CLINICAL PROFILE OF IDIOPATHIC ORBITAL INFLAMMATORY SYNDROME

DISSERTATION SUBMITED FOR

MS (Branch III) Ophthalmology

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

CHENNAI

APRIL – 2016

CERTIFICATE

This is to certify that dissertation entitled "**CLINICAL PROFILE OF IDIOPATHIC ORBITAL INFLAMMATORY SYNDROME "** is a bonafide done by **Dr.T.UMARANI** under our guidance and supervision in the Department of Orbit, Aravind Eye Hospital and Post Graduate Institute of Ophthalmology in Madurai during his residency period from July 2013to April2016.

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DECELARATION

I**, Dr.T.UMARANI** solemnly declare the dissertation titled "**CLINICAL PROFILE OF IDIOPATHIC ORBITAL INFLAMMATORY SYNDROME "** has been prepared by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other university board either in India or aboard

This dissertation is submitted to the **Tamil Nadu Dr. M.G.R Medical University**, Chennai in Partial Fulfilment of the rules and regulation for the award **of M.S. Ophthalmology (BranchIII**) to be held in April 2016.

Place: Madurai **Dr.T.UMARANI**
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ACKNOWLEDGEMENT

I take this opportunity to pay my respect and homage to **Dr.G.Venkatasamy,** our founder, whose ideals and philosophy have guided this institution in all its successful endeavours.

It is a proud privilege and pleasure to express my gratitude towards my esteemed guide **Dr. Usha Kim,** Chief- Orbit and Oculoplasty, Aravind Eye Hospital, Madurai for her constant support throughout the duration of my thesis.

I am deeply indebted to **Dr.N.Venkatesh Prajna**, Director of Academics and Head of the Department of Corneal Services for his constant encouragement, guidance and support throughout my residency.

I am very grateful to **Dr.R.D.Ravindran**, chairman of Aravind Eye Hospital for having created an environment enriched with all the facilities for learning and gaining knowledge. I am privileged to have on my side **Dr.P.Namperumalsamy,** Chairman Emeritus director of research, **Dr.G.Natchiar,** Director Emeritus (Human Resource Department), **Dr.M.Srinivasan,** Director Emeritus Director and other scholars of

Ophthalmology at Aravind eye Hospital, Madurai.

I am much grateful to **Mrs.R.Kumaragurupari,** Sr.Librarian**,** **Mr.R.Govindarajan,** Librarian**,** and **Mr. K. Ranjithraja,** Asst. Librarian**,** for their prompt and efficient response to my in numerable requests for articles and information.

I extent my sincere thanks to **Mr.B.Vijayakumar,** Biostatistician, for his valuable help in the statistical analysis of the study.

I would like to thank Mrs.Sundari, Mrs.Regitha and Miss. Geetha and other paramedical staff of orbit clinic for their cooperation during the study.

I extend my heartfelt words of gratitude to my beloved Husband and daughter for all their sacrifices and unfailing love towards me.

Last but not least, I thank my parents, Grandma, in-laws, friends and colleagues for their unwavering support.

CONTENT S

INTRODUCTION

Idiopathic orbital inflammatory syndrome or Non specific Orbital Inflammatory Disease, refers to a marginated mass-like enhancing soft tissue involving any area of the orbit. It is a benign, non-granulomatous orbital inflammatory process with characteristics of extra ocular, orbital and adnexal inflammation with no known local or systemic cause[1,6]. Idiopathic orbital inflammatory syndrome, also called as orbital pseudo tumor[2], was first described by Gleason in 1903 and by Busse and Hochhmein. In 1905, Birch-Hirschfield[2,3], characterized it as a distinct entity. In 1954 Umiker et al. named it as inflammatory pseudo tumor because of its propensity to resemble a malignant process. The terms, Nonspecific orbital inflammation and orbital inflammatory pseudo tumor can be used interchangeably.

Orbital inflammatory pseudo tumor accounts for 10 % of orbital tumors[3]. It most commonly occurs in the age group of 40 to 60 years, but it can also occur in children. There is no sex predilection. Though some bilateral involvement is possible in children, it is mostly unilateral. NSOI can either be localized or diffuse. For the case of localized, inflammation may affect the extra ocular muscles (termed as orbital myositis), lacrimal gland (termed as dacryoadenitis), sclera(termed as scleritis), uvea (termed as uveitis), and

the superior orbital fissure and cavernous sinus (termed as Tolosa-Hunt syndrome). In diffuse cases of NSOI, orbital fatty tissues are involved.

NSOI is found to be third most common orbital disease, only after thyroid eye disease and orbital lymphoma[1,3-7]. It remains a Diagnosis of exclusion, as it is diagnosed after excluding orbital tumor, thyroid eye diseases and systemic inflammatory diseases.[1,3,6]

Its histological characteristic is primarily inflammation. Despite a benign condition, it may show up as an aggressive clinical course, accompanied by severe vision loss and Oculo motor dysfunction. Various treatment options are available including surgery, steroids, chemotherapeutic agents and Irradiation.

Here we describe and evaluate clinical characteristics, orbital-imaging findings, treatment and outcome in patients with Non specific orbital inflammation.

REVIEW OF LITERATURE

Niphon chirapapaisan, Wanicha chuenkongkaew et al [1] described the clinical features and outcome of Orbital pseudo tumor. In a retrospective analysis for 10 years duration on patients with orbital pseudo tumor, they had identified 49 patients with a mean age of presentation at 43.75 years. The most common presenting symptom and sign was proptosis followed by ocular motor deficit. All patients were treated with corticosteroids, with clinical improvement seen in 40 patients.

Sonia J. Ahn Yuen et al[4] reported Distribution, Clinical Features, and Treatment Outcome of Idiopathic Orbital Inflammation. They studied 65 patients and reported that pain and periorbital swelling were the most common clinical features and were observed in 45 (69%) and 49 (75%) patients, respectively. The mean age at presentation was 45 years. Isolated dacryoadenitis followed by isolated myositis was the most common type.

Imtiaz A .Chaudhry, Farrukh A. Shamsi et al [5] provided an overview of spectrum of orbital pseudo tumor. They reviewed relevant literature and summarised findings regarding epidemiology, pathophysiology, diagnosis and treatment of IOIS. They had categorized the IOIS as myositis,

dacryoadenitis, anterior apical and diffuse processes. They had concluded that understanding the clinical features and differentiating it from other orbital diseases and timely implementation of treatment may help to prevent visual loss and other associated morbidity.

IN Br J Ophthalmology 2007: 91 (12): 1667-1670, **B N Swamy, P McCluskey et al**[6] described clinical features and treatment outcome of Idiopathic orbital inflammatory syndrome. They reviewed 98 patients with IOIS, diagnosed in Sydney eye hospital between 1995-2005.They had characterized the clinical and pathological features of 24 patients with biopsy proven IOIS. Swelling and mass was the most common presentation and Orbital fat was most commonly involved site. Sclerosing orbital pseudo tumor was the most common histo-pathological presentation in their study. They found that clinical and pathological features do not correlate treatment out come.

R.A Nugent and J. Rootman et al[7] described classification and CT features of acute orbital Pseudo tumor in AJR 137: 957-962. A retrospective computed tomographic study of 16 patients demonstrated that lesions occur in one of five specific anatomic patterns: anterior, posterior, diffuse, lacrimal, or myositic. The most common location was lacrimal followed by anterior pseudo

tumor. Posterior, diffuse, and myositic forms were equally frequent. Role of CT scan in evaluating therapeutic response was also discussed.

Imtiaz A .Chaudhry, Saif AL –Obaisi et al [12] described a case of unilateral recurrent orbital pseudo tumor presented with decreased vision, pain on eye movement. They attributed it to optic neuritis and macular edema, caused as a result of scleritis, which was rare presentation of orbital pseudo tumor. In their study they had insisted the importance of imaging in the establishment of correct diagnosis.

Taijitakanashi , Sunao Uchida et al [13] reported two cases of Churg strauss syndrome with orbital inflammatory pseudo tumor. They had described that orbital inflammatory Pseudo tumor and ischemic vasculitis may represent two essential characteristics of Churg strauss syndrome.

WH Chan, S Biswas, J Ashworth et al [14] reported a rare case of Bilateral Idiopathic orbital inflammatory syndrome involving all EOM with elevated Creatinine kinase level.

Michael E. Bernardino, Robert D. Zimmerman et al[15] described thickening and enhancement of scleral uveal rim,that represents a specific CT sign for diagnosis of Orbital Pseudo tumor.

Michael A.MAHR, Diva R.Salomao et al[16] Reported 4 cases of inflammatory orbital Pseudo tumor presented with extension beyond the orbit.

Luciana Souza cruzcaminha, Elisa Rebelo Pinto et al [17] reported a case of orbital pseudo tumor -a differential diagnosis of graves Ophthalmopathy.

F- X Borruat, P Vuilleumier et al[18] reported rare association of Transient ischemic attack with Idiopathic orbital inflammation (due to intra cranial extension).

TohruNishi, Yuji Saito et al [19] presented a rare case of histologically verified orbital pseudo tumor with intra cranial extension, accompanied by complete occlusion of Internal carotid Artery.

Satoshi Tsutsumi, Takuma Higo et al [20] reported a case of Orbital pseudo tumor associated with Retro bulbar hematoma.

Jack Anderson et al[21] reported a case of orbital pseudo tumor presenting as orbital cellulitis.

RecaiTurkoglu, NaciBalak [22] reported a case of orbital pseudo tumor with atypical presentation of vision loss as an initial manifestation.

Yu-Hung Lai, HweiZu Wang et al[23] reported a case of Bilateral orbital pseudo tumor with supra sellar and pulmonary involvement.

Norman C Charles, Roger E.Turbin[24] reported a case of bilateral sclerosing orbital pseudo tumor with intra cranial spread.

Jeffrey w.Berger, Peter A.D .Rubin [25] described a case report and review of literature of Pediatric orbital pseudo tumor.

ANATOMY OF THE ORBIT [8] [11]

The two bony orbits are quadrangular in shape resembling truncated pyramids and is located between the anterior cranial fossa and the maxillary sinuses, former above and latter below it. Each orbit has been formed by seven bones .The bones forming orbit includes frontal, lacrimal, ethmoid, palatine, zygomatic, maxilla, and sphenoid. The medial walls of the both orbits are in parallel to each other and they have in contact with the ethmoid and sphenoid sinuses. These two sinuses separates the orbits from the nasal cavities. The lateral wall of orbit lies at 45 degrees to the medial wall. Each lateral walls of the each orbits are at 90 degrees to each other. Posteriorly middle cranial fossa and anteriorly muscular temporal fossa are separated from orbit by lateral wall.

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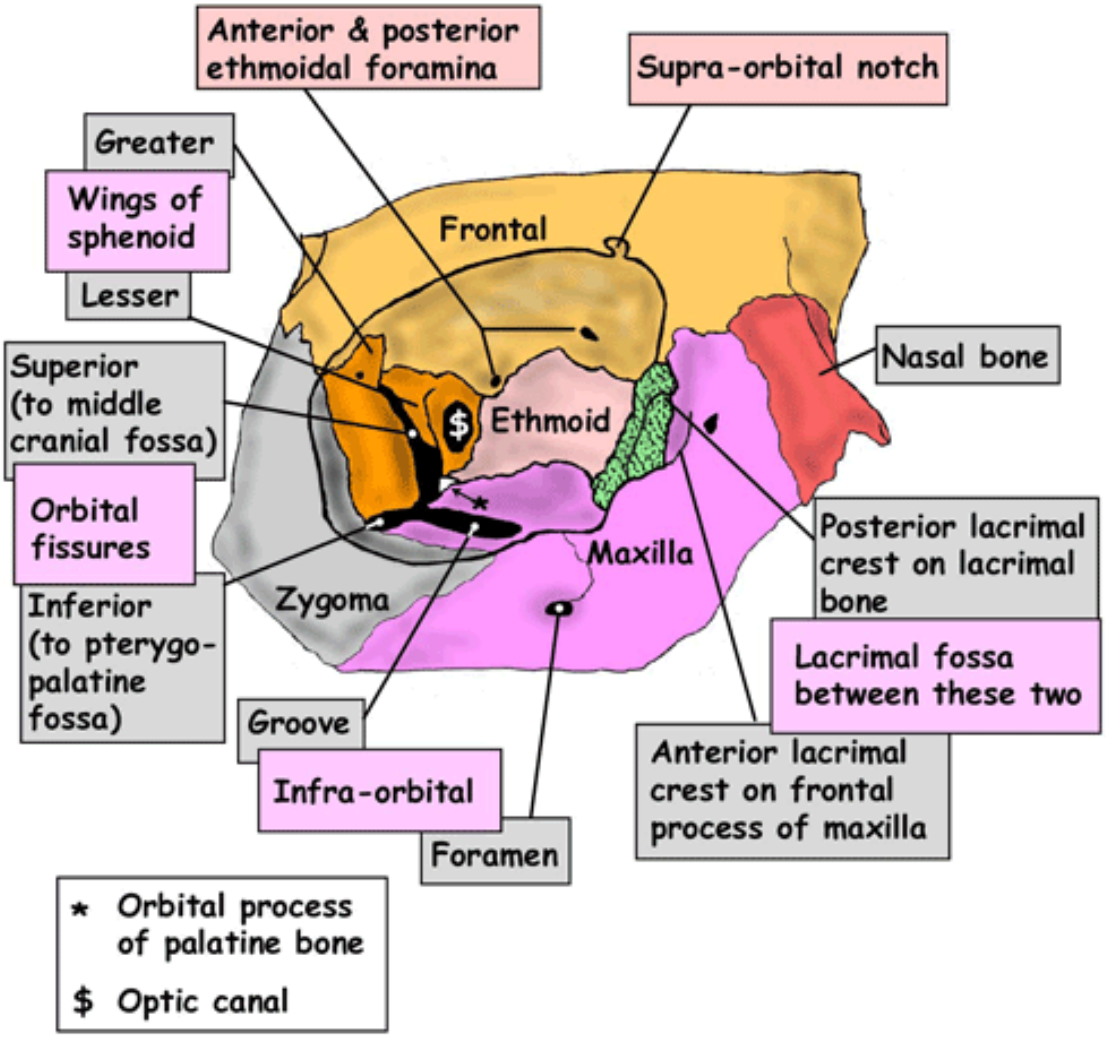
The depth of the orbit is 42 mm and 50 mm along with the medial wall and lateral wall respectively. The width of t he base of the orbit is 40 mm in width and its height is 35 mm . The intra orbital width is 25mm and the extra orbital width is 100 mm. The volume of each orbit is about 29 ml. The volume of the orbit is 4.5 times that of the eyeball (a ratio of 4.5:1)

EYE - BONES OF RIGHT ORBIT

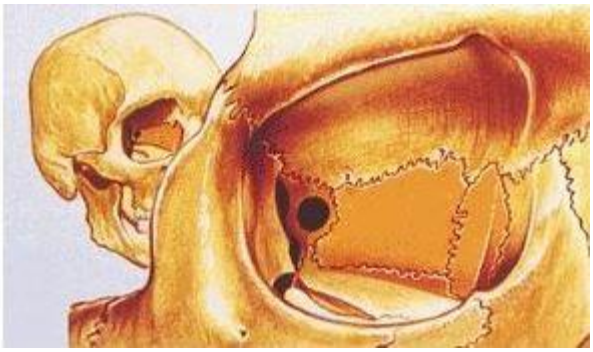


WALLS OF THE ORBIT

The four walls of orbit are medial wall, lateral wall, roof and floor. These walls meet at the superior internal, superior external, inferior internal and inferior external angles of the orbit.

Medial Wall

The medial wall of the orbit is quadrilateral in shape and is formed by the frontal process of the maxilla, the orbital plate of the ethmoid bone ,the lacrimal bone and the body of the sphenoid from front to back. The anterior part of the medial wall bears the lacrimal sac fossa present in anterior part of medial wall ,and it is continuous inferiorly with the naso lacrimal canal. The lacrimal fossa is formed anteriorly by the anterior lacrimal crest of the maxillary bone and posteriorly by the posterior lacrimal crest of the lacrimal bone.

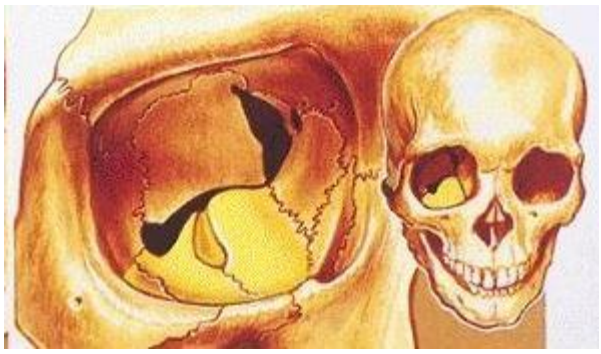


Medial wall of Orbit

The medial wall is the thinnest wall of the orbit and is frequently eroded by chronic inflammatory lesions, cysts and neoplasms that originate from adjacent air sinuses. It is easily fractured during injuries and during orbitotomy operations. During surgery ,due to injury to ethmoidal vessels along this wall, hemorrhage will occur. The medial wall can be easily visualized with routine PA radiographs of the orbit.

Inferior orbital wall (floor)

The floor of the orbit is triangular in shape and it is the shortest of all the walls. Floor is formed by orbital surface of the maxillary bone medially, the orbital surface of the zygomatic bone laterally and the palatine bone posteriorly. Inferior orbital fissure separates posterior part of the floor of the orbit from the lateral wall. The infra orbital groove extends from inferior orbital fissure and it further extends down anteriorly as a canal. Infra orbital canal opens at the infra orbital foramen located just below the infra orbital rim. The Infra orbital foramen transmits the infra orbital nerve ,its artery and its vein . Infra orbital vein is the one, which connects the inferior ophthalmic vein to the facial vein.



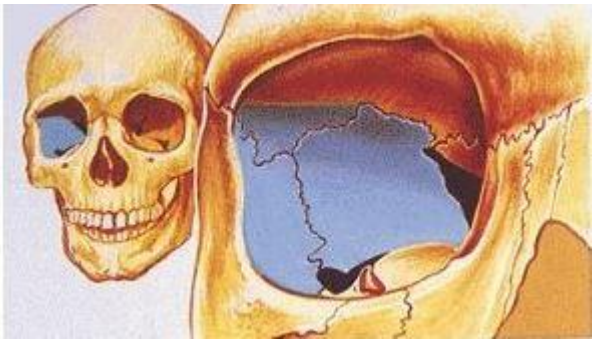
Floor

The orbital floor is easily invaded by tumors of the maxillary antrum and also commonly involved in ‘blow out fractures’. It can be best visualized with postero anterior radiographs of Orbit. The orbital floor can be approached by inferior orbitotomy (antral approach) easily.

Lateral Wall

The lateral wall of the orbit is triangular in shape. It is formed anteriorly and posteriorly by the zygomatic and the greater wing of the sphenoid bone respectively. On the posterior part of the lateral wall there is small bony projection called (spina recti lateralis and it gives origin to a part of the lateral rectus muscle. A projection, called lateral orbital tubercle of Whitnall is present on anterior part of lateral wall. It also gives attachment to the check ligament of the lateral rectus muscle, and to the suspensory ligament of the eyeball.

The lateral wall is separated from the roof by the superior orbital fissure posteriorly and from the floor by the inferior orbital fissure.



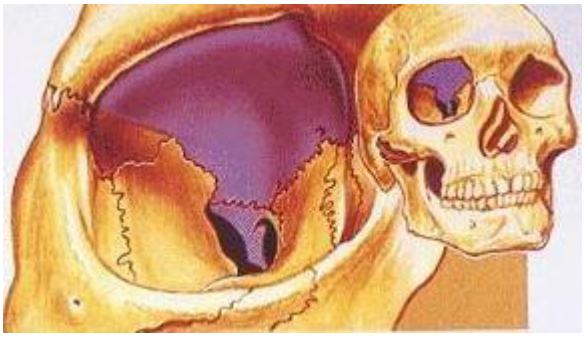
Lateral wall of Orbit

Palpation of retro bulbar tumors is easier from the lateral side as lateral wall of orbit protects only posterior half of eyeball and anterior half not covered . Because of its anatomical position a lateral orbital surgical approach is easier. The lateral rim of the orbit is the strongest portion of the orbit and it has to be sawed during lateral orbitotomy.

Roof

The roof or vault of the orbit is triangular in shape and is formed by the orbital plate of the frontal bone mainly and also by lesser wing of sphenoid. There is a depression in anterolateral part of the roof called the fossa for the lacrimal gland. It is usually quite smooth but may be pitted by the attachments of the suspensory ligament of the lacrimal gland. Trochlear fossa is a small

depression situated close to the orbital margin, at the junction of the roof and the medial wall which forms pulley for superior oblique muscle.



Roof of orbit

BASE OF THE ORBIT

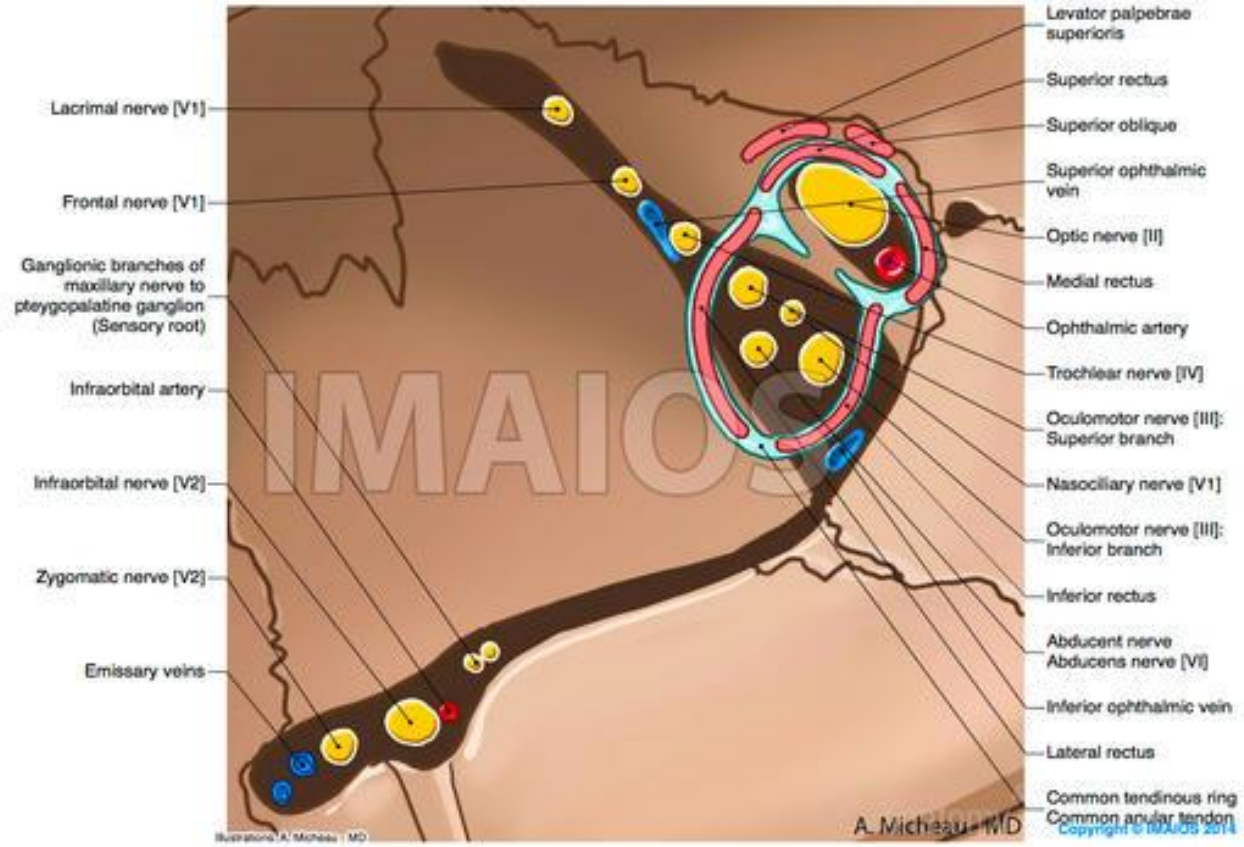
The anterior open end of the orbit is called as base and is bounded by thick orbital margins .The margins are formed by a ring of compact bone.

Apex of Orbit:

Posterior end of Orbit is orbital apex where four walls of the orbit converge. It has two openings namely Optic canal and Superior orbital fissure. The optic canal transmits Optic nerve and Ophthalmic artery. Superior orbital fissure transmits number of nerves and vessels.

ORBITAL CONTENTS

LOCATION		CONTENTS
Optic canal	Lesser wing of sphenoid	Optic nerve Meninges Ophthalmic artery Sympathetic fibers
Superior orbital fissure	Lesser and greater wing of sphenoid	**Upper and lateral part** Frontal lacrimal and Trochlear nerve Superior ophthalmic vein and recurrent branch of the ophthalmic artery. **Middle part** Superior and inferior divisions of oculo motor nerve. Nasociliary nerves Abducent nerve **Lower and Medial part** Inferior ophthalmic vein
Inferior orbital fissure	Greater wing of sphenoid, palatine, zygomatic, and maxillary bones	Sensory: V2 infra orbital and zygomatic Parasympathetic Branches from pterygopalatine ganglion Vessels: Inferior ophthalmic vein and branches to pterygoid plexus
Anterior ethmoid canal	Frontal and ethmoid	Nerve: Anterior ethmoid becomes dorsal nasal Vessel: Anterior ethmoid artery
Posterior ethmoid canal	Frontal and ethmoid	Nerve: Posterior ethmoid Vessel: Posterior ethmoid artery
Nasolacrimal fossa	Lacrimal and maxillary bones	Nasolacrimal sac and duct



ANATOMY OF SUPERIOR ORBITAL FISSURE

ANATOMY OF EXTRA OCULAR MUSCLES

The six striated extra ocular muscles, including the four recti and two oblique muscles, control eye movements.

Origin:

The rectus muscles arise from the common tendinious ring the annulus of Zinn at the apex of orbit which encircling the optic foramina and medial part of superior orbital fissure. The annulus has an upper tendon (of Lockwood)

and lower tendon (of Zinn). Because of this intimate relationship apical disease frequently affects all of these structures simultaneously. Medial rectus and superior rectus arises from the medial part and superior part of the ring respectively . Superior rectus also arises from the adjoining dura that covers the optic nerve. Due to these attachments characteristic pain will occur in case of retro bulbar optic neuritis, during upward and inward movements of globe. Inferior rectus and lateral rectus arises from the inferior part and lateral part of the annulus of Zinn respectively.

All recti muscle run forward around the eyeball and are inserted in to sclera at different distances from limbus. Anteriorly, the recti insert on the globe 5 mm to 7 mm posterior to the limbus.

The superior oblique arises from the bone above and medial to the optic foramina just superior to the annulus, and it passes forward through the trochlea (4 mm posterior to the orbital margin just medial to the supra orbital notch) and it is inserted in the upper and outer parts of sclera behind Equator.

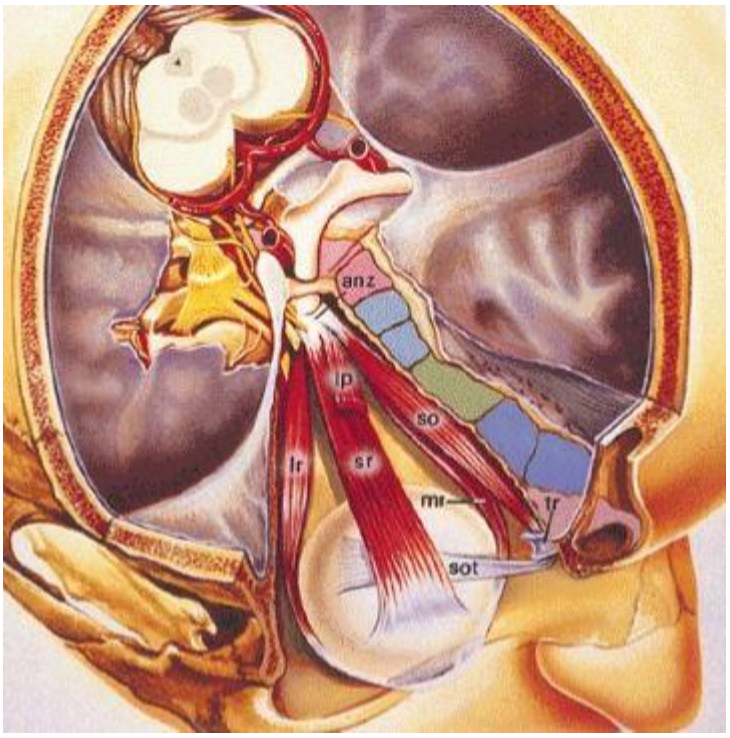
The inferior oblique arises by rounded tendon from the orbital plate of maxillary bone just postero lateral to the naso lacrimal fossa, and then extends laterally and backwards and its direction coursing beneath the inferior rectus and inserted in to lower and outer part of the sclera behind equator.

The levator palpebrae superioris (oculo motor innervation) originates from the annulus and inserts on the upper lid. Müllers' muscle, a sympathetically innervated smooth muscle and it is attached to the levator muscle and aponeurosis anteriorly. Disease, like Graves orbitopathy, Orbital inflammation affecting either of these muscles may alter lid position or function.

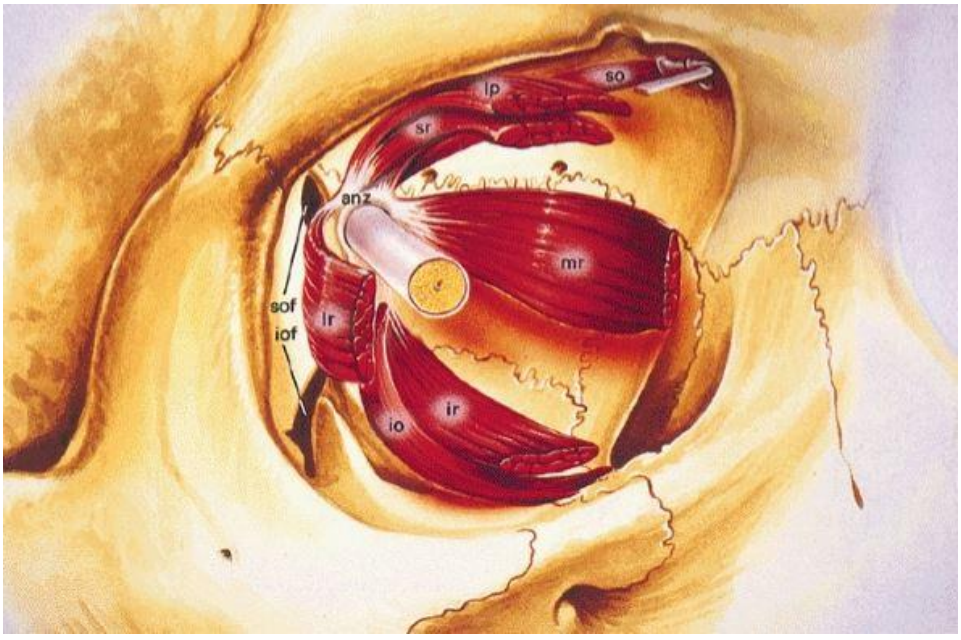
Nerve Supply:

The superior oblique is innervated by the trochlear nerve.

The lateral rectus by the abducens nerve, and the remaining muscles by the branches of the oculo motor nerve.



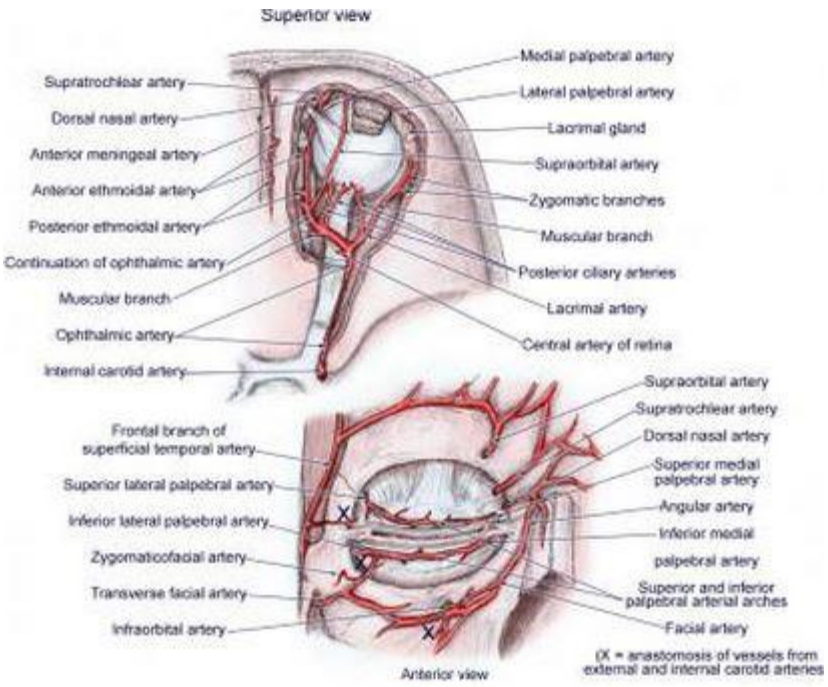
PANOROMIC VIEW OF EXTRA OCULAR MUSCLES



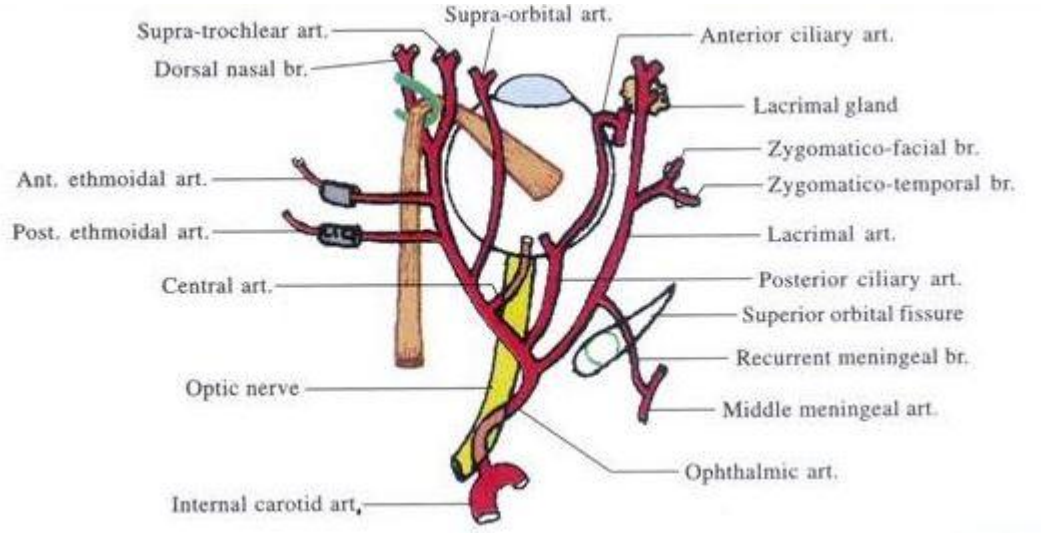
ANTEROLATERAL VIEW OF EXTRAOCULAR MUSCLES

ARTERIAL SUPPLY

The major arterial supply of the orbit is from branches of the ophthalmic artery, which generally arises from the internal carotid artery just inferomedial to the optic nerve .Rarely, the ophthalmic artery may arise from the middle meningeal and enter the orbit through the superior orbital fissure. In the optic canal it courses forward and laterally within the dural sheath, and at the orbital apex penetrates laterally through the dura and then crosses in 80% to 85% of subjects to the medial orbit over the nerve. In the remaining 15% to 20% of subjects the artery courses under the nerve. The branches of the ophthalmic artery with some variations in origin are as follows: central retinal, lateral posterior ciliary, lacrimal, medial posterior ciliary, muscular, supra orbital, posterior and anterior ethmoidal, naso frontal, supra trochlear, and dorsonasal arteries. The ophthalmic artery frequently has anastomotic branches to the external carotid system by means of the middle meningeal and lacrimal arteries, which pass through the superior orbital fissure, and by means of the anterior deep temporal, superficial temporal, and lacrimal arteries . The major distribution and anatomic variations of the ophthalmic artery have been described by Hayreh.



ARTERIAL SUPPLY



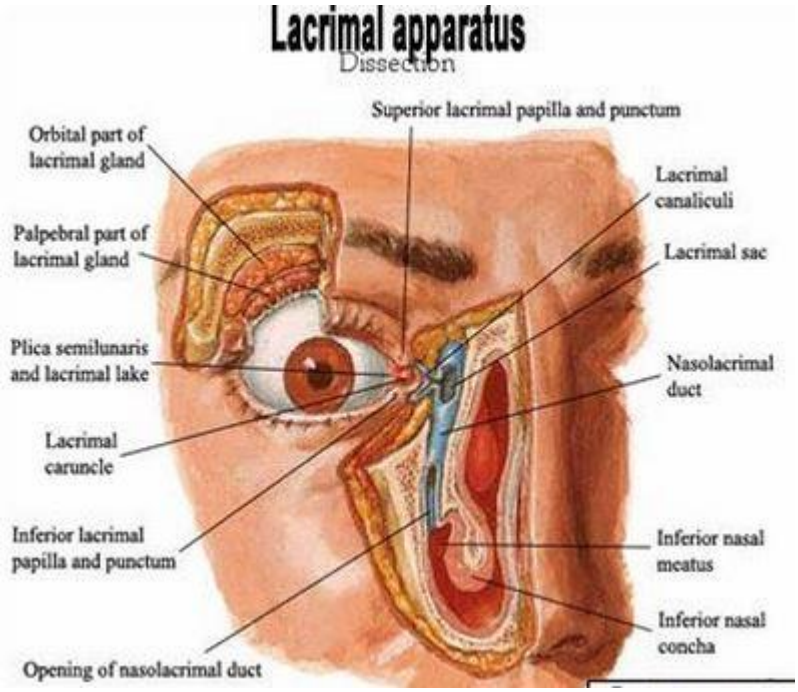
OPHTHALMIC ARTERY AND ITS BRANCHES

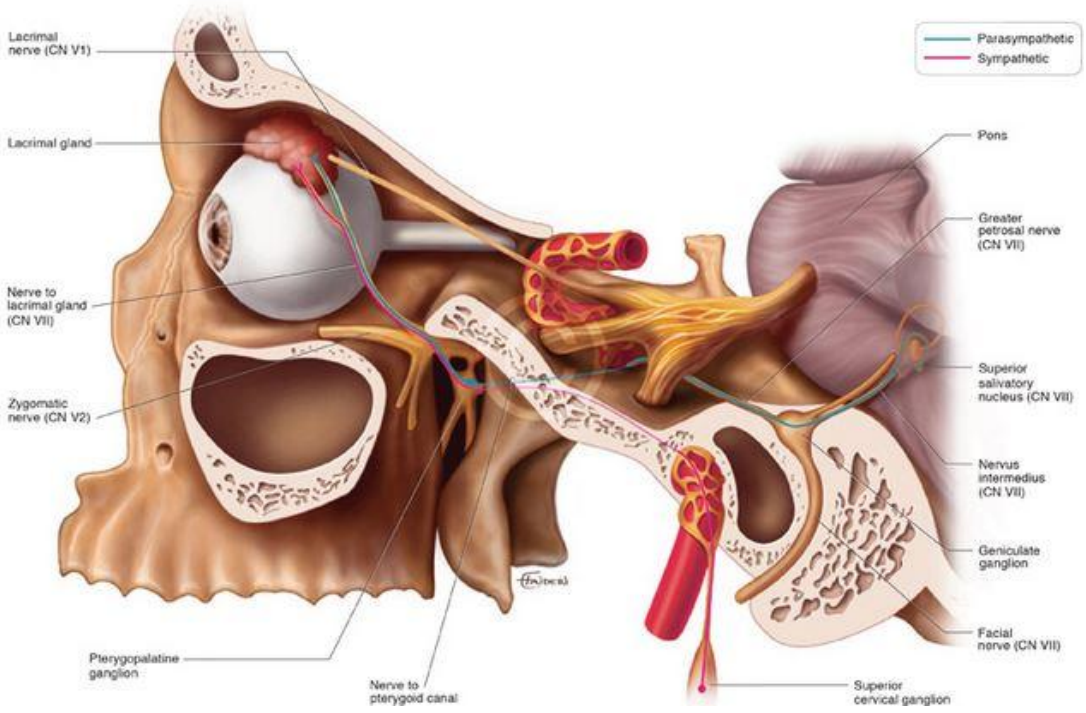
ANATOMY OF LACRIMAL SYSTEM

The lacrimal gland is situated in a shallow fossa in the superolateral orbit. Lacrimal gland weighs 78 g and measures 20 mm X 12 mm X 5 mm. It has been divided into palpebral and orbital (larger) lobes by the lateral horn of the levator aponeurosis. The orbital lobe is superior to the palpebral, and the isthmus lies between the two lobes. The ducts of lacrimal gland (10 to 12) pass through the palpebral lobe, and open in the superolateral conjunctival fornix 4 mm to 5 mm from the tarsal border. Thus, resection of the palpebral lobe functionally destroys the gland. The borders of the gland are related anteriorly to the orbital septum, posteriorly to the orbital fat, and medially to the superior rectus, globe, and lateral rectus. The inferior surface rests on the lateral rectus. The lacrimal gland is a serous gland, and the acini have a tubule racemose arrangement.

The acini consist of columnar cells surrounded by an incomplete layer of myo epithelial cells. Acinar secretions drain into intra lobular, then interlobular, and then into the main ducts. Grossly the lacrimal gland has a nodular surface with a fine connective tissue pseudo capsule. It is pinkish-gray, in contrast to orbital fat, which is yellow-gray. The gland is supported by Whitnall's ligament and the lateral horn of the levator aponeurosis as well as by septal attachments to the superior periorbita. The lacrimal artery penetrates

it posteriorly, and the vein from it drains into the superior ophthalmic. Lymphatic drainage is by means of the lid and conjunctiva to the preauricular nodes. The lacrimal nerve and sometimes branches of the zygomatic carry the sensory afferents, the parasympathetic efferents (by the nervus intermedius, facial, greater superficial petrosal, vidian, sphenopalatine ganglion, infraorbital, and lacrimal nerves), and the sympathetic efferents (from the carotid plexus through the sphenopalatine ganglion). This pathway is believed to account for reflex tearing. In addition to the lacrimal gland, there are accessory glands (of Krause and Wolfring) in the lids and conjunctiva.





ANATOMY OF LACRIMAL GLAND AND ITS NERVE SUPPLY

CLASSIFICATION

There are various classification[3,6,7] systems, due to the highly variable clinical and pathological features of Non specific orbital inflammation. However none are universally accepted.

CLASSIFICATION BASED ON ONSET:

- Acute
- Sub acute
- Chronic

CLASSIFICATION BASED ON TARGET TISSUE INVOLVED

- Diffuse
- Localised
- Anterior orbit
- Posterior Orbit
- Extra ocular muscle
- Optic nerve
- Lacrimal gland

HISTO PATHOLOGICAL CLASSIFICATION[6]

- Classical or cellular
- Eosinophilic
- Granulomatous
- Vasculitic
- Desmoplastic /Fibrous

CAUSE AND PATHOGENESIS

The Cause and pathogenesis of NSOI, still remains to be unknown [3,5,6,11]. Various causes proposed are Infections, post infectious, auto immune, genetic and environmental factors. Some studies suggest that onset of orbital pseudo tumor can occur simultaneously or after few weeks of upper respiratory infections [26]. The association of orbital pseudo tumor with some systemic diseases and successful treatment with corticosteroids or other immune suppressive therapy suggest that an auto immunological process is the mechanism[3]. It s acute form is characterized by infiltration of polymorphs and Sub acute and Chronic forms are characterized by increasing Fibro vascular stroma[3].

Orbital pseudo tumor is also observed, associated with Crohn's disease, systemic lupus erythematosus, diabetes mellitus, rheumatoid arthritis, myasthenia gravis, and ankylosing spondylitis, all of which strengthen the basis of NSOI being an immune-mediated disease [38].

Trauma has also been seen to precede some cases of orbital pseudo tumor. However, one study suggests that the release of circulating antigens caused by local vascular permeability triggers an inflammatory cascade in the affected tissues.

HISTOPATHOLOGY [3,6,11,32]

The histo pathologic spectrum of NSOI is typically non diagnostic. Its presentation can vary from typical diffuse polymorphous infiltrate to lymphoid, sclerosing, granulomatous, eosinophilic , or vasculitic inflammation[6]. Four forms have been suggested[6].

The classic form of orbital pseudo tumor is cellular variety which presents acutely and consists of inflammatory cells, mature lymphocytes admixed with plasma cells, neutrophils, eosinophils and occasionaly macrophages and histiocytes.

Atypical findings are tissue eosinophilia, granulomatous inflammation, vasculitis and desmoplasia.

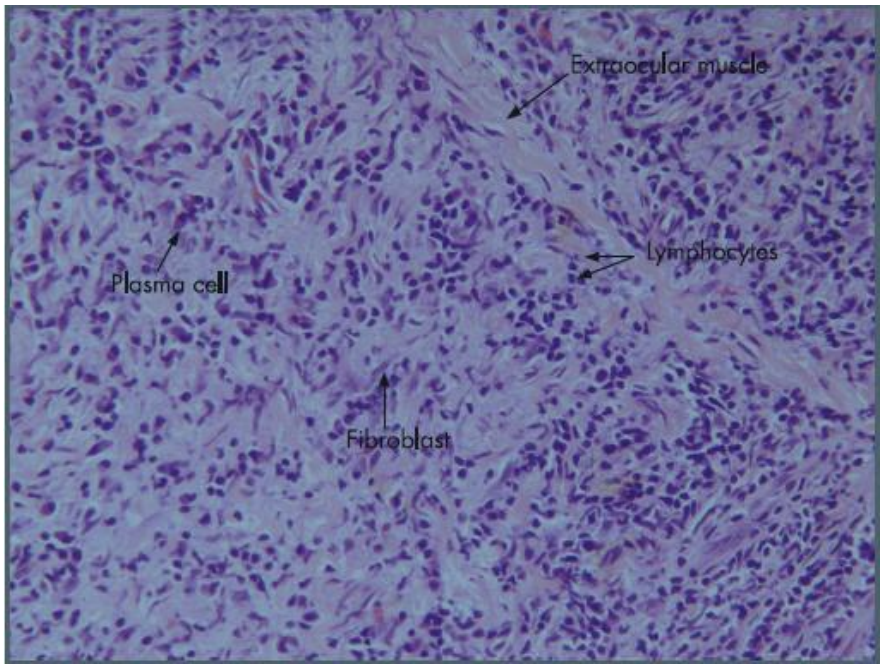
Eosinophils are present in pediatric pseudotumor in particular. Eosinophili degranulation contributes to tissue fibrosis.

Granulomatous inflammation consists of multinucleated giant cells and non caseating granuloma can mimic sarcoidosis.

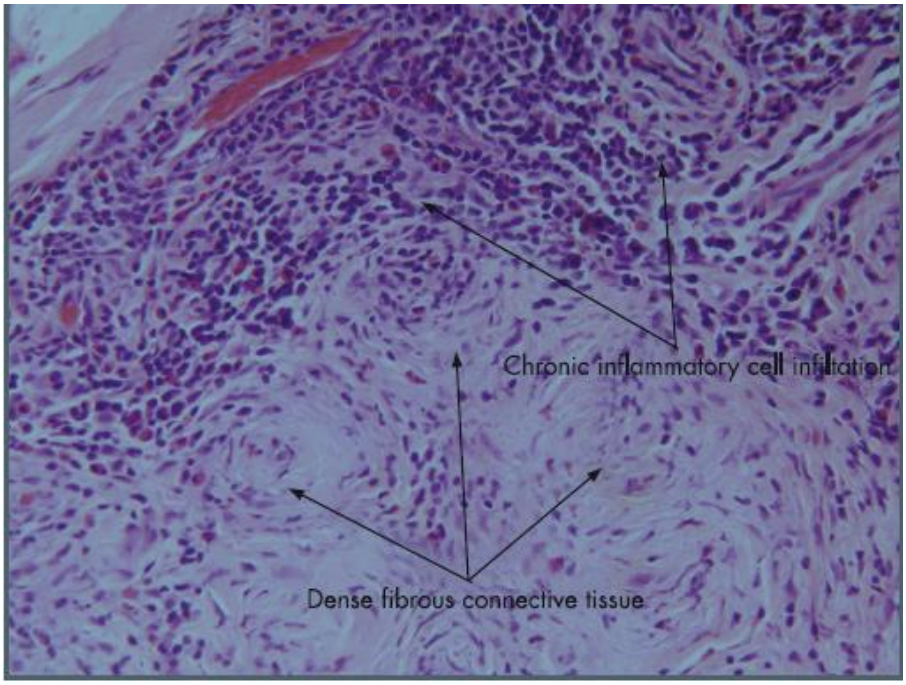
Vasculitic form is limited to the orbit and is rarely found. It primarily affects small arteries and aretrioles.

Chronic forms of the disease are characterized by increasing fibrous component . Lymphoid follicles with germinal centers are also observed in the chronic phase. Extra ocular muscle, lacrimal gland and fat are replaced with fibrous tissue. The desmoplastic response can eventually result in dense fibrosis, entrapment of orbital structures and mass effect. Certain cases are primarily sclerotic in nature presenting insidiously without prior acute phase. They have scant cellular infiltrate with dense desmoplastic stroma.

However, till date, no one particular classification has been universally accepted[6]



CLASSICAL IDIOPATHIC ORBITAL INFLAMMATORY DISEASE



SCLEROSING ORBITAL INFLAMMATORY DISEASE

CLINICAL PRESENTATION[8-11,30,31,33]

Acute & Subacute Nonspecific Myositic Inflammation: Orbital Myositis [27-29]

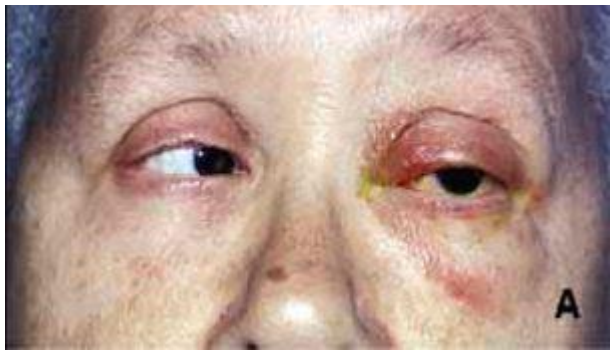
Orbital myositis is a most common nonspecific inflammatory condition of the Extra ocular muscle. It could be divided into three profiles: isolated, recurrent, and atypical.

Signs and symptoms :

The isolated and recurrent disease typically present with periorbital inflammation and swelling, retrobulbar pain, and pain on movement, .diplopia, conjunctival injection (often focal over muscle insertions), and proptosis. Isolated cases involve one muscle. Recurrent disease involve multiple muscle and is often bilateral. Virtually all of the vertical and horizontal muscles are involved. Those patients who go on to recurrent episodes usually have involvement of new muscles that differ from the primary presentation and may be either recurrent or progressive in terms of activity.

Atypical Myositis may lack pain, may lack restriction of movement, have unusual or abnormal CT patterns, are progressive, or may be associated with optic neuropathy. The atypical cases may require biopsy.

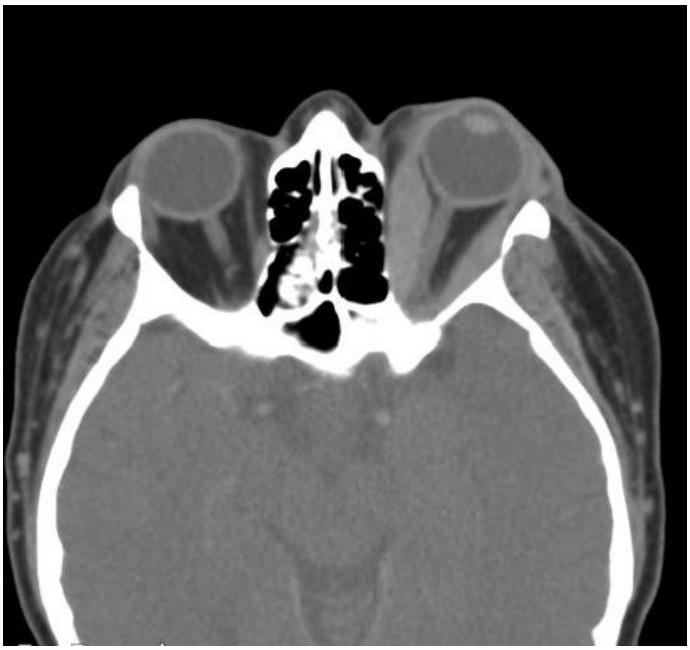
Most cases of myositis do not typically have direct systemic associations other than with underlying immune disorders in some (allergy, collagen vascular disease). Rarely, this disorder is associated with giant cell myocarditis or seen as a para neoplastic syndrome.



PICTURE SHOWING PERIORBITAL EDEMA WITH CONJUNCTIVAL INJECTION IN CASE OF MYOSITIS.

Imaging

CT scan and USG B scan shows enlargement of extra ocular muscles mostly, unilateral. On CT scan shows involvement of both muscle belly and tendon and diffuse inflammatory shadow extends up to orbital fat.



CT SCAN SHOWING MYOSITIC INFLAMMATION OF BOTH MUSCLE BELLY AND TENDON OF LEFT MEDIAL RECTUS .

DIFFERENTIAL DIAGNOSIS

The major differential diagnosis is Graves' orbitopathy[17].

Thyroid myopathy is usually painless in onset (unless severe and infiltrative), asymmetric, slowly progressive, and associated with a systemic diathesis. Lid retraction, limitation of gaze and deterioration of visual function (color vision, visual fields, and visual acuity) may occur in contrast to orbital myositis. On CT scan in thyroid myopathy the extraocular muscle enlargement is usually fusiform and tendons are usually spared. USG will demonstrate enlargement of the extra ocular muscle and local infiltration of surrounding

structures in myositis. These differences constitute a guideline for the majority of cases since there may be some overlap.

Additional diseases that included in the differential diagnosis are Arteriovenous fistulas and malformations,- It is associated with injection of the globe and enlargement of extra ocular muscles on CT scan. Generally it does not have pain or inflammatory features.

Orbital metastasis, and lymphomas[39], Amyloid depositions, and primary tumors of the extra ocular muscles can cause enlargement of extra ocular muscles but sharp pain is rare presentation in metastasis and enophthalmous is frequent.

Tolosa-Hunt syndrome, has deep orbital pain and multiple neuropathies and Trichinosis associated with dermatopathy, and Trochleitis have localized tenderness over globe.

Myasthenia gravis, usually does not have inflammatory signs and has reversible ptosis using edrophonium.

Differential diagnosis of Graves' orbitopathy versus orbital myositis[11]

	IDIOPATHIC MYOSITIS	GRAVES' ORBITOPATHY	
CLINICAL			
Onset	Sudden onset	Slower-weeks to months	
Pain	Frequent especially on eye movement	Rare initially; generally gritty irritation	
Ptosis	Frequent	Rare, except in markedly congested orbits	
Lid Retraction	Rare	Frequent	
Stare	Absent	Frequent	
Lid Lag	Absent	Frequent	
Chemosis	Localized and injected	Generalized, but may be localized	
Extraocular movements	Limitation and pain on eye movement of involved muscle or antagonist	Limitation; painless in field of movement opposite to involved muscle	
Visual function	Rarely affected	May be impaired	

Response to steroids	Dramatic with complete resolution; may recur	Incomplete and slow	
ORBITAL IMAGING			
Bilaterality	Infrequent	Frequent	
Number of muscles	More than one in approximately 54%	Typically more than one	
Muscle borders	Irregular	Regular	
Extension into orbital fat	Frequent	Little or none	
Tendon involvement	Frequent	Infrequent	
Scleral and Tenon's capsule enhancement	Occasional and localized	None	
Site	Any muscle	Inferior, superior, and medial most frequent	

TREATMENT

Patients with unilateral single-muscle disease of typical onset can be treated either with Non steroidal anti-inflammatory drugs or relatively low-dose(up to 30 -40 mg prednisone tapered rapidly on a symptomatic basis) corticosteroids. They will improve rapidly and are unlikely to have a recurrence.

Patients with bilateral or multiple muscle disease with a typical acute or subacute onset are expected to have recurrences and require more follow-up and should be carefully analyzed for potential systemic associations and has to be treated more aggressively, either with Pulsed intravenous corticosteroids (1g Methyl prednisolone) or high-dose oral corticosteroids (60 -80 mg prednisone) tapered over a 4- to 6-week period. If they fail to respond or persist, the patient should undergo biopsy. Patients who have persistent, recalcitrant, and recurrent disease may require intervention with immunosuppressive drugs[40]. Those cases with an atypical onset warrant early biopsy and management .

Radiotherapy is considered to be much less effective in the treatment of myositis.

Nonspecific Lacrimal Inflammation –Dacryo adenitis

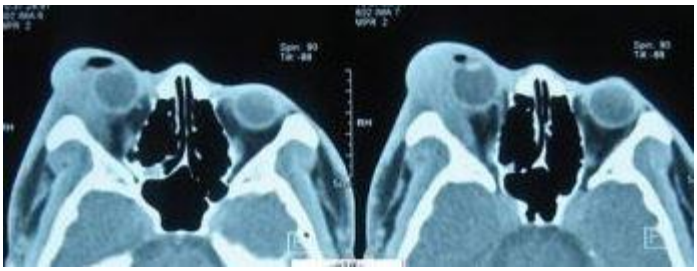
Dacryo adenitis is second most common nonspecific inflammatory disease of the orbit.

SIGNS AND SYMPTOMS:

The typical presentation of Dacryo adenitis includes pain, tenderness, and injection of the temporal portion of the upper lid and conjunctival fornix with an associated tender, palpable lacrimal gland, an S-shaped deformity of the lid. Sometimes there may be some minimal evidence of proptosis with downward, inward displacement of the globe.

IMAGING:

Lacrimal gland is typically enlarged, and has irregular margins and shows enhancement on contrast. It is confined to the supero lateral orbit, obscuring the lateral aspect of the globe and often displacing it infero medially. On USG B scan the mass may have internal reflectivity with an echolucent area adjacent to the scleral shell, associated with thickening of the adjacent muscles anteriorly.



CT SCAN SHOWING FEATURES OF LACRIMAL GLAND ADENITIS.

DIFFERENTIAL DIAGNOSIS

- viral and bacterial dacryoadenitis,
- rupture of a dermoid cyst
- Orbital lymphoma [39]and hematopoietic malignancy, and
- Specific lacrimal inflammations include sarcoid, Sjögren's syndrome, Wegener's granulomatosis, sclerosing inflammation, and a myriad of autoimmune disorders, which need biopsy. On biopsy, it shows polymorphous cellular infiltration with edema and vascular dilatation and do not have a marked degree of destruction of the lacrimal gland. If there is evidence of destruction of the lacrimal gland, we should rule out possibility of organ specific immune disorder.

TREATMENT

Treatment consists of oral corticosteroids, usually moderate tapering doses (40 mg prednisone), with majority of case resolving over a 1- to 3-month period. In non responding patients and patients with atypical features such as bony erosion and longer duration of disease requires Incision Biopsy to rule out other specific lacrimal inflammation. The biopsy should be taken through percutaneous route to avoid damaging the lacrimal ducts.

ACUTE AND SUBACUTE NONSPECIFIC ANTERIOR AND DIFFUSE ORBITAL INFLAMMATION

Non specific orbital inflammation may not be structure specific and instead it involves orbital tissue anteriorly or it involves whole extent of orbit. This syndrome most commonly present in children and young adult..

Signs and symptoms:

The patient presents with pain, proptosis, ptosis, lid swelling, and conjunctival congestion and chemosis and limitation of ocular motility.Some patients present with features of decreased visual acuity,.,Relative afferent pupillary defect ,and defective colour vision. The other important findings may include uveitis, sclerotenonitis, papillitis, and exudative retinal detachments. Patients with diffuse disease have the same features of anterior orbital

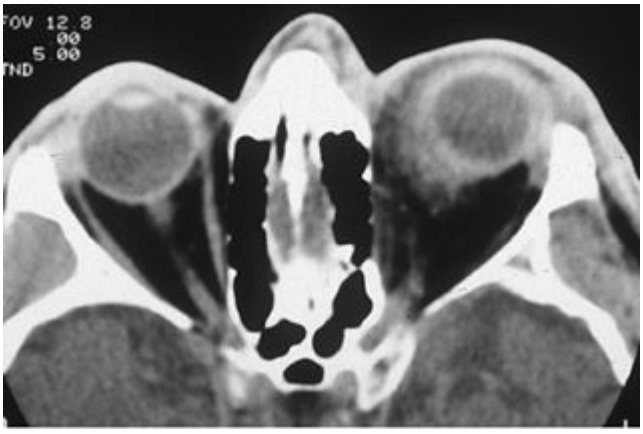
inflammation, with involvement of the extraocular muscles and neurosensory structures .

IMAGING:

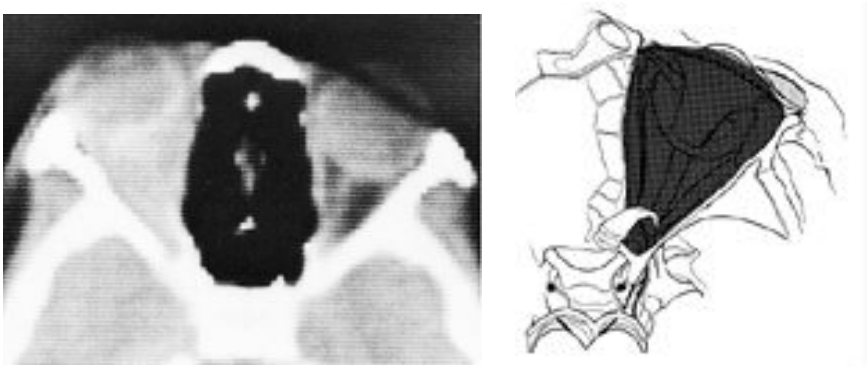
The imaging characteristics of anterior non specific inflammation consist of an irregular orbital infiltration limited to anterior orbit with scleral and choroidal thickening with obscuration of the junction of the globe and optic nerve and variable extension along its sheath.

In diffuse NSOID, the whole of the orbit is obscured by infiltration. Ragged shadows may surround the optic nerve and causing optic neuritis and accounting for defective visual function..

On ultrasonography, there is accentuation of Tenon's space and doubling of the optic nerve shadow (T sign).



CT SCAN SHOWING FEATURES OF ANTERIOR NSOI



CT SCAN SHOWING FEATURES OF DIFFUSE NSOI

DIFFERENTIAL DIAGNOSIS:

Differential diagnosis of anterior and diffuse nonspecific orbital inflammation includes.

Orbital cellulitis- bacterial cellulitis more likely to have an acute onset, systemic fever and malaise and leukocytosis.

A sudden event in a preexisting lesion (hemorrhage within a vascular lesion, rupture of dermoid cyst), Scleritis, and uveitis, and systemic inflammatory syndromes like collagen vascular diseases.

In younger patients, leukemic infiltration, metastatic neuroblastoma, and rhabdomyosarcoma may mimic orbital inflammation.

TREATMENT

The patient can be treated with NSAID, generally oral prednisone (usually in doses starting at 60 mg tapered over 2 to 3 months), which usually

produces a dramatic improvement of symptoms. The majority of patients improve substantially within weeks. And younger patients, may have recurrent episodes .In non responding and recurrent patients repeat imaging and biopsy is mandatory.

ACUTE AND SUBACUTE NONSPECIFIC APICAL ORBITAL INFLAMMATION

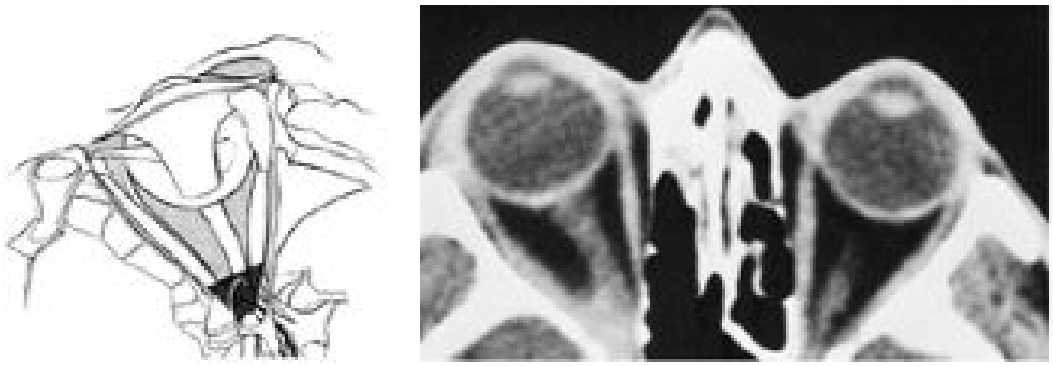
Inflammatory disease in orbital apex may affect multiple nerves and vessels.

Signs and symptoms:

Apical inflammation is characterized by less proptosis and less inflammatory signs and more signs of optic neuritis and multiple extraocular muscle paresis. Limitation of ocular movement, pain on movement diplopia and defective vision may be the presenting features. Thus, the cardinal features are a disproportionate functional abnormality compared to the degree of inflammatory signs.

IMAGING

CT scan shows inflammatory soft tissue shadow at the apex of orbit.



CT SCAN SHOWING FEATURES OF APICAL NSOI

DIFFERENTIAL DIAGNOSIS[36]

The differential diagnosis includes the orbital apex syndromes such as Tolosa-Hunt syndrome and Cavernous sinus thrombosis.

Lymphoma, secondary tumors from adjacent sinuses, sclerosing inflammation, fungal infections, metastases, Wegener's granulomatosis, meningioma, and mucormycosis also present with such features.

MANAGEMENT

Inflammation may be treated with systemic steroids.

It is difficult to obtain a biopsy from orbital apex without causing significant morbidity, the risks and benefits have to be weighed before the management decision.

COMPARATIVE FEATURES OF ACUTE AND SUBACUTE NONSPECIFIC IDIOPATHIC INFLAMMATIONS OF THE ORBIT[8]

	MYOSITIC	LACRIMAL	ANTERIOR	DIFFUSE	APICAL
Pain	On movement	With tenderness	Moderate	Moderate	may be severe
Ocular and orbital features	Painful Ocular movement restrictions Localized injection and chemosis	Lacrimal gland.swelling S-shaped Ptosis. Tenderness and localized injection.	Diffuse injection and lid swelling. Decreased vision Uveitis Retinal detachment Decreased ocular movement and anterior inflammation	Uveitis Retinal detachment ocular movement restriction, Decreased vision and diffuse injection and swelling of lid and - Chemosis swelling of lid	Decreased vision ocular motility restriction, Mild proptosis and chemosis

IMAGING					
CT & MR	Muscle irregularly enlarged with tendon. Fusiform enlargement of whole muscle	Irregular swelling of lacrimal gland with adjacent tissues	Anterior: enhancing with irregular margins intimate to scleral tissue. Variable extension along optic nerve with Decreased fat density	Diffuse: enhancing lesion with with decreased fat density	Apical irregular infiltration Extends through muscle and optic nerve
Ultrasonography	extra-ocular muscle thickness increased	Localised swelling with increased Tenon's space	Sclero tenonitis with T sign	T sign	Negative

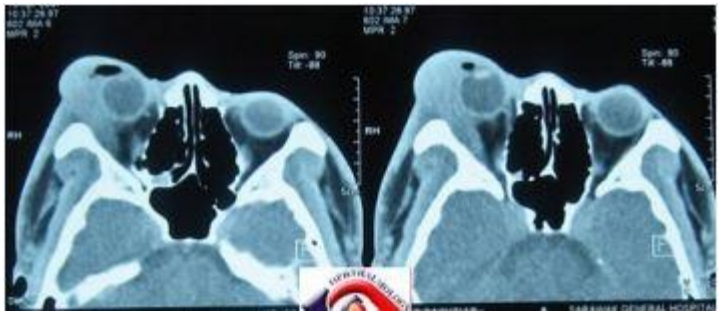
DIAGNOSIS

All suspected NSOI patients will require a complete ophthalmic evaluation. Pediatric NSOI is characterized by bilateral manifestation, uveitis, disc edema and eosinophilia. According to a study, Pain is the most common symptom in adult NSOI followed by diplopia. Most common sign is periorbital edema followed by proptosis. As Connective tissue diseases and NSOI are closely associated and laboratory work-up is needed for suspected NSOI, including complete blood count, thyroid function studies, ESR, , anti neutrophil cytoplasmic antibodies ,antinuclear antibodies, rapid plasma reagin test, angiotensin-converting enzyme level, and rheumatoid factor.

CT/MRI Scan[7]

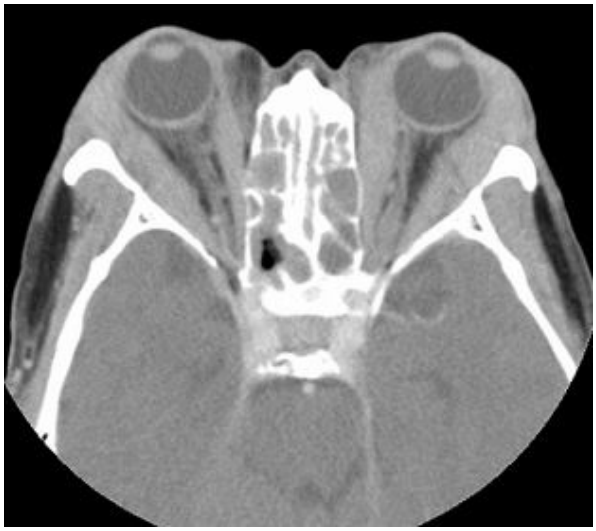
High resolution CT and contrast enhanced MRI is preferred in evaluation of NSOI CT is preferred in most of the cases because of its good inherent contrast of muscle, bony structures, orbital fat, and air in the adjacent paranasal sinuses. MRI is preferred in case of soft tissue lesions in the orbit. Neuro imaging is more useful in diagnosing asymptomatic presentation .

On imaging, lacrimal gland will be diffusely enlarged with blurred irregular margin .



CT Scan - Diffusely Enlarged Lacrimal Gland with Blurred Irregular Margin

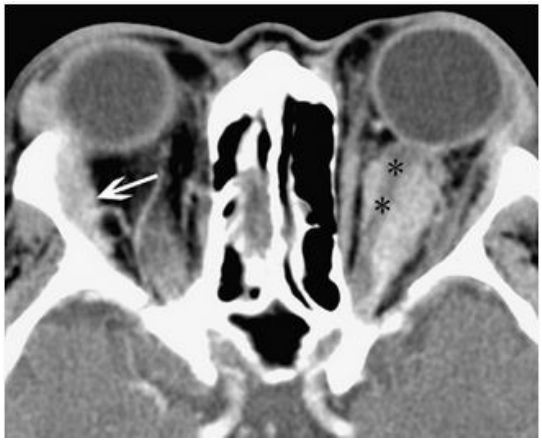
Unilateral single muscle enlargement with tendon involvement is most common in mys. The enlargement of tendon may also occur and this along with the muscle belly, leads to a tubular configuration and blurred muscle margin.



Tubular enlargement of extra ocular muscles.

OPTIC NERVE

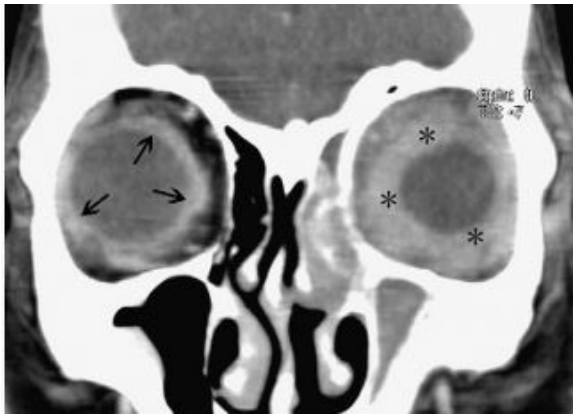
Inflammatory mass that surrounds an unenhanced optic nerve may give rise to classical appearance of, "tramline".



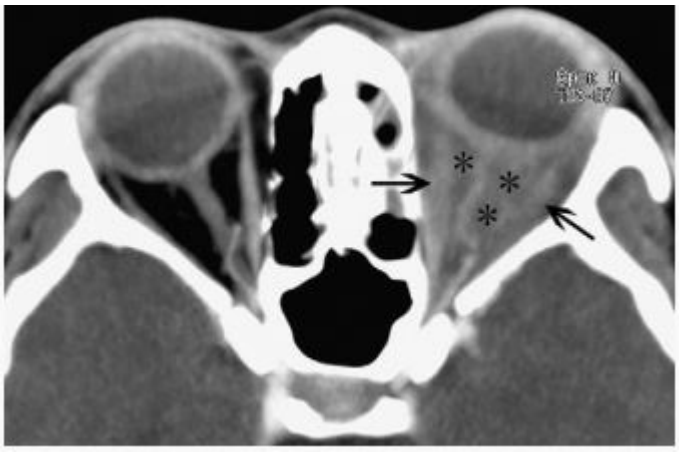
CT Scan Showing Optic Nerve Involvement With Tramline Sign

SCLERA, EPISCLERA, TENON'S CAPSULE, AND UVEA:

Imaging will demonstrate non-specific blurring and thickening of scleral uveal rim following contrast injection.

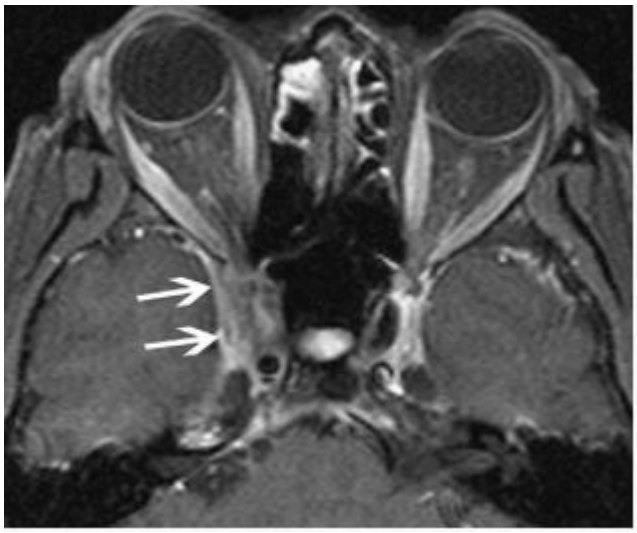


In imaging, the orbital fat is surrounded by diffuse inflammatory infiltrate which envelopes the globe and optic nerve sheath complex.



ORBITAL APEX, CAVERNOUS SINUS AND INTRACRANIAL INVOLVEMENT [34-36,48]

In Imaging obliteration, compression or displacement of the optic nerve can be seen. In intracranial extension of NSOI, the cavernous sinus and middle cranial fossa are the two most common locations. In certain cases imaging shows intracranial extension of the disease with pronounced narrowing of the right internal carotid artery in its intra cavernous portion, which causes transient ischemic attack[18].

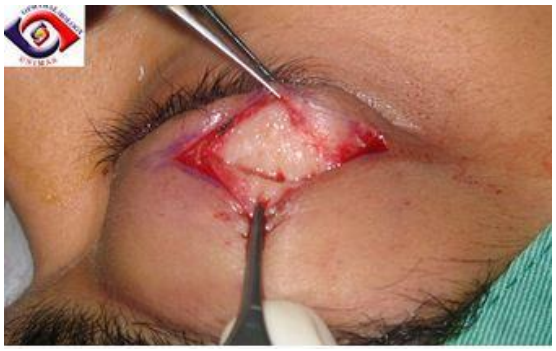


BIOPSY:[6]

Biopsy is usually not required for NSOI as there may not be a distinct mass or approachable lesion to biopsy .

Indication of Biopsy are as follows:

- Lack of steroid responsiveness
- Progressive neurologic deficits,
- Persistent imaging abnormalities.



DIFFERENTIAL DIAGNOSIS

Most common differential diagnosis of NSOI are thyroid eye disease and orbital cellulitis . They can mimic NSOI, both radiologically and clinically.

Below table outlines common differential diagnosis f or NSOI.

Type	Laterality	Onset	Differential findings	Associated signs/symptoms	Labs	Imaging
Lymphoproliferative disesase	Unilateral	Variable,typically insidious	Mass,Swelling,Ptosis,Ocular motility restriction,painless	Fatigue, Malaise	Anemia,Elevated serum lactate dehydrogenase	Extraconal and intraconal,Extraocular muscle involvement rare,No bony erosion
Metastatic disease	Unilateral	Variable,typically insidious	Mass, Subcutaneous nodules,Proptosis,Enophtalmos,painless	Variable given primary source: Breast>Lung>Prostrate	Variable given primary source	Bony metastasis,Infiltrative process, Obscuration of landmarks vs Focal lesions
NSOI	Usually Unilateral	Typically acute	Variable given anatomical location,Localtenderness,Eyelidswelling, Ocular motility restriction, pain present	Minimal consitituti onal symptoms	Increased erythrocyte sedementation rate(ESR), Increased C-reactive protein	Variable given anatomical location, Multiple muscles enlarged with tendon involvement, irregular anatomical borders

Orbital Cellulitis	Unilateral	Acute	Erythema, Chemosis, Ptosis,Sinusitis,Trauma,Ocular mobility restriction, Pain pronounced	Fever	Leukocytosis,Negative or positive blood cultures	Decreased orbital fat signal,concurrent sinus disease,bony erosions, subperiosteal abscess, venous thrombosis
Sarcoiodosis	Unilateral or Bilateral	Acute or subacute	Mass/Swelling,Ptosis, Ocular mobility restriction, Pain +	Shortness of breath or persisistentcough, Erythremanodosum,Arthralgias,Hilar lymphadenopathy	Elevated angiotensin converting enzyme levels,Decreased pulmonary function testing	Hilar lymphadenopathy, diffuse soft tissue mass
Thyroid Eye Disease	Typically bilateral but can be asymetric	Variable	Lid retration,Lid lag, Optic neuropathy,Ocular motility restriction, Pain +	Fatigue, Goiter, Heatintolerance.Increase sweating , Nervousness,Weightloss,Diarrhea,Hair loss,Hand tremor,Tachycardia	Elevated T4 and T3,Decreased TSH, Presence of stimulating autoantibodies	Multiple enlarged muscles ,Tendon sparing, Increased orbital fat
Wegener's Granulomatosis	Unilateral or Bilateral	Acute or subacute	Dacryocystitis,Saddle nose,Epistaxis,Chronic rhinosinusitis,Pain+	Shortness of breath ,Myalgias/Arthragl as,Subcutaneousnodules,Peripheral neuropathy	Positive anti-neutrophil cytoplasmic antibody (ANCA) ,Anti-pR3 >>> MPO	Extraconal and intraconal,Concurrent sinus disease,Bone erosion, mass lesions other organ systems

MANAGEMENT[3,8-11]

OBSERVATION

In mild cases of inflammation observation alone may be required. Non improvement or worsening of clinical features may require additional therapy.

Non-steroidal anti-inflammatory drugs (NSAIDs)

In mild cases of NSOI **NSAIDs**, can be used and in refractory cases steroids are indicated. The side effects of NSAIDs depends on the amount of dose and most of the patients experience dyspepsia, which can be reduced by proton pump inhibitor, such as esomeprazole or omeprazole.

CORTICOSTEROIDS

For NSOI, Systemic steroids are considered as mainstay of therapy. There will be rapid, dramatic improvement of symptoms with steroid treatment. Treatment doses that differ in ranges between1.0-1.5 mg/kg or 50-100 mg/day are initiated. Recommended therapy includes High dose oral steroid for a period of two to three weeks followed by slow tapering. Intra venous steroids -pulse dose are recommended for rapid progressive cases.

Failure in steroid treatment are termed as primary, if there is no improvement in adequate steroid dosage. It is defined as recalcitrant, if a

breakthrough inflammation occurs during steroid tapering and recurrent if symptoms recurs after a remission period.

Systemic Side effects:

- Cushingoid symptoms and signs
- Growth retardation
- Weight gain, diabetes
- Risk of development of gastrointestinal bleeding.

IMMUNO SUPPRESSANT[3,41]

Calcineurin inhibitors: Cyclosporine-A (CsA)

It is an immunosuppressant that acts on T-lymphocytes and inhibits synthesis of T-cells. Cyclosporine can be administered in NSOI patients with diabetes as they are intolerant to steroids. NSOI can be treated with 5mg/kg/ day then tapering to 2mg/kg/day over ten months.

CYCLOPHOSPHAMIDE:

Cyclophosphamide is an alkylating agent. Dose- 200 mg/day is used to treat patient with recurrence on steroid therapy.

Side effects of Cyclophosphamide includes nausea and vomiting, alopecia ,lethargy, bone marrow depression, gastrointestinal disturbances and Hemorrhagic cystitis(which can be prevented by fluid intake and mesna). Most common complication can be transitional cell carcinoma of the bladder.

METHOTREXATE

Methotrexate is an inhibitor of an enzyme dihydrofolate reductase, essential for folic acid synthesis and in turn causes of suppression of T-cell and B-cell functions. For ocular immune suppression dose of Methotrexate is 7.5 – 12.5 mg /kg. Adverse drug reactions of methotrexate include gastrointestinal distress, arthralgia, liver function abnormalities, alopecia and headache. Dietary supplementation of folate, alcohol restriction and parenteral administration can decrease the side effects.

IMMUNO MODULATORS

Biological immune modulators have revolutionized the treatment of autoimmune diseases.

Infliximab-chimeric monoclonal antibody is a TNF-α blocker, given in dose of 3 to 5 mg per kg for 6 weeks. It has been recently introduced for the treatment of NSOI.

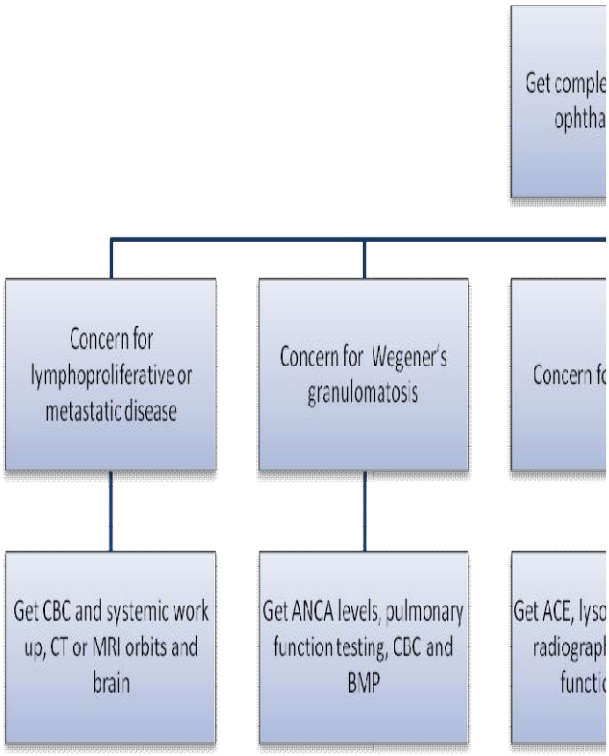
RADIATION THERAPY[42-44]

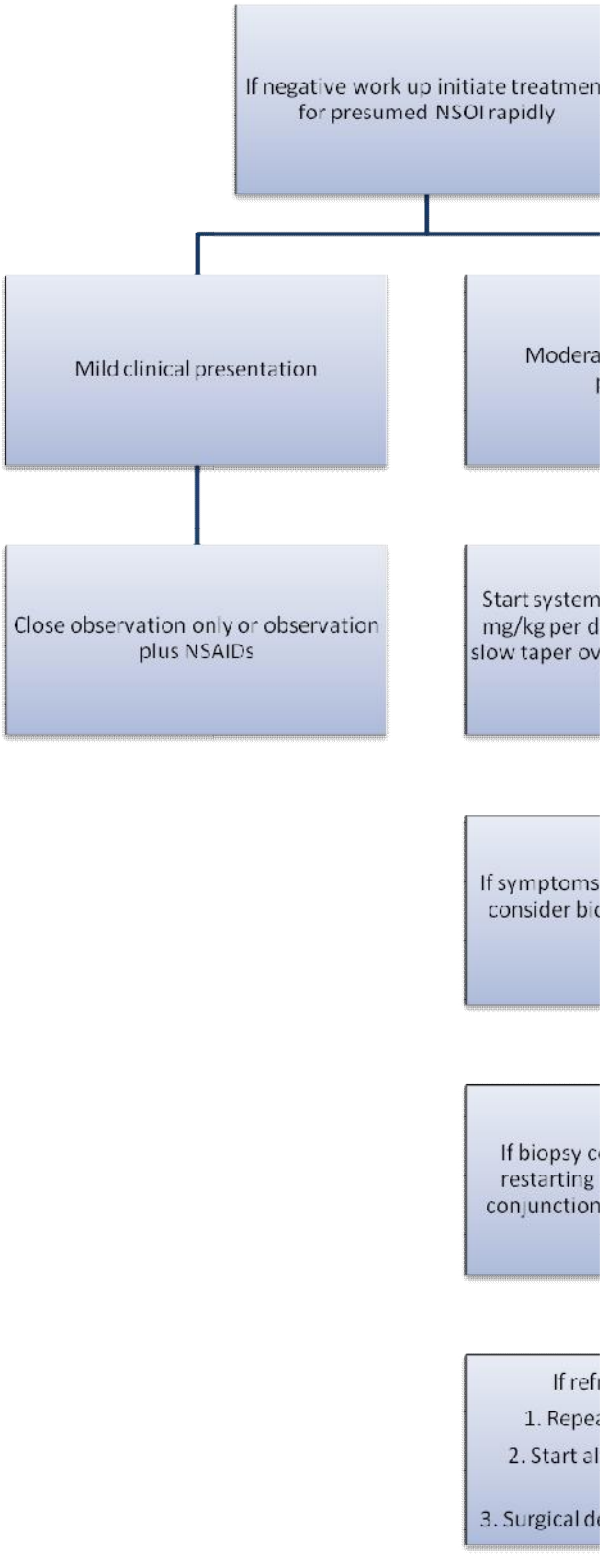
Radiation therapy can also be used in the NSOI treatment, when disease is resistant or intolerant to corticosteroid therapy. success rates of radiotherapy ranging between 50-75%.

Dose ranging from 1500- 2500 c Gy over 10-15 days. Time taken for response of treatment is 3 -8 months.

Localised mass, presence of lymphoid follicles, absence of eosinophils and initial response to steroid are good prognostic factors.

DECISION TREE




If negative work up initiate treatmen
for presumed NSOI rapidly
Mild clinical presentation
Modera
Close observation only or observation
plus NSAIDs
Start system
mg/kg per d
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AIM

- To study the demographic distribution of NSOI-Age wise, sex wise and state wise.
- To study the presenting complaints, presenting eye and associated symptoms.
- To study the presence of past and family history and associated systemic illness.
- To study the clinical features of NSOI including visual acuity , laterality of lesion and other associated features.
- To study the type of investigative modality used in the diagnosis of the disease.
- To study the treatment modality used in the treatment of disease and its outcome.
- To study the classification and type of NSOI and its nature of course.
- To compare the results with international studies.

MATERIAL AND METHODS

- **Study Design:** This is a comprehensive hospital based prospective study.
- **Source of Data:** Department of Orbit and Oculoplasty, Aravind Eye Hospital, Madurai.
- **Study Period:** January 2014 to June 2015 including follow up periods of 3 months.
- All patients with orbital pseudo tumor, those who presented to the orbit clinic were included in the study. Patients who are being recruited in the study, satisfying the inclusion criteria and not falling into the exclusion criteria.

INCLUSION CRITERIA:

- Study includes patients presented with orbital pseudo tumor between January 2014 to June 2015
- Only newly diagnosed cases and CT (radiologically) confirmed cases of orbital pseudo tumor, were taken into account.

- All patients after initial detailed clinical evaluation and assessment were subjected to treatment and a periodical review was done. A minimum of three months follow up was done.

EXCLUSION CRITERIA

- Recurrent cases and follow up cases of orbital Pseudo tumor, presenting initially during this period were excluded.
- Patients having other identifiable and similar condition, such as orbital tumor, Thyroid orbitopathy, Rheumatoid arthritis, Wegners granulomatosis and sarcoidosis and orbital cellulitis were excluded.

METHODOLOGY

- Patient clinically diagnosed with Detailed Ophthalmic & Medical History
- General Physical Examination
- Examination of ENT and PNS
- Comprehensive Ophthalmic Evaluation
- Visual Acuity , Refraction, IOP
- Anterior Segment Examination – Slit Lamp
- Proptosis-Hertels exophthalmometer

- Fundus Examination-90 D Lens
- Colour Vision – Ishihara's Chart, Visual Fields – Bjerrum's screen
- Investigation
    - Hematological- P ANCA , C- ANCA , RA factor and Sr.ACE , In selected cases to rule out other identifiable cause.
    - Thyroid function test in all cases.
    - USG B Scan.
    - CT Scan Orbit/ MRI scan.
    - Histopathology if indicated.

EXPECTED OUTCOME

- Mean Age of presentation
- Male to Female Ratio
- Laterality
- Most common clinical symptoms
- Most common clinical features
- Most common type of Pseudo tumor.
- Indication of biopsy
- Response to the treatment

RESULTS

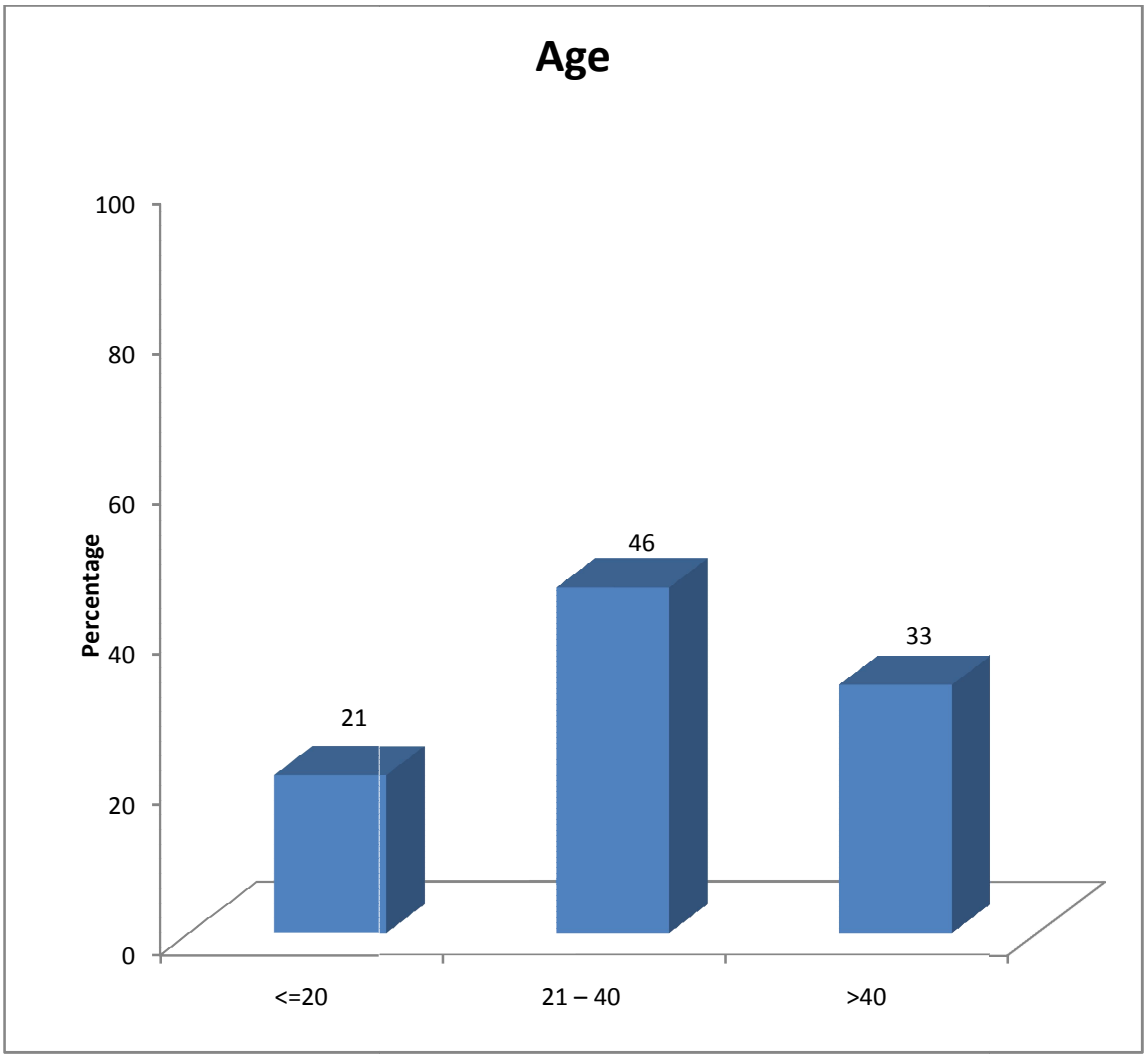
A total of 24 patients with Non specific orbital inflammation were identified.

AGE

Mean (SD) of the age is 34.63(14.0) and the range is 7 to 62 years

Age category	N	%
<=20	5	20.8
21 – 40	11	45.8
>40	8	33.3
Total	24	100.0

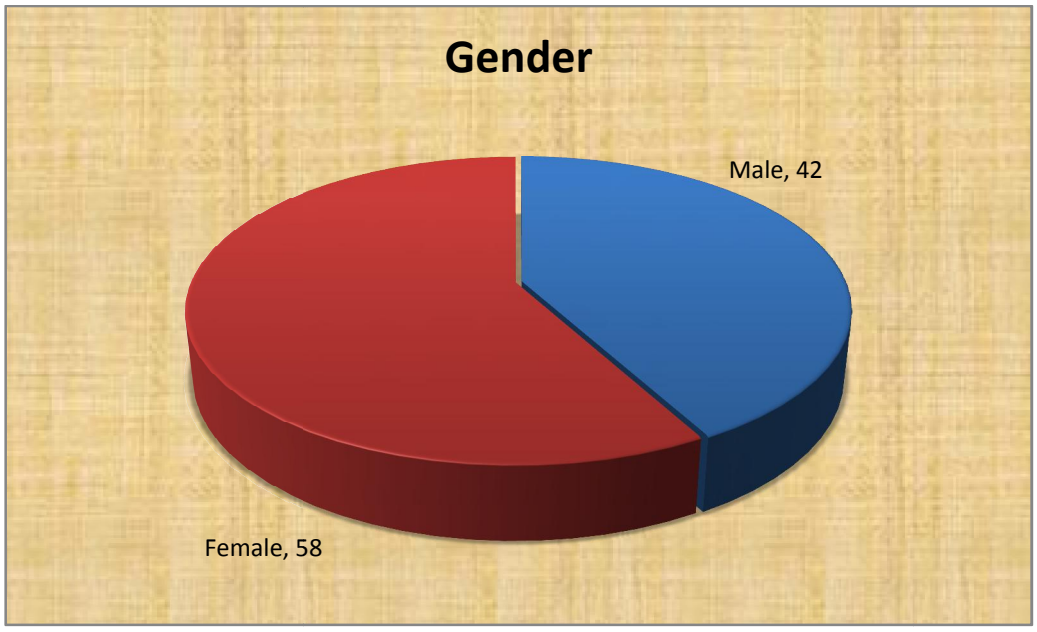
Age wise distribution showed 20 % in the age group of less than 20 years , 45.8 % in21 – 40 years,33.3 % in the age group of more than 40 years.


Age
100
80
60
40
20
0
Percentage
21
46
33
<=20
21 – 40
>40

SEX

Sex	N	%
Male	10	41.7
Female	14	58.3
Total	24	100.0

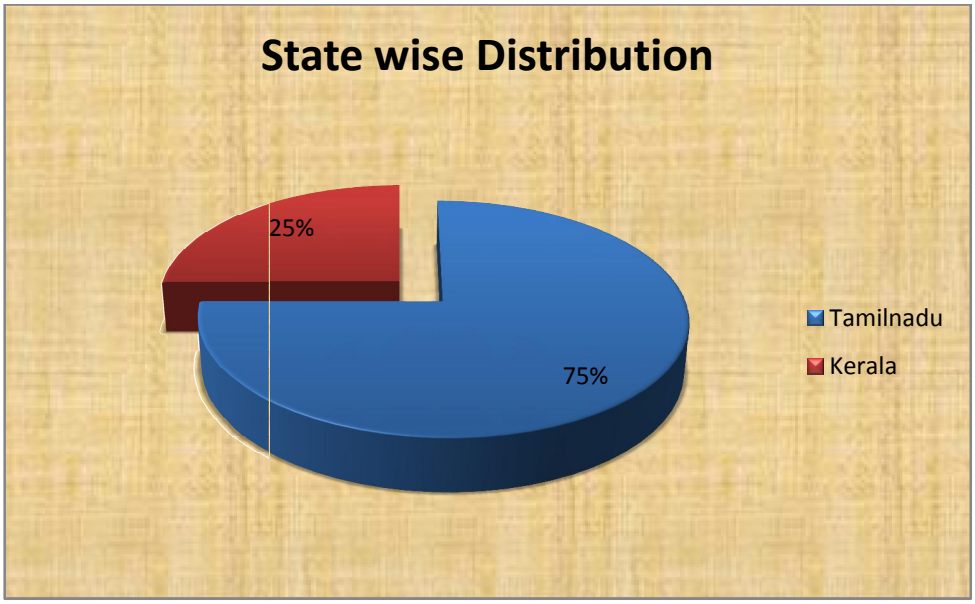
Sex wise distribution of NSOI, showed 41.7 % in males and 58.3% in females.



PLACE

Place	N	%
Tamil nadu	18	75.0
Kerala	6	25.0
Total	24	100.0

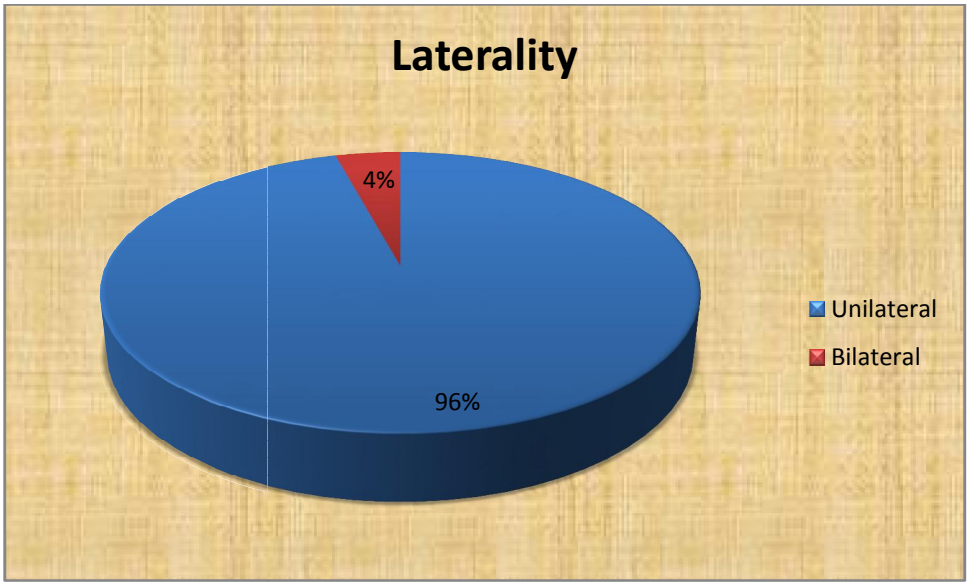
State wise distribution of cases showed that 25% of cases were from Kerala and 75 % from Tami lnadu.



LATERALITY

Laterality	N	%
Unilateral	23	95.8
Bilateral	1	4.2
Total	24	100.0

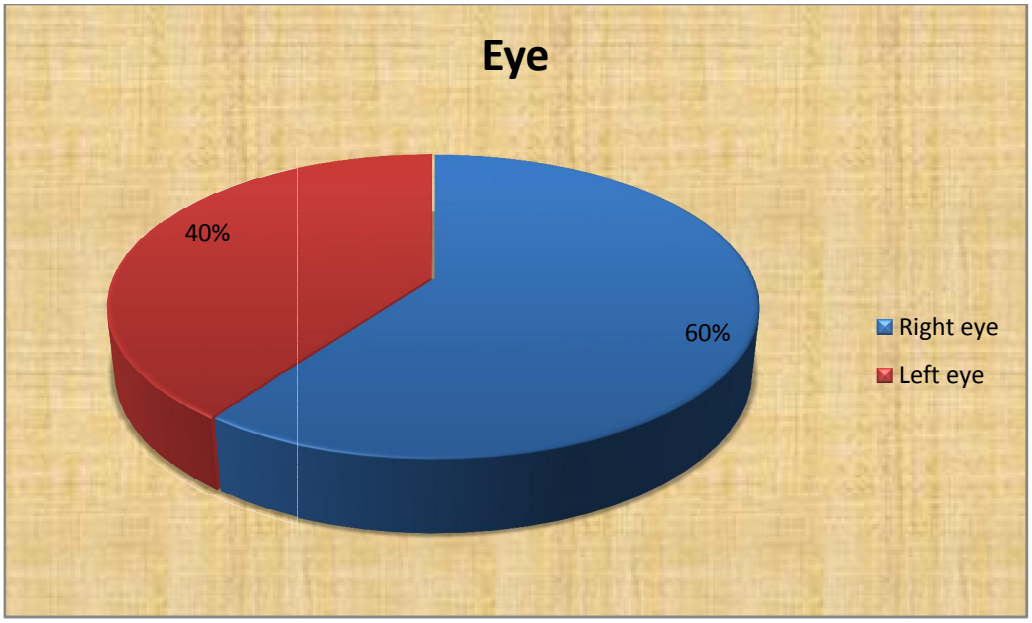
95.8 % cases were unilaterally involved and bilateral involvement was present in 4.2 % case.



EYE

Eye	N	%
Right eye	15	60.0
Left eye	10	40.0
Total	25	100.0

Right eye was involved in 60 % of cases and Left eye was involved in 40 % of cases.



PRESENTING COMPLAINTS

DEFECTIVE VISION

Defective vision	N	%
No	23	92.0
Yes	2	8.0
Total	25	100.0

DOUBLE VISION

Double vision	N	%
Absent	21	84.0
Present	4	16.0
Total	25	100.0

DROOP

Droop	N	%
Absent	22	88.0
Present	3	12.0
Total	25	100.0

PROTRUSION

Protrusion	N	%
Absent	19	76.0
Present	6	24.0
Total	25	100.0

PAIN

Pain	N	%
Absent	5	20.0
Present	20	80.0
Total	25	100.0

REDNESS

Redness	N	%
Absent	20	80.0
Localized	4	16.0
Diffuse	1	4.0
Total	25	100.0

WATERING

Watering	N	%
Absent	22	88.0
Present	3	12.0
Total	25	100.0

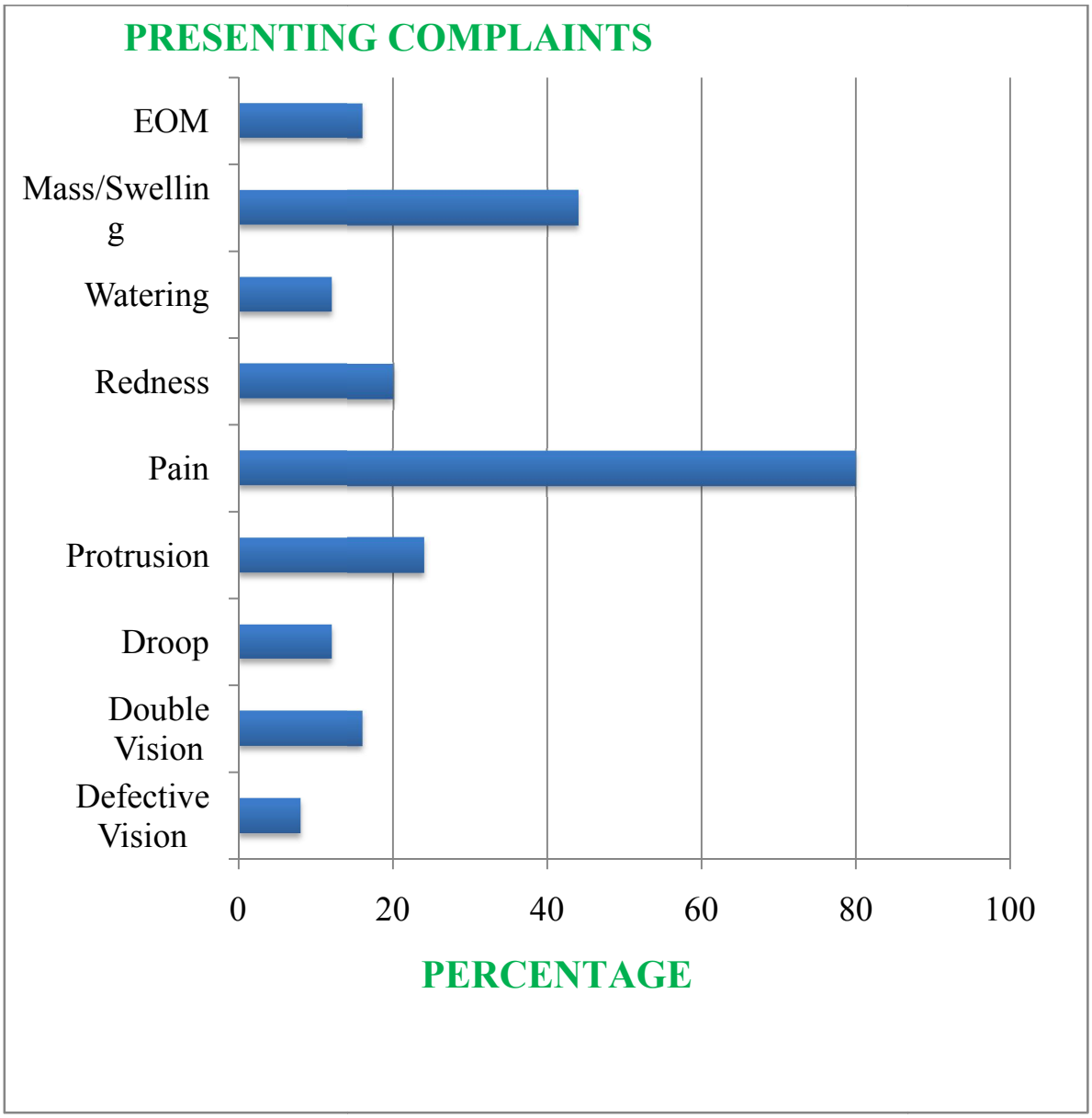
MASS/SWELLING

	N	%
Absent	14	56.0
Present	11	44.0
Total	25	100.0

EOM

	N	%
Absent	21	84.0
Present	4	16.0
Total	25	100.0

Vision was affected in 8% of cases and double vision was present in 16% of cases, Drooping of eyelids seen in 12% , protrusion in 24% , pain in 80 % ,redness in 20% ,Watering in 12% , and mass was felt in 44% of cases.EOM restriction was seen in 16% of patients.

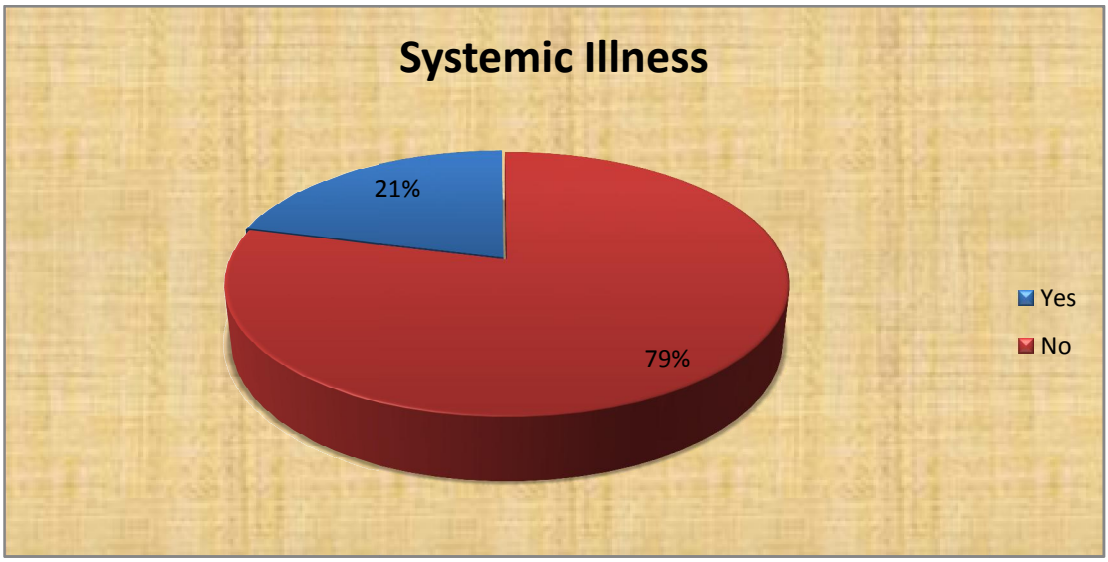


SYSTEMIC ILLNESS

	N	%
Yes	**5**	**20.8**
Diabetes	2	40.0
HT	2	40.0
Diabetes/HT/Cardiac	1	20.0
No	**19**	**79.2**
Total	24	100.0

Associated systemic comorbidity present in 20.8 % .

General examination, systemic examination and ENT Examination were normal in all patients.



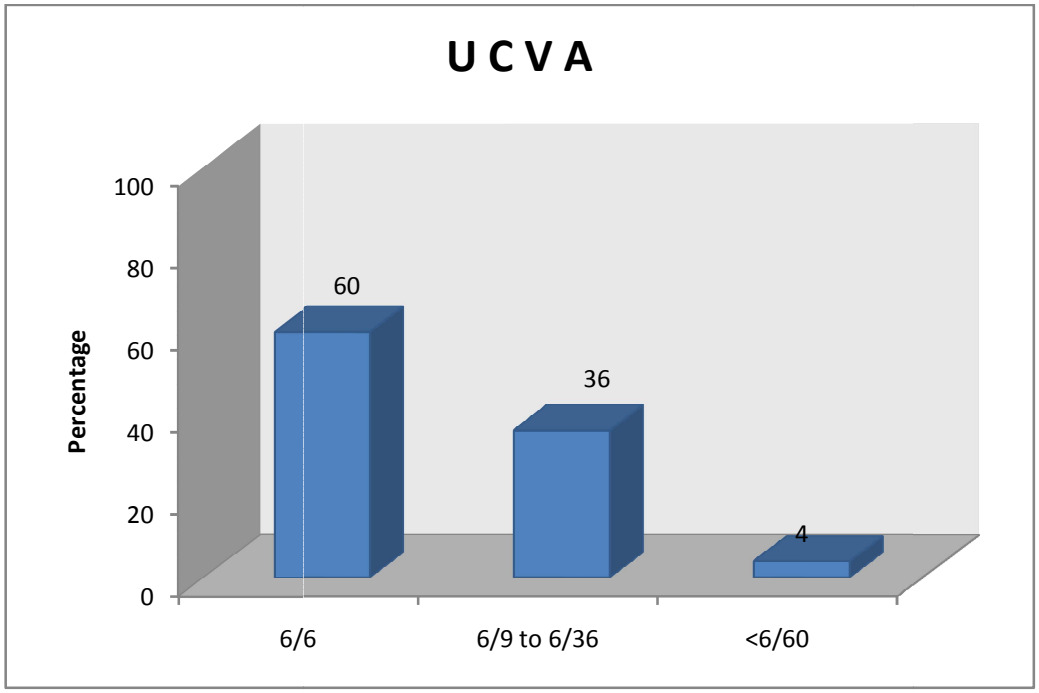
VISUAL ACUITY

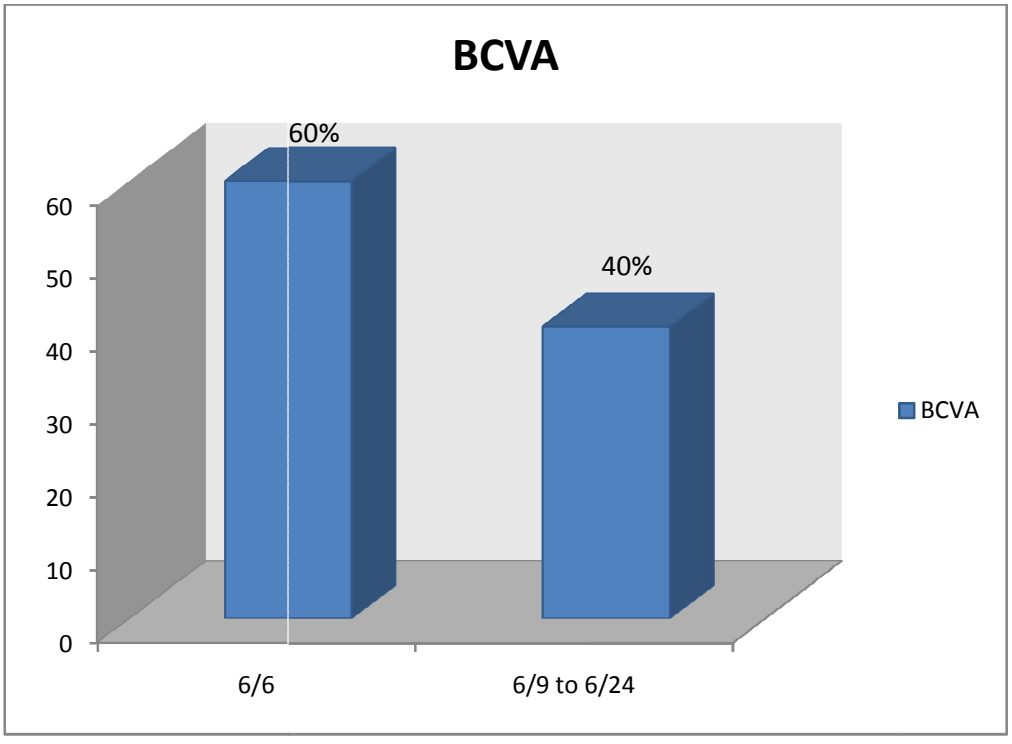
UCVA	N	%
6/6	15	60.0
6/9 to 6/36	3	12.0
5/60	1	4.0
Total	25	100.0

BCVA	N	%
6/6	6	60.0
6/9	1	10.0
6/18	1	10.0
6/24	2	20.0
Total	10	100.0

60% of patients had uncorrected visual acuity of 6/6. 36 % of patients showed UCVA of 6/9 to 6/36. 1 patient showed visual acuity of 5/60.

60% of patients showed Best corrected visual acuity of 6/6, and 40 % patient showed BCVA between 6/9 to 6/24.





IOP

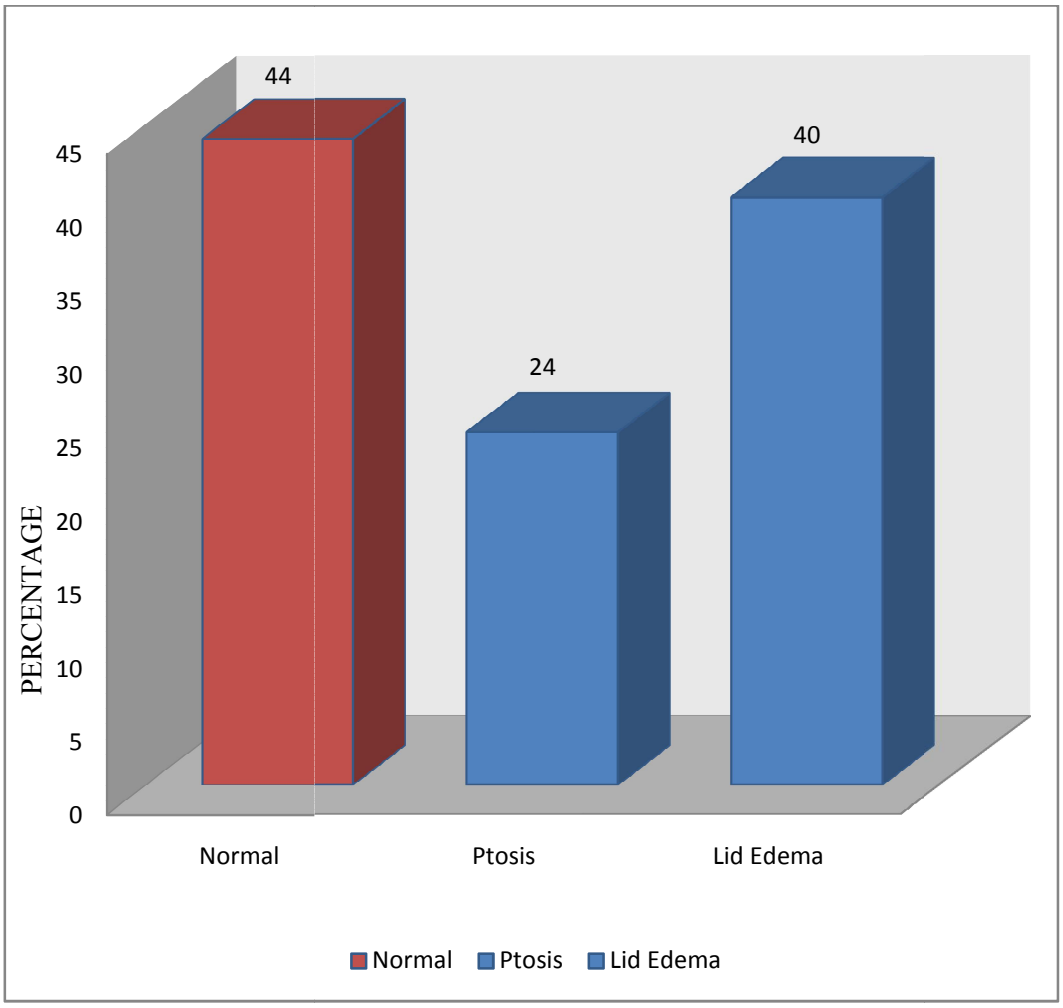
Mean intraocular pressure was 15.88 ranging from 10 to 20.

	N	Mean(SD)	Min-Max
IOP	25	15.88(2.89)	10 to 20

OCULAR EXAMINATION

Lids	Present N(%)	Absent N(%)	Total N(%)
Lids signs	11(44.0)	14(56.0)	25(100.0)
Ptosis	6(24.0)	19(76.0)	25(100.0)
Lid lag	-	25(100.0)	25(100.0)
Lagophthalmus	-	25(100.0)	25(100.0)
Lid edema	10(40.0)	15(60.0)	25(100.0)

Eye lids were normal in 44% of cases. Ptosis was present in 24% of cases and lid edema was presented in 40% of cases.

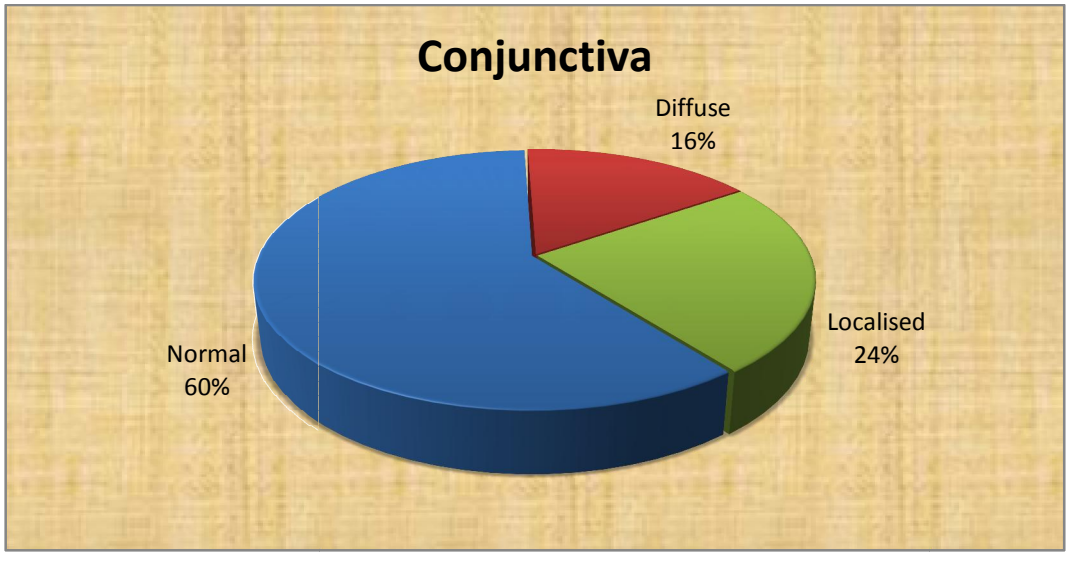

44
40
24
45
40
35
30
25
20
15
10
5
0
PERCENTAGE
Normal
Ptosis
Lid Edema
Normal
Ptosis
Lid Edema

CONJUNCTIVA

	N	%
Normal	15	60.0
Congested (abnormal)	**10**	**40.0**
Diffuse	4	16.0
Localized	6	24.0
Total	25	100.0

Conjunctiva was normal in 60 % of cases, while it was congested in 40% of cases.

Localised congestion was present in 24% and diffuse congestion was present in 16%.



ANTERIOR SEGMENT

	N	%
Cornea		
Clear	25	100.0
ACD		
Normal Depth	25	100.0
ACD Reaction		
No Reaction	25	100.0
Iris		
Normal	25	100.0
Pupil		
Normal	24	96.0
RAPD	1	4.0
Lens		
Clear	25	100.0

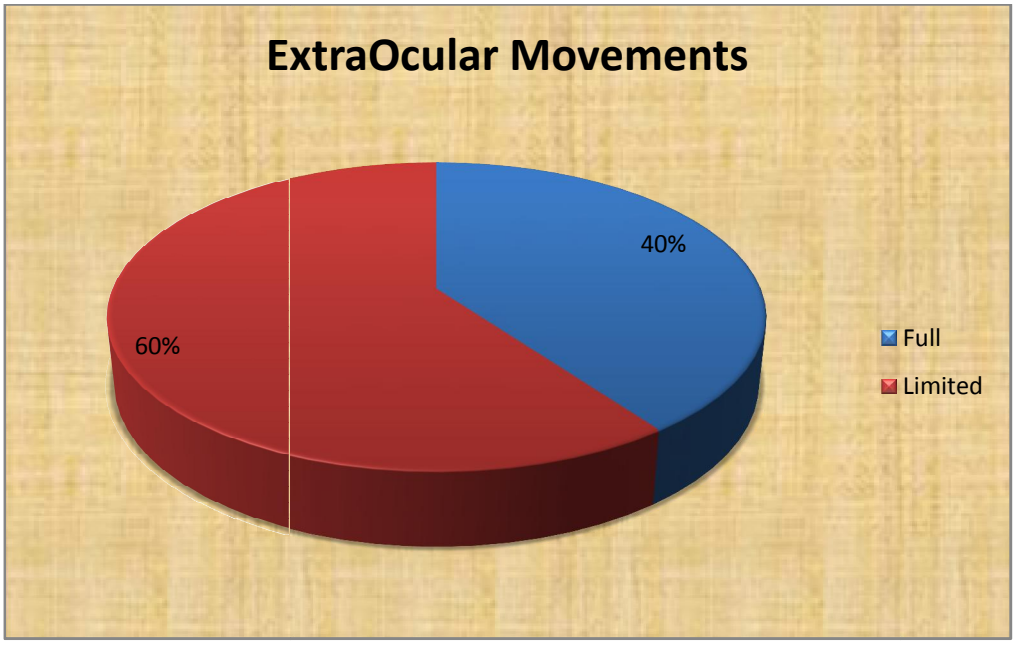
Other anterior segment examinations including cornea , Anterior chamber ,Iris And Lens were normal.

Pupillary reaction was normal in 96% of cases and 1 patient(4%) showed RAPD.

EXTRAOCULAR MOVEMENTS

	N	%
Full	10	40.0
Limited	15	60.0
Total	25	100.0

Extra ocular movements were full in 40% of cases and limited in 60% of cases.

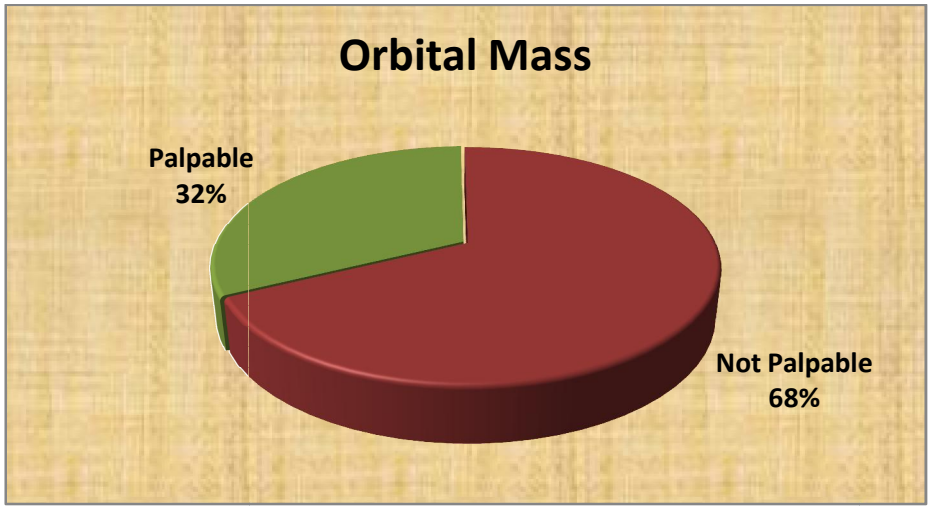


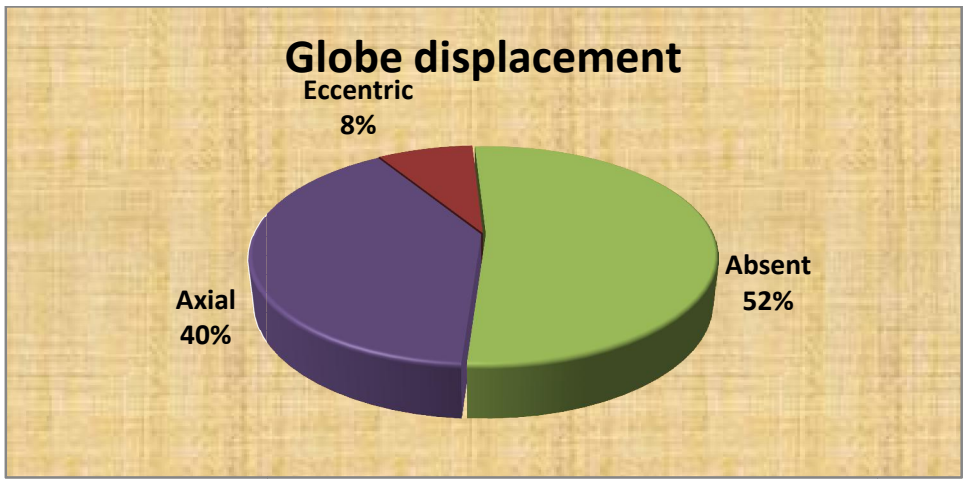
ORBITAL MASS

	N	%
Orbital mass		
Not Palpable	17	68.0
Palpable	8	32.0
Pre Orbital rim		
Continuous	25	100.0
Interrupted	-	-
Globe displacement		
Present	12	48.0
Absent	13	52.0
If Present,		
Axial	10	83.3
Eccentric	2	16.7
Globe retropulsion		
Present	1	4.0
Absent	24	96.0
Thrill/Pulsations		
Present	-	-
Absent	25	100.0
Valsalva		
Positive	-	-
Negative	25	100.0

Mass was felt in 32% of cases and 4% of cases showed resistance to retropulsion.

Proptosis was present in 48% of cases and absent in 52% of cases. Axial proptosis was seen in 83.3%,Eccentric proptosis was seen in 16.7%. Variation with Valsalva manouever was absent in all cases and bruit was absent in all cases.





POSTERIOR SEGMENT

	N	%
Normal	24	96.0
Abnormal (Pale disc)	1	4.0
Total	25	100.0

Fundus was normal in 96% of cases , 1 patient (4 %) showed pale disc.

COLOUR VISION, CENTRAL FIELDS

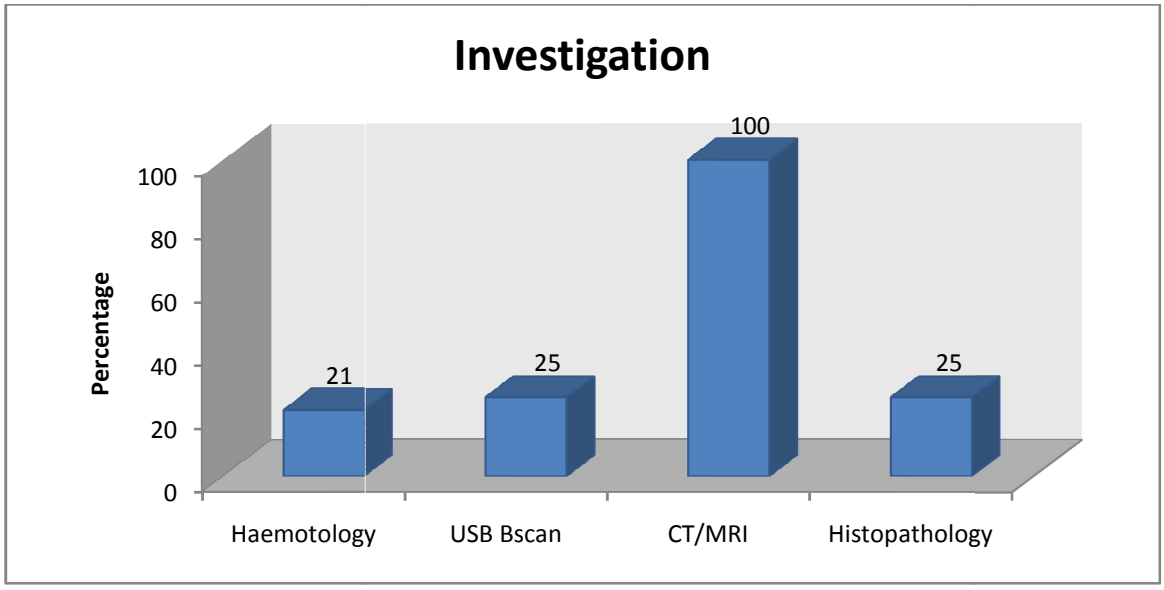
	Normal	Abnormal	Total
Colour vision	23(95.8)	1(4.2)	24(100.0)
Central fields	23(95.8)	1(4.2)	24(100.0)
TFT	24(100.0)	-	24(100.0)

Colour vision and central fields were normal in 95.8 % of cases and 4.2 % showed defective color vision and fields.

INVESTIGATION

Investigation	N	%
Hematology	5	20.8
USB Bscan	6	25.0
CT/MRI	24	100.0
Histopathology	6	25.0

Hematological examination was done in 20.8 % of cases, USG B scan was done in 25% of cases and histopathology was done in 25% of cases.



HISTOPATHOLOGY

	N	%
Not done	18	75.0
Done	6	25.0
Total	24	100.0

On Histo pathology, sclerosing form of orbital pseudo tumor was seen in 3 cases(50%). Inflammatory Myositis was seen in 1 case(16.6%). Inflammatory.

Pseudo tumor with granulomatous eosinophilic angitis was seen in 1 case(16.6%).

1 case(16.6%) showed only mature adipocytes (in-conclusive).

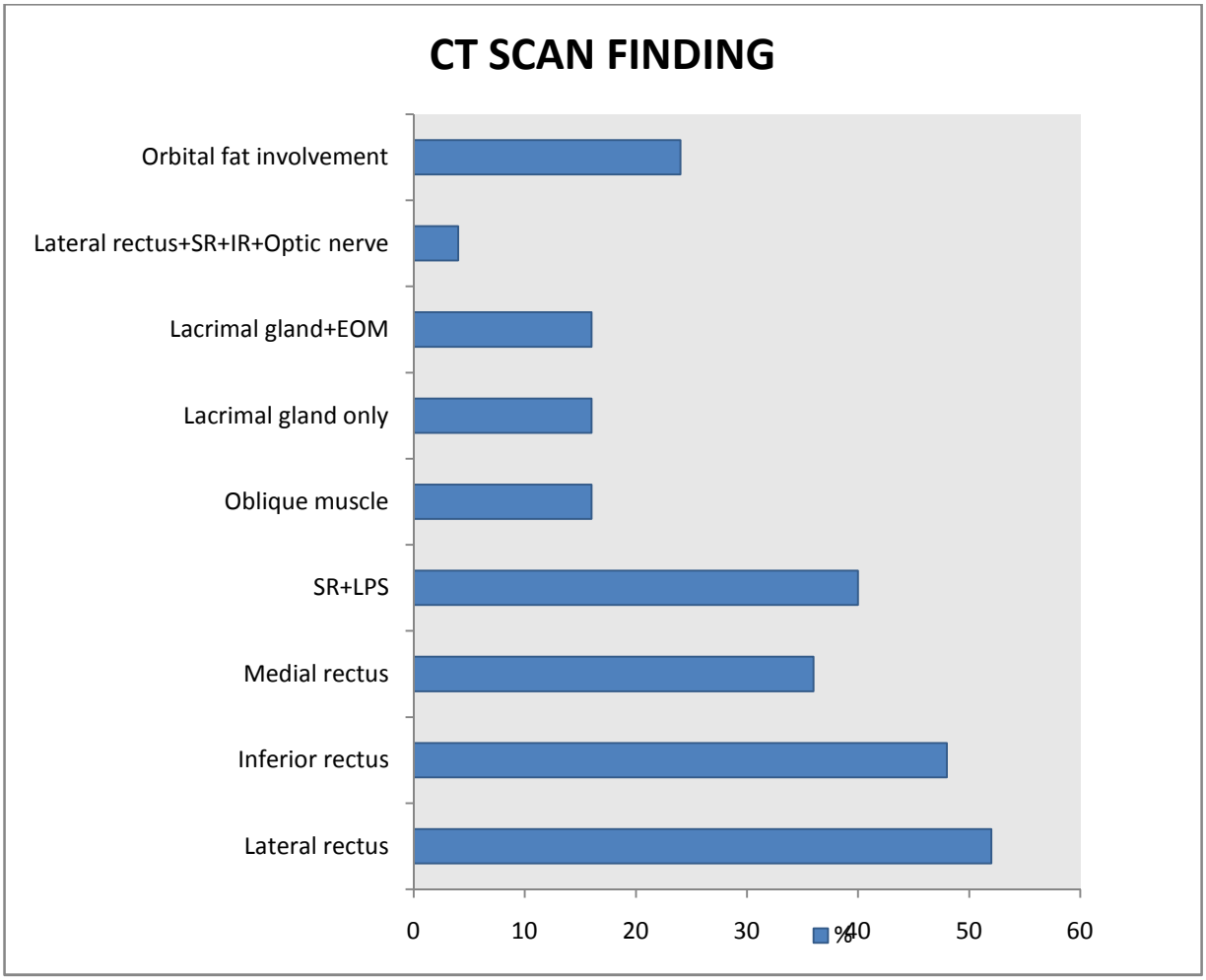
CT SCAN

CT scan finding	N	%
Lateral rectus	13	52.0
Inferior rectus	12	48.0
Medial rectus	9	36.0
SR+LPS	10	40.0
Oblique muscle	4	16.0
Lacrimal gland only	4	16.0
Lacrimal gland+EOM	4	16.0
Lateral rectus+SR+IR+Optic nerve	1	4.0
Orbital fat involvement	6	24.0

On CT Scan enlargement of lateral rectus was seen in 52 % of cases ,inferior rectus in 48% , medial rectus in 36% , oblique muscle involved in 16% and superior muscle complex involved in 40% of cases. Lacrimal gland

involvement only seen in 16% of cases and lacrimal gland with EOM involved in 16% of cases.

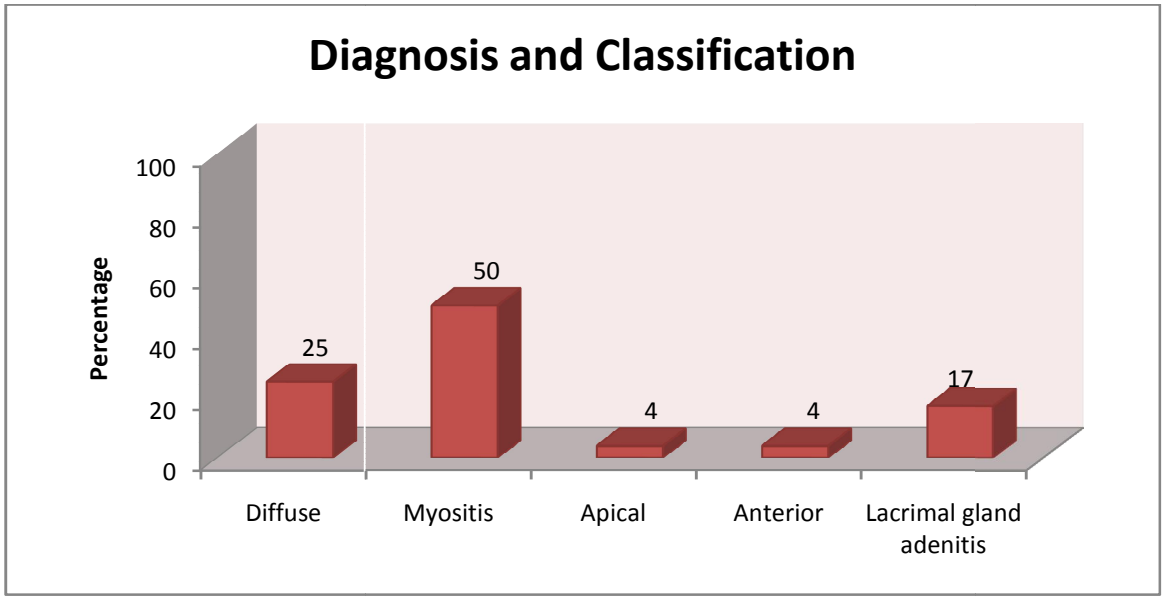
Optic nerve involved in 4% of cases. Involvement of orbital fat was seen in 24% of cases.



DIAGNOSIS AND CLASSIFICATION

	N	%
Diffuse	6	25.0
Myositis	12	50.0
Apical	1	4.2
Anterior	1	4.2
Lacrimal gland adenitis	4	16.7
Total	24	100.0

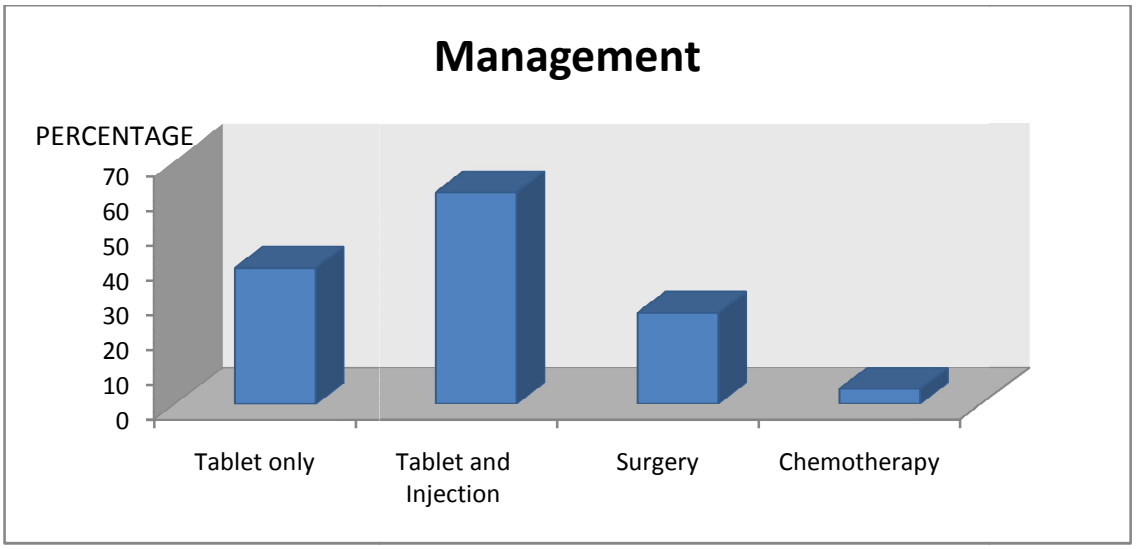
Myositis form of pseudotumor was seen in 50 % of cases .Diffuse form was seen in 25 % of cases ,and lacrimal gland involvement was seen in 16.7% of cases. Apical and anterior form of pseudotumor was seen in 4.2 % in each form.



MANAGEMENT

Management	N	%
Tablet only	9	39.1
Tablet and Injection	14	60.9
Surgery	6	26.1
Chemotherapy	1	4.3

In medical therapy, tablets were given in 39.1 % of cases, tablets and injection were given in 60.9 % of cases. Surgical treatment was given in 26.1 % of cases. Chemotherapy was indicated in 4.3 % of cases.



MEDICAL MANAGEMENT

	Tablet	Injection	Injection, Tablet	Total
Baseline	10(45.5)	-	12(54.5)	22
1st visit	17(77.3)	-	5(22.7)	22
2nd visit	9(90.0)	-	1(10.0)	10
3rd visit	1(100.0)	-	-	1
Study period	9(39.1)	-	14(60.9)	23

SURGICAL

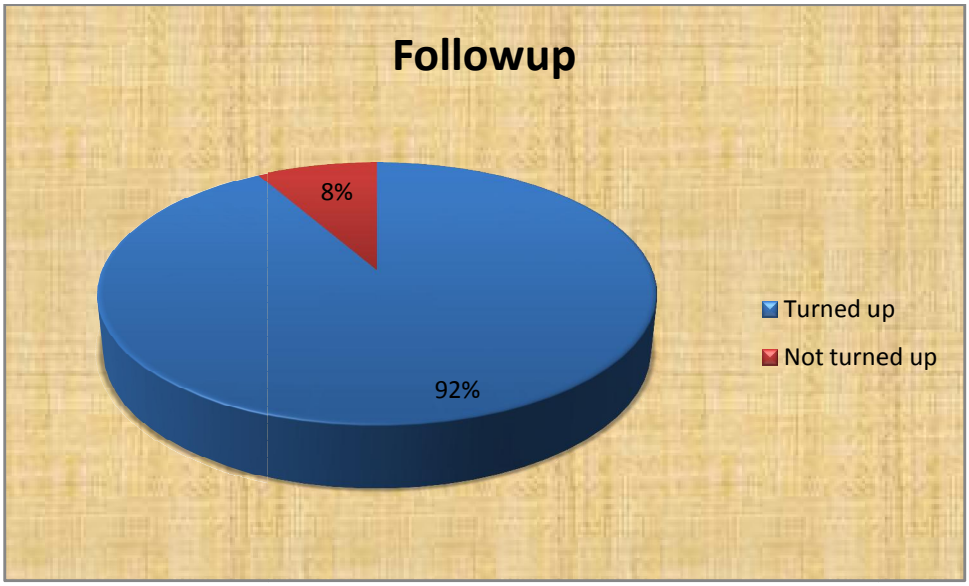
	N	%
Incision biopsy	4	50.0
Medial Orbitotomy	1	16.7
Supero lateral Orbitotomy with mass excision biopsy	1	16.7
Total	6	100.0

In surgical therapy incision biopsy was instituted in 50% of cases. Medial orbitotomy was done in 16.7 % and lateral orbitotomy in 16.7% of cases.

FOLLOWUP

Came for Followup	N	%
Yes	22	91.7
No	2	8.3
Total	24	100.0

In review 91.7% of cases were come for followup and 8.3 % of cases lost their follow up.

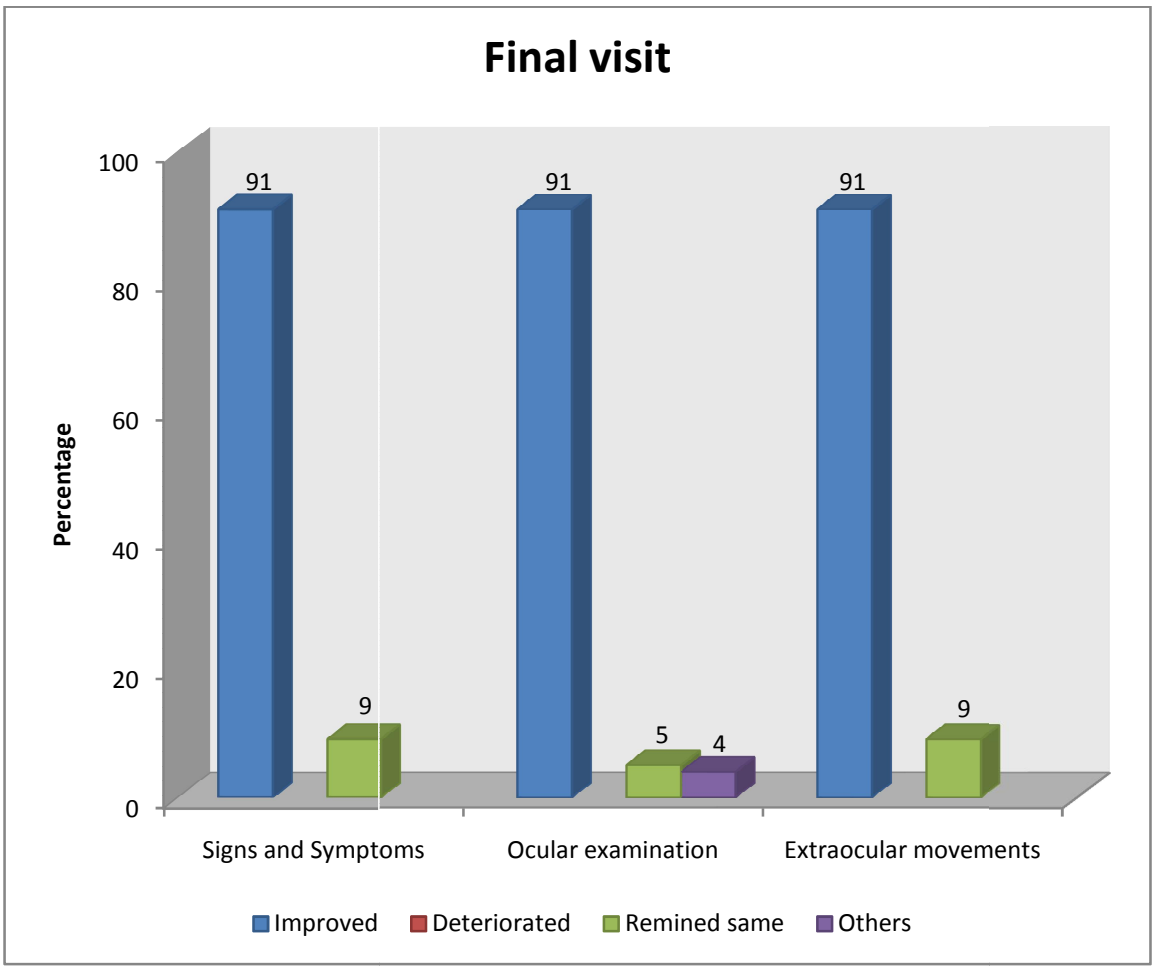


Followup 1	Improved	Deteriorated	Remained same	Others	Total
Signs and Symptoms	17(73.9)	-	6(26.1)		23
Ocular examination	17(73.9)	-	5(21.7)	1(4.4)	23
Extraocular movements	18(78.3)		5(21.7)		23
Followup2					
Signs and Symptoms	10(90.9)	-	1(9.1)		11
Ocular examination	9(81.8)	-	1(9.1)	1(9.1)	11
Extraocular movements	10(90.9)		1(9.1)		11
Final Visit					
Signs and Symptoms	21(91.3)	-	2(8.7)		23
Ocular examination	21(91.3)	-	1(4.4)	1(4.4)	23
Extraocular movements	21(91.3)		2(8.7)		23

On first follow up 17 (73.9%) patients showed improvement and 6 patients (26.1%) were remained in same condition.

On second follow up 10 (90.9%) patients showed improvement and 1 patients (9.1%) were remained in same condition.

On final visit 21 (91.3) patients showed improvement and 2 patients (8.7%) were remained in same condition and were subjected to revised plan of treatment.



COMPLICATION

	N	%
Steroid Induced CSCR	1	33.3
Steroid Induced Diabetes	1	33.3
Nil	1	33.3
Total	3	100.0

In complication due to steroid treatment 33.3 % showed CSCR and other 33.3% showed diabetes.

RECURRENCE SEEN

	N	%
Present	2	8.3
Nil	22	91.7
Total	24	100.0

Recurrence was seen in 8.3 % of cases in 3 months of follow up.

DISCUSSION

In our study, results corresponded to the clinical features reported in literature. In our study, totally 24 patients (25 eyes) were enrolled in duration of 18 months. Enrolled patients were in the age range of 7 to 60 years and the mean age of presentation was 34.63 years. 10 patients were male and 15 patients were female, 23 had unilateral involvement and 1 patient had bilateral involvement. RE involvement was seen in 15 and LE involvement was seen in 10 in our study. Similar study conducted by Niphon Chirapapaisan et al[1] retrospectively for 10 years included 49 patients with mean age of 43.75 years. Their study involved 24 males and 25females, out of which 36 Patients had unilateral involvement.

B N Swamy et al[6] conducted a retrospective study of 24 patients in western population, age ranging from 14 to 75 years, (14 male and 10 female patients) and 23 patients had unilateral involvement.

RA Nugent et al[7] conducted a retrospective study on 16 patients in western world, age ranging from 6 to 79 years, in which 10 were male and 6 were female.

Sonia J .Ahn Yuen[4] et al did a retrospective study of 90 eyes in 65 patients in 10 year duration .Mean age of presentation was 45 years and 17 patients had bilateral involvement.

Most common complaints in our study was pain reported in 80% (20/ 24) followed by mass/swelling reported in 44%(11 /24) and protrusion presented in 24% (6/24). and most common presentation was EOM restriction in 60%, proptosis in 48%, lid edema 40% ,congestion 40% , Ptosis in 24% .

In comparison Niphon Chirapapaisan et al[1] in their study, found proptosis in 49%,lid swelling or lid mass in 22.4% ,orbital pain in 12.2% were most common clinical features and most common presentation was proptosis in 79.6%, Ophthalmoplegia in 61.2% and lid swelling in 44.9 %.

BN Swamy et al[6] found that swelling /mass was the most common presentation followed by proptosis and pain.(in Australian population).

In one literature[4], it is reported that pain is most common symptom in 69% and periorbital swelling in 75% of patients was most common sign.

So, it can be inferred that proptosis and ophthalmoplegia is the most common clinical feature in asian population whereas periorbital swelling is the most common clinical feature in western population.

In our study vision was affected in 8% of cases, Fundus was abnormal in showing disc pallor in 1 patient , whereas Niphon et al study showed vision loss in 24.5 % and Fundus examination revealed optic disc swelling in 4.8% and disc pallor in 1 patient (1.6%) . In Western literature[6] decreased vision and RAPD was present in 20.8% .

In our study myositis form of pseudo tumor was present in 50% of cases, followed by diffuse form of orbital pseudo tumor in 25 % ,lacrimal gland adenitis in 16.7 % , apical and anterior form of pseudo tumor in 4% of cases each. In contrast to our study, BN Swamy et al [6] found that orbital fat is the most common orbital structure involving 75% of patients, followed by lacrimal gland in 54% of patients and extra ocular muscle in 50%.

In other study R.A.Nugent et al[7] found that most common location was lacrimal followed by anterior pseudo tumor. Posterior , diffuse and myositic pseudo tumor were equally frequent.

Sonia J.Ahn Yuen et al[4] found that most common type of NSOI was dacryoadenitis in 21% ,myositis in 19 % of western population.

In our study Lateral rectus and inferior rectus involvement was seen almost equally followed by medial rectus and superior muscle complex .Other study[4] showed medial recti followed by superior recti are commonly involved.

In our study , On Histopathology it was found that sclerosing form of pseudo tumor was most common . In western population sclerosing form was present in 50 % followed by classical form in 41.2 %.[6]

The mainstay of therapy includes corticosteroids. The response to steroid is typically rapid and good and the same was observed in our study. All our patients were treated with systemic steroid and clinical improvement was seen in 91.3% of cases . Only two patients showed complication due to steroids and Chemotherapy (methotrexate) was started for one patient. Recurrence was found in 8.3 % of cases. In Niphon Chirapapaisan et al study 85% of patients showed clinical improvement, with recurrence rate of 20%.

In some study it was reported that recurrence rate in orbital myositis is15 -23%. In BJO, BN Swamy et al reported that 79.2% of patients were treated with steroids and 7 (29.2%) patients received chemotherapeutic drugs.

The differences of responses and recurrence might be due to differences in subtypes of NSOI ,diagnostic criteria, dose ,duration of treatment and natural history of disease.

	Our study	N.Chirapapaisan et al[1]	BN Swamy et al[6]	R.A.Nugent et al[7]	Yuen SJA et al[4]
DESIGN	Prospective	Retrospective	Retrospective	Retrospective	Retrospective
DURATION	18 months	10 years	10 years	10 years	10 years
SAMPLE SIZE	24	49	24	16	65
AGE RANGE(years)	7-60		14-75	6-79	
MEAN AGE	34.63	43.75			45
MALE: FEMALE	10:15	24:25	14:10	10:6	
COMMON CLINICAL PRESENTATION	Pain 80% and EOM restriction 60% and Proptosis in 48%	Proptosis in 79.6% and ocular motor deficit 61.2%	Swelling/mass	Pain and Proptosis	Pain 69%and periorbital swelling 75%
MOST COMMON TYPE OF NSOI	Myositic form		Orbital fat followed by Lacrimal	Lacrimal adenitis	Dacryo adenitis

CONCLUSION

A total of 24 patients with orbital pseudo tumor were enrolled in our study.

Age wise distribution of the study, showed that most common age group was second to fourth decade, with mean age of presentation being 34.63 years.

In this study Females were more commonly involved than males. In this study the most common state from where patients presented to us was Tamil Nadu followed by Kerala.

In this study 23 patients had unilateral involvement, with Right eye being more commonly involved than left eye.

The most common presenting symptom in our study was orbital pain followed by mass /swelling and protrusion.

Visual acuity was 6/6 in most of the patients .Common presentation in eyelids was lid edema followed by Ptosis.

Pupil examination was normal in all cases, except in one case which showed RAPD.

Fundus was normal in all cases except in one case which showed pale disc.

The most common presenting sign was Extra ocular movements restriction (60%) followed by proptosis . Axial proptosis was more common than Eccentric proptosis.

CT Scan was done on all cases and histopathology was indicated in selected cases.

In classification Myositis form of pseudo tumor was most common followed by diffuse form and lacrimal adenitis., Anterior and apical forms were equally frequent.

In myositis form Lateral rectus muscle was most commonly involved, followed by inferior rectus and medial rectus muscles.

On Histopathological subtypes, sclerosing form of Orbital Pseudo tumor was most common presentation.

Medical treatment with systemic steroids are mainstay of therapy in most of the cases. Most of the patients showed clinical improvements with systemic steroid only. Surgical therapy was indicated in selected cases. One patient was treated with chemotherapy (methotrexate).

In our study 91.7% of patients came for follow up , whereas 8.3 % lost their follow up.

In this study 2 patients showed steroid related complication like CSCR and diabetes.

In our study 2 patients showed recurrence of disease.

LIMITATIONS

Our study has number of significant limitations as it was a short duration study.

Long term follow up is needed as it is a diagnosis of exclusion. Spontaneous remission and recurrences can occur and a follow up of at least 2 years is needed

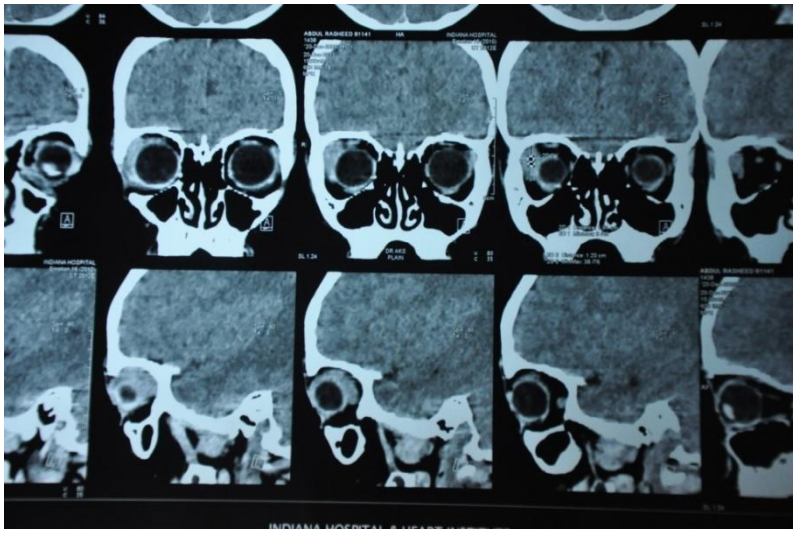
Further study with large number of patients and long term follow up is needed to understand the clinical manifestations and standard protocol for management of Orbital Pseudo tumor from other differentiating condition.

CLINICAL PHOTOS

PATIENT NAME: ABDUL RASHEED/7 YEARS /M

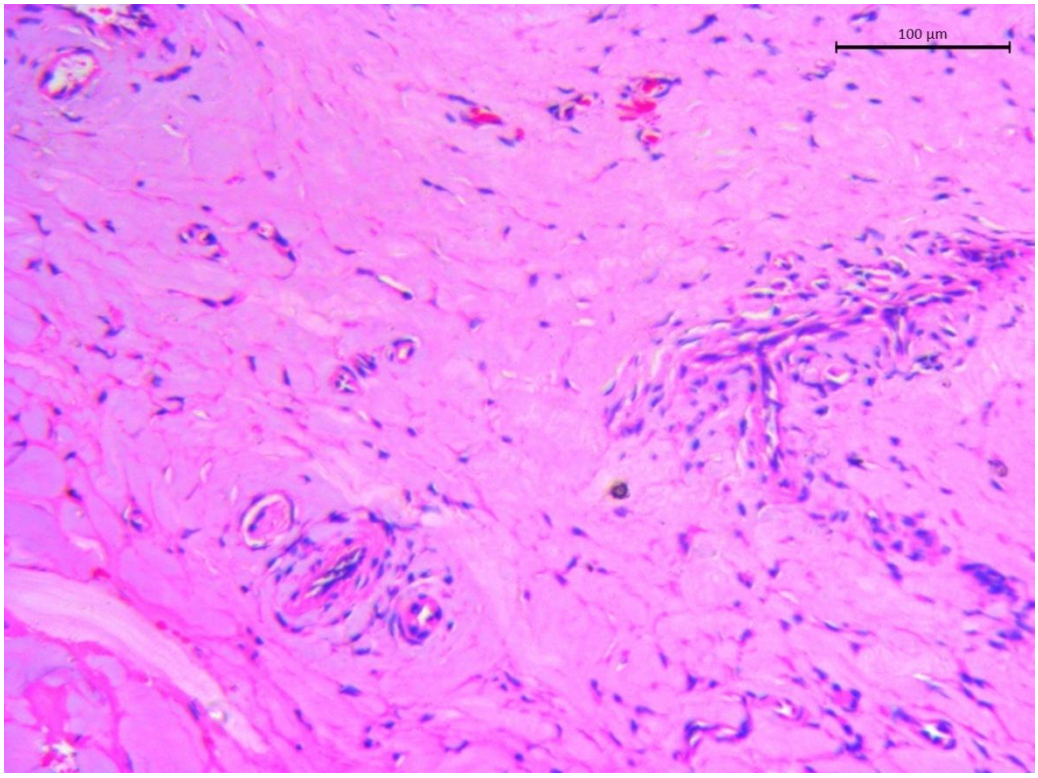
Presented with C/O. Swelling right eye

O/E. → 'S' Shaped lid deformity (+)



CT Orbit:

Hyper dense lesion and enlargement of Right Eye - lacrimal gland blends with lateral rectus.



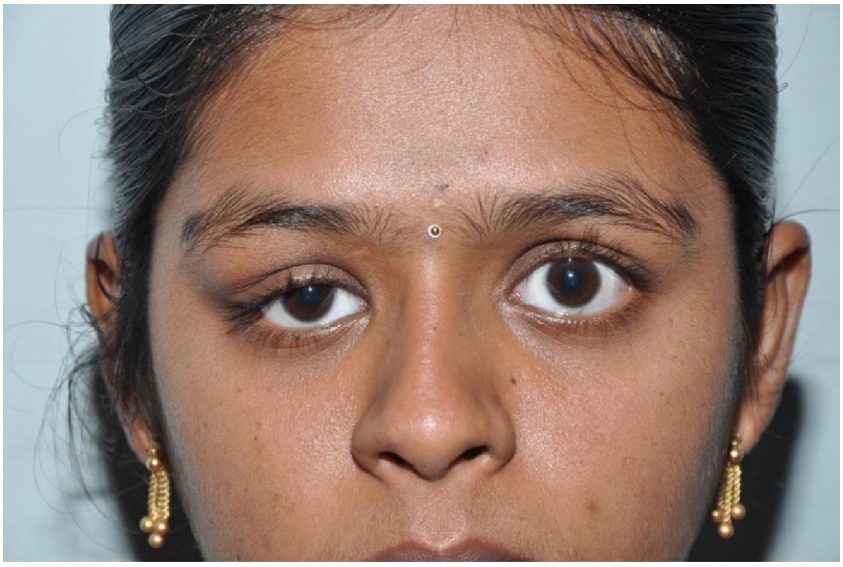
HPE:

Structure of fibrofatty tissue with dense hyalinised collagen and vascular proliferation. Perivascular lymphocytic infiltration with interspersed eosinophils are seen suggestive of Idiopathic sclerosing orbital inflammation.

Immunohistochemistry:

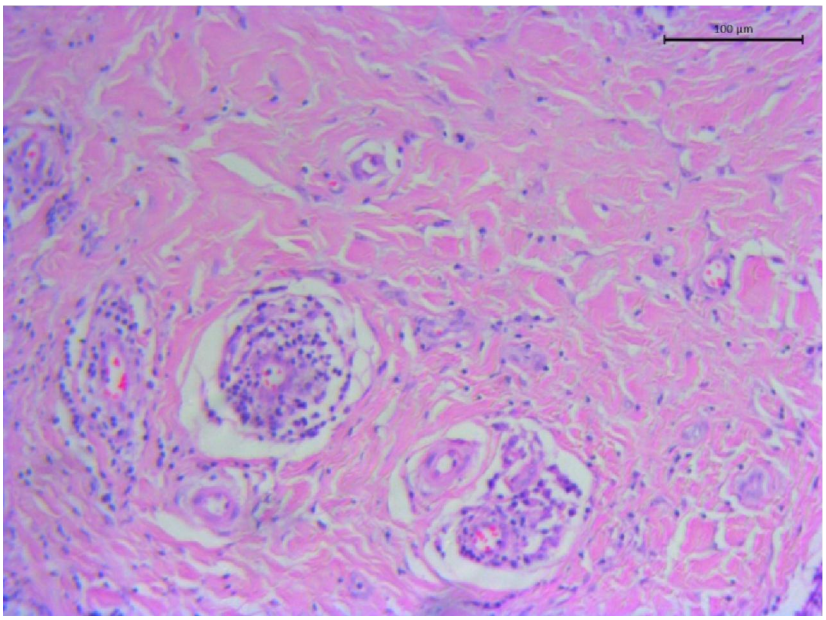
LCA: Diffuse positive reactivity

PATIENT: GAYATHRI DEVI/18/F



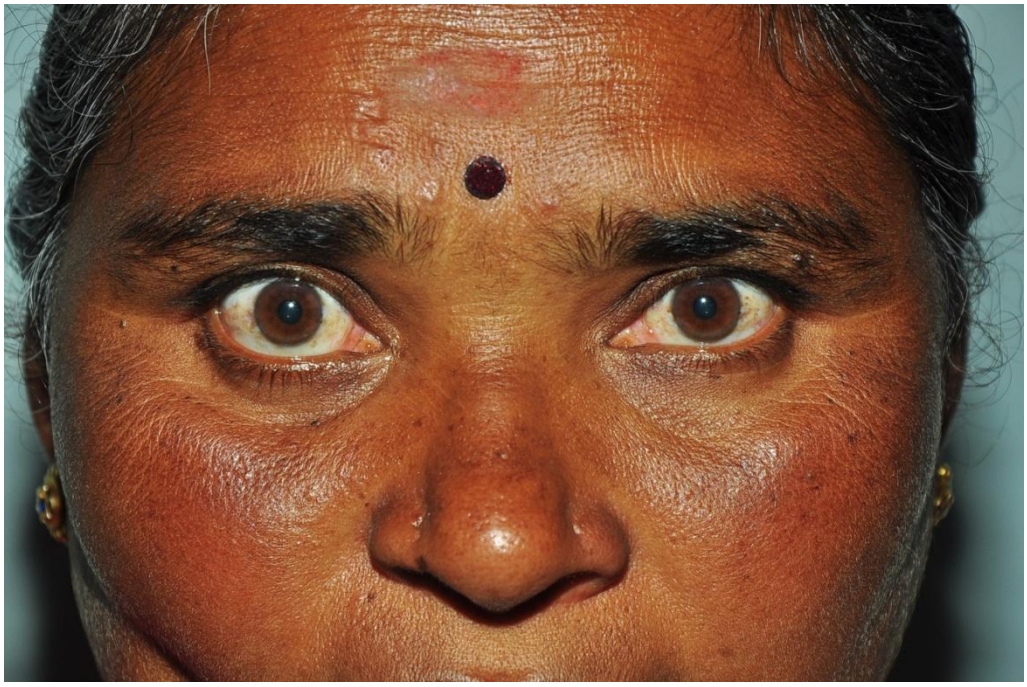
Presented with C/O. Swelling right eye

O/E. → 'S' Shaped lid deformity (+)

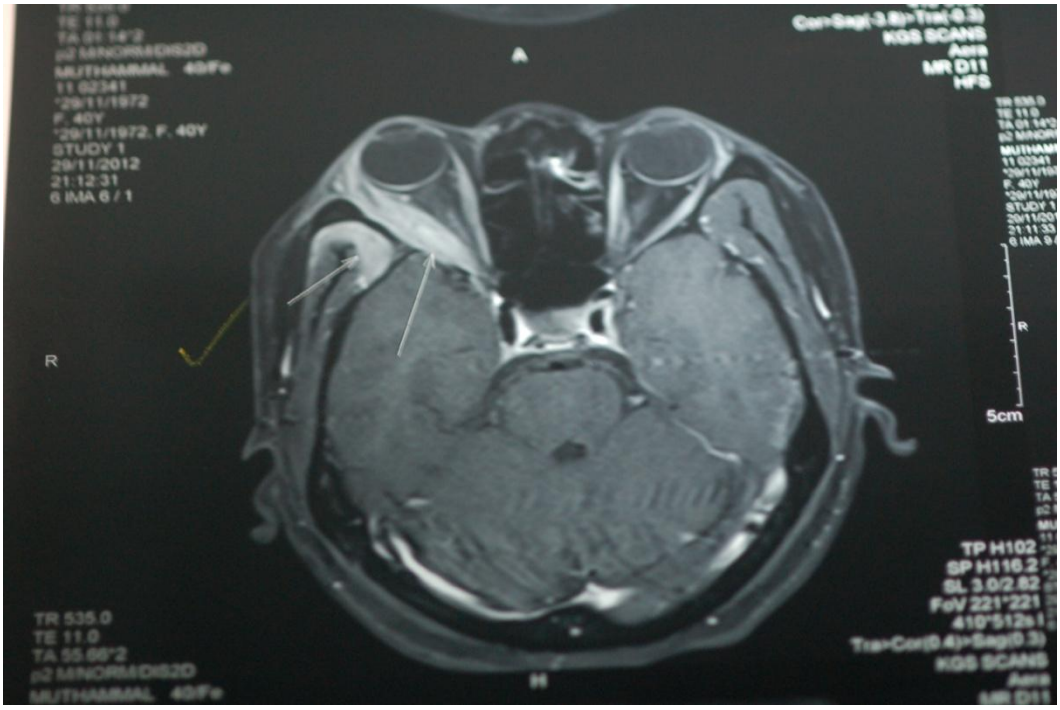


HPE report show features of inflammatory Pseudotumour with granulomatous eosinophilic necrotizing angitis.

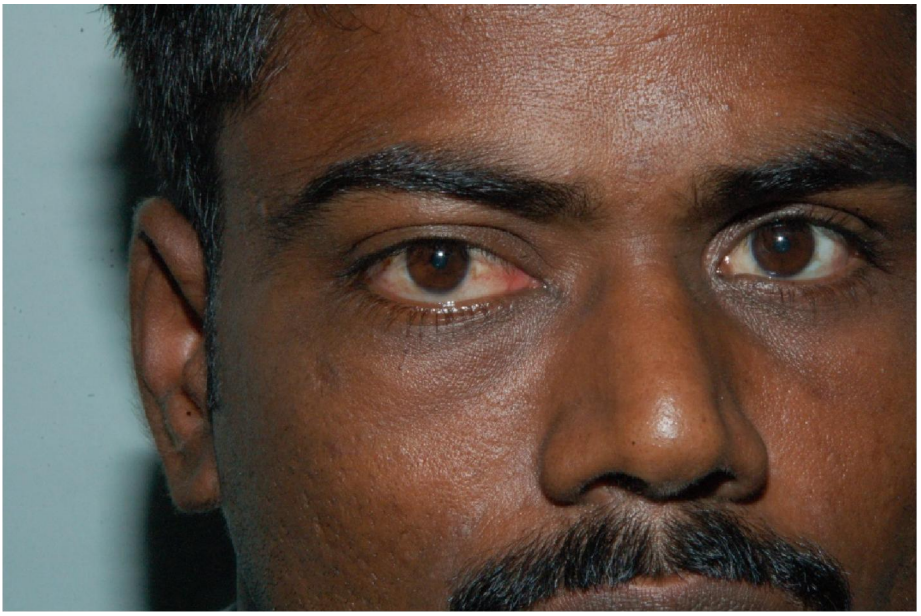
PATIENT: MUTHAMMAL/40/F



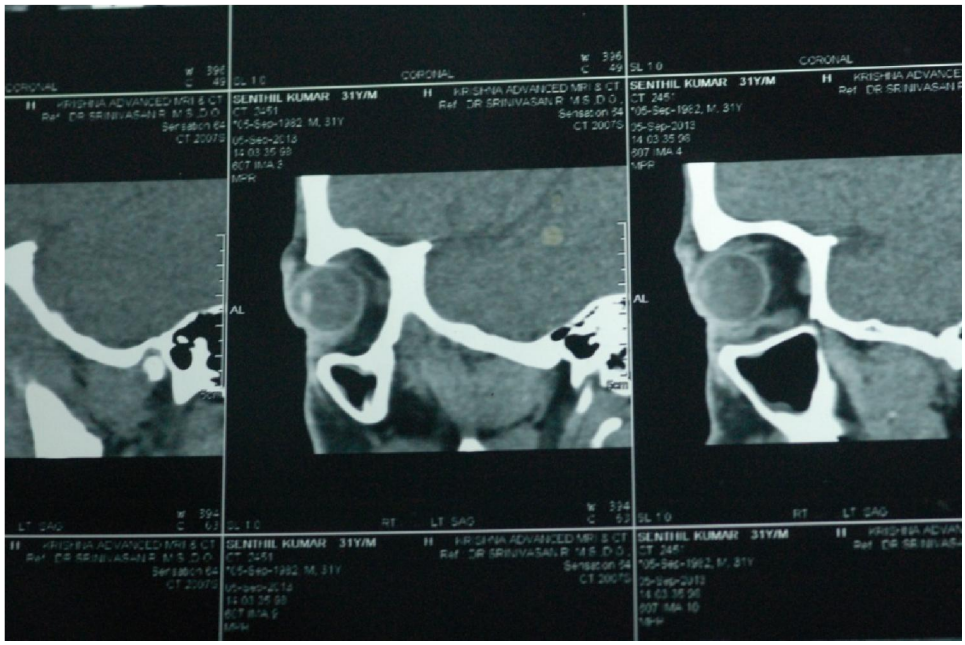
CASE OF MYOSITIS



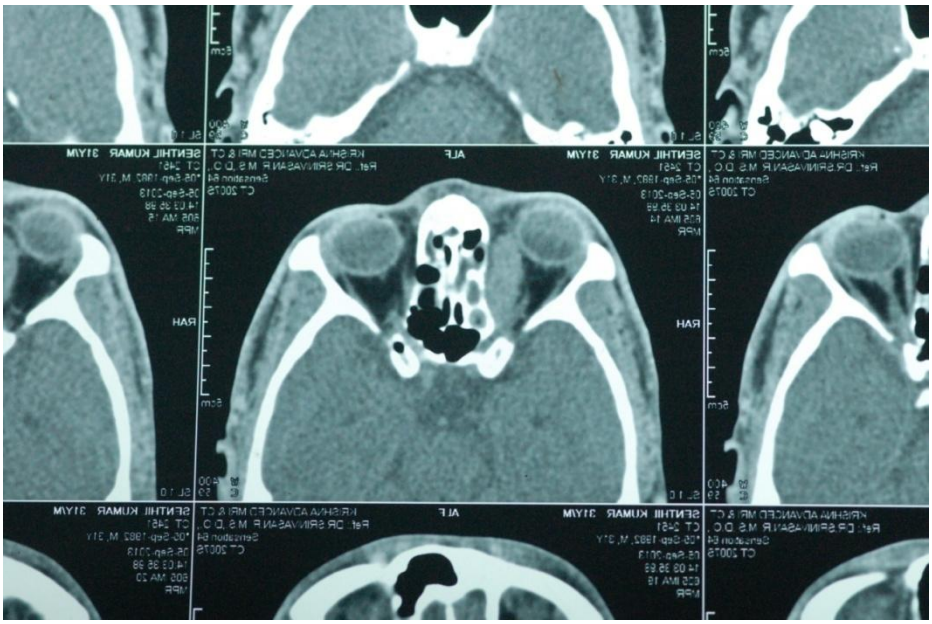
PATIENT: SENTHIL KUMAR

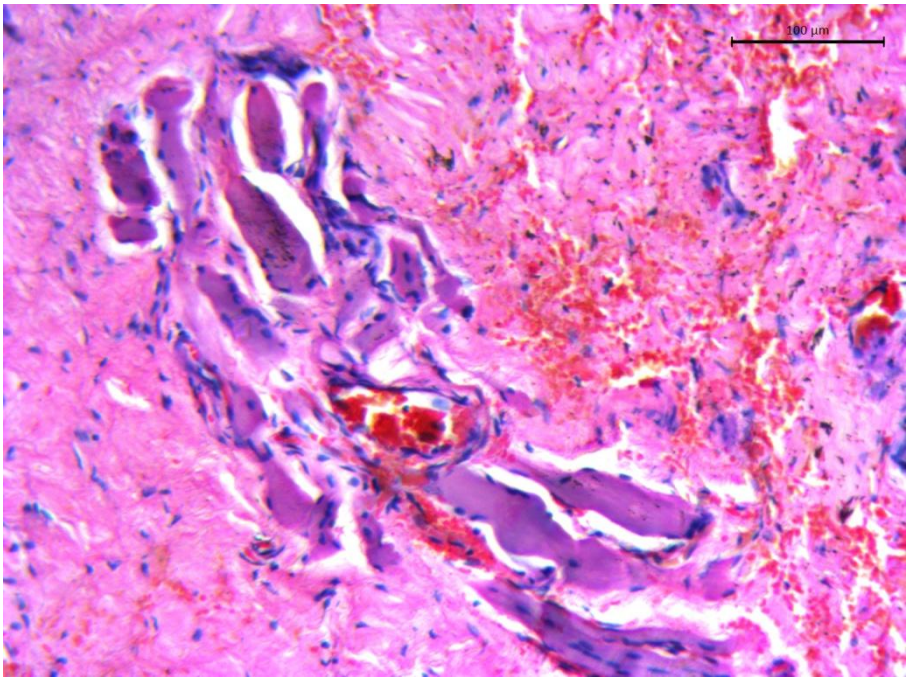


Presented with C/o. Protrusion of right eye



Thickened right medial rectus muscle and optic nerve

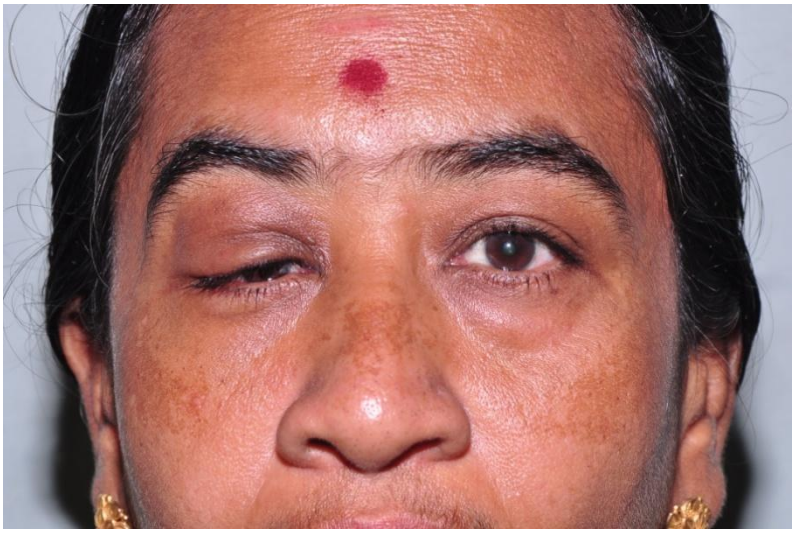




HPE Mature adipocytes and vessles with fibrotic and skeletal muscle fibres.

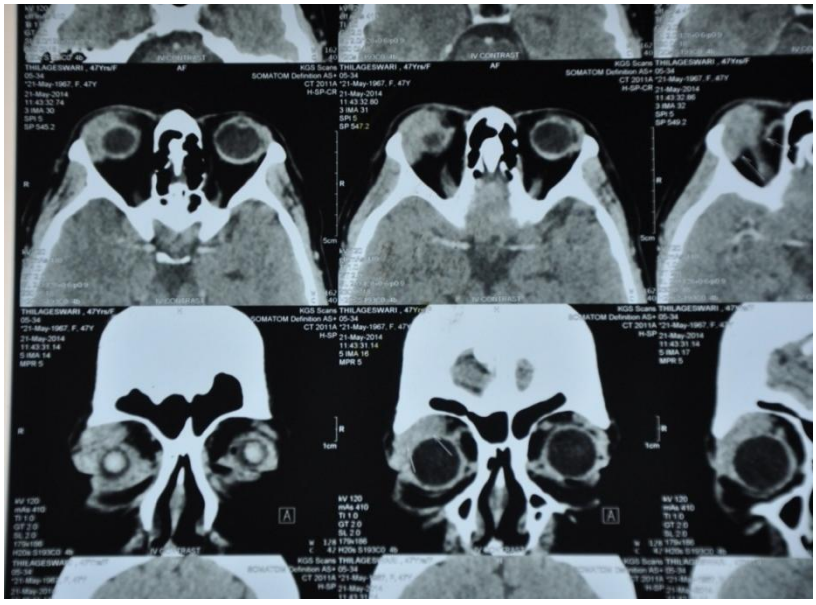
PATIENT: THILAGESHWARI

PRE OP



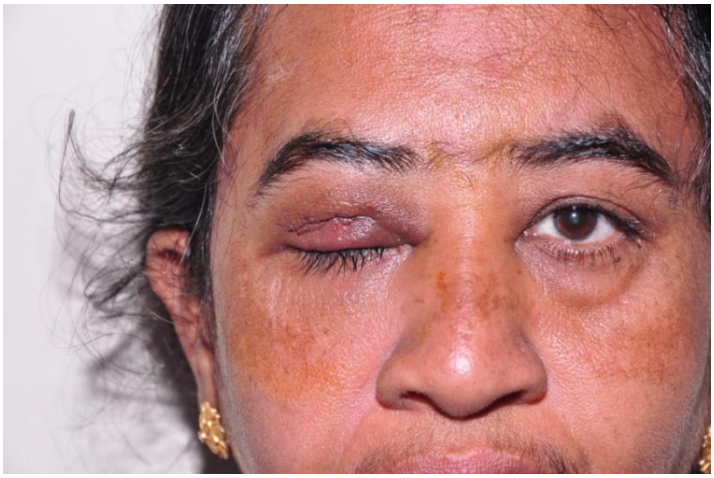
Presented with C/O. Swelling right eye

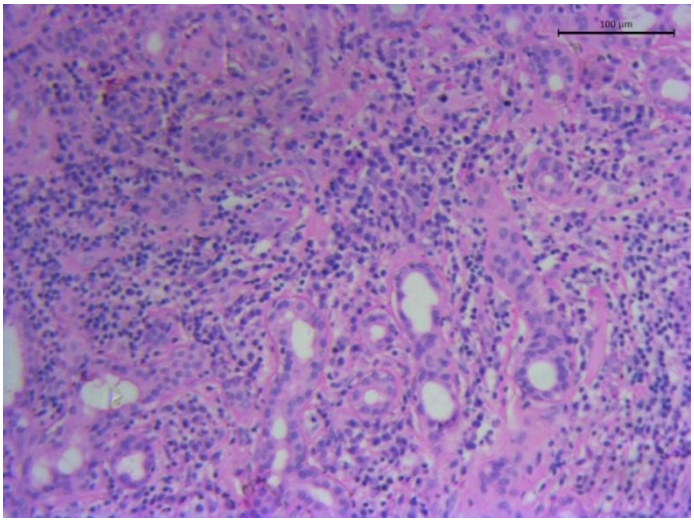
O/E. → 'S' Shaped lid deformity (+)



CT scan shows evidence of heterogeneously enhancing nodular lesion involving right side orbit along the supero lateral quadrant blends with lacrimal gland.

POST OP





HPE:

Structure of lacrimal gland tissue with dense hyalinised collagen and vascular proliferation. Perivascular lymphocytic infiltration with interspersed eosinophils are seen suggestive of Chronic sclerosing Dacryoadenitis.

Immunohistochemistry:

CD3 and CD20: Diffuse positive reactivity

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PROFORMA

Orbital PseudoTumor – Proforma

Name: **Age:** **Sex:**

MR No: **Address:**

Presenting C/O

	RE	LE	
Defective vision			
a.Onset			(1- Sudden 2- Insidiuous 3- Subacute)
b.Progression			(1-Gradual , 2- Rapid)
Double vision			
a.Binocular/Uniocular			
b.Horizontal/Vertical			
Drooping of eyelid			
Protrusion of eyeball			
Pain			
Redness			
Localised/Diffused			
Watering			
Discharge			
Nasal discharge/bleeding			
Trauma			
Fever			
Mass			
a) Onset			(1- Sudden 2- Insidiuous 3- Subacute)
b) Progression			(1-Gradual , 2- Rapid)
c) Associated with Pain			
d) Associated with trauma			
e) Intermittent remission			
f) Associated with Systematic illness			

EOM Restriction | | |

Past History ☐ None ☐ DM

HT ☐ Cardiac ☐ Asthmatic

Allergy ☐ Others,if others specify

If YES Treated OR Not

Family History:
YES/NO

If Yes Specify

General Examination

□Anaemic□ Cyanosis □Icteric□ General lymphadenopathy

□Pedal edema□ No Signs

BP ____ mm Hg Pulse Rate ___ per min Temperature ___(Febrile/Afebrile)

Weight ___ kgs

Cardiovascular System (1-Normal ,2-Abnormal)

If abnormal, specify _____

Respiratory System (1-Normal ,2-Abnormal)

If abnormal, specify _____

Abdomen (1-Normal ,2-Abnormal)

If abnormal, specify _____

Examination of CNS (1-Normal ,2-Abnormal)

If abnormal, specify _____

ENT Examination

Ear _____ (1- Normal, 2-Abnormal) If abnormal, specify

Nose _____ (1- Normal, 2-Abnormal) If abnormal, specify

Throat _____ (1- Normal, 2-Abnormal) If abnormal, specify

Examination of Paranasal sinuses

Sinus Tenderness _____ (1-Absent, 2- Present)

Crepitus _____ (1-Absent, 2- Present)

Ocular Examination

Visual Acuity

	RE	LE
UCVA		
BCBA		
IOP		

Anterior Segment:

	RE	**LE**	
Lid Ptosis			(1-Absent 2-Present)
Lid Lag			(1-Absent 2-Present)
Lagophthalmous			(1-Absent 2-Present)
Lid Edema			(1-Absent 2-Present) If present localized/diffused UpperLid/Lowerlid
			(1-Normal 2-Congested 3-Chemosis)
Conjunctiva			(1-Diffuse 2-Ciliary)
If congested specify type Of congestion			(1-Clear 2-Hazy 3-Could not be evaluated)
Cornea			(1-Edema 2-Opacity 3-Degeneration 4-Dystrophy 5-Ulcer,6-Infiltrate,7-Others
If Hazy, specify			

Anterior Chamber:

	RE	LE	
(1)			(1-Normal Depth 2-Shallow,3-Flat,4-Details not made out
			(1-NO reaction,2-Reaction,3-Exydates,4-Hyphaema,5-Hypopyon,6-Others)
(2)			

If others specify

Iris

RE	LE	
		(1-Normal,2-Abnormal,3-Could not be specified)

If abnormal specify

Pupils

RE	LE	
		(1-Normal,2-RAPD,3-AAPD,4-EPD,5-Could not be evaluated,6-Others)

If others specify

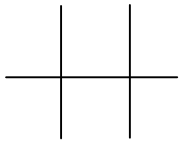
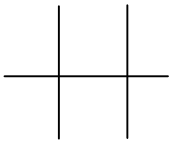
RE	LE	

Lens

		(1-Clear,2-Cataractous,3-Pseudophakia,4-Aphakia,5-Could not be evaluated,6-Others)

If others specify

ExtraOcular movements (1-Full,2 –Limited,3-Frozen globe)

Mass/Proptosis

	RE	**LE**	
Orbital Mass			(1-Not Palpable ,2- Palpable
PreOrbital Rim			(1-Continuous 2-Interrupted)
Globe displacement			(1-Absent , 2-Present)
If present			(1-Axial,2-Eccentric)
Hertel's			Base:
Globe retropulsion			(1-Absent 2-Present)
Thrill/Pulsations			
Valsalva			(1-Negative 2-Positive)

RE	**LE**	

Posterior

		(1-Normal,2-Abnormal ,3-Details not made out)	**Segment**

If others specify

Color vision _____ (1-Normal, 2-Defective) If defective specify

_____

Central Fields _____ (1-Normal, 2-Defective) If defective specify

_____

Hess Charting _____ (1-Normal, 2-Defective) If defective specify

_____

Diplopia Charting _____ (1-Normal, 2-Defective) If defective specify

_____

Investigations

Hematological CBC _____

TFT _____

If Others specify

_____

CT Scan/MRI

_____

USG
BSCAN_____
___

X Ray Orbit/Optic Foramen
_____

Histopathology Done/Not .If done specify
_____

Diagnosis

Type of tumor
_____

Classification
_____

Management

Medical
_____
_____
_____

Surgical _____

Chemotherapy /RT _____

Referral_____

Follow Up - First

	RE	LE	
Visual Acuity			
Signs and Symptoms			1-Improved,2-Deteriorated ,3-Remained Same
Ocular examination			
Extraocular movements			
Investigations (Not Done/Done) If Done Specify _____ _____ If Abnormal Specify _____			

Treatment (Changed/Not)	

Follow Up - Second

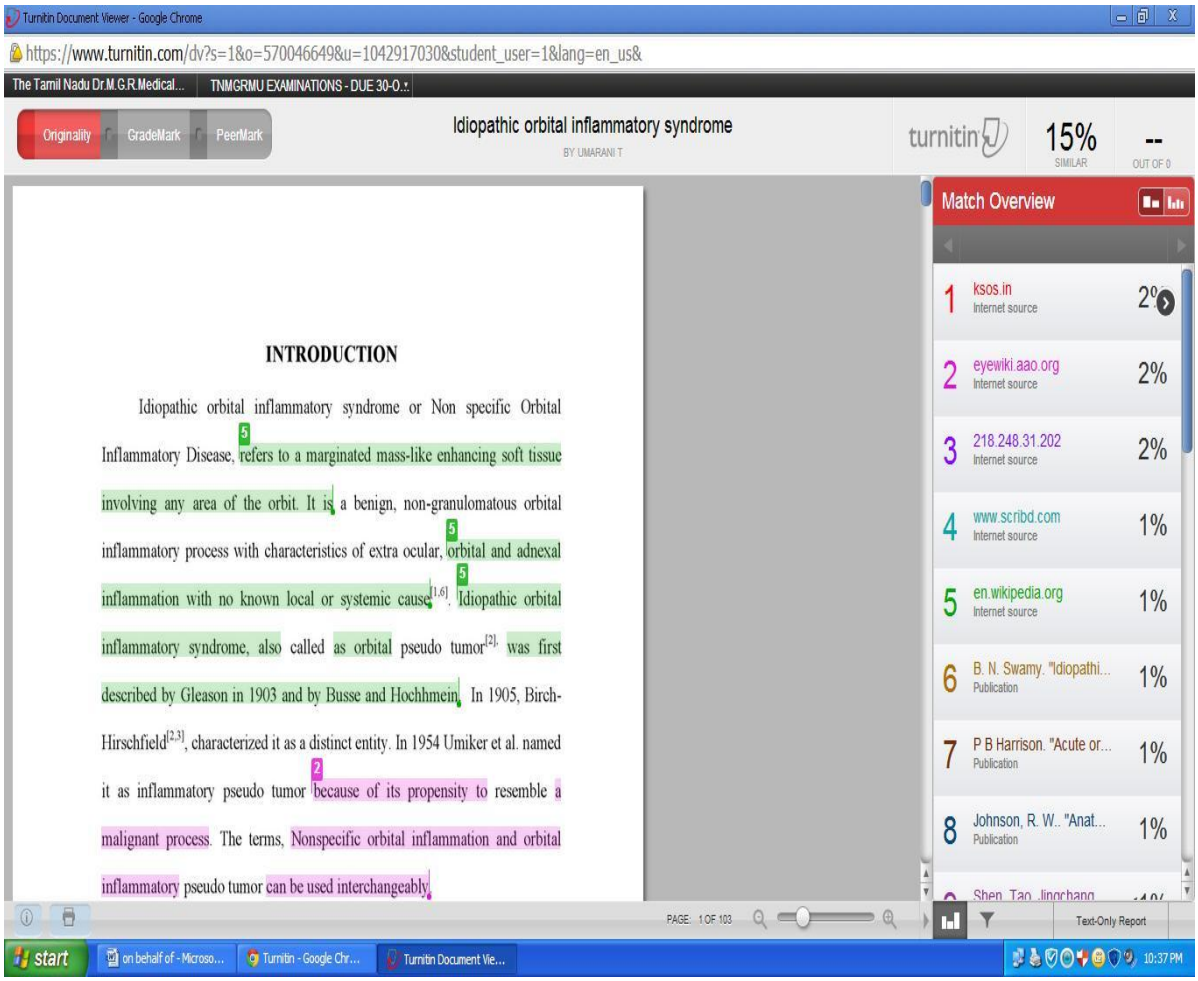
	RE	LE	
Visual Acuity			
Signs and Symptoms			1-Improved,2- Deteriorated ,3- Remained Same
Ocular examination			
Extraocular movements			
Investigations (Not Done/Done) If Done Specify _____ _____ If Abnormal Specify _____			
Treatment (Changed/Not)			

Follow Up - Third

	RE	LE	
Visual Acuity			
Signs and Symptoms			1-Improved,2- Deteriorated ,3- Remained Same
Ocular examination			
Extraocular movements			
Investigations (Not Done/Done) If Done Specify _____ _____ If Abnormal Specify _____			
Treatment (Changed/Not)			

ABBREVIATION

NSOI	-	Non Specific Orbital Inflammation
IOID	-	Idiopathic Orbital Inflammatory Disease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INTRODUCTION
Idiopathic orbital inflammatory syndrome or Non specific Orbital Inflammatory Disease, refers to a marginated mass-like enhancing soft tissue involving any area of the orbit. It is a benign, non-granulomatous orbital inflammatory process with characteristics of extra ocular, orbital and adnexal inflammation with no known local or systemic cause[1,6]. Idiopathic orbital inflammatory syndrome, also called as orbital pseudo tumor[2], was first described by Gleason in 1903 and by Busse and Hochhmein. In 1905, Birch-Hirschfield[2,3], characterized it as a distinct entity. In 1954 Umiker et al. named it as inflammatory pseudo tumor because of its propensity to resemble a malignant process. The terms, Nonspecific orbital inflammation and orbital inflammatory pseudo tumor can be used interchangeably.
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Submission ID:	**570046649**

INTRODUCTION

Idiopathic orbital inflammatory syndrome or Non -specific Orbital Inflammatory Disease, refers to a non-painful mass like enhancing soft tissue involving any area of the orbit. It is a benign, non-granulomatous orbital inflammatory process with characteristics of extra ocular, orbital and adnexal inflammation with no known local or systemic cause[1-3]. Idiopathic orbital inflammatory syndrome, also called as orbital pseudo tumor[4-7] was first described by Ghosten in 1903 and by Reese and Hershbaum. In 1905, Birch-Hirschfeld[8-9], characterized it as a distinct entity. In 1950 Dunken et al. named it as inflammatory pseudo tumor because of its propensity to resemble a malignant process. The terms, Nonspecific orbital inflammation and orbital inflammatory pseudo tumor can be used interchangeably.

Orbital inflammatory pseudo tumor accounts for 10 % of orbital tumors[10]. It most commonly occurs in the age group of 30 to 50 years, but it can also occur in children. There is no sex predilection. Though some bilateral involvement is possible in children, it is mostly unilateral. NSOI can affect the localized or diffuse. For the case of localized inflammation may affect the extra ocular muscles (termed as orbital myositis), lacrimal gland (termed

1

Sno	Name	Age	Sex	MRNo	Place	Weight	Study eye	Onset_def	Ocular_dou	Droop	Protrusion	Pain	Redness	Redness specify	Watering	Discharge	Nasal	Trauma	Fever	Mass/swelling	EOM	Systemic illness	Systemic illness specify	Past ocular history	Family history	General exam	Cardiovacular	Respiratory	Abdomen	CNS exam	ENT & PNS	UCVA RE	UCVA LE	BCVA RE	BCVA LE	IOP RE	IOP LE	Ptosis	Lid Lag	Lagophthalmous	Lid edema	Lid edema specify	Conjunctiva	Conjunctiva specify	Cornea	Cornea specify	ACD	AC reaction	Iris	Iris specify	Pupil	Pupil specify	Lens	Lens specify	Extraocular	Extraocular specify	Orbital mass
1	suresh george benjamin	44	M	3083482	Kerala	82	LE	1	1	1	1	2	1		1	1	1	1	1	1	2	1		1	1	1	1	1	1	1	1	6/6	6/6			13	17	1	1	1	1		1		1		1	1	1		1		1		2	FULL MOVEMENT RESTRICTION	1
2	Nithya	22	F	3093984	Madurai	50	LE	1	2	1	1	2	2	1	1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			20	14	1	1	1	1		2	1	1		1	1	1		1		1		2	Medial restriction	1
3	pethu raja	38	M	3359313	Kodaikkanal	70	LE	1	1	1	1	2	1		1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/6	6/6			14	18	1	1	1	2	upper lid	1		1		1	1	1		1		1		2	Superior ,lateral, Inferior Restriction	1
4	Muruganandham	26	M	3374940	Trichy	68	RE	1	1	1	1	2	1		1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/6	6/6			19	15	1	1	1	1		2	1	1		1	1	1		1		1		1		2
5	karthiga	36	F	3408257	Madurai	66	LE	1	2	1	1	1	2		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			18	20	1	1	1	1		2	nasal congestion	1		1	1	1		1		1		2	Medial and lateral restriction	1
6	palani kumar	17	M	3410808	Sivakasi	44	LE	1	1	2	1	2	1		1	1	1	1	1	1	2	1		1	1	1	1	1	1	1	1	6/6	6/6			18	16	2	1	1	2	Upper lid	2	2temporal congest	1		1		1		1		1		2	,superior and lateral restriction	1
7	kowsalya	15	F	3478168	Dindigul	50	?BE	1	1	1	2(BE)	2(BE)	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			15	16	1	1	1	1		1		1		1	1	1		1		1		2RE) 1 (LE)	RE MEDIAL RESTRICTION	BE RE > LE 2
8	muthammal	40	F	3491781	Kodaikkanal	68	RE	1	1	1	1	2	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			16	14	1	1	1	1		2	1	1		1	1	1		1		1		2	superior and lateral restriction	1
9	Rani	50	F	3646989	madurai	73	LE	1	1	1	1	2	1		1	1	1	1	1	2		2	HT	1	1	1	1	1	1	1	1	6/6	6/6			14	16	2	1	1	2	upper lid	1		1		1	1	1		1		1		2	lateral restriction	1
10	senthil kumar	31	M	3656394	Madurai	65	RE	1	1	1	2	2	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/12	6/6	6/6	6/6	10	12	1	1	1	1		2	2	1		1	1	1		1		1		2	superior and lateral restriction	1
11	Abdul Rasheed	7	M	3725059	Kerala	25	RE	1	1	1	1	1	1		1	1	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	6/6	6/6			12	13	2	1	1	1		1		1		1	1	1		1		1		1		2
12	Gayathiri devi	18	F	3726918	Theni	63	RE	1	1	1	1	2	1		1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/6	6/6			13	13	2	1	1	2	UPPER lid fullnes	1		1		1	1	1		1		1		2	superior restriction	2
13	Thilageswari	47	F	3807738	Madurai	75	RE	1	1	2	1	1	1		1	1	1	1	1	1	1	2	DIABETES	1	1	1	1	1	1	1	1	6/6	6/6			13	14	2	1	1	2	upper lid	1		1		1	1	1		1		1		2	superior restriction	1
14	Muniyandi	36	M	3817629	madurai	78	RE	1	1	1	1	2	1		2	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/24	6/6	6/9	6/6	2o	18	1	1	1	1		1		1		1	1	1		1		1		1		2

15	Boominathan	26	M	3881614	Ramanathapuram	60	RE	1	1	1	1	2	2	2	1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/24	6/6	6/6	6/6	18	16	1	1	1	2	Both lid	2	2	1		1	1	1		1		1		2	all muscles restriction	1
16	uppammal	40	F	3906067	Pudukottai	56	LE	1	1	1	1	2	2	1	1	1	1	1	1	1	2	2	DIABETES	1	1	1	1	1	1	1	1	2/60	6/9	2/60	6/6	11	16	1	1	1	1		1		1		1	1	1		1		1		1	xt	1
17	vimala	49	F	3915390	Kerala	65	LE	1	1	1	1	1	2	1	2	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/18	6/18	6/6	6/6	18	13	1	1	1	2	lower lid	2	1	1		1	1	1		1		1		1		2
18	Brindhu	48	F	3935045	Kerala	76	RE	1	2	1	2	2	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			12	12	1	1	1	1		1		1		1	1	1		1		1		2	Medial and Inferior restriction	1
19	shankar	26	M	3947035	Krishnagiri	54	LE	2	1	1	2	2	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/36	6/6	6/24	17	13	1	1	1	1		1		1		1	1	1		1		1		1		1
20	Geetha P.Nair	48	F	3972462	Kollam	72	RE	2	1	1	2	2	1		2	1	1	1	1	1	1	2	HT	1	1	1	1	1	1	1	1	5/60	6/6	6/24	6/6	20	16	1	1	1	1		1		1		1	1	1		2	RAPD	1		1		2
21	Mariammal	40	f	3993963	Madurai	60	RE	1	1	1	1	2	1		1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/36	6/6	6/18	6/6	16	18	1	1	1	2	upper lid and lower lid	1		1		1	1	1		1		1		1		1
22	Jeganathan	48	M	4023072	Dindigul	72	RE	1	1	1	1	2	1		1	1	1	1	1	2	1	2	Diabetic/Cardiac/HyperTension	1	1	1	1	1	1	1	1	6/9	6/9	6/6	6/6	20	19	1	1	1	2	Both Lid	1		1		1	1	1		1		1		2	Superior and lateral restriction	1
23	Sosamma George	62	F	4029614	Kerala	86	RE	1	1	2	1	1	1		1	1	1	1	1	2	1	1		1	1	1	1	1	1	1	1	6/9	6/9	6/6	6/6	15	16	2	1	1	2	Upper Lid Edema	2	temporal congest	1		1	1	1		1		1		1		1
24	Sangeetha	17	F	4071232	Krishnagiri	67	RE	1	2	1	1	2	1		1	1	1	1	1	1	1	1		1	1	1	1	1	1	1	1	6/6	6/6			19	15	1	1	1	1		2	temporal congest	1		1	1	1		1		1		2	Superior and lateral restriction	1

Pre-orbital rim	Globe displacement	Globe displace specify	Globe retropulsion	Thrill	Valsalva	PS	PS specify	Colour vision	Colour vision specify	CF	CF specify	TFT	Other Investigations	CT Scn/MRI	USG Bscan	Histopathology	Histopathology specify	Diagnosis and Classification	Medical	Surgical	Chemotherapy/Radiotherapy	Followup	BCVA RE 1v	BCVA LE 1v	Sign sym 1v	ocular 1v	Extraocular 1v	investigation 1v	Investigation specify 1v	Treatment 1v	BCVA RE 2v	BCVA LE 2v	Sign sym 2v	ocular 2v	Extraocular 2v	investigation 2v	Investigation specify 2v	Treatment 2v	Complication	Recurrence seen	BCVA	Presenting complaint	Anterior Segment Eamination	Investigation	Treatment
1	1		1	1	1	1		1		1		1	RA-Negative ACE-Nega	Enlarged musle BELLY and tendon of IR ,LR & SR muscle		2	inflammatory myositis	Myositis	T.wysolone 10 mg od x 15 days , 5 mg od x 15 days.	Incision biopsy			6/6	6/6	1	(CSCR) REFRACTORY	1			T.METHOTREXATE 10 MG /WEEK X 1 months.	6/6	6/6	1	Resolved CSCR	1			T.Methotrexate 10 mg /week x 3 months	Steroid Induced CSCR	Nil						
1	1		1	1	1	1		1		1		1		myositis form of pseudo tumor with involvement	Intra conal cystic lesion	1		Myositis	T.wysolone 10 mg od x 15 days , 5 mg od x 15 days.				6/6	6/6	1	1	1	1		t. wysolone 10 mg od 10 days										Nil						
1	2	1	1	1	1	1		1		1		1		Thickening and enhancement of belly and tenden of SR		1		myositis	T.wysolone 20 mg od and 10 mg od each 2 weeks				6/6	6/6	1	1	1	1		.t.wysolone20 mg od 15days and 10 mg od 15 days.										Nil						
1	1		1	1	1	1		1		1		1	RA-Negative ACE-Nega	PSEUDOTUMOR involving right lacrimal gland		1		lacrimal gland adenitis	T.wysolone20 mg od x 15 days and T.wysolone 10 mg od x 15				6/6	6/6	1	1	1			t.wysolone 5 mg od x 1 month	lost follow up									Nil						
1	2	2	1	1	1	1		1		1		1		Diffuse Thickening of EOM with involvement of MR SR-		1		Myositis	Inj.Dexa 2cc im OD T.wysolone 10 mg bd x 15 days				6/6	6/6	1	1	1			t.wysolone 5 mg bd x 1 month and 5 mg od x 1month										Nil						
1	2	1	1	1	1	1		1		1		1		myositis form of pseudo tumor with involvement		1		Myositis	INJ.Dexa 2 cc 3 days. T.wysolone 20 mg od 15 days and 10 mg od				6/6	6/6	1	1	1	1		t.wysolone 10 mg od on altrnate days										Nil						
1	2(BE)	1	1	1	1	1		1		1		1		Thickening and enhancement of belly and tenden of		1		Diffuse	INJ.Dexa 2 cc 3 days. T.wysolone 10 mg BD 15 days and 10 mg od				6/6	6/6	1	1	1	1		t.wysolone 10 mg od on altrnate days	6/6	6/6	1	1	1		T.wysolone 5 mg od x 1 m0nth	nil	Nil						
1	2	1	1	1	1	1		1		1		1		pseudo tumor involving LR IR & SR muscle &	Moderate EOM thickness	2	sclerosing inflammatory pseudo	? DIFFUSE	T.wysolone 20 mg od and 10 mg od 1 week each	incision biopsy			6/6	6/6	1	1	1			t.wysolone 5 mg od 1 month & 5 mg on alternate										Nil						
1	1		1	1	1	1		1		1		1	ra-Negative ACE-Nega	MYOSITIS FORM OF PSEUDOTUMOUR		1		MYOSITIS	Inj.IV MP 500 mg 3 days. T.wysolone 60 mg ,50 mg ,40 mg each 1				6/6	6/6	1	1	1	1		t.wysolone 30mg 20 mg 10 mg od 1 week each	6/6	6/6	1	1	1		t.wysolone 5 mg od x 1 month		Present	6/6	Pain and Redness	Upper lid edema ,conjunctival		T.wysolene 10 mg bd 15 days,10 mg od 15 days
1	2	1	1	1	1	1		1		1		1	RA-Negative ACE-Nega	thiickened MR muscle and ON lateral displacement	IR and MR enlarged.Intraconal	2	Mature adipocytes and vessels with	Myositis	T.Wysolone 10 mg od x 15 days , 5 mg od x 15 days.	Medial Orbitotomy			6/6	6/6	3	3	3	USG B SCAN	persisting MR &IR lesion RA -	inj.MP 500 mg od x 3 days.t.wysolone 20 mg od15 days	6/6	6/6	1	1	1		T.wysolone 10 mgbd x 1 week and 10 mg od x 15 days	Steroid Induced diabetes	present	6-Jun	Double vision	T. conjunctival congestion		T.wysolone 5 mg od x 15 days .Inj.dexa 2 cc x 3 days.
1	1		1	1	1	1		1		1		1		Lacrimal gland adenitis		2	Idiopathic sclerosing orbital inflamm	lacrimal gland adenitis		Mass incision biopsy			6/6	6/6	1	1	1	1		T.Wysolon 10mg OD 15days 5 mg OD 15 days										Nil						
1	2	2	1	1	1	1		1		1		1		Diffuse enlargement of Lacrimal gland with calcification.		2	Inflammatory pseudo tumor with	DACRYOADENITIS	INJ.IV MP 500 mg pulse dose. T.wysolone 10 mg bdx 1	Incisional biopsy			6/6	6/6	1	1	1			t.wysolone 10 mg bd x 1 month	6/6	6/6	1	1	1		t.wysolone 5 mg bd x 1month		Nil						
1	1		1	1	1	1		1	1	1		1		Pseudotumour involving lacrimal gland.Dacryoadenitis		2	chronic sclerosing dacryoadenitis	Dacryoadenitis		superolateral orbitotomy with mass			6/6	6/6	1	1	1	1		T.Wysolon 10mg bd 1 wk 10 mg od 1 wk	Missed follow up									Nil						
1	2	1	1	1	1	1		1		1		1		inflammatory etiology/ pseudotumor		1		?DIFFUSE	T.Wysolon 10mg bd 1 wk 10 mg od 1 wk				lost follow up																			Nil						

1	1		1	1	1	1		1		1		1		Myositis form of pseudotumor	Medial intraconal cystic lesion	1		Myositis	IV Methyl pred 500 mg x 3 days. T.wysolone 30mg od x 10				6/6	6/6	3	3	3	1		inj,dexa 2cc im 3 days..T.Wysolon 10mg bd 1 month	6/6	6/6	1	1	1			t.wysolone 5 mg bd x 1month and5 mg od x 1 month		Nil					
1	1		1	1	1	1		1		1		1		Thickening and enhancement ofleft MR and IR		1		Myositis	Inj.dexa stat T.wysolone 10 mg bd x 15 days				2/60	6/6	3	1	1	1	RE Amblyopic	ttab.wysolone 10 mg bd x 15 days	Missed follow up									Nil					
1	1		1	1	1	1		1		1		1		preseptal soft tissue swelling with edematous IR and IO		1		Anterior with myositis .	Inj.dexa 2cc 3days, T.wysolone 10 mg bd x 15 days.				6/6	6/6	1	1	1	1		ttab.wysolone 10 mg od x 30 days	nil									Nil					
1	2	1	1	1	1	1		1		1		1		Soft tissue lesion in inferomedial quadrant with		1		Myositis	Inj.IV Methyl pred 500 mg od x 3 days. T.wysolone 10 mg bd x				6/6	6/6	1	1	1	1		1.wysolone 5 mg od x 1month										Nil					
1	2	1	1	1	1	1		1		1		1		evidence of thickening of medial rectus & IR WITH SOFT TISSUE		1		DIFFUSE	Inj.methyl pred 500 mg bd x 5 days. T.wysolone10 mgbdx 15				6/6	6/6	3	3	1	1		Inj.IV Methyl pred 500 mg pulse dose,t.wysolon 10 mg bd x	6/6	6/6	1	1	1			t.wysolone 5 mg bd x 15 days and t.wysolone od 5 mg x 15 days		Nil					
1	2	1	2	1	1	2	pale disc	2	not able to read all	2	not able to seen	1		thickened posterior tendon of right lateral rectus,SR , IR		1		Diffuse	Inj.IV .Methyl pred 1 g od pulse dose T.wysolone 10 mg bd x				6/12	6/6	3	3	3	1		Inj.IV methyl pred 500mg pulse doseT.wysolone 10mg bd x	6/6	6/6	1	1	1			T.wysolone 10 mg od x 15 days and t.wysolone od 5 mg x 15 days		Nil					
1	1		1	1	1	1		1		1		1		enhancing soft tissue lesion involving preseptal		1		Anterior	Inj.dexa 2cc im x 3 days. T.wysolone 10 mg od 15 days.			missed follow up																		Nil					
1	1		1	1	1	1		1		1		1		RE mysotis form of pseudo tumor	LR 6.41 mm thickness	1		Myositis	IVMP 500 mg 3 days. T.wysolone 10mg BD 1 month				6/6	6/6	1	1	1	USG B SCAN	Resolving Myositis	t.wysolone 10 mg od x1 month	6/6	6/6	1	1	1			T.wysolone 5 mg od x 1 month and T.wysolone 5 mg od		Nil					
1	1		1	1	1	1		1		1		1		RE granulamatous inflammation of Lacrimal	SR +LPS+ IR Muscles more thickene	1		Diffuse	T.wysolone 10 mg bd x 15 days				6/6	6/6	1	1	3	USG B SCAN	Resolving EOM Thickness	t.wysolone 10 mg od 15 days 5 mg od 15 days ,,	nil									Nil					
1	1		1	1	1	1		1		1		1	RA-Negative ACE-Nega	RE LR & MR muscle and tendon diffusely thickening		1		Myositis	Inj.IV MP 500 mg 3 days T.wysolone 10 mg bdx 1 month				6/6	6/6	3	3	3	RA-Negative ,SR-ACE -Negative ,ANCA-		Inj.IV methyl pred 500 mg pulse dose.T.wysolone 2o mg bd	6/6	6/6	3	3	3			T.wysolone 2o mg od x 1 month and Inj.IV methyl pred 5oo mg		Nil					