“A STUDY TO CORRELATE OCULAR MANIFESTATIONS OF CLOSED HEAD INJURY WITH GLASGOW COMA SCALE AND VISION AND NEUROLOGICAL OUTCOME IN A RURAL TERTIARY CARE CENTER”

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DISSERTATION SUBMITTED TO

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MASTER OF SURGERY (M.S.)

IN

OPHTHALMOLOGY

Under the Guidance Of

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APRIL 2020

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I hereby declare that this dissertation entitled **"A STUDY TO CORRELATE OCULAR MANIFESTATIONS OF CLOSED HEAD INJURY WITH GLASGOW COMA SCALE AND VISION AND NEUROLOGICAL OUTCOME IN A RURAL TERTIARY CARE CENTER."** is a bonafide and genuine research work carried out by me under the direct guidance of **DR.SANDHYA R.**, Professor and Head of the Unit, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamaka, Kolar, in partial fulfilment of the requirement for the degree of **M.S. IN OPHTHALMOLOGY**. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

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LIST OF ABBREVATIONS

SL NO	ABBREVATIONS	FULL FORM
1	TBI	Traumatic Brain Injuries
2	GCS	Glasgow Coma Scale
3	RTA	Road Traffic Accidents
4	IOF	Inferior Orbital Fissure
5	SOF	Superior Orbital Fissure
6	CHI	Closed Head Injury
7	TON	Traumatic Optic Neuropathy
8	SCH	Sub-Conjunctival Haemorrhage
9	CF	Counting Fingers
10	VA	Visual Acuity
11	SICU	Surgical Intensive Care Unit
12	PICU	Paediatic Intensive Care Unit

ABSTRACT

"A STUDY TO CORRELATE OCULAR MANIFESTATIONS OF CLOSED HEAD INJURY WITH GLASGOW COMA SCALE AND VISION AND NEUROLOGICAL OUTCOME IN A RURAL TERTIARY CARE CENTER."

BACKGROUND:

Traumatic head injury is increasingly recognised as a major cause of morbidity and mortality worldwide.[1]

Head injuries are responsible for 50% of trauma deaths and 60% of road traffic accident deaths.[2]

Due to the close proximity of eyes to skull,Head injuries are frequently associated with ophthalmic manifestations and consequent morbidity.[3]

Ocular trauma is the cause of blindness in more than half a million people worldwide. Partial loss of sight is observed in many more. It is often the leading cause of uniocular loss of vision.[4]

Hence, the role of ocular injuries secondary to head injury as a cause of blindness and overall prognosis of patients has become a subject of immense importance.

Ocular manifestations and complications following head injury vary from time to time and patient to patient.

Clinical correlation of the ophthalmic findings with head injury is important in early localization of the site of injury for better management, and improved visual prognosis of the patient.[5]

In spite of the significant problems associated with ocular manifestations of head injury, only a few reviews of the whole spectrum are available in the literature.

The aim of this study is to evaluate and document various ocular manifestations in patients having closed head injury and correlate them with the patients' neurological status assessed by Glasgow Coma Scale (GCS) scoring and to compare any association between them.

OBJECTIVES:

- To evaluate and document various ocular manifestations in patients with closed head injury.
- To evaluate and document the neurological status by Glasgow Coma Scale (GSC) score at the time of presentation.
- To compare the association between them.

MATERIALS AND METHODS:

This prospective study was examined 85 patients of ocular trauma, reporting to the emergency department at R.L.J. HOSPITAL AND RESEARCH CENTRE,TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE between January 2018 and May 2019. Detailed history regarding injury was taken. Patients were examined at the time of presentation , at 1 week, at 4 weeks and at 6 weeks after the injury Consciousness status was assessed using Glasgow Coma Scale. Detailed Ophthalmic examination was done. And the neurological and visual outcome was evaluated and documented and the association, if any, between the two was analysed.

RESULTS:

Out of the total 85 cases, predominantly young male patients was the most affected group. Highest number of the patients showed periorbital oedema as the most common presentation. It was seen in 46 patients out of 85, i.e 54.11%, followed by eyelid lacerations, which was seen in 22 patients, i.e 25.88 %. Ecchymosis was seen in 18 patients i.e 21.17% .

SCH was seen in 15.29% patients , corneal tear was present in 7.05%. Pupillary abnormalities were present in 3.52 % and fundus findings were there in 4.70%. And eyelid abrasions were present in 1 patient. GCS score at presentation was normal for 56 patients, 3 patients had severe category of head injury and 6 patients had moderate head injury. At 1 week of follow up, out of 85 patients, follow up of 26 patients was lost. 3 patients died because of the head trauma complications.**,** 1 on day1 of admission, other 2 on day2. The most common ocular findings were periorbital oedema and ecchymosis, both seen in 10 patients,out of 56 patients ,with the frequency of 17.85 % each.GCS score of 55 patients out of 56 was 15.One patient

scored 12 on the GCS scale. At 4 weeks of follow up, 1 more patient died and the examination was done for 47 patients, among which only 1 patient had ecchymosis,while all others didn't have any ocular finding, with GCS score of 15. At 6 weeks, 45 patients were followed up, with no ocular findings and GCS score of 15.

At various follow up visits, ocular findings improved for the patients, as well as GCS score. 4 Patients with low GCS score at presentation, died. And we lost follow up for total 36 patients during the period of study.

CONCLUSION:

The correlation of the severity of head injury with the posterior segment and neuro-ophthalmic manifestations , showed that the more severe form of head injury is related with severe ocular findings and thus resulting in poorer neurological and visual outcomes.

Therefore, a detailed ocular assessment during the first presentation in all cases of head injury patients is mandatory as it helps in better management and the final outcome could be improved with better diagnosis and management.

Keywords- ocular manifestation, closed head injury, GCS score.

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INTRODUCTION

INTRODUCTION

Traumatic head injury is increasingly recognized as a major cause of morbidity and mortality worldwide.[1]

Industrialization and modern modes of transportation has amplified the frequency of accidents and consequently, trauma to the skull [2] .These injuries are responsible for 50% of deaths related to trauma and 60% of motorway traffic accident deaths.[3]

The propinquity of eyes to the brain , leads to the high frequency of association of skull injuries with findings of eye as well and consequent morbidity.[4]

Half a million or so people are blinded by ocular trauma, worldwide. Partial loss of sight is documented in many others. Mono-ocular blindness is also one of the consequence of trauma.[5]

Therefore, traumatic eye manifestations due to trauma to the head, play a major part in causation of blindness and as a prognostic factor for patients, has become a matter of immense importance.

Secondarily due to head trauma, eye findings and associated complications, may have varied presentations and may change within a short period of time.

Clinical correspondence of the findings of eye associated with head injury is vital in sooner localization of the site of injury for better management, and improved visual outcome of the patient.[6]

Despite of the significant problems coupling with ocular manifestations of head trauma , literature does not have enough data which shows the whole picture of the problem.

This study aims to evaluate and document various ocular manifestations in patients having closed head injury and correlate them with the patients' neurological status assessed by Glasgow Coma Scale (GCS) scoring and to compare any association between them.

OBJECTIVES

AIMS AND OBJECTIVES:

- To evaluate and document various ocular manifestations in patients with closed head injury.
- To evaluate and document the neurological status by Glasgow Coma Scale (GSC) score at the time of presentation.
- To compare the association between them.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The propinquity of the orbit to the brain, and the neuronal connections between the two may be accountable for skull injury (which has dura mater and skull intact), resulting in low vision.[7] The neurological outcomes of the head injury may be directly or indirectly contributing to the vision loss. Soft tissue incarceration in the orbit also may be to blame for the ocular findings like restricted movements. Thus, a wide range of ocular manifestations may be observed clinically in patients who have suffered with skull trauma. Increasing number of motor vehicle accidents in India in the recent past may be explained by the exponential progressing cosmopolitan standards, and this accounts for approximately ten percent of road traffic accidents (RTA) worldwide. High velocity impact because of RTA is the most accountable reason of head injury reported.[7,8] Universally, younger generation of male patients are the ones who are more prone to head injuries, according to a wide range of studies.[7,8,9]

According to a study[8] ,at a tertiary care hospital ,observations done on patients having closed head injury (where skull and dura mater remains intact),it was observed that 67.44% patients had visual complications. Among these, the most frequently occurred ocular finding was soft tissue injury to the globe and adnexa (48.83%). The probable mechanism leading to injuries in these cases is that, force is transmitted to the above mentioned parts from the boundries of the orbit laterally and from the frontal bones superiorly, during the shock of the trauma.[10,11] The most frequently recorded neuro-ophthalmic manifestation was papillary involvement(31.39%).[7]

Misalignment of the ocular posture are considered as a consequence of direct orbital trauma, usually involving the floor or the medial wall of the orbit, which results in soft tissues incarceration and entrapment of muscles.[7]

Studies have shown that there is a significant co-relation of the Glasgow Coma Scale (GCS), neuro-deficit and the ocular signs with the outcome of the injury.[8] Pupillary abnormalities, papilloedema and extra ocular muscle palsy pointed towards a poor neurological outcome.[8]

ANATOMY OF SKULL [12]

Skull- The skeleton, consists of large number of separate bones, which are united by sutures (fibrous immovable joints). It consists of 28 bones which are-

It can be divided into-

- Cranium
- Facial skeleton

Cranium- It is the uppermost part of the skull which contains the brain.

Calvaria- Composed of 14 bones including 3 paired ear ossicles.

Paired- Parietal, Temporal, Malleus,Stapes and Incus

Unpaired- Frontal, Occipital, Sphenoid and Ethmoid

Figure 1 shows the anterior view of the skull, consisting cranium and facial skeleton.

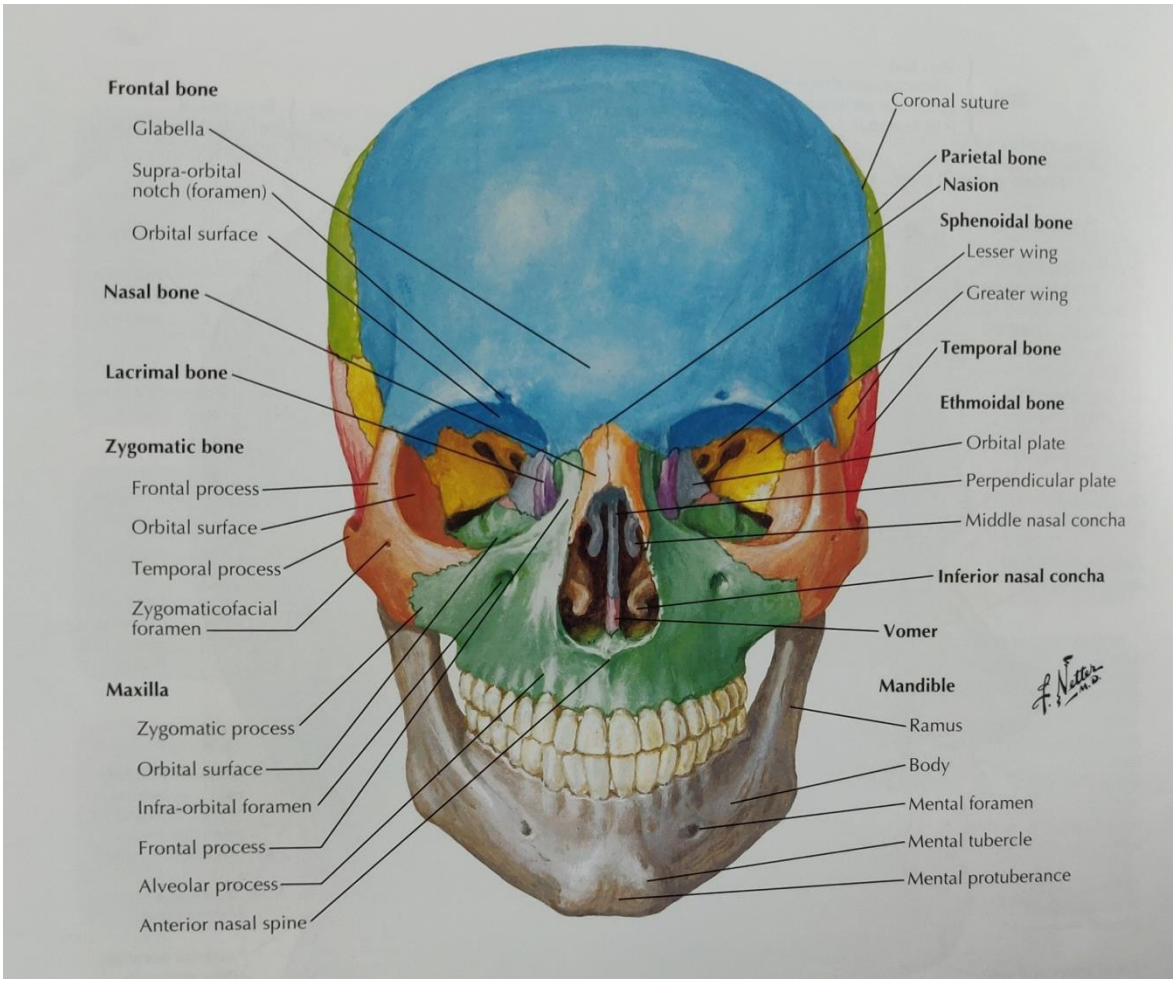

Figure 1.Anterior view of the skull.

The structure of the face is comprised of 14 bones-

Paired – Maxilla, Zygomatic, Nasal, Lacrimal, Palantine, Inferior nasal concha.

Unpaired- Mandible and Vomer

In the lateral view of the skull,as seen in figure 2, paired and unpaired bones of the skull can be seen.

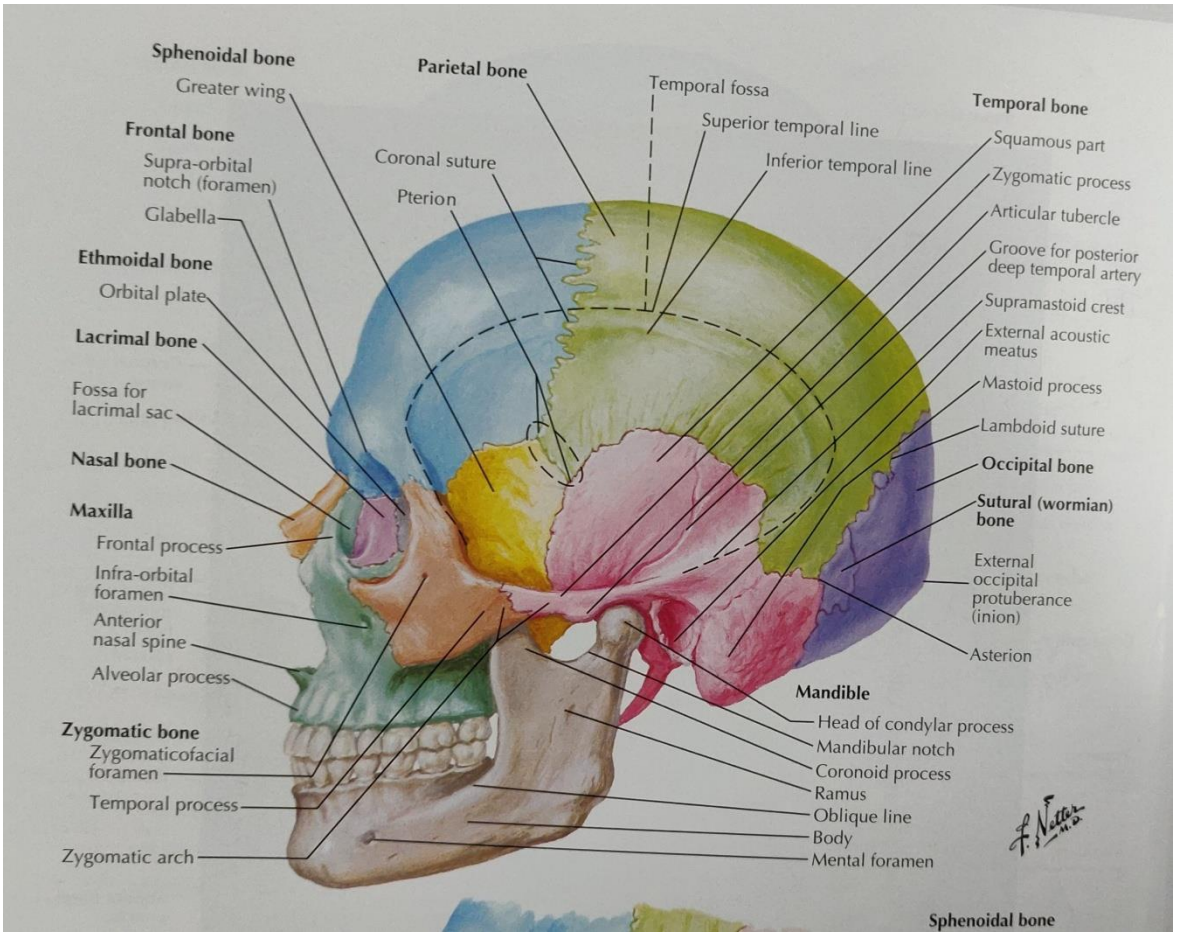

Figure 2. lateral view of the skull.

Parietal bones- Roof and the lateral walls of the skull is formed by them. Midline articulation of two parietal bones forms the sagittal suture. Posteriorly, lamboid suture is formed by joining with the occipital bone, and coronal suture, in the front, by attaching with frontal bone. As seen in the figure 3.

Occipital bone- Posterior part of the skull is formed by this bone. Interiorly,it forms the posterior cranial fossa, where the cerebellum, pons and medulla oblongata resides.

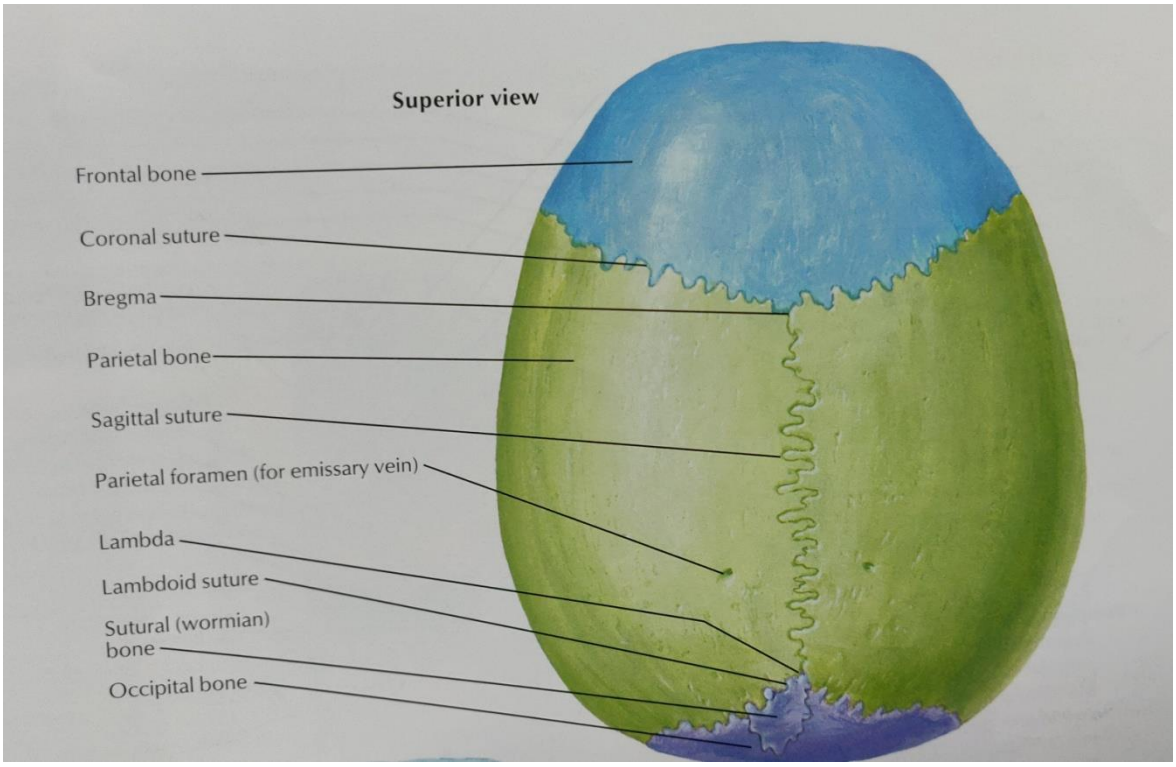

Figure 3. Various sutures of the skull are seen.

Temporal bones- Paired bones, each one contains two portions-

- Squamous- this part articulates with the sphenoid and the parietal bone. And it forms the lateral walls of the skull.
- Petrous- this is the part in which the middle and inner ear structures lie and it has extension in the cranium.

The two projections from the inferior aspect of the skull are the mastoid process and the styloid process. Stylomastoid foramen is located amid the two processes, which has exit for the seventh nerve (facial nerve).

Frontal bone- It is an unpaired bone forming the anterior portion of the skull, superior part of the facial structure, floor of the cranial cavity. Parietal bones are joined by the frontal bone at the top of the skull; it articulates inferiorly with the lacrimal, sphenoid and ethmoid bone; and infero-anteriorly with the nasal, maxillary and zygomatic bones. Anterior cranial fossa is fromed by the inner surface of the bone.

Sphenoid bone-Also an unpaired bone, base of the cranium is formed through sphenoid bone articulating with the occipital and the temporal bones. It articulates with the zygomatic bone, laterally. Sphenoid bone joins with the maxillary and palantine bones, anterio-inferiorly. And with the parietal bones superior-anteriorly and superiorly with the ethmoid and frontal bones. As depicted in figure 4.

The hypophyseal fossa, which is formed by the depression on the superior cranial surface of the body of the sphenoid bone, contains the pituitary gland. Pairs of lesser and greater wings project from the body of the sphenoid. Anterior portion of the sphenoid body projects the lesser wings which are attached to the body by two small roots, and this gap between the two roots forms the optic foramen, which forms the exit gate for the first cranial nerve (optic nerve). Sphenoid has two other projections from its body which are called greater wings, and they project from the lateral portion of its body.

Three important foramina are located

- Foramen rotundum- Maxillary nerve passes through it
- Foramen ovale – opening for Mandibular nerve
- Foramen spinosum – through this foramen Middle Meningeal artery exits

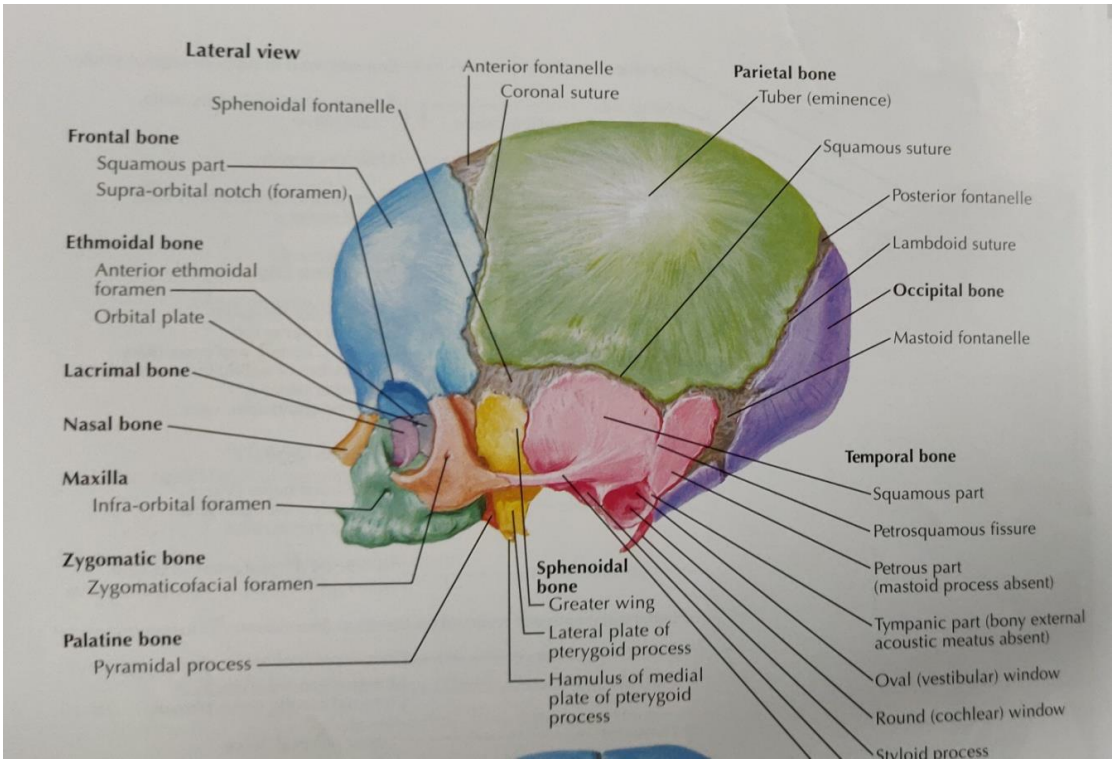

Figure 4. Lateral view depicting the parts of temporal and sphenoid bone

Ethmoid bone-

An unpaired bone, which looks like a rectangular box containing a midline straight plate, bisecting the top of the box, is the horizontal cribriform plate, which is perforated for the passage of the olfactory nerves. Orbital plates are the lateral walls of rectangular box parallel to the perpendicular plate which are separated from the perpendicular plate by the ethmoid air cells.

Facial skeleton- viscerocranium ,consists of 14 bones

Forehead is formed by the unpaired frontal bone. Its articulation with the nasal bones, the maxillae, and the zygomatic bones makes the face.

Maxillary bones- two maxillae form the upper jaw, the lateral walls of the nasal cavity, the hard palate and the orbital floors.

Nasal bones- the bridge of the nose is formed by the nasal bones by articulating with each other, with the frontal bone, and with the of the maxillary bones through their frontal processes.

Vomer bone - it is an unpaired bone forming bony nasal septum,basically the posterior portion.

Palatine bones - each is an L-shaped bone that extends from the hard palate at the back of the mouth to the orbit.

Lacrimal bone - smallest bone of the face.

Zygomatic bones- they form the sides of the cheek bones and zygomatic arches of facial skeleton by attaching with zygomatic process of the temporal bones.

Mandible- mobile lower jaw is formed by this bone.

Cavities of the skull

- Cranial cavity
- Nasal cavity
- Oral cavity
- Orbits

Cranial cavity- contains the brain and its meninges and their associated blood vessels with the intracranial portions of the cranial nerves.

Division of cranial cavity can be done as 3 fossae,which are -

- Anterior cranial fossa
- Middle cranial fossa
- Posterior cranial fossa

Anterior cranial fossa- anteriorly and laterally, it is formed by the frontal bone and posteriorly,by the lesser wing of the sphenoid. Orbital plate of the frontal bone,the cribriform plate of the ethmoid, lesser wings of the sphenoid and front portion of the body of the sphenoid, forms the floor of the anterior cranial fossa. Olfactory nerves pass through the perforations in the cribriform plate. Orbit is separated by the orbital plates of the frontal bones from the frontal lobes of the cerebral hemispheres above, whose sulci and gyri cause surface impressions on the bone.

Middle cranial fossa- It is at a lower plane compared to the anterior cranial fossa but is situated at a higher level compared to posterior cranial fossa. Its floor is shaped like a butterfly, it consists of a narrow central or median part and expanded lateral parts. Anteriorly,it is limited by the posterior free edge of the lesser wing of the sphenoid, the anterior clinoid processes and the front boundary of the sulcus chiasmatis.

Posteriorly,its extension is the superior borders of the petrous temporal bones and dorsum sellae of the sphenoid and laterally it is bounded by the squamous portion of the temporal bone, part of the parietal bones, and the greater wings of sphenoid. Pituitary fossa is situated in middle cranial fossa, there is an indentation in the roof of the body of the sphenoid bone which is bounded in front by the tuberculum sellae, which has posterior clinoid processes, in front of it.

The fossa is roofed by a sheet of dura mater, the diaphragm sella which is attached in front to the tuberculum and behind to the dorsum sellae. There is a small opening in the roof, through which pituitary stalk transverses . Laterally,lie the two cavernous sinuses.

Posterior cranial fossa –

This is the deepest of the three cranial fossae, its floor lying below the level of the middle fossa. Tentorium cerebella forms its roof.It lodges the hindbrain ,the cerebellum , pons, and medulla oblongata.

Bounded anteriorly by the superior borders of the temporal bone, mainly its petrous portion and the dorsum sella,and the foramen magnum is surrounded by it.

All the three fossae and the bones forming them are shown in figure 5.

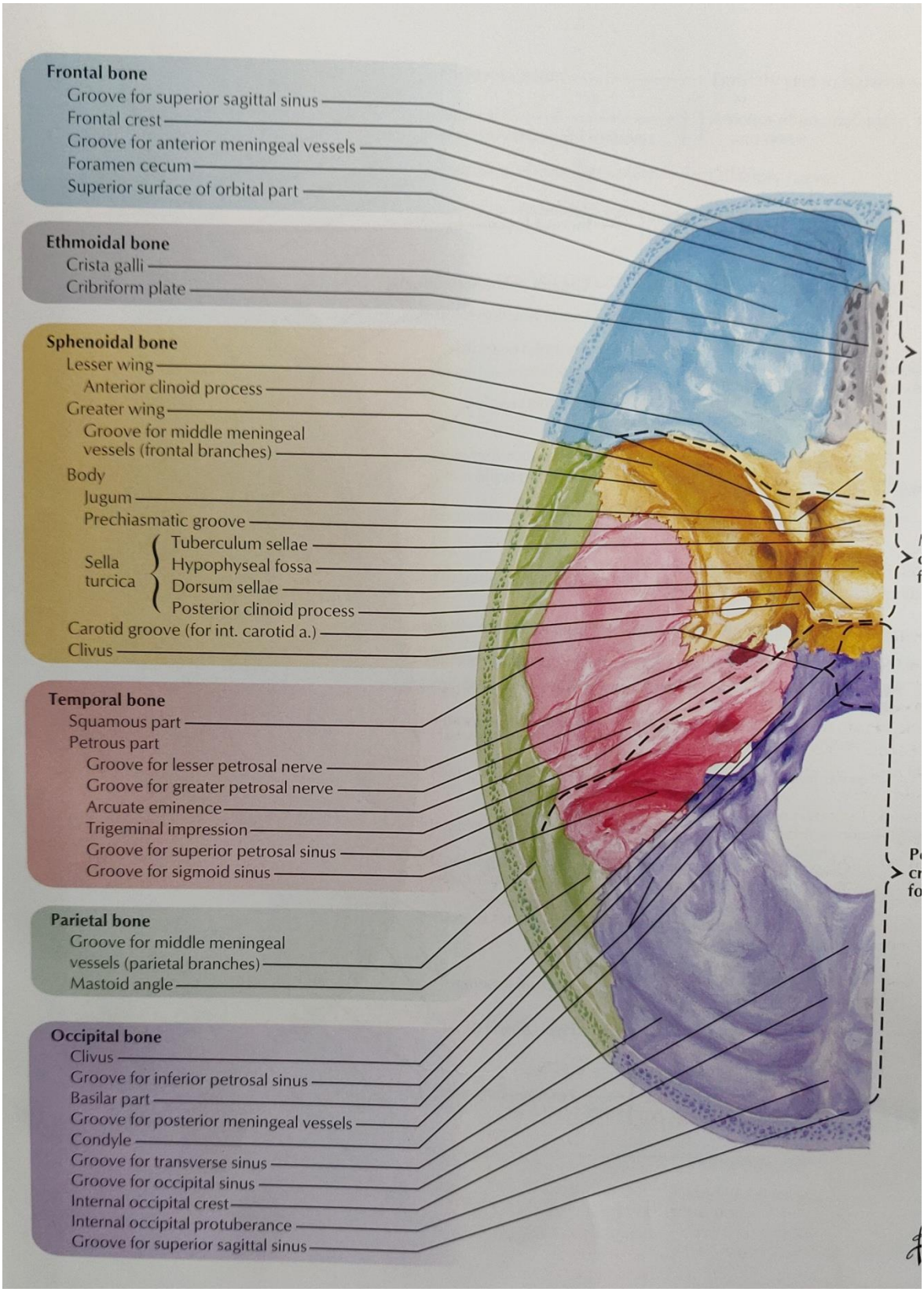

Figure 5. showing the base of the cranium

Anatomy of Orbit- [13]

- Bony orbit is formed from the mesenchyme that encircles the optic vesicle beginning as early as the 6-week of embryonic stage.
- Each orbit is formed by seven bones- Frontal,Ethmoid, Lacrimal,Palatine,Maxilla,Zygomatic and Sphenoid as seen in figure 6.
- The two bony orbits are shaped like a quadrangular truncated pyramids which lie between anterior cranial fossa above and the maxillary sinuses below.
- Medial walls are parallel to each other.
- Lateral wall of each orbit lies at an angle of 45º to the medial wall. And both orbital lateral walls are at 90º to each other.
- Depth is about 42 mm along the medial wall and about 50 mm laterally.
- Intraorbital width-25mm
- Extraorbital width-100mm
- Volume of each orbit- 29-30ml, and the ratio between the orbital volume and of the eyeball is 4.5:1

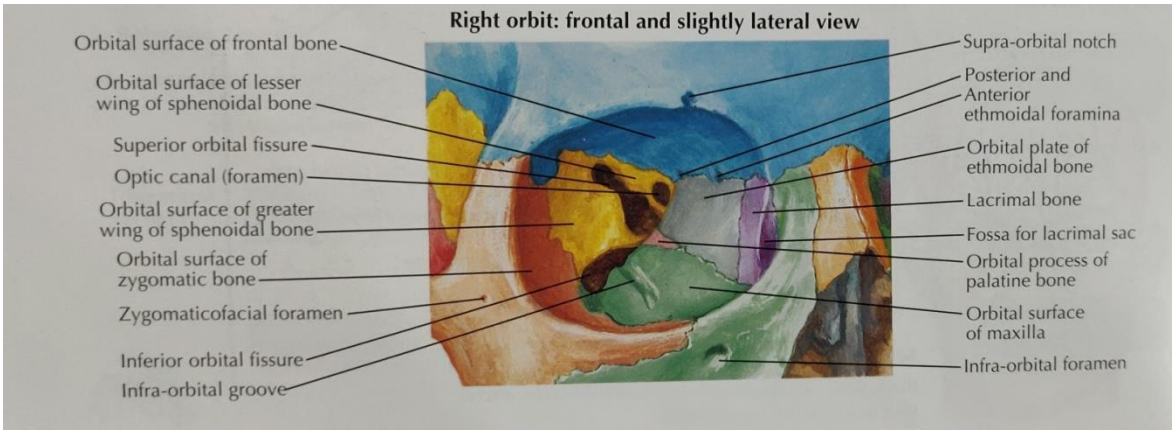

Figure 6. frontal view of the orbit is shown

Walls of the orbit

Medial wall- It is also called lamina papyracea as it is the thinnest among all the walls. It has quadrilateral shape and is formed by the frontal process of the maxilla, the lacrimal bone,the orbital plate of the ethmoid bone and the sphenoid bone's body. Front portion of the medial wall bears the lacrimal sac fossa-bounded anteriorly by the anterior lacrimal crest of the maxillary bone and posteriorly by the posterior lacrimal crest of the lacrimal bone. Medial to the lacrimal fossa lie the anterior ethmoidal sinuses in the upper part and middle meatus of the nose in the lower part. Just behind the posterior lacrimal crest,following structures have their attachments-

- Horner's muscle
- Septum orbitale
- Check ligament of medial rectus muscle

Medial to medial wall- lie anterior ehtmoidal air sinuses,middle meatus of nose,middle and posterior ethmoidal sinuses and sphenoidal air sinus.

Medial wall of orbit has orbital surface which is related to superior oblique muscle in the upper part near the roof and medial rectus muscle in the middle part. In between two muscles ,lie the anterior ethmoidal nerve,posterior ethmoidal nerve, infratrochlear nerve and terminal branch of the ophthalmic artery.

Inferior orbital wall (floor)- It is triangular in shape, and is the shortest wall of the orbit.

Consists of these bones - orbital surface of the maxillary bone, medially, the orbital surface of the zygomatic bone laterally and the palatine bone posteriorly.It slopes downward approximately 20ºfrom posterior to anterior.Inferior orbital fissure is situated in between the posterior part of the floor of the orbit and the lateral wall of

the orbit. Posteriorly ,the floor extends upto the posterior limit of maxillary sinus,therefore,does not extend to the orbital apex. Inferior wall relations-

Below,maxillary air sinus and palantine air cells.

Above,inferior rectus muscle,inferior oblique muscle and nerve to inferior oblique.

Lateral wall- It is the thickest and strongest,triangular in shape, it consists of two bones, anteriorly zygomatic bone and greater wing of sphenoid,posteriorly. Posterior part has a small bony projection-spina recti lateralis, which gives origin to a part of lateral rectus muscle. Anteriorly, on the wall ,a projection is situated, known as , the lateral orbital tubercle of Whitnall, which gives attachment for the check ligament of the rectus muscle, suspensory ligament of the eyeball, lateral palpebral ligament and aponeurosis of levetor muscle. Posteriorly ,it is separated from the roof by the superior orbital fissure and by the inferior orbital fissure, from the orbital floor.Separation of the orbit from temporal fossa anteriorly is done by the orbital lateral wall and it also seperates orbit from the middle cranial fossa, posteriorly. Medial associations of the wall are with the lacrimal nerve, lateral rectus muscle, and vessels, zygomatic nerve and the communication between zygomatic and lacrimal nerves.

Roof- orbital plate of the frontal bone forms the roof of the orbit,which is triangular in shape. Formation of the roof at apex is done by lesser wing of sphenoid, posteriorly. Roof has a depression called the fossa for the lacrimal gland, in the anterolateral part. Fovea for the pulley of the superior oblique (trochlear fossa) is a small depression situated close to the orbital margin. Roof slopes backward and downward towards the apex,where it ends at the optic canal and superior orbital fissure. Superior relations are with the frontal lobe of cerebrum and meninges. Below the roof are

periorbita,frontal nerve,levetor palpebrae superioris,superior rectus,superior oblique,trochlear nerve and lacrimal gland.

Base –It is defined as the anterior opening of the orbit. It is bounded by the orbital margins.

Superior orbital margin- lateral 2/3 portion is sharp and medial 1/3 portion is rounded. This arch is created by the orbital portion of the frontal bone. Where the two parts,there lies the supraorbital notch,which transmits the supraorbital nerve and artery. About 10mm medial to the notch,lies the supratrochlear groove,transmitting supratrochlear nerve and artery.

Lateral orbital margin- formed by zygomatic process of the frontal bone and the zygoatic bone. And is the strongest portion of the orbit.

Inferior orbital margin- created by the zygomatic bone laterally and maxilla, medially.

Medial orbital margin- below, it is fashioned by the anterior lacrimal crest on the frontal process of maxilla and above by the frontal bone.

Orbital apex is the posterior end of the orbit, where the four orbital walls converge.

It consists two openings -i) The optic canal, ii) The superior orbital fissure. At the orbital apex, inferior orbital fissure joins the superior orbital fissure,just below the optic canal and is continous with the foramen rotundum.

Between the roof and the lateral wall, there is superior orbital fissure (SOF), which separates the two pairs of wings of the sphenoid bone ,and is closed by the frontal bone, laterally. Wider at the medial end below the optic foramen, it is usually described as comma shaped. Sometimes,it tapers regularly towards its lateral extremity,but usually it shows a narrow lateral and a wider medial part,at the junction of which is the spine for the lateral rectus. The SOF is about 22mm long and is the largest communication between the orbit and middle cranial fossa. The common

tendinous ring of the rectus muscles spans the SOF between its medial and lateral parts, as seen in figure 7.

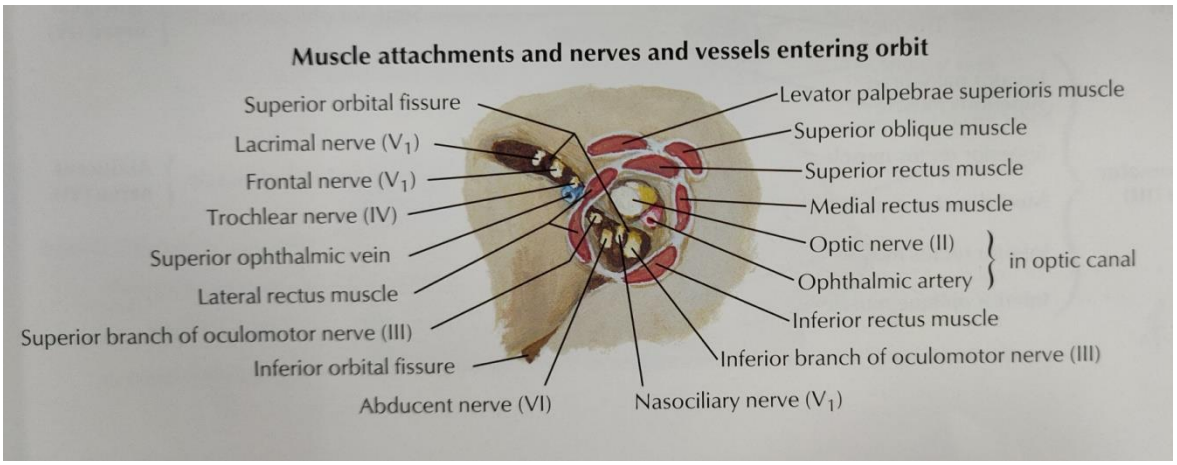

Figure 7. superior orbital fissure

The inferior orbital fissure (IOF)-

It is the structure between the orbital floor and its lateral wall. It joins the orbit to the pterygopalantine and infratemporal fossae. It extends inferolateral from theforamena of optic nerve ,at the medial side of the superior orbital fissure. Anteriorly, its boundary is the maxilla and the orbital processes of the palantine bone, and the entire lower margin of the sphenoid, posteriorly.

The IOF transmits the infraorbital and zygomatic nerves, orbital periosteal branches from the pterygopalantine ganglion ,and a branch from the inferior ophthalmic vein to the pterygoid plexus. And is close to the foramen rotundum and the sphenopalantine foramen.

The optic foramen- it is the structure created by the two roots of the lesser wing of the sphenoid bone and is the connection between the middle cranial fossa and the orbital apex. The passage is directed anterolaterally and slightly downwards,at an angle of about 36 degrees.

Orbital fascia- intraorbital structures are lined by the thin connective tissue membrane which is called orbital fascia.

Surgical spaces in the orbit-

Subperiosteal space- the space in between the orbital bones and its periorbita, limited anteriorly by the strong adhesions of periorbita to the orbital rim.

Peripheral orbital space (anterior space)- bounded peripherally by periorbita,internally by the four extraocular muscles with their intermuscular septa and aneriorly by the septum orbitale. Posteriorly it merges with the central space. Contents are peripheral orbital fat,superior oblique,inferior oblique and levetor palpebrae superioris muscles;lacrimal, frontal,trochlear,anterior ethmoidal and posterior ethmoidal nerves;superior and inferior ophthalmic veins;lacrimal gland ;and half of the lacrimal sac.

Central space- it also known as the muscular cone or posterior or retrobulbar space. It is bounded anteriorly by tenon's capsule lining the posterior part of the eye and peripherally by the extraocular rectus muscles and their intermuscular septa.

In the posterior part,where intermuscular septa are imperceptible,this space becomes continuous with the peripheral orbital space. Contents include, optic nerve and its meninges,superior and inferior divisions of oculomotor nerve,abducent nerve,nasociliary nerve,ciliary ganglion, ophthalmic artery ,superior ophthalmic vein and the central orbital fat.

Sub- Tenon's space- potential space around the eyeball between the sclera and Tenon's capsule.

Traumatic brain injury (TBI)-

Also known as intracranial injury,occurs when an external force traumatically injures the brain. Various classifications are based on severity ,anatomical features of the injury and the mechanism.[14] Mechanism – related classification divides TBI into closed and penetration head injury [15]. A closed injury, also called non-penetrating or blunt[16] occurs when the brain is not exposed[17.] There are three categories in which head injuries can be categorised and there can be some overlapping in which more than one may be present in a patient[18]. These are :

Diffuse- the brain has been shaken

Blunt- a direct non-penetrating blow

Penetrating – the cranium has been breached

The most frequently occurring type of traumatic brain injury (TBI) is the closed head injury(CHI) which is a significant cause of morbidity and mortality,worldwide.

The outcome of the CHI is affected by the two important factors,which are-

The initial impact causes the primary injury,defined as the immediate injury to neurons from transmission of the force of impact.

The long delicate axons of the neurons can shear as they undergo differential acceleration or deceleration along their projecting pathways.

Prevention strategies,such as wearing helmets,remain the best means to decrease disability from primary injury.

Secondary damage is considered as the subsequent neuronal damage caused due to the sequelae of the trauma.

Hypoxia, hypotension, hydrocephalus, intracranial hypertension, thrombosis, and intracranial hemorrhage may all be the mechanisms of seconday injury.

A specific check should be made for any loss of consciousness at the time of trauma and its duration.

The Glasgow Coma Scale (GCS) score and reaction of the pupil at the accident location and on the arrival to the hospital triage area should be evaluated and documented. They should be checked regularly thereafter. The deterioration in the GCS score is an important index of developing,secondary injury. the extent of amnesia, retrograde or anterograde,can be assessed by GCS score. Classification of the head injury severity is done according to the GCS score.

Glassgow coma scale [19,20,21]- it was introduced in 1974 as a method for determining sincerely the severity of brain dysfunction and coma six hours after the occurrence of head trauma. Nowadays, it is by far the most widely used score to assess the rigourness of head trauma. Ongoing impaired conscious level, beyond isolated confusion and amnesia implies a moderate injury and coma suggests a severe injury.

Initial assessment- The first three elements of the ABCDs of resuscitation- airway, breathing, and circulation Hypoxia and hypotension are considered as the negative prognostic factors which worsen outcome in TBI. Motor activity, speech and eye opening can be assessed by a GCS score. In all three categories, the best responses of the patient are recorded and documented, and the addition by the scores is done, and the final score is determined.

Eye opening	Verbral	Motor
Spontaneously - 4 To verbal command- 3 To painful stimulus -2 Do not open-1	Normal oriented conversion-5 Confused- 4 Inappropriate words- 3 Sounds only- 2 No sounds- 1 Intubated- T	Obeys command-6 Localizes to pain-5 Flexion-4 Abnormal flexion-3 Extension- 2 No response-1

The total GCS score ranges from 3-15.

14-15- mild Head injury

9-13- moderate Head injury

3-8- severe Head injury

The presence or absence of gross visual acuity ,may be prognostic when it is interpreted in the context of radiologic and neuro-ophthalmic findings.[22,23]Localization of the injury and the prognosis of the patient with head injury can be done by evaluating the ocular signs in the patient. Direct ocular trauma can also lead to the visual loss in head injury patients.

Various forms of ocular findings in closed head injury [24]–

The pathogenesis of ocular damage by blunt trauma is seen in figure 8.

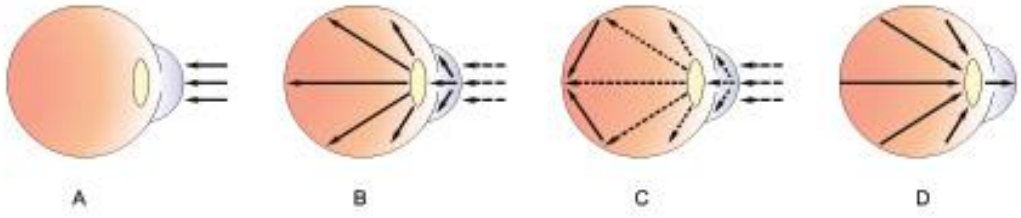

Figure 8.

Mechanics of blunt trauma to eyeball:
A, direct impact; B, compression wave force; C, reflected compression wave; D, rebound compression wave

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1. Eye Lid trauma- leads to 'black eye', consisting of a haematoma(focal collection of blood) and/or periocular ecchymosis(diffuse bruising) and oedema is the most common manifestations of the blunt injury to the eyelid. Figure 9 shows the various manifestations of eye lid trauma.

a)Edema and Haemorrhage- Laxity of structure and paucity of the subcutaneous fat permits an almost instantaneous swelling due to edema and hemorrhage which it creates enough pressure to cause pain. Extravasation of blood in the subcutaneous tissue leads to black eye.

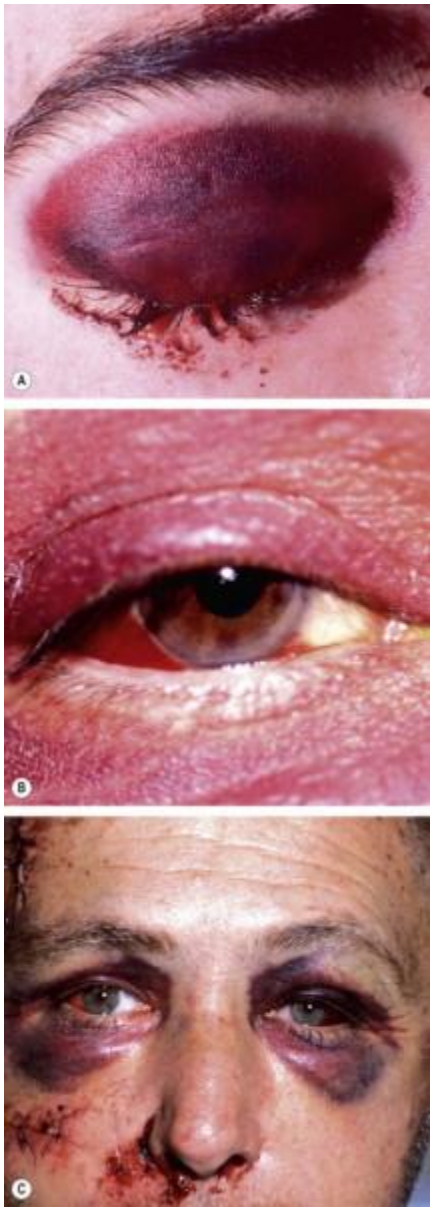


Figure .9 A) Periocular haematoma and oedema; (B) Periocular haematoma and subconjunctival haemorrhage; (C) 'panda eyes'

b) Crush wounds- Blunt force striking the orbital margins compress the tissue between the impinging object and the bone, due to which the tissue is stretched, ruptured or torn.

c) Laceration- the existence of a lid laceration,mandates careful exploration of the wound and assessment of the globe and adnexal structures. They can be-

- Superficial lacerations.
- Lid margin lacerations
- Lacerations with mild tissue loss
- Lacerations with extensive tissue loss

2.Injuries to the lacrimal system- The most at risk structure of the lacrimal system is canaliculus. Blunt force applied medially to laterally can tear the lid at the medial canthus.

3.Injuries to conjunctiva-

a)Sub conjunctival haemorrhage -it appears as a bright red patch of conjunctival tissue with distinct or feathered borders. Generally resolves spontaneously in 7-10 days, its color evolves from bright red to yellow green. Occassionally, when the hemorrhage involves the perilimbal conjunctiva, greenish discoloration in the anterior peripheral corneal stroma can be seen, which comes as the blood products breakdown.

b).Chemosis- as the outcome of increased orbital pressure from contusion,the accumulation of transudate in the subconjunctival tissue occurs. If it is severe, the conjunctiva may become elevated and prolapsed through the palpebral fissure.

c). Laceration –following direct impact thin conjunctiva tends to give away.

4.Lesions of sclera- Scleral rupture occurs as a result of the direct impact on the globe by a slowly moving blunt force which is adequately powerful. There are two types of contusion ruptures; direct and indirect. On the anterior part of the sclera,the direct ruptures are found and are more common and tend to occur when the sclera is thin. When the force hits the eye with sufficient intensity the site of impact is suddenly compressed and ruptures.

5. Corneal injury-

a)Abrasion- involves a breach of the epithelium, and stains well with fluorescein.

b)Corneal tears- complete or partial due to elasticity of Bowman's membrane.

c)Acute corneal edema- due to traumatic dysfunction of endothelial cells.

d)Corneal rupture- since the corneoscleral junction is weak, gives way producing a rupture at that site.

e) Blood staining of cornea- massive hyphema and raised IOP can cause blood staining of entire cornea,where the periphery is not stained due to entry of blood into corneal stroma. It gives a shape of a ring.

6. Hyphaema- blunt trauma can lead to hyphaema commonly. The source of bleeding is typically the iris root or ciliary body face.[25]

7. Pupillary changes- evaluating and documenting the reaction of the pupil in head injury cases is one of the most important step for the assessment.[26,27] the iris may momentarily be compressed against the anterior surface of the lens by severe anteroposterior force,with resultant imprinting of pigment from the pupillary margin.

a)Traumatic miosis- This condition is due to spasm of the pupillary sphincter muscle. It accompanies the compression, as evident by the outline of pigment, Vossius ring. The differential of this condition in a comatose patient after head trauma includes narcotic ingestion, parasympathomimetic ingestion or topical use and pontine haemorrhage.

b)Traumatic mydriasis- occurs from anatomical injury to the iris, mostly occurring as tears in the iris sphincter. Differentiation of traumatic mydriasis from a pupillomotor defect and pharmacological blockade is possible with pharmacologic testing. A mydriatic pupil caused by iris sphincter tears fails to constrict after instillation of 1% pilocarpine.

c) Rupture of pupillary margin

d)Radiating tears in the iris

e)Iridodialysis

Figure 10. Iris complications of blunt trauma(A) Vossius ring;**(B)** radial sphincter tears; **(C)**iridodialysis

f)Antiflexion of the iris

g)Retroflexion of the iris

h)Traumatic aniridia or iridemia

i)Angle recession

j)Horner's syndrome- the clinical signs of the syndrome include anhidrosis, relative miosis, pupillary dilatation lag,and narrowing of the palpebral fissure caused by upper lid ptosis, and higher than normal lower eyelid position due to reverse ptosis.[28]

Traumatic Horner's syndrome may be due to the damage to the preganglionic or postganglionic neurons.

k)Pupil involving oculomotor nerve paresis- traumatic injury of the oculomotor nerve may result in pupillary mydriasis and the loss of the accommodation as a result of interruption of parasympathetic innervation to the pupillary sphincter and the ciliary body.

l)Hutchinson's pupil- this ominous sign of a unilateral,fixed,dilated pupil is pathognomic of ipsilateral uncal herniation and cerebral edema and often precedes brainstem death. Evaluation of this sign is of great importance and relevance in a neurosurgery and polytrauma patients. It can due to kinking of the third cranial nerve across a displaced posterior cerebral artery because of any abnormal lesion and subsequent compression against the hiatus of the tentorium and the dural roof of the cavernous sinus.[29]

It has four stages, stage 1- ipsilateral miosis, stage 2- dilatation of ipsilateral pupil with contralateral miosis, stage3- incremental dilatation of the same sided pupil with contralateral miosis, stage 4- bilateral pupillary dilatation.[30]

8.Lens –

a)Vossius ring – it's a circular ring of brown pigment seen on the anterior capsule,due to striking of the contracted pupillary margin against the crystalline lens.

b)Concussion cataract- due to imbibition of aqueous and partly due to direct mechanical effects of the injury on lens fibres.

c)Traumatic absorption of the lens

d)Subluxation of the lens

e)Dislocation of the lens

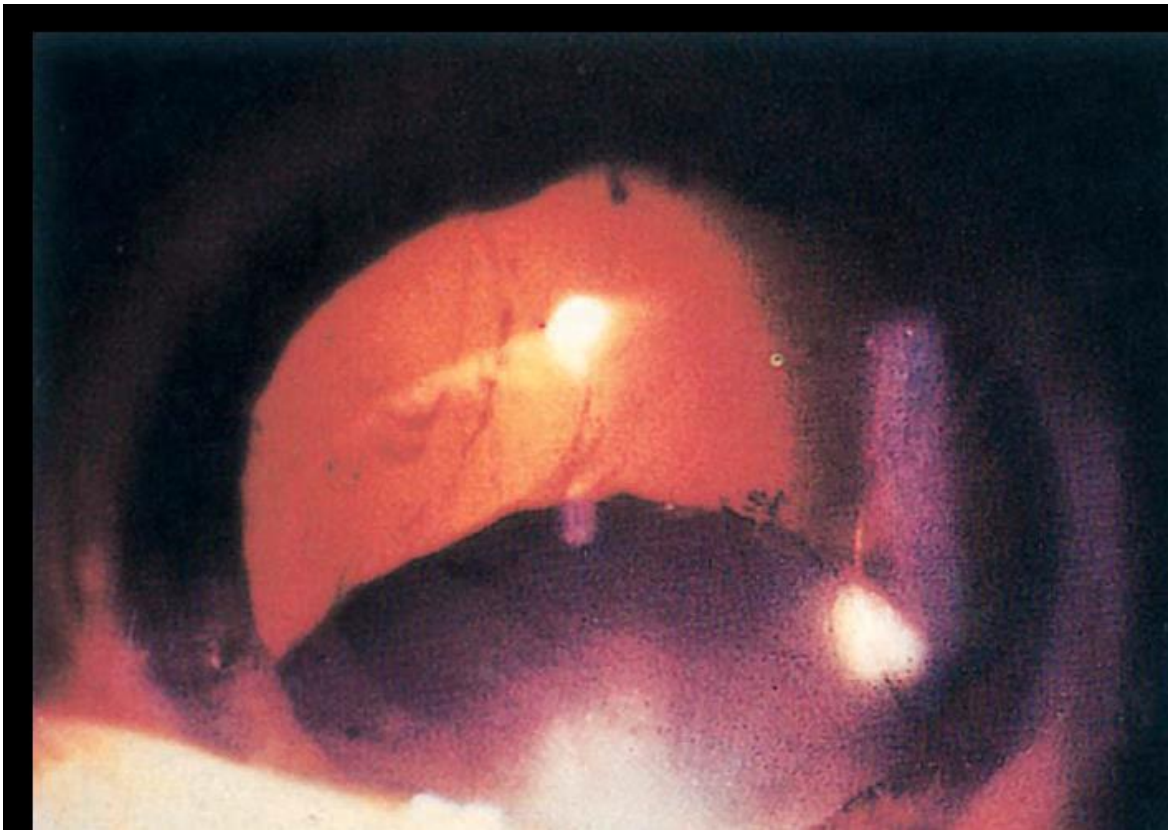


Figure 11. traumatic subluxation of the lens.

9.Vitreous -

a)Liquefaction

b)Detachment

c)Vitreous haemorrhage- pigment cells,tobacco dust, may be seen floating in the anterior vitreous.

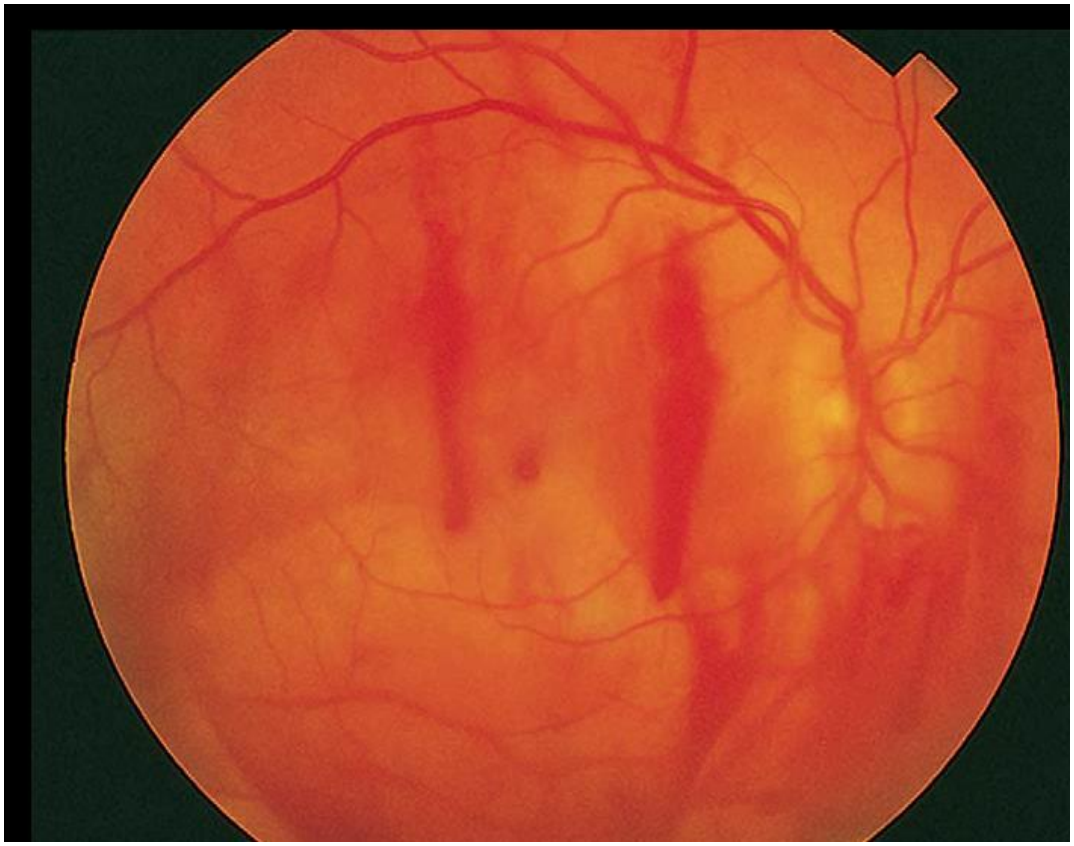


figure 12. traumatic vitreous haemorrhage

d)Vitreous herniation

10.Choroid –

a)Rupture of the choroid- involves the choroid,Bruch membrane and retinal pigment epithelium. Direct ruptures are more common at the site,which is anteriorly to the location of impact and run parallel with the ora serrata. Indirect ruptures occur to the reversed side of the site of impact.

b)Choroidal haemorrhage

c)Choroidal detachment

d)Traumatic choroiditis

11. Retinal manifestations – trauma is responsible for about 10% of all cases of retinal detachment.

a)Commotio retinae (Berlin's oedema)- Following a blow on the eye, it is one of the common manifestations , seen in figure 9. It manifests as milky white cloudiness involving a considerable region of the posterior pole with a 'cherry red spot'in the foveal region. The outer segments of the photoreceptors are the main location of retinal damage.[31]

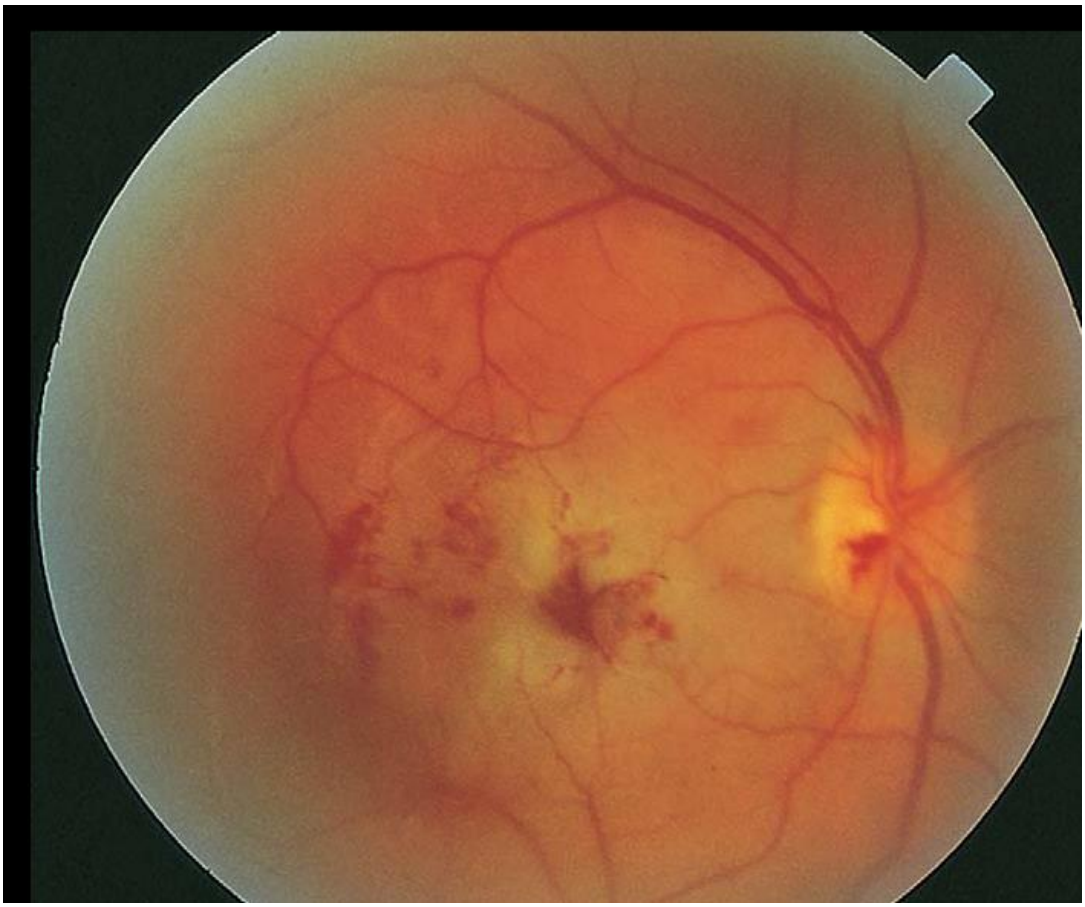


Figure 13.Shows the Berlin's oedema.

b)Retinal dialysis- is a break occurring at the ora serrata,caused by traction from the relatively inelastic vitreous gel.

c)Traumatic retinal breaks- occur when the vitreous is violently shifted away fron the retina.Direct or coup-type forces applied to the retina can result in retinal necrosis and atrophy. There is usually accompanying choroidal rupture, with subretinal and intaretinal hemorrhages.

d)Equatorial breaks and macular holes

e)Papilloedema- it is defined as a swelling of the optic nerve head, secondary to an increased intracranial pressure. Optic nerve comprises axons of the retinal ganglion cells that pass from the eye and are surrounded by the CSF and the meninges ,which forms the optic nerve sheath. The subarachnoid space around the optic nerve is continuous with the subarachnoid space around the brain. This, therefore,is susceptible to fluctuations of the CSF pressure that occur around the brain. Normally the intracranial pressure is less than intraocular pressure,but when this reverses the axoplasmic flow is disturbed and stasis occurs. When there is an abnormal autoregulation in the pressure gradient,papilloedema occurs. The most frequently occurring warning signs of raised intra cranial tension is Papilloedema.[32]

f)Traumatic optic neuropathy-It follows ocular,orbital or head trauma as sudden visual loss that can not be explained by other ocular pathology.Optic nerve distress is frequently connected with severe head trauma and multisystem imjury. Traumatic optic neuropathy (TON) occurs in most of the cases of motor vehicle accidents, which produce high energy deceleration type head trauma.[33,34] Patients with unilateral traumatic neuropathy demonstrate a relative afferent pupillary defect and decreased vision.

g) Injury of the optic nerve- direct or indirect mechanisms can lead to optic nerve injury. Direct trauma to the optic nerve may cause hemorrhage within the sheath or may produce traumatic arachnoid cysts.[35,36] Indirect,in which force is transmitted secondarily to the nerve without apparent direct disruption due to impacts upon the eye,orbit or other cranial structures. Due to blunt trauma, with high momentum, to the forehead, the orbital and intracranial contents shift, thus applying tractional forces on the tightly bound intracanalicular part.[37,38] This may lead to both shearing of axons

and blockage of the blood supply of the optic nerve.Indirect neuropathy is more common than direct.

h)Optic nerve avulsion- rare, typically occurs when an object intrudes between the globe and the orbital wall.

i)Purtscher's retinopathy[39]- Indirect ocular injury can result from compression injury to the chest, head or lower limbs. This is a retinal vascular injury with damage to the endothelium with intravascular clotting and occlusion of peripapillary arterioles by fibrin clot,fat or air emboli and platelet-WBC. Patchy whitening of the inner retinal layers with retinal hemorrhages and macular sparing in seen during the first 24 hours after trauma.

Figure 14. Purtscher's retinopathy

j) Terson's syndrome- this syndrome refers to the occurrence of vitreous haemorrhage in addition with sub-arachnoid hemorrhage[40] It is observed in many cases with post traumatic and spontaneous subarachnoid hemorrhage. A sudden increase in intracranial hemorrhage may lead to a sudden increase in retinal venous pressure with resulting venous bleeding into the retina and vitreous.[41,42] Hemorrhages are seen around the optic disc and are usually below the internal limiting membrane.

Figure 15. Terson's syndrome

12.Cranial nerve injury-

a) Oculomotor nerve- As extremely severe head trauma is present in patients who have associated oculomotor nerve paresis, they may also have reduced level of consciousness. Patients may have diplopia[43]. Infarction, contusion and odema of brain stem due to the injury may cause the third nerve nucleus damage.[44]

b)Trochlear nerve[45,46,47]- This is the smallest cranial nerve and has the longest course of approximately 75mm. only nerve that exits dorsally from the brainstem. In adults the most frequently occurring reason of trochlear nerve paresis is trauma. With blunt head trauma, rapid deceleration causes dislocation of the brain and brain stem within the cranium.

c)Abducens nerve[48,49,50]- although unilateral cases are more common, bilateral cases are also seen in association with head trauma. Trauma causes rapid displacement of the brain within the cranium, inducing shearing forces on the nerve. Patients present with horizontal diplopia.

d)Facial nerve – In cases with closed head injury , skull base fracture is one of the main cause of facial nerve paresis.[51] if the seventh cranial nerve palsy is delayed by 2-6 days ,then there may be a possible mechanism of denervation.[51] Paresis of the orbicularis oculi muscle which is the protractor of the eyelids causes incomplete eyelid closure or lagophthalmos.

Kulakarni et al[7] saw 200 successive patients of closed head injury admitted to a hospital.the major reason of head injury were motor vehicle accidents 52.5%. Which was second by assaults 34%. Eye was affected in 167 cases. These included corneal and sclera tears in 2%,subconjunctival haemorrhage or ecchymosis in 46%,orbital fractures 12%,pupillary involvement 6.5%, papilloedema 5.5%,intraocular trauma

5.5%,proptosis 3%,lateral rectus palsy 2%,lacrimal gland prolapse 1% and injury to first cranial nerve 0.5%. Early assessment in correlation with the GCS aids in prognosticating outcomes. Pupillary involvement, papilloedema and ocular motor paresis pointed to a more serious traumatic head injury.

Odebode et al[6] did a prospective study containing a total of 225 patients who had head injuries. Soft tissue damage to the globe and adnexae consisted of periorbital ecchymosis,subconjunctival haemorrhage, lid laceration and seldom ruptured globe in twenty- nine patients. Neuro-ophthalmic findings as cranial nerves of eye, paresis or paralysis was present in twenty- eight patients while walls of orbit fratures were documented intwo cases. Most frequently occurring ocular compilications following skull trauma were the damage to the lids and adnexa and structural damage to the cranial nerves related to eye, leading to their paresis or paralysis .

According to Rao KVM et al[8] and observations made by them, it was inferred that there were total number of 108 patients of head injury were examined of which 38 patients had ocular manifestations (35.18%). Of these, 85.18% were males, 84% of injuries were due to road traffic accidents and 16% were due to fall from a height. The ocular manifestations were as follows- 6 patients (15.8%) were affected by complications of the orbit secondary to the injury. Anterior segment manifestations incorporated black eyes as observed in ten patients (26.3%), subconjunctival haemorrhage was recorded in ten point five percent patients (4 patients), corneal involvement was seen in twenty one percent patients (8 patients) and atypical pupil reaction in fifty percent patients (19 patients). Posterior segment was involved in 26.3% of patients (10 patients) and the findings observed and recorded were -

Purtscher's retinopathy in two patients and atrophy of optic nerve in five patients. Cranial nerve palsies were seen in fifteen patients (39.47%) and supranuclear movement disorders were seen in three patients (8%).

METHODOLOGY

MATERIALS AND METHODS

SOURCE OF DATA:

Total of 85 patients of ocular trauma, reporting to the emergency department at R.L.J. HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE between January 2018 and May 2019,were included in the study.

STUDY DESIGN: Prospective study.

INCLUSION CRITERA:

1. All patients reporting to the emergency department with closed head injury.

EXCLUSION CRITERIA:

1. Previous history of any neurological disease.

2. Previous history of ocular trauma

METHOD OF COLLECTION OF DATA

The study comprised the analysis of 85 patients who were diagnosed of closed head injury. Detailed history regarding injury was documented . Patients were examined at the time of presentation , at 1 week, at 4 weeks and at 6 weeks after the injury and the neurological and visual outcome was evaluated and documented and the association, if any, between the two was analysed.

Consciousness status was assessed using Glasgow Coma Scale.

Ophthalmic examination included assessment of external injury, anterior segment examination, pupillary reaction, visual acuity, assessment of extra-ocular movements, visual field assessment by finger confrontation, fundus examination by direct ophthalmoscopy.

If necessary, assessment of visual acuity using Snellen's chart, assessment of intra ocular pressure using Goldmann applanation tonometer, and diplopia charting was done.

Sample size calculation:

Sample size was estimated based on the study done in India, Ocular manifestations in head injury patients- A prospective study. [8]

Sample size was estimated by using the following formula:

$n=Z^2_{1-\alpha/2}p(1-p)/d^2$

where ,

p : expected proportion

d :absolute precision

1-α/2 : desired confidence interval

At 10% absolute error and 95% Confidence interval, sample size

of 85 was obtained.

85 cases of closed head injury were assessed in this study.

STATISTICAL ANALYSIS:

Collected data was documented on an excel spread sheet and will be analyzed using SPSS 22 version software. All quantitative measures were presented with Mean Standard Deviation ,Confidence Interval and qualitative data was presented by percentage. Chi-square test was the test of significance to compare between the sub groups. P value ≤ 0.05 was considered as statistically significant.

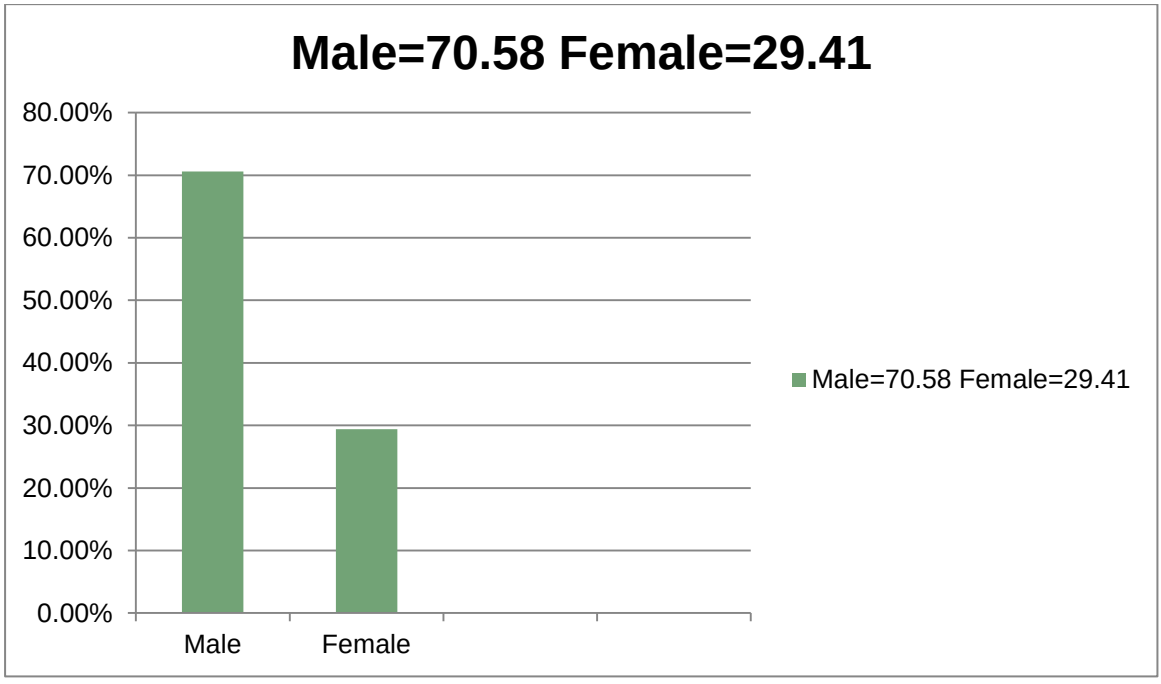
RESULTS

OBSERVATION AND RESULTS

TABLE1. GENDER DISTRIBUTION

Gender	Frequency	Percentage
Male	60	70.58
Female	25	29.41
Total	**85**	**100**

CHART1. BAR DIAGRAM SHOWING GENDER DISTRIBUTION OF 85 PATIENTS

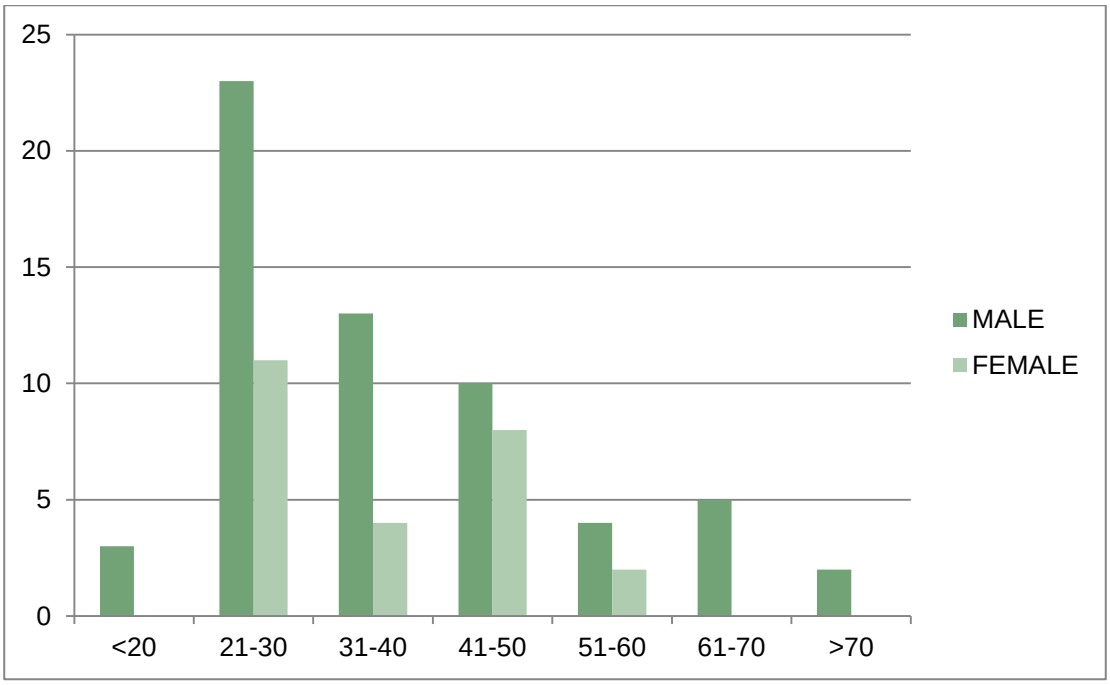


Out of the total 85 patients examined, 60 were male and the rest 25 were females.

TABLE 2. DISTRIBUTION OF THE STUDY POPULATION ACCORDING TO AGE AND SEX

AGE(YEARS)	MALE	FEMALE	TOTAL
<20	3	-	3
21-30	23	11	34
31-40	13	4	17
41-50	10	8	18
51-60	4	2	6
61-70	5	-	5
>70	2	-	2

CHART 2. DISTRIBUTION OF AGE OF THE STUDY POPULATION WITH RESPECT TO GENDER AS FREQUENCY



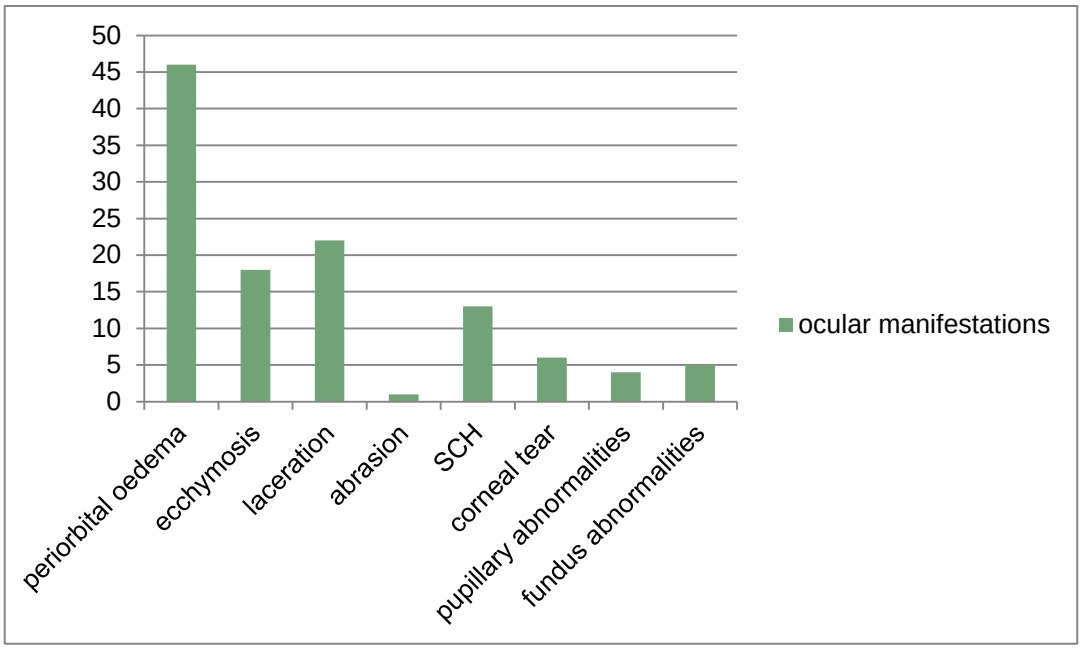
As it is seen, that the age distribution of the patients ranged from 4 months to 76 years with mean age of 35.20 years.

For men the age ranged from 4 months to 70 years with the mean age of 34.14 years and for females the age ranged from 21 to 75 years with the mean age of 45.43 years. The age range of 21 to 30 years accounted for the maximum number of patients in male category and same age range is the commonest in female category,as well.

TABLE3. FREQUENCY OF THE OCULAR MANIFESTATIONS AT THE TIME OF PRESENTATION AS SEEN IN CASUALTY/SICU/PICU

Ocular manifestation	Frequency	Percentage
Periorbital oedema	46	54.11
Ecchymosis	18	21.17
Eyelid laceration	22	25.88
Abrasion	1	1.17
Subconjuctival haemorrhage (SCH)	13	15.29
Corneal tear	6	7.05
Pupillary abnormality	3	3.52
Fundus	4	4.70

CHART 3. BAR DIAGRAM DEPICTING THE FREQUENCY OF OCCURANCE OF OCULAR MANIFESTATIONS AT PRESENTATION IN CASUALTY/SICU/PICU.

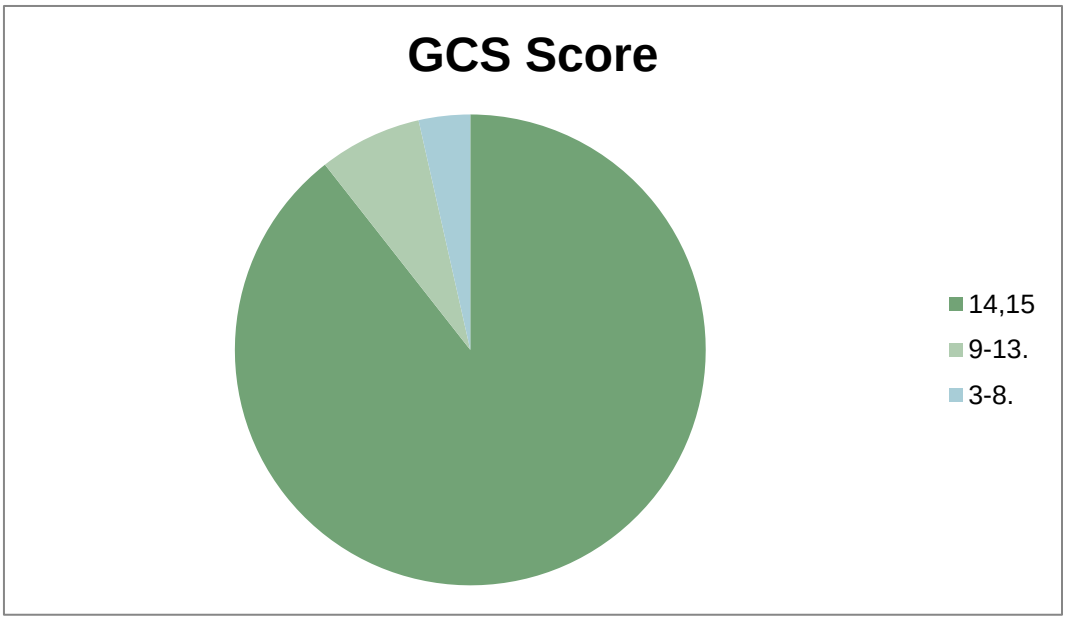


Highest number of the patients showed periorbital oedema as the most common presentation. It was seen in 46 patients out of 85, i.e 54.11%, followed by eyelid lacerations, which was seen in 22 patients,i.e 25.88 %. Ecchymosis was seen in 21.17%. SCH was seen in 15.29% patients ,corneal tear was present in 7.05%. Pupillary abnormalities were present in 3.52 % and fundus findings were there in 4.70% patients, as 1 had Traumatic Optic Neuropathy, 1 had Purtscher's retinopathy , 1 had macular oedema and 1 more had Papilloedema. And eyelid abrasions were present in 1 patient.

TABLE 4. DISTRIBUTION OF CASES IN CATEGORIES GLASGOW COMA SCALE(GCS) SCORE AT PRESENTATION.

GCS score	Frequency	Percent
14,15 (Mild)	76	89.4
9-13 (Moderate)	6	7.1
5-8 (Severe)	3	3.5
Total	85	100.0

CHART 4. SHOWING THE FREQUENCY OF GCS SCORE AT PRESENTATION

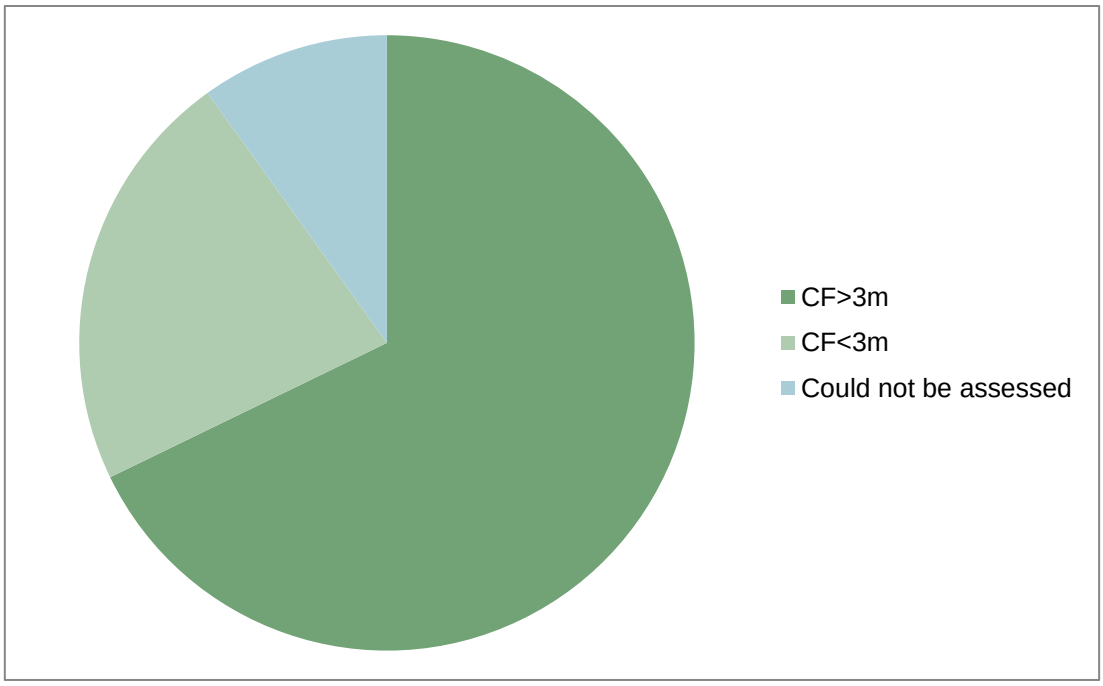


Out of 85 patients , 76 had GCS score 14,15 that is 89.4% patients with closed head injury had normal GSC score when they presented . Six of them (7.10%) had GCS score in the range of 9-13, categorizing head injury as moderate. 3.5% patients had GSC score in the range of 5-8, showing that they had severe head injury.

Table 5. VISUAL ACUITY AT THE TIME OF PRESENTATION

Visual acuity	Frequency	Percent
More than counting finger 3m	62	72.94
Less than counting finger 3m	13	15.29
Could not be assessed	10	11.76
Total	85	100

CHART 5. PIE CHART SHOWING THE FREQUENCY OF VISUAL ACUITY AT THE TIME OF PRESENTATION.



In this study, Observation was made that the visual acuity was greater than counting fingers (CF) 3m in 72.94% patients, and vision was lesser than CF 3m in 15.29% patients. Vision could not be assessed in 11.76% patients, at the presentation.

TABLE 6. GSC CATEGORY AND TRAUMA TO THE OCULAR ADNEXAE

GCS category	Cases with trauma to ocular adnexae	Percent
Mild	67	72.94
Moderate	2	2.35
Severe	0	-

Total 69 patients out of 85, had injury to ocular adnexa.72.94 % cases with trauma to ocular adnexa had mild head injury, whereas 2.35% cases had moderate head injury.

TABLE 7. GSC CATEGORY AND ANTERIOR SEGMENT TRAUMATIC FINDINGS

GCS category	Cases with trauma to the anterior segment	Percent
Mild	16	18.82
Moderate	3	3.53
Severe	0	-

19 patients, i.e 22.35% had injured anterior segment. Among these,18.82 % patients had mild head injury and only 3.53% patients were presented with moderate head injury. And none of the patients with trauma to anterior segment had severe nature of the head injury.

TABLE 8. GSC CATEGORY AND TRAUMA TO THE POSTERIOR SEGMENT

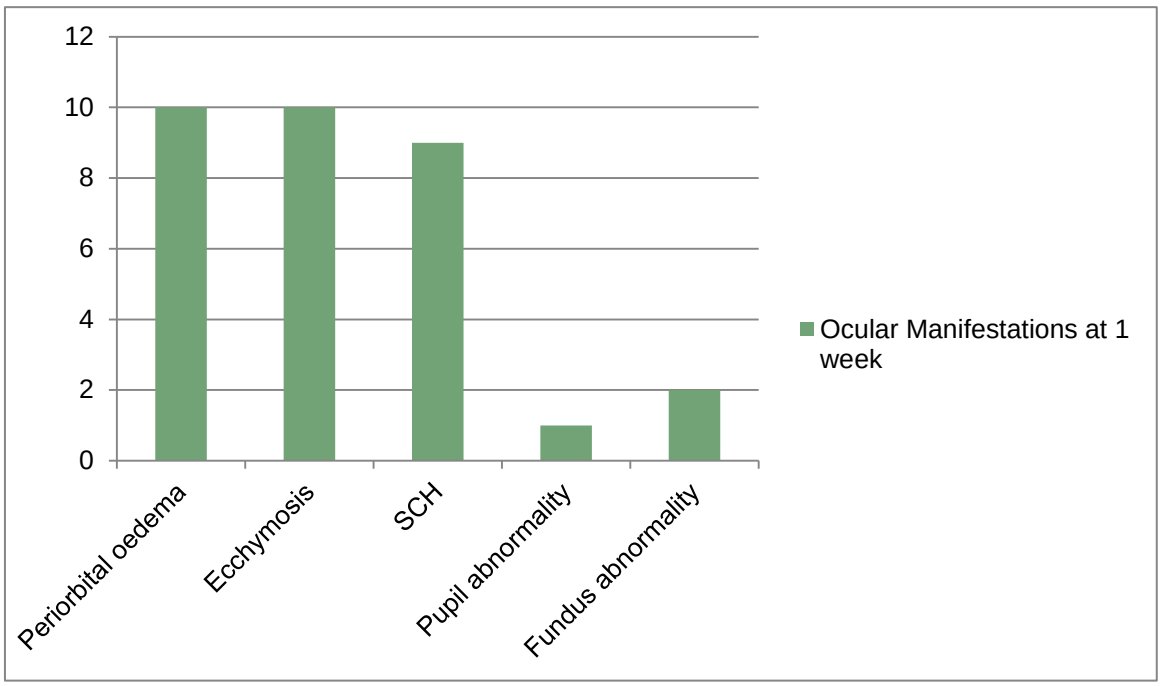
GCS category	Cases with trauma to the posterior segment	Percent
Mild	-	-
Moderate	1	1.17
Severe	3	4.70

Out of 85 patients, 4 had posterior segment manifestations, i.e 4.70%. 3 severe head injury patients had associated posterior segment findings . And 1 patient with posterior segment findings had moderate category of head injury.

TABLE 9. OCULAR MANIFESTATIONS AT 1 WEEK AS SEEN DURING FOLLOW UP IN SICU/OPHTHALMOLOGY OPD

Ocular manifestations	Frequency	Percent
Periorbital oedema	10	17.85
Ecchymosis	10	17.85
SCH	9	16.09
Pupil	1	1.78
Fundus	2	3.57
Nil	26	46.42

CHART 6. OCULAR FINDINGS AT 1 WEEK AS SEEN DURING FOLLOW UP



At 1 week of follow up, out of 85 patients, follow up of 26 patients was lost.

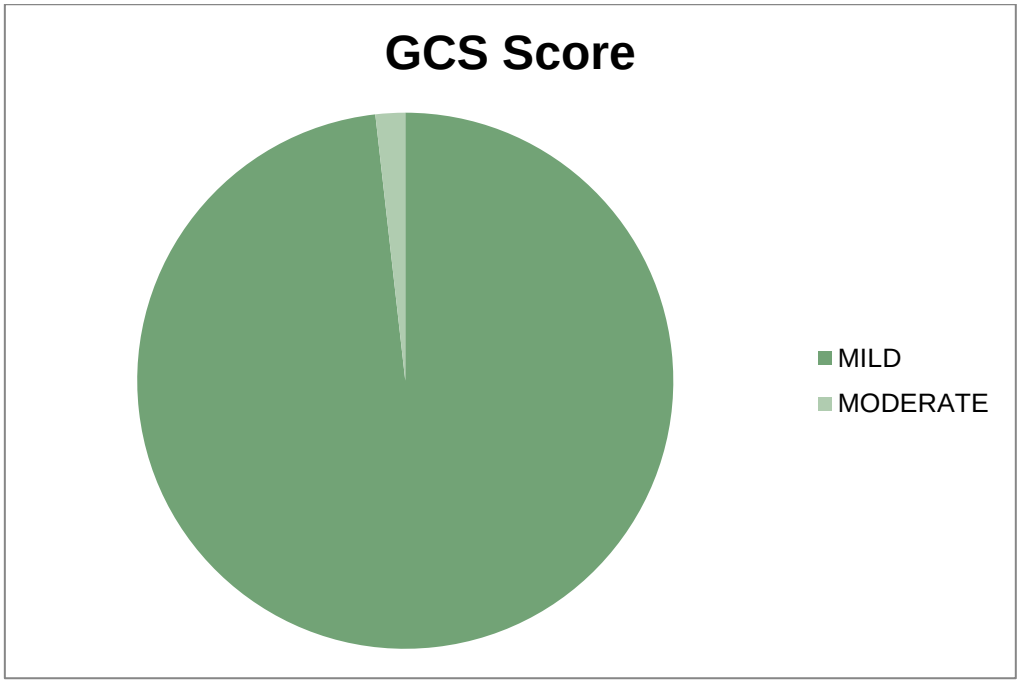
3 patients died because of the head trauma complications. 1 ON DAY1 OF ADMISSION, OTHER 2 ON DAY 2.

The most common ocular findings were periorbital oedema and ecchymosis, both seen in 10 patients,out of 56 patients ,with the frequency of 17.85 % each.

TABLE 10. DISTRIBUTION OF CASES IN CATEGORIES ACCORDING TO GLASGOW COMA SCALE(GCS) SCORE AT 1 WEEK.

GCS score	Frequency	Percent
14,15 (Mild)	55	98.21
9-13 (Moderate)	1	1.79
5-8 (Severe)	-	-
Total	56	100.0

CHART 7. PIE CHART SHOWING THE GCS SCORE AT 1 WEEK



GCS score of 55 patients out of 56 was 15.One patient scored 12 on the GCS scale, who was admitted in SICU of the same tertiary care centre where study was undertaken and follow up was done in SICU while other patients were followed up on OPD basis.

TABLE 11. GCS CATEGORY WITH LIDS AND ADNEXAE

MANIFESTATIONS OF INJURY AT 1 WEEK

GCS category	Cases with injury to ocular adnexa	Percent
Mild	20	35.71
Moderate	-	-
Severe	-	-

All patients with lids and adnexa manifestations at 1 week follow up, had head injury categorized as mild.

TABLE 12. GCS CATEGORY WITH ANTERIOR SEGMENT

MANIFESTATIONS OF INJURY AT 1 WEEK

GCS category	Cases with injury to anterior segment	Percent
Mild	9	16.07
Moderate	-	-
Severe	-	-

Anterior segment manifestations patients had mild head injury at 1 week of follow up

TABLE 13. GCS CATEGORY WITH POSTERIOR SEGMENT

MANIFESTATIONS OF INJURY AT 1 WEEK

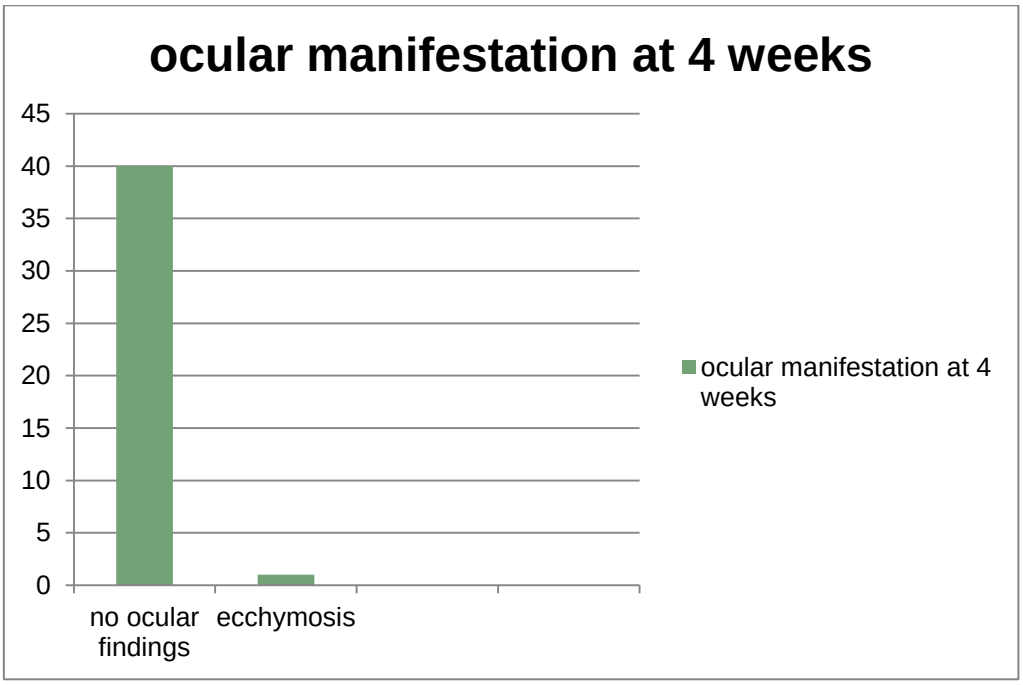
GCS category	Cases with injury to posterior segment	Percent
Mild	-	-
Moderate	-	-
Severe	1	1.78

With posterior segment findings, one patient had severe head injury at first follow up visit.

TABLE 14.SHOWING FREQUENCY OF OCULAR MANIFESTATIONS AT 4 WEEKS

	Occurrence	Percent
No ocular findings	46	47.1
Ecchymosis	1	1.2
Death	4	4.7
Lost follow up	34	40.0
Total	85	100.0

CHART 8. FREQUENCY OF OCULAR MANIFESTATIONS AT 4 WEEKS



At 4 weeks of follow up,only 47 patients were followed up, out of which,1 had ecchymosis and other 46 had no ocular findings, either due to head injury or due to direct trauma. Follow up was lost for another 7 patients and 1 more patient died , since the last follow up, who had the GCS score of 12 at 1 week follow-up.

All patients examined at 4 weeks had GCS score of 15.

TABLE 15. SHOWING THE OCCURENCE OF OCULAR MANIFESTATIONS AT 6 WEEKS

	Occurrence	Percent
No ocular findings	45	52.9
Death	4	4.7
Lost follow up	36	42.4
Total	85	100.0

At 6 weeks, 45 patients were followed up, none of them had any ocular finding ,either due to primary head injury or due to direct ocular trauma,

All patients had GCS score of 45.

TABLE 16. GCS score and VA correlation at the time of presentation.

Head injury severity As GCS score		VA(at presentation)			Total
		CF>3m	CF<3m	Could not be assessed	
	Mild	61	11	4	76
	Moderate	1	2	3	6
	Severe	0	0	3	3
	Total	62	13	10	85

Maximum patients, categorized as having mild head injury had vision CF>3m, i.e 71.76%. 12.94 percent patients had vision CF lesser than 3m with mild head injury. Only 4.70% patients' vision could not be assessed.

Vision could not be assessed for 3.52 percent patients who had moderate head injury, 2.35% had vision CF<3m and 1.17% patients had vision CF>3m.

And in all 3 patients of severe head injury, vision could not be assessed.

TABLE 17. SHOWING THE VISUAL ACUITY AT FOLLOW UP VISITS

BCVA	AT 1 WEEK		AT 4 WEEKS		AT 6 WEEKS	
	OD	OS	OD	OS	OD	OS
6/6-6/18	29	21	28	20	28	20
6/18-6/60	26	34	19	27	17	25
6/60-3/60	1	1	-	-	-	-
3/60-1/60	-		-	-	-	-
1/60-PL	-		-	-	-	-
NPL	-		-	-	-	-

Visual Acuity At 1 Week Of Follow Up , Vision Better Than 6/60 was there for fifty-five patients, and only one patient had Lesser Than 6/60.

And At Subsequent Follow Up Visits, All Patients Had Visual Acuity Better Than 6/60.

DISCUSSION

DISCUSSION

According to the undertaken study, patients who were in the age range of 21 to 30 years were the ones who got affected the most. There were 60 males and 25 females. For men the age ranged from 4 months to 70 years with the mean age of 34.14 years and the age range for female patients was, from 21 to 75 years, mean being the 45.43 years. The majority of males who were affected had age ranging from 21-30 years (23 cases) and the majority of affected females were also in the age group 21-30 years (11 cases).

Age and demographic

In the study by Odebode et al[6], showed the high frequency of affected male patients compared to female patients; i.e male -37 (64.9%) > 20 (35.1%) female subjects.

At the time of the, first examination, it was observed that the youngest patient was 9 months old and the oldest patient was 57 years old, so the mean age of the cases was recorded as 28 years.

In the study by Kulkarni et al[7], a total number of two hundred patients were examined of CHI ,out of which 194 i.e 97% were males patients and 6 i.e 3% were females. The age ranged from 5 to 67 years, with a mean of 28.08 years. 5–67 years was the age range of men with a median of 27.85 years. And for female patients it was 14 - 47 years with mean age as 35.33 years. Male patients with the age range of (21–30 years) were more prone to head trauma, i.e 62%.

In the study by Smitha et al[2], men formed the major portion of the study population which was 92% patients and 8% were the female patients.

16 cases(32%) had their ages in the range of 16-30 years, 13 cases (26%) in the age group of 31-45 years, 15 cases(30%) in the age range of 46-60 years, 4 cases (8%) to

the age group of <15 years and 2 cases(4%) were in the age group of greater than 60 years.

The demographic report of closed head injury cases of the undertaken study correlates with the conclusions of various other studies, showing that male population is more commonly involved, mainly because men more frequently go out and travel more for work and men have more probability to be involved in industrial activity. They are more likely to go through from alcohol abuse which can lead to motor traffic catastrophies and assaults.

Ocular findings

In this study, we saw that more commonly occuring ocular finding was seen as periorbital oedema, in 46 patients out of 85, i.e. 54.11%, followed by eyelid lacerations, seen in 22 patients, i.e 25.88 %. Ecchymosis was seen in 18 patients,21.17%. Subconjuctival haemorrhage was present in 13 patients. Periorbital skin abrasions were present in 1 patient.

In 6 patients, corneal tear was recorded.

Atypical pupil reaction was observed in 3 patients.

A total of four patients, were obsereved to have posterior segment findings i.e 4.70%, as Papilloedema in 1 patient, macular edema 1, Purtschers retinopathy in one case and Traumatic optic neuropathy in 1patient.

According to a study done by Kulkarni et al [7], two hundred patients of skull trauma were examined and the commonest eye finding was observed as ecchymosis in 54 (27%) patients followed by subconjunctival haemorrhages in 38 (19%) patients. 24 patients (12%) were observed to have orbital wall fractures . Papilloedema was seen in 11/200 cases (5.5%),macular edema in 4/200 cases (2%), retinal haemorrhage in

1(0.5%) case, vitreous haemorrhage in 1(0.5%) case, corneal tear in 2(1%) cases, scleral tear in 2(1%) cases, lacrimal gland prolapse in 2 (1%) cases.

Odebode et al[6] conducted one study and inferred, that the soft-tissue injuries to the globe and adnexa were the most frequently occuring eye findings , observed in 29(12.89%) patients, and orbital fracture with rupture of the eye was present in 2(0.89%) patients. Periorbital ecchymosis was the most frequent soft tissue injury, recorded in 17 patients (7.56%), chemosis in 20 patients (8.89%), subconjuctival haemorrhage in 21 patients (9.33%), ten cases had lid laceration, i.e (4.44%), corneoscleral laceration in five cases(2.22%), haemorrhages on the retina were observed in 2 patients (8.89%), and commotio retinae in 3(1.33%) patients. Various other neuro-ophthalmic complications observed were atypical pupil reaction in twelve cases (5.33%), partial or complete ptosis in 10 patients (4.44%), and lagophthalmos in 1 patient (0.44%).

According to the study of Masila et al[5] the commonest findings were in the ocular adnexae with ecchymosis occurring most frequently, in 65 (36.1%) eyes. In the anterior segment, corneal epithelial defects (superficial punctuate erosions and epithelial defects) were the most frequent finding and occurred in 30 (16.7%) eyes. Corneal and scleral lacerations occurred in 1 eye (0.6%) each. Cataract which was developed due to trauma was recorded in 2 eyes (1.1%). In the posterior segment, findings included one patient who had bilateral vitreous haemorrhage (1.1%) and retinal findings included papilloedema 8(4.4%), optic atrophy 19(10.6%), retinal haemorrhage 10 (5.6%), retinal detachment 1(0.6%), commotio retinae 2 (1.1%), macula hole/scar 2 (1.1%), choroidal rupture 2 (1.1%). Fracture of the orbital walls were seen in 9 patients (5%).

In a study by Ramachandran.S et al[50], 60 patients with ocular trauma were included, 31 out of sixty cases had head injury in association of the eye trauma , severity of the head injury was categorized by GCS score, and the observations made were, 25 had mild head injury, moderate head injury was seen in two cases and 4 patients had severe injury. The most common eye finding due to head trauma was ecchymosis of lids and periorbital edema,i.e 58%, followed by subconjuctival haemorrhage.

Study conducted by Abbasi et al.[51] lids and conjunctiva were structures which were more commonly involved.

Observations of the undertaken study, correlate with the findings of other studies.

Neuro-ophthalmic manifestations

Our study showed , 3 patients with pupillary abnormalities,i.e 3.52 %. And 1.17% patients had traumatic optic neuropathy

In the study by Masila et al[5] 39 eyes had atypical pupil reaction. Two patients had third cranial nerve palsy.

According to Kulkarni et al[7], involvement of pupil was present in 10/200 cases (5%) and it was the most frequently occurred neuro-ophthalmic sign. Sixth cranial nerve palsy was recorded in 2% of head injury cases. Traumatic optic neuropathy is seen in 0.5% of cases and third nerve palsy is seen in 1.5% of the cases.

According to observations made by Odebode et al[6] involvement of the sixth cranial nerve was the most common ocular motor nerve palsy, which was present in 8 patients, followed by third and fourth cranial nerve palsies, which were obsereved and documented in six cases each. Other cranial nerves, which were commonly involved in this case series were the seventh cranial nerve in six patients and auditory nerve in 2 patients. Various other neuro-ophthalmic complications recorded were

atypical pupil reaction in 12, partial or complete ptosis in 10, and lagophthalmos in 1case.

In the study by Smitha et al[2],traumatic optic neuropathy was seen in 6 cases (12%).Ocular motor palsy was noted in 6% of cases, of which one had 3rd nerve palsy, one had 6th nerve palsy and one had combined 3rd, 4th and 6th nerve palsy.

Abbasi et al[52] found traumatic motor nerve deformity being the most frequently occurring neuro-ophthalmic manifestation, where Abducens was the one , usually involved in 41% patients.

Correlation of ocular manifestations with the severity of head injury

According to our observations, 72.94 % cases with injured head ,who were classified having mild injury by GCS score had trauma to ocular adnexa, and findings in anterior anterior segment of the eye were associated with mild injury in 18.82% patients. trauma to ocular adnexa was associated with moderate head injury in 2.35% cases.

3.52% cases had anterior segment manifestations, and 1.17% cases had posterior segment manifestations . Severe head trauma and posterior segment findings had constant relation, without any exception . Severe head trauma was also positively correlated with the neuro-ophthalmic findings.

According to Odebode et al[6] and the observations made by them, severe head injury patients had only soft tissue injury to the eye, adnexa and periorbital region,50% of the times. 43.75% patients with severe head injury had neuro-ophthalmic manifestations in association with damage to ocular soft tissue, adnexa and periorbital region,as well. Patients with extreme severity of head inury had rupture of the globe and fracture orbit as well as neuro-ophthalmic manifestations and soft tissue

damage to eye, adnexa and periorbital region in 6.25 % cases. Moderately severe head injury cases had only soft tissue damage to the eye, adnexa and periorbital region, 40% of subjects of moderate head injury had neuroophthalmic manifestations in association with soft tissue of eye damage, adnexa and periorbital region,6.67% subjects of moderately severe trauma to the head had ruptured globe and orbital waal fracture in association with neuro-ophthalmic manifestations and soft tissue of eye damage, adnexa and periorbital region 85.71% of subjects with less severe head injury had only soft tissue damage to the eye, adnexa and periorbital region,38.1% cases of mild head injury had neuroophthalmic manifestations in addition to soft tissue injury to eye,adnexa and region around the eye and mild head injury had no association with any of the following findings- globe rupture and fracture orbit in association with neuroophthalmic manifestations and damage of soft tissue of the eye, adnexa and periorbital region .

According to Kulkarni et al[7] and their observations, 82.7% patients with less severe head injury had eye involvement of no neurological significance, including unilateral and bilateral ecchymosis 49 (32.67%), subconjunctival haemorrhages 38 (25.33%), orbital margin fractures in 12 (8%), proptosis in 6 (4%), blow out fractures of the orbit in four (2.67%), macular oedema in three (2%) cases, unilateral traumatic mydriasis, lacrimal gland prolapse and scleral and corneal tears in 2(1.33%) cases each, and hyphaema and haemorrhage in vitreous in one (0.7%) case each. 82.8% of patients of moderately injured head had ocular involvement. 9(31.03%) patients had pupillary signs. Papilloedema was observed in three (10.34%) cases, lateral rectus palsy in 4(13.79%) cases, ecchymosis and orbital margin fractures in 2(6.9%) cases each, retinal haemorrhage, macular oedema, ptosis, and traumatic optic neuropathy in 1(3.45%) case each. 90.48% patients of extremely severe head injury had involvement

of eye. 2 (9.52%) patients had pupillary involvement. Papilloedema was seen in 8 (38.1%) cases, orbital fractures were observed in six (28.57%) cases and ecchymosis was seen in 3(14.29%) cases

The findings of these studies largely correlate with the findings of our study. The mild discrepancy can be contributed to the fact that all subjects of our study were cases of closed head injury who had involvement of the eye as well whereas the cases in other studies were cases of head injury in which the majority had ophthalmic manifestations.

In this study, visual acuity greater than CF 3m was present in 59 cases, less than CF 3m in 18 cases and vision was unrecordable in 8 cases at presentation.

In the study done by Masila et al[5] ,70.8% eyes had the normal visual acuity ,21.90% had visual impairment, and 2.4% had severe visual impairment.

These findings correlate with our study.

Follow up visits-

At 1 week of follow up, we lost the follow up for 26 patients,and 3 patients died. So the follow up was done for 56 patients ,commonly occurring ocular findings were periorbital oedema and ecchymosis, documented in 10 patients each, with the frequency of 17.85 % each followed by SCH, in 16.07%. and in 1 patient optic nerve atrophy secondary to trauma was seen. And the remaining twenty-six subjects had no ocular findings.

The whole lot of cases with lids and adnexa manifestations at 1 week follow up had head injury categorized as mild. Anterior segment manifestations patients had mild head injury at 1 week of follow up. Severe head injury with posterior segment findings, were recorded in one patient.

Visual acuity less than 6/60,was recorded in one patient at the first follow up visit. others had visual acuity greater than 6/60 in both eyes.

At 4 weeks of follow up, we lost the follow up for 8 more patients, and 1 more patient died. So the follow up was done for 47 patients and 1 patient had ecchymosis and all other patients had no ocular finding.

All patients examined at 4 weeks had GCS score of 15.

All followed up patients at 4 weeks had their vision were better than 6/60.

At 6 weeks of follow up, only 45 patients were followed up,and none of them had any ocular finding. All of them had GCS score of 15.

All followed up cases at 6 weeks had their vision greater than 6/60.

So, the observation made was that at various follow up visits,the patients had improvement in the ocular manifeatations of the head injury and of the direct trauma to the eye. There was in the improvement in the GCS score as well.

Refractive errors, cataract and posterior capsular opacification were accounted for the less vision in the patients at the follow-up visits.

CONCLUSION

CONCLUSION

With the inferences drawn from the study we conclude that,

There can be various ocular manifestations in closed head injury patients, such as injury to the lids and adnexa, anterior segment manifestations, manifestations which involved posterior segment of the eye and neuro-ophthalmic manifestations as well.

The correlation of the severity of head injury with the posterior segment and neuro-ophthalmic manifestations , showed that the more severe form of head injury is related with severe ocular findings and thus resulting in poorer neurological and visual outcomes.

Therefore, a detailed ocular assessment during the first presentation in all cases of head injury patients is mandatory as it helps in better management and the final outcome could be improved with better diagnosis and management.

SUMMARY

SUMMARY

A total of 85 patients fulfilling the inclusion criteria were selected, who presented at the emergency department ,at R.L.J. HOSPITAL AND RESEARCH CENTRE, TAMAKA, KOLAR attached to SRI DEVARAJ URS MEDICAL COLLEGE between January 2018 and May 2019.

Neurological assessment was done by using Glasgow Coma Scale and further grading of head injury was done as,mild (GCS 14-15),moderate (9-13) and severe (3-8).

Detailed history regarding injury was documented . Patients were assessed at the time of presentation, Ophthalmic examination included assessment of visual acuity , external injury assessment , anterior segment examination, pupillary reaction, assessment of extra-ocular movements, visual field assessment was done by finger confrontation.Posterior segment was examined using direct and indirect ophthalmoscope.

If necessary, assessment of visual acuity using Snellen's chart, assessment of intra ocular pressure using Goldmann applanation tonometer, and diplopia charting was done.

Follow up was done at 1 week, at 4 weeks and at 6 weeks after the injury and, neurological assessment was done using GSC score. Assessment of best corrected visual acuity was done and ocular findings were documented.

The highest incidence of head injury was seen in the age group of 21-30 years. Men were more affected than women,as it was observed, total number of affected males was sixty and females was twenty-five.

The most common ocular finding at presentation was seen as periorbital oedema,in 46 patients i.e 54.11%, followed by eyelid lacerations,seen in 22 patients,i.e 25.88 %.

Ecchymosis in ,21.17%. Subconjuctival haemorrhage in 13 patients. Periorbital skin abrasions in 1 patient. In 6 patients, corneal tear was recorded.Atypical pupil reaction was observed in 3 patients.A total of four patients, were obsereved to have posterior segment findings i.e 4.70%, as Papilloedema in 1 patient, macular edema 1, Purtschers retinopathy in one case and Traumatic optic neuropathy in 1patient.

Head injury was mild for 76,moderate for 6 and severe for 3 patients.

At 1 week of follow up, we lost the follow up for 26 patients,and 3 patients died. So the follow up was done for 56 patients , periorbital oedema and ecchymosis,was documented in 17.85 % patients each followed by SCH, in 16.07%. and in 1 patient optic nerve atrophy secondary to trauma was seen. And the remaining twenty-six subjects had no ocular findings.All cases with lids and adnexa manifestations at 1 week follow up had mild head injury. Anterior segment manifestations patients had mild head injury at 1 week of follow up. Severe head injury with posterior segment findings, were recorded in one patient.Visual acuity less than 6/60,was recorded in one patient at the first follow up visit. others had visual acuity greater than 6/60 in both eyes.

At 4 weeks of follow up, we lost the follow up for 8 more patients, and 1 more patient died. So the follow up was done for 47 patients and 1 patient had ecchymosis and all other patients had no ocular finding.All patients examined at 4 weeks had GCS score of 15 and had their vision were better than 6/60. At 6 weeks of follow up, only 45 patients were followed up,and none of them had any ocular finding. All of them had GCS score of 15 and had their vision greater than 6/60.

There should be detailed early Ophthalmological assessment in all patients with head injury. In severe head injury patients,it is important to look for neuro-ophthalmic and posterior segment findings, as they have positive correlation.

This correlation can be of prognostic value for the visual as well as overall outcome of the patient.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Ventura RE, Balcer LJ, Galetta SL. The neuro-ophthalmolgy of head trauma. Lancet Neurol 2014;13:1006-16.
2. Smitha K S, Patil SB. Ocular and visual complications of head injury- a cross-sectional study. National Journal of Medical and Dental Research 2015 Jan-March ;3(2): 78-82.
3. Van GP, Biousse V, Lynn MJ, Simon DJ, Newman NJ. Neuro-ophthalmic manifestations of head trauma. J Neuro Ophthalmol 2001;21:112-17.
4. Sharma B, Gupta R, Anand R, Ingle R. Ocular manifestations of head injury and incidence of post traumatic ocular motor nerve involvement in cases of head injury: a clinical review. Int Ophthalmol 2014;34:893-900.
5. Faith M, Githinji KJ, Sheila M, Margaret N. Ocular findings in patients with head injury. International Journal of Medical and Clinical Sciences 2014;1(2):009-017.
6. Odebode TO, Ademola-Popoola DS, Ojo TA, Ayanniyi AA. Ocular and visual complications of head injury. Eye 2005;19:561-66.
7. Kulkarni AR, Aggarwal SP, Kulkarni RR, Deshpande MD, Walimbe PB, Labhsetwar AS. Ocular manifestations of head injury: A clinical study.Eye (Lond) 2005;19:1257-63.
8. Kumari R, Saha BC , Saha KK , Sinha BP. Ocular Manifestations in Head Injury Patients -A Prospective Study. International Journal of Contemporary Medical Research. 2017;4: 1648-51.
9. Cohen M, Groswasser Z, Barchadski R, Appel A. Convergence insufficiency in brain injured patients. Brain Injury 1989;3(2):187-91.
10. Malik A, Gupta A, Luthra N, Gupta V. Ocular manifestations of head injury: A clinical study. Sudanese J Ophthalmol 2016;8: 46-50.

11. Rao KVM, Kumari B. Ocular manifestations of head injuries. J Evid Based Med Health 2016;3:5545-49.
12. Garg K, Mittal P, Chandrupatla M. Human Anatomy.7th ed. New Delhi:CBS Publishers;2016.Pp4-12.
13. Khurana AK, Khurana I.Anatomy and physiology of eye. 3rd ed.New Delhi:CBS Publishers;2017.Pp570-85.
14. Saatman KE, Duhaime AC et al. Classification of traumatic brain injury for targeted therapies. Journal of Neurotruama 2008; 25(7):719-38.
15. Mass Al, Stocchetti N, Bullock R. Moderate and severe traumatic brain injury in adults. Lancet Neurology 2008 ;7 (8); 728-41.
16. Blissitt PA. Care of the critically ill patient with penetrating head injury.Critical Care Nursing Clinics of North America 2006;18 (3):321-32.
17. Hannay HJ, Howieson DB, Loring DW, Fischer JS, Lezak MD."Neuropathology for neuropsychologists". In Lezak MD,Howieson DB,LOring DW. Neuropsychological Assessment. Oxford : Oxford University Press; 2004. Pp.158-62.
18. Williams N, Bulstrode C, O'Connell P. Shot practice of surgery.26th ed.Boca Raton: CRC press,2013.Pp310-14.
19. G.Matis and T.Birbilis, "The Glasgow Coma Scale- A briefreview. Past,present,future," Acta Neurologica Belgica 2008;108:75-89.
20. Jennett B. The Glasgow Coma Scale: History and current practice.trauma 2002;4:91-103.
21. Batchelor J,Mcguiness A. A meta-analysis of GCS in 15 head injured patients with loss of consciousness or post-traumatic amnesia. Emerg.Med.J.2002;19:515-19.

22. Keane JR. Neurologic eye signs following motor vehicle accidents. Arch Neurol 1989;46:761-2.
23. Clinical Guideline 4. Head injury:Triage, Assessment, Investigation and Early Management of Head Injury in Infants, Children and Adults. National Institute for Clinical Excellence :London 2003.
24. Khurana A.K, Khurana A, Khurana B. Ocular injuries.In: A.K Khurana. Comprehensive Ophthalmology. NewDelhi :JP;2019.pp 446-49.
25. Bowling B.Ocular trauma. In: Bowling B. Kanski's Clinical Ophthalmology.China:Elsevier:2016.p862-73.
26. Sabates N, Gonce M, Farris B. Neuro-ophthalmological findings in closed head trauma. J Clin Neuroophthalmol 1991;11:273-7.
27. Moster ML,Volpe NJ, Kresloff MS. Neuro-ophthalmic findings in head injury. Neurol 1999;52: A23.
28. Davagnanam I, Fraser CL, Miszkiel K, Daniel CS,Plant GT. Adult Horner's syndrome: a combined clinical,pharmacological and imaging algorithm. Eye 2013; 27: 291-98.
29. Stanley Thompson H. The Pupil. In: Adler's Physiology of the Eye. Hart Jr,WM, Ed, Mosby year Book, St Louis, 1992; 412-41.
30. Rabiu T. Neuroclinical Anatomy of the third cranial nerve. The Internet Journal of Neurology.2009;12 (2).
31. Yamada SM, Teramoto A, Yamada S,Hoshiai YC. Severe papilledema identified 3 weeks after head injury. Neurol Med Chir (Tokoyo).2002;42 (7):293-6.
32. Kumar J, Tiwari P,Sharma J. Berlin's edema in Bundelkhand region.IOSR journal of dental and medical sciences 2018;17:19-22.

33. Baker RS, Epstein AD. Ocular motor abnormalities from head trauma. Surv Ophthalmol 1994 ;35:245-67.
34. Smith JL. Some neuro-ophthalmoological aspects of head trauma. Clin Neurosurg 1966;13:181-96.
35. Sarkies N. Traumatic optic neuropathy. Eye 2004 ; 18:112-15.
36. Steinspair KD. Traumatic optic neuropathy. Curr Opin Ophthalmol 1999; 10:340-42.
37. Buckley SA, James B. Purtscher's retinopathy. Postgrad Med J 1996;72(849):409-12.
38. Mc Carron M,Alberts M, McCarron P. A systemic review of Terson's syndrome:frequency and prognosis after subarachnoid haemorrhage. Journal of neurology,Neurosurgery and Psychiatry. 2004;75 (3):491-93.
39. Gauntt CD, Sherry RG, Khanna C. Terson syndrome with bilateral optic nerve sheath haemorrhage. J Neuroophthal 2007;27(3):193-94.
40. Frizzel RT, Kuhn F, Morris R,et al. Screening for ocular haemorrhages in patients with ruptured cerebral aneurysms: a prospective study of 99 patients. Neurosurgery 1997;41: .529-34.
41. Eyster EF, Hoyt WF, Wilson CB. Oculomotor palsy from minor head trauma. JAMA 1972;220:1083-86.
42. Memon MY, Paine KWE. Direct injury of the oculomotor nerve in craniocerebral trauma. J Neurosurg 1971;35;461-64.
43. Tiffin PAC, MacEwen CJ,Craig EA,et al. Acquired Palsy of the ocuolomotor,trochlear and abducens nerves. Eye 1996;10:377-84.

44. Jacobson DM, Warner JJ, Choucair AK ,Ptacek LJ. Trochlear nerve palsy following minor head trauma. A sign of structural disorder. J clin Neuroophthalmol.1988 ;8 (4):263-68.
45. Brazis,P.W. Palsies of the trochlear nerve (diagnosis and localization- Recent concepts). Mayo Clin Proc 1993;68:501-9.
46. Advani RM , Bauann MR. Bilateral sixth nerve palsy after head trauma. Ann Emerg Med. 2003:41:27-31.
47. Cho MS, Kim SH. Delayed bilateral abducens nerve palsy after head trauma . J Korean Neurosurg Soc.2008;44:396-98.
48. Salunke P, Savardekar A,Sura S. Delayed-onset bilateral abducens paresis after head trauma. Indian Journal of Ophthalmology. 2012:60(2):149-50.
49. Puvanendran K, Vitharana M, W.P.K. Delayed facial palsy after head injury . Journal of Neurology, Neurosurgery, and Psychiatry:1997,40: 342-50.
50. Ramachandra S, Yeturu S, Pujar C, Sagara S,Veerakanellore S, Kailasam D. Profile of head injuries in road traffic accident cases with ocular trauma in a rural tertiary care hospital. Ind J of clin and experimental Ophthlmology. 2015;1(4):245-51.
51. Abbasi K, Qadeer B, Raza A. Ocular manifestations associated with head injury. Pakistan Journal of Ophthalmology.2016;32(2):111-16

ANNEXURES

ANNEXURE I :

PROFROMA

STUDY PROFORMA

Case no: **Date:**

Name: **OP no:**

Age: **IP no:**

Sex: **DOA:**

Occupation: **DOS:**

Address:

Brief history:

Past history:

Family history:

Personal history:

GPE:

Vital signs:

Pulse –

RR –

BP –

Temp –

Systemic examination:

a) Cardiovascular system –

b) Respiratory System –

c) Per Abdominal examination –

d) Central Nervous System –

GLASGOW COMA SCALE

		1	2	3	4	5
EYE		DOES NOT OPEN EYES	OPENS EYES IN RESPONSE TO PAINFUL STIMULI	OPENS EYES IN RESPONSE TO VERBAL COMMANDS	OPENS EYES SPONTANEOUSLY	N/A
VERBAL		NO SOUNDS	SOUNDS ONLY	INAPPROPRIATE WORDS	CONFUSED	NORMAL ORIENTED CONVERSATION
MOTOR		NO MOTOR RESPONSE	EXTENSION (DECEREBRATE)	ABNORMAL FLEXION (DECORTICATE)	WITHDRAWAL /FLEXION	LOCALIZES TO PAIN

GCS SCORE= $E_4 V_5 M_6$

OCULAR EXAMINATION

TESTS **RE** **LE**

1. HEAD POSTURE
2. OCULAR POSTURE
3. FACIAL SYMMETRY
4. EXTRAOCULAR MOVEMENTS
    a) Ductions
    b) Versions
5. VISUAL ACUITY:
    a) Distant
    b) Near
6. **ANTERIOR SEGMENT**
    a. Lids and Adnexa
    b. Conjunctiva
    c. Cornea
    d. Anterior chamber
    e. Iris
    f. Pupil
    g. Lens
    h. Anterior Vitreous
7. FUNDUS
    a. Distant direct ophthalmoscopy
    b. Direct ophthalmoscopy
    c. Indirect ophthalmoscopy
8. IOP (mm Hg GAT)
9. Diplopia charting

FOLLOW UP ASSESSMENT:

FOLLOW UP VISITS	**1 WEEK**	**4 WEEKS**	**6 WEEKS**
VISUAL ACUITY			
GSC score			
Ocular findings			

ANNEXURE II: INFORMED CONSENT

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

Case No:
IP No:

TITLE: A STUDY TO CORRELATE OCULAR MANIFESTATIONS OF CLOSED HEAD INJURY WITH GLASGOW COMA SCALE AND VISION AND NEUROLOGICAL OUTCOME IN A RURAL TERTIARY CARE CENTER.

I, the undersigned, agree to participate in this study and authorize the collection and disclosure of personal information as outlined in this consent form.

I understand the purpose of this study, the risks and benefits of the technique and the confidential nature of the information that will be collected and disclosed during the study. The information collected will be used only for research.

I have had the opportunity to ask questions regarding the various aspects of this study and my questions have been answered to my satisfaction.

I understand that I remain free to withdraw the participation from this study at any time and this will not change the future care.

Participation in this study does not involve any extra cost to me.

Name	Signature	Date	Time
Patient:			
Witness:			
Primary Investigator/ Doctor:			

ಮಾಹಿತಿ ಕಾನ್ಸೆಂಟ್ ಫಾರ್ಮ್

ಪ್ರಕರಣ ಸಂಖ್ಯೆ :

ಐಪಿ ಸಂಖ್ಯೆ :

ಶೀರ್ಷಿಕೆ: ಗ್ಲಾಸ್ಗೋ ಕೋಮಾ ಸ್ಕೇಲ್ ಮತ್ತು ವಿಷನ್ ಮತ್ತು ರೂರಲ್ ಟೆರ್ಟಿಯರಿ ಕೇರ್ ಸೆಂಟರ್ನಲ್ಲಿನ ನ್ಯೂರೋಲಾಜಿಕಲ್ ಹೊರಗುತ್ತಿಗೆಗಳೊಂದಿಗೆ ಮುಚ್ಚಿದ ತಲೆಯ ಗಾಯದ ಒರಟಾದ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್ಗಳನ್ನು ಸರಿಪಡಿಸಲು ಒಂದು ಅಧ್ಯಯನ.

ನಾನು, ಸಹಿ ಮಾಡದವರು, ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ಈ ಒಪ್ಪಿಗೆಯ ರೂಪದಲ್ಲಿ ವಿವರಿಸಿರುವಂತೆ ವೈಯಕ್ತಿಕ ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ಅಧಿಕೃತಗೊಳಿಸುತ್ತೇನೆ.

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶ, ತಂತ್ರದ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರ ಯೋಜನಗಳು ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿ ಬಹಿರಂಗಪಡಿಸುವ ಮಾಹಿತಿಯ ಗೌಪ್ಯ ಸ್ವರೂಪವನ್ನು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳಿಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳುವ ಅವಕಾಶ ನನಗೆ ಸಿಕ್ಕಿದೆ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ.

ಈ ಅಧ್ಯಯನದಿಂದ ಭಾಗವಹಿಸುವಿಕೆಯನ್ನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂತೆಗೆದುಕೊಳ್ಳಲು ನಾನು ಮುಕ್ತನಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ಭವಿಷ್ಯದ ಆರೈಕೆಯನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಿಕೆಯು ನನಗೆ ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ವೆಚ್ಚವನ್ನು ಒಳಗೊಂಡಿರುವುದಿಲ್ಲ

ಹೆಸರು	ಸಹಿ/ಹೆಬ್ಬೆಟ್ಟಿನಗುರುತು	ದಿನಾಂಕ	ಸಮಯ
ರೋಗಿಯಹೆಸರು			
ಸಾಕ್ಷಿಗಳಹೆಸರು			
ಪ್ರಾಥಮಿಕಸಂಶೋಧಕರು/ ವೈದ್ಯರು			

ANNEXURE III: PATIENT INFORMATION SHEET

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.
PATIENT INFORMATION SHEET

Case no:

IP no:

TITLE: A STUDY TO CORRELATE OCULAR MANIFESTATIONS OF CLOSED HEAD INJURY WITH GLASGOW COMA SCALE AND VISION AND NEUROLOGICAL OUTCOME IN A RURAL TERTIARY CARE CENTER.

- Head injuries are a cause of hospitalization of 200-300 persons per 100,000 population per year and about 25% of these are associated with ocular and visual defects.
- Ocular trauma is the leading cause of loss of vision.
- Hence, the role of ocular injuries secondary to head trauma in the causation of blindness and overall prognosis of patients has become a subject of immense importance.

If you agree to participate in the study we will examine you for the anterior segment and fundus changes. We will collect the relevant details about you from your hospital record. The information collected will be used only for research purpose. This study will be reviewed by local ethical board and will be started only after their formal approval. The care you will get will not change if you do not wish to participate. You are required to sign/thumb impression only if you voluntarily agree to participate in this study. Participation in this study will not

involve any cost for you. This also does not affect the care you will receive in the hospital.Participation in this research study may not change the final outcome of your eye condition. However, patients in the future may benefit as a result of knowledge gained from this study. You will not be charged extra for any of the procedures performed during the research study. Your taking part in this study is entirely voluntary. You may refuse to take part in the study or you may stop your participation in the study at any time, without a penalty or loss of any benefits to which you were otherwise entitled before taking part in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information/ clarification please contact Dr.Sandhaya R, SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101. Contact no: 9844177487

CONTACT DETAILS:

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ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಪ್ರಕರ ಸಂಖ್ಯೆ :

ಐಪಿ ಸಂಖ್ಯೆ :

ಶೀರ್ಷಿಕೆ: ಗ್ಲಾಸ್ಗೋ ಕೋಮಾ ಸ್ಕೇಲ್ ಮತ್ತು ವಿಷನ್ ಮತ್ತು ರೂರಲ್ ಟೆರ್ಟಿಯರಿ ಕೇರ್ ಸೆಂಟರ್ನಲ್ಲಿನ ನ್ಯೂರೋಲಾಜಿಕಲ್ ಹೊರಗುತ್ತಿಗೆಗಳೊಂದಿಗೆ ಮುಚ್ಚಿದ ತಲೆಯ ಗಾಯದ ಒರಟಾದ ಮ್ಯಾನಿಫೆಸ್ಟೇಶನ್ಗಳನ್ನು ಸರಿಪಡಿಸಲು ಒಂದು ಅಧ್ಯಯನ.

ಗಾಯಗಳು ವರ್ಷಕ್ಕೆ 100,000 ಜನಸಂಖ್ಯೆಗೆ 200-300 ಜನರನ್ನು ಆಸ್ಪತ್ರೆಗೆ ದಾಖಲಿಸಲು ಒಂದು ಕಾರಣವಾಗಿದೆ ಮತ್ತು ಇವುಗಳಲ್ಲಿ ಸುಮಾರು 25% ರಷ್ಟು ಆಕ್ಯುಲರ್ ಮತ್ತು ದೃಷ್ಟಿ ದೋಷಗಳಿಗೆ ಸಂಬಂಧಿಸಿವೆ.

ದೃಷ್ಟಿ ಕಳೆದುಕೊಳ್ಳಲು ಆಕ್ಯುಲರ್ ಆಘಾತ ಪ್ರಮುಖ ಕಾರಣವಾಗಿದೆ.

ಆದ್ದರಿಂದ, ಕುರುಡುತನ ಮತ್ತು ರೋಗಿಗಳ ಒಟ್ಟಾರೆ ಮುನ್ನರಿವು ಉಂಟಾಗುವುದರಲ್ಲಿ ತಲೆ ಆಘಾತಕ್ಕೆ ದ್ವಿತೀಯಕ ಆಕ್ಯುಲರ್ ಗಾಯಗಳ ಪಾತ್ರವು ಅಪಾರ ಪ್ರಾಮುಖ್ಯತೆಯ ವಿಷಯವಾಗಿದೆ.

ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ಮುಂಭಾಗದ ವಿಭಾಗ ಮತ್ತು ಫಂಡಸ್ ಬದಲಾವಣೆಗಳಿಗಾಗಿ ನಾವು ನಿಮ್ಮನ್ನು ಪರಿಶೀಲಿಸುತ್ತೇವೆ.

ನಿಮ್ಮ ಆಸ್ಪತ್ರೆಯ ದಾಖಲೆಯಿಂದ ನಿಮ್ಮ ಬಗ್ಗೆ ಸಂಬಂಧಿಸಿದ ವಿವರಗಳನ್ನು ನಾವು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಗ್ರಹಿಸಿದ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನವನ್ನು ಸ್ಥಳೀಯ ನೈತಿಕ ಮಂಡಳಿಯು ಪರಿಶೀಲಿಸುತ್ತದೆ ಮತ್ತು ಅವರ formal ಪಚಾರಿಕ ಅನುಮೋದನೆಯ ನಂತರವೇ ಪ್ರಾರಂಭವಾಗುತ್ತದೆ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಆರೈಕೆ ಬದಲಾಗುವುದಿಲ್ಲ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ ಮಾಡಬೇಕಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದರಿಂದ ನಿಮಗಾಗಿ ಯಾವುದೇ ವೆಚ್ಚವನ್ನು ಒಳಗೊಂಡಿರುವುದಿಲ್ಲ.

ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನಿರಾಕರಿಸಬಹುದು ಅಥವಾ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವ ಮೊದಲು ನಿಮಗೆ ಅರ್ಹತೆ ದೊರೆತ ಯಾವುದೇ ಪ್ರಯೋಜನಗಳ ದಂಡ ಅಥವಾ ನಷ್ಟವಿಲ್ಲದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವುದನ್ನು ನಿಲ್ಲಿಸಬಹುದು.

ಗೌಪ್ಯತೆ

ನಿಮ್ಮ ವೈದ್ಯಕೀಯ ಮಾಹಿತಿಯನ್ನು ಅಧ್ಯಯನ ವೈದ್ಯರು ಮತ್ತು ಸಿಬ್ಬಂದಿ ಗೌಪ್ಯವಾಗಿಡುತ್ತಾರೆ ಮತ್ತು ಸಾರ್ವಜನಿಕವಾಗಿ ಲಭ್ಯವಾಗುವುದಿಲ್ಲ.

ನಿಮ್ಮ ಮೂಲ ದಾಖಲೆಗಳನ್ನು ನಿಮ್ಮ ವೈದ್ಯರು ಅಥವಾ ನೈತಿಕ ಪರಿಶೀಲನಾ ಮಂಡಳಿಯು ಪರಿಶೀಲಿಸಬಹುದು

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ / ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ದಯವಿಟ್ಟು ಡಾ.ಸಂಧಯ ಆರ್, ಶ್ರೀ ದೇವರಾಜ್ ಯುಆರ್ಎಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ ಅಂಡ್ ರಿಸರ್ಚ್, ತಮಾಕಾ, ಕೋಲಾರ್ - 563101 ಅನ್ನು ಸಂಪರ್ಕಿಸಿ. ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9844177487.

ಸಂಪರ್ಕ ವಿವರಗಳು:

ಡಾ.ಸಂಧ್ಯಾ ಆರ್, ಎಂ.ಬಿ.ಬಿ.ಎಸ್., ಎಂ.ಎಸ್.,

ಪ್ರೊಫೆಸರ್ ಮತ್ತು ಯುನಿಟ್ ಹೆಡ್

ನೇತ್ರಶಾಸ್ತ್ರ ವಿಭಾಗ,

ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು

ತಮಾಕಾ, ಕೋಲಾರ - 563101

ದೂರವಾಣಿ ಸಂಖ್ಯೆ -9844177487

ಡಾ.ಮಾನು ಸೈನಿ

ಕಿರಿಯ ನಿವಾಸಿ

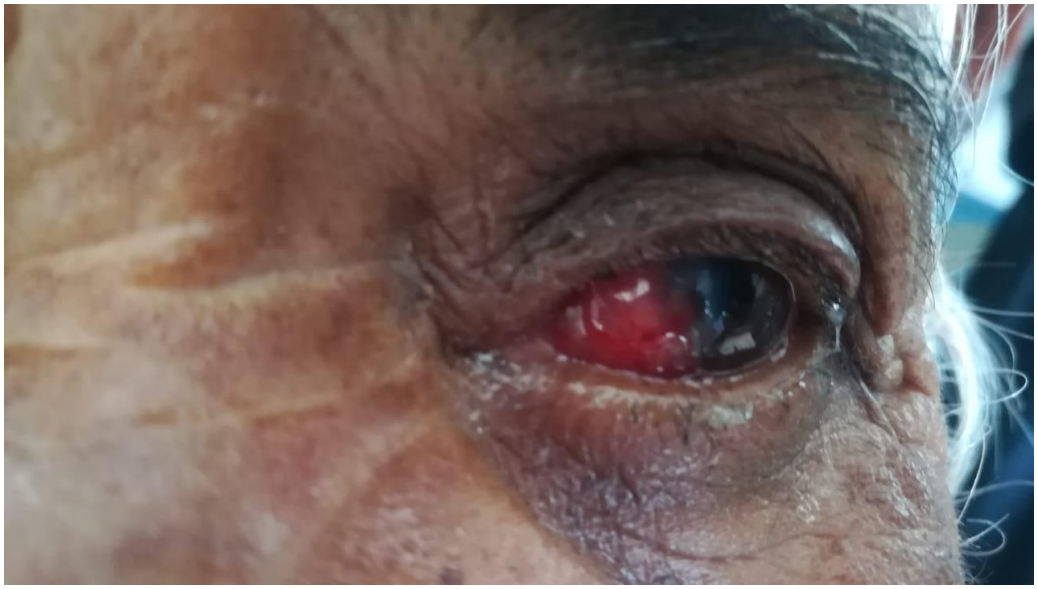
ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು

ದೂರವಾಣಿ ಸಂಖ್ಯೆ-8630635150

ANNEXURE IV

PHOTOGRAPHS

Photograph 1. Right eye lid laceration with periorbital oedema and ecchymosis.



Photograph 2. Left eye- subconjunctival haemorrhage is seen.

Photograph 3. Right eye- Laceration of the upper eye lid with periorbital oedema and ecchymosis.

KEY TO MASTER CHART

SEX – 1= MALE

2= FEMALE

EYE- 1=RIGHT

2=LEFT

3=BOTH

OCULAR MANIFESTATIONS-

0= NO OCULAR MANIFESTATIONS

1=PERIORBITAL OEDEMA

2=ECCHYMOSIS

3=EYELID LACERATION

4=ABRASION

5=OEDEMA AND LACERATION

6=OEDEMA AND ECCHYMOSIS

7=LACERATION,ECCHYMOSIS AND OEDEMA

8=SCH

9=SCH AND OEDEMA

10=SCH AND ECCHYMOSIS

11=OEDEMA ECCHYMOSIS AND SCH

12=OEDEMA,ECCHYMOSIS,SCH AND LACERATION

13=CORNEAL TEAR

14=PUPILLARY ABNORMALITIES

15=FUNDUS FINDINGS

16= PUPIL AND FUNDUS INVOLVEMENT

17=DEATH

18=LOST FOLLOW UP

LOCATION OF OCULAR MANIFETATION- 1 OCULAR ADNEXA

2-ANTERIOR SEGMENT

3-POSTERIOR SEGMENT

4-PUPILLARYABNORMALITY

VISUAL ACUITY - A- MORE THAN 3M

(AT PRESENTATION) B- LESS THAN 3M

C-COULD NOT BE ASSESSED

GCS SCORE(HEAD INJURY SEVERITY) –A- 14,15 (MILD)

B- 9-13 (MODERATE)

C- 3-8(SEVERE)

VISUAL ACUITY - (AT 1 ,4 AND 6 WEEKS)

A-6/6

B-6/9

C-6/12

D-6/18

E-6/24

F-6/36

G-6/60

op	age	sex	eye	ocular mani(prsn)	VA(at prsn)	GCS (at prsn)	ocular mani (1 week)	VA 1 week(re)	le	GCS 1 wk	ocular mani (4 weeks)	VA 4 weeks (re)	le	GCS 4 weeks	ocular mani (6 wks)	VA 6 wks (re)	le	GCS 6 wks
557213	65	2	2	1	a	a	1	f	e	a	0	f	e	a	0	f	e	a
598080	22	1	2	3	a	a	0	a	a	a	0	a	a	a	0	a	a	a
565748	40	1	2	1	a	a	1	a	a	a	0	a	a	a	0	a	a	a
617532	18	1	2	8	a	a	8	a	a	a	0	a	a	a	0	a	a	a
628492	40	1	2	8	a	a	8	a	a	a	0	a	a	a	0	a	a	a
561976	6	1	2	4	c	a	0	a	a	a	0	a	a	a	0	a	a	a
559907	26	2	1	6	a	a	2	a	a	a	2	a	a	a	0	a	a	a
559357	23	1	1	1	a	b	0	a	a	a	0	a	a	a	0	a	a	b
559129	4m	1	3	16	c	c	17	17	17	17	17	17	17	17	17	17	17	17
625769	60	1	3	16	c	c	17	17	17	17	17	17	17	17	17	17	17	17
530046	55	1	1	7	b	a	2	e	e	a	0	e	e	a	0	e	e	a
530410	55	1	1	7	a	a	2	d	c	a	0	d	c	a	0	d	c	a
530533	42	1	1	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
531099	35	1	1	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
531832	50	1	1	1	a	a	0	a	a	a	0	a	a	a	0	a	a	a
531753	35	2	2	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
531378	38	1	2	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
532574	30	1	2	3	a	a	0	a	a	a	0	a	a	a	0	a	a	a
533092	22	1	2	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
534164	50	2	1	10	a	a	8	b	a	a	0	a	a	a	0	a	a	a
535117	40	1	1	2	a	a	18	18	18	18	18	18	18	18	18	18	18	18
535142	6	1	1	3	c	b	18	18	18	18	18	18	18	18	18	18	18	18
531838	30	1	2	13	c	a	0	a	a	a	0	a	a	a	0	a	a	a
539164	40	1	2	1	a	a	0	a	a	a	0	a	a	a	0	a	a	a
535950	28	1	2	1	a	a	1	a	a	a	0	a	a	a	0	a	a	a
535954	19	1	2	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
535955	55	1	2	3	b	a	0	b	d	a	0	b	d	a	0	b	d	a
536347	24	1	2	10	a	a	18	18	18	18	18	18	18	18	18	18	18	18
537358	22	1	2	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
537572	33	1	1	1	a	a	1	a	a	a	b	a	a	a	0	a	a	a
538980	24	1	1	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
539806	40	1	1	2	a	a	2	18	18	18	18	18	18	18	18	18	18	18
540049	11	1	1	13	a	a	0	a	a	a	0	a	a	a	0	a	a	a
541705	60	2	1	1	b	a	1	b	f	a	0	b	f	a	0	b	f	a
542991	30	1	1	1	a	a	1	a	a	a	0	a	a	a	0	a	a	a
543093	75	2	1	1	b	a	0	g	e	a	18	18	18	18	18	18	18	18
543594	7	2	3	13	c	a	18	18	18	18	18	18	18	18	18	18	18	18
544011	24	2	3	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
544259	61	1	3	2	b	a	18	18	18	18	18	18	18	18	18	18	18	18
544423	65	1	1	3	a	a	0	d	f	a	0	d	f	a	0	d	f	a
545676	22	1	1	8	a	a	8	18	18	a	18	18	18	18	18	18	18	18
543093	75	2	3	1	a	a	0	h	g	a	0	h	g	a	0	h	g	a
549931	24	1	2	1	a	a	0	18	18	18	18	18	18	18	18	18	18	18
543424	58	1	2	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
551326	34	1	2	8	a	a	8	a	a	a	c	a	a	a	0	a	a	a
553084	50	1	2	9	a	a	8	18	18	18	18	18	18	18	18	18	18	18
553083	40	1	2	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
553443	55	1	1	1	b	a	18	18	18	18	18	18	18	18	18	18	18	18
553853	62	1	1	15	b	b	15	c	d	b	18	18	18	18	18	18	18	18
555064	35	2	1	1	a	a	0	a	a	a	18	18	18	18	18	18	18	18
555481	28	1	1	3	a	a	0	a	a	a	0	a	a	a	0	a	a	a
593099	18	1	1	12	a	a	9	a	a	a	0	a	a	a	0	a	a	a
593131	28	1	1	6	a	a	2	a	a	a	0	a	a	a	0	a	a	a
593123	35	1	1	8	a	a	8	a	a	a	0	a	a	a	0	a	a	a
593212	32	2	1	7	a	a	1	a	a	a	18	18	18	18	18	18	18	18
594056	7	1	1	13	c	b	0	a	a		0	a	a	a	0	a	a	a
594027	27	1	3	7	a	a	2	a	a	a	0	a	a	a	0	a	a	a
595676	26	1	1	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
596150	39	2	3	6	a	a	0	a	a	a	0	a	a	a	0	a	a	a
596970	24	1	1	11	a	a	2	a	a	a	c	a	a	a	0	a	a	a
597043	24	1	2	3	a	a	0	a	a	a	0	a	a	a	0	a	a	a
597042	21	1	3	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
597655	22	1	1	1	a	a	0	a	a	a	0	a	a	a	0	a	a	a
598067	68	2	1	8	b	a	0	d	c	a	0	d	c	a	0	d	c	a
598057	35	1	1	5	a	a	1	a	a	a	0	a	a	a	0	a	a	a
598928	50	1	1	9	b	a	8	b	c	a	0	b	c	a	0	b	c	a
598920	55	1	3	11	b	a	2	b	d	a	0	b	d	a	0	b	d	a
601027	32	1	3	2	a	a	2	a	a	a	0	a	a	a	0	a	a	a

601127	27	1	3	13	c	b	0	a	a	b	0	a	a	a	0	a	a	b
602964	18	1	3	16	c	c	17	17	17	17	17	17	17	a	17	17	17	17
602984	27	1	1	7	a	a	1	a	a	a	d	a	a	a	0	a	a	a
604968	45	1	1	1	a	a	0	a	a	a	0	a	a	a	0	a	a	a
604963	50	1	1	3	a	a	0	a	a	a	0	a	a	a	0	a	a	a
606361	63	1	1	1	b	a	0	b	d	a	0	b	d	a	0	b	d	a
607607	35	1	1	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
607639	27	1	1	13	a	a	18	18	18	18	18	18	18	18	18	18	18	18
607656	28	1	1	3	a	a	18	18	18	18	18	18	18	18	18	18	18	18
608138	22	1	1	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
607656	24	1	1	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
568799	45	2	2	1	a	a	18	18	18	18	18	18	18	18	18	18	18	18
608529	20	1	2	1	a	a	0	a	a	a	0	a	a	a	0	a	a	a
611936	4	1	2	1	c	a	0	a	a	a	c	a	a	a	18	18	18	18
618743	70	1	2	1	b	a	18	18	18	18	18	18	18	18	18	18	18	18
479280	41	1	2	6	a	a	2	a	a	a	c	a	a	a	18	18	18	18
619156	48	1	1	16	b	b	16	c	c	c	17	17	17	17	17	17	17	18