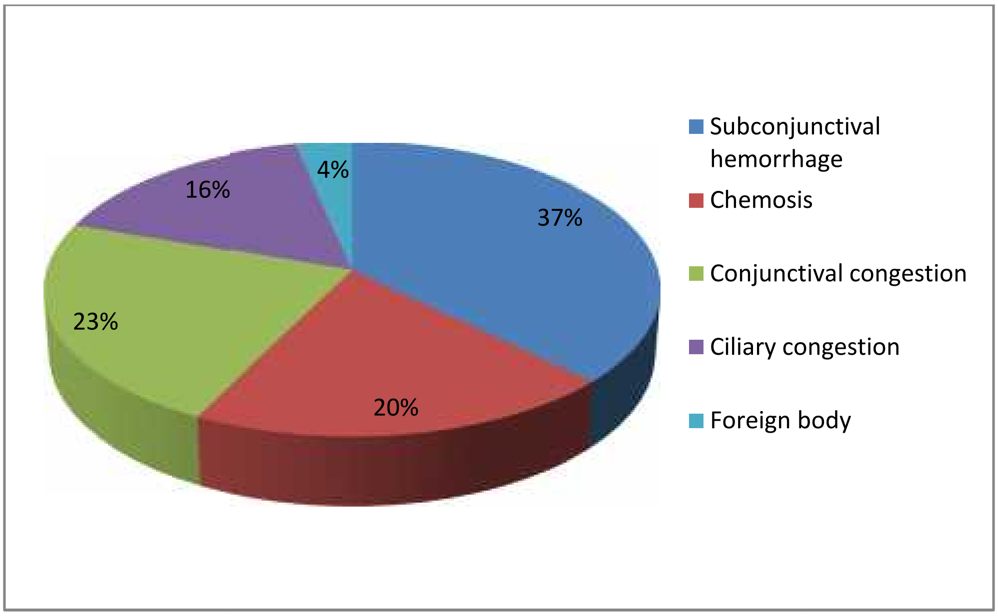


Out of total 217 ocular trauma patients, 184 (84.79%) patients had eyelid involvement. Out of 368 eyes of 184 patients, 269 eyes had eyelid lesions. Out of 269 involved eyes, 142 (57.79%) eyes had Ecchymosis, 73 (27.14%) eyes had lid edema, 41 (15.24%) eyes had partial thickness laceration, 3 (1.12%) eyes had full thickness laceration and 10 (0.37%) eyes had abrasions over the lid.

Table 12 – Conjunctival findings

Findings	Number of eyes	Percentage
Subconjunctival hemorrhage	78	37.32
Chemosis	42	20.10
Conjunctival congestion	48	22.97
Ciliary congestion	34	16.27
Foreign body	7	3.35
Total	209	100

Chart 12 – Conjunctival findings

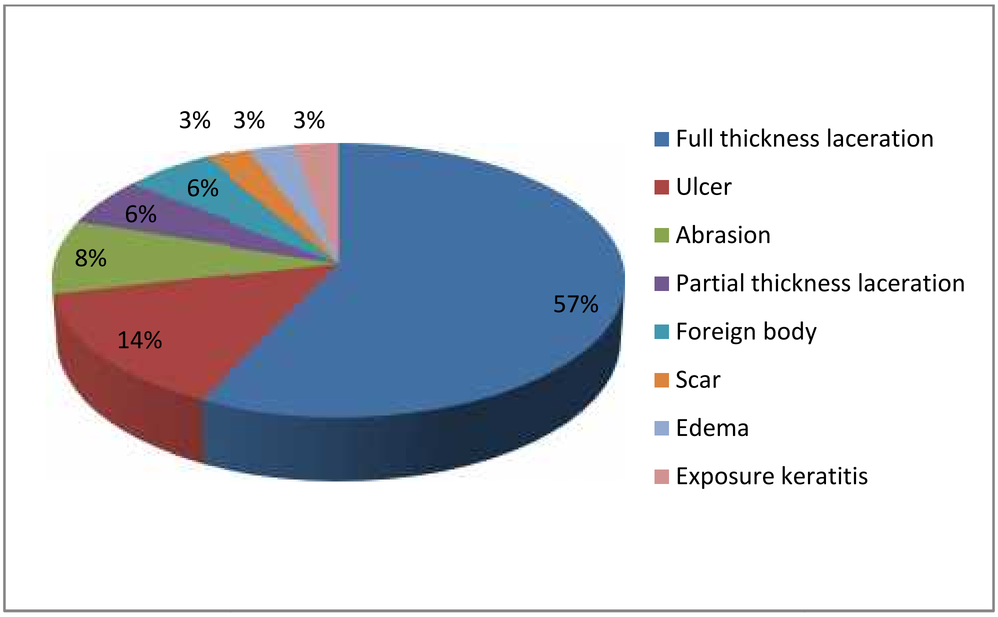


155 (71.43%) patients out of 217 patients had findings in the conjunctiva as well apart from other structures. Out of 310 eyes of these 155 patients, conjunctiva was involved in 209 eyes. Amongst the 209 eyes, 78 (37.32%) eyes had subconjunctival hemorrhage, 42 (20.10%) eyes had chemosis, 48 (22.97%) eyes conjunctival congestion, 34 (16.27%) eyes had ciliary congestion and 7 (3.35%) eyes had foreign bodies in them.

Table 13 – Corneal findings

Findings	Number of eyes	Percentage
Full thickness laceration	20	57.12
Ulcer	5	14.29
Abrasion	3	8.57
Partial thickness laceration	2	5.71
Foreign body	2	5.71
Scar	1	2.86
Edema	1	2.86
Exposure keratitis	1	2.86
Total	35	100

Chart 13 – Corneal findings

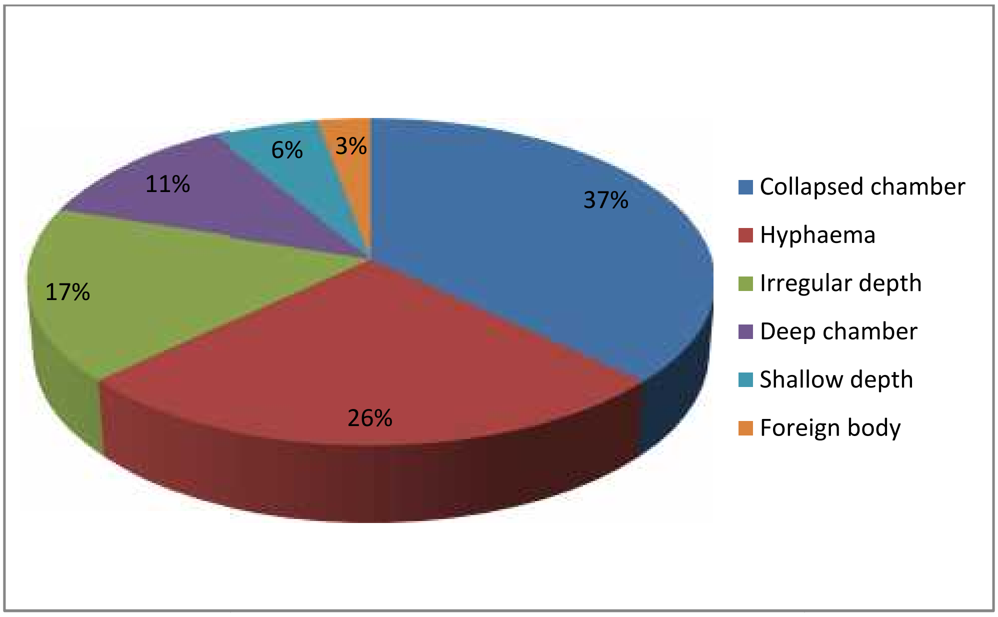


Out of 217 patients, 34 (15.67%) patients had corneal findings. Out of the 68 eyes of 34 patients, 20 (57.12%) eyes had full thickness laceration, 5 (14.29%) eyes had an ulcer, 2 (5.71%) eyes had partial thickness laceration and another 2 eyes had foreign bodies. 3 eyes had corneal scar, exposure keratitis and corneal edema respectively.

Table 14 – Anterior chamber findings

Findings	Number of eyes	Percentage
Collapsed chamber	13	37.14
Hyphaema	9	25.71
Irregular depth	6	17.14
Deep chamber	4	11.43
Shallow depth	2	5.71
Foreign body	1	2.86
Total	35	100

Chart 14 – Anterior chamber findings

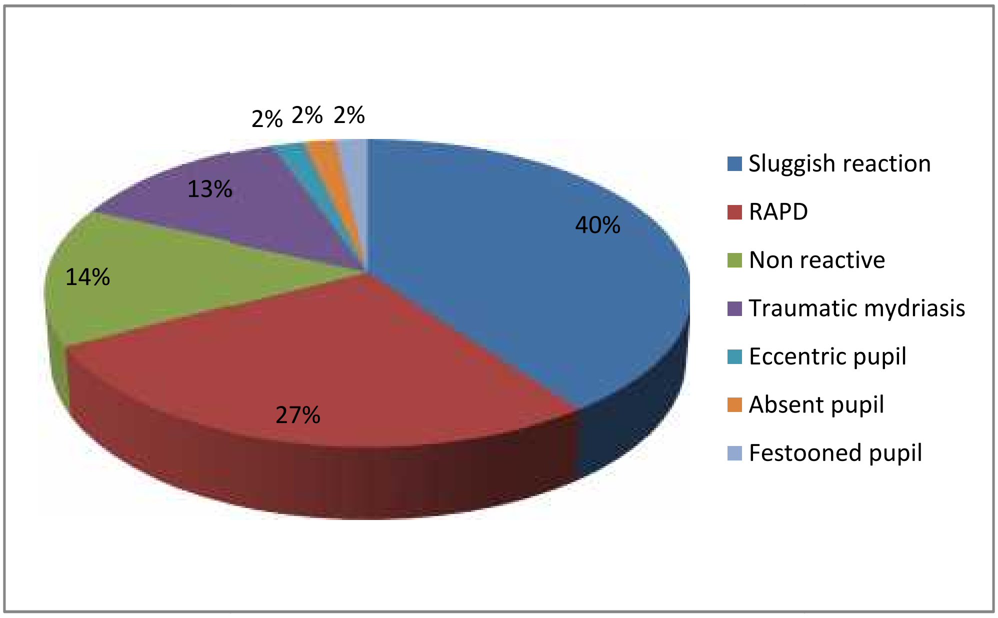


27 (12.44%) patients out of 217 patients had finding in their anterior chambers. Out of 54 eyes of 27 patients, 13 (37.14%) eyes had a collapsed chamber, 9 (25.71%) eyes had a hyphaema, 6 (17.14%) eyes had an irregular chamber depth, 4 (11.43%) eyes had a deep chamber, 2 (5.71%) eyes had shallow chambers and 1 (2.86%) eye had an intra ocular foreign body in the anterior chamber.

Table 15 – Pupil findings

Findings	Number of eyes	Percentage
Sluggish reaction	22	40.00
RAPD	15	27.27
Non reactive	8	14.55
Traumatic mydriasis	7	12.73
Eccentric pupil	1	1.82
Absent pupil	1	1.82
Festooned pupil	1	1.82
Total	55	100

Chart 15 – Pupil findings

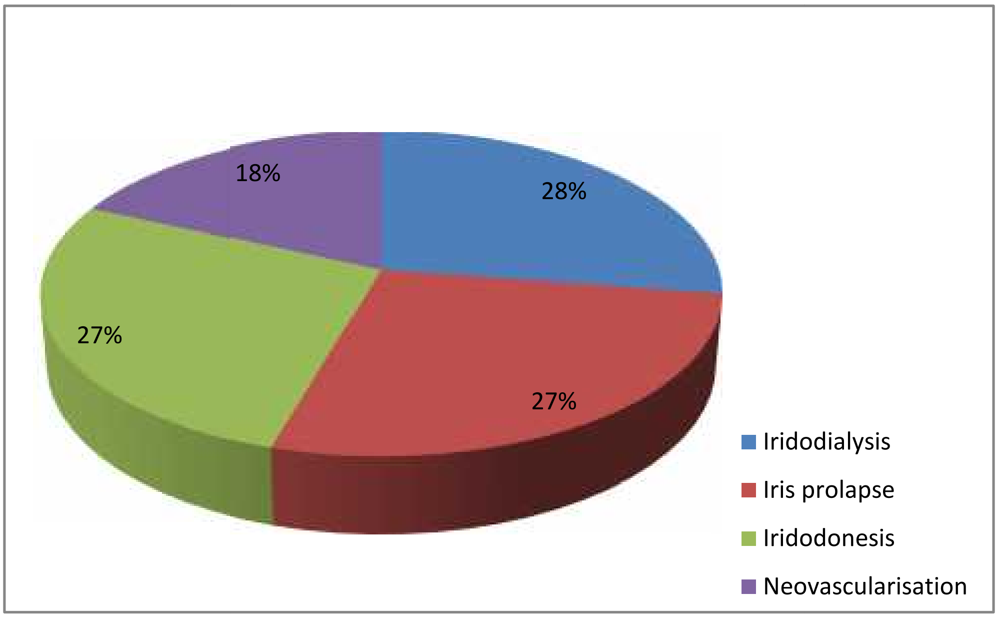


Out of 217 ocular trauma patients, 54 (24.88%) had pupillary findings. Out of 108 eyes of 54 patients, 55 (50.93%) eyes had pupillary findings. Out of the 55 eyes, 22 (40%) eyes had a sluggish reaction to light, 15 (27.27%) had a relative afferent pupillary defect and 8 (14.55%) eyes had no reaction to light at all. Traumatic mydriasis was present in 7 (12.73%) eyes and 3 eyes had an eccentric pupil, no pupil due to complete iridodialysis and a festooned pupil, respectively. 20 (4.61%) eyes out of 434 eyes of 217 ocular trauma patients developed Traumatic Optic Neuropathy and all cases were treated medically.

Table 16 – Iris findings

Findings	Number of eyes	Percentage
Iridodialysis	3	27.27
Iris prolapse	3	27.27
Iridodonesis	3	27.27
Neovascularisation	2	18.18
Total	11	100

Chart 16 – Iris findings

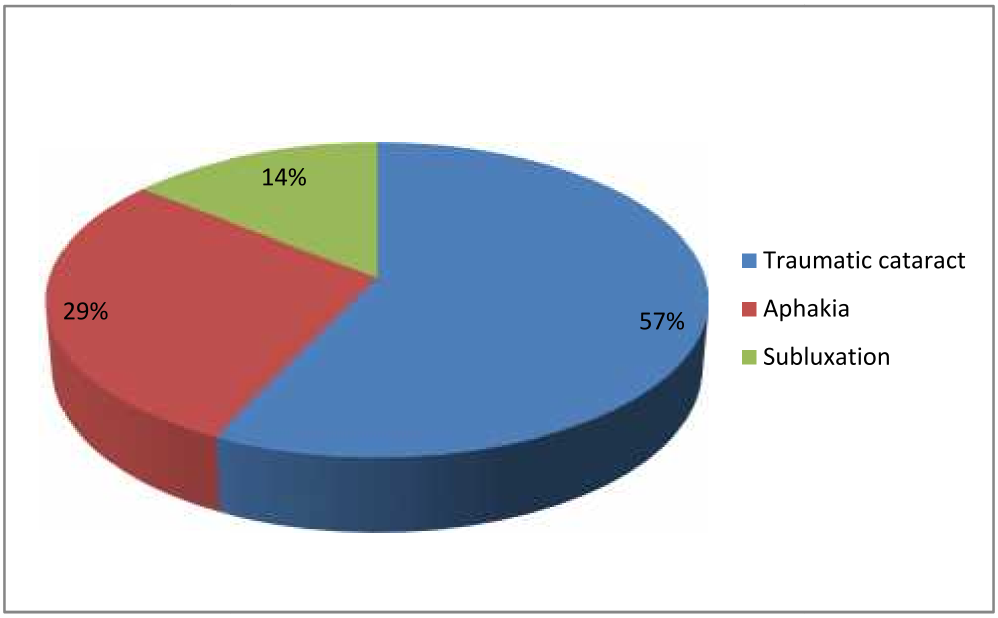


In our study, 11 (5.07%) patients out of 217 patients having ocular trauma had iris findings. 11 (50%) eyes out of 22 eyes of 11 patients had iris findings. Out of 11 eyes, 3 (27.27) eyes had iridodialysis, iris prolapse and iridodonesis respectively. 2 (18.18%) eyes had neovascularisation of the iris.

Table 17 – Lens findings

Findings	Number of eyes	Percentage
Traumatic cataract	8	57.14
Aphakia	4	28.57
Subluxation	2	14.29
Total	14	100

Chart 17 – Lens findings

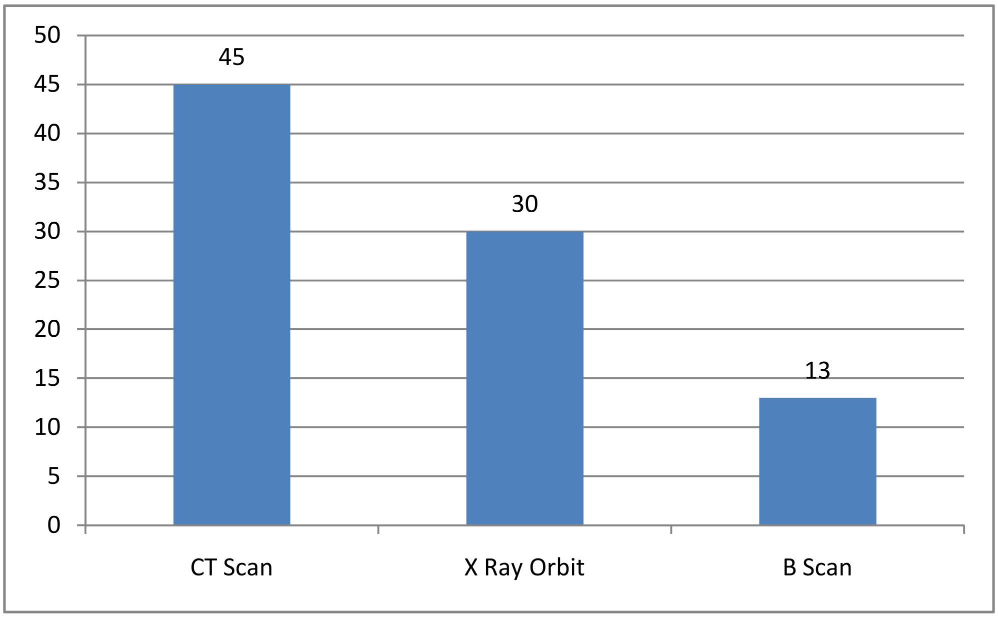


Out of 217 ocular trauma patients, 14 (6.45%) patients had lens involvement. Out of 28 eyes of 14 patients, 14 (50%) eyes had lens findings. Out of the 14 eyes, 8 (57.14%) eyes developed traumatic cataract, 4 (28.57%) eyes had aphakia secondary to trauma and 2 (14.29%) eyes developed subluxation of the lens.

Table 18 – Investigations

Investigation	Number of patients	Percentage
CT Scan Brain Plain	45	51.14
X ray Orbit Plain	30	34.09
B Scan	13	14.77
Total	88	100

Chart 18 – Investigations

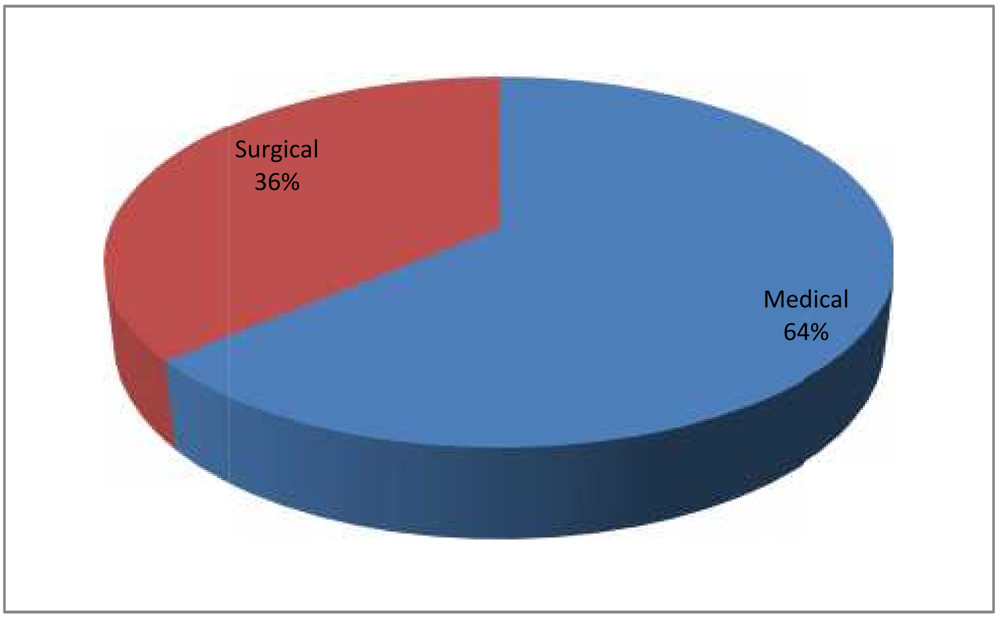


Out of 217 ocular trauma patients, 88 (40.55%) patients underwent investigations. Out of 88 patients, CT Scan brain plain was done for 45 (51.14%) patients, X- ray orbit plain was done for 30 (34.09%) patients and B scan was done for 13 (14.77%) patients. Since most RTA cases had head injuries, CT scan brain plain was ordered by the Neurosurgery consultant for most patients.

Table 19 – Treatment

Treatment	Number of patients	Percentage
Medical	138	63.59
Surgical	79	36.41
Total	217	100

Chart 19 – Treatment



Out of 217 ocular trauma patients, 138 (63.59%) patients underwent medical treatment and the rest 79 (36.41%) patients underwent surgical treatment.

DISCUSSION

Prevalence rate

This study was done on 2308 trauma patients, out of whom 217 patients had ocular trauma resulting in a prevalence rate of 9.40%.

The prevalence rate is in conformity with The Andhra Pradesh Eye Disease Study[6] where 824 patients out of 7771 patients had ocular trauma giving a prevalence rate of 10.6%. The Beaver Dam Eye Study[4] included 4926 patients out of whom, 972 patients had ocular trauma resulting in a prevalence rate of 19.8%. Another study, The Aravind Comprehensive Eye Study[5] calculated the prevalence rate to be 4.5% which included 5150 patients out of whom 229 patients had ocular trauma.

Age and Sex

The mean age of patients was found to be 34.47±19.49 years. Maximum patients (27.19%) were in the age group of 21-30 years and minimum patients (0.92%) belonged to the age group of 71-80 years.

182 (83.87%) patients were males and 35 (16.13%) patients were females and male:female ratio was 5.2:1 in our study.

According to a study conducted by Parul Desai et al[62], a bimodal peak occurs in the older age groups. They also reported higher rates of injury in the age group of 15-64 years and lower rates for the age group of 0-14 years and 65 years and older age group. Males were affected in 80% (32 patients) cases and females in 20% cases (8 patients), which is consistent with other studies. The Aravind Comprehensive Eye Study[5] reported that blunt trauma (n = 124; 54.9%) was the major cause of ocular trauma in the South Indian rural population. They also reported a higher odds ratios (OR) for trauma in males (OR, 2.2; 95% CI, 1.6-3.0).[5] This is well supported by a study conducted by V K Gothwal[63] in Hyderabad where males were involved in 86.8% of cases of ocular trauma. The most likely cause for a higher number of males being affected could be attributed to the fact that males are more likely to be involved in outdoor activities.

Etiology

In our study, most common cause of ocular trauma was RTA (66.36%), followed by occupational and fall from height (6.91%).

Foreign bodies entering the eye accounted for 6% of injuries.

Other causes which were responsible for 1-2% of injuries included assault, vegetative matter, stick, knife, firecracker, bull horn, glass, ball, sports related, stone chip and blast injury.

According to Guly C M et al[1], 57.3% of ocular injuries were caused due to road traffic accidents (RTAs). In a study conducted by Vats S et al[64], they found out that majority of the ocular injuries were sustained at work and home, and blunt trauma (41.7%) was more commonly implicated than sharp objects (19.6%). In The Beaver Dam Eye Study[4], it was found that the most common cause of injury was trauma resulting from sharp objects which occurred in 54.1% of the participants.

Eye laterality

In our study, right eye was involved in 106 (48.85%) patients and the left eye in 78 (35.94%). In 33 (15.21%) patients, both eyes were involved.

This coincides with The Beaver Dam Eye Study[4] where, both eyes were involved in 15.4%, right eye in 30.5% and the left eye in 27.8% of participants. Rest 26.3% participants couldn't remember which eye was involved.

Vision

145 eyes (58%) had clinically good vision whereas no perception of light was present in 5 eyes (2%) in our study. Clinically good vision was considered as vision of counting fingers at a distance of more than 3 meters.

51 (20.4%) eyes had vision less than 3/60, 6 (2.4%) eyes had a visual acuity between 3/60 and 5/60, 14 (5.6%) eyes had vision between 6/60 and 6/24, 15 (6%) eyes had vision between 6/18 and 6/9, 10 (4%) eyes had 6/6 vision and 5 (2%) eyes presented with no perception of light.

In the Andhra Pradesh Eye Disease Study[6], 4.61% patients presented blind (best corrected distance visual acuity less than 6/60 in the better eye) due to trauma and another 3.4% had visual impairment worse than 6/12 to 6/60 in one eye due to trauma. According to The Aravind Comprehensive Eye Survey[5] identified trauma as the underlying cause for vision impairment (vision between 6/18 and 3/60 after best correction for that eye) in 27.2% eyes including 15.2% of these eyes that were blind (vision worse than 3/60 after best correction for that eye). In a study conducted by McCarty et al[65], out of the 1197 injures eyes, 6.3% eyes had visual acuity less than 6/18.

Clinical manifestations of ocular trauma

Ocular adnexal findings

Out of 217 ocular trauma patients in our study, 28 (12.90%) patients had orbital fractures.

Out of 269 eyes with eyelid involvement, 142 (57.79%) eyes had ecchymosis, 73 (27.14%) eyes had lid edema, 41 (15.24%) eyes had partial thickness laceration, 3 (1.12%) eyes had full thickness laceration and 10 (0.37%) eyes had abrasions over the lid.

Anterior segment findings

Amongst 209 eyes where the conjunctiva was affected, 78 (37.32%) eyes had subconjunctival hemorrhage, 42 (20.10%) eyes had chemosis, 48 (22.97%) eyes conjunctival congestion, 34 (16.27%) eyes had ciliary congestion and 7 (3.35%) eyes had foreign bodies in them.

Out of the 68 eyes with corneal involvement, 20 (57.12%) eyes had full thickness laceration, 5 (14.29%) eyes had an ulcer, 2 (5.71%) eyes had partial thickness laceration and another 2 eyes had foreign bodies. 3 eyes had corneal scar, exposure keratitis and corneal edema respectively.

Out of 54 eyes where the anterior chamber was involved, 13 (37.14%) eyes had a collapsed chamber, 9 (25.71%) eyes had a hyphaema, 6 (17.14%) eyes had an irregular chamber depth, 4 (11.43%) eyes had a deep chamber, 2 (5.71%) eyes had shallow chambers and 1 (2.86%) eye had an intra ocular foreign body in the anterior chamber.

Out of 55 eyes with pupillary findings, 22 (40%) eyes had a sluggish reaction to light, 15 (27.27%) had a relative afferent pupillary defect and 8 (14.55%) eyes had no reaction to light at all. Traumatic mydriasis was present in 7 (12.73%) eyes and 3 eyes had an eccentric pupil, no pupil due to complete iridodialysis and a festooned pupil, respectively. 20 (4.61%) eyes out of 434 eyes of 217 ocular trauma patients developed traumatic optic neuropathy and all cases were medically treated.

Out of 11 eyes having iris involvement, 3 (27.27) eyes had iridodialysis, iris prolapse and iridodonesis respectively. 2 (18.18%) eyes had neovascularisation of the iris.

Out of the 14 eyes with lens findings, 8 (57.14%) eyes developed traumatic cataract, 4 (28.57%) eyes had aphakia secondary to trauma and 2 (14.29%) eyes developed subluxation of the lens.

As per The Andhra Pradesh Eye Disease Study[6], amongst the 39 cases of blindness from ocular trauma, 33.3% eyes had traumatic cataract, another 33.3% had traumatic corneal scars, 20.5% were phthisical, 10.3% had traumatic optic atrophy and 2.6% had surgical anophthalmia. In a study conducted by Vats et al[64], amongst 158 participants, 39.9% had corneal opacities following trauma, 19.6% had traumatic cataract, 12.1% had iris tears, 3.2% had iridodialysis, 0.6% had hyphaema, 1.9% had corneal epithelial defects, 1.3% had subconjunctival hemorrhage, 5.1% had conjunctival tears, 8.2% had lid scars, 4.4% had angle recession, 1.3% had retinal detachment and 0.6% each had a macular hole, a macular scar and optic neuropathy, 3.2% had subluxated lenses and 2.6% had phthisis bulbi.

Management

Out of 217 ocular trauma patients in our study, 138 (63.59%) patients underwent medical treatment and the rest 79 (36.41%) patients underwent surgical treatment.

These findings nearly co-relate with the study conducted by Vats et al[64] where 85.3% patients were treated with eye drops and/or oral drugs, and surgery was performed in 11%. Hospitalization was required in 3.7% cases.

CONCLUSION

The present study was conducted in the Department of Ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi on 2308 trauma patients presenting to the Ophthalmology OPD and the casualty of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi between 1st January 2014 and 31st December 2014. Out of the 2308 patients, there were 217 patients who had ocular trauma.

On conclusion of this study, prevalence of ocular trauma was found to be 9.40%.

Most of the injuries involved the ocular adenexa, which while causing certain degree of cosmetic disfigurement did lead to permanent visual sequelae in some patients, injuries involving cornea or sclera had bad prognosis and those with optic neuropathy had the worst prognosis.

Most common cause of ocular trauma was found to be road traffic accidents (RTA), seen most commonly amongst males in the age group of 21-30 years. As seen from study data, the people of the most active and productive age groups were involved in RTA, which leads to cosmetic disfigurement and serious economic loss to the community. Males are commonly injured in RTA which is probably related to both exposure and risk taking behavior.

Several human and environmental risk factors were found to be associated with increased risk of RTA e.g. lack of awareness of traffic rules, inadequate enforcement of existing laws, easy accessibility to license and driving under the influence of alcohol. If we control the factors appropriately, mortality and morbidity can be prevented.

Recommendations for the prevention of ocular injuries in RTA

1. Passengers sitting in the front seats more commonly sustain ocular trauma.
    - The use of safety seat belts must be made compulsory.
    - All road vehicles must have laminated glass windscreens.
2. The practice of seating younger children on the lap of a parent on one of the front seats should not be allowed.
3. There is an urgent need for education of the public through the use of news media and television programmes.
    - The requirement of wearing seat belts in four wheelers and helmets on two wheelers.
    - Observation of the safety rules of the road.
    - Punishment for reckless driving and dangerous overtaking.
4. The use of unbreakable plastic spectacles should be encouraged.
5. Road markings guiding traffic and drivers need to be re-painted more frequently. Paint should be fluorescent so as to be clearly visible even in darkness.

Apart from RTA, occupational hazard was the next major cause of ocular trauma. Certain work place laws and the use of protective eye wear while working can go a long way in reducing this cause of trauma.

Hence with the data of this study, preventive and management strategies can be formulated to manage ocular trauma in a better way.

SUMMARY

Although the eyes represent only 0.1% of the total body surface, most of the information reaches humans through vision. *Ocular trauma* causes a great socio-economic impact. Those affected by eye injury often have to face loss of career opportunities, major lifestyle changes and occasionally permanent disfigurement.

The present study was conducted on 2308 trauma patients presenting to the casualty and Ophthalmology OPD of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from 1st January 2014 till 31st December 2014. Out of the 2308 patients, 217 patients were found to have ocular trauma which resulted in a prevalence rate of 9.40%.

Road traffic accident was the most common cause; male patients within the age group of 21 to 30 years were involved. Ecchymosis of the lid and subconjunctival hemorrhage were the most common findings, followed by partial thickness lid laceration and corneo-scleral laceration.

The patients underwent CT scan, X ray and B scan for better diagnosis of the lesions. More than half of the patients were medically managed and the rest required surgical intervention.

With the help of certain laws, legislations and public awareness, maximum causes of ocular trauma can be reduced drastically. Hence with the data of this study, preventive and management strategies can be formulated to manage ocular trauma in a better way.

BIBLIOGRAPHY

1. Guly CM, Guly HR et al. Ocular injuries in patients with Major trauma. Emergency Med Journal. 2006; 23: 915-917.
2. Rose J, Raju R. Cause and Clinical Profile of 379 cases of Ocular Trauma. Delhi Journal of Ophthalmology. 2010 Oct-Dec; 21(3): 48-50
3. Shtewi M EI et al. Road traffic accidents and ocular trauma. Journal of Community Eye Health. 1999; 12 (29): 11-12.
4. Wong T Y, Klien B E K, Klein R. The Prevalence and 5-year Incidence of Ocular Trauma. *The Beaver Dam Eye Study.* Ophthalmology. 2000; 107: 2196-2202.
5. Nirmalan P K et al. Ocular Trauma in a Rural South Indian Population. *The Aravind Comprehensive Eye Survey*. Ophthalmology. 2004; 111: 1778-1781.
6. Krishnaiah S et al. Ocular Trauma in a Rural Population of Southern India. *The Andhra Pradesh Eye Disease Study.* Ophthalmology. 2006; 113: 1159-1164.
7. Sumana Kamath J, Kamath MG. A study of orbital fractures in a Tertiary Health Care Centre. Journal of Health and Allied Sciences. Jan-Mar 2007; 6(1): 1-4.
8. Soong TK, Koh A, Subrayan V, Loo AV. Ocular trauma injuries: a 1-year surveillance study in the University of Malaya Medical Centre, Malaysia. Graefes Arch Clin Exp Ophthalmol. 2011 Dec; 249(12): 1755-60.
9. Serinken M et al. Causes and characteristics of work-related injuries in western Turkey. Indian Journal of Ophthalmology. 2013 Sept; 61(9): 497-501
10. Kuhn F et al. A standardized classification of ocular trauma terminology. Ophthalmology. 1996; 103: 240-243

11. Sihota Ramanjit, Tandon Radhika. Parsons' Diseases of the Eye: Injuries to the Eye. 20th edition. Uttar Pradesh, India: Elsevier; 2007. Page No.364.
12. Greenwald MJ, Boston D, Pensler JM, et al. Orbital roof fracture in childhood. Ophthalmology 1989; 96: 491-496.
13. Nolasco FP, Mathog RH. Medial orbital wall fractures: classification and clinical profile. Otolaryngol Itcod Nec Surg. 1995; 112: 49-56.
14. Kersten RC. Blowout fracture of the orbital floor with entrapment caused by isolated trauma to the orbital rim. Am J Ophthalmol. 1987; 103: 215-219.
15. White house RW, Batterbury M, Jackson A, et al. Prediction of enophthalmos by computed tomography after blow out orbital fracture. Br J Ophthalmol 1994; 78: 618-620.
16. Slingsby JG and Forstot SL. Effect of blunt trauma on the corneal endothelium. Arch Ophthalmol. 1981; 99: 1041-1043.
17. Topping TM et al. Traumatic wound dehiscence following penetrating keratoplasty. Br J Ophthalmol. 1982; 66: 174-178.
18. Elizabeth M Eagling. Ocular damage after blunt trauma to the eye. Br J Ophthalmol. 1974; 58: 126-140.
19. Thomas C. Spoor, Geoffrey M. Kwitko, John M O'Grady and John M.O' Grady and John M Ramocks. Traumatic hyphaema in urban population. American Journal of Ophthalmology. 1990; 109: 23-25.
20. James P. Ganley, Michoel J. Geiger, Joh, R. Clement Perry G. Rigby and Gory J. Levy. Aspirin and recurrent hyphema after blunt ocular trauma; American Journal of Ophthalmology. 1983; 96; 124-127
21. Edwards WC, Layden WF. Traumatic hyphaema. Am L Ophthalmol. 1993; 75: 110-116.

22. Wolfe SM and Zimmerman LE. Chronic secondary glaucoma associated with retro displacement of iris root and deepening of the anterior chamber angle secondary to contusion. Am J Ophthalmol. 1962; 54: 547-563.

23. Read J and Golberg MF. Comparison of medical treatment for traumatic hyphema. Trans Aad Opthalmol Otolaryngol 1974; 78: 799-815.

24. Kennedy RH and Brubaker RF. Traumatic hyphaema in a defined population, Am J Ophthalmol 106; 123-130, 1988.

25. Agapitos PJ, NOCL LP, and Clarke, WN. Traumatic hyphema in children, Ophthalmology. 1987; 94: 1238-1241.

26. Wilson FM. Traumatic hyphema: Pathogenesis and Management. Ophthalmology. 1962; 54: 547-563.

27. Deutsh TA and Goldberg MF. Traumatic hyphaema: Medical and surgical management. In Focal Points: Clinical modules for Ophthalmologists San Francisco: American Academy of Ophthalmology; 1984: no.5.

28. Hoskins HD and Kass MA. Becker-Shaffer's Diagnosis and Therapy of the Glaucomas: Secondary open angle glaucoma. 8th Edition. St. Louis: Elsevier; 2007. Page no. 276.

29. Shields MB. Shields Text book of Glaucoma. 2nd Edition. Baltimore: Williams and Wilkins; 1987. Page 300

30. Bleiman BS and Schwartz AL. Paradoxical, intraocular pressure response to pilocarpine: A proposed mechanism and treatment. Arch Ophthalmol 1979; 97: 1305-07

31. Richardson K. Acute glaucoma after trauma. In freeman HM, Editor: Ocular trauma, New York, 1979, Appleton – Century – Crofts

32. Howard GM Hutchinson BT, and Frederick AR Jr. Hyphaema resulting from

blunt trauma: gonioscopic, tomographic, and ophthalmoscopic observations following resolution of the hemorrhage. Trans Am Acad Ophthalmol. 1979; 12: 345-348

33. Tonjum AM. Gonioscopy in traumatic hyphema. Acta Ophtlamol. 1966; 44: 650-664.

34. Blanton FM. Anterior chamber angle recession and secondary glaucoma: A study of the after effects of traumatic hyphaema. Arch ophthalmol 1964; 72: 39-43.

35. Kaufman JH and Tolpin DW. Glaucoma after traumatic angle recession: a ten-years prospective study. Am J Ophthalmol 1974; 78: 648-654.

36. Their H J, Aden G, and Pulhorn G. Changes in the chamber angle following ocular contusions. Klin Monatsble Angenheilkd 1988; 177: 165-67.

37. Tonjum AM. Intraocular pressure and facility of outflow late after ocular contusion. Acta Ophthlamol. 1968; 46: 886-908.

38. D' Ombrain A. Traumatic monocular chronic glaucoma. Trans Ophthalmol Soc Aust 1945; 5: 116-121.

39. Wolter JR. Coup - contrecoup mechanism of ocular injuries. Am J Ophthalmol 1963; 56: 785-796.

40. Weidenthal DT and Schepens CL. Peripheral fundus changes associated with ocular contusions. Am J Ophthalmol 1966; 62: 465-477.

41. Gregor Z and Ryan SJ. Blood – retinal barrier after blunt trauma to the eye. Graefes Arch Clin Exp ophthalmol 1982; 219: 205-208.

42. Bunt – Milam AH, Black RA and Bensinger RE Breakdown of the outer blood – retinal barrier in Experimental commotio retinae. Exp Eye Res 1986; 43: 397-412.

43. Mansour AM, Green WR, Hogge C. Histopathology of commotio retinae. Retina 1992; 12: 24-28.

44. Sipperley JO, Quigley HA, Gass JDM. Traumatic retinopathy in primates : the explanation of commotio retinae. Arch ophthalmol 1978; 96: 2267-2273.

45. Von Graefe, A.Zwei. Falle Von ruptur der choroidea. Graefes arch ophthalmol 1854; 1: 402-403.

46. Aguilar IP, Green WR. Choroidal rupture: a histopathologic study of 47 cases. Retina 1984; 4: 269-275.

47. Siejrest NIH. Traumatiscen Ruptur von ciliaraterien Klin U Med Inst Schweie 1895; 3: 554-571.

48. Fuller B, Gitter KA. Traumatic choroidal rupture with late serous detachment of macula: report of successful argon laser treatment. Arch ophthalmol 1973; 89: 354-355.

49. Gass JDM. Stereoscopic atlas of macular disease: diagnosis and treatment. 4th edition. St. Louis: Mosby: 1997. Page 1243-49

50. Smith RE, Kelley JS, Harbin TS. Late Macular complications of choroidal rupture. Am J Ophthalmol. 1974; 77: 650-658.

51. Cox Mx, Schepens CL, Freeman HM. Retinal detachment due to ocular contusion. Arch Ophthalmol. 1966; 76; 678-685.

52. Wedenthal DT, Schepens CL. Peripheral fundus changes associated with ocular contusion. Am J Ophthalmol. 1966; 62: 465-477.

53. Giovinazzo VJ, Yannuzzi LA, Sorenson JA et al. The ocular complication of boxing. Ophthalmology. 1987; 94: 587-596.

54. Goffstein R, Burton TC. Differentiating traumatic from non-traumatic retinal detachment. Ophthalmology. 1982; 89: 361-368.

55. Malbran E, Dodds R, Hubsbus R. Traumatic retinal detachment. Mod Probl Ophthalmo 1972; 10: 479-489.

56. Tulloh CG, Trauma in retinal detachment. Br J Ophthalmology. 1968; 52: 317-321.

57. Tasman W. Peripheral retinal changes following blunt trauma. Trans Am Ophthalmol Soc. 1972; 70: 190-198.

58. Cooling RJ. Traumatic retinal detachment mechanisms and management. Trans Ophthalmol Soc VK. 1986; 105: 575-579.

59. Magheria RR, Schepens CL. Macular breaks: diagnosis, etiology, and observation. American Journal of ophthalmology. 1972; 74: 219.

60. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. *Surv Ophthalmol*. 1994 May-Jun. 38(6):487-518.

61. Foster Bs, March GA, Lucareri MJ et al. optic nerve avulsion Arch Ophthalmol 1997; 115: 623-630.

62. Parul Desai, Caroline J Mac Ewen, Paul Baines, Darwin C Minassian. Incidence of cases of ocular trauma admitted to hospital and incidence of blinding outcome. Br J Ophthal .1996; 80:592-596.

63. Gothwal V, Adolph S, Jalali S, Naduvilath T. Demography and prognostic factors of ocular injuries in South India. Australian and New Zealand journal of ophthalmology. 1999;27(5):318--325.

64. Vats S et al. Epidemiological study of ocular trauma in an urban slum population in Delhi, India. Indian J Ophthalmol 2008; 56; 313-16

65. McCarty C A, H L Cara and Taylor H R. Epidemiology of Ocular Trauma in Australia. Ophthalmology. 1999; 106: 1847-1852.

ANNEXURE-I

CONSENT FOR PARTICIPATION IN RESEARCH STUDY

ID NO. ☐☐☐

Mr/Mrs/Ms____ you are invited to participate in our research study titled **"Prevalence of Ocular Trauma in KLES Hospital – A 1 year cross sectional study"**

Respected Sir/Ma'am we request you to enroll yourself in our study as you are eligible for participation. Your participation in this research is voluntary. If you decide to participate you are free to withdraw at any time later on.

Purpose of the Study: The purpose of research is to find the prevalence, causes, demographic and clinical profile of ocular trauma in patients attending our hospital.

Need for the Study: This study will help to get a clear picture of the various causes of ocular trauma and measures can be taken for their prevention.

Procedure Involved: If you agree to enroll yourself in this study, you will be asked your present and past history. You will be clinically examined and if at all required, relevant investigations will be done according to the treatment protocol. Pre treatment and post treatment photographs will be taken.

Risks and Benefits: There are no major risks. However you can have some discomfort while examination. Your participation may benefit you and others by establishing certain facts about the study.

Alternatives: If you are not willing to participate you will be treated according to the existing protocol & it will not affect your relationship with this hospital.

Costs for participating in this research: There will not be any extra cost incurred by you. You will however, have to pay for the investigations which are part of the existing management protocol for the condition. There is no commitment for any reimbursement or any other compensation.

Privacy and Confidentiality: Your privacy is guaranteed. However, your medical records can be directly accessed and reviewed by authorized individuals or by the ethics committee. Records which could reveal your identity, will be kept confidential. Personal data will remain anonymous if data is being published or written as a dissertation.

Authorization to Publish Results: When the results of the research are published or discussed in a conference, no information will be displayed that would disclose your identity.

Compensation: In the event of injury related to the study, treatment will be made available through KLES Dr. Prabhakar Kore Hospital & MRC, Belagavi. There is no compensation or payment for such medical treatment by law. The doctors and the staff will provide facilities and medical attention to you.

Questions

If you have any questions about the research you may please contact:

1. Dr. (Mrs.) Ganga S. Pilli, Chairperson, Institutional Ethics Committee.

   Contact No.(0831) 2471350

Consent for participation in research trial

I, Mr./Ms./Mrs ________________________________ voluntarily agree for the participation as a subject of this study. By signing this consent form, I am not giving up any of my legal rights. I may withdraw from the study at anytime. I am signing the consent form after having read or been read for me in my own vernacular language, including the risks and the benefits and having all my questions answered.

Subject Name : ____

Signature or the Left Thumb Print of Subject : ______________________________

Witness Name : ____

Signature of Witness: ____

Investigator's Name: _______________________

Signature of Investigator: ____

Name of the Guide: _____________________

Signature of the guide: ____

Date:

Place:

Assent for participation in research trial

I, Mr./Mrs ________________________________ parent/guardian of_________________________________ voluntarily agree for the participation of my child as a subject of this study. By signing this consent form, I am not giving up any of my legal rights. I may withdraw my child from the study at anytime. I am signing the consent form after having read or been read for me in my own vernacular language, including the risks and the benefits and having all my questions answered.

Child's Name : ____

Signature or Left Thumb Print of the Child : ______________________________

Parent's/Guardian's Name : _______________________________________

Signature of Parent/Guardian: ____

Investigator's Name: _______________

Signature of Investigator: ____

Name of the Guide: _________________

Signature of the Guide: ____

Date:

Place:

ANNEXURE-II

PROFORMA

NAME		AGE/SEX	
ADDRESS		OCCUPATION	
DATE OF INJURY		ECONOMIC STATUS	
MECHANISM OF INJURY		TIME OF INJURY	
OTHER INJURIES			

HISTORY	
• EYE INVOLVED • WHETHER WORK RELATED • OTHER THAN WORK RELATED • UNINTENTIONAL/ASSAULT/UNKNOWN • UNDER THE INFLUENCE OF ALCOHOL • WEARING SEATBELT/HELMET • DIMINUTION OF VISION • FLASHES OF LIGHT • FLOATERS • REDNESS • WOUND IN THE LIDS • PAIN IN THE EYES	

• DIPLOPIA • PRIOR OCULAR HISTORY (Operation, Prev. trauma, Ocular medications) • TETANUS IMMUNISATION • COMPOSITION OF INTRAOCULAR FOREIGN BODY • IDENTITY OF CHEMICAL AGENT	
SYSTEMIC EXAMINATION	

EXAMINATION FINDINGS	
VISUAL ACUITY • **Unaided** • **Pin hole**	

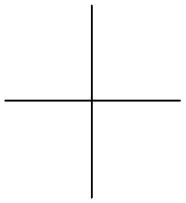
TISSUES INVOLVED

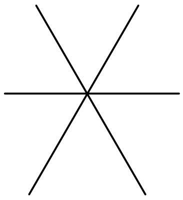
1. **LIDS** OD OS

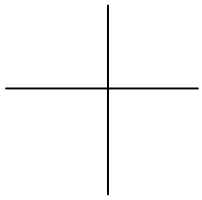
2. **GLOBE** OD OS

3. **MOTILITY**

OD BINOCULAR OS







4. CONJUNCTIVA OD OS

5. CORNEA OD OS

6. SCLERA OD OS

7. ANTERIOR CHAMBER OD OS

8. IRIS	OD	OS
9. PUPIL	OD	OS
10. CRYSTALLINE LENS	OD	OS
11. INTRAOCULAR PRESSURE	OD	OS
12. OPHTHALMOSCOPY	OD	OS
Glow		
Media		
Disc		
C: D ratio		
Blood vessels		
Background		
Macula		

13. **INVESTIGATIONS**

B Scan

X-ray

CT Scan

SYSTEMIC INVESTIGATIONS

DIAGNOSIS

TREATMENT

ANNEXURE III – PHOTOGRAPHS

Photo 1 – Left eye Traumatic extrusion of the eyeball

Photo 2 – Post enucleation photo of left eye traumatic extrusion of the eyeball

Photo 3 – Right eye Partial thickness lid laceration

Photo 4 – Right eye sutured partial thickness lid laceration

Photo 5 – Left eye full thickness corneal laceration with iris prolapsed

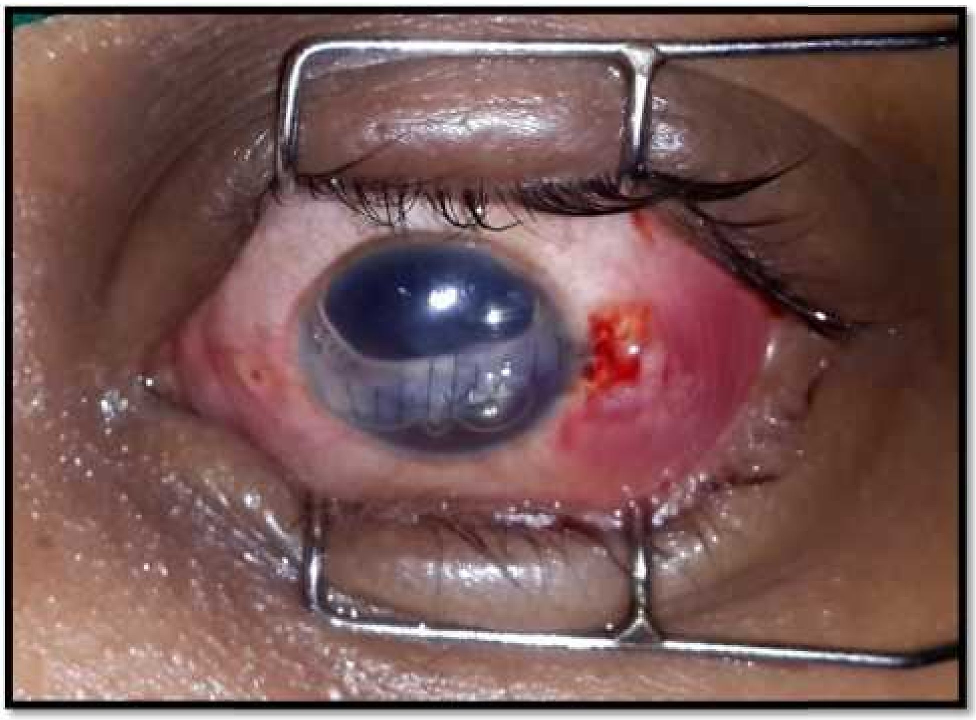


Photo 6 – Left eye primary globe repair

Photo 7 – Left eye Ecchymosis

Photo 8 – Right eye Subconjunctival haemorrhage

Photo 9 – Left eye Chemosis

Photo 10 – Left eye Corneal Foreign Body

Traumatic Cataracts

Photo 11 – Right eye Traumatic cataract

Photo 12 – Left eye Traumatic cataract

Photo 13 - Left eye Traumatic cataract

Photo 14 - Right eye Traumatic cataract

Photo 15 - Left eye Traumatic cataract

Photo 16 - Right eye Traumatic cataract

Photo 17 – Right eye Iridodialysis with Traumatic cataract

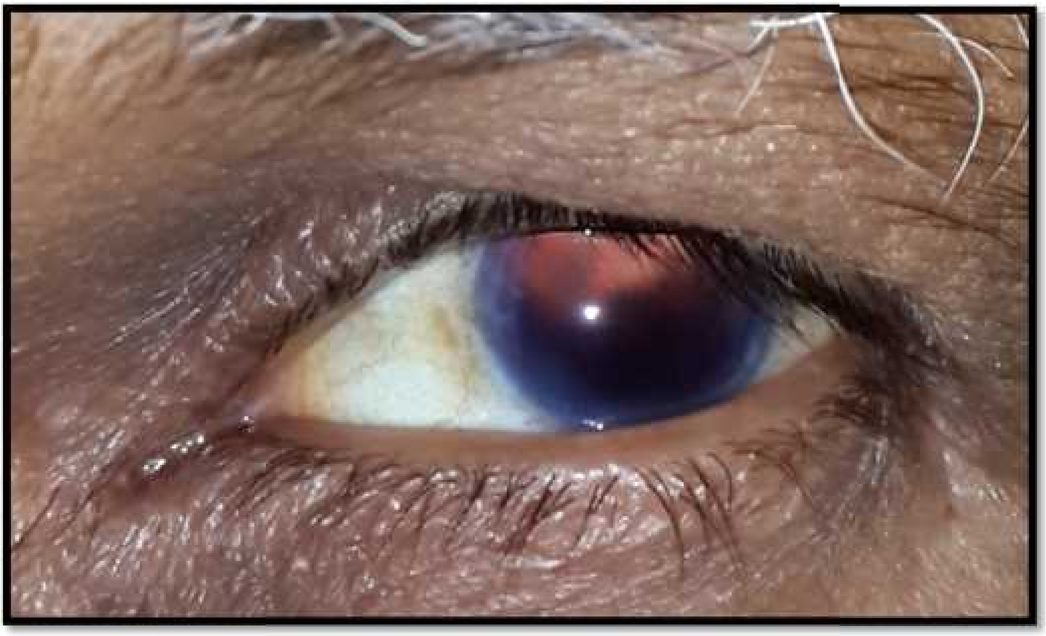


Photo 18 – Left eye Traumatic Aphakia

Subluxated Lenses

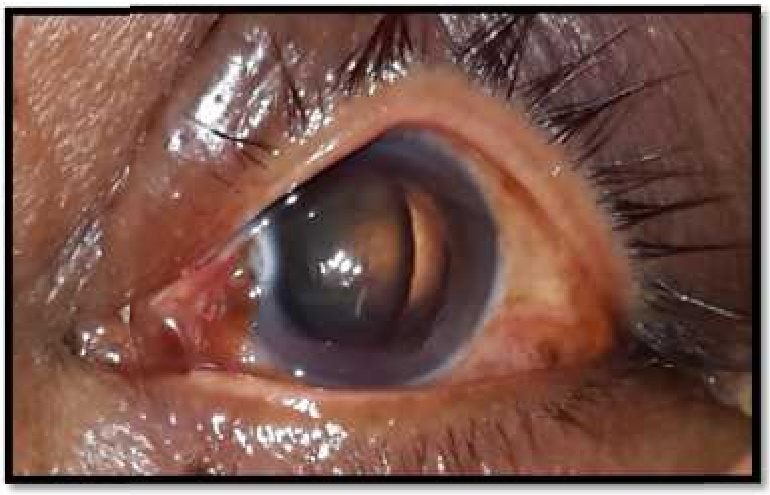


Photo 19 – Left eye Nasal Subluxation

Photo 20 – Left eye Inferior Subluxation

Photo 21 – Right eye Nasal Subluxation

B Scans

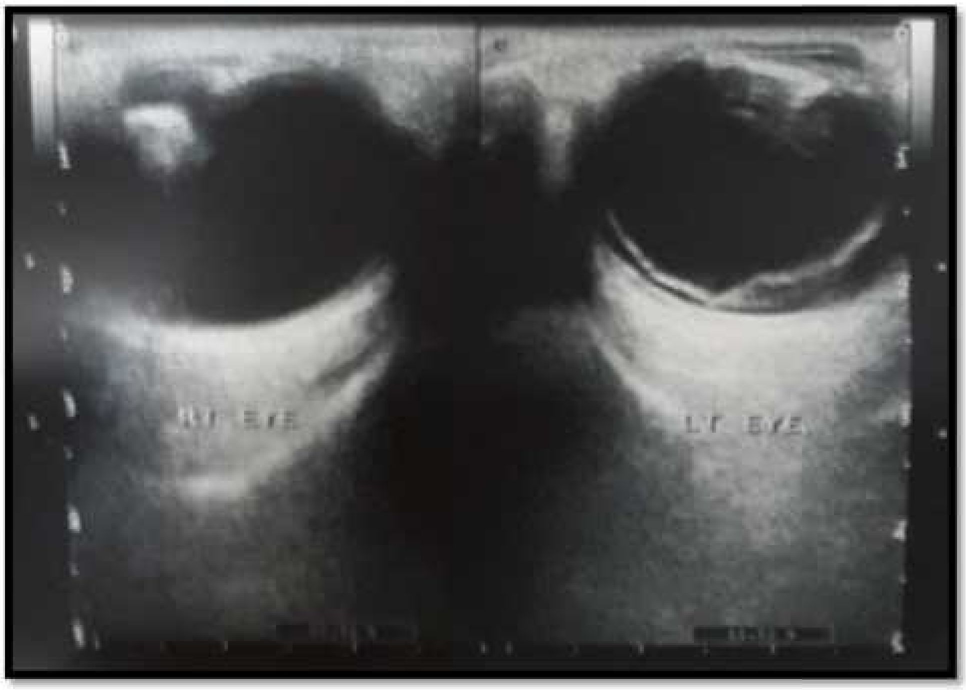


Photo 22 – Left eye Retinal Detachment

Photo 23 – Right eye Traumatic cataract

X rays

Photo 24 – A P view of Orbit showing IOFB

Photo 25 – Left Lateral view of Orbit showing IOFB

KEY TO MASTER CHART

#	-	Fracture
AC	-	Anterior chamber
Acc	-	Accurate
BE	-	Both eyes
BL	-	Bilateral
BS	-	B Scan
CF	-	Counting fingers
CFCF	-	Counting fingers close to face
Conj Cong	-	Conjunctival Congestion
CTS	-	CT Scan
DNMO	-	Details not made out
Epi def	-	Epithelial defect
F	-	Female
FB	-	Foreign body
FT	-	Full thickness
HMCF	-	Hand movements close to face
Inacc	-	Inaccurate
Inv	-	Investigation
LE	-	Left eye
LR	-	Lateral Rectus
M	-	Male
m	-	Meters
MED	-	Medical management
NVI	-	Neovascularisation of iris
PL	-	Perception of light
PR	-	Projection of rays

PT	-	Partial thickness
RAPD	-	Relative Afferent Pupillary Defect
RE	-	Right eye
RRR	-	Round, regular, reactive
RTA	-	Road traffic accident
SUR	-	Surgical management
Tr myd	-	Traumatic mydriasis
Tr cat	-	Traumatic cataract
TON	-	Traumatic Optic Neuropathy
WNL	-	Within Normal Limits
XRO	-	X ray Orbit