A CLINICAL STUDY OF OCULAR MANIFESTATIONS

IN HEAD INJURY

DISSERTATION

SUBMITTED FOR M.S. [OPHTHALMOLOGY]

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DEPARTMENT OF OPHTHALMOLOGY

THANJAVUR MEDICAL COLLEGE

THANJAVUR

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI - TAMILNADU

CERTIFICATE

This is to certify that this dissertation entitled "**A CLINICAL STUDY OF OCULAR MANIFESTATIONS IN HEAD INJURY"** is a bonafide record of work done by **Dr. ARUNA.G**, under my guidance and supervision in the Department of Ophthalmology, Thanjavur Medical College, Thanjavur during her Post Graduate study for the degree of M.S. OPHTHALMOLOGY from May 2017- May 2020.

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DECLARATION

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"A CLINICAL STUDY OF OCULAR MANIFESTATIONS IN HEAD INJURY" is a bonafide record of work done by me in the Department of Ophthalmology, Thanjavur Medical College, Thanjavur under the guidance and Supervision of my Professor **Dr.J.Gnanaselvan M.S.,D.O.,** the Head of the Department, Department of Ophthalmology, Thanjavur Medical college, Thanjavur between May 2017 – May 2020.

This dissertation is submitted to The Tamilnadu Dr.M.G.R Medical University, Chennai in partial fulfillment of University regulations for the award of M.S Degree (Branch III) in Ophthalmology to be held in May 2020.

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CERTIFICATE FOR PLAGIARISM

This is to certify that this dissertation work entitled **"A CLINICAL STUDY OF OCULAR MANIFESTATIONS IN HEAD INJURY"** is a bonafide record of work done by **Dr.ARUNA.G** with Registration number 221713151 for the award of **MASTERS DEGREE** in the branch of **OPHTHALMOLOGY**.I personally verified the urkund.com website for plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 5 percentage of plagiarism in the dissertation.

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CONTENTS

PART - I

INTRODUCTION

Head injury is the common cause of death and major disabilities in trauma patients. The visual system is one of the system involved in Traumatic brain injury. The eye is frequently involved due to proximity of the eye to the head as well as due to neural connections between the eye and the brain. Ocular trauma is the important preventable cause of blindness and visual impairment in the world.

Eyeball is anatomically well protected inside bony orbit socket and orbital margin, covered anteriorly by eye lids with lashes, embedded in cushion of retrobulbar pad of fat behind. Inspite of all these protections eye is vulnerable to injuries.

Many theories have been proposed of how the eye is injured in traumatic brain injury. In penetrating brain injury, there may be physical damage to the visual pathway, visual cortex and other vision related structures of the brain. In non-penetrating or closed head injury displacement, stretching and shearing forces may damage areas of brain, including those associated with vision. Visual dysfunction in patients with head injury is also due to direct ocular trauma.

RTA forms a major part of accident and emergency care not only in cities but also in rural areas. Improved affordability, change in socio economic conditions, mind set and personality of the younger generation etc., have influenced the behavioural pattern directly and indirectly and these have resulted in increased head injuries, which may often have associated eye injuries.

In head injury many of the ocular manifestations are often missed and present much later to ophthalmology department. Hence clinical correlation of the ophthalmic findings is important in early localization of the site of injury, ongoing assessment, management and prognosis with head injury.

Early diagnosis of visual problems following traumatic brain injury is essential to maximize overall rehabilitation potential. Strengthening of preventive measures in terms of need for medical care, loss of income, cost of rehabilitation services on ocular trauma victims are needed.

REVIEW OF LITERATURE

Although many ophthalmologists have contributed enough literature regarding ocular manifestations in head injury, few were given below.

Rudolf Berlin (1873) was the first person to interpret the importance of clinical observation through crucial test of experiments. His name is immortalised in “Berlins Oedema”, the dramatic changes that appear in the retina following injury.

Ogilivie (1900) has put forward a detailed theory to explain the mechanism of contusion injury of the globe, which is circumferential lateral distention to compensate for the sudden antero-posterior compression of the globe.

Rowbotham et al reported that the proportion of head injuries resulting from RTA is higher than 44% , while in other studies it can be as high as 84.2%.(1)

Masila et al., in his study reported a positive correlation between severe head injury (GCS<8) and occurrence of ocular signs.(2)

Moster et al reported that oculomotor nerve palsy is seen in 30% of head injury patients, trochlear nerve palsy in 26% of cases and abducent nerve palsy in 22% of cases.(3)

Mariak, after brain autopsy in 12 patients found serious cranial nerve involvement in 75% of the fatal closed head injury cases.(4)

Brahm et al (2009) found that of the inpatients (with moderate to severe head injury), vision of 20/60 or better was present in 77.8% patients ,vision of 20/70-20/100 was seen in 6.3% patients, 12.7% cases had a visual acuity of worse than 20/100 and 3.2% cases had no perception of light in both eyes. Of the outpatients (with mild head injury), 98.4% had a visual acuity of 20/60 or better, none of the patients had a visual acuity between 20/70-20/100, 1.6% had a visual acuity worse than 20/100 and no patient had no perception of light. (5)

Kulkarni and Aggarwal (2005) found a higher prevalence of ocular involvement of 83.5% in closed head injury patients. (6)

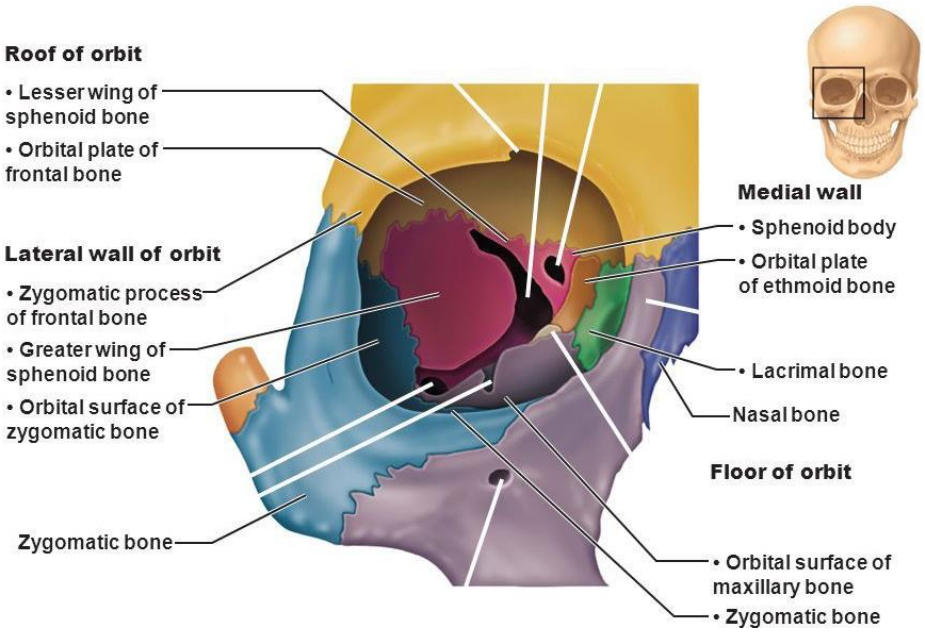
Odebode et al (2005) reported globe rupture in 2 patients with concomitant orbital wall fracture. On admission patients with GCS $\leq$ 8 (severe head injury) were presented with severe ocular manifestations. In mild head injury (GCS – 13-15) nearly half of the patients were presented with visual and ocular injuries. (7)

ANATOMY

ANATOMY RELATED TO OCULAR TRAUMA

There is an eyeball, some fat, muscles, nerves, vessels, fasciae and lacrimal glands in the orbit. Frontal sinus, ethmoid sinus, maxillary sinus and sphenoid sinus are located adjacent to orbit. Orbital walls are derived from cranial neural crest cells and they are quadrangular truncated pyramidal in shape. Each orbit is made of 7 bones.

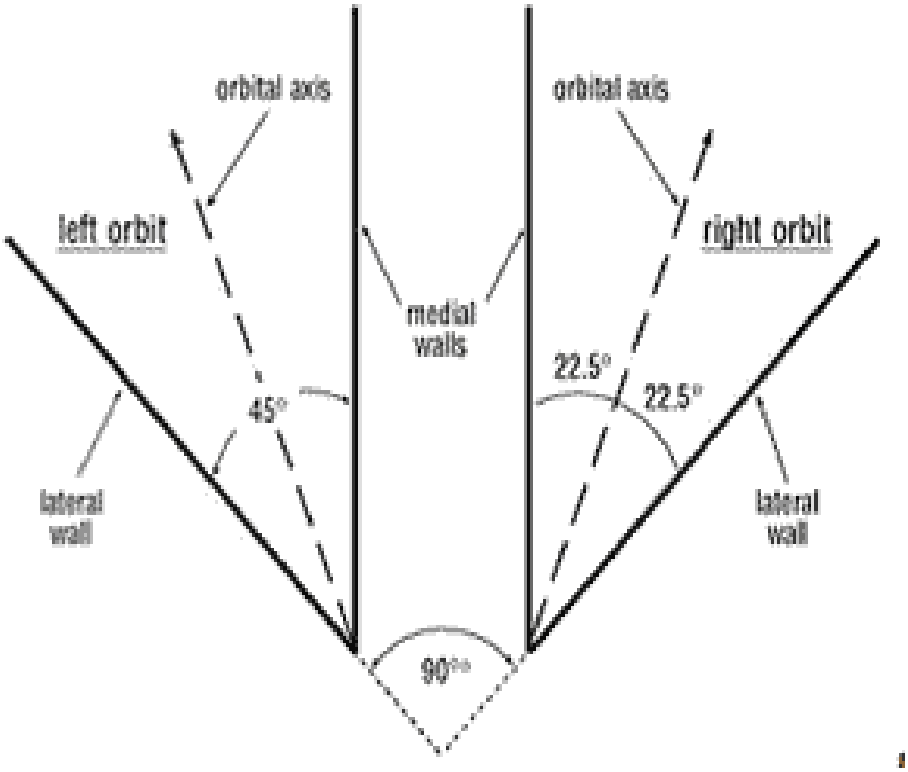
- Maxillary bone
- Ethmoidal bone
- Lacrimal bone
- Palatine bone
- Zygomatic bone
- Frontal bone
- Sphenoid bone



The walls of the orbit are

- LATERAL WALL: Zygomatic bone, Greater wing of sphenoid.
- FLOOR: Maxillay bone, Zygomatic bone and Palatine bone..
- ROOF: Lesser wing of sphenoid, Orbital plate of frontal bone
- MEDIAL WALL: Lacrimal bone, Sphenoid bone, Ethmoid bone, Maxillary bone.

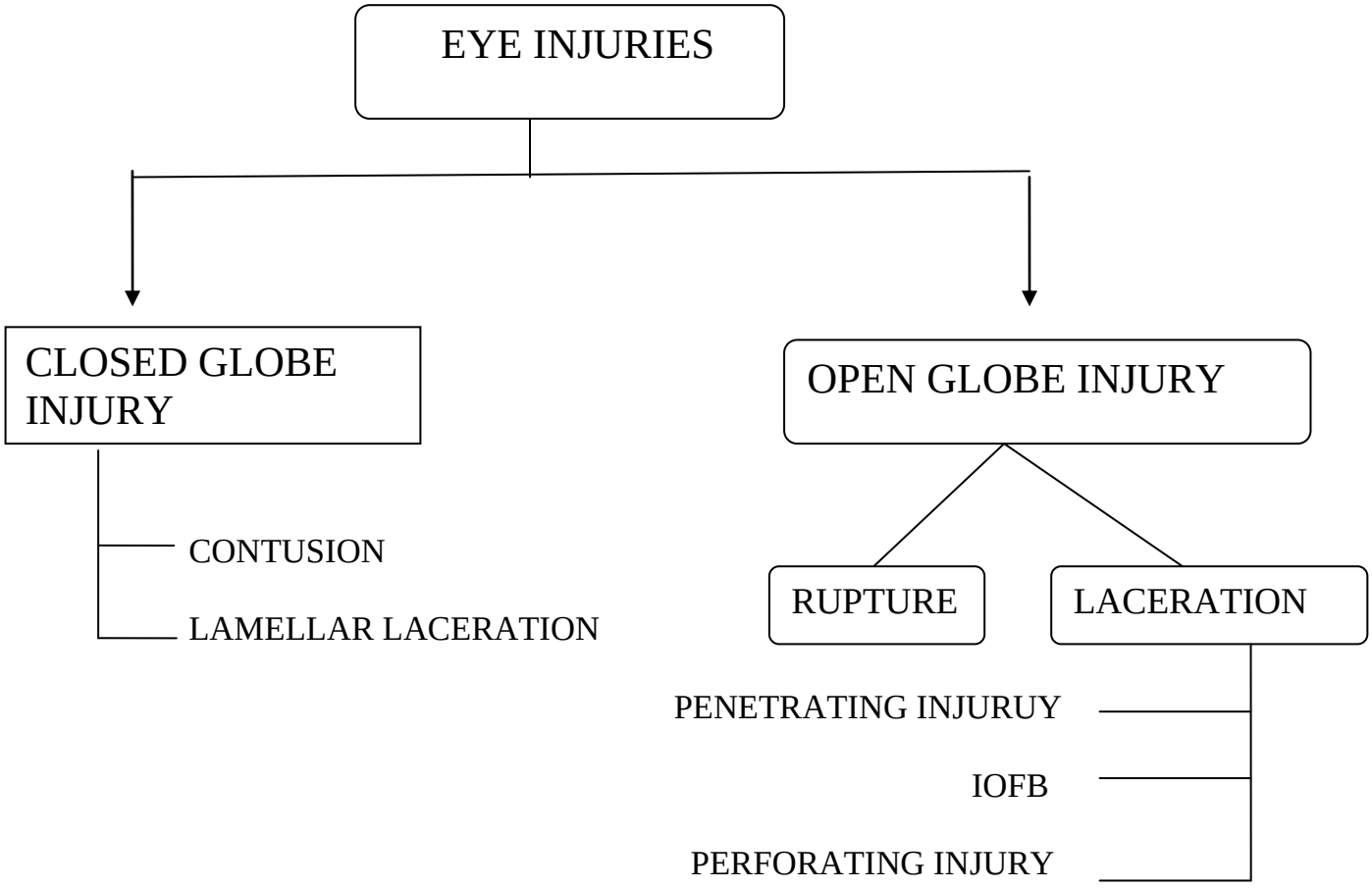
The volume of the adult orbit is 30 ml. The rim of the orbit measures 40 mm horizontally and 35 mm vertically. The orbit depth is 42 mm medially and 50 mm laterally. Lateral walls of the orbit are 90° to each other and medial walls are almost parallel to each other.



CLASSIFICATION OF OCULAR TRAUMA

1) THE BIRMINGHAM EYE TRAUMA TERMINOLOGY SYSTEM (BETTS)

CLASSIFICATION:



TERMS AND DEFINITIONS IN BETTS:

TERM	DEFINITION AND COMMENT
Eyeball	Sclera and cornea. Although technically the eyeball has three coats posterior to limbus for clinical and practical purposes, violation of only the most external structure is taken into consideration.
Closed globe injury	No full thickness wound of the eyeball.
Open globe injury	Full thickness wound of the eyeball.
Contusion	There is no (full thickness) wound. The injury is due to either direct energy delivery by the object (e.g., choroidal rupture) or the changes in the shape of the globe (e.g., angle recession).
Lamellar laceration	Partial thickness wound of the eyeball
Rupture	Full thickness wound of the object, caused by a blunt object. Because the eye is filled with incompressible liquid, the impact results in momentary increase in IOP. The eyeball yields at its weakest point (at the impact site or elsewhere: e.g., an old cataract wound dehisces even though the impact occurred elsewhere); actual wound is produced by an inside-out mechanism.
Laceration	Full thickness wound of the eyeball, caused by a sharp object.

	The wound occurs at the impact site by an outside – in mechanism.
Penetrating injury	Entrance wound. If more than one wound is present, each must have been caused by different agent Retained foreign object(s) Technically a penetrating injury but grouped separately because of different clinical implications
Perforating injury	Entrance and exit wounds. Both wounds caused by the same agent.

2) OCULAR TRAUMA CLASSIFICATION SYSTEM (OTCS):

I.TYPE

	OPEN GLOBE INJURY	CLOSED GLOBE INJURY
A	Rupture	Contusion
B	Penetrating	Lamellar laceration
C	Intraocular foreign body	Superficial foreign body
D	Perforating	Mixed
E	Mixed	

II. GRADE (visual acuity):

	OPEN GLOBE INJURY	CLOSED GLOBE INJURY
A	≥20/40	≥20/40
B	20/50-20/100	20/50-20/100
C	19/100-5/200	19/100-5/200
D	4/200 to light perception	4/200 to light perception
E	No light perception	No light perception

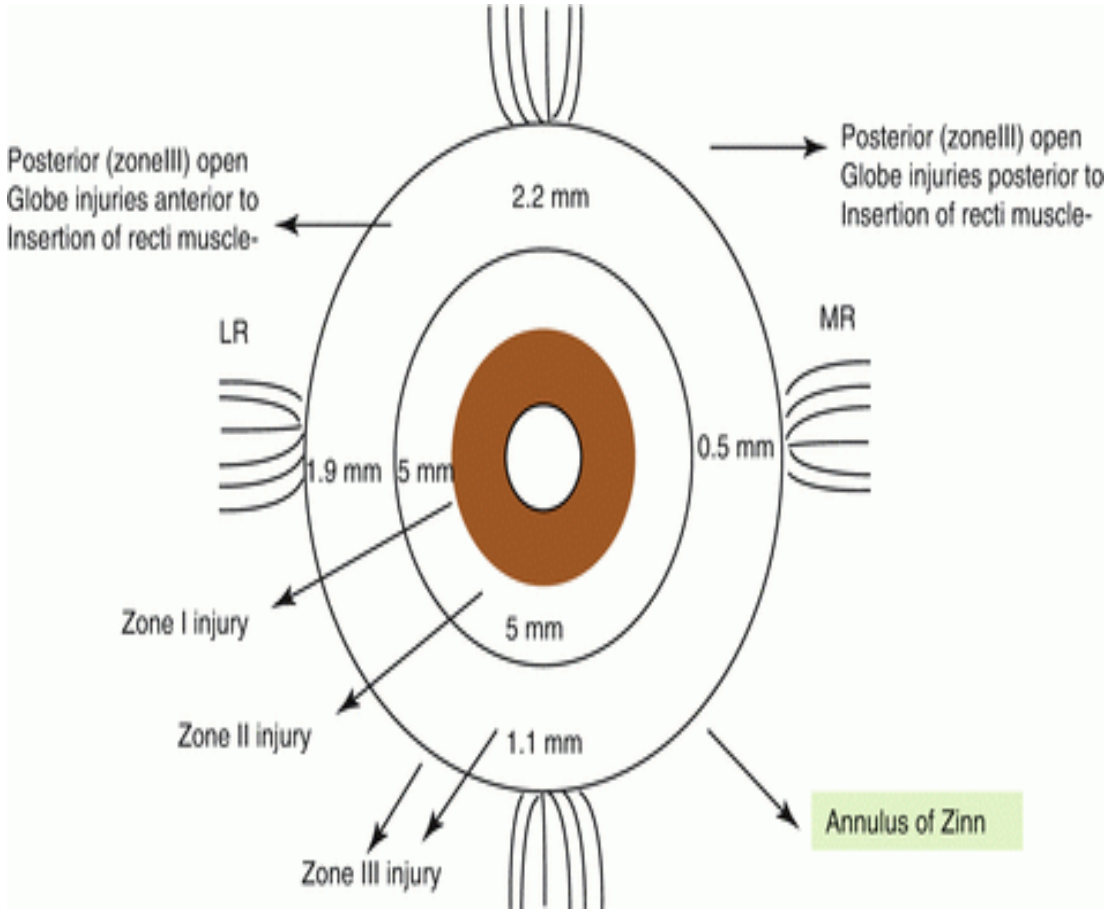
III.PUPIL:

	OPEN GLOBE INJURY	CLOSED GLOBE INJURY
A	RAPD(+)	RAPD(+)
B	RAPD(-)	RAPD(-)

IV.ZONE:

	OPEN GLOBE INJURY	CLOSED GLOBE INJURY
I	Isolated to cornea (including corneoscleral limbus)	External (limited to bulbar conjunctiva, sclera, cornea)
II	Corneoscleral limbus to a point 5	Anterior segment (involving structures in

	mm posterior into sclera	anterior segment internal to the cornea and including posterior lens capsule; also includes pars plicata but not pars plana)
III	Posterior to anterior 5 mm of the sclera	Posterior segment (all internal structures posterior to the posterior lens capsule)



OCULAR TRAUMA SCORE:

The chief concern of all patients with ocular trauma is visual prognosis. This score is used to calculate prognosis assuming that the trauma has been managed optimally. It is based on the Brimingham Eye Trauma Technology System. The OTS ranges from 1 (most severe injury and worst prognosis at 6 months follow up) to 5 (least severe injury and least poor prognosis at 6 months). The predictive accuracy of ocular trauma score is around 80 %.

CALCULATION OF OCULAR TRAUMA SCORE:

TABLE 1:

INITIAL VISUAL FACTOR	**RAW POINTS**
A. Initial Visual acuity	NPL = 60 PL or HM = 70 1/200 to 19/200 = 80 20/200 to 20/50 =90 ≥20/40= 100
B. Globe rupture	-23
C. Endophthalmitis	-17
D. Perforating injury	-14
E. Retinal detachment	-11
F. Relative afferent pupillary defect	-10

SUM OF ALL RAW POINTS = RAW SUM SCORE

TABLE 2: Conversion of raw score points into ocular trauma score category and calculating the likelihood of final visual acuity in five categories

Raw Score Sum	OTS Score	NLP	PL/HM	1/200-19/200	20/200 to 20/50	≥ 20/40
0-44	1	74%	15%	7%	3%	1%
45-65	2	27%	26%	18%	15%	15%
66-80	3	2%	11%	15%	31%	41%
81-91	4	1%	2%	3%	22%	73%
92-100	5	0%	1%	1%	5%	94%

PROCEDURE TO CALCULATE OTS:

1. Assign an initial raw score based on the initial visual acuity

2. From this initial raw score subtract points for each factor as mentioned in table 1

3. Once the raw score sum has been calculated find the relevant category in table 2 and mention the corresponding OTS score.

The OTS score enables us to efficiently plan, manage and monitor the range of ocular injuries due to mechanical trauma.

PATHOPHYSIOLOGY OF HEAD TRAUMA

Ophthalmic complications in head injury depends on

1. Brain trauma itself (concussion, contusion, laceration) with visual complications.

2. Skull fracture in the occiput or base with damage to the visual cortex or the nerves subserving the eyes.

3. Cerebral compression due to meningeal hemorrhage (acute or chronic, extradural, subdural, subarachnoid or intracerebral)

PROGRESSIVE CEREBRAL COMPRESSION DUE TO INTRACRANIAL BLEED GOES THROUGH FOUR STAGES:

Harvey cushing proposed the following stages:

1. Stage of physical compensation: CSF displaced, BP rises and circulation maintained – symptomless

2. Stage of venous compression – irritative signs appear

3. Stage of capillary anemia –paralytic signs appear

4. Irreversible sign of perivascular hemorrhages and progressive brain edema

CONSEQUENCES OF A SPACE OCCUPYING LESIONS (SOL)

1. Herniation of the cingulated gyrus under the falx

2. Tentorial herniation of the para hippocampal gyrus

3. Herniation of cerebellar tonsil through the foramen magnum

4. Impingement of the crus against the tentorium

5. Hemorrhage into the midbrain

BIOMECHANICS OF BRAIN INJURY

By definition head trauma is an anatomical damage or physiological disturbance caused by application of mechanical force to the head.

Therefore the nature, site and magnitude of the mechanical force are the main determinants of head injury. The modification or elimination of this force is the major preventive strategy.

PRIMARY BRAIN INJURY

1. Contact injuries
2. Inertial injuries

1. CONTACT INJURIES

A direct impact causes complex mechanical events both near and distant to the point of contact. They typically cause focal injuries and do not cause diffuse brain injury. Contact forces produce injury in the immediate vicinity of skull injury. Since most impacts set the skull in motion, there are associated acceleration injuries also.

Local contact effects:

1. Depressed fractures
2. Linear fractures
3. Basilar skull fractures
4. Extradural hematoma
5. Coup contusions

Remote contact effects:

Remote contact effects are caused by deformation of skull and also by stress waves transmitted at high velocity

1. Remote linear fracture (base or vault)

2. Remote brain contusions and hematoma

3. Brain herniation caused by transient distortion, particularly in infant skulls

Small high velocity objects may shutter the skull and drive bone fragments deeper. When the contact surface is larger or the skull hits a fixed surface the result depends on the nature of mechanical loading.

MECHANICAL LOADING -TYPES

i. Static loading:

Static loading is less common type. Input force applied is more than 200ms duration. e.g. slow moving vehicles

ii. Dynamic loading:

Dynamic loading is the most common type. Input force applied is less than 50ms. e.g. most RTAs, fall from height. In dynamic loading there can be contact or inertial force. There are two subtypes

1. Impulsive loading

2. Impact loading

Impulsive loading:

Only inertial forces

Head is accelerated or decelerated without being struck directly

e.g. blow to thorax or face,

Fall from height

Impact loading:

Both inertial and contact forces

Accelerates or decelerates when there is short impact to head

In common, it is local skull injury

2. INERTIAL INJURIES:

Head acceleration causes functional or structural damage to neural and vascular structure in relation to the dura and skull by the relative movement of the brain.

Acceleration types are

1. Translational

2. Rotational

3. Angular i.e. rotational and translational

Most commonly encountered is angular acceleration. Angulation center is usually mid cervical spine. It is most injurious as it causes shear strain deformation because of differential motion of one portion of the brain with respect to another.

SECONDARY CEREBRAL INJURY

Secondary cerebral injury is the result of intracranial space occupying lesion, either by edema or hematoma, in contrast to primary brain injury which occurs at the movement of the impact. Effects of secondary damage are usually preventable by active management of head injuries in the early stages and by close observation of clinical signs

TYPES OF PRIMARY LESION

1. Diffuse axonal injury and contusion
2. Subdural hematoma
3. Extradural hematoma
4. Intracerebral hematoma

Important note:

Increased shear deformation and subdural hemorrhage is seen in senile cerebral atrophy

BRAIN LESIONS AND ITS MECHANISM OF INJURY:

1. Skull fractures, Epidural hematoma, coup contusion – Contact forces
2. Contrecoup contusions - inertial effects (impact or impulsive)
3. Intra cerebral hematoma - acceleration induced strains and stress waves
4. Subdural hematoma - disruption of bridging vein
5. Cerebral concussion – angular accelerations with electro physiologic dysfunction
6. Diffuse axonal injury-angular acceleration of long duration especially in coronal plane.

GLASGOW COMA SCALE (GCS):

GCS is a neurological scale, aims to give reliable and objective way of recording the state of person’s consciousness for initial and subsequent assessment.

FEATURE	RESPONSE	SCORE
Eye response	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No eye opening	1
Verbal response	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No verbal response	1
Motor response	Obeys commands	6
	Localising pain	5
	Withdrawal from pain	4
	Flexion to pain	3
	Extension to pain	2
	No motor response	1

Brain injury is classified as:

Severe, GCS<8-9

Moderate, GCS 8 or 9-12

Minor, GCS≥13

THE REVISED TRAUMA SCORE (RTS):

RTS is a physiologic scoring system, based on the initial vital signs of the patient. Lower score indicates higher severity of injury.

GLASGOW COMA SCALE (GCS)	SYSTOLIC BLOOD PRESSURE (SBP)	RESPIRATORY RATE (RR)	RTS VALUE
13-15	>89	10-29	4
9-12	76-89	>29	3
6-8	50-75	6-9	2
4-5	1-49	1-5	1
3	0	0	0

CONTUSION AND CONCUSSION (BLUNT INJURY)

Concussion indicates that there is no disorganization and all changes are reversible. The term contusion is used when tissues are bruised and disorganized by perivascular hemorrhages with intact surface. If they are torn or disrupted it is laceration. The effects on the eye are due to

Direct impact: where maximum effect occurs at the point where the blow is received.

Transmitted force: In this wave of pressure due to sudden compression of the globe travels throughout its fluid contents in all directions and maximum effect is seen point distal to force of impact (countercoup).

Indirect force: due to sudden hurling of globe against the elastic content of the orbit and its resistant bony walls.

MECHANISM:

Damage to eye is due to impinging force that acts in antero posterior axis resulting in sudden anterior –posterior compression of the globe, relief being sought in lateral distension . If the force impinges on cornea, it is thrust inwards, anterior chamber is compressed and aqueous humor moves towards periphery. Pupil also contracts spasmodically. Pressure wave of aqueous pushes iris and lens backwards, forces the vitreous against the posterior pole, thereby pushing choroid and retina against sclera.

OCULAR MANIFESTATIONS OF HEAD INJURY

1. EYELIDS:

a) Periorbital edema and ecchymosis:

Collection of blood in the subcutaneous tissue can result in periorbital ecchymosis. It is the most common manifestations of blunt injury. Ecchymosis is usually purplish red in color. Difference between orbital hemorrhage from fracture of anterior cranial fossa and black eye are as follows,

FEATURES	ANTERIOR CRANIAL FOSSA FRACTURE	LOCAL CAUSE
Limitation	Extravasated blood is limited sharply by palpebral fascia to orbital margin	No limitation
Color	Purplish	Buffy red
On movement of conjunctiva	No movement of hemorrhage	Hemorrhage moves with conjunctiva

If black eye is seen following head injury, it indicates base of skull fracture. Hemorrhage from skull fractures usually spreads diffusely in to the eyelid tissues after 24 to 48 hours. Ecchymosis is considered significant only when it causes edema of conjunctiva, posterior limit of hemorrhage cannot be made out or hemorrhage is large which results in restriction of ocular movements.

b) Emphysema:

Communication between orbital tissue and nasal sinuses results in the collection of air in the subcutaneous tissue of the orbit. Three types of ocular emphysema

1. Palpebral or Preseptal Emphysema: It is due to lacrimal bone fracture in front of tarso-orbital fascia. The air from the nose enters the lids via the nasolacrimal canal and there may be rupture of the lacrimal sac.

2. Orbital Emphysema: It occurs in orbital wall fracture behind tarso-orbital fascia and accumulation of air behind intact septum.

3. Orbitopalpebral Emphysema: In this type, air from the orbit travels along the tarso-orbital fascia, produces swelling of lid and conjunctiva and also rise in intra orbital pressure.

c)Traumatic Ptosis:

Ptosis can be due to

1. Oculomotor nerve palsy

2. Levator palpebrae superioris aponeurosis disinsertion

3. Edema (mechanical ptosis)

3. Damage to levator palpebrae superioris muscle

d) Laceration of lid:

Partial thickness or full thickness laceration can occur in lid. If the laceration involves medial fourth of lids, it may damage the puncta, canaliculi, lacrimal sac or naso lacrimal duct and also impairs the tear drainage to nose by direct injury.

e) Orbital fracture:

Orbital fractures are usually associated with adnexal and acute ocular injuries. On palpation discontinuity in bone(step off), abnormal mobility, pain at fracture sites, depression or subcutaneous emphysema in fractures involving paranasal sinuses is noted. Numbness in the area supplied by inferior orbital or supraorbital nerve indicates fracture passing through superior or inferior orbital margin involving the nerves.

1. Medial orbital wall fracture: If there is bleeding from nose with or without surgical emphysema, naso-orbital injuries (naso ethmoid or naso maxillary) should be suspected. It is most commonly associated with orbital floor fracture.

2. Inferior orbital wall fracture: It is divided into orbital rim fracture and inferior orbital floor fracture. Blow out fracture occurs due to objects greater than the orbital rim resulting in increased intra orbital pressure and fracture by direct force.

3. Lateral orbital wall fracture: Separation of articulation of zygoma with greater wing of sphenoid and zygomatico-frontal suture with depression of zygomatic arch occurs. Because of three characteristic sites of bony separation: lateral orbital rim, zygomatic arch and infraorbital rim, zygomatic bone fractures are commonly called tripod or trimalar fracture.

4. Superior orbital wall fracture: It includes frontal sinus and orbital roof fracture. Anesthesia in the area of supraorbital nerve with superior oblique muscle involvement from dislocation of trochlea, palpable deformity of superior orbital rim, superior rectus muscle weakness and damage to optic nerve may occur occasionally.

2. CONJUNCTIVA

a) Subconjunctival hemorrhage:

If the blood vessels of the conjunctiva are torn following injury it results in bleeding into the subconjunctival space. Hemorrhage occurs in the bulbar conjunctiva, but it can spread to the palpebral area because of anatomical predisposition. It is flat with sharply defined limits and appears as dark red patches or bright red color of various sizes.

Subconjunctival hemorrhage helps in localization of bony fracture. Example: In apex of the orbit fracture, blood tracks along the lateral rectus muscle. In orbital floor fracture, blood tracks into the lower fornix and lid. Grading of subconjunctival hemorrhage is as follows:

Grade I – Hemorrhage in one quadrant

Grade II – Hemorrhage in two quadrants

Grade III – Hemorrhage in three quadrants

Grade IV – Hemorrhage in four quadrants

b) Chemosis:

Conjunctiva appears swollen and may even protrude between the lids or folds over the cornea, which is due to the transudate accumulation in the subconjunctival space.

c) Conjunctival laceration:

As conjunctiva is very thin, it gets lacerated by direct impact. Conjunctiva is freely mobile, hence laceration is rarely extensive.

d) Conjunctival emphysema:

Presence of air under the conjunctiva is associated with fractures of lamina papyracea of the ethmoid or other paranasal sinuses and also laceration of their mucosa.

3. CORNEA:

a) Corneal abrasion:

Patient presents with photophobia, pain and redness. Corneal abrasion are generally sharp and it stains with fluorescein. It usually heals within 24 hours.

b) Recurrent epithelial erosion/Recurrent traumatic keratalgia:

Following trauma patient presents with recurrent attack of photophobia, lacrimation and pain on waking up in the early morning. Corneal abrasion damages the epithelium- basement membrane complexes and leads to recurrent epithelial defects.

c) Non penetrating corneal lacerations:

When stroma is exposed to tears and aqueous fluid after breakdown of epithelial barrier, small non penetrating corneal injuries usually self seals due to stromal swelling. Siedel test should be done to check for microscopic leaks.

d) Full thickness corneal tear:

In full thickness corneal tear aqueous humor leaks from the anterior chamber and is frequently associated with uveal tissue and iris proplase.

e) Corneal blood staining:

Corneal blood staining usually occurs after prolonged hyphema and elevated intraocular pressure. Cornea appears greenish or reddish brown in color. The process of

clearing starts slowly from periphery towards center and is due to scavenging action of the leucocytes.

4. ANTERIOR CHAMBER

a) Traumatic Hyphema:

Hyphema is due to accumulation of blood in the anterior chamber. Obstruction of trabecular meshwork by inflammatory debris and blood clot can result in rise in intraocular pressure.

Grading of hyphema:

0	Microhyphema (red blood cells within anterior chamber)
I	Blood occupying less than one third of the anterior chamber
II	Blood filling one third to one half of the anterior chamber
III	Blood filling one half to less than total of the anterior chamber
IV	Total clotted blood/blackball/8 ball hyphema

b) Traumatic iridocyclitis:

Iridocyclitis is due to breakdown of blood aqueous barrier resulting in accumulation of fibrin and protein in the anterior chamber. Patient presents with history of pain, redness and photophobia. Slit lamp examination shows anterior chamber cells and flare. Treatment is with corticosteroids and cycloplegics.

5. IRIS AND CILIARY BODY

a) Iridodialysis:

Iridodialysis is due to separation of iris from its root at the ciliary body. It act as second pupil and causes uniocular diplopia. It results in D- shaped pupil.

b) Pigmentary changes:

10-30 years after trauma, iris becomes darker than the iris in the non-traumatized eye known as “inverse heterochromia”.

C) Cyclodialysis:

It is due to detachment of ciliary body from the scleral spur and is characterised by severe hypotony. It is also associated with corneal edema, shallow anterior chamber and peaked pupil.

6. PUPIL:

a) Sphincter tear:

In sphincter tear, mid dilated pupil is seen and pupillary margins are irregular in shape and appears serrated.

b) Traumatic mydriasis and miosis:

Sphincter tear results in traumatic mydriasis and any inflammation in anterior chamber causes traumatic miosis.

7. LENS:

a) Subluxation and dislocation of lens:

In subluxation, displaced lens remains in the plane behind the iris in the patellar fossa. If the lens is completely away from the patellar fossa due to complete rupture of zonules it is called dislocation.

b) Traumatic cataract:

In trauma, aqueous humor enters the lens due to anterior capsule disruption causing cataract. Early maturation of senile cataract, Rosette cataract, Traumatic Zonular cataract, Diffuse concussion cataract, Opacities in the sub epithelial layer are the various types of cataract seen in trauma.

c) Vossius ring:

Vossius ring appear as circular ring of pigment deposition over the anterior lens capsule. It can be complete or incomplete ring which is due to compression of posterior pigment iris epithelium against anterior lens capsule following blunt injury.

8. VITREOUS

a) Vitreous detachment:

In trauma, posterior to the vitreous base there will be detachment of cortical vitreous from the retina. Retinal/vitreous hemorrhage may be the associated finding.

b) Vitreous hemorrhage:

There will be history of new onset floaters and sudden painless loss of vision in case of vitreous hemorrhage. Margins will be distinct and appears red in color. If it is associated with acute posterior vitreous detachment, fundus should be examined by

indirect ophthalmoscopy with scleral depression. Red blood cells are de-hemoglobinized and usually appears as khaki color in chronic cases.

c) Vitreous opacification:

Opacification usually occurs after hemorrhage from torn choroidal, retinal, or ciliary body vessels. Release of pigments from iris and retinal pigment epithelial cells may also lead to vitreous opacification. Pigment in the vitreous indicates underlying retinal tear or dialysis.

9. RETINA:

a) Commotio retinae or Berlin's edema:

After trauma edema occurs due to extravasation of blood between the choroid and sclera. This results in edema due to mechanical pressure on the choriocapillaries producing ischemia and transudation of edematous fluid into the retina. When macula is involved "cherry red spot" is seen.

b) Traumatic retinal detachment:

1. Immediate retinal detachment: It usually occurs at the time of injury in eyes that have undergone degenerative changes due to myopia, senile or post inflammatory.

2. Delayed retinal detachment: This occurs following contraction of the organized vitreous hemorrhage and cicatrization of the scleral rupture.

c) Purtscher's retinopathy

It occurs following occlusion of small arterioles by intravascular microparticles generated by underlying systemic conditions like fat embolism.

d) Traumatic retinal vascular occlusion:

Vascular occlusion may be seen rarely after a blunt trauma due to retinal vascular constriction or central retinal artery or vein occlusion.

e) Traumatic macular hole:

Macular hole is due to full thickness defect of neurosensory retina and trauma is the most common cause. It results in central vision loss.

f) Retinal dialysis:

Following trauma if nonpigmented pars plana epithelium is detached from the ora serrata, it can cause retinal dialysis. Inferotemporal and superonasal quadrant are the most common location. They are of two types: Anterior and posterior retinal dialysis.

g) Retinal tears:

Retinal tear occurs at the site of impact and it may predispose to retinal detachment. It is known as giant retinal tear, if the break extends for at least 3 clock hours or 90 degrees.

h) Macular edema:

In macula, large quantity of fluid gets absorbed due to thick Henle's layer and vasodilatation of underlying rich choroidal capillary bed. Resorption of fluid is difficult due to foveal avascular zone.

i) Terson's syndrome:

Intraocular hemorrhage is seen along with acute, spontaneous or trauma induced intracranial bleeding due to sudden rise in intraocular hemorrhage.

10. CHOROID:

a) Choroidal hemorrhage:

Choroidal hemorrhage appears as rounded, dark red -blue mount with pinkish edges.It is usually located at the equator or adjacent to the disc.

b) Choroidal detachment:

Choroidal detachment appears dome shaped due to its attachment at vortex veins. In this there will be accumulation of blood in between sclera and choroid

c) Choroidal rupture:

Choroidal rupture occurs following trauma due to tear of the inelastic bruch's membrane along with choriocapillaries and retinal pigment epithelium. It can be single or multiple and is usually located concentric to the disc.

Direct choroidal rupture	• Occurs due to direct impingement of force • Broad, large and irregular in shape
Indirect choroidal rupture	• Occurs due to countercoup force • Present within posterior pole

d) Chorioretinitis sclopetaria:

It occurs when high velocity projectile object penetrates the globe adjacent to the site of injury, which results in tearing of the choroid and neurosensory retina.

11. SCLERA:

a) Scleral rupture:

Scleral ruptures have more chances for developing intraocular bacterial contamination. They are usually solitary but multiple ruptures can occur in severe cases. If sclera ruptures at the impact site it is called direct scleral rupture, whereas if it occurs at weakened scleral area then it is called as indirect scleral rupture.

12. INTRAOCULAR PRESSURE:

In case open globe injury is present IOP measurement should be avoided.

a) Low intraocular pressure-causes:

Retinal and choroidal detachment, ciliary body ischemia and wound leak.

b) High intraocular pressure-causes:

Angle recession glaucoma, retrobulbar hemorrhage, hyphema, anterior chamber inflammation and hemolytic glaucoma.

13. OPTIC NERVE INJURY:

The intracranial portion of optic nerve is fixed and firmly adherent to the underlying sheaths, so they are more vulnerable to trauma. There are several mechanisms responsible for vision loss:

- Optic nerve transection
- Optic nerve avulsion
- Optic nerve sheath hemorrhage
- Orbital emphysema
- Localized orbital hemorrhage

- Diffuse orbital hemorrhage
- Bony fracture may impinge on optic nerve

14. OTHER CRANIAL NERVES

a) Oculomotor nerve:

In head injury third neve damage can be either due to direct or indirect effect resulting in palsy. Direct oculomotor injury may be the result of rootlet avulsion, distal fascicular damage extreme distraction of the nerve or defective blood supply. Indirect injury is due to displacement, compression or deformity of oculomotor nerve by space occupying lesions such as cerebral aneurysm or expanding hematoma.

b) Trochlear nerve:

Trochlear nerve runs at the free tentorial edge after decussating around the dorsal midbrain and contrecoup injury usually occurs when the nerve is compressed against tentorium. Unilateral trochlear nerve injury occurs after frontolateral impact whereas bilateral injury can occur from midfrontal impact.

c) Abducent nerve:

Long intracranial course of the abducent nerve makes it more vulnerable to injuries and usually occurs at the site of dural entry point and at the petrous apex. Traumatic sixth nerve palsy is usually seen in combination with skull fracture, facial fracture, intracranial hemorrhage or elevated intracranial pressure, but it may occur in the absence of such lesions.The mechanism of injury is stretching of nerve by acceleration in

the mid-sagittal plane. Apex of the petrous bone acts as the fulcrum, so the nerve is contused, compressed and stretched at this point.

d) Facial nerve:

Facial nerve injury is usually seen in blunt or penetrating trauma to the petrous portion of the temporal bone. Facial nerve weakness can be partial or complete and it can manifest immediately or as a delayed manifestation. Late presentation is due to pressure effect from edema, hemorrhage or granulation tissue. Patient complains of inability to close the lids and immobility of entire half of face.

15. INTRAOCULAR FOREIGN BODY:

Ocular foreign bodies should be examined and removed properly as it may cause serious complications in the eye. Modes of damage are post traumatic iridocyclitis, foreign body reaction, infection, mechanical effects and finally sympathetic ophthalmitis. The foreign body can be either extraocular or intraocular.

i) Extraocular foreign body:

Foreign body is usually seen in lid, sclera, conjunctiva and cornea. Foreign bodies that cause extraocular injuries are dust, sand, iron particles, wings of insects etc. If foreign body is not eliminated by tears, it produces discomfort to the patient and introduction of infection may cause permanent damage to vision.

ii) Intraocular foreign body:

In this foreign body is seen in the angle of anterior chamber, iris, lens, vitreous, retina and intraorbital. Foreign body that penetrates the eye are chips of iron, wood,

copper, steel, stone, glass, lead particles etc. If the velocity is great foreign body may traverse the eye causing a double perforation and gets lodged in the orbital tissues.

- **FB in the anterior chamber**: It usually rests in the bottom of anterior chamber. It may remain quiet or produces iridocyclitis and corneal edema.
- **FB in lens**: Foreign body rests in the nucleus. Effect in the crystalline lens varies from localized opacity to total opacification of lens. It may produce secondary glaucoma, uveitis or lens may be absorbed.
- **FB in posterior segment**: Usually metals with high velocity enters the posterior segment causing intraocular hemorrhage and infection as a major complication or it may lie quietly in vitreous for years.

EFFECT OF RETAINED FOREIGN BODIES:

Ocular reaction to the foreign body depends upon the composition of the particle. There are three types of ocular reactions:

i) No specific reaction – Non organized substances

ii) Non specific or occasionally specific – chemical reaction

iii) Proliferative response – Organized materials

COPPER:

In its pure form, copper causes acute iridocyclitis with hypopyon, retinal detachment, vitreous abscess and phthisis bulbi. Other manifestations are Kayser-Fleischer ring, Sunflower cataract and deposition of golden plaques at the posterior pole.

Copper deposits are usually extracellular whereas iron deposits are intracellular causing degeneration of cells. Blindness is usually never seen in copper deposition whereas it is the rule in iron deposition.

IRON:

Deposition of iron in ocular tissues produces brown deposit known as ‘rust ring’.Chemical effects of iron in the ocular tissues is called as ‘siderosis’.They are of two types:

1. Direct siderosis: Iron is deposited near the foreign body.
2. Indirect siderosis: Iron metal is diffusely deposited throughout the ocular tissue

PART - II

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES:

- To evaluate the pattern of ocular manifestations in head injury
- To study the final visual outcome
- To educate patients about safety measures

INCLUSION CRITERIA:

- All head injury patients
- Presenting for first time
- Unilateral and bilateral
- All mode of injuries
- All age group
- Male and female

EXCLUSION CRITERIA:

- Previous H/O head and ocular injury
- Congenital anomalies
- Unconscious patients who did not subsequently recover adequate consciousness

MATERIALS AND METHODS

MATERIALS AND METHODS

All head injury patients with ocular manifestations attending casualty, neurosurgery and ophthalmology department in Thanjavur medical college from December 2017 to May 2019 were taken up for study after obtaining informed consent.

This is a prospective and analytical study of 164 head injury patients who presented to our hospital with ocular manifestations. All head injury patients were examined, which includes present history of trauma, past history of any ocular and systemic disease.

In proforma all details were recorded for each patient with head injury: age and sex, mode of injury, history of head injury and ocular injury, presence or absence of safety measures and alcohol intake, history of loss of consciousness, any symptoms and signs following the injury.

Following ocular examinations were done in head injury patients:

- A thorough clinical examination using torch light
- Visual acuity by Snellen chart. In few patients vision examination was done bed side considering their general condition
- Color vision examination by Ishihara's plate
- Pupillary assessment
- Slit lamp examination, whenever it was possible
- Fundus examination by direct and binocular indirect ophthalmoscopy
- B scan -if there is any posterior segment pathology

- X ray or CT scan/MRI scan
- Gonioscopy and Intraocular pressure measurement (if required)

Patients with head trauma with multiple injuries were hospitalized for further management and specialized care. All the patients were reviewed for follow up 1 week after being seen last in the hospital. For those who had unexplained visual loss long term follow up was advised every month. If patients required higher centre management, they were immediately referred.

Patients were also examined by other specialities like neurosurgery, orthopaedics, general surgery, plastic surgery, ENT and Oro - maxillo -facial surgery depending on the presentation.

CLINICAL PROCEDURES AND INVESTIGATIONS

1) VISUAL ACUITY ASSESSMENT:

Snellen chart was used to assess the visual acuity of the patient.

2) DIRECT OPHTHALMOSCOPY:

A convergent beam of light is reflected into the patient's pupil. The emergent rays from any point of the patient's fundus reach observer's retina through the viewing hole in the ophthalmoscope. It works on the basic optical principle of glass plate ophthalmoscope introduced by Von Helmholtz. This is dependent on the refractive state of the patient.

3) INDIRECT OPHTHALMOSCOPY:

Indirect ophthalmoscopy involves making the eye highly myopic by placing a high power convex lens (+ 20 D) in front of the eye so that a real, inverted and laterally reversed image is formed in front of the lens. The image is formed close to the principle focus of the lens, between the lens and the observer. To view the retinal periphery and ora serrata scleral indendation was done.

4) GONIOSCOPY:

Structures in the anterior chamber angle can be visualized using gonioscopy (goldmann three mirror lens). The central part provides 30 degree upright view of the posterior pole. Equatorial mirror (73°, largest and oblong shaped) enables visualization from 30 degree to the equator. Peripheral mirror (67°, intermediate and square shaped) used for visualization between equator and the ora serrata. Gonioscopy mirror (59°, smallest and dome shaped) may be used for visualizing the anterior chamber angle.

5) B-SCAN ULTRASOUND:

B-Scan or brightness modulation scan provides two dimensional images of a series of dots and lines. It provides the topographic information of shape, location, extension, mobility and gross estimation of thickness of the tissue.

It is useful in patients with opaque media and also to detect vitreous hemorrhage, choroidal detachment, retinal detachment, subluxated or dislocated lens, localization of intraocular foreign body and vitreous incarceration in the wound.

6) PLAIN X-RAYS:

Plain x-rays are used in case of orbital wall fractures and to localize foreign body

1. X-Ray skull PA

2. Rheese method-parieto orbital oblique projection: This view demonstrates optic canal in cross section, lying in the lower outer quadrant of the orbital shadows.

3. Modified Waters's nose-chin position: This view shows a fracture of the orbital floor and prolapse of soft tissue into the maxillary antrum.

4. Caldwell's view: This view will provide a clear view of frontal sinus, ethmoidal sinus and also superior orbital rim can be evaluated.

5. Lateral view: To view orbital roof fracture.

6. Submento vertex view: It demonstrates lateral orbital wall fracture.

7) COMPUTED TOMOGRAPHY SCAN (CT):

CT is a technique of thin slice radiography, which is performed in a rotating basis through a 360 degree and then data are computed, assimilated and reconstructed to create two dimensional images of any part of the body. It is the standard imaging technique in traumatized eye and orbit.

CT scan is important in case of head injury with ocular manifestations for detecting intracranial abnormalities, orbital wall fractures and to locate any intraocular foreign body. Retained foreign bodies of sufficient size and radiodensity can be easily located.

8) MAGNETIC RESONANCE IMAGING (MRI):

MRI provides high resolution of any part of the body without ionizing radiation. It characterizes tissue by spectroscopical analysis. MRI is less useful in evaluation of trauma than CT and ultrasound because magnetic foreign body, if present could be dislodged and produce further damage to the eye.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

This study includes 164 cases of head injury with ocular manifestations who presented at Thanjavur Medical College from December 2017 to May 2019.

TABLE 1: AGE DISTRIBUTION

Age (years)	No of cases	Percentage (%)
1-10	2	1.2%
11-20	13	7.9%
21-30	61	37.2%
31-40	40	24.4%
41-50	22	13.4%
51-60	12	7.3%
61-70	9	5.5%
71-80	5	3.0%

In this study young adult groups were most commonly affected. Out of 164 head injury patients with ocular manifestations, only 2 cases (1.2%) were seen in the age group of 1-10 years, 13 patients (7.9%) in 11-20 years, 61 cases (37.2%) in 21-30 years, 40 patients (24.4%) in 31-40 years, 22 cases (13.4%) in 41-50 years, 12 cases (7.3%) in 51-60 years, 9 cases (5.5%) in 61-70 age group, 5 cases (3%) in 71-80 age group.

FIGURE 1: AGE DISTRIBUTION

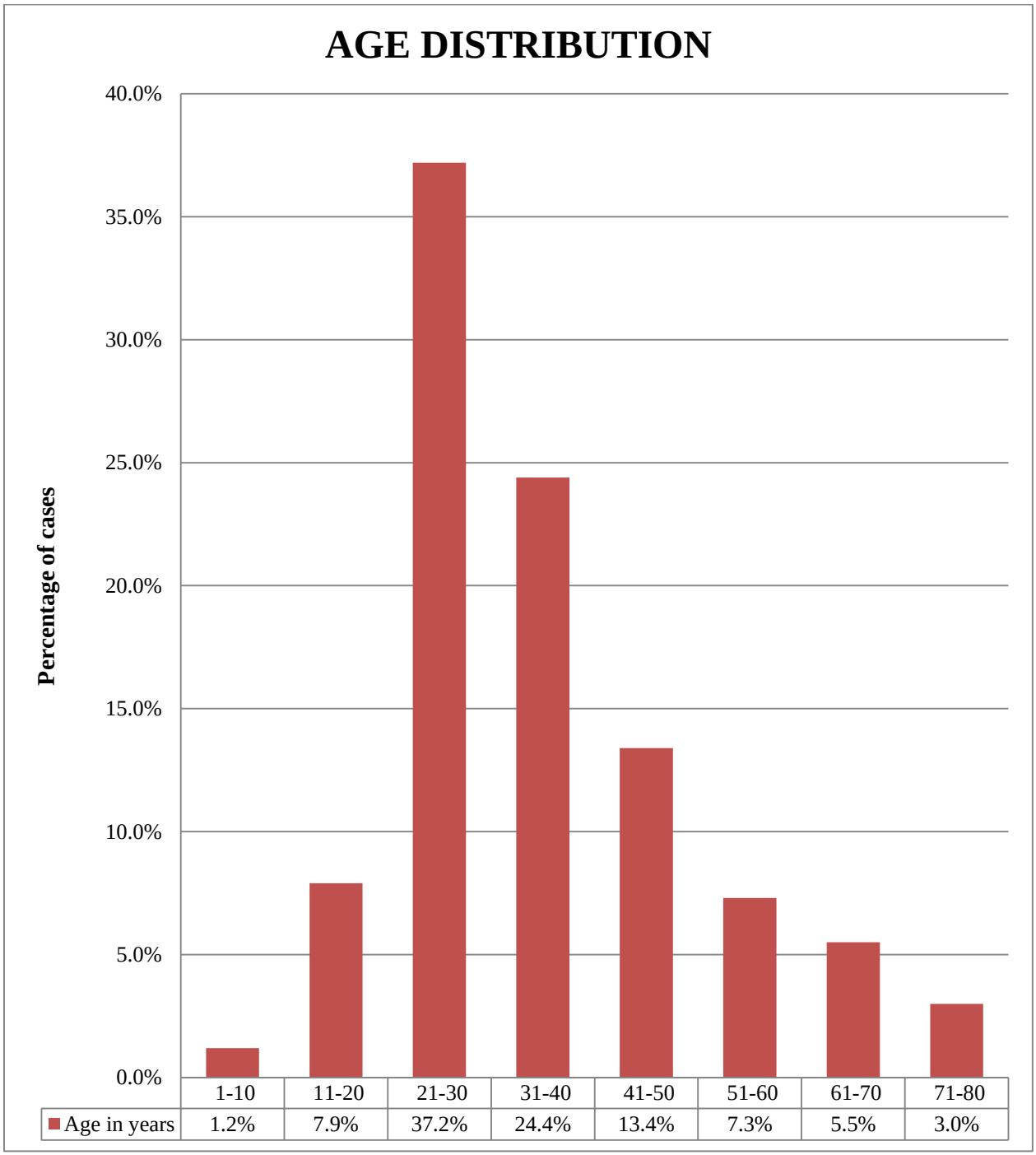


TABLE 2: SEX DISTRIBUTION

Sex	No of cases	Percentage (%)
Male	144	87.8%
Female	20	12.2%

Out of 164 head injury patients, 144 cases were males (87.8%) and 20 cases were Females (12.2%).

FIGURE 2: SEX DISTRIBUTION

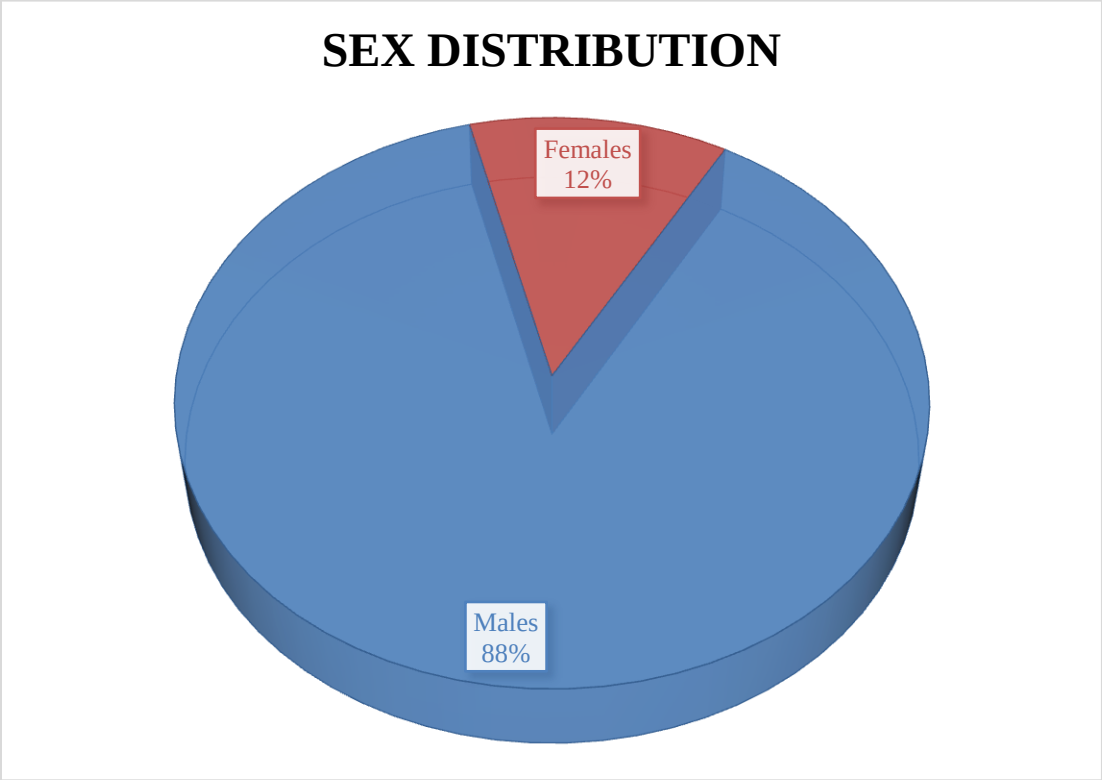


TABLE 3: MODE OF INJURY

Mode of injury	No of cases	Percentage (%)
RTA	123	75%
pedestrian	6	4%
2 wheeler	107	65%
4 wheeler	10	6%
Assault	24	14.6%
Accidental self fall	17	10.4%

Out of 164 head injury patients, road traffic accident was the common mode of head injury which is seen in 123 patients (75%) followed by assault in 24 patients (14.6%) and accidental self fall in 17 patients (10.4%). Among two wheeler users, helmet was worn only by 13 patients (12%) and not worn by 94 patients (88%).

FIGURE 3: MODE OF INJURY

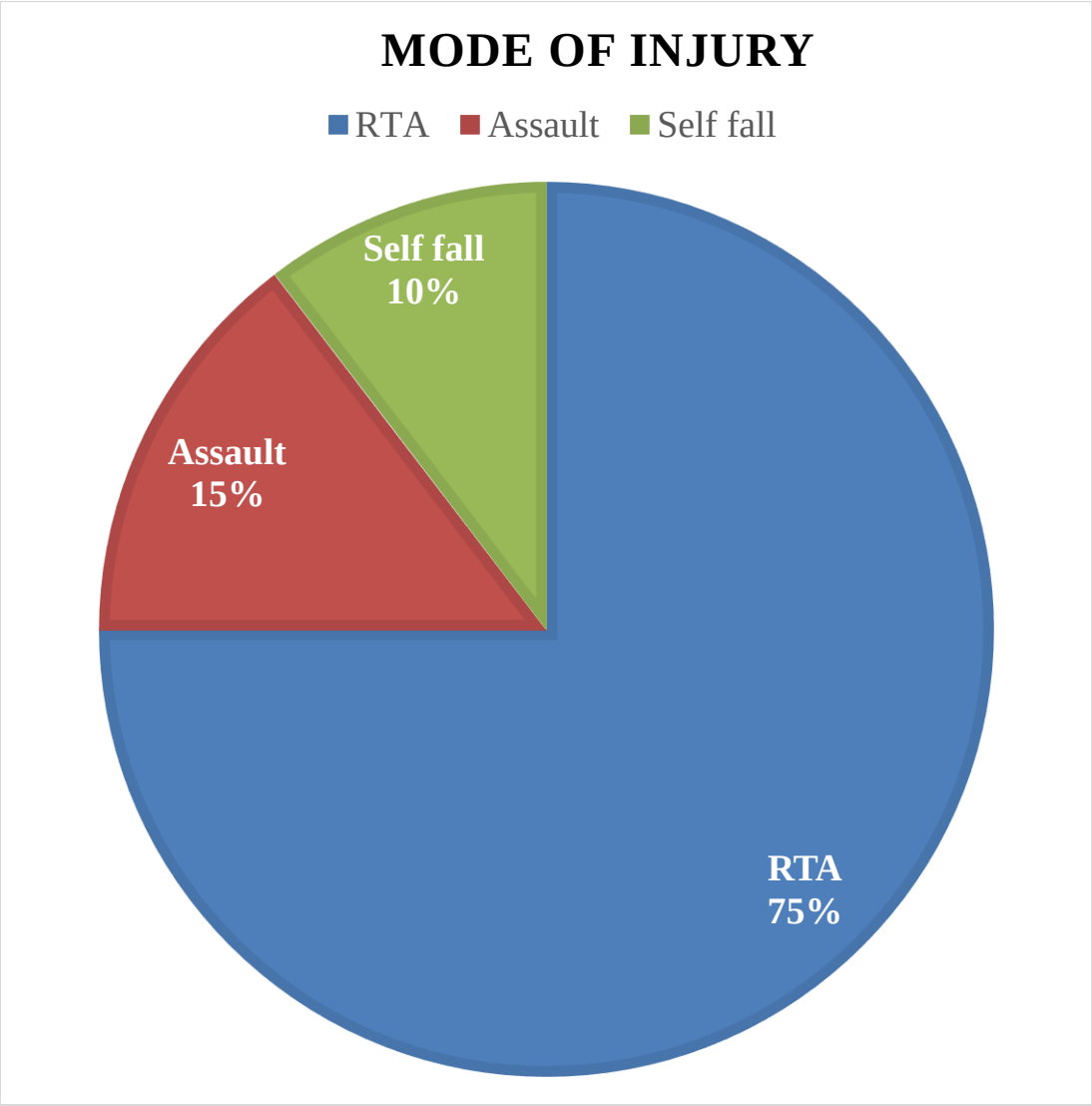


TABLE 4: EYE INVOLVED

Eye	No of cases	Percentage (%)
Unilateral involvement	156	95.1%
Right eye	42	25.6%
Left eye	114	69.5%
Bilateral involvement	8	4.9%

Out of 164 head injury patients, unilateral ocular involvement was seen in 156 cases (95.1%) in which right eye was involved in 42 cases (25.6%) and left eye in 114 patients (69.5%) and bilateral ocular involvement was seen in 8 patients (4.9%).

FIGURE 4: EYE INVOLVED

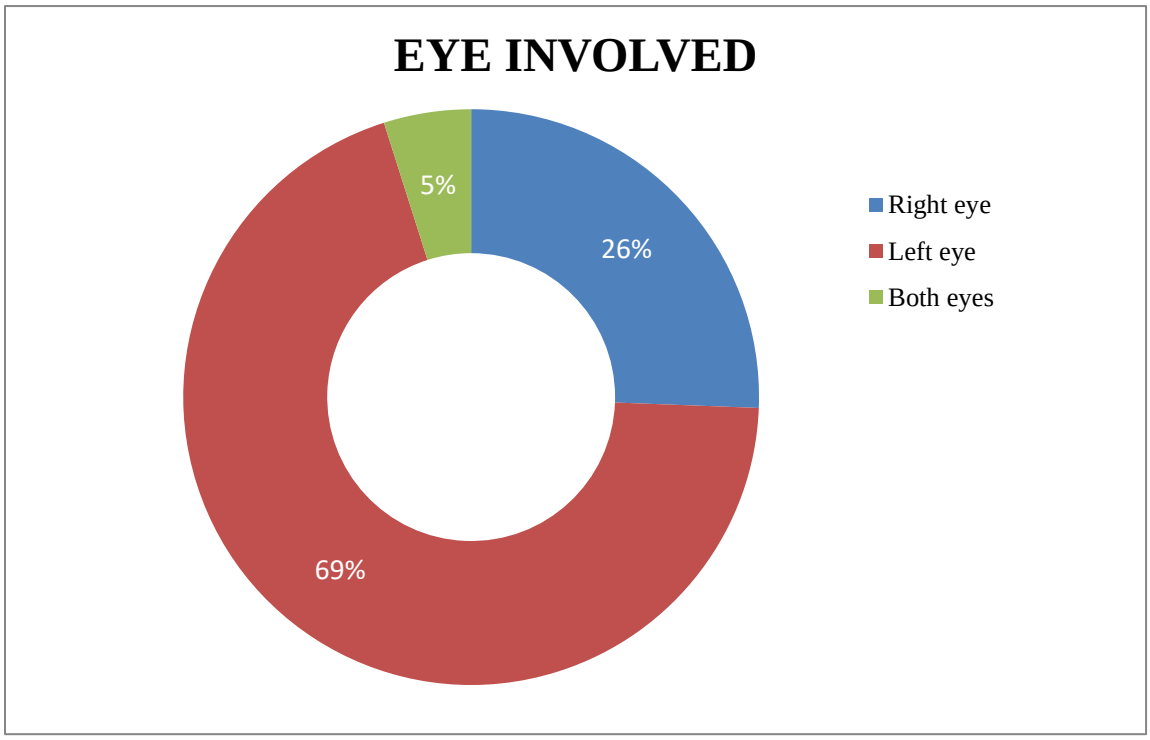
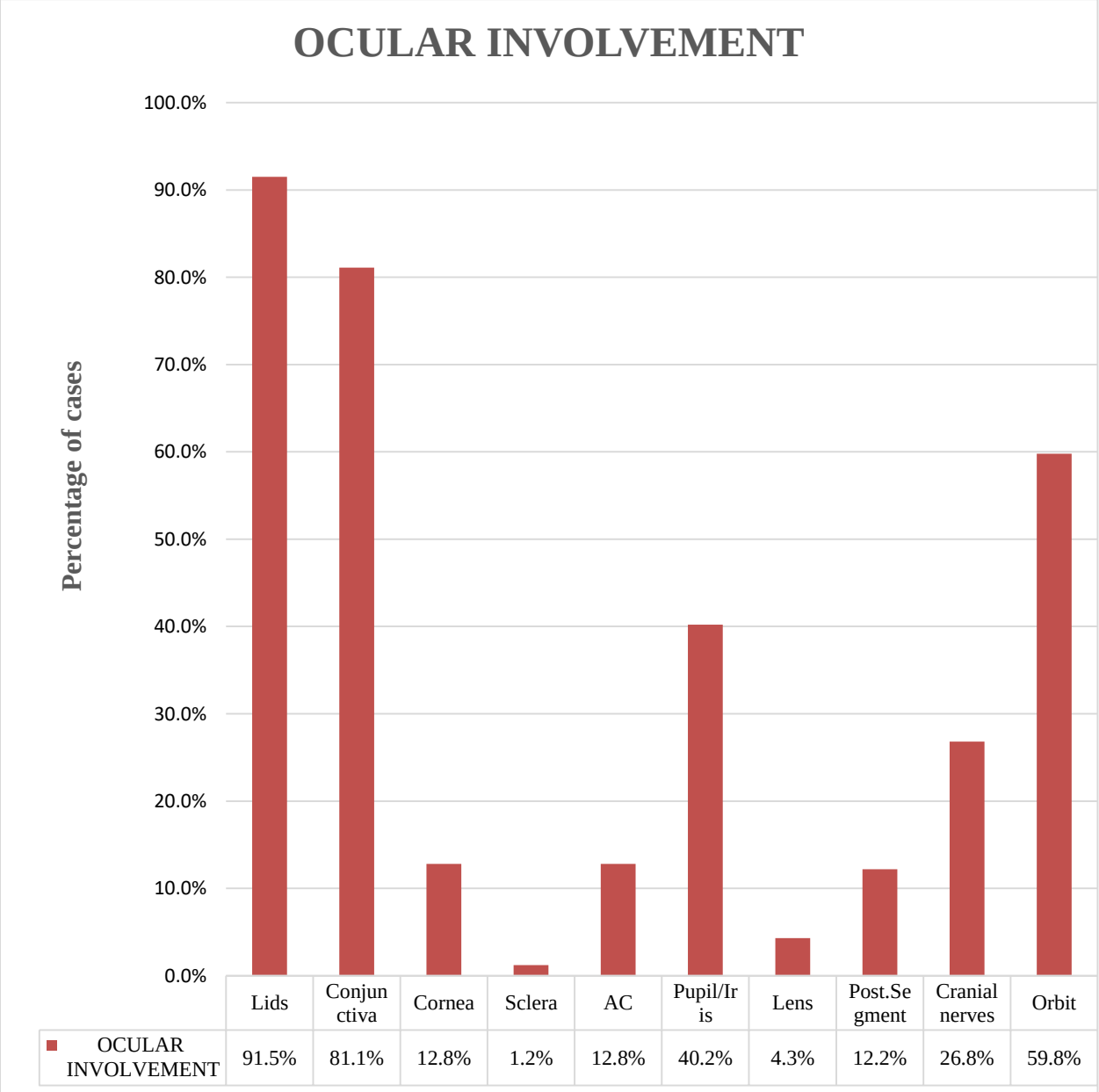


TABLE 5: OCULAR INVOLVEMENT

Ocular involvement	No of cases	Percentage (%)
Lids	150	91.5%
Conjunctiva	133	81.1%
Cornea	21	12.8%
Sclera	2	1.2%
Anterior chamber	21	12.8%
Pupil/iris	66	40.2%
Lens	7	4.3%
Posterior segment	20	12.2%
Cranial nerves	44	26.8%
Orbital wall	98	59.8%

Out of 164 head injury patients, lids were involved in 150 patients (91.5%), conjunctiva in 133 patients (81.1%), cornea in 21 cases (12.8%), sclera was involved in 2 patients (1.2%), anterior chamber manifestations was seen in 21 cases (12.8%), pupil and iris manifestations was seen in 66 cases (40.2%), lens was involved in 7 cases (4.3%), posterior segment manifestations was seen in 20 cases (12.2%) cranial nerves involvement in 44 cases (26.8%) and orbital wall fractures was seen in 98 patients (59.8%).

FIGURE 5 : OCULAR MANIFESTATIONS



	Lids	Conjunctiva	Cornea	Sclera	AC	Pupil/Iris	Lens	Post.Segment	Cranial nerves	Orbit
OCULAR INVOLVEMENT	91.5%	81.1%	12.8%	1.2%	12.8%	40.2%	4.3%	12.2%	26.8%	59.8%

TABLE 6: LID MANIFESTATIONS

Lids	No of cases	Percentage(%)
Edema	124	75.6%
Ecchymosis	125	76.2%
Lid tear	9	5.5%
Upper lid	4	2.4%
Lower lid	5	3.0%
Canaliculi	3	1.8%
Lagophthalmos	3	1.8%
Ptosis	13	7.9%

In this study, 124 patients had periorbital edema (75.6%), 125 patients had periorbital ecchymosis (76.2%), 9 patients had lid tear (5.5%) with canalicular involvement in 3 patients (1.8%), 3 patients had lagophthalmos (1.8%) and 13 patients had ptosis (7.9%).

FIGURE 6: LID MANIFESTATIONS

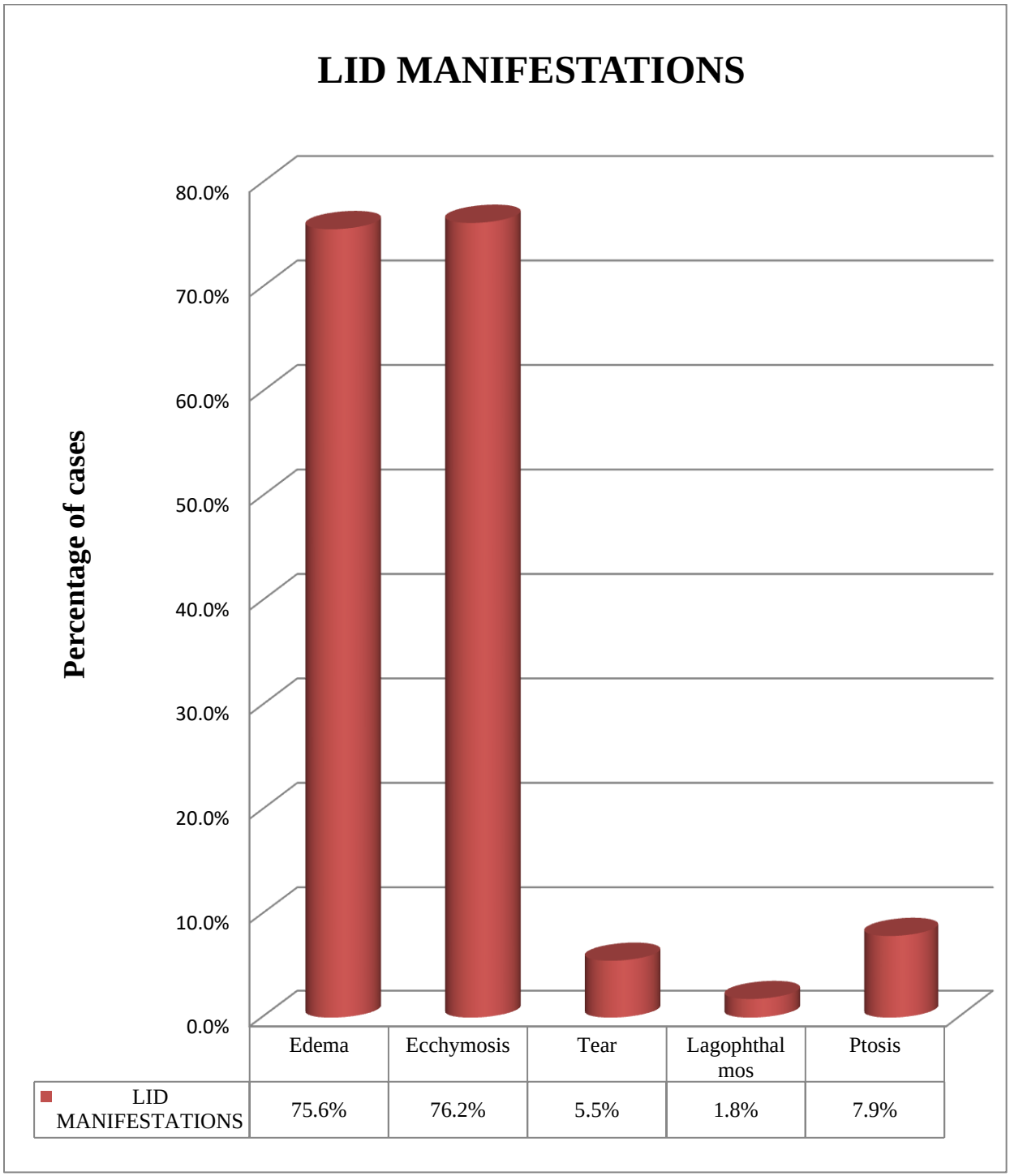


TABLE 7: CONJUNCTIVAL MANIFESTATIONS

Conjunctiva	**No of cases**	**Percentage (%)**
Subconjunctival hemorrhage	95	57.9%
Chemosis	15	9.1%
Congestion	17	10.4%
Conjunctival tear	6	3.7%

Out of the 164 head injury patients, 95 patients had subconjunctival hemorrhage (57.9%), 15 cases had conjunctival chemosis (9.1%), conjunctival congestion is seen 17 patients (10.4%) and conjunctival tear in 6 patients (3.7%).

FIGURE 7: CONJUNCTIVAL MANIFESTATIONS

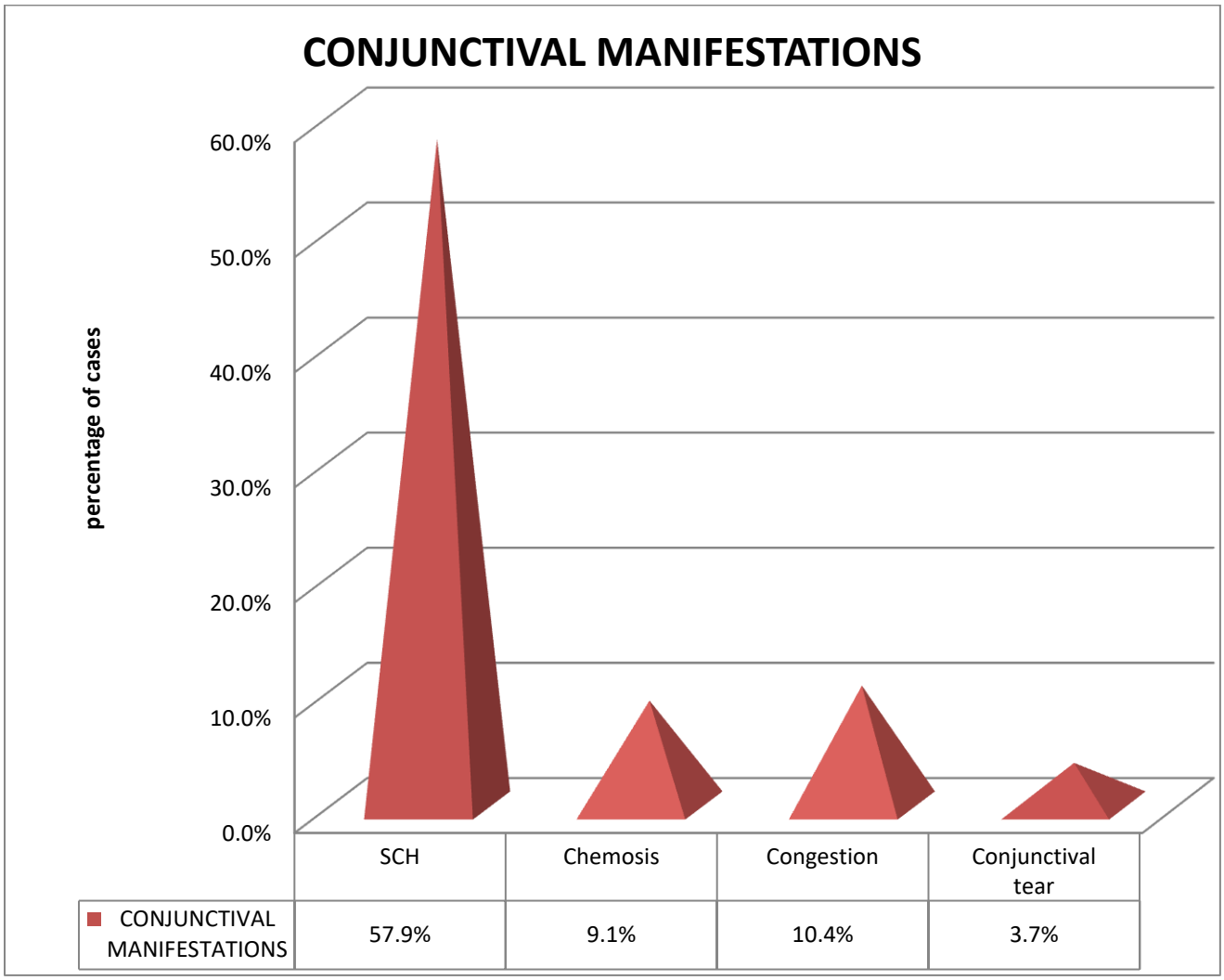


TABLE 8: CORNEAL MANIFESTATIONS

Cornea	**No of cases**	**Percentage (%)**
Abrasion	5	3.0%
Edema	8	4.9%
Tear	4	2.4%
Foreign body	2	1.2%
Exposure keratitis	2	1.2%

Out of the 164 patients, 21 patients had corneal involvement. Out of these 5 patients had corneal abrasion (3.0%), 8 patients had corneal edema (4.9%), 4 patients had corneal tear (2.4%), Foreign body in 2 patients (1.2%) and Exposure keratitis in 2 patients (1.2%).

FIGURE 8: CORNEAL MANIFESTATIONS

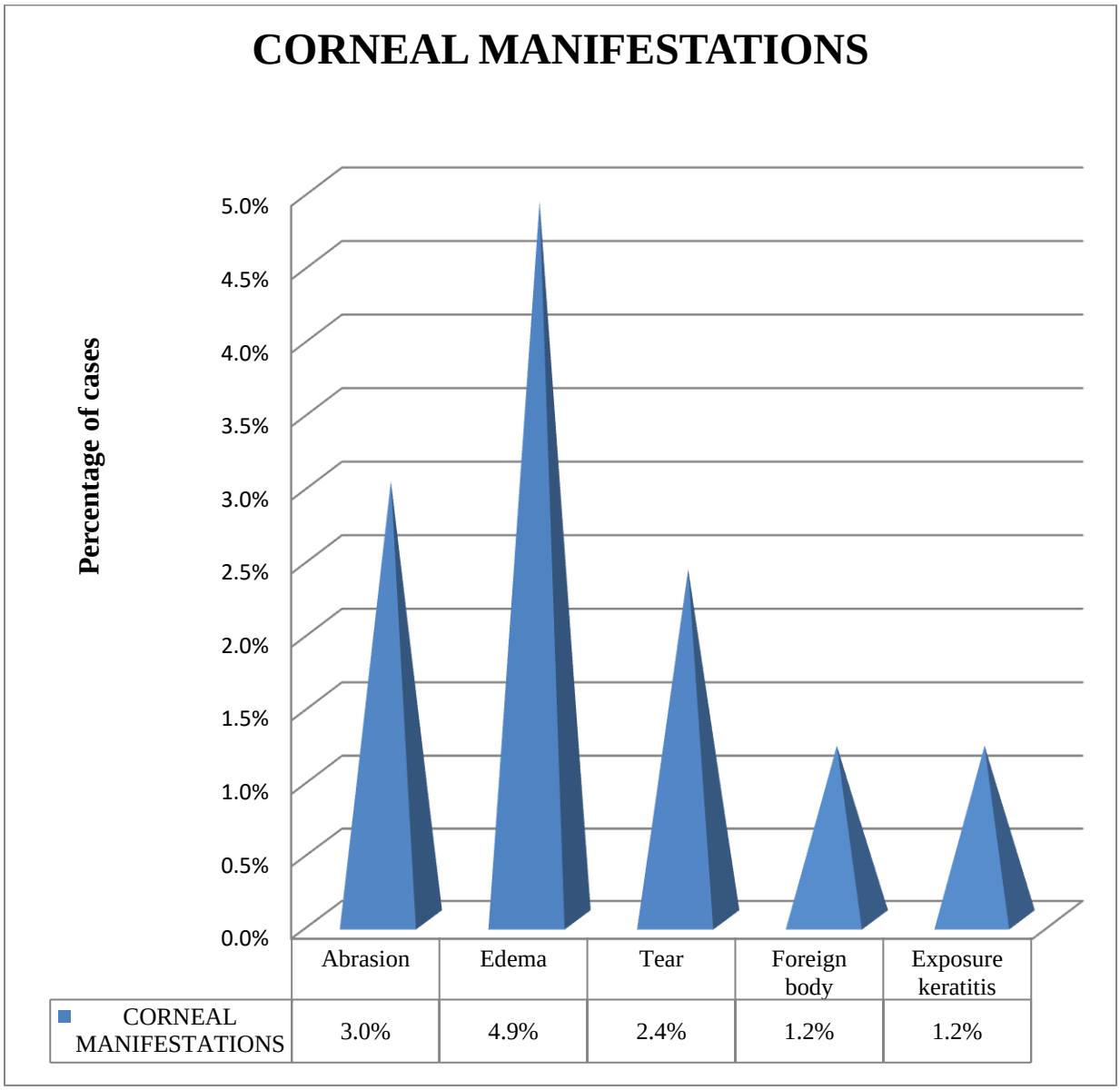


TABLE 9: SCLERAL MANIFESTATIONS

Sclera	**No of cases**	**Percentage (%)**
Tear	2	1.2%

Out of 164 head injury patients, 2 patients (1.2%) had scleral tear.

TABLE 10: ANTERIOR CHAMBER MANIFESTATIONS

Anterior chamber	No of cases	Percentage (%)
Hyphema	5	3.0%
Traumatic uveitis	12	7.3%
Vitreous	4	2.4%

Of the 164 patients, 5 had hyphema (3%) and 12 patients had Traumatic uveitis (7.3%) and 4 patients had vitreous in anterior chamber (2.4%).

FIGURE 9: ANTERIOR CHAMBER MANIFESTATIONS

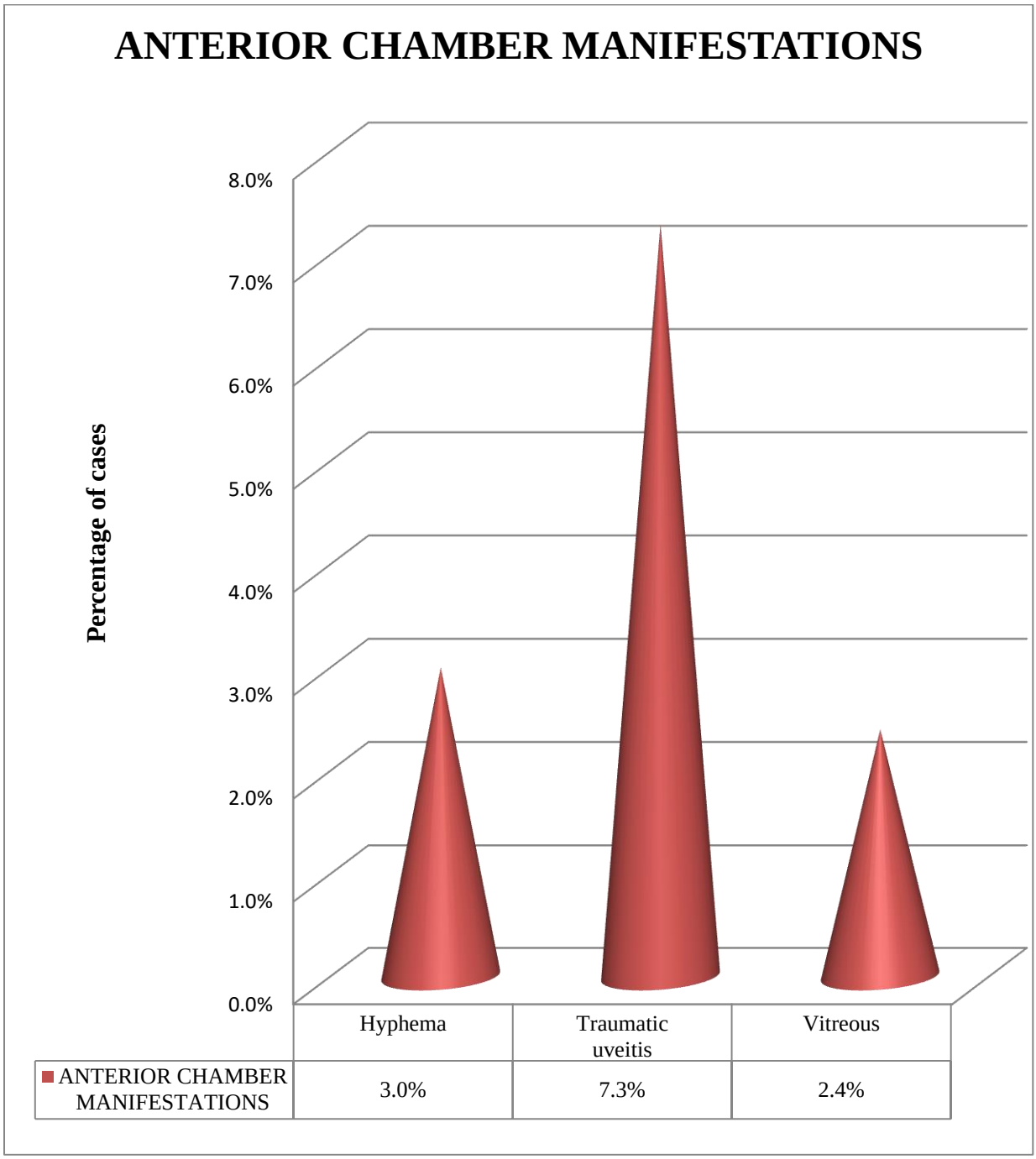


TABLE 11: PUPIL/IRIS MANIFESTATIONS

Pupil/Iris	No of cases	Percentage (%)
RAPD	21	12.8%
NRTL	14	8.5%
SRTL	21	12.8%
D-Shaped	3	1.8%
Iris prolapse	3	1.8%
Sphincter tear	4	2.4%

In this study, out of 164 patients 21 cases had relative afferent pupillary defect (12.8%), 14 patients had pupil which is not reacting to light (8.5%), 21 patients had pupil which is sluggishly reacting to light (12.8%), D shaped pupil in 3 patients (1.8%) , Sphincter tear in 4 patients (2.4%) and 3 patients had iris prolapse (1.8%).

FIGURE 10: PUPIL/IRIS MANIFESTATIONS

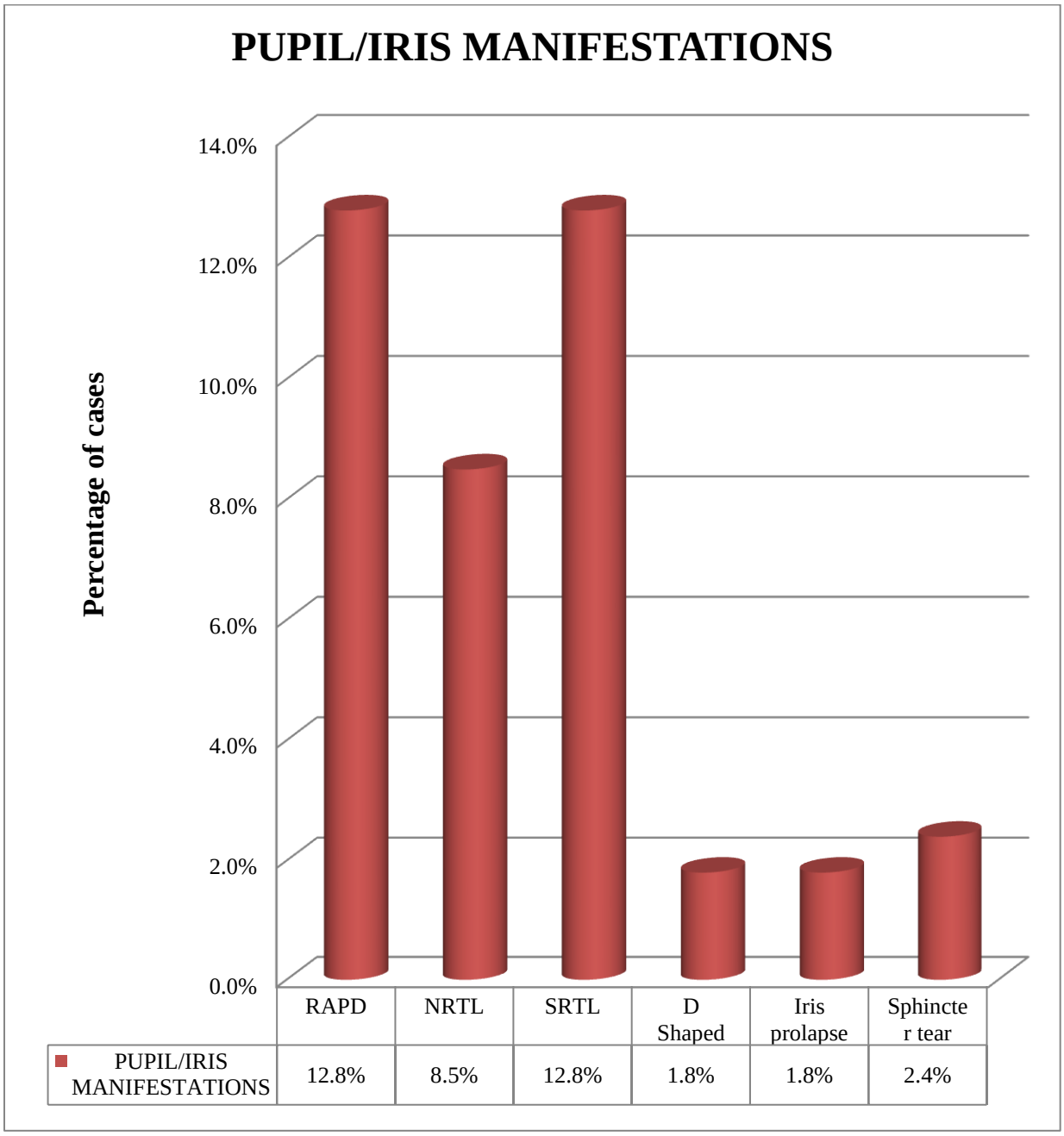


TABLE 12: LENS MANIFESTATIONS

Lens	No of cases	Percentage
Traumatic cataract	2	1.2%
Subluxation of lens	3	1.8%
Dislocation of lens	2	1.2%

Among 164 head injury cases, 2 patients had traumatic cataract (1.2%), 3 patients had subluxation of lens (1.8%) and 2 patients has dislocation of lens (1.2%).

FIGURE 11: LENS MANIFESTATIONS

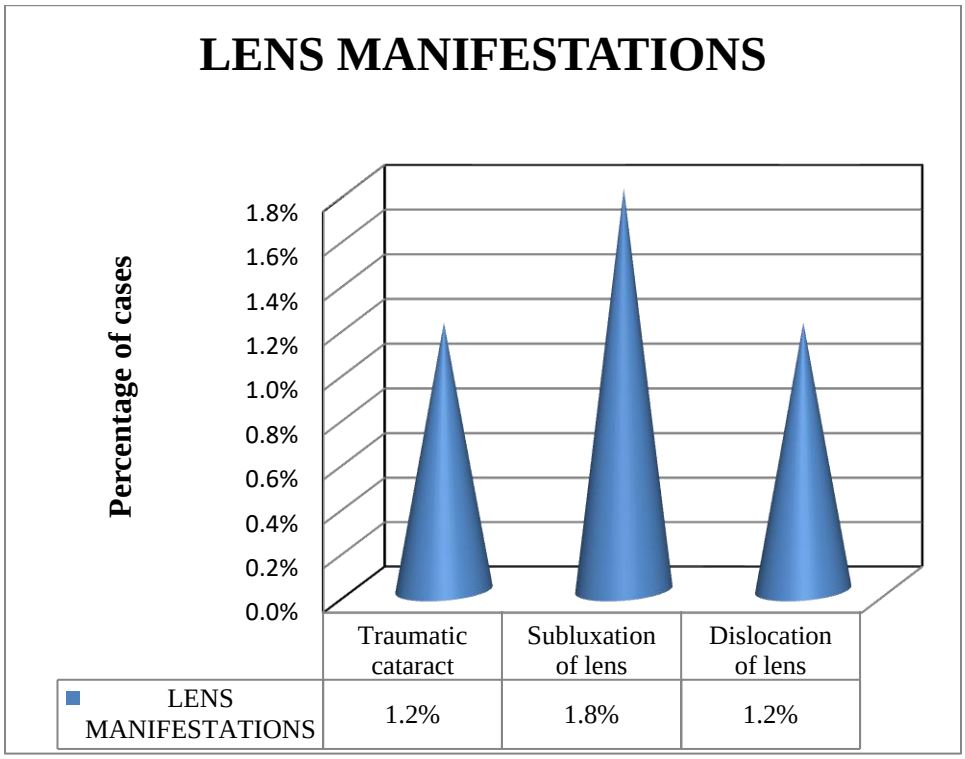


TABLE 13: POSTERIOR SEGMENT MANIFESTATIONS

Posterior segment	No of cases	Percentage (%)
Macular edema	7	4.3%
Berlin's edema	3	1.8%
Vitreous hemorrhage	4	2.4%
Retinal tear	2	1.2%
Retinal hemorrhage	2	1.2%
Purtscher retinopathy	1	0.6%
Papilledema	1	0.6%

Out of 164 patients, macular edema was seen in 7 patients (4.3%), Berlin's edema in 3 patients (1.8%),Vitreous hemorrhage in 4 patients (2.4%), Retinal tear in 2 patients (1.2%), Retinal hemorrhage in 2 patients (1.2%), Purtscher retinopathy in 1 patient (0.6%) and 1 patient had papilledema (0.6%).

FIGURE 12: POSTERIOR SEGMENT MANIFESTATIONS

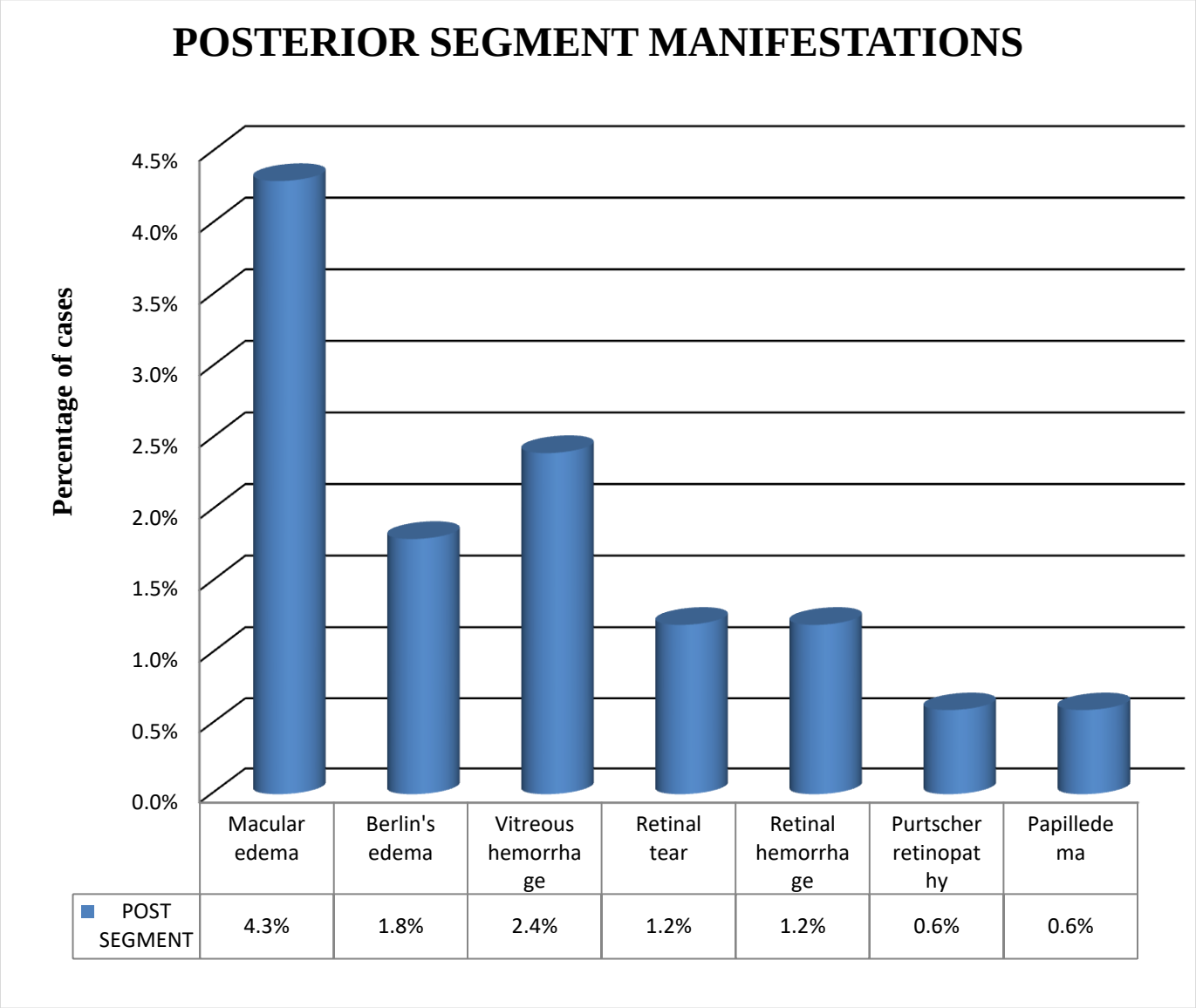


TABLE 14: CRANIAL NERVES INVOLVEMENT

Cranial nerves	No of cases	Percentage (%)
Optic nerve	21	12.8%
Oculomotor nerve	13	7.9%
Trochlear nerve	3	1.8%
Abducent nerve	4	2.4%
Facial nerve-LMN	3	1.8%

Out of 164 patients, 44 patients had cranial nerve involvement (26.8%).Traumatic optic neuropathy was seen in 21 patients (12.8%). Oculomotor nerve was involved in 13 cases (7.9%), Trochlear nerve in 3 patients (1.8%), Abducent nerve in 4 patients (2.4%) and LMN Facial nerve palsy in 3 patients (1.8%).

FIGURE 13: CRANIAL NERVE INVOLVEMENT

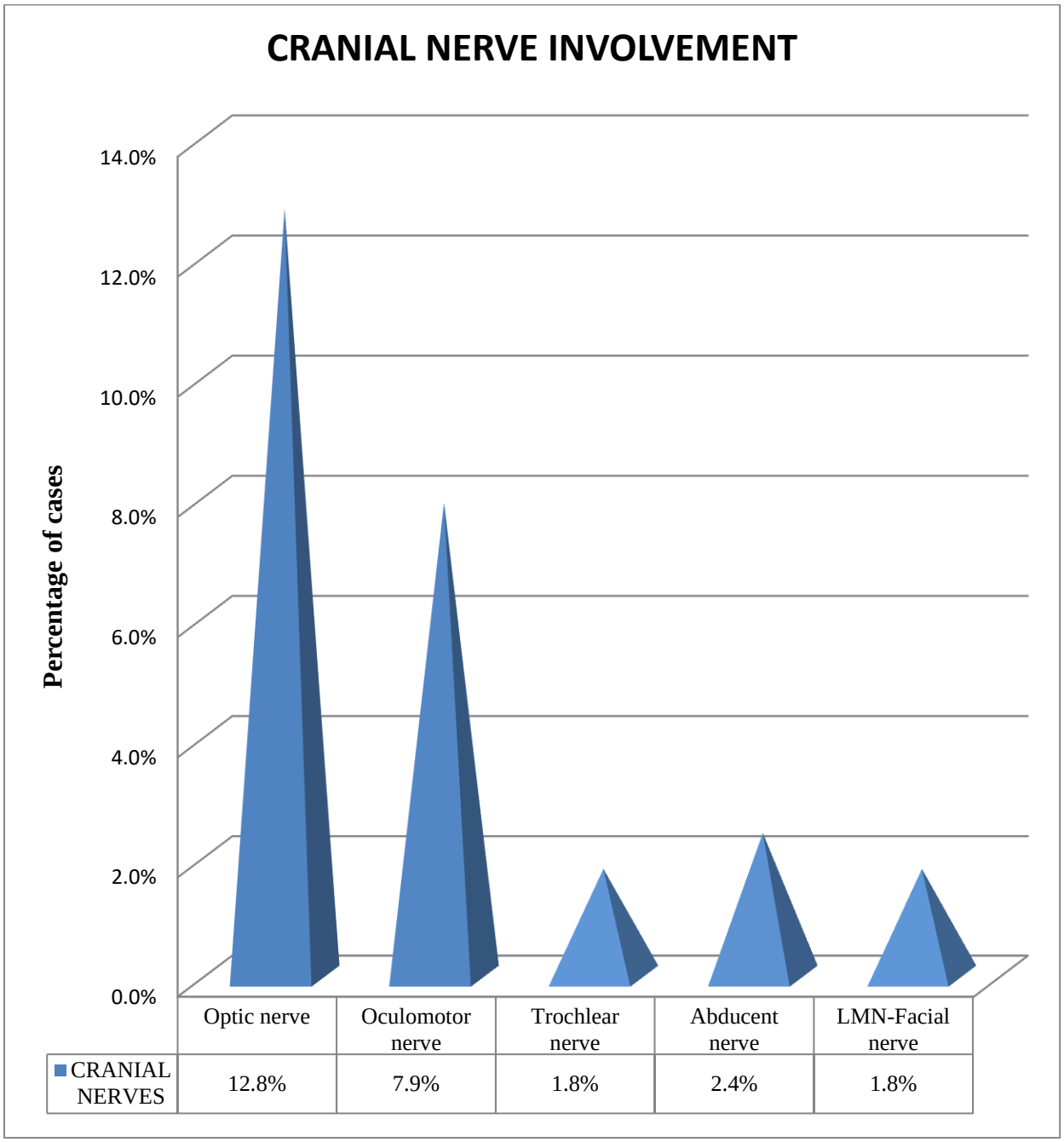


TABLE 15: ORBITAL WALL MANIFESTATIONS

Orbital wall	No of cases	Percentage (%)
Roof	26	15.9%
Lateral wall	38	23.2%
Medial wall	9	5.5%
Floor	25	15.2%
Combined	12	7.3%

In this study, out of the 164 patients 26 had orbital roof fracture (15.9%), lateral wall fracture was seen in 38 cases (23.2%), medial wall fracture in 9 patients (5.5%), orbital floor fracture in 25 cases (15.2%) and combined fracture in 12 patients (7.3%). Inferior rectus muscle entrapment was seen in 1 patient with orbital floor fracture.

FIGURE 14: ORBITAL WALL MANIFESTATIONS

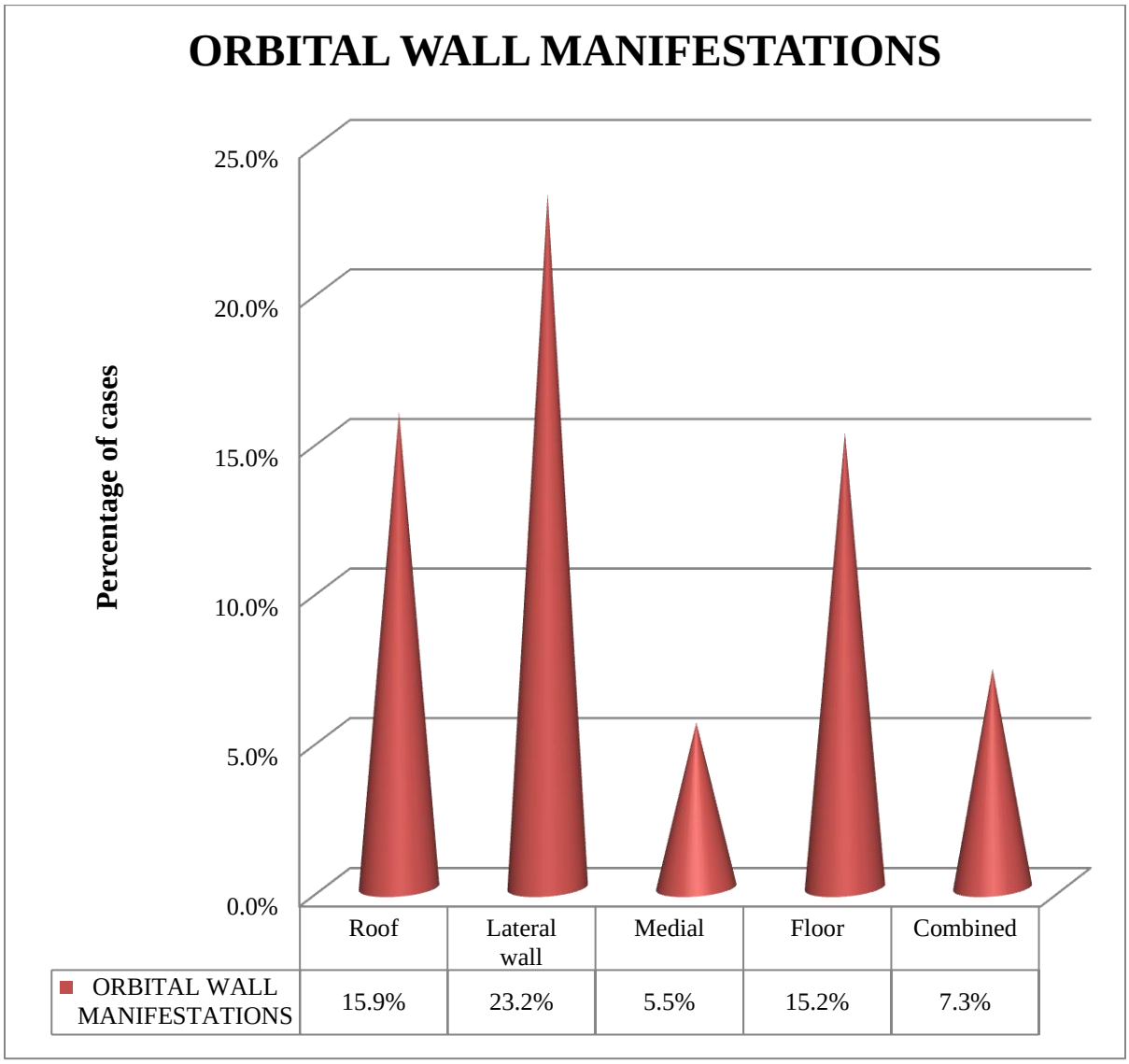


TABLE 16: TYPE OF INJURY

Type of injury	No of cases	Percentage (%)
Closed globe injury	160	97.6%
Open globe injury	4	2.4%

In this study, 4 patients had open globe injury which is due to corneal tear in 2 cases and corneoscleral tear in 2 cases.160 patients (97.6%) had closed globe injury.

FIGURE 15: TYPE OF INJURY

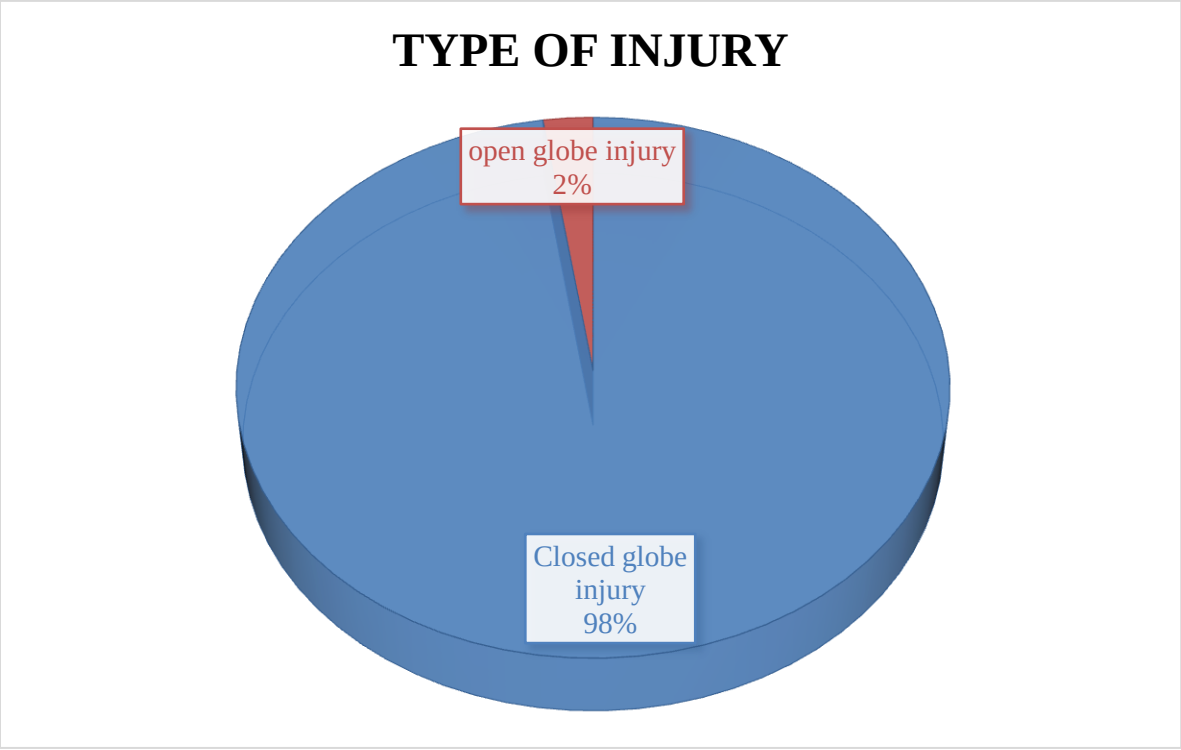


TABLE 17: NEUROIMAGING FINDINGS

Neuroimaging findings	No of cases	Percentage(%)
Brain Hemorrhage	41	25%
EDH	6	3.7%
SDH	14	8.5%
ICH	8	4.9%
SAH	13	7.9%
Contusion	3	1.8%
Pneumocephalus	5	3.0%
Diffuse Axonal injury	3	1.8%
Cerebral edema	4	2.4%
Isolated skull fracture	26	15.9%

In this study, 82 patients had CT scan findings. Epidural hematoma occurred in 6 patients (3.7%), followed by subdural hematoma in 14 cases (8.5%), intracerebral hemorrhage in 8 cases (4.9%) and subarachnoid hemorrhage in 13 patients (7.9%). Contusion was seen in 3 patients (1.8%), Pneumocephalus in 5 patients (3%), Diffuse

axonal injury in 3 patients (1.8%), Cerebral edema in 4 patients (2.4%), Isolated skull fracture occurred in 26 patients (15.9%).

FIGURE 17: NEUROIMAGING FINDINGS

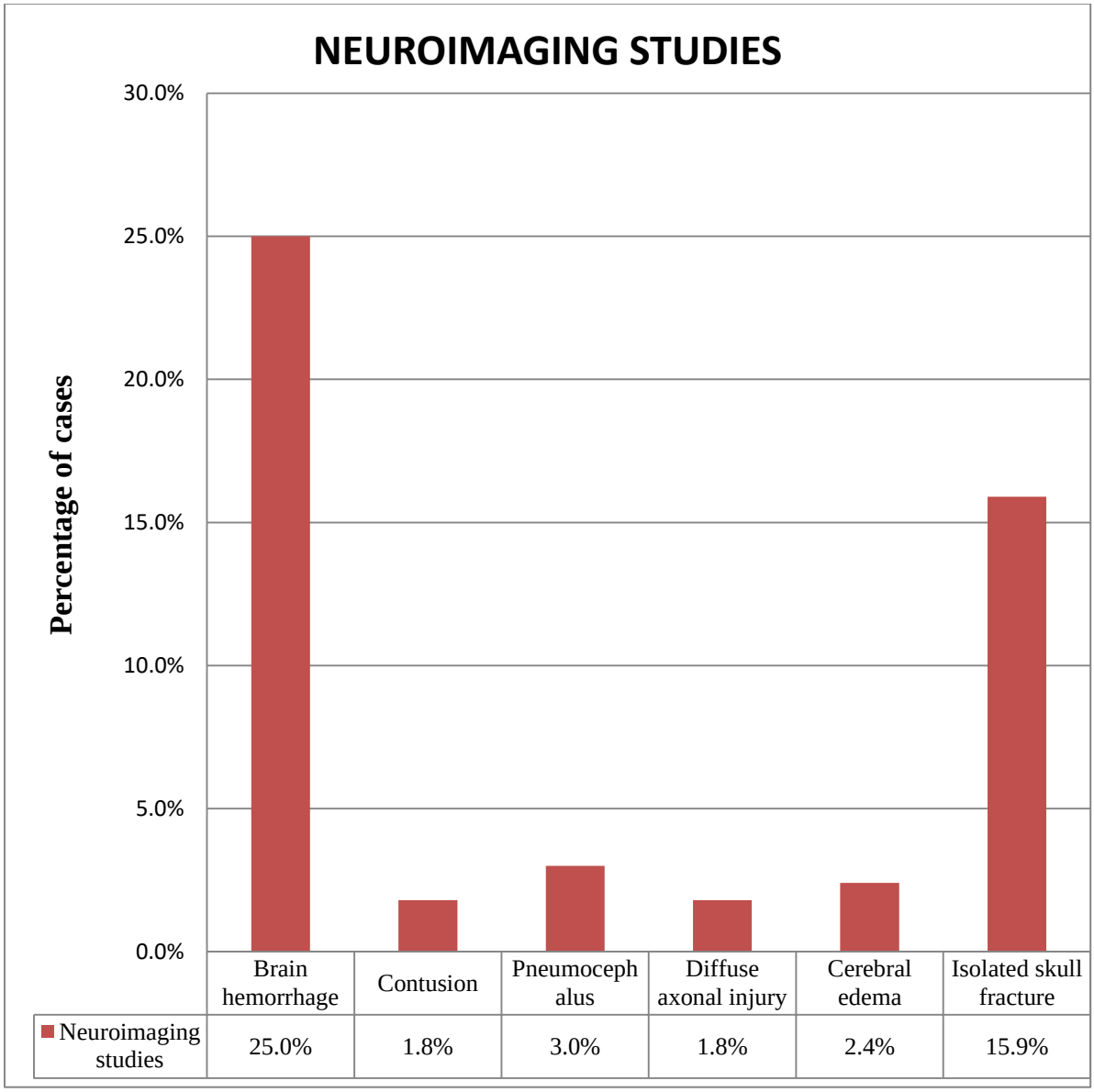


TABLE 18: VISION AT PRESENTATION

Vision at presentation	**No of cases**	**Percentage (%)**
NO PL	6	3.7%
PL	7	4.3%
HM	12	7.3%
CFCF	4	2.4%
6/60 – 1/60	26	15.9%
6/18-6/36	16	9.8%
6/9-6/12	20	12.2%
6/6	73	44.5%

Out of the 164 head injury patients, no perception of light was seen in 6 patients (3.7%), Perception of light in 7 patients (4.3%), Hand movements in 12 patients (7.3%), Counting finger close to face in 4 patients (2.4%), Vision of 6/60-1/60 in 26 cases (15.9%), visual acuity of 6/18-6/36 in 16 patients (9.8%), visual acuity of 6/9-6/12 in 20 patients (12.2%) and 73 patients had vision of 6/6 (44.5%).

FIGURE 17: VISION AT PRESENTATION

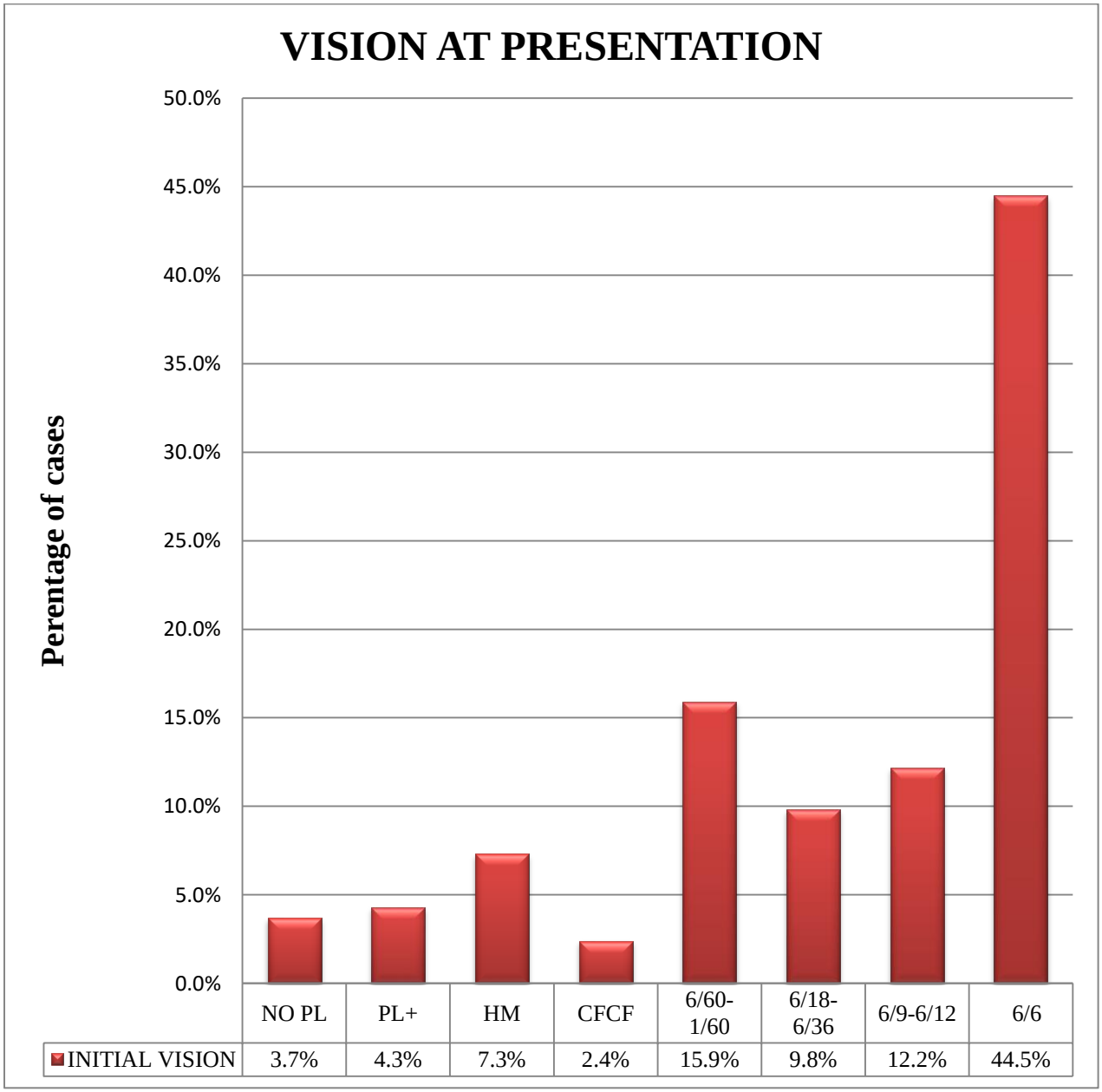
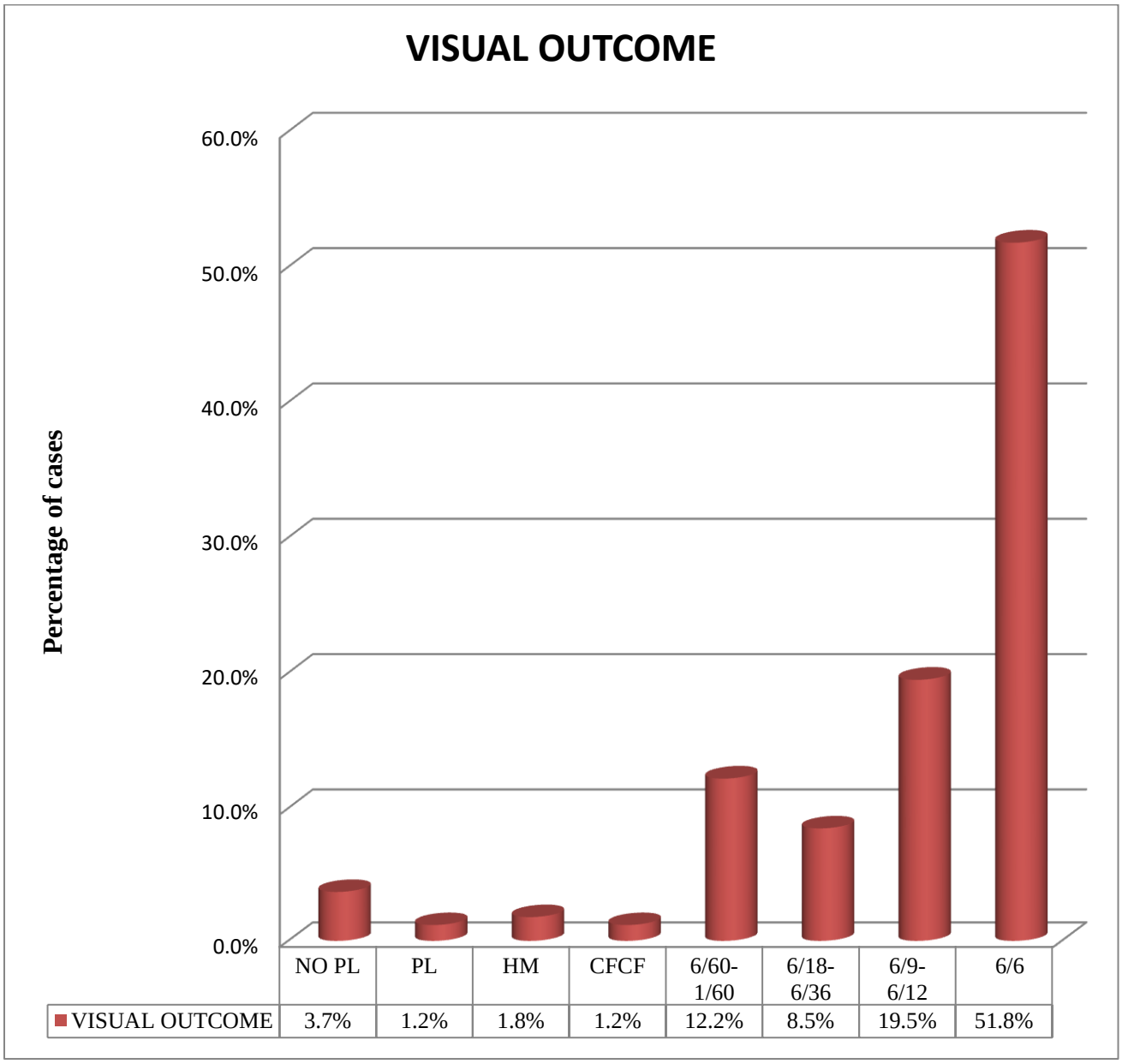


TABLE 18: VISUAL OUTCOME

Visual outcome	No of cases	Percentage (%)
NO PL	6	3.7%
PL	2	1.2%
HM	3	1.8%
CFCF	2	1.2%
6/60-1/60	20	12.2%
6/18-6/36	14	8.5%
6/9-6/12	32	19.5%
6/6	85	51.8%

Out of the 164 head injury patients, no perception of light was seen in 6 patients (3.7%), Perception of light in 2 patients (1.2%), Hand movements in 3 patients (1.8%), Counting finger close to face in 2 patients (1.2%), Vision of 6/60-1/60 in 20 cases (12.2%), visual acuity of 6/18-6/36 in 14 patients (8.5%), visual acuity of 6/9-6/12 in 32 patients (19.5%) and 85 patients had vision of 6/6 (51.8%).

FIGURE 18: VISUAL OUTCOME



DISCUSSION

DISCUSSION

In this study involving 164 head injury patients with ocular manifestation, a wide spectrum of serious ocular injuries were seen.

In our study most of the head injury patients with ocular manifestations were males, which is about 87.8% and 12.2% patients were females. In a study done by Sharma B et al (2014) 81.6% patients were males and 18.4% were females, with a male to female ratio of 4:1.(9) and in a study done by Anu Malik et al (2016), 91% patients were Males and only 9% of patients were females.(8)

In our study most of the patients were in the young adult male age group 21-40 years which is same as in the study conducted by Sharma B et al(2014).(9)

In our study right and left eye were involved in 25.6% and 69.5% cases respectively and bilateral ocular involvement was seen in 4.9% cases, whereas Hendge et al (2016) reported that right eye was involved in 35% head injury patients, left eye in 62% of patients, bilateral ocular involvement in 3% of patients.(10)

In our study Road traffic accident (75%) was the most common cause for head injury followed by assault (14.6%) whereas in the study done by Anu Malik et al (2016) 68.8% head injury cases were due to Road traffic accident and 18% of cases was due to assault.(8)

In this study periorbital ecchymosis was seen in 76.2% of head injury patients, in comparison to the study done by Masila Faith et al (2014), in which periorbital ecchymosis was seen in 36.1% of head injury patients. (11)

Periorbital edema was seen in 75.6% head injury patients in our study, whereas in the study done by Sharma B et al (2014), periorbital edema was seen in 41.48% head injury patients.(9)

In our study lid tear was present in 5.5% head injury patients, whereas in the study done by Masila Faith et al (2014), lid tear was seen in 8.9% of head injury patients.(9)

In our study, 57.9% patients had subconjunctival hemorrhage while in the study by Sharma B et al(2014), 44.44% patients were found to have subconjunctival hemorrhage.(9)

Corneal tear was seen in 2.4% of patients in this study, while in the study by Masila Faith et al (2014), corneal tear was present in 0.6% of patients.(11)

In this study Exposure keratitis was seen in 1.2% patients, but it is 4.21% cases in the study done by Sharma B et al(2014).(9)

3% patients had traumatic hyphema in our study, while in the study done by Anu Malik et al(2016), hyphema was present in 1.59% of patients.(8)

Traumatic cataract was seen in 1.2% of head injury patients in this study, which is similar (1.1%) to the study done by Masila Faith et al (2014).(11)

In our study, dislocation of lens was seen in 1.2% of head injury patients, while in the study done by Abha Gahlot et al (2015), lens dislocation was seen in 0.82% of patients.(12)

In our study vitreous hemorrhage was seen in 2.4% of head injury patients, while in the study by Masila Faith et al (2014) 1.1% patients had vitreous hemorrhage.(11)

12.8% of head injury patients had traumatic optic neuropathy in our study, while it was seen in 10.6% of patients in the study done by Masila Faith et al (2014).(11)

In this study orbital fractures were seen in 59.8% of patients, among which lateral wall was most commonly affected (23.2%). Sharma B et al (2015) reported that 54% patients had orbital wall fractures and among that lateral wall fracture is most common.(29%).(9)

In our study no perception of light were seen in 3.7 % of cases following treatment. El ShtewI et al reported that post treatment, 3.28% had no perception of light.(13)

SUMMARY

SUMMARY

164 head injury patients with ocular manifestations who presented to Thanjavur medical college were studied during a period of December 2017 to May 2019.

- The commonest age group involved were young adult males (21-30 years)
- Males have a significantly higher frequency of head injury with ocular manifestations when compared to females.
- Unilateral eye involvement is more common than bilateral involvement, of which is left eye was most commonly affected than the right eye
- Road traffic accident was the most common cause for head injury followed by assault.
- The most common ocular manifestation in head injury was seen in eyelids followed by conjunctiva.
- The most common ocular presentation in head injury was periorbital ecchymosis and periorbital edema.
- The most common conjunctival manifestation in head injury was subconjunctival hemorrhage
- The most common corneal manifestation in head injury was corneal edema
- The most common lenticular manifestation was subluxation of lens
- Posterior segment was involved in 20 patients and macular edema is the most common manifestation.
- The most common cranial nerve to be affected in head injury was optic nerve

- Among orbital wall, lateral wall fracture was more common.
- In head injury patients closed globe injury was more common than open globe injury
- No perception of light was seen in 6 head injury patients which was due to traumatic optic neuropathy.

CONCLUSION

CONCLUSION

The immense increase in problem of head injury in present era has necessitated this study aimed at analyzing cause, pattern and identification of clinical features. Head injury can result in varied ocular manifestations. Saving the life of the patient may seem to be the immediate goal, but saving the sight should also be an important target of treating surgeon.

Methods of prevention are

- The use of helmets, protective goggles and seat belts must be made compulsary
- Early diagnosis, referral and treatment are important factors associated with visual prognosis.
- The public must be made aware of dangers of driving under influence of alcohol.
- Along with public awareness stricter road traffic legislation is required.

RTA related trauma has significant morbidity associated with it and this morbidity can be drastically reduced if precautionary measures are taken such as wearing seat belts and helmets. They also reduce the possibility of head injury.

Ocular involvement in cases of head injury should also be carefully evaluated, which can go a long way in saving the life and sight of the patient.

PART III

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A CLINICAL STUDY OF OCULAR MANIFESTATIONS IN HEAD INJURY

PROFORMA

Name of the patient: **Age:** **sex: M / F** **Date:**

Hospital no.: **Address:** Rural / Urban

Occupation: Agriculture / Industrial worker / Driver / Officer / Students / Others

H/O Presenting illness:

Mode of injury: RTA (Pedestrian / 2 wheeler / 3 wheeler / 4 wheeler / others)

Assault

Accidental self fall

others

Date / time of injury:

Date first seen in Hospital:

H/O alcohol intake: victim / driver

H/O use of safety measures: Helmet / seat belt

H/O Head injury

H/O Loss of consciousness

H/O Ocular injury

H/O other injury

Ocular history:

H/O pain

H/O defective vision

H/O Photophobia / redness /watering

H/O Headache / vomiting

H/O Diplopia

H/O Floaters

Past History:

H/O Previous ocular injury

H/O Previous ocular surgery

H/O previous spectacle/contact lens use for refractive error

H/O Systemic medications

General examination:

Anemia: Pulse Rate:

Lymphadenopathy: Respiratory Rate:

GCS Score: Blood pressure:

RTS Score:

Type of ocular injury:

Systemic examination:

Cardiovascular system:

Respiratory system:

Abdominal examination:

Central nervous system:

Ocular examination:

	OD	**OS**
Vision-unaided		
Near vision		
Best spectacle-corrected visual acuity		
Colour vision		
Forehead/Adnexa		
Orbit		
Wall		
Margin		
Lid		
Position (upper/lower lid)		
Tear		
Edema		
Ecchymosis		
Others		
Canalicular injury		
Extra ocular movements		
Cornea		
Edema		
Keratic precipitates		
Tear		

- Ulcer
- Opacity
- Others

Anterior chamber

- Depth
- Cells
- Flare
- Hyphema
- Others

Pupil

- Size
- Shape
- Reaction to light
  - Direct
  - Consensual
- Others

Iris

Lens

Tonometry

Fundus

Gonioscopy (if needed)

Investigation:

Complete blood investigation:

Radiological investigation (X ray/CT scan/MRI):

Slit lamp examination:

B Scan:

Others:

Provisional diagnosis:

Treatment:

Medical:

1. Mydriatics/cycloplegics
2. Antibiotics
    - Topical
    - Systemic
3. Steroids
    - Topical
    - Systemic
4. Others

Surgical:

Visual outcome:

Prognosis:

Follow up:

KEY TO MASTER CHART

ABBREVIATION	EXPLANATION
S.NO	Serial number
MOI	Mode of injury
RTA	Road traffic accident
CONJ	Conjunctiva
AC	Anterior chamber
EOM	Extra ocular movement
RE	Right eye
LE	Left eye
PL	Perception of light
CFCF	Counting finger close to face
NO PL	No perception of light
E	Edema
EC	Ecchymosis
LAG	Lagophthalmos
CREP	Crepitus
SCH	Subconjunctival hemorrhage
CCC	Circumcorneal congestion
CONG	Congestion
CHEM	Chemosis

ABR	Abrasion
CT	Corneal tear
Ex.K	Exposure Keratitis
FB	Foreign body
HYP	Hyphema
VIT	Vitreous
C/F	Cells/Flare
IRR	Irregular
SHA	Shallow
DL	Dislocation of lens
SL	Subluxation of lens
TC	Traumatic cataract
NRTL	Not reacting to light
SRTL	Sluggishly reacting to light
RAPD	Relative afferent pupillary defect
IP	Iris prolapse
S.TEAR	Sphincter tear
ID	Iridodialysis
ME	Macular edema
BER.E	Berlin’s edema
VH	Vitreous hemorrhage

RH	Retinal hemorrhage
RT	Retinal tear
PR	Purtscher’s retinopathy
PE	Papilledema
IMC	Immature cataract
PCIOL	Pseudophakia
R	Restricted
T.O.N	Traumatic optic neuropathy
C.E	Cerebral edema
EDH	Epidural hematoma
SDH	Subdural hematoma
SAH	Subarachnoid hemorrhage
ICH	Intracerebral hemorrhage
DAI	Diffuse axonal injury
PNEUM	Pneumocephalus
#R	Orbital roof fracture
#L	Lateral wall fracture
#M	Medial wall fracture
#F	Orbital floor fracture
E/D/Ad-	Elevation,depression,adduction restricted
Ab-	Abduction restricted

In-	Intorsion restricted
#FR	Frontal bone fracture
#T	Temporal bone fracture
#P	Parietal bone fracture
#OCC	Occipital bone fracture

MASTER CHART

S.No	Name	Age	Sex	Occ	M.O.I	Eye	Vision	Lids	Conj	Cornea	sclera	AC	Iris/ pupil	Lens	Fundus	EOM	Orbit	Neuro imaging	Diagnosis	Final Vn
1	Babu	57	M	Agri	Assault	RE	HM	E+EC	SCH			VIT	SRTL	DL				C.E	Dislocation of lens	6/36
2	Azhaguraja	25	M	Agri	RTA	LE	PL+	E+EC	SCH				RAPD				#R	#FR	T.O.N	PL+
3	Thenmozhi	55	F	Agri	Self fall	LE	6/18		SCH					IMC					Subconj.Hemorrhage	6/18
4	Iyyapan	30	M	Agri	RTA	RE	6/6	E+EC											Edema+Ecchymosis	6/6
5	Arun	17	M	Student	RTA	LE	6/18	E+EC	SCH	E		C/F	SRTL				#L		Trumatic uveitis	6/9
6	Natarajan	43	M	Agri	Assault	LE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
7	Govindaraj	50	M	Agri	RTA	LE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
8	Vaithiyan	55	M	Agri	RTA	LE	6/6	E+EC	SCH										Sunconj.Hemorrhage	6/6
9	Badrinath	24	M	Student	RTA	RE	6/6	E+EC	SCH								#F,R		#Floor and roof	6/6
10	Prema	38	F	Agri	Assault	LE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
11	Braman	40	M	Driver	Assault	RE	6/6	E+EC	SCH										Subconj.Hemorrhage	6/6
12	Prem	24	M	Agri	RTA	RE	6/6	E+EC	SCH								#R	EDH	#Roof	6/6
13	Muthu kumar	22	M	Student	RTA	LE	PL-	E+EC					RAPD				#R		T.O.N	PL-
14	Anandham	40	M	Agri	Assault	RE	6/6	E+EC	SCH										Subconj.Hemorrhage	6/6
15	Kumar	42	M	Driver	RTA	LE	6/6	E+EC	SCH								#R		#Roof	6/6
16	Rajendran	37	M	Agri	RTA	LE	CFCF	E+EC	SCH				RAPD				#R	#T	T.O.N	1/60
17	Eswaran	7	M	Student	RTA	RE	6/6	E+EC	SCH								#F		#Floor	6/6
18	Vignesh	19	M	Student	RTA	LE	6/6	E+EC	SCH								#L,M		#Lateral and Medial	6/6
19	chandrasekar	32	M	Driver	RTA	LE	6/6	E+EC									#L	#T/ICH	Edema+Ecchymosis	6/6
20	Arun kumar	20	M	Student	RTA	LE	6/60	E+EC	SCH						BER.E			C.E	Berlin's edema	6/12
21	Gunasekaran	65	M	Agri	RTA	LE	PL+	E+EC	SCH				RAPD				#L		T.O.N	HM
22	Manikandan	11	M	Student	RTA	LE	6/6	E+EC	SCH									DAI	Edema+Ecchymosis	6/6
23	Senthil	50	M	Agri	RTA	LE	6/18	Lag		Ex.K								#T	VII N Palsy-LMN	6/18
24	Kaarthi	20	M	Student	RTA	LE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
25	Dinesh	18	M	Student	RTA	LE	6/9	Lid tear											Upper Lid tear	6/6
26	Manohar	25	M	Driver	RTA	RE	6/6	E+EC	SCH								#R		#Roof	6/6
27	Pughal	24	M	Officer	Assault	LE	PL+	E+EC	SCH				RAPD				#R	C.E	T.O.N	PL+
28	Uma	40	F	Agri	RTA	BE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
29	Inban	12	M	Student	RTA	LE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
30	Kannappan	26	M	Agri	RTA	L	6/6	E+EC	SCH								#R		#Roof	6/6

						E														
31	Sumathy	26	F	Officer	RTA	LE	6/12	E+EC	SCH						ME				Macular edema	6/9
32	Karupusamy	25	M	Agri	Assault	LE	6/6	Crep	SCH								#I		#Inferior wall	6/6
33	Vignesh	25	M	Student	RTA	LE	6/6	E+EC	SCH								#I,L		# Inferior/Lateral wall	6/6
34	Rajesh	24	M	Agri	RTA	LE	PL+	E+EC					RAPD				#I,M		T.O.N	1/60
35	Ayyapan	22	M	Agri	RTA	RE	6/18	Ptosis					NRTL			E/D/Ad-	#R	Pneum	III N Palsy	6/9
36	Murugesan	40	M	Agri	Assault	RE	HM	E+EC	SCH				ID		VH/RT		#L		Retinal Tear/VH	6/60
37	Chandraboss	25	M	Driver	RTA	LE	PL-	E+EC	SCH				RAPD				#L	#FR	T.O.N	PL-
38	Nelson	21	M	Student	RTA	LE	PL+	E+EC	SCH				RAPD				#R		T.O.N	CFCF

39	Rajasekar	55	M	Agri	RTA	LE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
40	kathar	38	M	Agri	Self fall	RE	6/9	Ptosis					NRTL			E/D/Ad-	#L	SAH	III nerve palsy	6/9
41	kalaiyarsan	20	M	Student	Self fall	LE	HM	E+EC	SCH	E		VIT	SRTL				#I		Subluxation of lens	6/36
42	Karthik	21	M	Driver	RTA	LE	6/9	E+EC								Ab-		#FR/EDH	VI N Palsy	6/6
43	Chinnaponnu	50	F	Agri	Self fall	RE	6/18	E+EC	SCH					IMC			#I		#Inferior wall	6/18
44	Thiyagarajan	26	M	Agri	RTA	RE	6/12	E+EC	SCH			C/F	SRTL					#F	Traumatic uveitis	6/6
45	Tamilarasan	26	M	Driver	RTA	BE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
46	Maran	24	M	Agri	RTA	RE	6/12	E+EC	SCH						ME			#T	Macular edema	6/9
47	Pandimuthu	48	M	Agri	RTA	RE	PL-	EC	SCH				RAPD				#R	#FR/SDH	T.O.N	P-
48	Vishwanathan	40	M	Agri	Self fall	LE	1/60	E+EC	Chem	CT	tear	IRR	IP					#P	corneoscleral tear	3/60
49	vannila	31	F	Agri	RTA	LE	6/6	E+EC	SCH								#I	SAH	#Inferior wall	6/6
50	Rajagopal	32	M	Driver	Assault	LE	6/6	Lid tear	SCH										Lower Lid tear	6/6
51	Girija	18	F	Student	Self fall	LE	6/6	Ptosis					NRTL			E/D/Ad-	#M		III nerve palsy	6/6
52	Palraj	42	M	Agri	Assault	RE	6/6	E+EC											Edema+Ecchymosis	6/6
53	Palanivel	62	M	Agri	RTA	LE	PL+	E+EC	SCH				RAPD				#I	SAH	T.O.N	HM
54	Chinnayan	25	M	Officer	RTA	RE	6/6	Ptosis					NRTL			E/D/Ad-			III nerve palsy	6/6
55	Ravikumar	26	M	Agri	RTA	BE	6/6	E+EC											Edema+Ecchymosis	6/6
56	Vaithiyanathan	38	M	Driver	Assault	LE	6/6	E+EC				C/F	SRTL				#L	#FR	Traumatic uveitis	6/6
57	Tamilarasu	27	M	Agri	RTA	LE	HM	E+EC	SCH				RAPD				#L	EDH	T.O.N	1/60
58	Satheesh	30	M	Officer	RTA	LE	6/60	E+EC	SCH						ME				Macular edema	6/9
59	Chinnaponnu	77	F	Agri	Self fall	RE	6/60	Ptosis					NRTL	IMC		E/D/Ad-	#L	SAH	III nerve palsy	6/60
60	Karunanithi	50	M	Agri	RTA	RE	6/6	Ptosis	SCH				NRTL			E/D/Ad-	#M,L	#OCC	III nerve palsy	6/6
61	Gomathi	42	F	Agri	RTA	LE	6/18		CCC			C/F	SRTL						Traumatic uveitis	6/6
62	Rajendhiran	50	M	Agri	RTA	RE	6/6	Lid tear	SCH										Lower Lid tear	6/6
63	Karthik	32	M	Officer	RTA	BE	6/6	E+EC	SCH								#R	#FR	#Roof	6/6
64	Saroja	60	F	Agri	Self fall	LE	6/9	Ptosis					NRTL	PCIOL		E/D/Ad-		FR -H	III nerve palsy	6/9
65	Palanisamy	30	M	Driver	RTA	RE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
66	Pandimuthu	48	M	Agri	RTA	RE	HM	E+EC	SCH				RAPD				#R	SDH	T.O.N	CFCF
67	Sudhakaran	34	M	Agri	Self fall	RE	6/24	E+EC					S.tear		ME			SAH	Macular edema	6/9
68	Arokiaraj	22	M	Student	RTA	LE	6/12	E+EC	SCH	Abr								#FR-T	Corneal abrasion	6/6
69	Palanivel	62	M	Agri	RTA	LE	HM	E+EC	SCH				RAPD				#F	SAH	T.O.N	HM
70	Bakkiyaraj	37	M	Agri	RTA	B	6/6	E+EC	SCH										Edema+Ecchymosis	6/6

						E														
71	Vignesh	37	M	Agri	RTA	RE	PL-	E+EC	SCH				RAPD				#L	FR-H	T.O.N	PL-
72	Mani	60	M	Agri	RTA	LE	6/18	E+EC	Chem	E							#R	SAH	Corneal edema	6/6
73	Velmurugan	40	M	Agri	Self fall	LE	6/12	Ptosis					NRTL			E/D/Ad-	#R	SDH	III nerve palsy	6/9
74	Udhaya	35	M	Officer	Self fall	LE	6/6	E+EC	SCH							In-	#L	SDH	IV nerve palsy	6/6
75	Madasamy	45	M	Agri	RTA	LE	6/6	Lid tear	Cong									P-ICH	Upper Lid tear	6/6
76	Arokiyasaamy	70	M	Agri	RTA	LE	6/12	E+EC	SCH					IMC			#M	ICH	# Medial wall	6/12
77	Azhagar	45	M	Driver	RTA	LE	HM	E+EC					SRTL	SL					Subluxation of lens	6/18
78	Ravichandran	50	M	Agri	Assault	LE	6/24	E+EC	CCC			C/F	SRTL						Trauamtic uveitis	6/9

79	Mathioli	27	M	Agri	RTA	RE	6/6	E+EC	SCH								#L	Pneum	#Lateral wall	6/6
80	Veerasaamy	26	M	Student	RTA	LE	1/60	E+EC	Chem	CT		SHA	SRTL					#FR	Corneal tear	6/60
81	Uthrapathy	66	M	Agri	Assault	RE	HM	Lid tear	Chem	E		HYP	NRTL		VH		#R		Vitreous hemorrhage	1/60
82	Senthil kumar	37	M	Agri	RTA	LE	6/6	E+EC	SCH							Ab-	#M,L,I		VI nerve palsy	6/6
83	Muthusamy	60	M	Agri	Self fall	LE	6/6	E+EC									#R	#FR	#Roof	6/6
84	Anandha kumar	35	M	Agri	Assault	LE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
85	Thirumuruga n	33	M	Officer	RTA	RE	2/60	E+EC	SCH					TC					Traumatic cataract	6/12
86	Manikandan	27	M	Student	RTA	LE	6/6	Lag										#T	LMN VII nerve palsy	6/6
87	Vinoth	28	M	Agri	RTA	LE	6/6	Ptosis	SCH				NRTL			E/D/Ad -	#F,R,L	#T	III nerve palsy	6/6
88	Mani	30	M	Agri	RTA	LE	PL-	E+EC	SCH				RAPD				#F		T.O.N	PL-
89	Murugan	27	M	Agri	Assault	BE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
90	Manoj	13	M	Student	RTA	LE	6/6									Ab-		Pneum	VI nerve palsy	6/6
91	Ambethkar	29	M	Agri	RTA	LE	6/60	E+EC							BER.E			SDH	Berlin's edema	6/12
92	Pushpam	62	F	Agri	RTA	LE	6/12	E+EC	CCC			C/F	SRTL					FR-T SDH	Traumatic uveitis	6/9
93	Rani	32	F	Agri	RTA	RE	6/18	Ptosis	Cong				NRTL			E/D/Ad -	#L	SG-H	III nerve palsy	6/9
94	Selva kumar	22	M	Agri	RTA	LE	6/60	E+EC	SCH						PR			FTP-SAH	Purtscher retinopathy	6/12
95	Padmashree	10	F	Student	Self fall	RE	6/6	E+EC					S.tear					#OCC	Sphincter tear	6/6
96	Baskar	26	M	Driver	RTA	LE	6/6	E+EC	SCH							In-	#R	T-P-ICH	IV nerve Palsy	6/6
97	Parvathi	29	F	Agri	RTA	LE	CFCF	E+EC	SCH				RAPD				#F	FR-Cont	T.O.N	5/60
98	Ashokan	25	M	Agri	Assault	LE	1/60	E+EC	SCH	E		HYP	SRTL				#L	TSAH	Traumatic Hyphema	6/12
99	Subramani	26	M	Agri	RTA	RE	1/60	E+EC		E		VIT	SRTL	SL				FR-ICH	Subluxation of lens	6/18
100	Saaminaatha n	61	M	Agri	RTA	LE	6/9	E+EC	SCH								#L	FR-T SDH	#Lateral wall	6/6
101	Gethiyan	21	M	Student	RTA	RE	6/6	E+EC	SCH								#I,L		#Inferior and Lateral	6/6
102	Raj kumar	40	M	Agri	RTA	LE	6/6	E+EC	SCH								#I		#Inferior wall	6/6
103	Kathirvel	75	M	Agri	Self fall	LE	6/60	E+EC	Chem	Ex.K				PCIOL				FR-T-P.SDH	Exposure keratitis	6/36
104	Saaminath	62	M	Agri	RTA	RE	6/12	E+EC	SCH			C/F	SRTL				#L		Traumatic uveitis	6/9
105	Jeyachandra	24	M	Driver	RTA	LE	6/6	Lid tear	Cong									#FR	Upper Lid tear	6/6
106	Thiru	48	M	Agri	RTA	RE	6/6		Tear								#I		Conjunctival tear	6/6
107	Mohan	27	M	Agri	RTA	LE	6/6	E+EC	SCH									DAI	Edema+Ecchymosis	6/6
108	Ramesh	32	M	Officer	RTA	LE	2/60	E+EC	SCH			C/F	SRTL		VH			FR-ICH	Vitreous hemorrhage	6/60
109	Nilavendhan	53	M	Agri	Assault	LE	6/60	E+EC	Cong						ME		#R		Macular edema	6/12
110	Kaviyarasan	20	M	Student	Assault	LE	6/6	E+EC	Cong	FB									Corneal Foreign Body	6/6

111	Thangayan	75	M	Agri	RTA	LE	PL+	E+EC	Chem				RAPD				#R	FR-SAH	T.O.N	4/60
112	Ramkumar	27	M	Driver	RTA	RE	6/9	Ptosis	SCH				NRTL			E/D/Ad -	#L	FR-Cont	III nerve palsy	6/6
113	Anandhan	24	M	Agri	RTA	LE	6/18	E+EC	Cong	Abr									Corneal abrasion	6/6
114	Naveen	29	M	Agri	RTA	LE	6/6	E+EC	Tear										Conjunctival tear	6/6
115	Velaiyaatham	46	M	Agri	RTA	LE	HM	E+EC	SCH				RAPD				#R,L		T.0.N	6/60
116	Sowmya	22	F	Agri	Assault	LE	6/9	Ptosis	SCH				NRTL			E/D/Ad -	#I	SDH	III nerve palsy	6/9
117	Kumaresan	28	M	Agri	RTA	LE	6/6	Crep	Chem								#M		#Medial wall	6/6

118	Rajagopal	45	M	Agri	RTA	LE	6/60	E+EC	SCH						RT			C.E	Retinal tear	6/18
119	Vasanth	16	M	Student	RTA	LE	1/60		Chem	CT	tear	SHA	IP				#R	FR-H	Corneoscleral tear/IP	4/60
120	Ziyavudeed	60	M	Agri	RTA	RE	HM	E+EC	SCH	E		VIT	SRTL	DL				EDH	Dislocation of lens	6/36
121	Kandhasamy	40	M	Agri	RTA	LE	6/18	Lid tear	Chem								#F	FR-T-SDH	Upper Lid tear	6/6
122	Naresh	27	M	Driver	Assault	RE	6/6	E+EC	SCH									SAH	Edema+Ecchymosis	6/6
123	Azhagaraajan	33	M	Agri	RTA	LE	6/6	E+EC	SCH				S.tear				#L	#T	#Lateral wall	6/6
124	Soundarajan	40	M	Agri	RTA	LE	3/60	E+EC	SCH	E		IRR	SRTL	SL			#L		Subluxated lens	6/60
125	Kumaravel	40	M	Agri	Assault	LE	6/6	E+EC	SCH								#L		#Lateral wall	6/6
126	Mani	72	M	Agri	RTA	LE	6/60	E+EC	Chem					IMC			#F		#Floor	6/60
127	Saravanan	27	M	Agri	RTA	LE	6/6	Lid tear	Cong									#FR	Lower Lid tear	6/6
128	Dhanasekar	42	M	Agri	RTA	LE	6/60	E+EC	CCC			C/F	SRTL		ME				Traumatic uveitis	6/18
129	Subramanian	50	M	Agri	RTA	LE	6/6	E+EC	SCH							Ab-	#L		VII N Palsy	6/6
130	Babulal	29	M	Agri	Assault	RE	HM	E+EC	CCC	CT		HYP	IP					Pneum	Corneal tear with IP	4/60
131	Hema	45	F	Agri	RTA	LE	6/6	Lag										#T	VII N Palsy	6/6
132	Sugan	19	M	Student	RTA	LE	6/9	E+EC	Chem							D-	#F,IRE		#F with IR entrapment	6/6
133	Palanivel	40	M	Agri	RTA	LE	6/60	E+EC	SCH						RH			#FR	Retinal hemorrhage	6/18
134	Rajan	30	M	Agri	RTA	LE	6/36	E+EC	SCH			C/F	SRTL						Traumatic uveitis	6/12
135	Sivanadhan	24	M	Agri	RTA	RE	PL-	E+EC	SCH				RAPD				#F,L,	FR-ICH	T.O.N	PL-
136	Thenmozhi	51	F	Agri	Self fall	LE	6/6	E+EC	Tear									Pneum	Conjunctival tear	6/6
137	Thiyagaraju	35	M	Driver	RTA	LE	6/6	Crep	SCH								#F,L,M		#Floor,Lateral,Medial	6/6
138	kannan	69	M	Agri	RTA	BE	6/12	E+EC	Chem					PCIOL	PE		#R	SAH	Papilledema	6/9
139	Julius cesar	75	M	Agri	Self fall	RE	6/18	E+EC	CCC	Abr				PCIOL				ICH	Corneal abrasion	6/9
140	Paneer	34	M	Agri	RTA	LE	6/6	E+EC	SCH							In-		EDH	IV nerve palsy	6/6
141	Srinivas	32	M	Agri	RTA	LE	HM	E+EC						TC					Traumatic cataract	6/12
142	Manohar	28	M	Officer	RTA	LE	6/6	E+EC	SCH										Edema+Ecchymosis	6/6
143	Nalini	34	F	Agri	RTA	LE	6/6	E+EC	SCH								#L	SDH	#Lateral wall	6/6
144	Prithiv	22	M	Student	RTA	RE	6/12	E+EC	CCC			C/F	SRTL						Traumatic uveitis	6/9
145	Raju	26	M	Agri	RTA	LE	6/60	E+EC					S.tear		ME				Macular edema	6/12
146	Latha	29	F	Agri	RTA	LE	6/6		Tear									#FR	Conjunctival tear	6/6
147	Sekar	47	M	Agri	RTA	LE	CFCF	E+EC	SCH				RAPD					DAI	T.O.N	6/60
148	Narayanan	31	M	Officer	Assault	LE	3/60	E+EC	SCH			HYP	ID				#I		Traumatic Hyphema	6/36
149	Bala	30	M	Agri	RTA	LE	6/6	Lid tear	Cong									FR-Cont	Lower lid tear	6/6

150	Paandi	28	M	Agri	RTA	LE	4/60								RH			FR-T-SDH	Retinal hemorrhage	6/60
151	John	32	M	Driver	RTA	LE	6/6		Chem								#I		#Inferior wall	6/6
152	Raghunaath	26	M	Agri	Assault	LE	6/36	E+EC		Abr									Corneal abrasion	6/9
153	Balaji	36	M	Agri	RTA	RE	6/12	Ptosis	SCH				NRTL			E/D/Ad-	#R,L	EDH	III nerve palsy	6/9
154	Harish	24	M	Student	RTA	LE	6/60	E+EC	Cong						BER.E			SAH	Berlin's edema	6/12
155	Jegan	39	M	Agri	RTA	LE	6/60	E+EC	Chem	Abr									Corneal abrasion	6/9
156	Balakrishna	26	M	Agri	RTA	LE	6/60	E+EC	SCH			HYP	ID					FR-T-SDH	Traumatic Hyphema	6/36
157	Selvam	54	M	Agri	Self fall	LE	6/6	Crep	SCH								#M		#Medial wall	6/6

158	Chitra	38	F	Agri	RTA	LE	6/36	E+EC	CCC			C/F	SRTL						Traumatic uveitis	6/9
159	Saminathan	36	M	Agri	RTA	RE	6/6	E+EC	Tear										Conjunctival tear	6/6
160	Kannaiyan	34	M	Driver	RTA	LE	6/9			FB								#FR	Corneal Foreign Body	6/6
161	Veeraiyan	42	M	Agri	RTA	LE	CF CF	E+EC	SCH				RAPD						T.O.N	2/60
162	Maanikam	55	M	Agri	RTA	LE	1/60		SCH				SRTL		VH			SDH	VH	6/60
163	Sundar	29	M	Agri	RTA	LE	6/6	E+EC	Chem								#L		#Lateral wall	6/6
164	Sasi kumar	25	M	Student	RTA	LE	6/6	E+EC	Tear										Conjunctival tear	6/6