EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE

REGIONAL INSTITUTE OF OPHTHALMOLOGY AND
GOVERNMENT OPHTHALMIC HOSPITAL
EGMORE, CHENNAI

Dissertation Submitted In Partial fulfilment
of the requirements for
M.S. DEGREE EXAMINATION
BRANCH III – OPHTHALMOLOGY

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY
MADRAS MEDICAL COLLEGE , CHENNAI
APRIL 2017

CERTIFICATE

This is to certify that this dissertation titled **" EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE "** is a bonafide original research work carried out by **DR. KEERTHANA J** , Post Graduate in the Regional Institute of Ophthalmology & Government Ophthalmic Hospital, Madras Medical College, Chennai in partial fulfilment of the regulations laid down by The Tamilnadu Dr.M.G.R Medical University for the award of M.S. Ophthalmology Degree - Branch III under my guidance and supervision during the academic year from 2014-2017.

Prof. Dr. Waheeda Nazir M.S., D.O.,
Chief, Orbit and Oculoplasty Services,
Regional Institute of Ophthalmology,
Madras Medical College,
Chennai – 600 008.

Prof.Dr. Waheeda Nazir M.S., D.O.,
Director and Superintendent,
Regional Institute of Ophthalmology,
Madras Medical College,
Chennai – 600 008.

Prof. Dr. M. K. Muralidharan., M.S., M.CH
Dean, Madras Medical College,
Rajiv Gandhi Government General Hospital,
Chennai – 03.

ACKNOWLEDGEMENT

I am grateful to **DR. M.K. Muralidharan M.S., M.Ch.,** Dean, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai for permitting me to do this study.

I have great pleasure in thanking **Prof Dr.Waheeda Nazir**, **M.S., D.O**., my Guide, Unit Chief, Director And Superintendent, Regional Institute Of Ophthalmology & Government Ophthalmic Hospital, Chennai for having assigned me this very interesting topic and her valuable guidance and immense support for doing this study.

I am grateful to my Co-Guides **Asst Prof Dr. K.S.T.Latha M.S.,** and **Dr.Vasumathy M.S.,** for their valuable guidance and support for this study.

My sincere thanks to **Prof Dr. Rajavelu Indhira M.D.,** Deparment Of Pathology, RIO-GOH, for the continued support during the course of the study.

My sincere thanks to **Prof Dr.Dharmender M.D.,** Deparment of Radiology, RIO-GOH, for the continued support during the course of the study.

I am extremely thankful to my Assistant professors **Dr Anuradha M.S.,** and **Dr.Sujatha M.S.,** for rendering their valuable suggestions and constant support for this study.

I would like to thank my colleagues, friends & family who have stood by me throughout the study. Finally I am indebted to all the children and their parents for their sincere cooperation for the completion of this study and above all the God almighty for his blessings.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.Keerthana.J.
Post Graduate in M.S. Ophthalmology
Madras Medical College
Chennai 600 003

Dear Dr.Keerthana.J.

The Institutional Ethics Committee has considered your request and approved your study titled **"EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS – A CLINICAL STUDY IN A TERTIARY CARE CENTRE " - NO.31032016.**

The following members of Ethics Committee were present in the meeting hold on **01.03.2016** conducted at Madras Medical College, Chennai 3

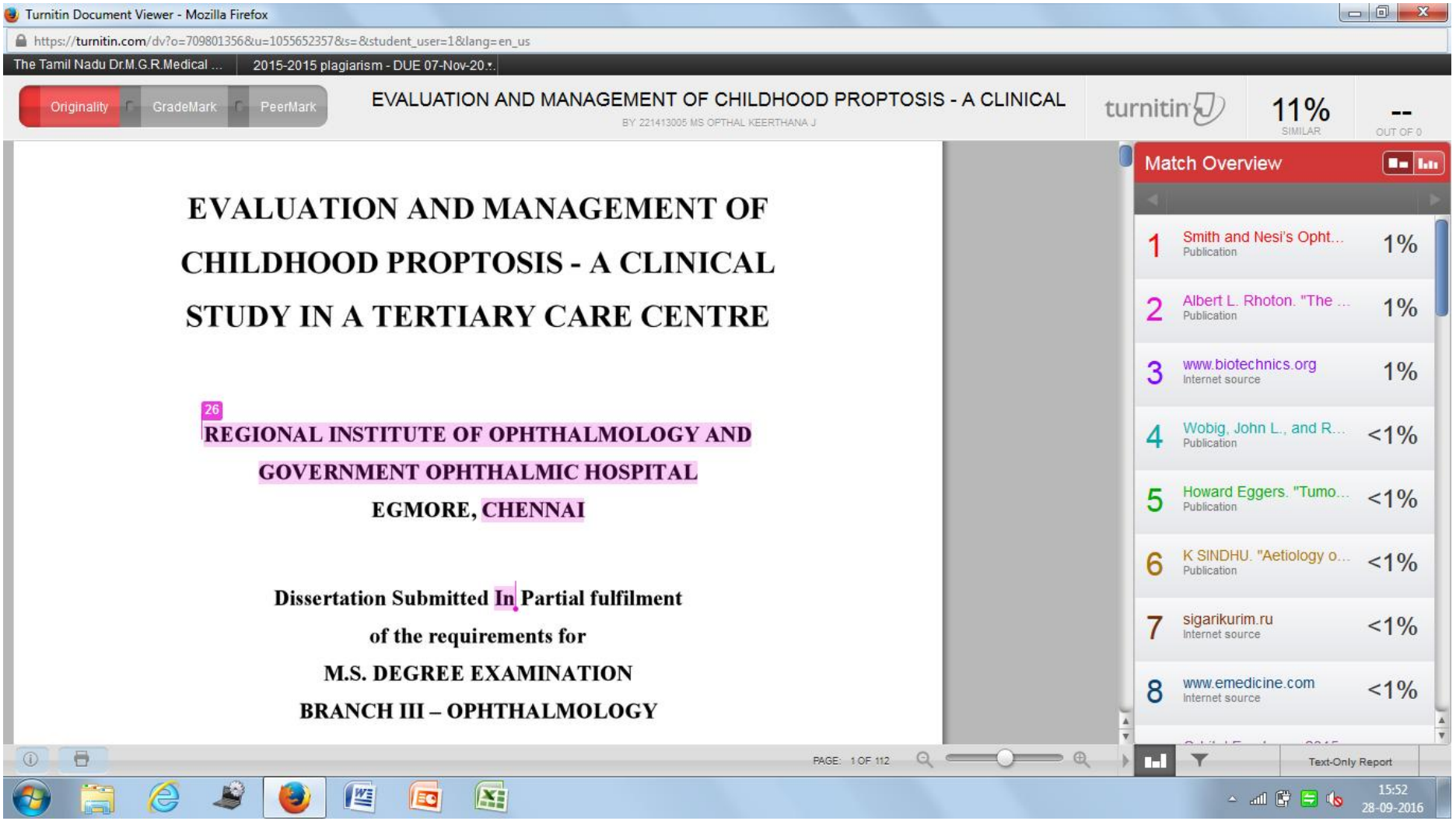
1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD.,Inst.of Pharmacology,MMC,Ch-3	: Member
5.Prof.P.Raghumani,MS, Dept.of Surgery,RGGGH,Ch-3	: Member
6.Dr.Baby Vasumathi, Director, Inst. of O&G,Ch-8	: Member
7.Prof.M.Saraswathi,MD.,Director, Inst.of Path,MMC,Ch-3	: Member
8.Prof.Srinivasagalu,Director,Inst.of Int.Med.,MMC,Ch-3	: Member
9.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
10.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
11.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE.
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

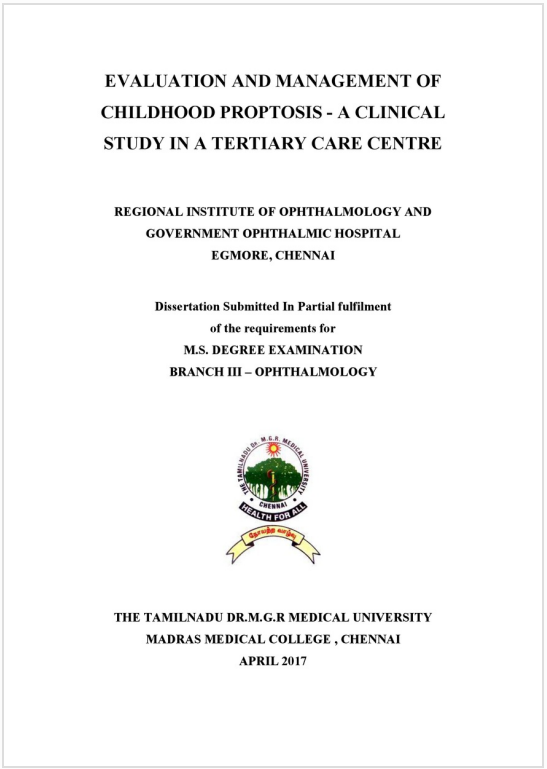

Turnitin Document Viewer - Mozilla Firefox
https://turnitin.com/dv?o=709801356&u=1055652357&s=&student_user=1&lang=en_us
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-20...
Originality
GradeMark
PeerMark
EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL
BY 221413005 MS OPTHAL KEERTHANA J
turnitin
11%
SIMILAR
--
OUT OF 0
EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE
26
REGIONAL INSTITUTE OF OPHTHALMOLOGY AND GOVERNMENT OPHTHALMIC HOSPITAL
EGMORE, CHENNAI
Dissertation Submitted In Partial fulfilment
of the requirements for
M.S. DEGREE EXAMINATION
BRANCH III – OPHTHALMOLOGY
Match Overview
1 Smith and Nesi's Opht... Publication 1%
2 Albert L. Rhoton. "The ... Publication 1%
3 www.biotechnics.org Internet source 1%
4 Wobig, John L., and R... Publication <1%
5 Howard Eggers. "Tumo... Publication <1%
6 K SINDHU. "Aetiology o... Publication <1%
7 sigarikurim.ru Internet source <1%
8 www.emedicine.com Internet source <1%
PAGE: 1 OF 112
Text-Only Report
15:52
28-09-2016

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221413005 Ms Opthal KEERTHAN…
Assignment title:	2015-2015 plagiarism
Submission title:	EVALUATION AND MANAGEMENT…
File name:	Whole_Dissertation.docx
File size:	4.78M
Page count:	112
Word count:	12,918
Character count:	74,532
Submission date:	28-Sep-2016 12:24PM
Submission ID:	709801356



EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE

REGIONAL INSTITUTE OF OPHTHALMOLOGY AND GOVERNMENT OPHTHALMIC HOSPITAL EGMORE, CHENNAI

Dissertation Submitted In Partial fulfilment of the requirements for M.S. DEGREE EXAMINATION BRANCH III – OPHTHALMOLOGY

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY MADRAS MEDICAL COLLEGE , CHENNAI APRIL 2017

DECLARATION BY THE CANDIDATE

I **Dr. Keerthana J**, hereby solemnly declare that this dissertation entitled **" EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE "** is a bonafied and genuine work carried out by me under the guidance and supervision of **Prof. Dr. Waheeda Nazir M.S., D.O.,** in the Regional Institute Of Ophthalmology, Madras Medical College, Chennai.

This dissertation is submitted to The **Tamilnadu Dr.M.G.R Medical University, Chennai** in partial fulfilment of the university rules and regulations for the award of the degree of **M.S Ophthalmology.**

Date:

Place : **Dr. Keerthana J**

CONTENTS

INTRODUCTION

Proptosis or exophthalmos is defined as protrusion of the globe beyond the orbital margins, with the patient looking straight ahead . Henderson[1] reserves the use of word exophthalmos for those cases of proptosis secondary to endocrinological dysfunction. Proptosis is the common presenting symptom of wide variety of diseases affecting the structures present in and around the orbit.

The orbits are a pair of bony cavities in the skull situated on either side. The orbit houses the eyeball and sub-serves the major function of protection of the eye. The orbit is pear shaped cavity, the stalk of which is the optic canal. The fact that the orbit is in intimate relationship with the cranial cavity, the nose and the para-nasal sinuses around it makes it vulnerable to many disorders that might involve the orbit.

Proptosis is the hall mark of orbital diseases. A lesion in the intra-conal region produces axial proptosis and lesions in the extra-conal region produces eccentric proptosis. A difference of more than 2 mm between the two eyes of any given patient is considered abnormal. The intra orbital portion of the optic nerve is longer (25mm) than the distance between the back of the globe and optic canal (18mm). This allows for significant forward displacement of the globe (i.e) proptosis.

The etiological basis of proptosis can be congenital, inflammatory, vascular, neoplastic, endocrinological and trauma. Proptosis occurs in both adults and children at any age. The symptoms and signs of proptosis are

manifested by displacement of the globe and are associated with local pain, redness and swelling. It is often accompanied by decreased vision, double vision, optic disc swelling and impaired ocular movements. Early diagnosis and management is the key to address the issue. The services of an ENT surgeon, Neurosurgeon and a Physician are sought whenever necessary and a thorough systemic examination should be done.

A conclusion can be arrived clinically noting the age of the patient, duration and presentation of proptosis, direction of proptosis, associated signs and symptoms and also by excluding certain diseases by the absence of their typical presentations. A final diagnosis may be possible in many cases only after the laboratory, radiological and pathological investigations.

LITERATURE REVIEW

- **Downie et al**[2] in 1998 conducted a retrospective study among 57 cases of children with proptosis presenting to The Children's Hospital, Hospital, Camperdown, Sydney showed that infective orbital cellulitis was the most common cause of childhood proptosis The most useful initial investigation was an orbital computed tomography scan. Treatment depended on the cause of the proptosis and was multidisciplinary.
- **Loganathan et al**[3] in 2014 in a study on childhood Proptosis conducted among 50 cases showed that Orbital cellulitis is the single most common cause for proptosis in children. Secondaries are common than the primary tumours in the orbit among malignancies. Orbital X-ray, B-Scan, CT Scan and MRI were helpful in diagnosis, treatment and follow up**.**
- **Chaudhry et al**[4] showed that pathological diagnosis remains the gold standard in diagnosis of orbital diseases when imaging studies are inconclusive and tissue biopsy provides the ultimate diagnosis.
- **Altonbary Y et al** showed that sub-acute and chronic cases of childhood proptosis should arise the suspicion of malignancy. They reported four cases with proptosis caused by different malignant lesions retrieved from Paediatric Oncology Unit, Oncology Centre of Mansoura University. They also concluded that invasive diagnostic tests should be preceded by non-invasive tests such as CBC, LDH, VMA and imaging studies.

- In 2016 **Snehal R Thakre et al**[5] from MGM Medical College, Aurangabad, Maharashtra, India reported a case wherein a child with sub-acute , bilateral and asymmetric Proptosis was diagnosed as pseudo-tumour based on imaging studies and was started on systemic steroids. But after a initial period of response there was recurrence. Later on peripheral blood smear and bone marrow biopsy confirmed it to be acute myeloid leukemia. They concluded that extra-ocular causes of proptosis should be remembered while dealing with a childhood proptosis.
- In 2001 **Sethi a et al**[6] cited in an article that basic blood investigations such as peripheral smear will clinch the diagnosis of proptosis before ordering for imaging and biopsy.
- In 2015 **Porubanova M,** et al stated that in case of orbital and peri-ocular capillary infantile haemangioma beta-blockers, both propranolol and metoprolol seems to be very effective in reduction of the tumour size.
- In 2013 **Sah KP, Saiju R et al**[7] described about the incidence, laterality, signs, age at presentation and outcome of retinoblastoma in a group of 42 children with retinoblastoma.
- In 1998- 2002 **Bhurgri Y** stated the higher prevalence of juvenile Graves' hyperthyroidism among smoking teenagers and also stated that the Anti-thyroid drugs remain the treatment of choice.

- **In 1997 Wright[8] JE** mentioned that orbital venous anomaly have features of both normal orbital veins and lymphatics and should be managed conservatively as possible.
- **Bakshi[9]** in 2008 conducted study among 104 cases of malignant childhood Proptosis wherein study concluded that careful analysis of symptomatology, haematological workup is mandatory before imaging studies and invasive orbital biopsy.
- In 1999 a study conducted by **Belmekki M[10]** revealed that retinoblastoma was the predominant cause of childhood Proptosis among Moroccan children.

ANATOMY OF THE BONY ORBIT

The eyes lie in two bony cavities of the skull situated on either side of the nose, the orbits Fossae orbitales[11]. The orbit resembles a quadrilateral pyramid with its base directed forwards outwards and slightly downwards. Posteriorly the cavity narrows to an apex of a triangular pyramid wherein the optic nerve and the muscles lie. The change in configuration from a quadrangular to a triangular pyramid is due to the failure of the floor of the orbit, to reach the apex. (Fig. 1)

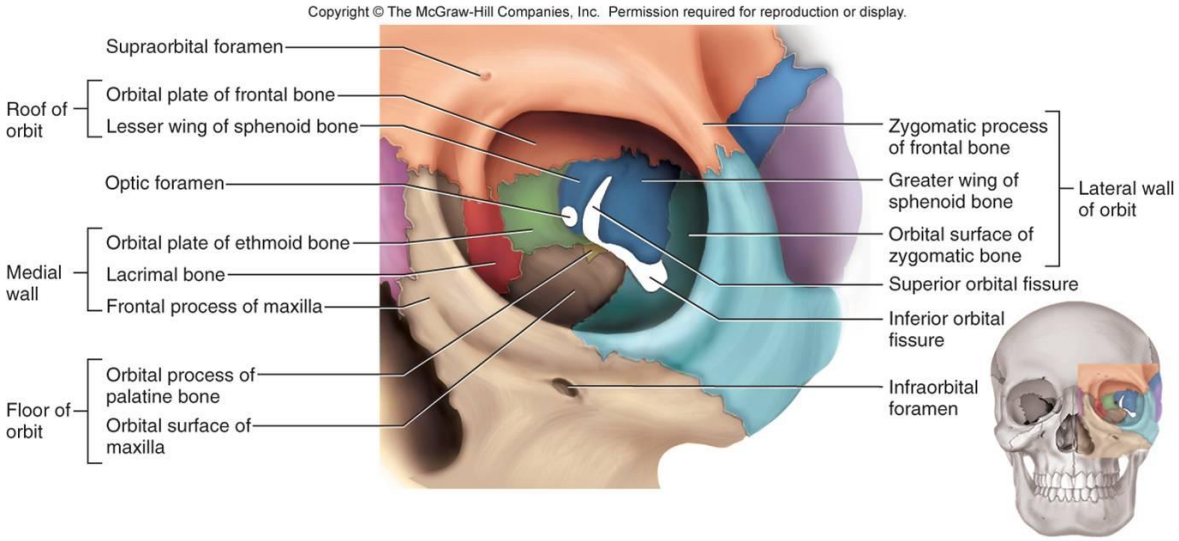


Fig .1 Anatomy of orbit

EMBRYOLOGY OF THE ORBIT

The bony walls of the orbit are formed from the mesoderm surrounding the developing eye. The floor and lateral wall of the orbit are derived from the visceral mesoderm of the maxillary process, the roof is developed in the paraxial mesoderm forming part of the capsule covering the forebrain; while the medial wall is developed from the lateral nasal process. All the orbital contents, its muscles, fascia and vessels as well as the mesodermal stroma of the globe are developed from the paraxial mesoderm[12]. Ossification centres for orbital bones appear between the 6th and 7th week of embryonic life.

ORBITAL WALLS

The orbital walls are composed of seven bones.

1. Ethmoid
2. Frontal
3. Lacrimal
4. Maxillary
5. Palatine
6. Sphenoid
7. Zygomatic

THE ROOF (Vault of the Orbit)

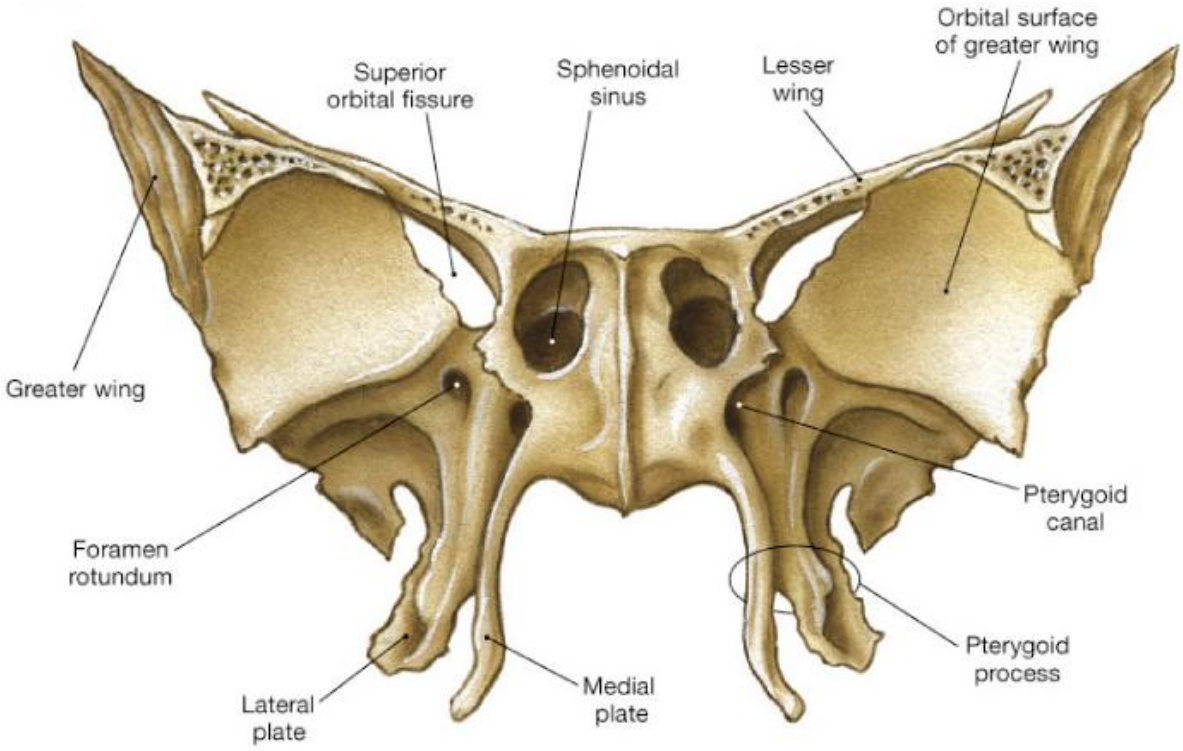


Fig.2 Roof of the orbit

It is approximately triangular in shape with the base placed anteriorly. It faces downwards and slightly forwards with concave anteriorly and flatter posteriorly. The roof is formed largely by the triangular orbital plate of the frontal bone, and behind this by the lesser wing of the sphenoid[13]. The fossa for the lacrimal gland lies in the anterolateral aspect of the roof. It contains not only the lacrimal gland but orbital fat, principally at its posterior (accessor fosa of Rochon-duvingneaud). Trochlear fossa situated in the anteromedial aspect of the roof above the frontolacrimal suture, 4 mm behind the orbital margin. The

fibrous pulley of superior oblique is attached to this fossa. The frontosphenoidal suture is usually obliterated in the adult, this suture lies between the orbital plate of the frontal bone and the lesser wing of the sphenoid. The orbital aspect of the roof is marked by small apertures and are marked in infants and children. The openings in the anterior part near the trochlear fossa transmit diploic veins. The foramina in and around lesser wing of sphenoid transmit vessels connecting dural veins and ophthalmic veins. **(Fig.2)**

RELATIONS

The frontal nerve is in contact with the periorbita along the whole roof. The supraorbital artery accompanies only in the anterior half. Inferior to both are levator palpebrae and the superior rectus. The trochlear nerve lies medially, in contact with the perorbita. The lacrimal gland adjoins the lacrimal fossa and the superior oblique the junction of roof and medial wall. Invading the roof to a variable extent are the frontal and ethmoidal sinuses. Above the roof are the frontal lobe of the cerebrum and its meninges.

THE MEDIAL WALL OF THE ORBIT

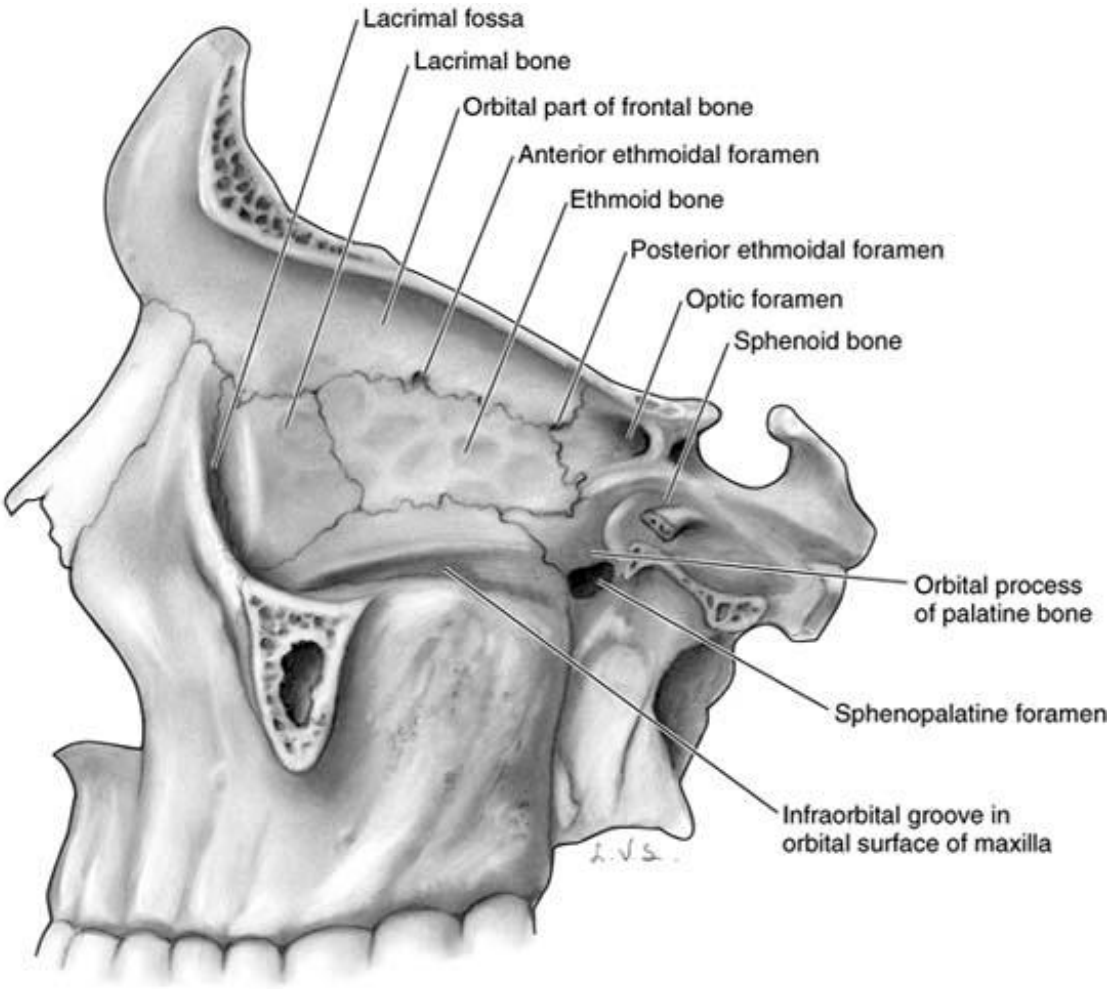


Fig.3. Medial wall of the orbit

The oblong medial wall is the thinnest (0.2 to 0.4 mm) and it is formed by four bones namely :

1. The frontal process of the maxilla
2. The lacrimal bone
3. The orbital plate of ethmoid
4. A small lateral aspect of the body of the sphenoid.

Anteriorly is the lacrimal fossa, formed by the frontal process of the maxilla and the lacrimal bone[14]. It is bounded by the anterior and posterior lacrimal crests. About 20 mm behind the anterior medial orbital margin is the anterior ethmoid foramen, and 12 mm behind this, the posterior ethmoid foraman, which is 5-8 mm from the optic canal.(Fig.3)

RELATIONS

Medially the lateral nasal wall, infundibulum, ethmoidal sinuses and sphenoidal air sinus. The optic foramen is located at the posterior end of the medial wall. The superior oblique muscle is in the angle between roof and medial wall.

THE FLOOR OF THE ORBIT

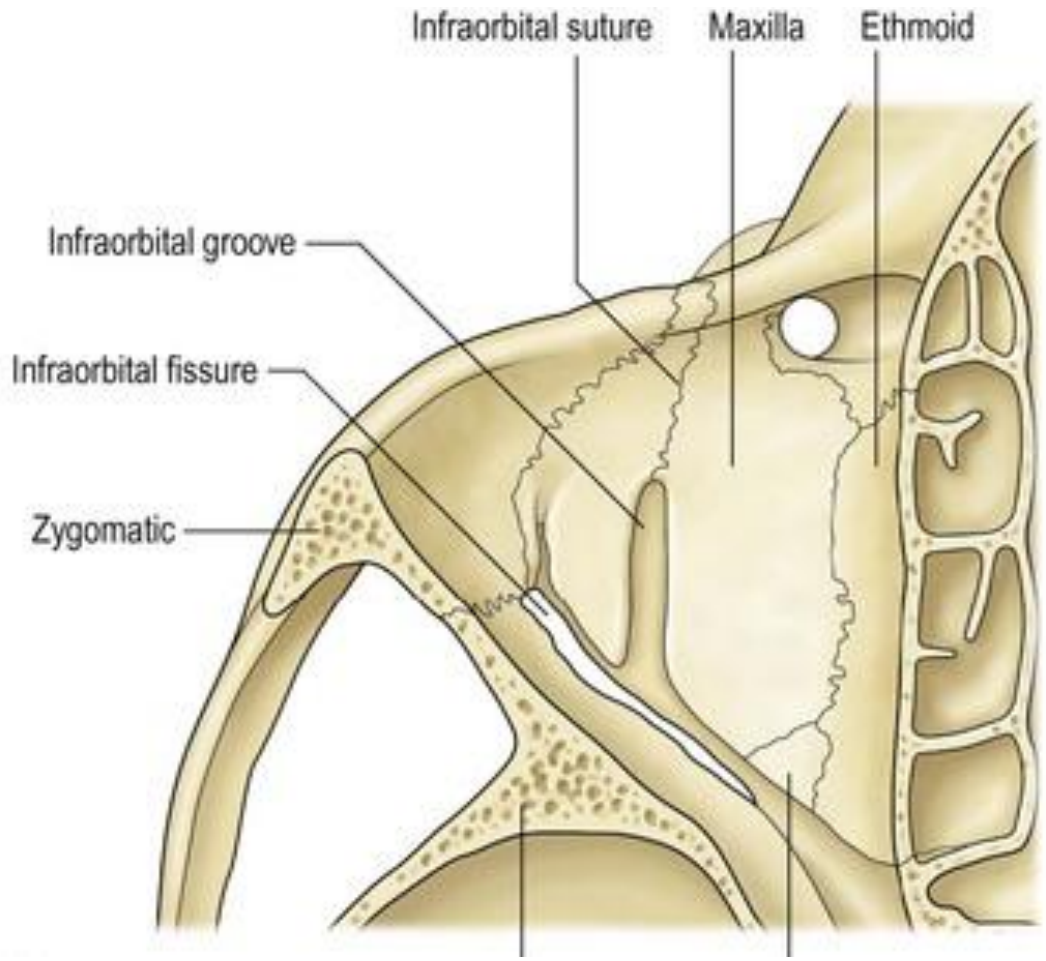


Fig. 4 Floor of the orbit

The infraorbital canal descends in the orbital floor to open at the infraorbital foramen about 4 mm below the orbital margin[15]. Posteriorly the lateral wall is separated from the floor by inferior orbital fissure (sphenomaxillary fissure). The floor is traversed by the infraorbital sulcus which runs forwards from the inferior orbital fissure. (Fig.4)

RELATIONS

Below most of the floor of the orbit is the maxillary sinus. The inferior rectus adjoins the floor near the apex of the orbit, but it is separated from it anteriorly by the inferior oblique muscle and fat. At the lateral edge of the inferior rectus, or between it and the lateral rectus, is the nerve to inferior oblique. The inferior oblique arises at the lateral edge of the opening of the nasolacrimal canal and passes posterolaterally and up near the floor. The infraorbital vessels and nerve occupy their sulcus canal.

INFERIOR ORBITAL FISSURE

It is situated at the junction of the lateral wall and floor. It is a gap bounded by maxilla and orbital process of palatine bone anteromedially and greater wing of the sphenoid posterolaterally and called the Spheno-Maxillary fissure. The inferior ophthalmic vein passes through its lower portion before entering the cavernous sinus.

THE LATERAL WALL OF THE ORBIT

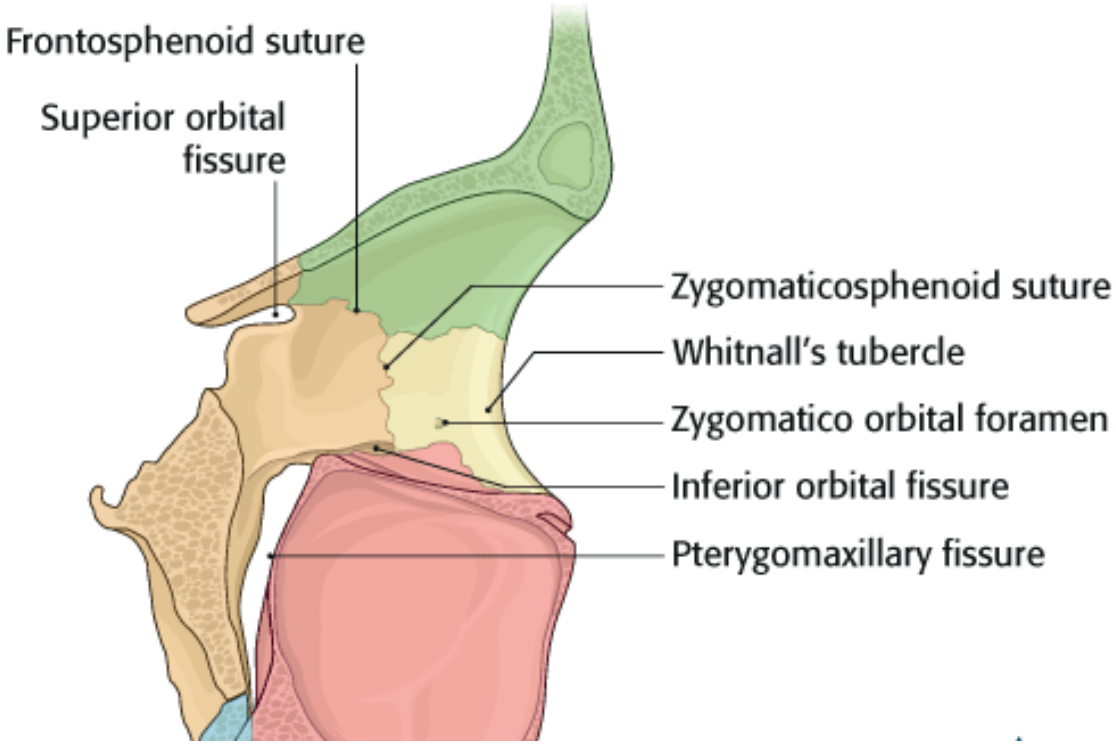


Fig.5 Lateral wall of the orbit

The lateral wall is the thickest among the four walls. It is triangular in shape with the base of the triangle placed anteriorly. It is directed inwards, forwards and slightly upwards and forms an angle of about 45° with the median sagittal plane[16]. The lateral wall of the orbit is formed by two bones

(i) Anteriorly by the orbital surface of the Zygomatic bone

(ii) Posteriorly by the orbital surface of the greater wing of the sphenoid.

The lateral orbital tubercle (Whitnall) is a small elevation on the orbital surface of the Zygomatic bone behind the lateral orbital margin and about 1 mm below the frontozygomatic suture. (**Fig.5**)

It gives attachment to (i) the check ligament of the lateral rectus muscle (ii) The suspensory ligament of the eye ball (iii) The aponeurosis of the levator palpebrae superiors (iv) The lateral palpebral ligament.

RELATIONS

The lateral wall separates the orbit anteriorly from the temporal fossa and muscle, posteriorly from the middle cranial fossa and temporal lobe of the cranium. The lateral rectus muscle is in contact with the whole of this wall, with the lacrimal nerve and artery above it. The inferior pole of the lacrimal gland reaches the lateral wall, where the lacrimal nerve receives a parasympathetic branch from the zygomatic, which with its vessels, also adjoins the wall.

SUPERIOR ORBITAL FISSURE (SPHENOIDAL FISSURE)

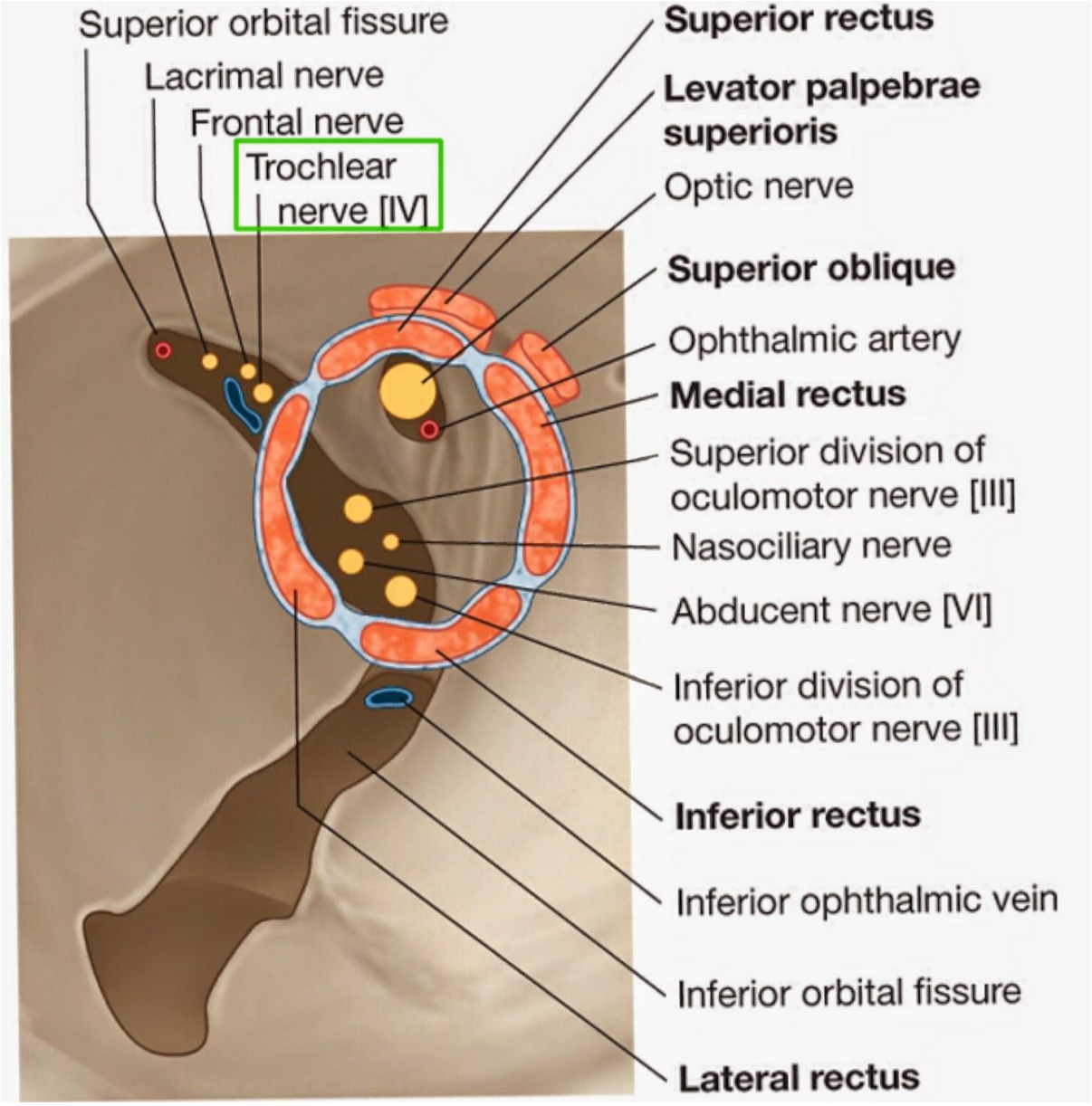


Fig.6 Superior orbital fissure

This connects the orbit and the middle cranial fossa and it is situated at the junction of the roof and the lateral wall. This is a gap between the lesser and greater wings of the sphenoid. It measures 22 mms in length[17]. The fissure is wide medially and narrow laterally (retort or comma shaped). The common tendinous ring of Zinn divides the fissure in to three parts. (i) The part lateral to the ring transmits lacrimal, frontal, trochlear nerves and superior ophthalmic vein and occasionally an arterial anastomosis between branches of middle meningeal artery and lacrimal artery. (ii) The intermediate part is called oculomotor foramen, it transmits superior division of oculomotor nerve, nasociliary, and a sympathetic twig to ciliary ganglion. (iii) The part below and medial to the ring transmits the inferior ophthalmic vein. (**Fig.6**)

THE RELATIONS OF THE ORBIT

Superiorly - Anterior cranial fossa

- Frontal Sinus

- Supra orbital sinuses

Inferiorly - Maxillary antrum

- Palatine air cells

Medially - Ethmoid sinus, Nasal cavity, Sphenoid Sinus (Posteriorly)

Laterally - Middle cranial fossa, Temporal fossa, Pterygopaltine fossa.

THE ORBITAL CONTENTS

While the globe of the eye occupies most of the space of the anterior segment of the orbit, the greater part of its posterior segment occupied by orbital fat and the functionally important tissues like muscles, blood vessels and nerves occupy a comparatively small volume.

The important orbital contents:

1. Muscles
2. Orbital fascia
3. Surgical spaces
4. Blood vessels
5. Nerves
6. Others

1. The Muscles Of The Orbit

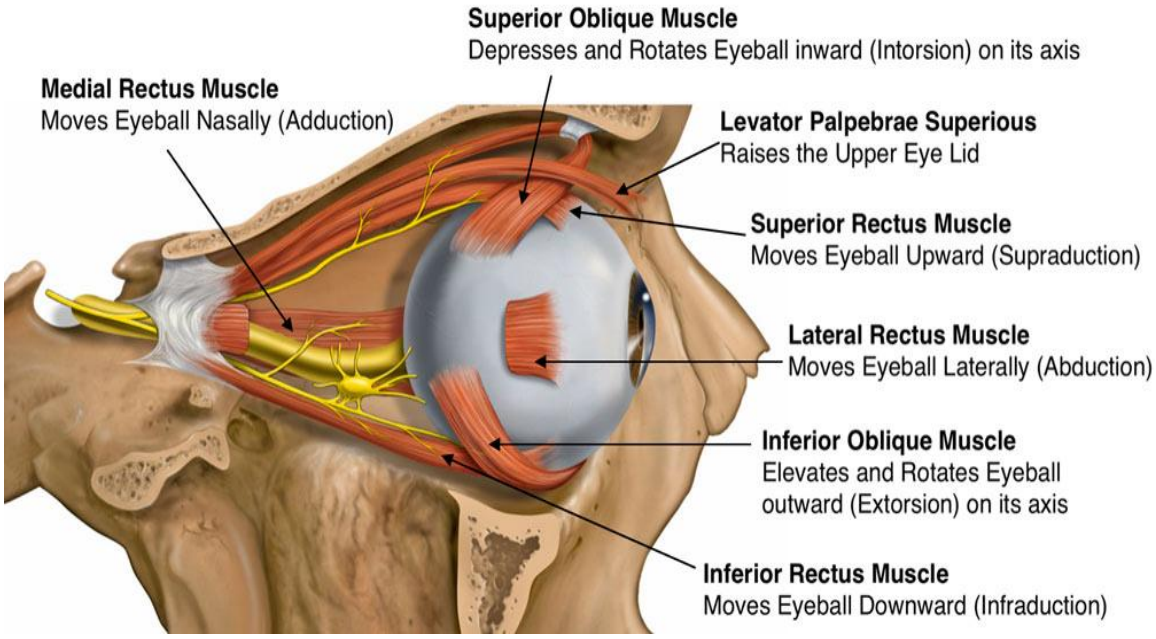


Fig.7 Muscles of the orbit

The muscles of the eyeball (**Fig.7**)

i. Extrinsic muscles of the eye ball

- Recti : lateral, medial, superior and inferior
- Obliques : superior and inferior

ii. Muscles of the lids, levator palperbrae superiors

iii. Plain muscles of the orbit

- Orbital muscle (of muller)
- Periorbital muscle

2. The Orbital Fascia

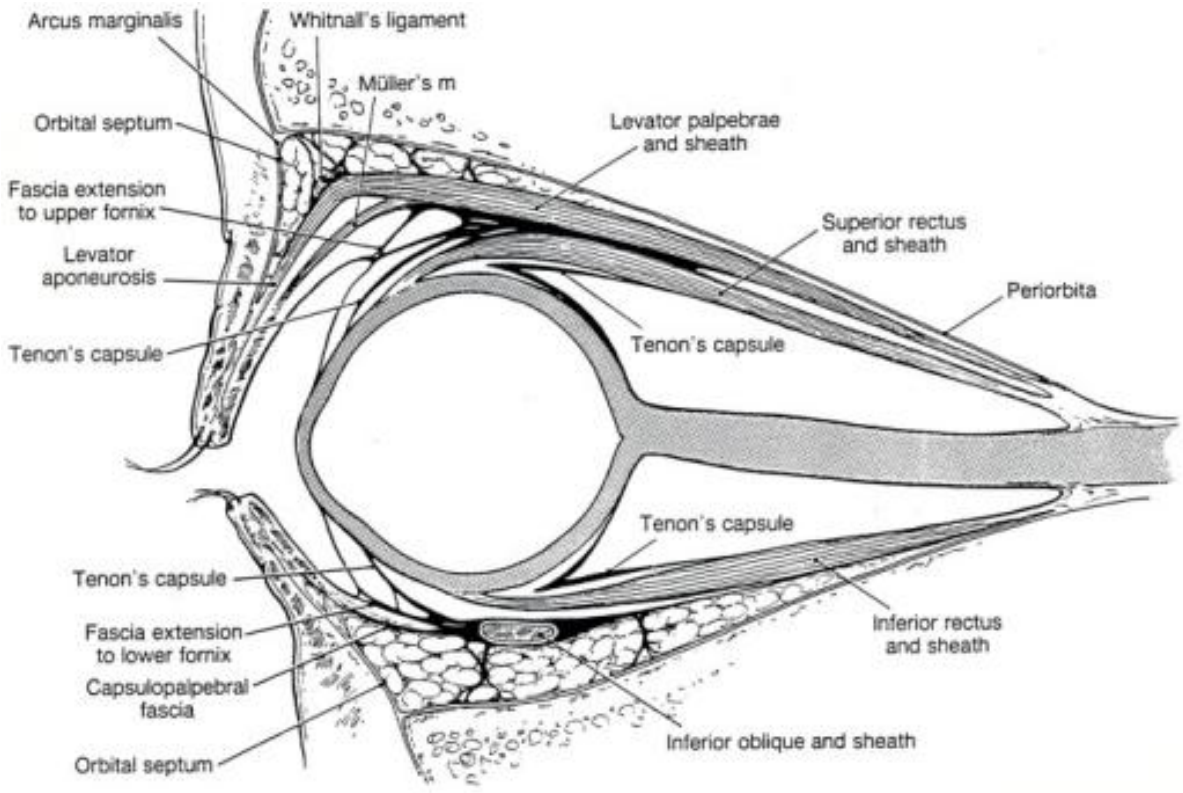


Fig.8 The orbital fascia

The orbital fascia comprises (**Fig.8**)

i. The fascia bulbi (Tenons capsule / Tunica Vaginalis occuli)

ii. The fascial sheaths of the muscles

iii. The check ligaments of the muscles

iv. The connective tissue supporting the orbital fat

v. The periorbital membrane (periosteum which lines the orbital cavity)

vi. The orbital septum.

3. Surgical Spaces of the Orbit

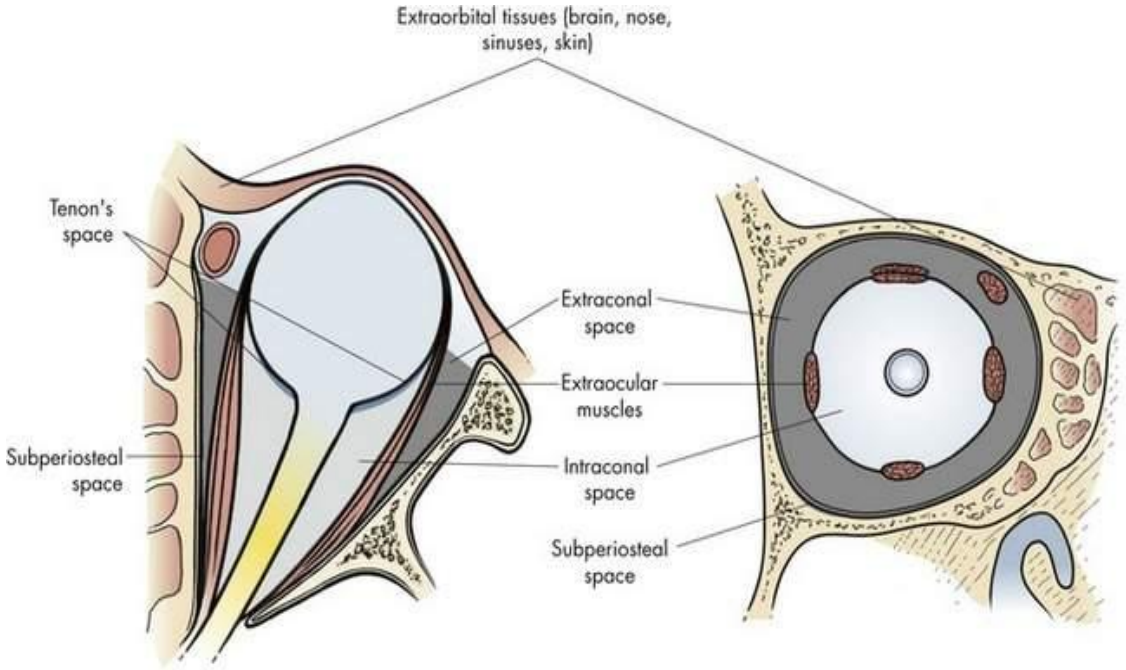


Fig.9 Surgical spaces of the orbit

There are four surgical spaces

i. The Episcleral (Tenon's) space –

lying between the Tenon's capsule and the globe.

ii. The central surgical space – lies within the muscle cone

iii. The peripheral surgical space –

lies between the partition formed by the muscles and the intermuscular membrane internally and the periosteum externally.

iv. The subperiosteal space –

potential space between the periosteum and the bone. (**Fig.9**)

4. Blood vessels of the orbit

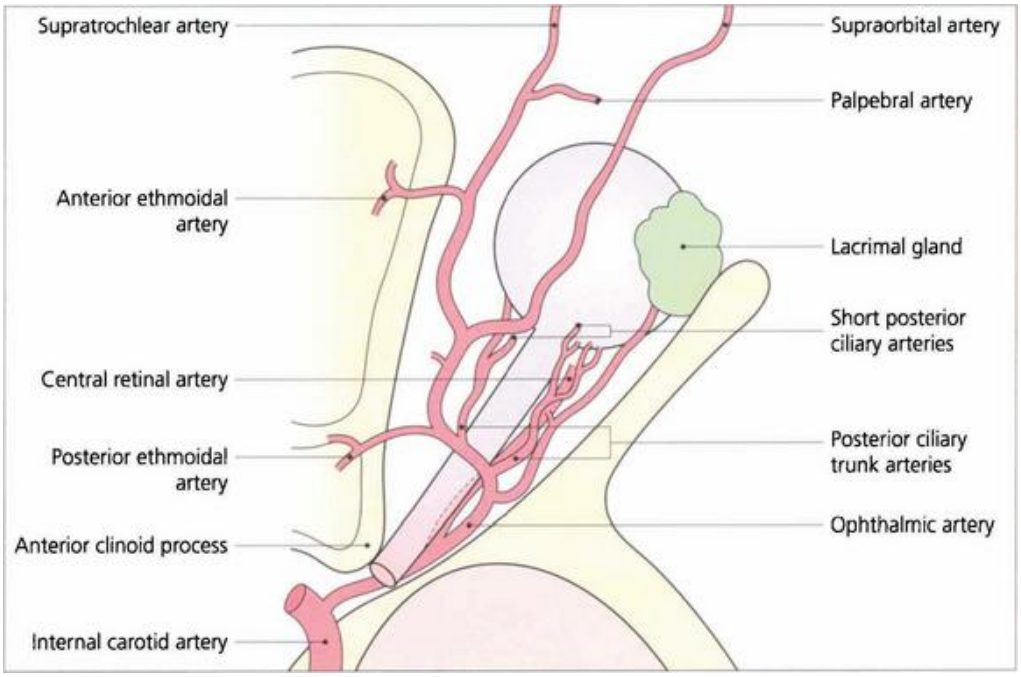


Fig.10 Arterial supply of the orbit

i. Arterial circulation[18] :

• Internal Carotid artery – gives off the ophthalmic artery

• External Carotid artery -

A. Internal maxillary artery through the infraorbital artery

B. Middle meningeal artery through the orbital branch

ii. Venous Circulation:

There are three main veins within the orbit

(i) Superior ophthalmic vein

(ii) Inferior ophthalmic vein

(iii) Central retinal vein. (**Fig.10**)

The superior ophthalmic vein is formed by the union of the supraorbital and angular vein of the face. It receives branches from large ethmoid branches, from the face, two superior vortex veins and the lacrimal vein. The inferior ophthalmic vein begins as a venous network on the orbital floor, receiving branches from the lower lid, the region of the lacrimal sac, the inferior rectus and inferior oblique muscles and the two inferior vortex veins.

iii. The Lymphatics

In the orbit there are no lymph nodes or lymphatic vessels have been demonstrated. Probably the main lymph drainage from the orbit accompanies the veins through the inferior orbital fissure to the internal maxillary nodes then to the superior deep cervical nodes.

5. Nerves of the Orbit

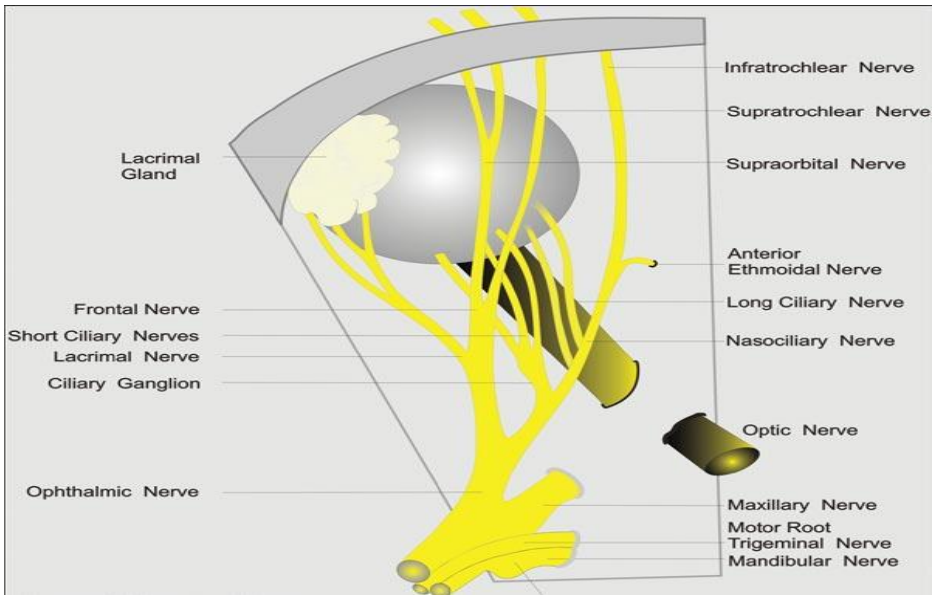


Fig.11 Nerves of the orbit

i. Optic nerve – the nerve of vision

ii. Cranial nerves III, IV, VI : Motor and parasympathetic fibres to the extra ocular muscles and the LPS and to the intra-ocular muscles

iii. 1st and 2nd division of cranial nerve V : Sensory to eye ball, the lacrimal gland, the conjunctiva, the lids and large areas of the surrounding skin of the face, as well as conveying parasympathetic fibres (**Fig.11**)

iv. Cranial nerve VII : Parasympathetic fibres essentially to the lacrimal gland.

v. Sympathetic : To the eye ball, lacrimal gland, the orbital plain (and striated) muscle and vasomotor to the orbit.

6. Other structures

- ❖ Lacrimal gland,
- ❖ Lacrimal sac and
- ❖ Orbital fat.

CLASSIFICATION OF PROPTOSIS

1. Proptosis may be acute or chronic.
2. It may be unilateral or bilateral.
3. It may be axial or eccentric[19].
4. It may be classified based on the aetiology.

AXIAL PROPTOSIS

- AV Malformations, Metastatic tumours
- Optic nerve glioma/ meningioma/ neurilemmoma,

ECCENTRIC PROPTOSIS

- **Downward:** Neurofibroma, Lymphoma, Thyroid ophthalmology, Frontal mucocele, Neuroblastoma, Schwannoma
- **Down and in:** Lacrimal gland tumour, Dermoid, Pseudotumour
- **Up ward:** Maxillary sinus tumour, Lymphoma, Lacrimal sac tumour, Metastatic tumour
- **Lateral:** Ethmoidal mucocele, Nasopharyngeal tumour, Midline Granuloma, Lacrimal sac tumour, Metastatic tumour
- **Medial:** Lacrimal fossa tumour, Sphenoid wing meningioma.

CAUSES OF CHILDHOOD PROPTOSIS

- CONGENITAL
- INFLAMMATORY
- VASCULAR
- NEOPLASTIC
- BENIGN
- MALIGNANT
- ENDOCRINE
- TRAUMATIC

1. CONGENITAL

- Malformations – Orbital , Ocular, Vascular
- Hamartoma – Capillary and Cavernous Hemangioma, Lymphangioma
- Choristoma – Dermoid cyst, Other Epithelial cyst
- Meningocele and Encephalocele[20]

2. INFLAMMATORY AND INFECTIOUS

- Orbital cellulitis
- Sinus mucocele and sinusitis
- Orbital abscess
- Osteomyelitis
- Cavernous sinus thrombosis

- Pseudotumour
- Parasitic cyst

3. VASCULAR

- Capillary haemangioma
- Cavernous hemangioma
- Lymphangioma
- Sturge -Weber syndrome
- Carotid – Cavernous fistula
- Orbital varix
- A-V Malformations

4. NEOPLASTIC

a. Benign

- Dermoid
- Epidermoid
- Teratoma
- Haemangioma
- Lymphangioma
- Neurofibroma
- Fibroma, Lipoma
- Rhabdomyoma
- Osteoma, Chondroma

- Neurilemmoma
- Meningioma
- Optic nerve glioma
- Eosinophilic granuloma
- Fibrous dysplasia
- Ossifying fibroma
- Juvenile fibromatosis

b. Malignant (Primary and Metastatic)

- Rhabdomyosarcoma
- Juvenile angiofibroma of nasophaynx
- Osteosarcoma, Chondrosarcoma
- Neurofibrosarcoma
- Metastatic neuroblastoma
- Extraocular Retinoblastoma
- Leukemic infiltration (chloroma)
- Lymphoma (Burkitt's Lymphoma)
- Malignant melanoma
- Ewing sarcoma, Wilms tumour
- Malignant histiocytosis
- Intracranial tumour involving orbit

5. METABOLIC AND DISEASE INVOLVING BONE :

- Grave's disease,
- Myxoedema
- Acromegaly
- Osteopetrosis
- Infantile cortical hyperostosis
- Fibrous dysplasia
- Histocytosis – X

6. TRAUMATIC

- Birth injuries
- Orbital Hematoma
- Orbital Emphysema
- Traumatic aneurysm
- Carotid cavernous fistula
- Encephalocele

PSEUDO PROPTOSIS

- Craniosynostosis
- Congenital Buphthalmos
- High axial myopia
- Staphyloma
- Congenital deformity - microblepharon

DESCRIPTION OF CAUSES

1. CONGENITAL AND DEVELOPMENTAL ANOMALIES

a. CRANIOFACIAL DYSOSTOSIS

Proptosis in this condition is the result of shallow orbit due to anterior displacement of greater wing of sphenoid[21]. In Crouzon's (Fig.12) and Apert's diseases ocular complications are related to coronal stenosis[22]. Papilledema, optic atrophy and Strabismus are the other features. Other syndromes causing proptosis are Gruber's syndrome, Turner's syndrome , Woife's syndrome , Morquio's disease .

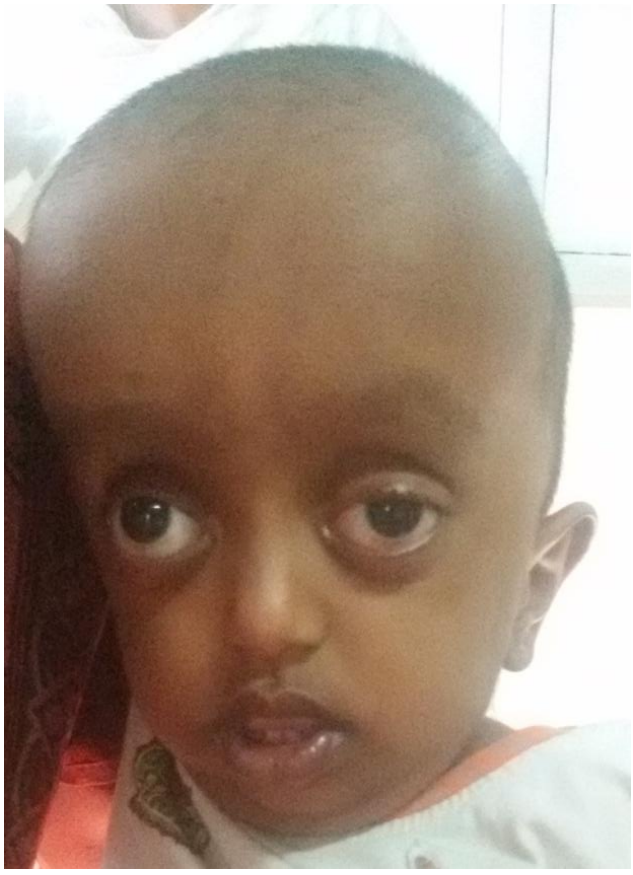


Fig.12 Pseudoproptosis in Crouzon's syndrome

b. TERATOMA

Contains multiple tissues which are representatives of more than one germinal layer. In addition to structures derived from ectoderm, tissues derived from endoderm and mesenchyme may be present. The cystic nature of these lesions is usually due to the presence of epidermoid cysts and embryonic formations of mucin secreting gastro intestinal mucosa. Other cysts may be limited by ependymal cells or even choroid plexus[23].

c. ENCEPHALOCELE AND MENINGOCELE

Due to congenital dehiscence in the bones, cerebral tissue herniates into the orbital cavity. If the meninges herniate causing a cystic tumour with cerebrospinal fluid, it is termed a meningocele. If the brain protrudes inside the meningeal sac, then it is termed encephalocele. Attempts to explore the orbit may lead to rupture of the cyst causing cerebral damage or meningitis. Facial anomalies characterized by hypertelorism, broad nasal root and increased bitemporal diameter should alert the clinician to the possibility of an encephalocele.

2. INFLAMMATION

a. ORBITAL CELLULITIS

Secondary to sinusitis, this is one of the most common causes of proptosis in childhood. The inflammatory process may spread from the adjacent sinus cavities into the orbit by means of communicating vessels or by direct erosion [24]. Toxic products of inflammation readily diffuse across the normally

thin bony barrier that separates the sinuses from the orbit causing exudation from the orbital vessels. Inflammatory exudates may get loculated to form orbital abscess. In rare instances, it can cause cavernous sinus thrombosis. Four cardinal signs are proptosis, lid swelling, chemosis and impaired ocular motility(Fig.13, 14). Other signs include decreased visual acuity, impaired colour vision, restricted fields, pupillary abnormalities. Local pain and symptoms of profound toxicity like fever, nausea, vomiting and prostration will be present. This clinical picture is most often caused by ethmoiditis[25].

Radiological Findings :

CT :

Diffuse fat infiltration characterized by increased intensity of extra and intraconal fat. There is obliteration of normal fat planes and swelling of anterior orbital tissues. There is no definite delineation among the orbital structures. There is extraocular muscles and optic nerve thickening.

MRI :

In **T1** weighted images high intensity of normal fat with dark inflammatory changes will be seen. In **T2** weighted images normal findings of dark orbital fat with increased intensity for inflammatory changes will be seen. Helps to assess the extension of orbital infection to orbital apex and cavernous sinus.

Treatment:

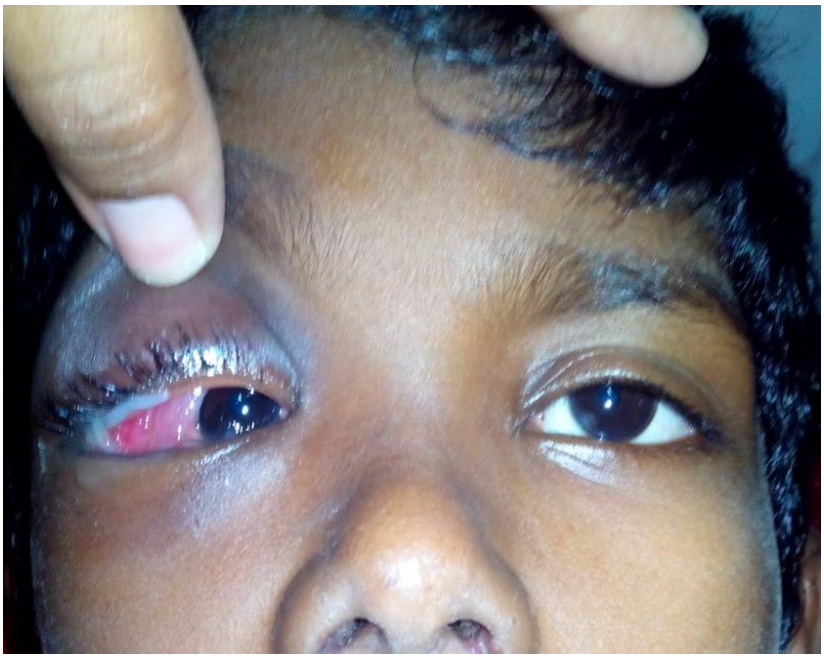
Once the child is diagnosed to have orbital cellulitis he/she should be admitted. Frequent monitoring of systemic and ocular parameters is essential. Intensive antibiotic therapy is started with the broad spectrum antibiotics. The chosen antibiotic should cover sinus pathogen, as well as exhibit beta lactamase resistance and have the ability to penetrate CSF. Intravenous antibiotics are given for 1-2 weeks and then oral antibiotics for 4 weeks.

Fig.13 Orbital cellulitis of the right eye

Fig.14 Orbital cellulitis of left eye

b. ORBITAL ABSCESS

Orbital Abscess is the collection of pus within the orbital tissue. It occurs due to penetrating injuries, post surgical complication and following systemic infections. It can be differentiated from orbital cellulitis by the occurrence of orbital apex syndrome which is consistent with organized infection in posterior orbit.



Signs and symptoms :

Symptoms are similar to orbital cellulitis – proptosis, swelling of eyelids, defective vision and redness. Signs include severe exophthalmos, chemosis, complete ophthalmoplegia, venous engorgement or papilloedema on fundus examination. Systemic toxicity is marked.

Complications:

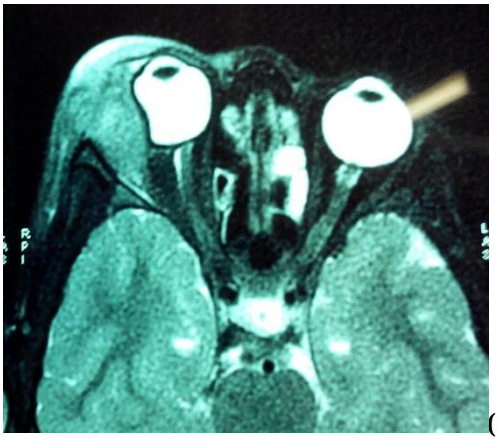
- Infective /toxic neuropathy
- Orbital apex syndrome
- Loss of vision
- Intracranial complications[26]

Radiological Findings:

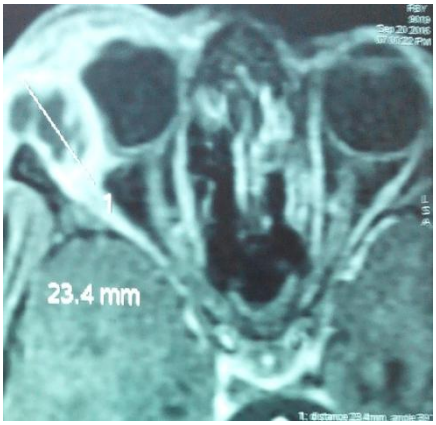
CT : With contrast, it is seen as a well defined lesion with central necrosis and peripheral enhancement.

MRI: Central T1 Hypointense, T2 Hyperintense lesion with thick irregular rim**.**

(Fig 15-16)



(15)



(16)

Figure 15, 16: MRI T2 weighted showing hyperintense lesion and MRI contrast showing ring enhancement in the superotemporal quadrant of right eye suggestive of abscess.

Treatment :

Initial and supportive treatment is similar to that of Orbital cellulitis. Surgical drainage of abscess is done in cases of non resolution of abscess.

c. PSEUDOTUMOUR

Group of inflammatory disorders that produce a pseudo neoplastic orbital mass[27]. This causes pain, proptosis, chemosis and diplopia with visual loss to a virtually asymptomatic ill defined orbital swelling. Non specificity of histology findings, worsening of disease following biopsy and marked improvement with systemic steroids forms the hallmark of this condition. Pseudotumour can mimic thyroid exophthalmos.

d. CYSTICERCOSIS

Caused by Cysticercus cellulosae, the larvae from tapeworm Taenia solium. It has site predilection for brain and eyes. In eyes it can cause cystic lesion in the conjunctiva, muscles and intraocular structures. Blood investigation reveals eosinophilia. B-scan (Fig.18) and CT scan(Fig.19) reveals cystic lesion with a scolex. Treatment includes albendazole with or without corticosteroids.

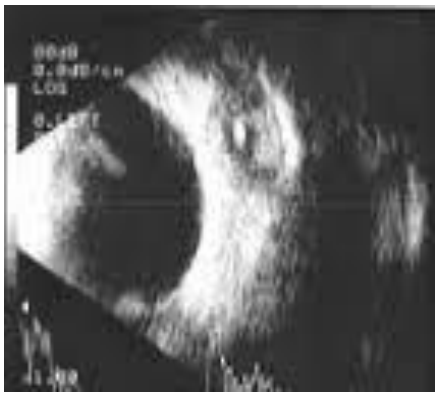


Fig.18 B scan showing cystic lesion with scolex

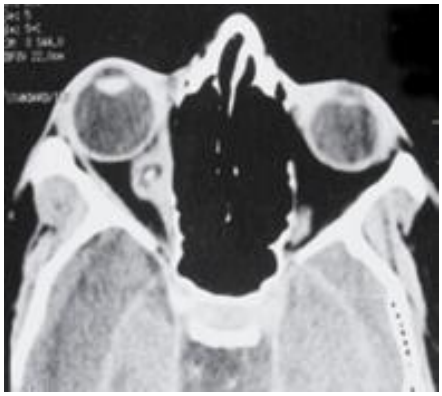


Fig.19 CT scan showing cystic lesion with scolex in right eye

3. VASCULAR LESIONS

a. HEMANGIOMA

Capillary Hemangioma : It is the most common orbital lesion of infancy. Often not recognized during birth, it appears during the first month of life as an ill defined, compressible bluish mass that has a predilection for the upper nasal quadrant of the orbit[29]. The proptosis increases in degree whenever the infant strains or cries. Capillary hemangioma of the eyelid is called a strawberry naevus. Capillary hemangioma (Fig.20) grows with alarming rapidity during the first six months of life followed by spontaneous regression during the next four to five years. With involution it becomes more circumscribed.. Histologically these involutional changes represent a transition from a densely cellular hemangio endotheliomatous tumour into a hypocellular atrophic lesion that contains obliterated capillaries. Although natural involution occurs therapy with topical and oral beta blocker has been advocated to induce a more rapid regression.

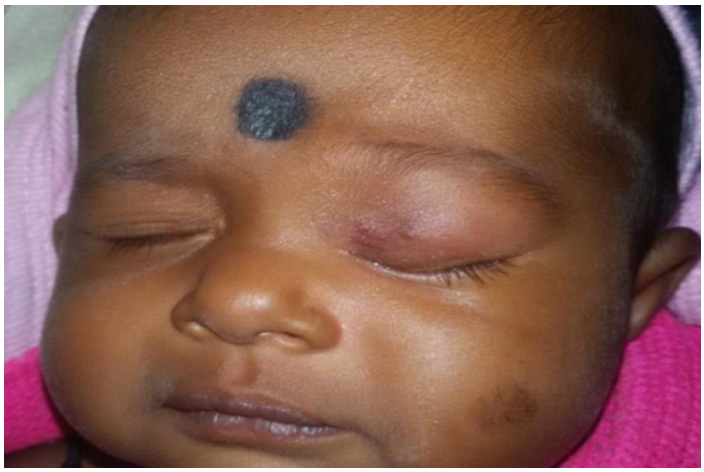


Fig.20 Capillary Hemangioma of Left eye

Cavernous Hemangioma : It represents a developmental anomaly. Enlargement of the tumour is due to the sequential and progressive opening and dilatation of preexisting vessels and sinuses. Symptoms begin in early adulthood. A slowly progressive unilateral proptosis develops. Often the tumour is within the muscle cone causing axial proptosis. It can also produce retinal striae by compressing the globe posteriorly. Visual loss, external ophthalmoplegia can also occur. Surgical removal of the mass is gratifying. Visual recovery is complete post operatively. It is usually easily dissected free of other surrounding structures without hemorrhagic complications. Gross specimen typically has a honeycomb appearance. A connective tissue capsule surrounds widely dilated vascular spaces that are filled with red blood cells.

b. LYMPHANGIOMA

It is a rare congenital tumour of the orbit, becoming classically apparent in the early years of childhood[30]. It tends to involve the superior orbit. It causes recurrent proptosis. Its slowly progressive relentless growth throughout childhood, the lack of spontaneous regression and its unresponsiveness to therapy serves to distinguish it clinically from hemangioma. Bleeding into a lymphangioma causes rapid enlargement of the tumour and an increase in the proptosis and onset of pain. Such blood cysts are called “chocolate cysts”. Periodic enlargement of lymphangioma often accompanies upper respiratory tract infections causing recurrent proptosis.

c. ORBITAL VARICES

True incidence of orbital varices remains controversial. Many Lymphangiomas have been mistakenly thought to be hemangiomas. Wright infact suggested that many lymphangiomas are in fact, congenital orbital varices [31].

Orbital varices are of two types

Type 1 - Represents congenital weakness in the venous wall.

Type 2 - Acquired weakness in the venous wall caused by an A-V shunt.

Signs and symptoms begin during the first five years of life. It continues to enlarge until the patient is 17 or 18 years of age after which there is little change. This also produces intermittent proptosis. Proptosis results from straining, crying, valsalva manoeuvre, placing the head in dependent position or compression of jugular veins. Proptosis is usually downwards, non pulsatile and with absent bruit. Enlargement of dilated veins in the eyelid may be present. Varices can be demonstrated by positive contrast venography.

d. A – V MALFORMATIONS

With congenital AV malformations, the communication (shunt) may be large or small, resulting in either high-flow or low-flow lesions[32]. The larger the communication, the more profound the orbital findings. A high flow malformation demonstrates pulsating exophthalmos , bruit, marked orbital swelling clinically due to retrograde flow into the venous system. Low flow lesions shows lesser degree of Proptosis, absent or minimal bruit, milder elevation of venous pressure leading to dilated episcleral, orbital, intraocular veins and raised intraocular pressure (Fig 21,22). Larger venous connections are distensible and small venous connections are non-distensible. Clinically non-distensible lesions has minimal flow (Fig 23) and tends to present with spontaneous thrombosis or hemorrhagic episodes.

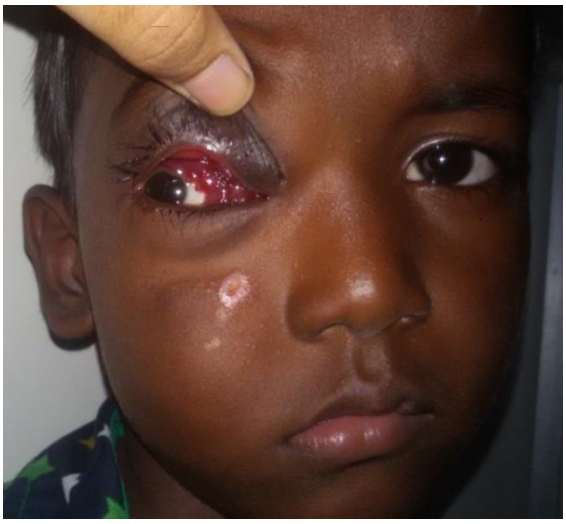


Fig.21 Showing vascular malformation Right eye

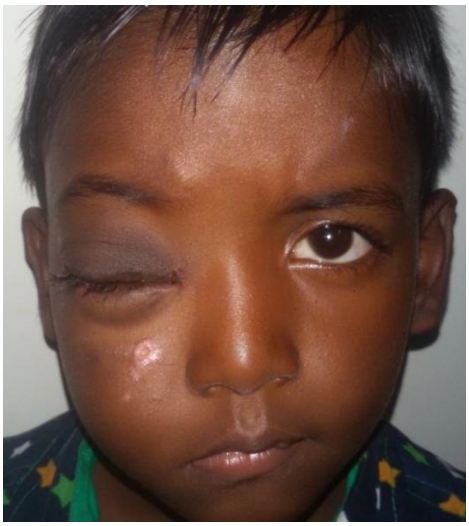


Fig.22 Showing Proptosis Right eye

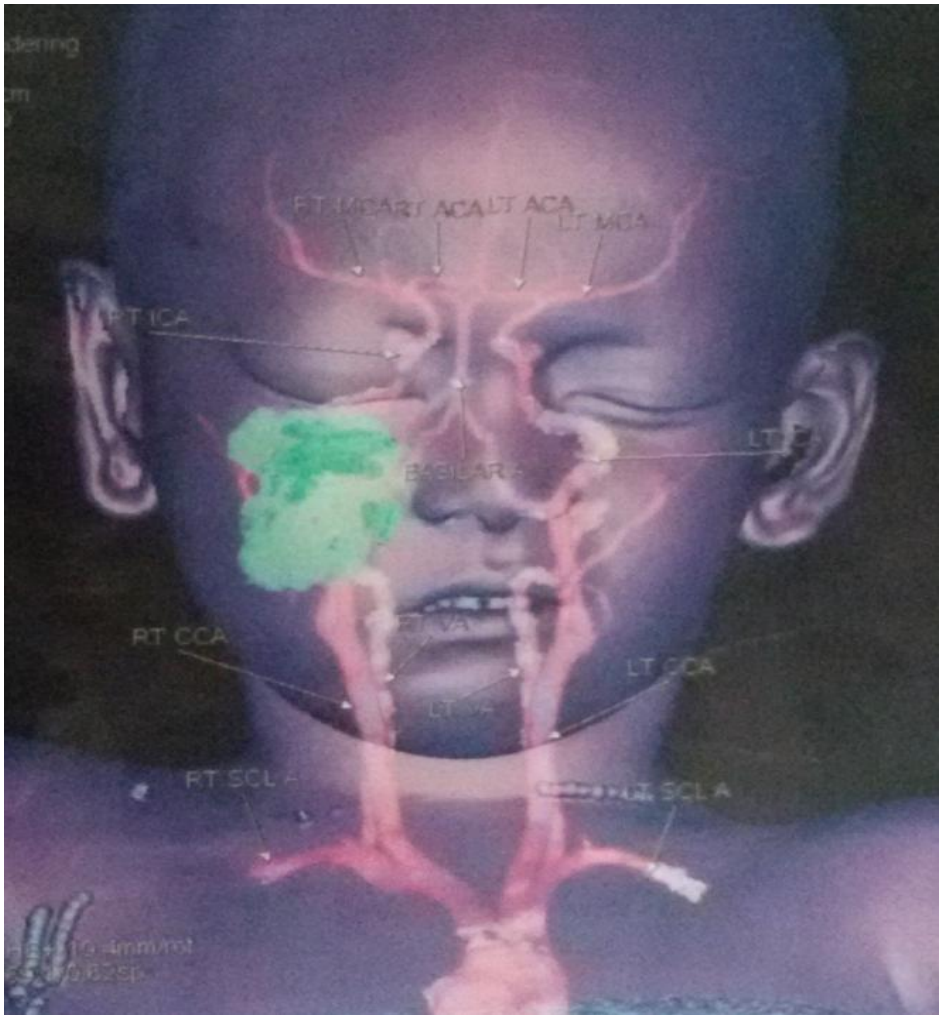


Fig.23 128 slice CT angiogram showing tortuous attenuating structures in the right orbit – suggestive of low flow vascular malformation

4. TUMOURS

a. PRIMARY MALIGNANCIES

MALIGNANT HEMANGIOENDOTHELIOMA

Malignant tumour of the endothelial cells of the blood vessels[34]. These tumours are uncommon. These tumours are infiltrative and locally aggressive and have a high metastatic potential.

HEMANGIOPERICYTOMA

This is a tumour of the pericyte which occupies a position outside of the endothelial cells but in close apposition to them in capillaries and post capillary venular channels[36]. Usually presents with proptosis of 6 months to 2 years duration. Tumour is usually situated within or outside the muscle cone and usually does not involve bone at the onset. Eyelid swelling, diplopia, a palpable mass in 60% of patients are present.

NEUROGENIC LESIONS

NEUROFIBROMATOSIS

Ocular manifestation occur in the first decade of life. Although hereditary, it is transmitted in an autosomal dominant fashion. Orbital involvement is invariably unilateral. One characteristic finding is developmental absence of orbital bones posteriorly. These defects usually involve the sphenoid bone and orbital roof posteriorly. Harkin and Reed have classified involvement in neurofibromatosis into 3 principal patterns.

1. Central neurofibromatosis with gliomas, ependymomas, meningiomas &

schwannomas of the nerves.

2. Peripheral neurofibromatosis with neurofibromas of the skin, plexiform type tumours and sparing of CNS and the viscera.

3. Visceral neurofibromatosis with neurofibromas ,schwannomas and ganglioneuromas occurring along the Gastero Intestinal tract.

MENINGIOMAS

Can be primary or secondary

Primary: Orginates from the intraorbital optic nerve sheath.

Secondary: Originates along the sphenoid wing or in the basofrontal region and subsequently invade the orbit.

GLIOMA

Optic nerve gliomas are uncommon but they are more common than meningiomas. The peak incidence is from 2-6 years of age[35]. Females are slightly more frequently affected. Clinical presentations fall into 2 patterns depending on whether the tumour is largely orbital or intracranial. Loss of vision is the most common initial symptom in intracranial gliomas. Proptosis is an early feature of intraorbital gliomas. It is usually very mild rarely exceeding 3mm (Fig.24,25). Proptosis is axial, non pulsatile and irreducible. Pain is unusual. Tumour is not palpable. Movements are mechanically restricted. Ophthalmoscopy reveals primary optic atrophy, CRVO is a rare complication. Intracranial gliomas disturb hypothalamic and pituitary function and produce symptoms of increased intracranial pressure.

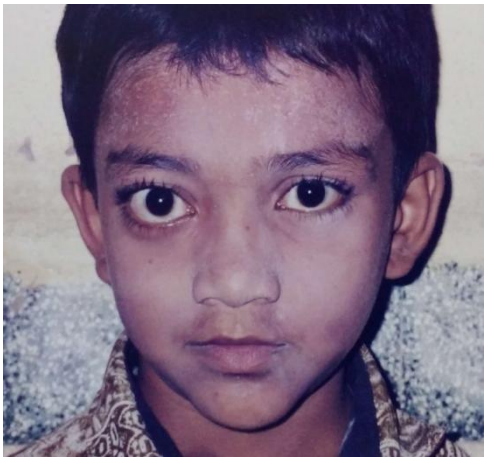


Fig.24 showing Proptosis due to Optic glioma Right

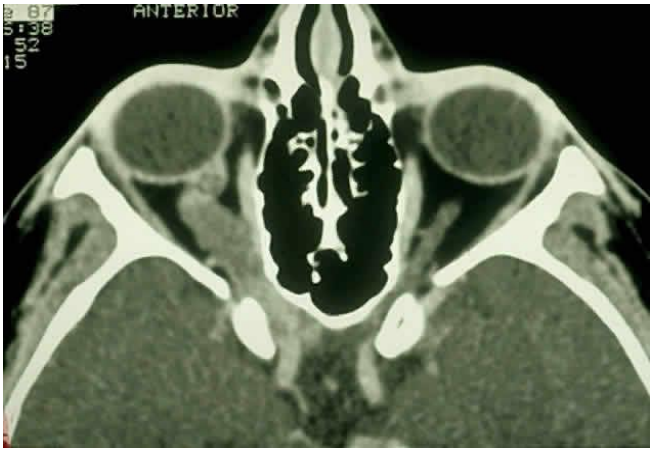


Fig.25 CT scan showing Optic Nerve glioma (intraconal lesion) Right eye

RHABDOMYOSARCOMA

It is the most common primary malignant orbital tumour of childhood. Produces a rapidly progressing unilateral proptosis of sudden onset in a Child . Although the proptosis, lid swelling and chemosis may be marked (Fig.26), the symptoms associated with this rapidly evolving and often horrifying clinical picture may be disproportionately meagre. It is a highly malignant neoplasm of pleuripotential embryonic Mesoderm [37].

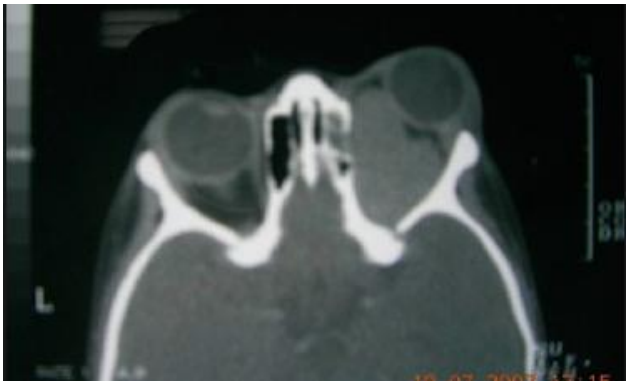


Fig.26 Rhabdomyosarcoma Left Eye

FIBROOSSEOUS LESIONS

OSTEOMA : Osteoma is a common benign tumour of the paranasal sinuses. It usually arises within the frontal sinus. It is less frequent in the ethmoidal and maxillary sinuses and rarely seen in sphenoidal sinus. They grow very slowly and is usually asymptomatic for long periods of time, frequently being discovered as an incidental finding. Headache, facial pain or swelling are the usual complaints. Nasal obstruction or discharge may be caused by an ethmoidal osteoma. Frontal and ethmoidal osteomas are most likely to encroach on the orbital space. When palpable it is firm hard, nontender, and noncompressible. Asymptomatic osteomas, especially in elderly require no therapy. Encroachment into the orbit requires surgery. Histologically it consists of dense intracommunicating bony lamellae.

FIBROUS DYSPLASIA : It is a non neoplastic disorder of the childhood that frequently involve the orbital bones. The frontal, sphenoidal and ethmoidal bones are most commonly involved. Thickening of these bones decrease the orbital volume and characteristically leads to a slowly progressive unilateral eccentric proptosis. Facial asymmetry will be conspicuous. The condition becomes inactive after puberty. Surgical intervention may become necessary, especially when there is optic nerve compression producing visual loss. Histologically poorly calcified, primitive islands, of bone are surrounded by a highly cellular stroma that is composed of benign fibroblasts[37].

SECONDARY ORBITAL NVOLVEMENT

FROM THE GLOBE - RETINOBLASTOMA

Retinoblastoma is the most common intraocular tumour of Childhood. Rb 1 gene, retinoblastoma gene located on the long arm of chromosome 13 is implicated in its oncogenesis. There are heritable and sporadic forms. Clinical presentation includes leucocoria, strabismus, buphthalmos, secondary glaucoma, pseudo-hypopyon, orbital cellulitis like picture, unexplained hyphema, unexplained phthisis. It can be exophytic or endophytic or mixed.

Histopathological Features: Round or oval nuclei with scanty cytoplasm with areas of necrosis and calcification. Flexner wintersteiner rosettes are seen. Orbital extension is associated with retinoblastomas that involve the choroid and optic nerve extensively. Once in the orbit, tumour grows luxuriantly causing rapidly advancing Proptosis.

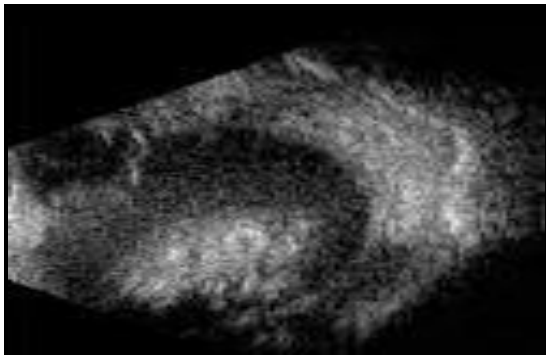


Fig.27 B scan showing diffuse mass filling the globe with areas of high echoreflectivity suggestive of calcification

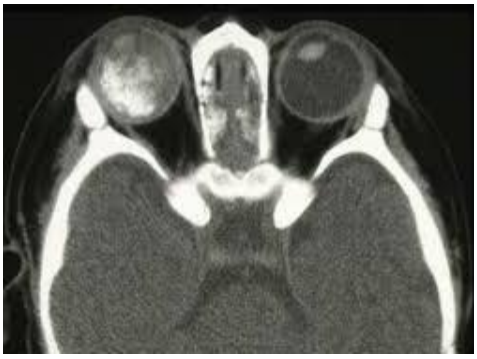


Fig.28 CT scan showing mass lesion with internal calcification in the right eye

b. METASTASIS

NEUROBLASTOMA

It is one of the most common childhood tumours[38]. It is a neoplasm of embryonic neuroblastic tissue. It may arise from anywhere such tissue is normally found. There is one report of a neuroblastoma having arisen within the orbit. Apart from proptosis, flushing, diarrhoea and hypertension are seen in some patients with secreting tumours. In the orbit the metastasis has a tendency to lodge in the zygomatic bone. Ecchymosis of lids is also present (Fig.29).

Fig.29 B/L Severe Proptosis with ecchymosis due to secondaries from Neuroblastoma

LYMPHORETICULAR MALIGNANCY

In over 75% of patients with leukemia the eyes and adnexa are invaded by leukemic cells, usually by lymphoblastic cells. The acute leukemias are more likely to cause orbital metastasis and lymphoblastic ones more so than myelogenous leukemias. Orbital involvement occurs in 2% of patients[38].

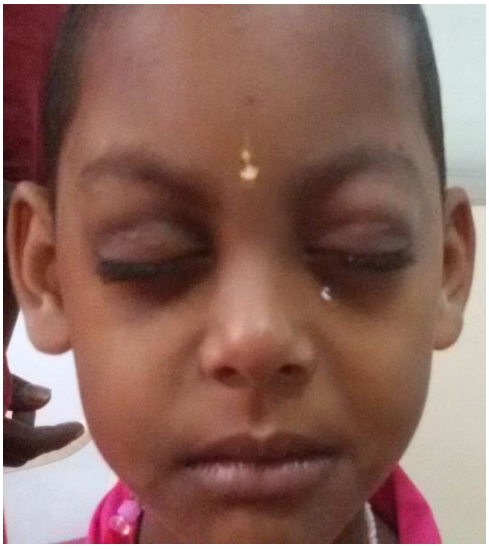


Fig.30 showing Bilateral Proptosis due to AML

Fig.31 showing Bilateral Proptosis due to Leukemia

5. ENDOCRINE:

GRAVE'S DISEASE

Thyroid dysfunction is the most common cause of unilateral proptosis[28]. The proptosis frequently becomes bilateral in the course of the disease. Hyperthyroidism contributes to 90% of cases. Euthyroid & hypothyroid individuals form the rest 10%. Typical clinical signs accompany the disease. Proptosis with lid lag and lid retraction are very characteristic (Fig.17). Lid swelling, chemosis conjunctival hyperemia, lagophthalmos and impaired ocular motility can also be present. Defective vision and lacrimation caused by exposure keratitis may also be present. Impaired ocular motility is restrictive and mechanical in origin. Restriction is confirmed by "Forced duction test". Morphologically there is massive enlargement of the muscles due to interstitial

edema. Individual muscle cells are normal. There will be associated inflammatory cell infiltration consisting of lymphocytes, macrophages, mast cells and plasma cells. These are manifestations of deranged immunologic system. Most patients have elevated levels of circulating thyroid stimulating immunoglobulins. These are long acting thyroid stimulator and human thyroid stimulator (HTS) as well as easily demonstrable circulating antithyroid antibodies. Systemic corticosteroids orbital radiotherapy and surgical decompression are the various methods adopted to treat Grave's exophthalmos.

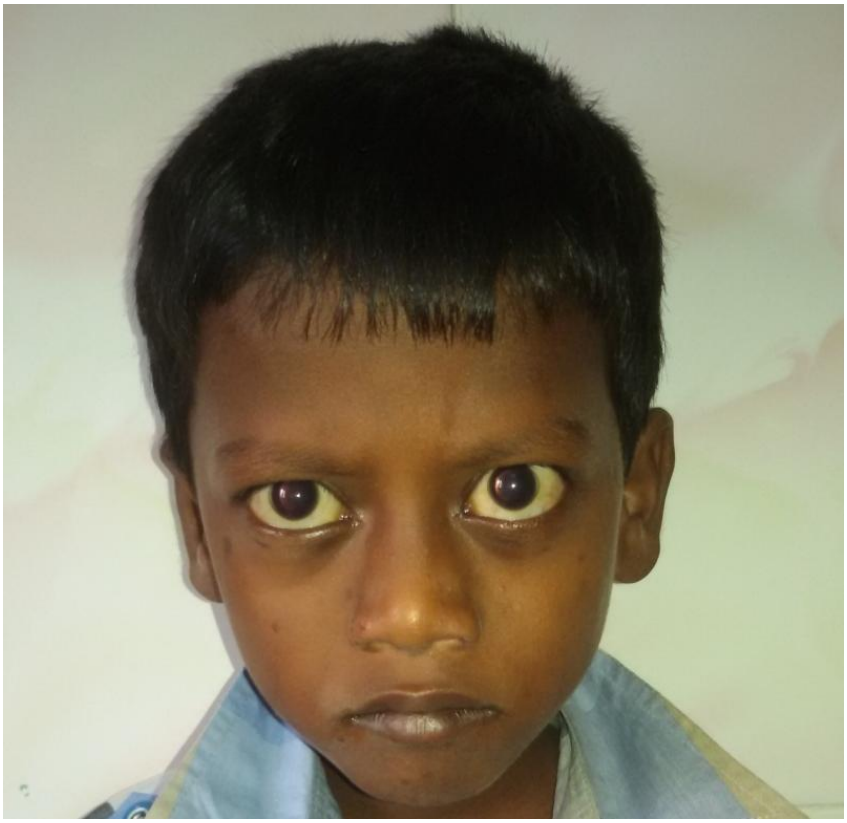


Fig.17 Grave's exophthalmos (B/L Proptosis)

EVALUATION OF A CHILD WITH PROPTOSIS

The workup is based on the following :

A. History

B. Clinical examination

C. Investigation

A. HISTORY

The following points are relevant:

1. Usual complaints of orbital disease are -

proptosis/ pain/ diplopia/ defective vision/ defective field of vision

2. Mode of onset may be acute/ subacute / chronic and nature of onset may be congenital/ acquired

3. Mode of progression - may be stationary/ gradually progressive or rapidly progressive. Remissions and exacerbations may also occur.

4. Diurnal variation- Painful proptosis in the early mornings which subsides by evening indicates orbital inflammatory diseases like pseudotumour, thyroid orbitopathy etc.

5. History of injury to eye or head injury

6. Systemic complaints:

    a. Thyroid related complaints like increased appetite with loss of weight, palpitations and chest pain, decreased appetite with weight gain,

hyperactivity/ lethargy, diarrhoea/ constipation, skin related problems, swelling in the neck region, tremors in the extremities.

b. History of fever and frequent upper respiratory infections are important in case of leukemias, orbital inflammations and sinusitis.

c. History of epistaxis and abnormal bleeding from other sites of body.

d. History of loss of weight and loss of appetite may be suggestive of systemic malignancy.

e. History of any other swellings in the body as in the case of neurofibromatosis, haemangioma, parasitic cysts[39].

B. CLINICAL EXAMINATION

SYSTEMIC EXAMINATION

1. Vital signs monitoring:

   pulse rate, respiratory rate, blood pressure and temperature recording

2. Lymphadenopathy which may be preauricular/ cervical/ generalized

3. Hepatosplenomegaly

4. Any other mass palpable in other sites of body

5. Thyroid swelling

6. Finger tremors/clubbing/cyanosis

7. Nasopharyngeal examination/ Dental examination

8. Any musculoskeletal disorders

9. Dermatological examination

10. CNS examination/ CVS/ Resp/ GIT Examination

LOCAL EXAMINATION

1. Visual status and refraction: Patient may be hypermetropic because retina is pushed forwards, or myopic if there is a pseudoproptosis.
2. Colour vision
3. Central field charting
4. Diplopia and Hess charting
5. Squint evaluation/ Forced duction test
6. Examination of anterior segment/ Slit lamp biomicroscopy
7. Pupillary reactions- Direct/ Consensual
8. Intraocular pressure should be done preferably with applanation tonometry. It increases in orbital inflammatory diseases. Differential intraocular pressure recording should be done in various gazes.
9. Examination of posterior segment by indirect/direct/ ophthalmoscope. Fundus examination to rule out papilloedema/ papillitis/ optic atrophy/ opticociliary shunt/ vascular anomalies/ retinal striae or folds, choroidal folds, exudative retinal detachments etc .

SPECIFIC EXAMINATION

This should be carried out for:

1. Facial assymmetry.
2. Position of eye brows/ eye lids-lid retraction/ bogginess of lids/ lid lag on Down gaze,
3. Examination of ptosis/ measurement of levator function,
4. Extraocular motility/ defective convergence,
5. Various thyroid orbitopathy signs

PROPTOSIS EXAMINATION

It may be axial or eccentric depending upon direction.

It may be unilateral or bilateral.

- Down and out proptosis occurs in ethmoidal mucocles.
- Down and in proptosis occurs in lacrimal gland tumours
- Up wards in maxillary involvement.
- Downwards in subperiosteal hematomas

EXOPHTHALMOMETRY (PROPTOMETRY)

Exophthalmometers are used to measure the proptosis, generally it measures the distance from outer orbital margin to the corneal apex, while the eyes look straight.

TYPES OF EXOPHTALMOMETRY

a. Absolute – The values are compared with the normal values .
b. Comparative – The values are compared from time to time.
c. Relative – As compared with other eye (The difference of >2 mm is important).

HERTEL'S EXOPHTHALMOMETER (1905)

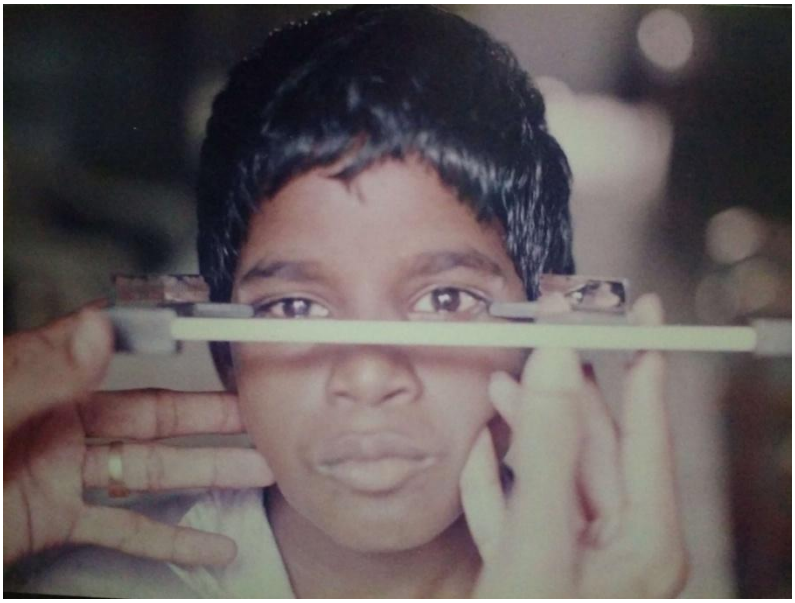


Fig.32 Hertel's Exophthalmometer

It is the most commonly used exophthalmometer[40]. This instrument is binocular and resting on each lateral bony orbital margin, allows an observer in front with the aid of mirrors to view the images of the corneal apex, superimposed upon measuring scale. The measurement is the distance between the apex of the uncovered cornea to the temporal margin of the orbit (Fig.32).

ON INSPECTION

Naffziger's sign: While looking tangentially over the forehead, the palpebrae of the proptosed eye is seen first. Other points to be noted are

• Fullness/ mass lesion in the orbit

• Any visible pulsation/ visible engorged vessels,

• Lagophthalmos with corneal exposure.

• Conjunctival congestion at recti muscle insertion indicates thyroid orbitopathy while diffuse congestion of conjunctiva indicates vascular anomaly.

ON PALPATION

Palpation of orbital rim for any irregularity, mass lesion, finger insinuation between globe and orbital bones is essential. Size, shape, surface, skin over swelling, consistency, signs of inflammation, tenderness, reducibility, margins ,motility etc., details of the mass lesion must be noted. Variation of proptosis with alteration in postures are like valsalva maneouvre/ bending down the head, should be looked for. Other features to be noted are

- Resistance to retropulsion,
- Pulsations over swelling/ over the eye ball,
- Infra orbital/ Supraorbital anaesthesia, Corneal anaesthesia

ON AUSCULTATION

For the presence of bruit over the mass lesion/over the eye ball/over the temporal vessels. It is positive for A-V malformations.

INTRAOCULAR PRESSURE

Intraocular pressure is measured in both eyes, in both primary and in differential gaze, using the SCHIOTZ tonometer.

FORCED DUCTION TEST

It is performed by using instruments to move the anesthetized eye mechanically in to various positions, thus determining resistance to passive movements in restrictive pathology but not in paralytic cases.

C. INVESTIGATIONS

Can be divided into

- **Lab investigations**
- **Non invasive investigations**
- **Invasive investigations**

LAB INVESTIGATIONS

- Total Count,
- Differential Count
- ESR, Mantoux Test
- Peripheral Smear
- Thyroid function tests
- LDH assay
- Urine : Albumin, sugar and deposits
- Motion : Ova and cyst.

NONINVASIVE INVESTIGATIONS

1. PLAIN X-RAY – ORBIT, OPTIC CANAL, SINUSES AND SKULL.

IMPORTANT RADIOLOGICAL VIEWS

- **Caldwell's view** : In this view , patient's forehead and nose touch the plate with incident ray angled at 10˚downwards. This view allows good visualization of orbital margins, medial wall, lesser wing of the sphenoid, superior orbital fissure, orbital portion of zygoma. The innominate line is prominent in this view and is discontinuity indicates lateral orbital wall fracture. It provides excellent view of frontal and ethmoid sinuses.
- **Water's view** : Patient nose and chin touch the plate in this view. This view provides the best image of the maxillary antrum and good images of the orbital rim, floor, lesser wing of the sphenoid and infra orbital foramen. **Rhese's view** (optic canal) : It allows good visualization of the optic canal, medial wall, lacrimal fossa and orbital apex.
- **Lateral view** : The orbital roof is the best evaluated in this view, all paranasal sinuses can be evaluated by this view. It also shows the sella tursica, anterior and posterior clinoid processes, nasopharynyx and cribriform plate.
- **Basal view** : This view allows good visualization of posterolateral wall of the orbit, maxillary sinus, greater wing of sphenoid and pterygoid fossa. History of neck injury is a contraindication to this view.

X RAY CHANGES IN ORBITAL PATHOLOGY

- **Enlargement of the orbit :** Localized enlargement in space occupying lesions outside the muscle cone and long standing neoplasms (lacrimal gland lesion is common). Generalized enlargement in long standing space occupying lesion causing increased intra orbital pressure. Downward bowing of the thin floor of the orbit below the infra orbital margin is the earliest sign. Concentric enlargement without bony destruction is seen in hemangioma and lymphangioma. Symmetric enlargement is seen incase of tumour within the muscle cone.
- **Diminution of orbital size :** It is seen in craniosynostosis and the therapeutic irradiation.
- **Hyperostosis :** The most common for hyperostosis is meningioma of the sphenoid bone. Fibrous dysplasia, Paget's diseases, chronic periostitis, malignant tumours of the lacrimal gland and osteoblastic metastasis also produce hyperostosis.
- **Calcification :** It can be seen in retinoblastoma, meningioma, cavernous hemangioma and orbital varices. Plexiform neurofibromatosis, tuberculosis, cyticercosis and hydatid disease also produce calcification.
- **Enlargement of sphenoid fissures :** Infraclinoid aneurysm, meningioma, caratico-cavernous fistula, extrasellar extension of pituitary tumour can produce enlargement of sphenoid fissure. Intracranial extension of orbital

space occupying lesion such as neurofibroma, reticulosis or pseudo tumour extending backward in to the middle cranial fossa.

- **Enlargement of optic foramen :** Normal adult dimension (4-6mm) being reached by the age of 4 years. Measurement of more than 7 mm is abnormal. Uniform enlargement is seen in glioma of the optic nerve and extension of orbital neurofibroma or retinoblastoma. Asymmetrical erosion of inferolateral margin is seen in infraclinoid aneurysm and meningioma. Erosion of upper margin is characteristic of raised intra cranial pressure.
- **Narrowing of Optic foramen :** Seen in fibrous dysplasia, Paget's disease, hyperostosis secondary to a meningioma, idiopathic hypercalcemia and microophthalmos.
- **Soft tissue and orbital emphysema :** Clouding of paranasal sinuses may be due to infection or neoplastic disease. The orbital emphysema may be seen in medial wall and floor fractures, aneurysms.

X RAY SINUSES :

Mucocele, Sinusitis

X RAY CHEST:

TB, Sarcoidosis, Rhabdomyosarcoma, Secondary deposits

2. ULTRASONOGRAPHY

Baum and Green wood in 1960 first used the ultrasound in orbital lesions. It is a dynamic test where diagnosis is best reached during examination and not from still pictures. There are three modes of echo display used in ophthalmic ultrasound.

i. **A Sound** – It is a time amplitude study. Primarily used for measuring the axial length and power of intraocular lens..

ii. **B Mode** – Gives real time two dimensional image of the eye[41].

iii. **M Mode** – Usually 5 or 10 MHz transducer are used for orbital diagnosis.

a. **Low reflectivity :** Organizing hematoma in peripheral space, mucocele, varix, benign mixed tumour, lymphoma, inflammatory tumour rhabdomyosarcoma, neurilemmoma, fibrous histiocytoma and capillary hemangioma (variable).

b. **Medium reflectivity :** Dermoid, optic nerve glioma and mengioma.

c. **High reflectivity :** Cavernous hemangioma, lymphangioma, most carcinomas and vascular neoplasm.

Advantages:

- ✓ It is not invasive and no exposure to radiation
- ✓ Assess kinetic properties of orbital lesions
- ✓ Performed as an OP procedure
- ✓ Can be performed by an ophthalmologist and ideal for follow-up.

Disadvantages :

- ❖ Lesion in the posterior aspect of the orbit may not be picked up.

ORBITAL COLOUR DOPPLER

It uses B mode coupled with Doppler technology to visualize flow in the vessels of the orbit. Useful in cavernous sinus fistula, orbital varices, A-V malformation and superior ophthalmic vein obstruction.

3. COMPUTED TOMOGRAPHY (CT SCAN)

CT scan is the most valuable technique for delineating the shape, location, extent and character of lesions in the orbit. CT scan not only helps to refine the differential diagnosis but also it guides the selection of the surgical approach by relating the lesion to the surgical space or spaces of the orbit. Generally slice width for orbital CT scanning is 3 mm (for both axial and coronal). Current CT scanners administers a dose of radiation of approximately 1-2 cGy per orbit scan. CT scan has resolution and tissue contrast capabilities allowing imaging of soft tissue, bones, contrast-containing blood vessels, and foreign bodies. Coronal CT scanning is especially useful in evaluating orbital floor and extra ocular muscle size in Grave's ophthalmopathy. Axial slices are parallel to Reid's baseline (from external auditory meatus to inferior orbital rim). The spiral CT scanning technique, using new imaging hardware and software technique moves the scanner in a spiral fashion around the patient,

generating data set. Three dimensional CT scan technique allows reformatting of CT information in to three dimensional projection of the bony orbital walls. Intra orbital contents cannot be visualized because of artifact.

3. MAGNETIC RESONANCE IMAGING (MRI)

MRI has revolutionalised the radiological imaging technique especially for soft tissue lesions. It is a non-invasive imaging technique that does not employ ionizing radiation and has no known adverse biological effects.MRI is based upon the interaction of three physical components in (i) atomic nuclei possessing an electrical charge. (ii) radio frequency (RF) waves
(iii) powerful magnetic field. Angiography using MRI (MRA) can be done on diagnosis of vascular lesions like ophthalmic artery aneurysm, AV malformation and vascular tumours. Use of surface coils increase the spatial definition of the images.

Contraindication to MRI :

- Suspected magnetic intraocular or intraorbital FB,
- Metallic surgical clips,
- Cardiac pace makers,
- Cochlear implant and
- Pregnancy.

INVASIVE INVESTIGATIONS

Other invasive procedures given below are also available :

- Orbital Angiography
- Orbital Venography
- Radioactive isotope scanning
- Radio isotope arteriography
- Fine needle aspiration cytology (FNAC) ▪ Orbital biopsy
- Incisional biopsy
- Excisional biopsy

AIM OF THE STUDY

- To evaluate the various causes of Proptosis in children upto 14 years.
- To analyse the incidence, mode of onset, various clinical presentations in patients with childhood Proptosis.
- To assess the various treatment modalities and the final outcome of treatment of childhood Proptosis.

MATERIALS & METHODS:

This descriptive study will be conducted at Orbit and Oculoplasty Services, RIOGOH, Egmore, Chennai for a period of 6 months from March 2016 to Sep 2016.

METHODOLOGY:

- ✓ First 30 Patient presenting to Orbit and Oculoplasty Services with proptosis will be registered, evaluated and followed up during the study period.
- ✓ A detailed history of the patient in relation to the mode of onset, progression, laterality, associated symptoms like pain, fever, birth trauma, prior medical and surgical treatment, family history of proptosis were noted.

- ✓ Complete general examination and ocular examinations including visual acuity, examination of orbit, eyelids, anterior and posterior segments were done.
- ✓ Slit lamp biomicroscopy, ophthalmoscopy, Hertel's exophthalmometry, fields, colour vision, refraction, intra ocular pressure and examination of proptosis were also done.
- ✓ Laboratory investigations were done to find out inflammatory and haematological causes of proptosis.
- ✓ Radiological investigations and tissue examination were done to aid in the etiological diagnosis and to plan the management.
- ✓ Patients were also referred to other departments like ENT, Haematology, Neurology, Oncology and Radiology to get expert opinion regarding diagnosis and for treatment whenever indicated.

INCLUSION CRITERIA:

- ❖ Children upto 14yrs presenting with proptosis were enrolled.

EXCLUSION CRITERIA:

- ❖ Children with severe debilitation were excluded.

STATISTICAL ANALYSIS :

- ❖ Descriptive analysis – here all variables will be described with frequency distribution and displayed using percentage.

RESULTS AND ANALYSIS

Total number of proptosis cases reported in RIOGOH during the study period between March 2016 to September 2016 were 90 cases, including both children and adults.

DISTRIBUTION OF PROPTOSIS IN GENERAL POPULATION :

Age	No.of cases	Percentage
Upto 14 yrs	30	33%
Above 14 yrs	60	66%

Table.1 Distribution of Proptosis in general population

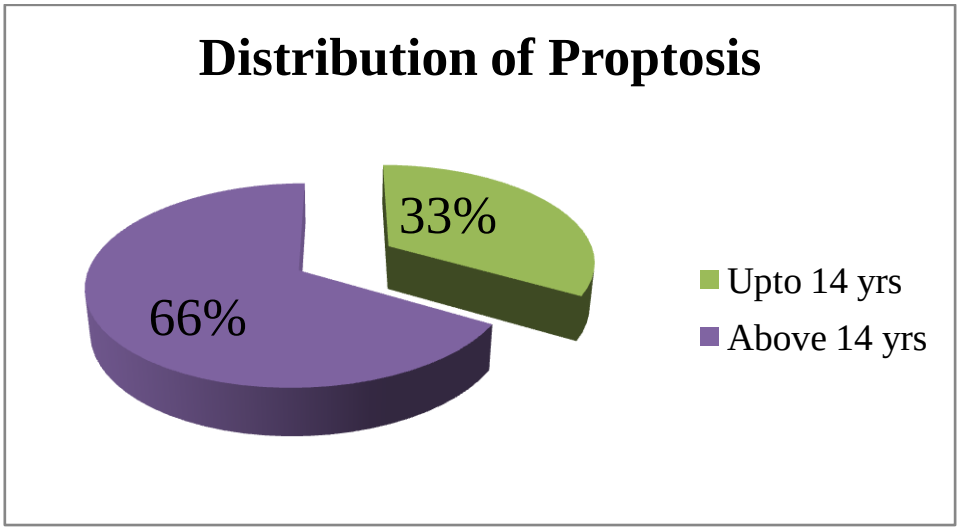


Chart.1 Pie chart showing distribution of proptosis in general population

The incidence of proptosis was highest in patients above the age group of 14 yrs (66%). Only 33% were under the age group of 14 yrs.

AGE DISTRIBUTION AMONG CHILDREN WITH PROPTOSIS:

Age in years	No. of cases	Percentage
0-4	17	56.7%
5-9	11	36.7%
10-14	2	6.7%

Table.2 Age distribution among children with proptosis

Of the proptosis in the pediatric age group in our study, the highest incidence was seen between the age group of 0-4 years (56.7%)

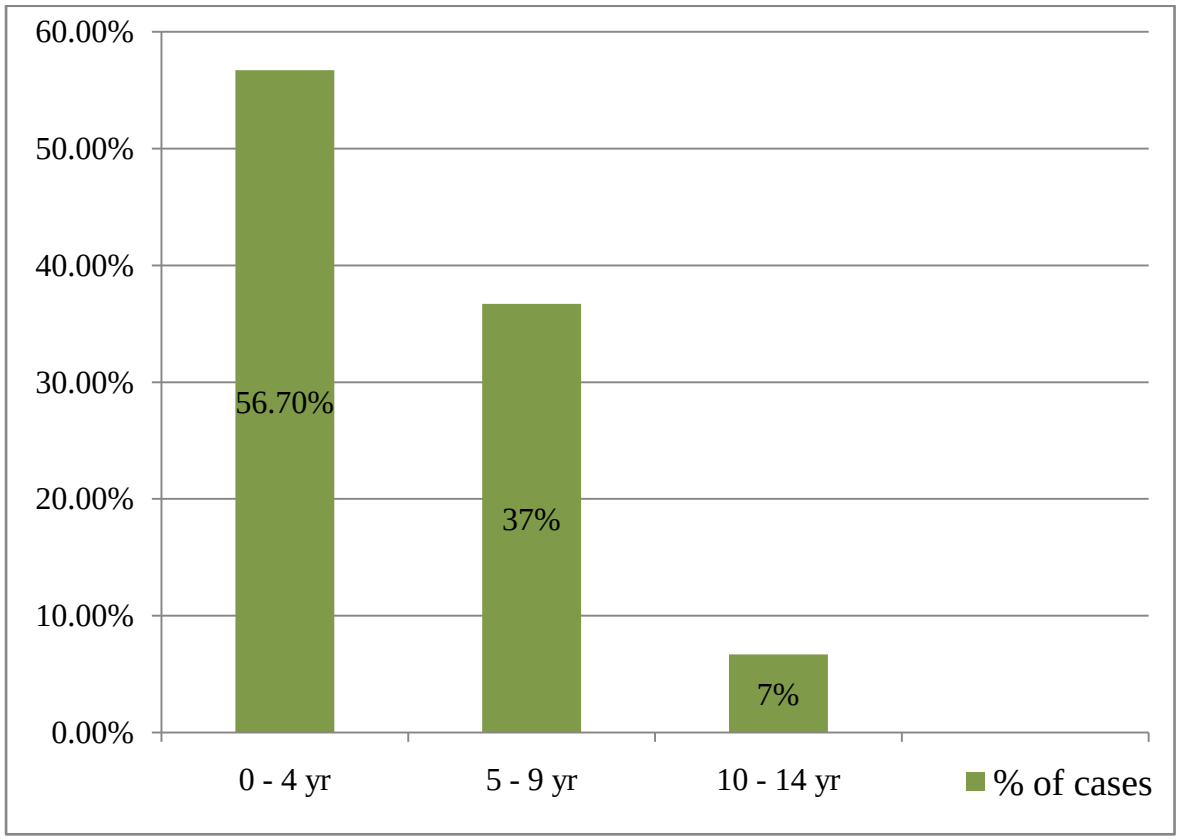


Chart.2. Histogram showing Age distribution among children with proptosis

SEX DISTRIBUTION AMONG CHILDREN WITH PROPTOSIS :

Sex	No. of cases	Percentage
Male	16	53.3%
Female	14	46.7%

Table.3 Sex distribution among Children with proptosis

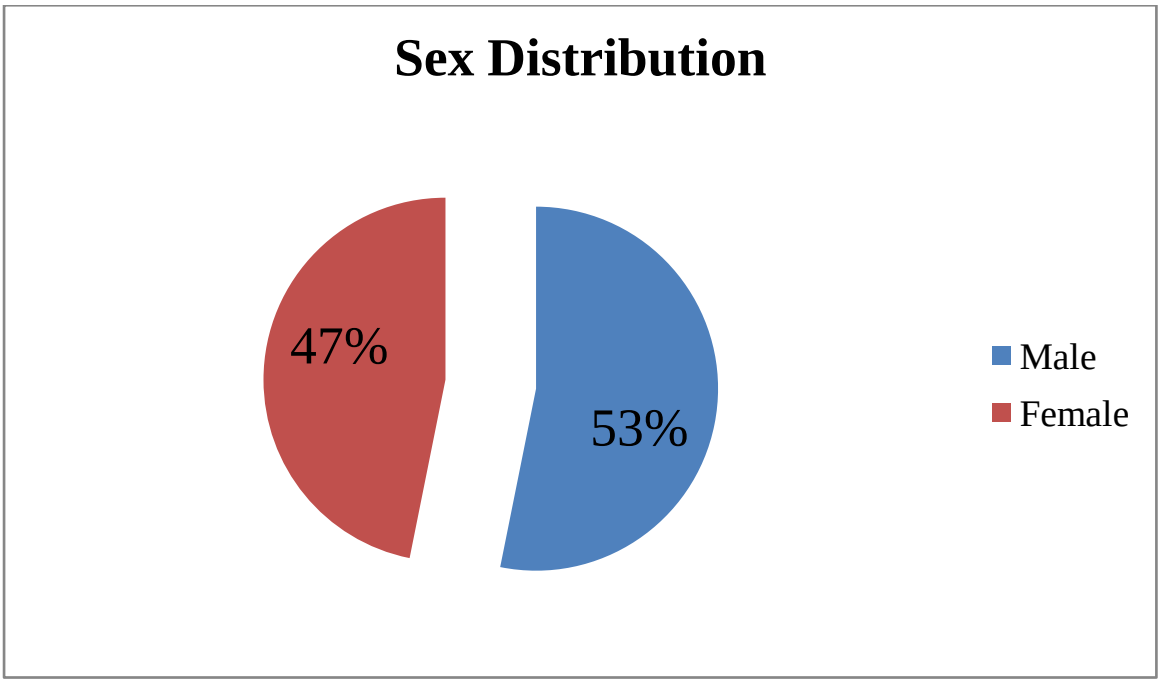


Chart.3 Pie Chart showing Sex distribution among Children

Of the reported childhood proptosis in this study 53.3% were males and 46.7% were females. There is no significant in incidence of proptosis among male female children.

DIRECTION OF PROPTOSIS:

Direction	No. of cases	Percentage
Axial	18	60%
Eccentric	12	40%

Table.4 Direction of Proptosis in Children

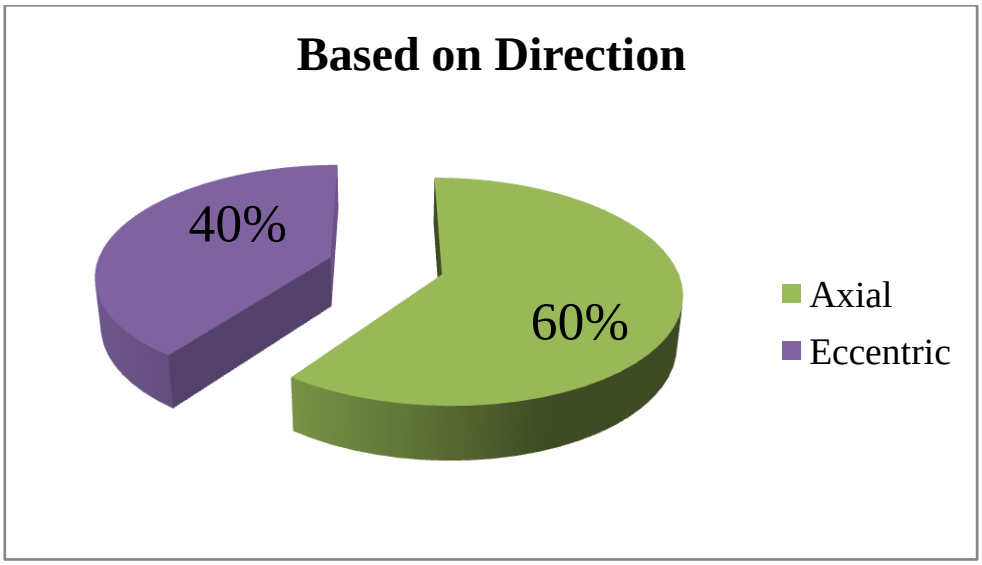


Chart.4 Pie chart showing Direction of Proptosis in Children

Axial proptosis was more common than the eccentric proptosis. Of the total 30 cases 18 cases (60%) were axial and only 12 cases (40%) were eccentric proptosis.

LATERALITY OF PROPTOSIS:

Laterality	No. of cases	Percentage
Unilateral	25	83.3%
Bilateral	5	16.67%

Table.5 Laterality of Proptosis in Children

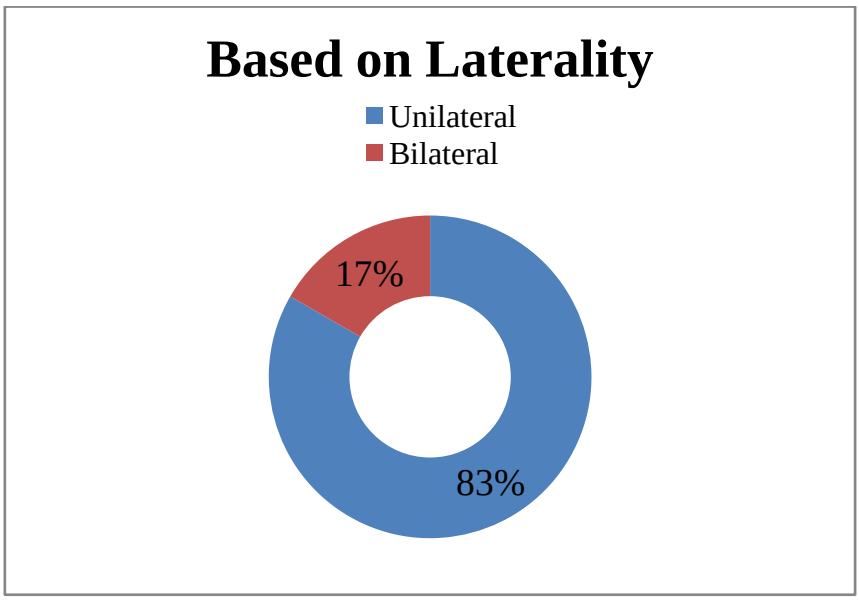


Chart.5 Doughnut chart Showing Laterality of Proptosis in Children

Incidence of unilateral proptosis was more common (83.3%) than the bilateral proptosis which is 16.67%.

ONSET OF PROPTOSIS:

Nature of onset	No. of cases	Percentage
Acute	11	36.7%
Chronic	19	63.3%

Table.6 Showing Onset of Proptosis in Children

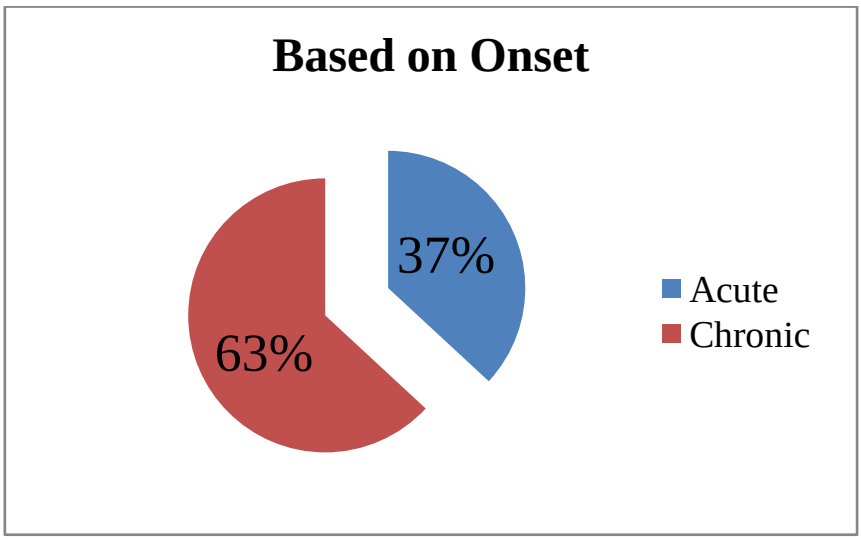


Chart.6 Pie Chart Showing Onset of Proptosis in Children

Acute onset of proptosis is less common than the chronic cases. Among the acute conditions orbital cellulitis was the most common cause. Among the chronic conditions benign tumours were more common followed by malignant tumours among which secondaries were more common.

CAUSES OF PROPTOSIS IN OUR STUDY POPULATION

Aetiology	No. of cases	Percentage
Inflammatory	12	40.00 %
Congenital	9	30.00 %
Malignant	6	20.00 %
Grave's eye disease	2	06.67 %
Traumatic	1	03.33 %

Table.7 Various Causes Of Proptosis In Our Study Population

- Among various causes of childhood Proptosis Inflammatory causes ranks the first(40%) followed by congenital conditions (26.7%) and neoplastic conditions (20%).
- Among the inflammatory conditions orbital cellulitis(30%) is the most common cause. Among the congenital causes hamartomas (viz. capillary hemangioma and lymphangioma) and choristomas(viz. dermoid and dermolipoma) have equal incidence of 12.9% followed by AV malformations(3%).
- Among the malignant causes secondaries(13.33%) are more common than the primary(6%). Among the secondaries, haematological malignancies (10%) are common followed by neuroblastoma (3%).

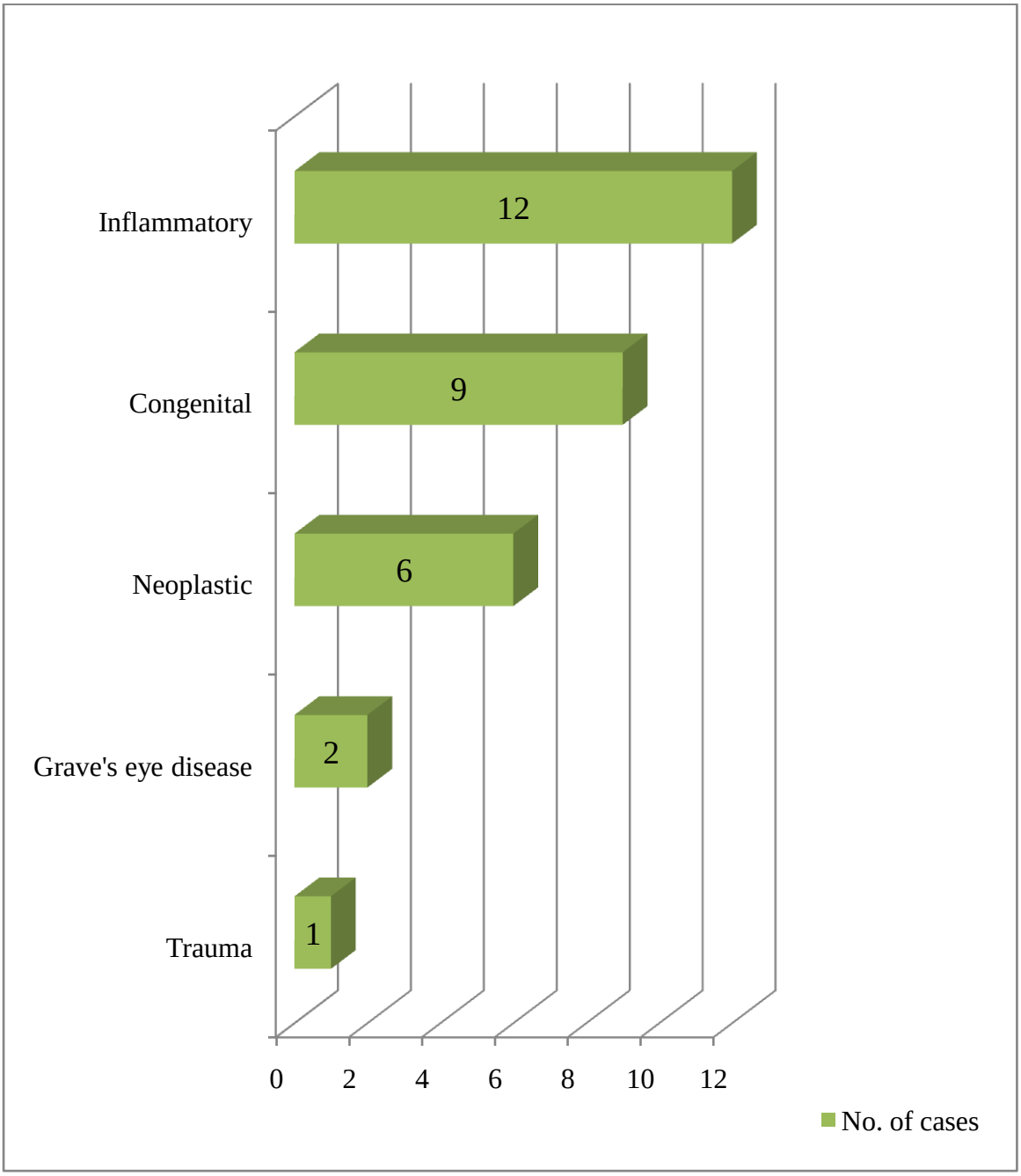


Chart.7 Bar Diagram showing Various Causes of Proptosis in our Study Population

PROPTOSIS DUE TO VARIOUS TUMOURS:

Causes	No. Of Cases	Percentage
Dermoid	4	13.33%
Capillary hemangioma	3	10.00%
Lymphangioma	1	03.33%
Retinoblastoma	1	03.33%
Glioma	1	03.33%
Secondaries	4	13.33%

Table.8 Proptosis due To Various Tumours

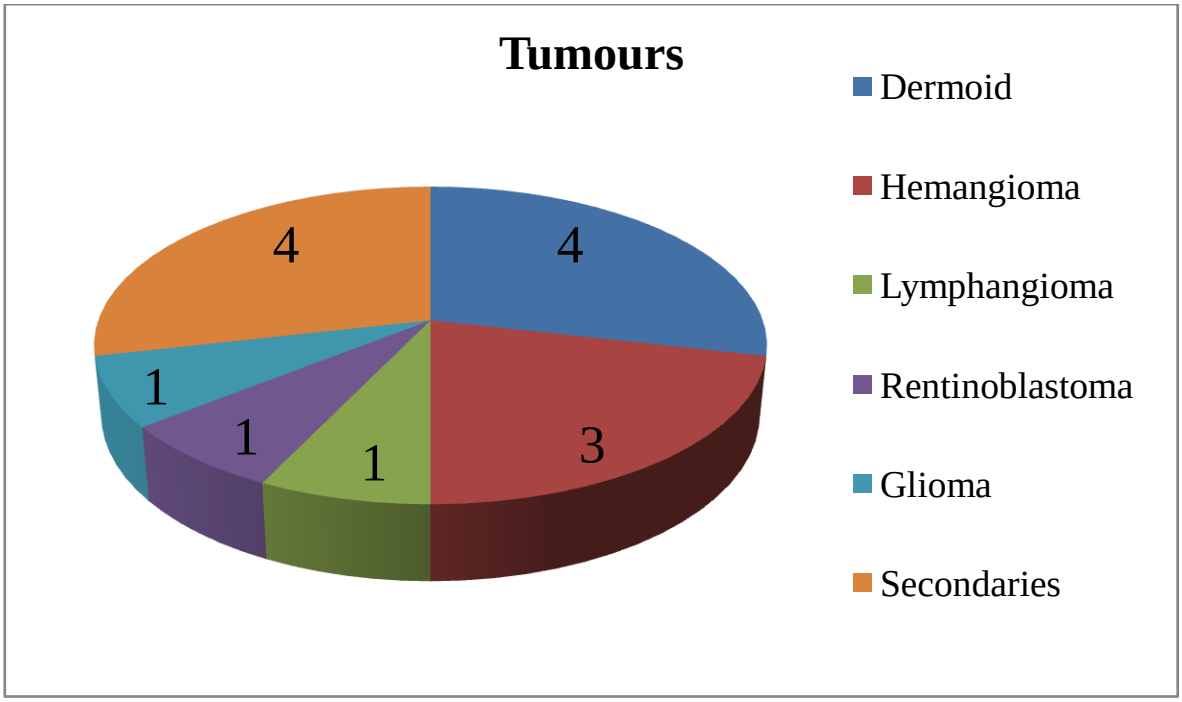


Chart.8 Pie chart showing Proptosis due To Various Tumours

- Among tumours benign tumours are more common than the malignant tumours.
- Among benign tumours dermoid (13.33%) is the most common cause followed by hemangioma (10%) .
- Among the malignant causes secondaries (13.33%) are more common than the primary(6.67%).
- Among the secondaries haematological malignancies(10%) are common followed by neuroblastoma (3%).

THE VARIOUS CLINICAL PRESENTATIONS OF PROPTOSIS :

Clinical Presentations	No Of Cases	Percentage of cases
Lid edema	18	60.00%
Chemosis of the conjunctiva	17	56.67%
Restriction of extraocular movements	16	53.30%
Pain	14	46.67%
Discharge	10	33.33%
Subconjuctival haemorrhage	5	16.67%
Dilated vessels over the lid	4	13.33%
Resistance to retropulsion	4	13.33%
Ecchymosis	2	06.67%
Pulsations	1	03.33%
Bruit	1	03.33%

Table.10 Various clinical presentations of Proptosis

- Analysing the various clinical presentations of proptosis, lid edema seems to be the most common presentation of proptosis.
- Apart from lid edema, chemosis , restriction of extra ocular movements and pain were the predominant complaints.
- All cases of orbital cellulitis presented with lid edema, conjunctival chemosis, pain and restriction of extraocular movements.
- Albeit, Proptosis without pain were more common among total cases than the painful one. Among the painless conditions dermoid was more common followed by capillary hemangioma. Among the painful

conditions orbital cellulitis was the most common cause followed by orbital secondaries due to haematological malignancies.

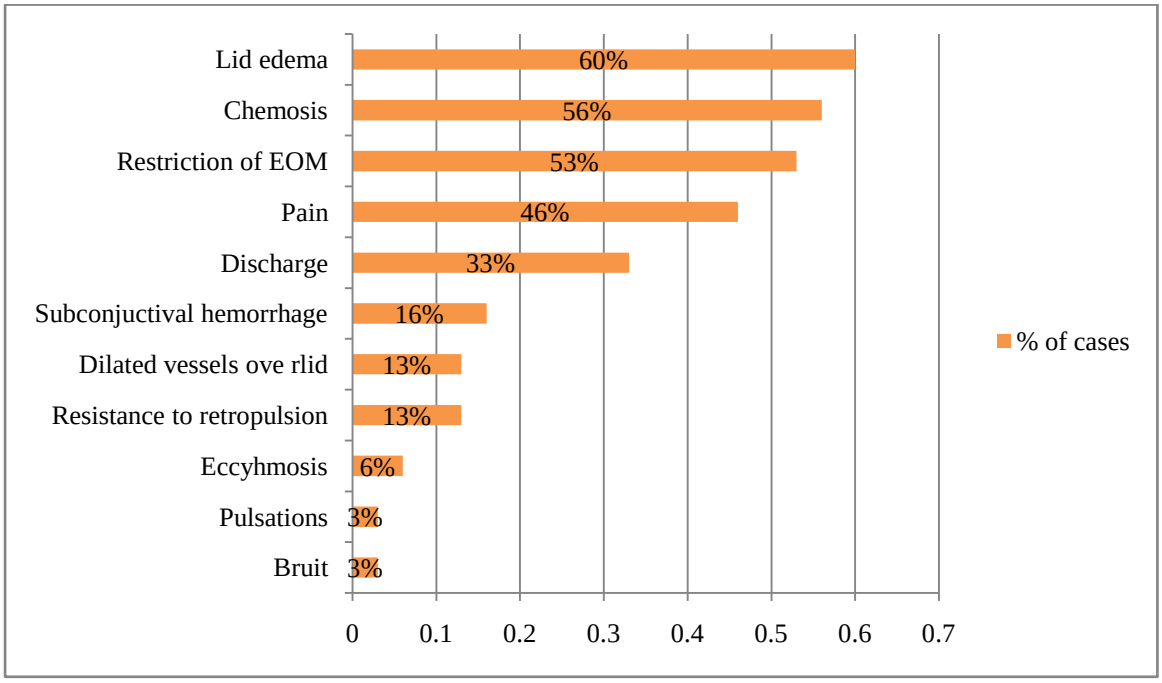


Chart.10 Various clinical presentations of Proptosis

- Dilated vessels were present over the lid in cases of capillary hemangioma and AV malformations.
- Subconjunctival haemorrhage was present in proptosis due to haematological malignancies and orbital hematoma following trauma.
- Eye discharges was present in cases of orbital cellulitis and orbital abscess.
- Resistance to retropulsion was present in orbital tumours due to haematological malignancies ,optic nerve glioma , secondaries due to neuroblastoma.
- Pulsations and bruit were present in case of AV malformations.

ANALYSIS OF VARIOUS RADIOLOGICAL INVESTIGATIONS

Investigations	No Of Cases	Percentage
X- RAY	5	16.67%
B - SCAN	11	36.67%
CT – SCAN	21	70%
MRI SCAN	9	30%

Table.11 Various Radiological Investigations done for Proptosis

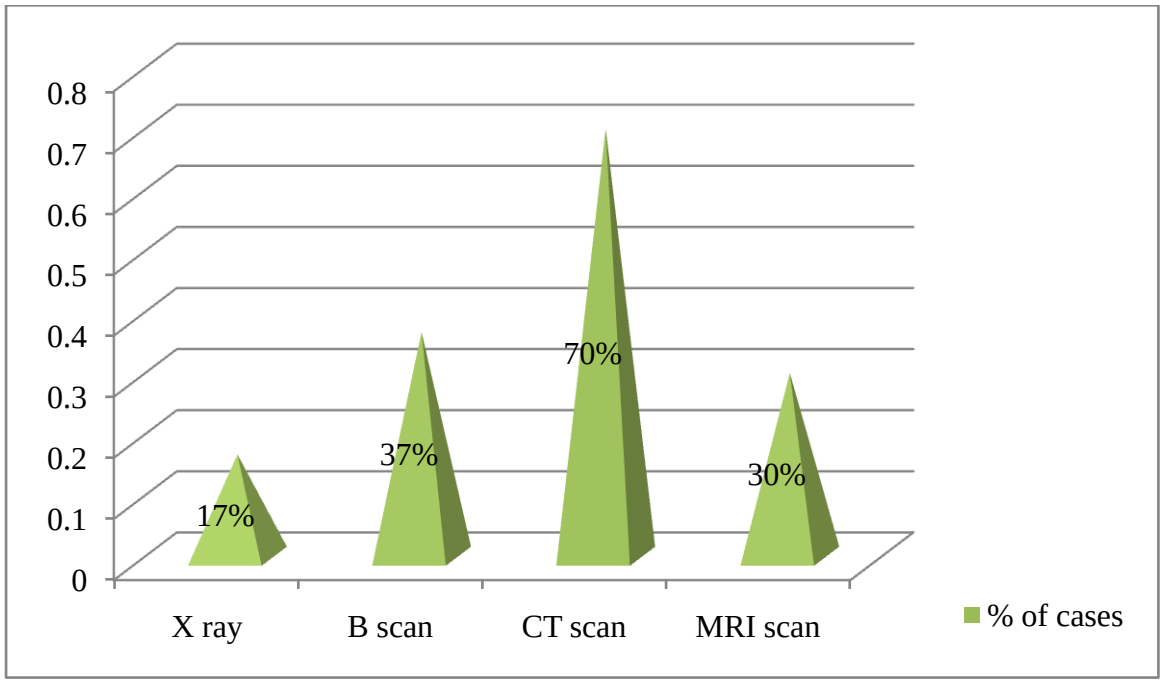


Chart.11 Various Radiological Investigations done for Proptosis

XRAY :

- 3 cases of orbital cellulitis revealed sinus haziness.
- 1 case of dermoid revealed bony defect.
- 1 case of trauma revealed haziness in extraconal space suggestive of hematoma which was later confirmed with CT scan.
- Also skeletal survey in neuroblastoma patient revealed widening of skull sutures and multiple lytic lesions in the long bones and skull suggestive of secondaries.

USG (B SCAN) :

- 3 Patients with orbital cellulitis were subjected to USG and they showed low reflective echoes of the soft tissues, with mottled appearance of orbital fat **"*dirty fat sign*".**
- 2 cases of thyroid ophthalmopathy showed a low to medium reflectivity echoes and enlargement of extraocular muscles with tendon sparing.
- 3 cases of capillary Hemangioma showed a typical well demarcated cystic lesion with high reflectivity and strong transmission.
- 2 patients of cysticercosis showed well circumscribed cyst with high reflective echoes inside the cyst wall suggestive of scolex.
- In case of Retinoblastoma diffuse mass filling the globe with areas of high echo reflectivity suggestive of calcification was noted.

- The safety and relative low cost of ultrasound on comparison to CT Scan and MRI gives it a distinct and practical advantage that will maintain its value in detecting orbital mass lesions.

CT SCAN :

- Among 30 cases of childhood Proptosis taken for study, in 21 cases diagnosis was confirmed with CT scan.
- Of all the orbital celluliti , 6 cases showed ethmoidal sinusitis, 2 cases showed sphenoidal sinusitis, 1 case showed both sphenoidal and ethmoidal sinusitis. All the cases of orbital cellulitis showed hyperdense lesions involving the soft tissues of the orbit.
- All 4 cases of dermoid showed non-enhancing cystic lesion with smooth margins. Bony defect was noted in 2 cases.
- Orbital abscess showed a ring lesion with wall enhancement with central hypodense region.
- Orbital hematoma revealed a isodense lesion laterally along lateral rectus muscle compressing the globe medially.
- Optic nerve glioma showed an isodense, fusiform mass lesion within the muscle cone arising from the optic nerve.
- CT scan of retinoblastoma showed calcification within the lesion.
- Most of the secondary malignant orbital tumours showed hyperdense, diffuse lesions with some orbital enlargement and bony involvement.

- 128 slice CT angiogram was done in child with suspected AV malformations which revealed the diffuse tortuous attenuating structures suggestive of low flow vascular malformation.

MRI SCAN :

- In 2 cases of dermoid, MRI done showed hyperintensity consistent with fat was seen in both T1W and T2W sequences.
- In 3 cases of capillary hemangioma, hypointensity in T1W images and iso to hyperintensity in T2W images were seen.
- In case of lymphangioma variable intensity was noted in T1W sequence.
- In 2 cases of cysticercosis both T1W and T2W revealed isointense lesion with eccentric scolex.
- In case of neuroblastoma heterogenous soft tissue mass displacing the globe both in right and left eye was noted along with reteroperitoneal hypointense mass originating from left adrenal gland consistent with primary neuroblastoma.

- Analysing the various radiological investigations used in proptosis evaluation, Plain x-ray is an easy investigation to carryout and it is extremely helpful in the diagnosis of the lesions involving and arising from the bones. But in proptosis due to soft tissue lesions it was not much helpful, as the increased soft tissue density was similar in most of the soft tissue lesions.
- Ultrasonography was very much useful in evaluation of orbital soft tissue lesions, but not in identifying the lesions arising from the bones. Ultrasound data has to be correlated with clinical findings to narrow down the differential diagnosis. This was consistent with the reports that USG is the most easily available and versatile test for evaluation of orbital soft tissues.
- CT scan proved to be the most important and essential one as it exactly delineate the mass by its size, shape, site and involvement of other adjacent structures[42]. Hence it helps a lot in narrowing down the causes of differential diagnosis and also help to outline the treatment modalities too. Hence among all the radiological investigations CT Scan was done more commonly than any other investigations i.e among 21 patients (70%) out of 30 patients.
- MRI has better resolution and soft tissue delineation than CT Scan hence it is preferred in vascular and soft tissue lesions more so in paediatric age group since it has no radiation hazard.

BLOOD INVESTIGATION

- Haemogram and differential count were done in all patients. Patients with orbital cellulitis and abscess showed an increase in neutrophil count and elevated ESR. Children with cysticercosis showed eosinophilia.
- Peripheral Smear was done in patients with haematological malignancies where in immature blasts cells with altered nuclear cytoplasmic ratio were seen.

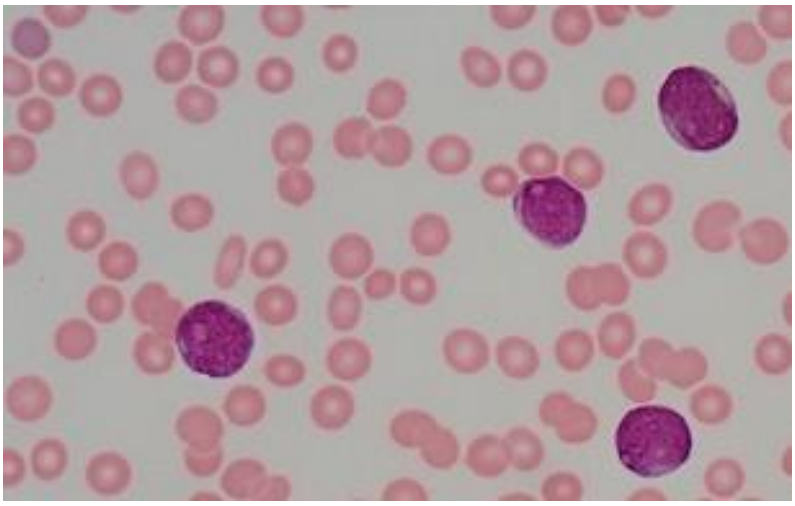


Fig. 33. Immature Blast cells with scanty cytoplasm and large nuclei suggestive of leukemia.

- Thyroid function test was done for two patients with suspected thyroid ophthalmopathy revealed low TSH and elevated T4, T3 suggestive of hyperthyroidism.

BIOPSY OF ORBITAL LESION

Most of the cases were diagnosed using non invasive laboratory and radiological investigations. Excisional biopsy of the lesion were done in dermoid for confirming the diagnosis and for therapeutic purpose. Retinoblastoma was confirmed by histopathological examination after enucleation.

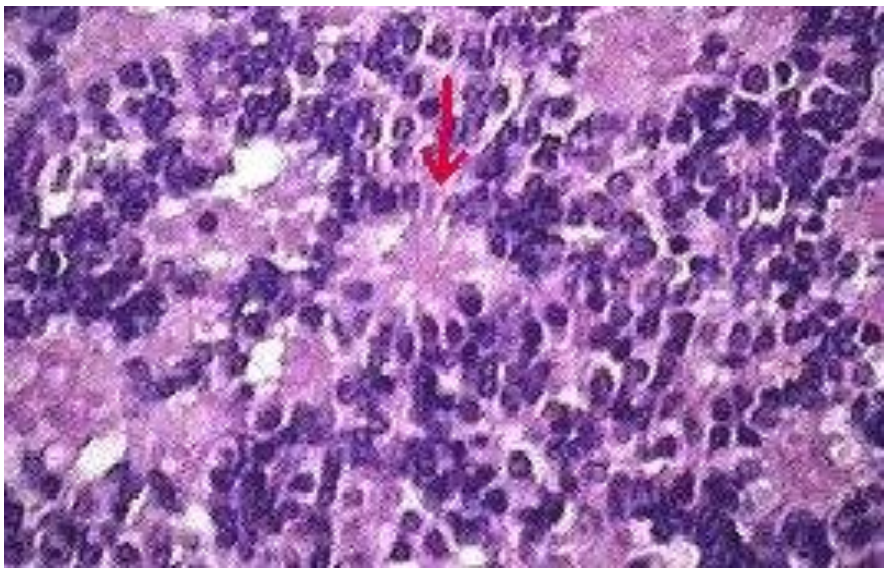


Fig. 34.HPE Showing rosettes suggestive of Retinoblastoma

ANALYSIS OF THE VARIOUS TREATMENT MODALITIES :

- All 9 cases of orbital cellulitis are treated with parenteral antibiotics , anti-inflammatory drugs along with topical antibiotics.
- The child with orbital abscess was started empirically on broad spectrum parenteral antibiotics and anti-inflammatory drugs along with topical antibiotic. After drainage of pus under general anaesthesia pus was sent for culture and sensitivity and antibiotic was changed accordingly.
- All cases of capillary hemangioma was treated with topical 0.05% timolol gel forming solution and oral propranolol. Two cases showed resolution in size of the proptosis.
- All cases of proptosis due to thyroid are treated medically with antithyroid drugs and they showed good respose to treatment during follow up.
- All the cases of dermoid showed good results following excision of the tumour with preservation of the vision.
- Proptosis secondary to retinoblastoma was treated by enucleation followed by radiotherapy and chemotherapy.
- Leukemic infiltration of the orbit was treated by chemotherapy. Two cases of leukemia showed poor prognosis inspite of treatment, because of their extensive metastasis.

- Secondaries due to neuroblastoma was managed with radiotherapy and chemotherapy.

Etiology	**Total Cases**	**Medical**	**Surgical**	**Chemo therapy**	**Radio therapy**	**Observation**
Congenital	9	3	4	-		2
Inflammatory	12	11	1	-	-	-
Malignancy	6	-	2	3	1	-
Trauma	1	-	-	-	-	1
Endocrine	2	2	-	-	-	-
Total	**30**	**16**	**7**	**3**	**1**	**3**

Table.12 Analysis Of The Various Treatment Modalities Used in Proptosis

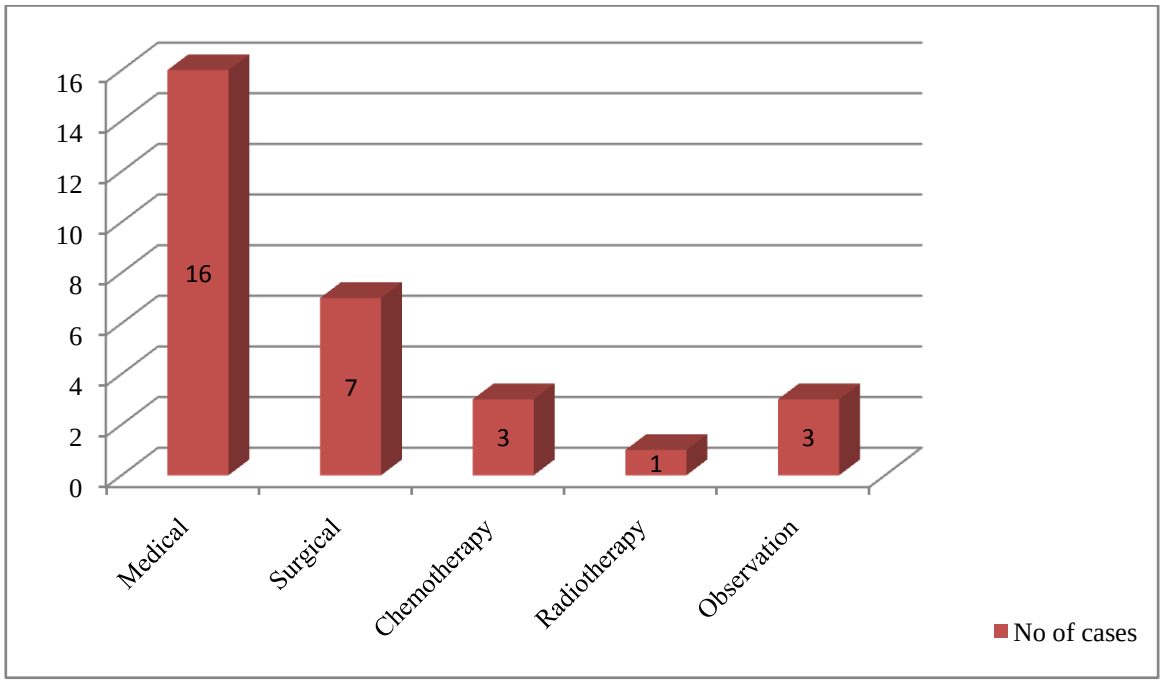


Chart.12 Analysis Of The Various Treatment Modalities Used in Proptosis

ANALYSIS OF OUTCOME OF VARIOUS TYPES OF PROPTOSIS

Various Causes	Total Cases	Cured	Improved	Static	Worsened
Orbital cellulitis	9	7	2	-	-
Orbital abscess	1	-	1	-	-
Orbital hematoma	1	1	-	-	-
Cysticercosis	2	1	1	-	-
Graves eye disease	2	-	2	-	-
Dermoid cyst	3	2	1	-	-
Dermolipoma	1	1	-	-	-
Hemangioma	3	-	2	1	-
Lymphangioma	1	-	-	1	
AV malformations	1	-	-	-	1
Retinoblastoma	1	-	1	-	-
Optic nerve glioma	1	-	1	-	-
Secondaries	4	-	1	-	3
Total cases	**30**	**12**	**12**	**2**	**4**

Table.13 Analysis Of Outcome Of Various Types Of Proptosis

- Out of the 9 cases of orbital cellulitis 7 cases got cured with parenteral broad spectrum antibiotics and 2 patients had improvement in symptoms.
- A case of orbital abscess got improved with surgical drainage of abscess and with broad spectrum iv antibiotics.
- Out of the 2 cases of orbital cysticercosis 1 got completely cured and 1 child improved clinically with residual lesion in the imaging.

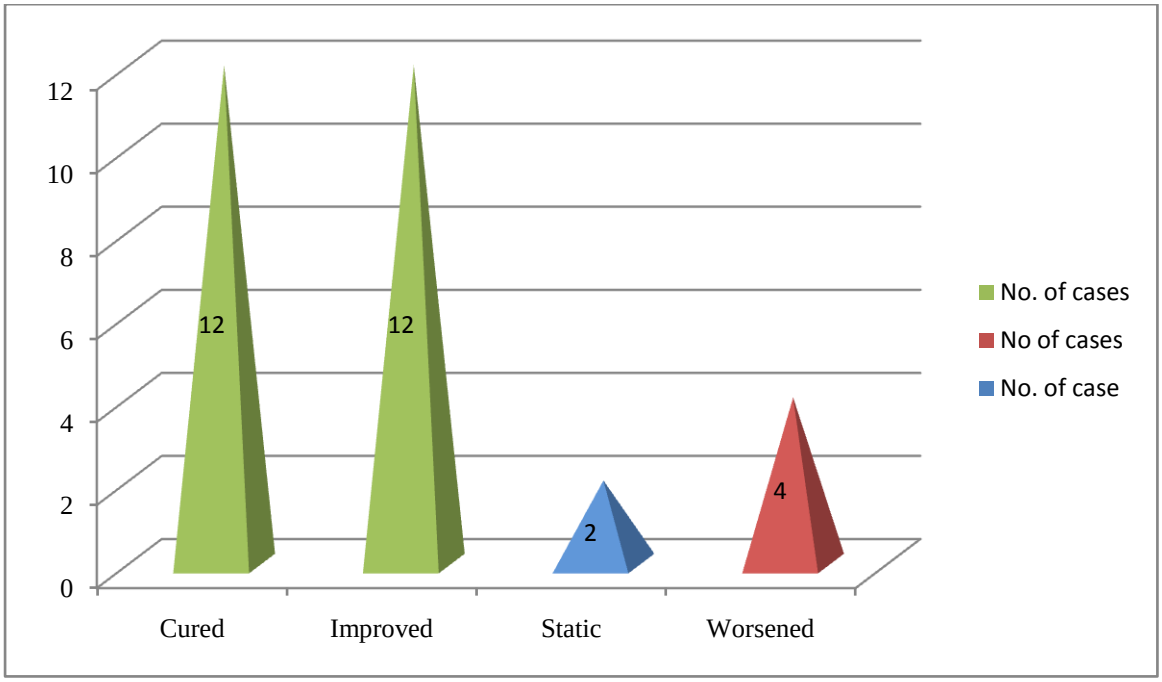


Chart.13 Analysis Of Outcome Of Various Types Of Proptosis

- All the dermoid, dermolipoma patients had got cured after surgical removal with good postoperative vision.
- 2 cases of capillary hemangioma had marked reduction in size of proptosis after topical timolol gel forming solution and oral propranolol. One child had no change in the size of proptosis.

- 2 cases of Grave's eye disease was treated medically with antithyroid drugs. Both of them showed good improvement .
- 1 case of orbital hematoma improved after three weeks of observation and steroid therapy.
- 1 case of optic nerve glioma was sent for neurosurgical intervention which got improved.
- 1 case of neuroblastoma was sent for chemotherapy and radiotherapy which got worsened.
- Among 3 cases of haematological malignancies which were treated with chemotherapy 1 got improved and 2 got worsened.

DISCUSSION

- **M loganathan et al[3]** in 2014 conducted a study on childhood Proptosis among 50 cases which revealed orbital cellulitis was the most common cause of childhood Proptosis which is similar to our study. This study also showed that axial Proptosis were more common than eccentric. Proptosis among malignancies, secondaries were more common than primary. These results were comparable to my study.
- According to the study conducted by **Bakshi et al[9] in 2008** among 104 cases of malignant childhood Proptosis secondary tumours (59.6%) were more common than primary tumours (51%) which is similar to my study.
- A study done by **Snehal r thakre[5] et al** in 2016 revealed malignant ocular and extraocular tumours being the most common cause of childhood proptosis. But in my study inflammatory lesions were the most common cause of childhood Proptosis.
- According to the study conducted by **Zahir Shah Et Al** "Diagnostic role of CT scan in Proptosis" showed that tumour were the most common (56 %) followed by inflammatory lesions (24%).Of these tumours primary malignancies like retinoblastoma and optic nerve glioma topped the list. But in my study inflammatory lesions were most common among childhood proptosis followed by congenital conditions. And also among tumours secondaries were more common.

- In a study done by **Belmekki M[10] In 1999** among 54 cases of Morocan children with proptosis retinoblastoma was found to be the leading cause of proptosis whereas in my study inflammatory lesions was found to be the leading cause of proptosis.
- In an unpublished study[43] conducted by IRCH (Institute Rotary Cancer Hospital) and AIIMS(All India Institute Of Medical Sciences) at NewDelhi among 45 children with proptosis , 80% were unilateral and 17% were bilateral which is similar to my study. This study also showed that retinoblastoma followed by rhabdomyosarcoma was the most common cause of proptosis but in my study inflammatory lesions were the most common cause of proptosis. This study also shows that AML was the most common cause of bilateral proptosis and this supported my data wherein haematological malignancies leads the cause of bilateral proptosis.

SUMMARY

30 Patients with proptosis in the paediatric age group who attended and referred to the outpatient department of Orbit and Oculoplasty Services in RIOGOH, Chennai between March 2016 and September 2016 were analysed.

The analysis includes the various causes of proptosis, variability in their presentation, the efficacy of various investigations to diagnose proptosis and the outcome of various treatment modalities.

The findings of the analysis are as follows:

i. Of the total 90 cases of proptosis attended the hospital during the study period , 30 were children upto 14 years of age (33.33%).
ii. The incidence of proptosis was more in males (53%) compared to females (47%) but the difference is not significant.
iii. 18 cases (60%) of patients presented with axial proptosis and 12 cases (40%) were eccentric.
iv. 25 cases (83%) presented with unilateral proptosis and 5 cases (17%) with bilateral proptosis.
v. Of the 30 cases, 11 (37%) were acute in onset and 19 (63%) were chronic.
vi. The common aetiologies for proptosis in children were inflammatory (33%) followed by neoplastic (20%) esp. secondaries.

vii. Among the inflammatory causes orbital cellulitis was the most common cause(30%) and all cases of inflammatory proptosis were acute in onset .

viii. Leukemic infiltration was the commonest cause of proptosis due to secondary orbital infiltration.

ix. Plain X ray and CT scan were useful tool in the diagnosis of proptosis due to bony lesions.

x. Ultrasonography was very useful in diagnosing soft tissue and cystic lesions but it has to be correlated with clinical findings and CT scan reports to narrow down the differential diagnosis and also to decide about treatment modalities especially in tumours.

xi. CT scan is a very useful investigation and it remains the essential of investigation in proptosis. It helps in locating the intracranial extension of the orbital lesions, which is necessary to plan the mode of treatment. It is also very helpful to detect early soft tissue and bony lesions which cannot be picked up by the USG.

xii. Most of the soft tissue inflammatory lesions were managed by medical treatment. The mass lesions (tumours and secondaries orbit) causing the proptosis has to be tackled by combined modalities viz. surgery, chemotherapy and or radiotherapy in association with various specialities like neurosurgery, ENT, oncologist.

xiii. Among all cases inflammatory lesions had better outcome.

CONCLUSION

- This study reveals that the aetiology of proptosis in children up to 14 years of age is definitely different from that in adults. Because in adults thyroid disease is the most common cause wherein children inflammatory condition is the predominant cause .
- There was no much sex difference in the incidence of childhood proptosis. Most of the cases were of axial and unilateral in presentation.
- Inflammatory lesions and congenital causes are the two broad groups which accounts for majority of cases of childhood proptosis. Overall the single most common aetiology of a childhood proptosis is orbital cellulitis.
- Among the malignant neoplasms the secondaries were more common than the primary orbital tumours and haematological malignancy accounts for majority of cases of secondaries in the orbit in children.
- X-ray and USG are earlier and cheaper investigations in diagnosing the proptosis. CT and MRI scans are the best modality of investigation in all cases of proptosis to detect the early lesions within the orbit and helps to identify the extent of lesion in planning the further management.

The exact early diagnosis of proptosis and timely referral to appropriate expert orbital surgeon is mandatory to save both vision and life of the patients .

BIBLIOGRAPHY

1. Frequency of orbital disease : In : Henderson JW, FarrowGM Orbital tumors; 3rd edition ; Newyork.
2. Sindhu K, Downie S, Ghabrial R, Martin F. Aetiology of childhood proptosis. J Paediatr Child health 1998; 34:374-6.
3. M. Loganathan, M. Radhakrishnan. "An Etiological Analysis of Childhood Proptosis". Journal of Evolution of Medical and Dental Sciences 2014; Vol. 3, Issue 22, June 02; Page: 6158-6162,
4. Chaudhry, Imtiaz A., Elzaridi, Elsanusi, Shamsi, Farrukh A., & Riley, Fenwick (2005). Childhood proptosis. Saudi Journal of Ophthalmology, 19(1), 15-25.
5. Thakre SR, Mishrikotkar JP, Wankhede SU,Deshpande SA. Childhood proptosis: A case of missed diagnosis. Sudanese J Ophthalmol 2014;6:33-5.
6. Sethi A, Ghose S, Gujral S, Jain P, Kumar R. Childhood proptosis: The invaluable but overlooked peripheral blood smear. Indian J Ophthalmology 2001; 49:121-3.
7. Sah KP[1], Saiju R[2], Roy P[1], Kafle S[1] JNMA J Nepal Med Assoc. 2013 Oct-Dec;52(192):576-9
8. Wright JE[1], Sullivan TJ, Garner A, Wulc AE, Moseley IF. Ophthalmology. 1997 Jun;104(6):905-13.
9. Bakhshi Sameer, Singh Preetpaul, Chawla Nikhil. Malignant Childhood proptosis. Study of 104 cases. J Paediatr Hematology Oncology 2008; 30:73-76.
10. Belmekki M, El Bakkali M, Abdullah H, et al. Epidemiology of orbital processes in. 54 cases; J Fr Ophthalmol 1999; 22: 394-8.
11. Last R.J: Eugene Wolff's Anatomy of eye and orbit. WB Saunders & Co. 1968, 6 edi , 9-4.
12. Williams P.C. Warwick R: Gray's Anatomy. 36 edi Churchill Livingstone, 1990: p-209.
13. Last R.J, Anatomy regional and applied . Churchill livingstone. 8th ed. Orbital contents.
14. Hogan M.J. Zimmerman L.E, Ophthalmic pathology, an atlas and textbook. 2 edi Philadelphia, W.B. Saunders 7 Co. 1962.
15. Jones IS, Jakobiec FA, diseases of the orbit. Hagerstown, MD, Harper & Row, 1979, pp 17-30.

16. Grossniklaus HE, Yanoff M; orbit. In Tasman W. Jaeger EA (eds);Foundations of Clinical ophthalmology. Philadelphia, JB Lippioncott, 1994, pp 1-36.
17. Rischbeith R.H.c, Bull J.W.D: Significance of enlargement of the superior orbital fissure. Br J Radial 1958, 31: 125.
18. Susal AL: Vascular studies of the orbital cavity. Ophthalmology88:548. 1981.
19. Rootman J: Diseases of the orbit. Philadelphian, JB Lippincott, 1988, pp 1-612.
20. Duke Elder S: System of ophthalmology. Vol 3, Normal and abnormal development. Part 2, congenital deformities .ST Louis Mosby, 1963.
21. Blodi F.C, Developmental anomalies of skull affecting the eye. Arch Ophthalmol 1957: 593.
22. Permberton J.W. Freeman: Craniosynostosis. Am J Ophthalmol 1962. 54: 641.
23. Appalanarasayya K, Devi O.B: Teratoma of Orbit Int Surg 1970, 54: 301.
24. Jakobiec FA, Jones IS: Orbital inflammations. In Duane TD (ed):Clinical Ophthalmology, 2nd ed, New York, Harper & Row 1980, pp 1-75.
25. Haynes R.E, Cramblett H.G, Acute ethmoiditis its relationship to orbital cellulites. Am J Dischild 1967 , 114 : 261.
26. Jarrett W.H, Geetman F.A; Ocular complications of infections in the paranasal sinuses. Arch Ophthalmol 1969, 81: 683.
27. Mottow LS, Jakobiec FA: Idiopathic inflammatory orbital pseudotumor in childhood, I: Clinical characteristics. Arch Ophthalmol 96: 1410, 1978.
28. Werner S.C.: The eye changes of Grave's disease. Overview Mayo clinic proc. 1972, 47:969 Retter
29. Lioyd G.A.S: Vascular anomalies in the orbit. CT and angiographic diagnosis. Orbit 1982, 1: 45.
30. Jones: Lymphangioma of ocular adnexa – An analysis of 62 cases. Trans Am Ophthalmol soc 1959, 57: 602.
31. Wright JE, Sullivan TJ, Garner A, et al orbital venous anomalies. Ophthalmology. 1997; 104: 905- 913.
32. Eckman P.B, Fountain F.M: Unilateral proptosis – Association with A.V. Malformations – I. Arch Neurol 1974, 31(50): 350.
33. Illiff WJ, Green WR: Orbital tumors in children. In Jakobie FA (ed): Ocular and Adnexal Tumors. Birmingham, Aesculapius, 1978, pp 699-684.
34. Jenkin D, Angyalfi S, Becker L, et al . Optic glioma in children: surveillance, resection, or irradiation? Int Radiation Oncology Biol. 1993 ; 25: 215- 225.

35. Mohan, Hari; Sen, D K "Primary Tumours of the Orbit causing Unilateral Proptosis: part incidence and clinical features" Indian J Ophthalmol Vol: 21 (4) 1973 Dec. p.161-170..
36. Crist W, Gehan EA, Ragab AH, et al . The Third Intergroup Rhabdomyosarcoma Study .J Clin Oncol. 1995; 13: 610-630.
37. Raldon Hugh "Unilateral proptosis due to monostotic fibrous dysplasia" Ann Ophthalmol Vol: 8 (1) 1976 Jan. p.45
38. Char DH, Miller T, Kroll S. Orbital metastases: diagnosis and course. Br J Ophthalmol. 1997; 81:386-390.
39. Kennedy RE: An Evaluation of 829 orbital cases. Trans Am Ophthalmol Soc: 82; 134-155, 1984Krohel G.R. Stewart W.B, Chavis R.M: Orbital diseases – A practical approach. Grune & Stralton. 1981, p 32.
40. Drews L.C: Exophthalmometry and a new exophthalmometer. Trans Am Ophthalmol Soc 1956, 54; 215.
41. Fischer Y.L, the current status of ophthalmic 'B' scan ultrasonography. J Clin ultrasound 1975, 3: 219.
42. Dallow RL: Momuse KJ, Seber AL, Wray SH; Comparison of ultrasonography, computerized tomography and radiography, ophthalmology 85; 1218, 1978.
43. Ganessan K, Bakhshi S. Proptosis in children: Approach. Indian J Med Pediatr Onc. 2004; 25 Supp : 33-34.

PROFORMA

Name :

Age/ Sex :

O.P/I.P No :

Complaints :

History of presenting illness:

History of Proptosis :

Age of onset

Onset: sudden/gradual

Duration: acute/chronic

Unilateral/ bilateral

Variability with cough/ posture/ valsalva maneuver/ crying/ sneezing

H/O Pain: onset/ nature/ progression/ severity/ aggravating and reliving factors

H/O Defective vision/ colour vision/ field of vision/ diplopia

H/O Fever/ loss of weight/ headache/ vomiting/ loss of consciousness

H/O Trauma

H/O ENT discharge, blockage of nose

H/O Bleeding gums/ epistaxis

H/O Contact with pet animals

H/O Thyroid symptoms

Past History:

H/O Similar episodes

H/O TB / Syphilis/ Malignancy

H/O Thyroid/ ENT problem

H/O Any ocular surgery

Personal History:

Vegetarian/non-vegetarian

Birth history-birth trauma/mode of delivery

Family History:

Tuberculosis / Thyroid disorder

Any similar problems in the siblings or other family members

Treatment History:

Medical/ Surgical/ Chemotherapy/ Radiotherapy/ Others

General Examination:

Built - well/ moderate/ ill

Nourishment - well/ moderately/ ill

Consciousness, Orientation

Anemia/ Jaundice/ Cyanosis/ Clubbing/ Lymphadenopathy

Vitals:

PR/BP/RR/temp

Local examination

Head posture

Facial asymmetry

RIGHT EYE	EXAMINATION	LEFT EYE
	Visual Acuity	
	Eyelids	
	Eyelashes	
	Extraocularmovements	
	Conjunctiva	
	Cornea	
	Anterior Chamber	

	Iris	
	Pupil	
	Lens	
	Proptosis	
	Axial/Eccentric	
	Compressibility	
	Resistance To Retropulsion	
	Valsalva Manoeuvre	
	Pulsations	
	Orbital Margins	
	Tension	
	Fields	
	Exophthalmometer	
	Fundus	

Other system examinations:

CVS/ RS/ ABDOMEN/ CNS

Other Consultations:

Paediatrics/ ENT/ Neurology/ Endocrinology/ Radiology/ Oncology

Treatment History :

Provisional Diagnosis:

Investigations:

Laboratory:

TC/ DC/ ESR/ Hb%

Urine albumin/ sugar/ deposits

Serum free T3, T4, TSH

Peripheral smear

Radiological:

Plain X-ray orbit/ sinuses/ optic foramen/ skull/ chest etc

USG A-Scan, B-Scan

USG abdomen/ head & neck

MRI/CT scan orbit/ sinuses/ brain (plain/contrast)

Orbital venogram

Biopsy: FNAC/ Incisional/ Excisional

HPE Report

Others:

Final Diagnosis:

Treatment: Medical/ Surgical/ Chemotherapy/ Radiotherapy/ Others

Followup:

INFORMATION TO PARTICIPANTS

INVESTIGATOR :

Dr. J. Keerthana
M.S. Ophthalmology PostGraduate,
Madras Medical College,
Chennai – 600008.

GUIDE :

Prof. Dr. Waheeda Nazir M.S., D.O.,
Chief, Orbit and Oculoplasty Services
Regional Institute of Ophthalmology,
Government Ophthalmic hospital,
Chennai – 600008.

NAME OF PARTICIPANT :

You are invited to take part in this research/ study titled **"EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE"**. The information in this document is meant to help you decide whether or not to take part in this study. Please feel free to ask if you have any queries or concerns.

PURPOSE OF THE STUDY :

To analyse the various causes of childhood proptosis, its various clinical presentations, investigations, treatment modalities and its outcome.

We have obtained permission from the Institutional Ethics Committee.

STUDY DESIGN : Descriptive study

STUDY PROCEDURE :

A detailed history of all the patients enrolled in the study, complete general examination, ocular examinations including visual acuity, examination of orbit, eye lids, anterior and posterior segments will be done. Slit lamp biomicroscopy, ophthalmoscopy, Hertel's exophthalmometry, visual fields, colour vision, refraction, intra ocular pressure and examination of proptosis will also be done. Laboratory investigations and Radiological investigations will be done to aid in etiological diagnosis. Patients will also be referred to

other departments like ENT, Haematology, Neurology, Oncology and Radiology to get expert opinion regarding diagnosis and whenever indicated. Patients will be treated accordingly and proptosis assessment will be done during follow up period.

Confidentiality of the information obtained from you?

You have the right to confidentiality regarding the privacy of your son/daughter ‘s medical information (personal details, results of physical examinations, investigations, and medical history).The information from this study, if published in scientific journals or presented at scientific meetings, will not reveal your son/daughter‘s identity.

How will your decision to not participate in the study affect you?

Your decision not to participate in this study will not affect your son/daughter‘s medical care or your relationship with the investigator or the institution. Your son/daughter will be taken care of and will not loose any benefits to which they are entitled.

Can you decide to stop participating in the study once you start?

The participation in this study is purely voluntary and you have the right to withdraw your son/daughter from this study at any time during the course of the study without giving any reasons. However, it is advisable that you talk to the research team prior to stopping the treatment/discontinuing of procedures etc.

Signature of Investigator Signature of Participant

INFORMED CONSENT FORM

Study title : **"EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE"**

Name of the Participant: _____

Name of the Principal investigator – **Dr. J. Keerthana**

Name of the Institution: Regional Institute of Ophthalmology,
Govt. Ophthalmic hospital,
Madras Medical College,
Chennai - 600008.

I _____ have read the information in this form (or it has been read to me). I am free to ask any questions and they have been answered. I exercise my free power of choice, hereby to give consent for my son/ daughter to be included as a participant in the study titled "**EVALUATION AND MANAGEMENT OF CHILDHOOD PROPTOSIS - A CLINICAL STUDY IN A TERTIARY CARE CENTRE".** I understand that my son/ daughter's participation in the study is voluntary and that I am free to withdraw him/her from the study at any time, without giving any reason, without his/her medical care or legal rights being affected.

I hereby give permission to the investigators to release the information obtained from my son/daughter as a result of participation in this study to regulatory authorities, Govt. agencies, and Ethics Committee. I understand that they are publicly presented.

(1) I have read this consent form and understood the information provided.

(2) I have had the consent document explained to me.

(3) I have been explained about the nature of the study.

(4) I have been explained about my rights and responsibilities by the investigator.

(5) I have informed the investigator of all the treatments my son/daughter is taking or have taken in the past including any native (alternative) treatments.

(6) I have been advised about the risks associated with my son/daughter's participation in the study.

(7) I agree to cooperate with the investigator and I will inform him/her immediately if my son/ daughter suffer unusual symptoms.

(8) I am aware of the fact that my son/ daughter can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital.

(9) I am also aware that the investigators may terminate my son/daughter's participation in the study at any time, for any reason, without my consent.

(10) I hereby give permission to the investigators to release the information obtained from my son/daughter as a result of participation in this study to the sponsors, regulatory authorities, Government agencies, and ethics committee. I understand that they may inspect his/her original records.

(11) I understand that my son/ daughter's identity will be kept confidential if his/her data are publicly presented.

(12) I have had my questions answered to my satisfaction.

(13) I consent voluntarily for my son/ daughter to participate as a participant in this study.

(14) I am aware, that if I have any questions during this study, I should contact the investigators. By signing this consent from, I attest that the information given in this document has been clearly explained to me and understood by me.

(15) I will be given a copy of this consent document.

Name and signature / thumb impression of the participant's parent/guardian :

(Name) _____

(Signature/Left thumb impression) _____

Date: _____

Name of the witness :

(Name) _____

(Signature) _____ Date: _____

Address and contact number : _____

Name and signature of the Investigator or his representative obtaining consent:

(Name) _____

(Signature) _____ (Date): _____

KEY TO THE MASTER CHART

1. Serial number
2. Name
3. Age
4. Sex
5. IP or OP number
6. Eye involved: R-right eye, L-left eye, B-both eyes
7. Laterality: U- unilateral, B- bilateral
8. Direction of proptosis: A-axial, E- eccentric
9. Onset of proptosis: A- acute, C –chronic
10. Associated signs:, DV_ dilated vessel, SCH- subconjunctival haemorrhage, D-discharge, E- ecchymosis, LE - lid edema, C - chemosis, MR- movements restricted, RP- resistance to retropulsion, PL-pulsation, P-pain, B-bruit
11. Vision: PL- perception of light, HM- hand movements, NC- not co-operative, R-right eye, L-left eye, V – both eyes.
12. Lab investigation: WNL- normal haemogram, LP- leukemic picture, PML- polymorpho leukocytosis, E- eosinophilia, L- lymphocytosis, A-anemia
13. Radiological Investigation - X-Xray, CT, U-B Scan, M-MRI
14. Speciality clinic: N-neurology, H-haematology, O-oncology,

R-radiology, OR-orthopaedics, P-Pathology, ENT- ear, nose & throat, P-paediatrician

15. Aetiology: PM- primary malignant neoplasm, SM- secondary malignant neoplasm, T-traumatic, C-congenital, INF-inflammatory, E-endocrine.

16. Provisional diagnosis

17. Treatment given: M-medical, S-surgical, R-radiotherapy, C-chemotherapy, NS-neurosurgical, COM-combined therapy, O-observation, E-endocrine,

18. Follow up: C-cured, I-Improved , S-Static, W-Worsened

MASTER CHART

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	Koush	46547	1	M	L	U	A	A	LE,C,MR,D,P	NC	PML	X,CT	ENT	INF	Orbital cellulitis	M	C
2	Parmeshwari	12987	7	F	B	B	A	C	LE,SCH,C,RP,MR,P	R 6/36 L 6/18	LP	CT	H	SM	Acute myeloid luekemia	C	I
3	Nandhitha	34573	1	F	R	U	A	A	LE,C,MR,D,P	NC	PML	X,CT	ENT	INF	Orbital cellulitis	M	C
4	Vishwa	23456	2	M	B	B	A	C	MR	NC	TFT	U	E	E	Hyperthyriodism	E	S
5	Dinesh	32167	8	M	B	B	A	C	LE	R 6/6 L6/6	TFT	U	E	E	Hyperthyriodism	E	S
6	Danush	23452	7	M	R	U	E	C	SCH,DV,MR,B,C,PL	R NO PL L 6/6	WNL	CT	V	C	Arteriovenous malformation	COM	W
7	Akshya	90864	2	F	R	U	E	C	LE,DV	NC	WNL	M,U	V	C	Capillary Hemangioma	M	I
8	Sowmya	56432	3	F	L	U	E	C	LE,DV	NC	WNL	M,U	V	C	Capillary Hemangioma	M	I
9	Pradeep kumar	54353	13	M	L	U	A	C	BD	R 6/9 L 6/6	WNL	X,CT,M	-	C	Dermoid Cyst	S	C
10	Sashmitha	12098	9	F	R	U	E	C	LE,DV,C	R6/18 L6/6	WNL	M,U	V	C	Capillary Hemangioma	M	S
11	Manjula	53782	12	F	R	U	A	C	LE,C,MR,D,P	R 6/60 L 6/6	PML	X,CT	ENT	INF	Orbital cellulitis	M	C
12	Krithika	38245	3	F	L	U	E	A	LE,C,MR,D,P	NC	PML	CT	ENT	INF	Orbital Abscess	M	C
13	Sameer	22097	2	M	L	U	A	A	LE,C,MR,D,P	NC	PML	CT,U	ENT	INF	Orbital cellulitis	M	C
14	Samuktha	45378	6	F	L	U	A	A	LE,C,MR,D,P	R6/6 L 6/9	PML	CT,U	ENT	INF	Orbital cellulitis	M	C
15	Rakesh	29634	4	M	B	B	A	C	LE,SCH,C,RP,MR,P	NC	LP	CT	H	SM	Lymphoreticular malignancy	C	W
16	Faruk	20987	1 1/2	M	R	U	A	A	LE,C,MR,D,P	R 6/18 L6/6	PML	CT,U	ENT	INF	Orbital cellulitis	M	C
17	Isakikumar	24543	9	M	L	U	A	A	LE,C,MR,D,P	R6/6 L 6/60	PML	CT	ENT	INF	Orbital cellulitis	M	C
18	Yeshwanth	83565	3	M	L	U	E	C	-	NC	WNL	CT,M	-	C	Dermoid Cyst	S	C
19	Srivadhan	10956	5	M	L	U	A	C	RP	R6/6 LHM	WNL	CT	O	PM	Optic Nerve Glioma	NS	I
20	Sristi	45035	1	F	R	U	A	C	WR	NC	WNL	CT,U	O	PM	Retinoblastoma	COM	I
21	Kumaresh	10643	6	M	R	U	E	C	-	R6/6 L6/6	E	U,M	-	INF	Cysticercosis	M	I
22	Neela	70565	3	F	B	B	E	C	LE,RP,MR,E,C,SCH,P	NC	WNL	X,M	O	SM	Neuroblastoma	COM	W
23	Kavya	10934	8	F	R	U	A	A	LE,C,MR,D,P	R 6/36 L6/6	PML	CT	E	INF	Orbital cellulitis	M	C
24	Dikshitha	56034	2 1/2	F	R	U	E	C	-	NC	WNL	M	-	C	Lymphangioma	O	S
25	Suganth	17407	5	M	L	U	E	C	-	R 6/6 L6/6	WNL	CT	-	C	Dermoid Cyst	S	C
26	Sengi	40286	4	F	L	U	A	A	SCH,MR,E,C,P	NC	WNL	X,CT	-	T	Orbital hematoma	O	C
27	Lokesh kumar	10735	4	M	B	B	A	C	LE,SCH,C,RP,MR	NC	LP	CT	H	SM	Leukemic Infiltration	C	W
28	Dheeran	64935	3	M	L	U	E	A	LE,C,MR,D,P	NC	PML	CT	ENT	INF	Orbital cellulitis	M	C
29	Glory	30867	8	F	L	U	A	C	-	R 6/6 L6/6	E	U,M	-	INF	Cysticercosis	M	C
30	Kameshwaran	10634	1	M	L	U	E	C	BD	NC	WNL	CT	-	C	Dermolipoma	S	C