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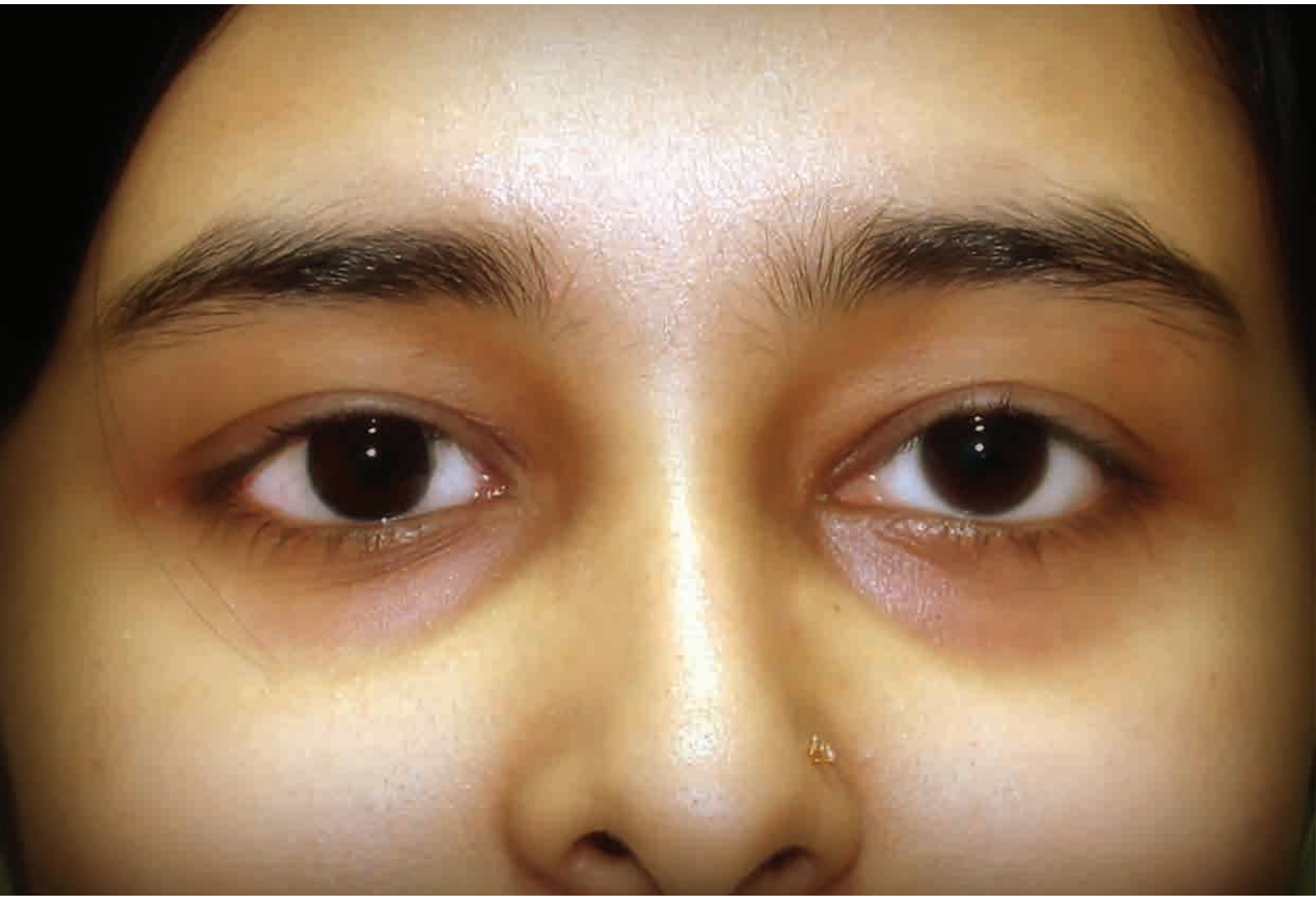

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Sankara Nethralaya – The Temple of the Eye.

It was in 1976 when addressing a group of doctors, His Holiness Sri Jayendra Saraswathi, the Sankaracharya of the Kanchi Kamakoti Peetam spoke of the need to create a hospital with a missionary spirit. His words marked the beginning of a long journey to do God's own work. On the command of His Holiness, **Dr. Sengamedu Srinivasa Badrinath**, along with a group of philanthropists founded a charitable not-for-profit eye hospital.

Sankara Nethralaya today has grown into a super specialty institution for ophthalmic care and receives patients from all over the country and abroad. It has gained international excellence and is acclaimed for its quality care and compassion. The Sankara Nethralaya family today has over 1400 individuals with one vision – to propagate the Nethralaya philosophy; the place of our work is an Alaya and Work will be our worship, which we shall do with sincerity, dedication and utmost love with a missionary spirit.

Contents

Inquiries or comments may be mailed to the editor at insighteditor@snmail.org

Cover Image: "Catch Me If You Can"
Loss of an eye can be psychologically devastating for anyone. Though the patients learn to cope with the functional deficit, the cosmetic blemish can encumber them for life. This is particularly tragic in young people. However, a providential encounter with an ocularist is all that they need. A custom–made prosthesis can transform their lives by enabling them to gain the confidence to go out back in the society knowing that no one can tell the difference between their good eye and the prosthetic eye.
Can you?
Mr. Ezakial D, Ocularist, Ocular Prosthetic Clinic,
Dr. Bipasha Mukherjee, Director, Orbit, Oculoplasty, Reconstructive & Aesthetic Services, Medical research Foundation, Chennai, India.

The Age of Aquarius

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I shall be telling this with a sigh
Somewhere ages and ages hence:
Two roads diverged in a wood, and I–
I took the one less traveled by,
And that has made all the difference.
– Robert Frost

Oculoplasty surgery originated in India more than 2500 years back.[1] Hence, it is ironical that Oculoplasty as a super-specialty has generally been ignored by the Indian ophthalmologists. Aravind eye hospitals, Madurai, started a full term fellowship in Orbit and Oculoplasty as late as 1998. Several institutions have been offering a rotation in Oculoplasty as a part of general Ophthalmology fellowship curriculum, or short-term observerships. However, it takes more than 3–6 months of training to develop the clinical judgment necessary to provide proper care for all eyelid, lacrimal, orbital, facial plastic and cosmetic problems.

In 2014, as Oculoplasty Association of India celebrates its silver jubilee, there are approximately only 200 trained Oculoplasty surgeons in India, and just about 10 fellowships on offering a year (Source: *Indian Journal of Ophthalmology*).

The term "plastic" in plastic surgery is derived from the Greek word "plastikos", which means to mold or shape. There are several specialized components under the spectrum of Oculoplasty surgery such as reconstructive surgery, which involves restoration of an eye that is abnormal due to trauma/accident, tumor or birth defect and cosmetic surgery, which is the esthetic enhancement or reshaping of normal tissue to improve appearance.

India, with 1.27 billion people, is the second most populous country in the world at present. With a projected growth rate of 1.58%, it is all set to take the number one position by 2030. With more than 65% of India's current population <35 years, India is poised on the threshold of a unique demographic phenomenon.

In 2020, the average age of an Indian will be 29 years.[2] It is high time we realize that in near future, an average Indian will not be needing management for "senile" cataracts or "age related" macular degeneration. These two conditions attract the maximum attention and funding, because it addresses concerns of an aging Western populace. While in India, the majority of the population consisting of millions of Gen Y (those born after 1980), also known as the "selfie" generation who thrive on social media networking, will need Ophthalmologists trained in Ophthalmic Plastic and Reconstructive surgery. Better Technology, minimally invasive procedures and greater accessibility will make esthetic procedures, surgical and non-surgical, far more attractive. USA with a population 314 million spent more than $12 billion on cosmetic procedures alone in 2013.[3] Oculofacial esthetics is a multibillion dollar industry. The Ophthalmic community of India is blissfully unaware of this potential as cosmetic dermatologists and plastic surgeons reap the benefits of their complacency.

The fallout of increase in working-age population, consumerism and motorization in India has resulted in worst road traffic accident rates in the world. Tamil Nadu has the dubious distinction of recording the most number of deaths.[4] Lack of traffic discipline combined with a lack of political will to implement "unpopular" safety measures have led to prodigious increase in accidents. This fact also underlines the need for personnel trained in tackling orbital trauma and reconstructive surgery.

This is an exciting time to be an Ophthalmic Plastic and Reconstructive surgeon. There is a tremendous scope and need for trained Oculoplasty surgeons in India. Ophthalmologists should realize the opportunities available to them in the esthetic realm. The challenge lies in clearing the misconceptions about this super-specialty. It is high time that we realize that Oculoplasty is more than just DCR. We need to raise awareness among Ophthalmologists and other clinicians so that patients are referred for optimal treatment by trained specialists.

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Orbital Infections

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Introduction

Orbit is at risk of developing infection either by endogenous or contiguous spread from surrounding structures. Several anatomical factors predispose the orbit for developing infections which include its close proximity to paranasal sinuses, thin bone of lamina papyracea and valveless veins of the midface.

The orbit can be invaded by a variety of microorganisms including bacteria, viruses and fungi, each presenting with different clinical features.

Chandler's classification[1]

Chandler's offered a staging system for complications resulting from sinusitis but now it is used for orbital infections in general. He classified the orbital infections into following five stages.

Stage 1. Preseptal cellulitis: Inflammation localized anterior to orbital septum without orbital signs.

Stage 2: Orbital cellulitis: Infection extending beyond the orbital septum leading to orbital edema and thereby orbital signs.

Stage 3: Sub-periosteal abscess (SPA): Accumulation of pus between the periorbita and the orbital bony walls, which typically appears as dome-shaped elevation on radiographic imaging.

Stage 4: Orbital abscess: Abscess formation within the orbital cavity.

Stage 5: Cavernous sinus thrombosis (CST): With further posterior extension of the infectious process the cavernous sinus gets affected and there is development of CST.

Bacterial infections

Bacterial infections are the most common cause of orbital infections and affect the orbit secondary to sinusitis, trauma, panophthalmitis, dental abscess and rarely endogenous spread.[2–8]

Ethmoidal sinusitis is the most common cause of orbital cellulitis amongst all age groups and results from septic thrombophlebitis of the valve less veins between sinuses and orbit.[1]

Staphylococcus aureus is the most common cause of orbital infections in both pediatric and adult population followed by Streptococcus.[9] *Haemophilus influenzae* used to be the commonest organism causing sinusitis and orbital infections in children, but its incidence has come down drastically following introduction of Hib vaccine.[10] Unlike children adults are more likely to harbor polymicrobial infections owing to the anatomical differences between pediatric and adult paranasal sinuses.

Streptococcus milleri causes multiple orbital abscesses.[11] *Pseudomonas aeruginosa* causes a fulminant orbital infection leading to extensive lid necrosis and formation of lid and orbital abscess. It generally affects neutropenic patients.[12]

Clinical features

Both preseptal and post septal orbital cellulitis present with swelling, signs of inflammation, with or without fever. It is important to distinguish an orbital infection (orbital cellulitis) from a preseptal infection (preseptal cellulitis) as the former is a potentially vision and life threatening condition. Whereas a preseptal infection presents with edema, signs of inflammation and fever, postseptal orbital cellulitis generally leads to diminution of vision, ophthalmoplegia, chemosis, proptosis and optic nerve compromise.[13] Orbital cellulitis can lead to multiple ophthalmic and systemic complications as depicted in Table 2.

Table 1: Differentiating clinical features between preseptal and orbital cellulitis

Clinical features	Preseptal cellulitis	Orbital cellulitis
Vision	Normal	Decreased
Proptosis	Absent	Present
Pupillary involvement	Absent	Variable
Chemosis	Absent	Present
Ophthalmoplegia	Absent	Present

Inflammatory fluid can get collected between the periorbita and orbital wall leading to SPA. A subperiosteal fluid collection can expand rapidly and compromise optic nerve.

Intraorbital abscess occurs when infection breaks through the periorbita into the intraconal and extraconal fat. Visual impairment can occur from direct inflammation of the optic nerve.[13]

Further progression of orbital cellulitis can cause thrombophlebitis and intracranial spread of infection leading to CST. Hallmarks of CST include bilateral cranial neuropathy, headache and neurological impairment.[13]

Investigations

Thin slice contrast enhanced CT scan is the first and foremost investigation to be ordered in any

case of orbital cellulitis. CT scan is preferred over MRI.[14] CT scan is essential for identifying sinusitis and the presence of a subperiosteal or orbital abscess. Use of contrast helps to differentiate an abscess from an inflammatory phlegmon. MRI plays a role in cases of possible CST, intracranial extension or for detecting non radiopaque foreign body.[14]

A complete and differential blood count, blood culture and sensitivity of pus or fluid from sinus drainage are some of the ancillary tests required. Lumbar puncture is generally reserved for patients with signs and symptoms of meningism.[15]

Management

Preseptal cellulitis

Preseptal cellulitis can be treated on an outpatient basis with broad spectrum oral antibiotics and antiinflammatory. A penicillin-clavulinic acid combination is the preferred choice. Hot fomentation helps in reducing the inflammation. Topical antibiotic ointment for local application can be prescribed for any burst hordeolum or abscess. The patient should be under close follow up and should be advised to report to emergency services in case of worsening of symptoms.

Orbital cellulitis

Any patient of orbital cellulitis either adult or a child warrants an urgent admission and needs to be started on broad spectrum intravenous antibiotics covering aerobes and anaerobes. In adults, a parentral cefuroxime 1.5 g or amoxicillin-clavulinic acid combination 1.2 g with metronidazole 500 mg thrice daily can be started on an empirical basis until culture results are available. Patients with culture proven MRSA should be started with vancomycin, cefotaxime and either metronidazole or clindamycin.[16] A nasal decongestant like oxymetazoline helps in drainage of the sinus and even sometimes an SPA.

A consultation with an ENT specialist is desirable in patients with documented sinusitis on CT scan. Unresponsive patients frequently need functional endoscopic sinus surgery. Vision, extra ocular motility and pupillary reaction should be closely monitored.

The use of steroids in orbital cellulitis remains controversial. Fungal infections and immunocompromised patients may further deteriorate if started on steroids. Steroids can be started under antibiotic cover on an individual basis once there is confirmation of a bacterial infection along with the sensitivity.[17]

Subperiosteal Abscess

Patients aged 9 years or younger can be observed on intravenous antibiotics for 24–48 h, under strict visual and pupillary monitoring, with surgery reserved for visual loss and failure to improve. Surgical intervention in SPA is indicated if one or more of the criteria as described in Table 2 are present.

Table 2: Complications of orbital cellulitis and criteria for draining SPA

Orbital cellulitis complications[18,19]	SPA drainage criteria[20]
Permanent blindness	Age more than 9 years
CST	Presence of frontal sinusitis
Optic neuropathy	Non medial location of SPA
Exposure or neurotrophic keratitis	Large SPA
Carotid occlusion	Suspicion of anaerobic subperiosteal infection (e.g. presence of gas within the abscess as visualized on CT scan)
Exudative retinal detachment	Recurrence of SPA after previous drainage
Central retinal artery occlusion	Evidence of chronic sinusitis
Septic embolus	Acute optic nerve or retinal compromise
Meningitis	Infections of dental origin
Brain abscess	

Orbital abscess

Once a diagnosis of orbital abscess is made it should be drained immediately, and adequately.[21] Postoperative monitoring is warranted to detect re-formation of abscess and complications. Patient should be continued on intravenous antibiotics based on the culture and sensitivity of the drained pus.[21]

Cavernous Sinus Thrombosis

Broad spectrum intravenous antibiotics with good CNS penetration for 3–4 weeks is the mainstay of treatment for CST

The infectious nidus should be drained as soon as possible.

Use of anticoagulants as a therapy in CST is controversial with studies citing both beneficial and adverse effects. The rationale behind using anticoagulants is to arrest the thrombotic process in the central venous sinuses thereby reducing bacterial sequestration, cerebral edema and venous infarction and extracranial embolic events like pulmonary embolism.[22]

Corticosteroids may be beneficial for patients of CST by reducing intraorbital congestion and edema. They are absolutely indicated in cases of Addisonian crisis caused due to pituitary ischemia complicated by CST. Corticosteroids also reduce cranial nerve inflammation in patients with cranial dysfunction.[23] However, steroids carry a risk of generalized spread of infection because of

their immunosuppressive effects.[22] The role of reducing intraorbital edema and congestion is also debatable as the cause is more vascular than inflammatory.[22]

Orbital tuberculosis

Orbital tuberculosis is a rare form of extrapulmonary tuberculosis which afflicts the orbit and its surrounding structures either by a hematogenous spread or by a spread from paranasal sinuses. Clinically, it presents as one of the following five characteristics presentations.[24]

1 *Classical periostitis*: It presents as chronic ulceration or a discharging sinus from the periorbital region with or without cold abscess formation and soft tissue swelling.[25]

2 *Orbital soft tissue tuberculoma with cold abscess with no bony destruction*: This group of patients present with pain, proptosis, palpable orbital mass and diplopia. There can be visual loss because of total destruction of globe or direct involvement of the optic nerve by the disease process.[26,27]

3 *Orbital tuberculosis with bony involvement not classified as classical periostitis*: In this group of cases, there is evidence of bony destruction radiologically, erosion, osteomyelitis and rarely bony sclerosis. The roof and lateral wall are most commonly involved.[25]

4 *Orbital tuberculosis secondary to spread from paranasal sinuses.*

5 *Tubercular dacryoadenitis.*[28]

Investigations and diagnosis

The diagnosis of orbital tuberculosis is clinical. Imaging is warranted in all cases of suspected orbital tuberculosis with CT scan being the modality of choice. A systemic workup is mandatory to rule out systemic tuberculosis.

Orbital biopsy is done to confirm the diagnosis. The hallmark histopathological feature is caseating granuolmatous inflammation with Langhans giant cells. Acid fast bacilli are generally demonstrated. Culture for Mycobacterium tuberculosis remains the gold standard for diagnosis. Automated commercial broth culture system utilizing colorometric or radiometric method for mycobacterial growth give results in 1–3 weeks.[29] Polymerase chain reaction is a rapid technique for diagnosing both pulmonary and extrapulmonary tuberculosis with specificity ranging from 93.7 to 100%.[30] However, the sensitivity is low ranging from 27 to 100%.[30]

Management

In general the regimen for orbital tuberculosis is same as that of pulmonary tuberculosis. A 6–9 month course of antitubercular treatment is recommended preferably under the care of an infectious disease specialist. Surgery is needed for excising the fistulous tract, debriding the bone[25] and clearing the paranasal sinuses. Neurosurgical interventions are required in cases of CNS spread.[31]

Orbital fungal infections

Fungal infections of the orbit are rare but can be devastating. They are potentially vision and life threatening. Orbit most commonly gets infected by two classes of fungi namely zygomycota and ascomycota. Both classes have different clinical presentations, risk factors and virulence potential.

Orbital zygomycosis

The orbital fungal infection by this class of fungi is commonly referred to as rhino-orbital-cerebral-zygomycosis (ROCZ), rhino-orbital-cerebral-mucormycosis (ROCM), zygomycosis, phycomycosis, mucormycosis and hyphomycosis.[32] The most common species to cause infection is *Rhizopus oryzae*. It is the cause of roughly 90% of all ROCM infections. Other species of this class known to cause the disease include *Absidia corymbifera, Mucor species, Rhizomucor pusilus, Apophysomyces elegans, Saksenaea vasiformis* and *Cunninghamella bertholletiae*. It is a non-septate filamentous fungus and is generally found in soil, decaying fruit and vegetables animal feces and old bread.[32]

Orbital mucormycosis commonly results from spread from paranasal sinuses, direct traumatic inoculation or rarely from hematogenous spread.

ROCM almost exclusively occurs in immunocompromised hosts (e.g. AIDS, Chemotherapy, steroids, bone marrow transplant). Other risk factors include diabetic ketoacidosis, intravenous drug abuse, iron over load (patients on hemodialysis, hemochromatosis) and desferoxamine therapy.[32]

A patient of orbital mucormycosis presents with all signs and symptoms of orbital cellulitis with or without a characteristic periorbital and/or nasal black eschar. Sudden blindness can result from central retinal artery occlusion, posterior ciliary artery thrombosis or infarction of the intraorbital optic nerve.[33] Infection can spread to brain through cribriform plate, orbital apex and ophthalmic vessels.[34] Alternatively a patient of ROCM can present as orbital apex syndrome without any other signs of orbital cellulitis.[35]

Investigations and diagnosis

The CT scan will demonstrate sinusitis, mucosal thickening, bone necrosis with or without involvement of pterygoid and infratemporal fossa and thrombosis of superior ophthalmic vein. Brain abscess can also be present in cases of intracranial spread.

The KOH mount shows non-septate branched fungal hyphae arranged at right angles.[35] Histopathology shows thrombosing arteritis with vessel walls invaded with fungal hyphae. Veins are relatively spared.[36]

Management

Proper treatment of mucormycosis requires early diagnosis, reversal of the predisposing and systemic factors, wide local excision of the necrotic tissues, establishment of adequate sinus drainage and systemic antifungals.[37] It is always better to involve an infectious disease specialist in the care of the patient.

1 *Amphotericin B*: The dose of intravenous amphotericin B is slowly increased from 0.7–1 mg/kg to a cumulative dose of 2–4 g over a period of weeks to months.[33] Fever, chills, headache, myalgia, anorexia, malaise, anemia, hypokalemia and vomiting are the most common side effects of the drug. Nephrotoxicity is the major dose limiting side effect, hence a constant monitoring of renal function test is required.[38] Lipid formulations of amphotericin B (amphotericin B lipid complex and liposomal amphotericin B) are newer preparations of amphotericin B with increased drug delivery to the site of infection, hence having increased efficacy, with fewer side effects. They are now the drug of choice for ROCM.

2 *Surgery and local debridement*: Wide local excision of the necrotic orbital tissues and sinuses should be carried out to decrease the fungal burden. Infected tissue typically bleeds less because of the ischemia caused by mucor arteritis and thrombophlebitis. Orbital exenteration proves to be life saving in cases of active fungal infection not responding to medical therapy.[39]

3 *Local irrigation and packing with amphotericin B*: Local irrigation and packing of the involved paranasal sinuses, orbit and exenterated socket with amphotericin B (1 mg/ml) can achieve excellent results by increasing the drug delivery to the site of infection.[40]

4 *Hyperbaric oxygen*: Hyperbaric oxygen works by increasing oxygenation, thereby decreasing acidosis and increasing the phagocytic activity. Its role in ROCM is still not clearly defined. The treatment regimen includes hyperbaric oxygen every 12 h with 2 h of 100% oxygen at two atmospheres absolute for 3 days, then daily treatments of 2 h duration. Total number of treatment depends upon the patient's response.[41] It is expensive, cumbersome and not readily available.

5 *Newer modalities*: Posaconazole is an oral anti-fungal agent which can be used as a combination salvage therapy with liposomal amhpotericin B in patients with refractory mucormycosis.[42] Caspofungin is an echinocandin which has shown increased survival in patients of ROCM when given in combination with polyenes.[43] Multiple immune augmentation strategies such as G-CSF, GM-CSF and Interferon-γ have also been proposed.[44]

Orbital aspergillosis

Orbital aspergillosis is an uncommon infection that can affect both immunocompromised and healthy immunocompetent hosts.[37] *Aspergillus fumigatus, Aspergillus flavus* and *Aspergillus niger* are the most common species affecting the orbit with *aspergillus fumigatius* being most common in HIV patients. Similar to mucormycosis the most common mode of infection is spread from adjacent paranasal sinuses.[45]

Total neutrophil count $<1000/mm^3$, T cell defects (e.g. AIDS), defective phagocytosis, hematologic malignancy, steroids and other immunosuppressive agents, diabetes mellitus, prosthetic devices, trauma, excessive environmental exposure (e.g. nearby demolition or restoration of buildings, yardwork, compost heaps), residence in endemic area (e.g. Sudan) and advanced age are some of the risk factors for orbital aspergillosis.[45] Marijuana can be contaminated with aspergillus and is a risk factor of sino-orbital aspergillosis especially in immunocompromised hosts.[46]

Clinically orbital aspergillosis can have one of the following three presentations.[45,47,48]

a Allergic sinusitis with or without orbital involvement.

b Fungal ball or aspergilloma.

c Invasive sino-orbital aspergillosis.

Investigations and diagnosis

CT scan shows heterogenous soft tissue masses with calcification and bony erosion. MRI shows contrast enhancing masses that are hypointense on T1- and T2-weighted images. In contrast, neoplasms and bacterial infections show hyperintensity on T2-weighted images.[37] MRI gives better details in areas such as posterior orbit, optic nerve and cavernous sinus.

Typical aspergillus hyphae are septate with uniform width and dichotomous branching.[49] The fungi can also be identified on hematoxylin-eosin and Gomori methenamine silver stains.[37]

Management

a *Allergic sinusitis with or without orbital involvement:* Patients are usually immunocompetent. Management includes surgical debridement of the involved sinuses followed

by systemic and topical corticosteroids. Systemic antifungals are usually not needed.[50]

b *Fungal ball or aspergilloma:* Surgical debridement of the involved sinus is the treatment of choice. Systemic antifungals are not needed.[50]

c *Invasive sino-orbital aspergillosis in immunocompetent individuals:* Treatment includes wide surgical debridement with aeration of the involved paranasal sinus.[51] Adjunctive systemic antifungal therapy is required and amphotericin B is the drug of choice. Oral itraconazole is an alternative therapy to amphotericin B with response rates almost equal to the latter.[49]

d *Invasive sino-orbital aspergillosis in immunosuppressed individuals*: Reversal of immunosuppression is the primary therapy in such cases. Treatment is otherwise similar to as in immunocompetent patients. Exenteration may be needed in advanced cases. Prognosis is poor if immunosuppression cannot be reversed.[52] Adjuvant local irrigation of amphotericin B with indwelling catheters is recommended to improve survival.[52]

Viral infections

A clinical picture of preseptal orbital cellulitis may occur with viral infections of the eye due to adenovirus, herpes simplex or herpes zoster. With adenovirus there may be a history of exposure to a patient with viral conjunctivitis and patient will have signs and symptoms of conjunctivitis. In herpetic infections vesicular or crusted lesions of the eyelids along with keratouveitis may be present.

Herpes zoster ophthalmicus may also present as orbital cellulitis. The patient presents with proptosis, chemosis and extraocular muscle palsy.[53] It results from ischemia and the resultant inflammatory mass.[54]

Management

Oral acyclovir in a dose of 800 mg five times a day for 7–10 days is the treatment of choice. Oral steroids tapered over a period of 2 weeks can be used in case of extensive inflammation but their role is controversial.

Conclusion

Orbit is at risk of getting infected by a variety of microorganisms because of its close proximity to paranasal sinuses, and spread of midfacial infection by valve less veins. Orbital infections are potentially vision and life threatening and a timely diagnosis and management may help in improving the prognosis.

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Thyroid Eye Disease: A Review

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Introduction

Thyroid eye disease (TED), also called Graves' ophthalmopathy, is an autoimmune disorder, which is an important extra-thyroidal manifestation of autoimmune thyroid diseases such as Graves' disease. TED may be the first noticeable warning sign of systemic thyroid dysfunction. Though mostly associated with hyperthyroidism, it has also been linked to Hashimoto thyroiditis and primary hypothyroidism.

TED results in lymphocytic and mast cell infiltration with edema in orbital connective tissues and collagen deposits in extraocular muscles. The disease is characterized by various clinical features. Inflammatory reactions in the orbit and the periocular tissues cause discomfort, swelling and pain. It can also produce eyelid malpositions and exophthalmos, significantly altering the patient's appearance, distorting self-image and provoking psychological and social problems. The disease is usually benign but can also cause visual handicap in a minority of patients. Clinically significant TED occurs in 10–45% of patients with Graves' disease.[1] It is the most common cause of adult proptosis, most cases affected bilaterally, with unilateral proptosis accounting for 15–28% of cases.

Epidemiology

TED occurs in genetically predisposed individuals, affecting females more commonly than males.[2] It most commonly occurs in the 4th or 5th decades of life. It can follow or precede systemic thyroid disease and can occur in hyperthyroid, euthyroid or hypothyroid states. TED is progressive but self-limiting in 3–5 years of onset. Risk factors for TED include smoking (tobacco chewing), advanced age, radio-active iodine therapy, post ablative hypothyroidism, drugs and stress.

Pathogenesis[3]

A cumulative role of genetic susceptibility, environmental factors and risk factors lead to the development of autoantibodies which result in thyroid gland stimulation causing goiter and hyperthyroidism. Similar factors affect the orbits in some patients producing TED, and produce pretibial myxedema in a minority of patients. The immunologic mechanism of TED is initiated by autoreactive T-lymphocytes directed against antigens that are common to the thyroid and the orbit. In addition, autoantibodies to thyroid stimulating hormone receptor and insulin-like growth factor-1 receptor have been found to play a role in TED. The target of the autoimmune stimuli are the orbital fibroblasts, leading to glycosaminoglycan (GAG) accumulation causing tissue edema, lymphocyte and mast cell infiltration, adipogenesis, fibroblast proliferation and secondary changes in extra-ocular muscles.

Clinical features

Patients with TED present with varied manifestations. Patients with mild disease usually are asymptomatic or have watering, redness, fullness in the eyes which is more prominent in the morning. They become better as the day progresses. Patients with active disease can present with congestion, chemosis, double vision and defective vision. The cause of visual loss in TED is due to exposure keratopathy and dysthyroid optic neuropathy (DON). It is an ocular emergency to prevent irreversible loss of vision.

Lid signs

The most common sign in TAO is eyelid retraction (Fig. 1A), which affects 90–98% of patients.[4,5] Contour of the retracted upper eyelid often shows lateral flare, an appearance that is almost pathognomic for TAO.[6] Lid retraction is multifactorial and is due to increased sympathetic stimulation of Muller's muscle, contraction of the levator muscle and scarring between the lacrimal gland fascia and levator which specifically gives rise to the lateral flare.[7] The excursion of the upper eyelid often lags behind eyeball movement on vertical downward pursuit (lid lag) (Fig. 1B) and remains high. They can have lagophthalmos (Fig. 1C).

Proptosis

Patients with TED have axial non-pulsatile proptosis (exophthalmos) (Fig. 2) secondary to orbital venous congestion, accumulation of GAG and adipogenesis. Exophthalmos can be quantified by the exophthalmometer or radiologically with axial orbital scans.

Restrictive myopathy

The inflammation and secondary GAG deposition previously described causes tissue swelling, which in muscles leads to dysfunction. The inferior rectus is most commonly involved, followed by medial, superior and lateral rectus. Movement is therefore usually worst in elevation or abduction. Once the active inflammatory phase of the disease is over, muscle fibrosis causes restricted motility (Fig. 3).

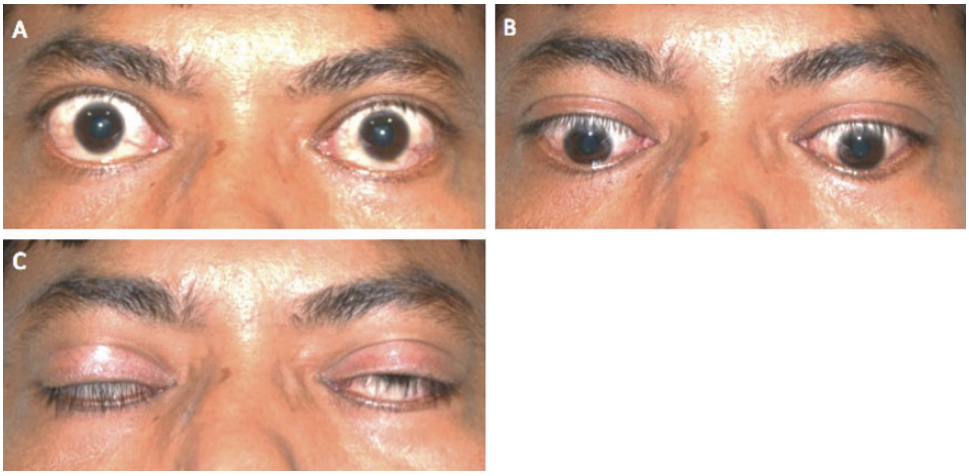

Figure 1: Lid signs of TED. (A) lid retraction and lateral flare, (B) lid lag and (C) lagophthalmos. (Courtesy: Dr. Bipasha Mukherjee.)

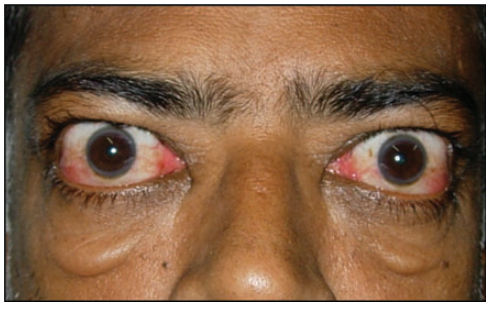

Figure 2: Bilateral proptosis. (Courtesy: Dr. Bipasha Mukherjee.)

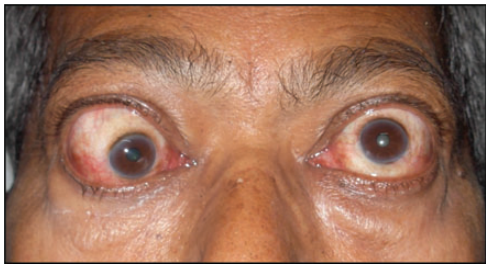

Figure 3: Ocular movement restriction. (Courtesy: Dr. Bipasha Mukherjee.)

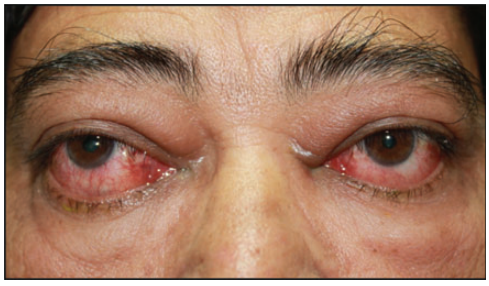

Figure 4: Soft tissue signs—lid edema, chemosis, congestion, caruncular edema. (Courtesy: Dr. Bipasha Mukherjee.)

Soft tissue signs

Patients commonly complain of grittiness, redness, irritation of the eyes and are found to have keratoconjunctivitis sicca (dry eyes) besides congestion, chemosis and eyelid edema (Fig. 4).

Dysthyroid optic neuropathy

DON is usually due to apical compression by enlarged extraocular muscles (Fig. 5). Other mechanisms involved include inflammation, ischemia or mechanical stretching. Signs of optic neuropathy include drop in vision, color vision, visual field and afferent papillary defect or optic disc swelling. Patients with existing diabetes or of Asian origin (due to shallow orbits) have higher chances of developing DON.

Gorman and Bartley's criteria for diagnosis of TED[8]

A clinical diagnosis of TED is made if:

1 There is eyelid retraction in association with objective evidence of thyroid dysfunction or exophthalmos, optic nerve dysfunction or extraocular muscle involvement.

2 If eyelid retraction is absent, then TED is diagnosed only if exophthalmos, optic nerve dysfunction or restrictive extraocular myopathy is associated with thyroid dysfunction and if no other cause for the ophthalmic features is evident.

Investigations

Investigations are aimed at establishing the diagnosis of TED, assessing thyroid function/dysfunction, assessing optic nerve status and assessing disease activity. A baseline thyroid function tests (TFT) during the first visit in all patients suspected to have TED is a must. TFT includes TSH, Free T3 and T4. In autoimmune thyroid disease, several serum antibodies can be measured: TSH-R antibodies, antithyroglobulin antibody, antimicrosomal antibodies/

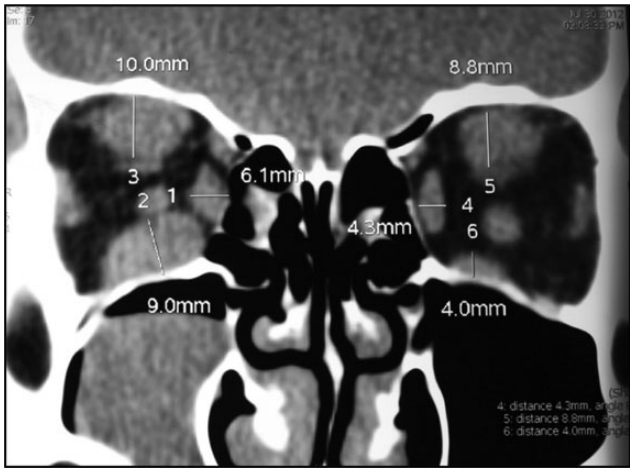

Figure 5: Computed tomography scan of orbits, coronal view, showing apical crowding due to enlarged extraocular muscles. (Courtesy: Dr. Bipasha Mukherjee.)

thyroid peroxidase antibodies (anti-TPO).[9] These tests point to the autoimmune nature of the disease. Ultrasonography to see for disease activity is not recommended as it is interpretor dependent.

Indications for imaging in the form of CT/ MRI include:[6]

- Unilateral involvement
- To rule out other causes of orbital inflammation or orbital mass lesions
- Clinical suspicion of optic nerve involvement
- VISA score of 4 or more
- Pre-operatively, if orbital decompression is planned

The typical characteristics on CT/MRI include enlargement of the extraocular muscles, which has a fusiform configuration, with sharply defined borders and sparing of the tendinous insertions (Fig. 6). The inferior rectus is most commonly involved followed by medial rectus, superior rectus and lateral rectus in that order.[9] Other findings include proptosis and anterior prolapse of the orbital septum due to excessive orbital fat and muscle swelling, lacrimal gland enlargement, severe apical crowding. Visual field assessment should be performed in all patients as a baseline record and in patients suspected to have compressive optic neuropathy.

Classifications of thyroid eye disease

Making decisions of management of patients with TED requires a detailed ophthalmic evaluation, understanding of the natural history of disease, an insight into the impact of the disease on the patient as well as the possible efficacy of proposed therapies.

The severity of thyroid eye disease is graded into broad categories to facilitate decision making with regard to management. The European group of Graves' orbitopathy (EUGOGO)[10] has recommended the following classification of patients with TED:

1 Sight-threatening GO: patients with DON and/ or corneal breakdown.

2 Moderate-to-severe GO: patients without sight-threatening GO, but with one or more of the following: lid retraction >2 mm, exophthalmos >3 mm above normal for race and gender, moderate or severe soft tissue involvement, inconstant or constant diplopia.

3 Mild GO: they usually have one or more of the following: lid retraction (<2 mm), exophthalmos

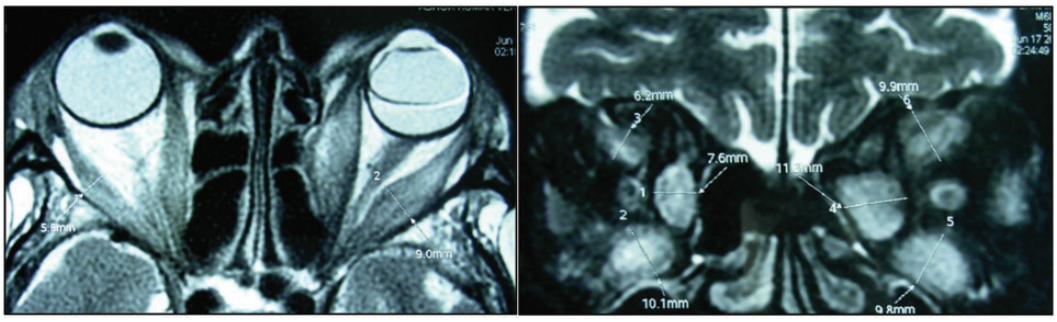

Figure 6: MRI–T_2-weighted axial and coronal images showing fusiform enlargement of extraocular muscles with sparing of tendons. (Courtesy: Dr. Bipasha Mukherjee.)

<3 mm above normal for race and gender, mild soft tissue involvement, transient or no diplopia, corneal exposure responsive to topical lubricants.

The intensity of inflammation is assessed clinically using a list of symptoms and signs that together provide a score, grading the patient's clinical activity of TED. This grades the disease as clinically active or inactive and determines the need and possible efficacy of immunosuppressive therapy.

The clinical activity score (CAS) described by Mourits and the Amsterdam orbitopathy group assigns one point for each of the following: orbital pain at rest, orbital pain with movement, chemosis, caruncular edema, conjunctival injection and eyelid injection. Patients thus scored with a CAS of 3 or more out of 7 are labelled as clinically active.

However, the VISA classification put forth by the International thyroid eye disease society (ITEDS) classifies all clinical manifestations of TED, including vision, clinical activity of inflammation, extraocular muscle restriction and appearance into the VISA form.[11] The clinical activity scoring of VISA modifies the CAS slightly by widening the grade for chemosis and eyelid edema from 0 to 2. VISA inflammatory score of 4 or more out of 10 is considered to be significant active disease.

Management of thyroid eye disease

The multidisciplinary approach in managing TED requires close collaboration between physicians and ophthalmologists for early diagnosis, triaging, referral, symptomatic topical and systemic anti-inflammatory therapies, as well as staged surgical rehabilitation.

General recommendations

Maintenance of euthyroid status is essential in all cases of thyroid eye disease. Additionally, the use of radioactive iodine (RAI) in active TED, or inactive TED with risk factors such as smoking or thyrotropin receptor Ab > 7.5 IU/L should be accompanied by concurrent steroid prophylaxis to prevent progression of the disease.[12]

Cessation of smoking is mandatory in all phases of TED because it worsens the outcome and it is the strongest modifiable risk factor.[13]

All patients with TED would benefit from topical lubricants including eyedrops in day and a gel at night. Additionally, propped up position and taping of lids while sleeping are helpful in reducing periorbital swelling and corneal exposure, respectively.

Immunosuppressants

Patients with moderate-to-severe disease having a significant clinical activity (VISA inflammatory score of 4 or more out of 10) generally require immunosuppression in the form of oral or intravenous steroids. Pulse therapy with intravenous methylprednisolone is more effective and better tolerated than oral steroids.[14] Pulse therapy is generally administered as a 500 mg infusion of i.v. methylprednisolone in 100 mL of physiological saline given weekly for 6 weeks followed by reducing the dose to 250 mg weekly for another 6 weeks. Oral prednisolone is given starting with a dose of 1 mg/kg body weight/day and then tapered over several weeks. Prolonged oral steroid therapy is associated with side effects in a significant proportion of patients. Patients with persistent active disease not responding to or intolerant to steroids may be prescribed steroid sparing agents, such as azathioprine, or newer biologic agents such as rituximab, a chimeric monoclonal anti-CD20 antibody.[15]

Patients with DON are managed by pulse therapy with intravenous methyl prednisolone administered in doses of 1 gm daily for 3 days. In cases with transient or insufficient response, additional EBRT may be administered. In cases with poor response to the above therapy or those intolerant to steroids should be offered urgent orbital decompression to relieve the optic nerve compression.[16]

Radiotherapy

Besides the role of radiotherapy in DON as mentioned above, fractionated EBRT has been shown to be effective in reduction of clinical activity of TED. Radiotherapy may not show benefit for several days to weeks, but its effects are longer lasting. It has been shown to enhance the benefit of oral or i.v. steroid therapy when administered together. It may be administered as primary therapy in patients intolerant to steroids. It is given in a dose of 20 gray in 10 fractions of 2 gray each.[17]

Anti-glaucoma medication

Anti-glaucoma medication may be required to control secondary increased intraocular pressure. Topical alpha-agonists (e.g. brimonidine) have an added benefit of decreasing conjunctival and episcleral congestion.

Surgical intervention

Stability of thyroid status as well quiescence of thyroid eye disease for a period of at least 6–9 months is recommended before considering surgical rehabilitative surgeries. A staged approach with orbital decompression followed by strabismus surgery, correction of lid retraction and then blepharoplasty or other aesthetic surgeries is ideal.[18]

Orbital decompressions are classified into bone removal orbital decompression (BROD) and fat removal orbital decompression (FROD), which can be performed in isolation or in combination. The design of BROD is by the choice of surfaces and

incisions for bone removal, e.g. medial (via transcaruncular, transcutaneous Lynch or endonasal), inferior (transconjunctival forniceal/swinging eyelid, transcutaneous subciliary or transantral) and lateral walls (transcutaneous upper lid crease/swinging eyelid, coronal). FROD involves removal of intraconal and sometimes extraconal orbital fat via a transcutaneous (upper lid crease or lower lid subciliary) or transconjunctival approach.

Strabismus correction in TED is challenging due to the fibrotic nature of EOM involvement. Usually EOM recession instead of resection is performed to correct the limitation of movement rather than the amount of ocular deviation at primary gaze.

The correction of upper lid retraction involves procedures such as mullerotomy, Muller's muscle extirpation, levator aponeurosis disinsertion/recession and levator muscle myotomy with/without the use of adjustable or hangback sutures. Recently full-thickness blepharotomy has gained popularity due to technical simplicity with predictable results. Lower lid retraction is managed with retraction disinsertion with the use of spacer grafts.

Temporary correction of eyelid retraction and lagophthalmos may be done with the use of botulinum toxin A injection as well as injectable fillers.[19]

Prognosis[20]

Upon achieving euthyroid status, up to 90% of eyelid retraction and 30% of restrictive myopathy show improvement. However, proptosis is rarely seen to improve. Clinically evident TED usually runs its active course for 12–24 months before becoming quiescent.

Conclusions

Thyroid eye disease is the most common cause of proptosis or lid retraction in adults and affects 25–50% of all patients with Graves' disease. Smoking is the most important risk factor for developing TED. Thyroid eye disease requires specialist management under the care of an ophthalmologist and an endocrinologist. Appropriate immunosuppression is needed for clinically active disease.

Vigilance is needed for any features possible optic neuropathy including teaching self-assessment of vision and color vision at home. Diagnostic pitfalls include uniocular presentation, a lack of history of Graves' disease and optic neuropathy without obvious proptosis.

Staged surgical rehabilitation is desirable in quiescent disease with stable thyroid status.

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Oculoplasty Crossword

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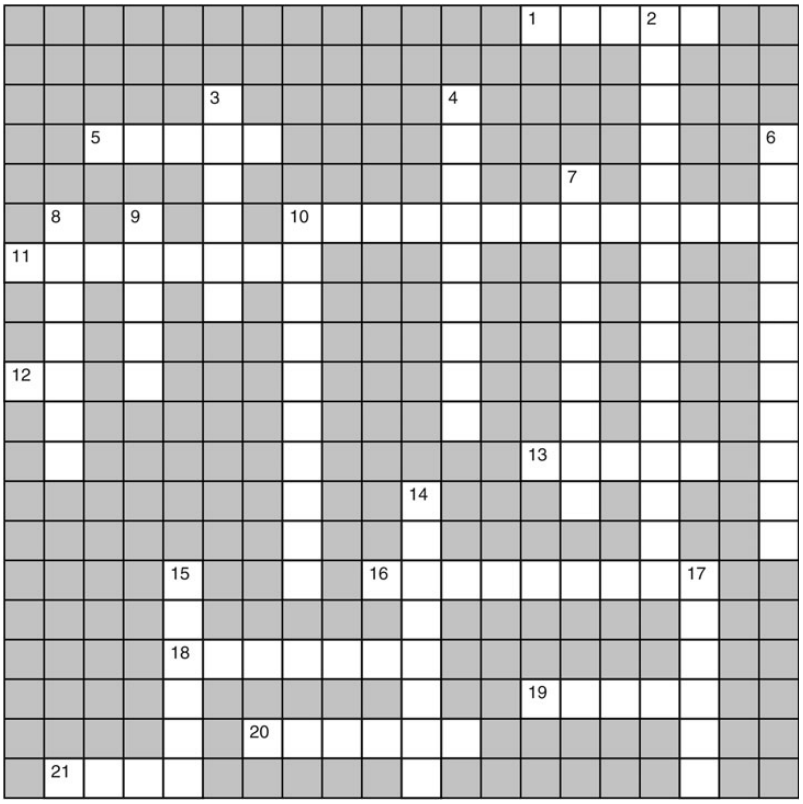

Across

I Ptosis clamp (5)

5 First evisceration(5)

10 Retention of nuclei in stratum corneum (13)

11 City in Japan - congenital muscular dystrophy (8)

12 Muir Torre inheritence (2)

13 Subacute blood is hyperintense on MRI due to _____(5) (abbrev)

16 John Abraham's 'smile' and 'wink' (5,4)

18 I play with snowball and drip candle wax. Wont let you cry! who am I? (7)

19 Tired alphabet - corrects ectropion (5)

20 Multiple trichilemmoma seen in_____ disease (6)

21 Fat suppression in MRI (4)

Down

2 Eyelid tumour which resolves spontaneously (14)

3 TR in MRI stands for time to_____(6)

4 'Hammock' for the eyeball (9)

6 Infratrochlear is a branch of ? (11)

7 Scar Scale - reference style - city in Canada (9)

8 Gripping instrument - fierce pet (7)

9 Measures 'popping out eyes (5)

10 'enclosing' in eyelid tumour (10)

14 Classify orbital infections (one of the 'Friends') (8)

15 Lacrimal canalicular trephine (6)

17 Giant cells in xanthogranuloma (6)

Evaluation of a Case of Ptosis

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Introduction

Blepharoptosis, from Ptosis (Greek: falling down), refers to drooping of upper eyelid in primary gaze. Apart from a cosmetic concern ptosis may lead to amblyopia, visual field defects, and brow ache due to chronic use of frontalis muscle.

Classification

1 Congenital ptosis
   a With normal superior rectus function
   b With superior rectus weakness
   c With blepharophimosis syndrome
   d Synkinetic ptosis
      i Marcus Gunn jaw-winking ptosis
      ii Misdirected third nerve ptosis (may also be acquired)
2 Acquired ptosis
   a Neurogenic
   b Myogenic
   c Traumatic
   d Mechanical

Pseudoptosis: anophthalmia/microphthalmia/phthisis bulbi, hypotropia, dermatochalasis; contralateral eye lid retraction/proptosis.

History

1 Previous photographs of the patient (family album tomography (FAT) scan)
2 Onset and duration of ptosis
3 Aggravating and alleviating factors
4 Diurnal variation
5 Progression of ptosis (increasing, decreasing, or constant)
6 Family history
7 Any history of
   A. Diplopia
   B. Weakness in any other body part (limb weakness or any difficulty in deglutition)
   C. Trauma (including birth trauma in case of congenital ptosis), or previous surgery (ptosis/eyelid surgery, orbital surgery, intraocular surgery)
   D. Chronic drug intake
   E. Long-term use of steroid drops
   F. Contact lens usage
   G. Bleeding tendency or usage of any blood thinning agents.

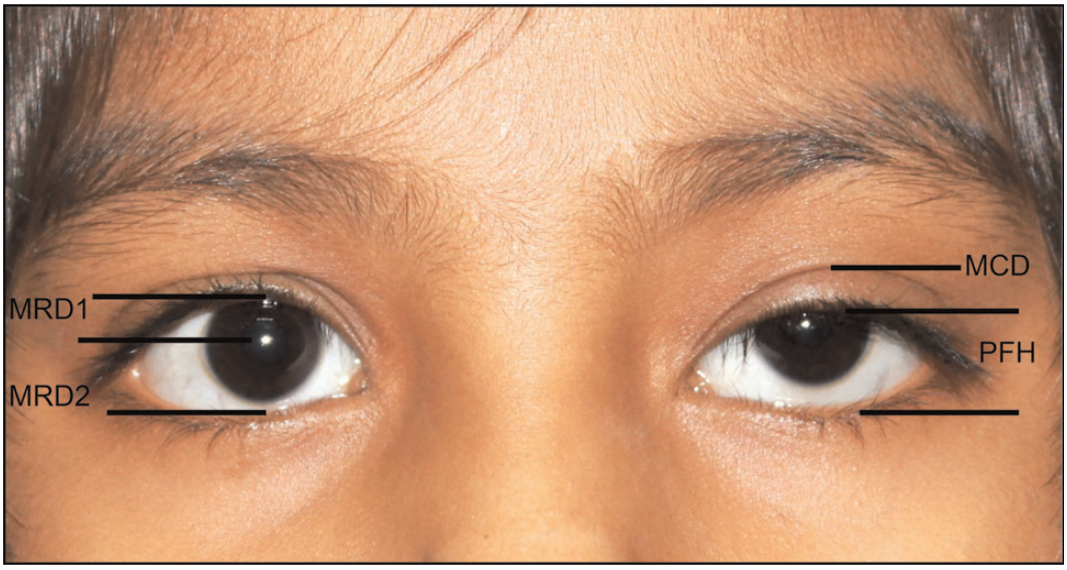
Examination of a ptosis patient

1 **Head posture:** a persistent chin up position
2 **Facial asymmetry/dysmorphism:** craniofacial syndromes like blepharophimosis syndrome.
3 **Periocular skin:** wrinkled tissue paper in blepharochalasis; mechanical ptosis in lid edema, hematoma or ecchymosis; associated brow ptosis / dermatochalasis.
4 **Visual acuity and refraction:** rule out amblyopia in a pediatric patient because of associated refractive error (commonly' with the rule' astigmatism), stimulus deprivation and/or strabismus.
Cycloplegic refraction is mandatory in children.
5 **Hirschberg/cover test:** rule out associated squint, particularly hypotropia.
6 **Pupillary evaluation:** abnormal in Horner's syndrome, Third nerve palsy and Myotonic dystrophy.
7 **Slit lamp evaluation:** Chronic conjunctivitis, uveits can cause ptosis. Cataracts should be ruled out in myotonic dystrophy. Heterochromia can be seen in congenital Horner's syndrome.
Examination of the upper palpebral conjunctiva is mandatory in ptosis patients.
8 **Fundus evaluation:** ptosis with pigmentary retinopathy is seen in Myotonic dystrophy, chronic progressive external ophthalmoplegia and Kearns–Sayre syndrome.
9 **Measurements in a case of ptosis (Fig. 1)**

A. Palpebral fissure height (PFH): the distance from the upper eyelid margin to the lower eyelid margin taken at the centre of the lid, with patient fixating at a distant target.
Normal is 8–10 mm.

Marginal reflex distance 1 (MRD1): the distance between the corneal light reflex and the upper eyelid margin with the eye in primary gaze.
Normal = 4–5 mm.

If the lid margin is below the corneal reflex the MRD-1 is measured by elevating the lid till the corneal light reflex is visible and expressing the distance in negative value.



Grading of severity of ptosis

Mild ≤ 2 mm.

Moderate = 3 mm

Severe ≥ 4 mm

B. Marginal reflex distance 2 (MRD2): The distance between the pupillary light reflex and lower lid margin with eye in primary gaze.

Normal = 5-6 mm.

Margin crease distance (MCD): the distance from upper lid margin to the eyelid crease. *Normally it is 8–10 mm in females and 5–7 mm in males.*

Levator excursion: the distance the eyelid travels from downgaze to upgaze while the frontalis muscle action is negated.

Grading of LPS action

≤4 mm: Poor

5–7 mm: Fair

8–12 mm: Good

>12 mm: excellent.

Normal LPS action: 13–17 mm.

Iliff test: Assesses levator function indirectly. If the levator action is good, everted upper eyelid of a child will revert on its own.

10 **Lid lag:** Seen when dystrophic LPS fail to relax on downgaze.

11 **Lagophthalmos:** Incomplete closure.

12 **Bell's phenomenon and corneal sensitivity:** normal protective phenomenon of upward and outward movement of the globe on closure of the eyes. Poor or absent Bell's phenomenon and corneal sensation would carry risk of post-operative exposure keratopathy.

13 **Marcus Gunn jaw-winking and other synkinetic movements:** the upper lid elevates on opening the mouth or on sideways movement of the jaw. Synkinetic movement of the lid with eye movements seen in misdirection syndromes. (3rd nv aberrant regeneration.)

14 **Extra ocular movement (EOM):** An elevation restriction is noted in many cases of congenital ptosis because of concurrent superior rectus weakness. EOM restriction is seen in CPEO, myotonic dystrophy, myasthenia gravis and third nerve palsy.

15 **Herring's phenomena:** Drooping of the contralateral lid on elevating the ptotic lid is noted as Herring's positive.

16 **Fatigability:** The patient is asked to look up at the examiner's finger without blinking for 30 s and the lid position is observed. Progressive increase in drooping is seen in ocular myasthenia gravis. It may also be positive in elderly patients with aponeurotic ptosis.

17 **Cogan's lid twitch sign:** the patient is asked to look at the examiner's finger for several seconds in downgaze and then asked to fixate quickly in primary gaze. The overshooting of upper eye lid before settling down to normal indicates a positive Cogan lid twitch sign signifying myasthenia.

18 **Ancillary tests**

A. Phenylephrine test: Improvement ≥2 mm after instillation of 10% phenylephrine eye drops in superior fornix in cases of mild ptosis is noted as positive.

B. Ice test: Ice test is a rapid, simple and inexpensive test to diagnose myasthenia with a high degree of specificity and sensitivity. The test is considered positive if the size of the palpebral fissure improves by 2 mm after cooling. The improvement typically lasts <1 min.

C. Schirmer's test

Investigations

	Test	Indications
A	Tensilon test	Ocular myasthenia gravis (OMG)
B	Electromyogram	OMG
C	Magnetic resonance imaging/angiography	Acute third nerve palsy secondary to an aneurysm
D	USG/CT/MRI	1. Acquired ptosis with atypical features 2. Progressive ptosis as evidenced on FAT scans 3. High degree of clinical suspicion of an underlying orbital pathology.

Postoperative complications

1 Undercorrection
2 Overcorrection
3 Eyelid contour asymmetry
4 Exposure keratitis

Side-effects

1 Lid lag
2 Lagophthalmos

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Centurion Syndrome

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Introduction

Centurion syndrome is an idiopathic medial canthal anomaly resulting in epiphora. These patients have prominent nasal bridge with an anteriorly inserted medial canthal tendon (MCT), which in combination result in an outward displacement of lacrimal puncta out of the lacus lacrimalis.[1] Patients commonly present in the second decade of their life. The term "Centurion syndrome" is derived from the Roman soldiers (centurions) who had a similar facial appearance.[1] The syndrome often goes unrecognized due to lack of awareness.

Case report

A 16-year-old male presented to our Oculoplasty clinic with complaints of watering from both the eyes for the last 2 years. There was no history of discharge, trauma, surgery or any episode of acute dacryocystitis. Patient had consulted many ophthalmologists before presenting to us and had been prescribed lubricants and antibiotic eye drops. The patient had no relief in his symptoms with any of the medications and was facing significant difficulty in his studies because of continuous watering.

On examination the best corrected visual acuity in both the eyes was 20/20. Tear film height was raised in both the eyes. Fluorescein dye disappearance test (FDDT) showed a minimal dye retention in the right eye (FDDT grade 1) and a significantly thick strip in the left eye (FDDT grade 3).[2,3] Slit lamp examination revealed that the patient had a prominent nasal bridge and the puncta of both the eyes were not apposed to the globe (out of lacus lacrimalis) (Fig. 1a and b). Regurgitation on pressure over lacrimal sac was negative and syringing was freely patent in both the eyes. Hertel's examination showed a reading of 17 mm in both the eyes. Rest of the anterior and posterior segment examination was within normal limits. Based on the clinical history and examination a diagnosis of Centurion syndrome was made. Patient was advised MCT disinsertion in both the eyes. Since patient was more symptomatic in left eye he wished to undergo surgery in the left eye. Postoperatively there was significant subjective improvement in watering (>80%) and the left eye puncta were found to be well apposed to the lacus lacrimalis. Postoperative FDDT grade was 0 (Fig. 2).

Discussion

"Centurion syndrome" or "Idiopathic anterior displacement of the medial canthus" was first reported by Sullivan et al. in 1993.[1] They for the first time pointed out that the anteriorly malpositioned MCT was responsible for displacement of puncta out of tear lake resulting in watering in these group of patients. They used the term "Centurion syndrome" to describe this anomaly recognizing the similarity of nasal structure between the patients and Roman Centurions, but also clearly stated that prominent nasal bridge

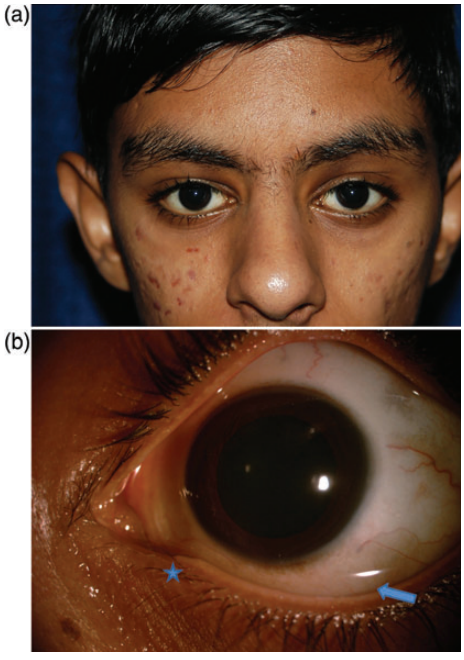

Fig. 1. (a) Clinical photograph of the patient showing prominent nasal bridge. (b) Close up photograph of the left eye showing punctum (asterisk) out of lacus lacrimalis (arrow) and raised tear film height.

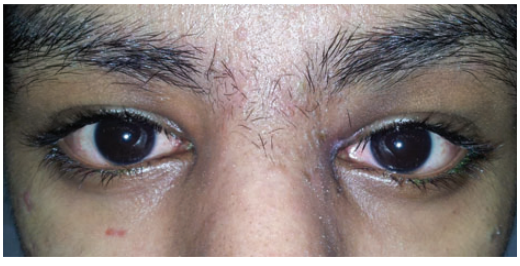

Fig. 2. Postoperative photograph of the patient showing scar in the left lacrimal sac area along with a normal FDDT.

was not a prerequisite for diagnosis and was only one of the several signs present.

The sharp inferior angulation of the medial canthus has been referred to as "beak sign".[4]

The mean age of onset of watering is 20 years.[4,5] The onset in second decade is attributed to the growth of the mid face during this period resulting in pulling of the MCT forward.[1]

Enophtahlmos is another important contributory factor noted in these patients, which by relative retrodisplacement of the globe results in the loss of punctal globe apposition.[6]

These patients seek medical help due to epiphora and the diagnosis is essentially clinical. The condition is managed surgically. Various surgical procedures for the management of this condition including isolated MCT release, or combined with other procedures like punctoplasty, medial conjunctivoplasty and lower lid retractor plication or posterior plication of the MCT stump, have been proposed with varying results.[1,4–6] The aim of surgery is to restore punctal globe apposition.

Sujatha et al.[5] achieved good results with release of anterior limb of MCT alone. However, Ma'luf et al.[6] found unsatisfactory results with isolated MCT release. They concluded that globe retroplacement is also an important contributory factor in many of these cases and mere anterior canthal tendon release might not suffice. There was no enophthalmos in our patient and hence an isolated MCT release resulted in good punctal globe apposition and relief in epiphora.

Conclusion

Centurion syndrome is an important cause of epiphora in young adults and often goes unrecognized. Careful examination of the patient reveals lack of punctal globe apposition and the typical facial features diagnostic of centurion syndrome. Release of anterior limb of MCT provides significant clinical improvement.

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Management Options for Acquired Contracted Socket

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The loss of an eye due to trauma, disease, malignancy or even congenital deformities causes significant psychological trauma to the patient as well as their near and dear ones. In cases where the visual prognosis is nil, the best solution that an ophthalmologist can offer is a cosmetically comparable appearance so that the patient can face the world confidently.

Contracted socket is the shrinkage of all or part of orbital tissue causing shallowing of the fornices and decrease in the orbital volume and thus causing the inability to retain prosthesis.[1]

Post Enucleation Socket Syndrome consists of superior sulcus depression, pseudoptosis of upper lid and ptosis of lower lid. This is mainly due to orbital fat shrinkage, cicatricial changes, fornix contraction and volume redistribution.[2]

Classification of contracted socket has been described by Krishna[3] in detail in the following manner:

Grade-0: Socket is lined with the healthy conjunctiva and has deep and well-formed fornices.

Grade-I: Socket is characterized by the shallow lower fornix or shelving of the lower fornix.

Grade-II: Socket is characterized by the loss of the upper and lower fornices.

Grade-III: Socket is characterized by the loss of the upper, lower, medial and lateral fornices.

Grade-IV: Socket is characterized by the loss of all the fornices, and reduction of palpebral aperture in horizontal and vertical dimensions.

Grade-V: Recurrence of contraction of the socket after repeated trial of reconstruction.

There are various factors leading to a contracted socket. They are as follows:

1. Intrinsic factors: These are patient-related factors making the socket prone to contraction.

a. Cancer therapy—radiotherapy (RT) or external beam RT given to children with retinoblastoma causes significant changes in their anophthalmic sockets. As we know, RT destroys normal cells along with the cancer cells and this greatly reduces the healing capacity of the tissues. The tissues remain in a chronic state of inflammation and are prone to infection. Chemotherapy (CT) also delays tissue healing.[1]

Various studies have concluded that children who receive CT or RT, alone or in combination have greater rates of implant exposure and extrusion, socket contraction, inflammation and infection compared with those children who have untreated sockets.[1,4]

b. Chemical burns—particularly alkali burns cause excessive tissue damage and inflammation that heals with fibrosis and leads to contracted socket.[1,3]

c. Trauma—extensive lacerations of conjunctiva and orbital tissue associated with tissue loss and fibrosis resulting in socket contraction.[1,3]

2. Extrinsic factors: These are factors related to surgery and postsurgical course which induce contraction of the socket.

a. Poor surgical technique—extensive orbital tissue dissection and failure to meticulously preserve the tissue planes leads to fibrosis during the healing. If the conjunctiva is sacrificed, the fornices shallow with time.[1,2]

b. Absence of implant—if no implant or an undersized implant is placed, the resultant volume deficit has to be tackled by using a heavy prosthesis. This further causes pulling of lower lid and its laxity, shallowing of inferior fornix and the levator palpebrae superioris loses its fulcrum of action, all this leading to PESS. In children, absence of implant means absence of stimulus for orbital growth and consequently bony contraction and facial asymmetry.[2]

c. Absence of conformer—a conformer keeps the conjunctiva stretched and prevents forniceal shallowing. Failure or delay in the placement of conformer after socket surgeries causes shortening of the fornices.[1,2]

d. Implant or prosthesis-related factors—the healing of orbital tissues after an implant exposure or extrusion occurs with formation of scar tissue. The use of ill fitting or improper prosthesis causes continuous irritation of orbital tissues and induces fibrosis and shortening of fornices.[1–3]

Management of contracted socket

Prevention is the first step in management of this condition. Meticulous surgical dissection preserving tissue planes and minimal damage to conjunctiva is the first step in prevention of contracted socket. This results in good healing of tissues and minimal fibrosis. An optimal size implant must be placed during the primary surgery to maintain orbital volume. A conformer should be placed immediately after surgery to keep the tissues stretched. The conformer should be of correct side, optimal size (maximum size that fills the fornices and allows comfortable eyelid closure) and have multiple holes through which postoperative medication can seep into the conjunctiva and also the secretions can be flushed out.[1,2]

Various techniques are used to handle the issue of contracted socket. They are as follows:

1. Orbital implants

If an implant is not placed in the first surgery a secondary ball implantation should be done for volume augmentation. The implant size to be placed depends upon whether it is wrapped or unwrapped. If unwrapped, Implant size = (Axial length of the other eye – 2 mm). If wrapped, an additional 1 mm has to be deducted.[1,2]

In cases of evisceration the implant is placed in the residual scleral space after removing the uveal tissue. This gives the best implant motility as it manipulates least with the dynamics of extraocular muscle action.[5] The different types of ball implants available are:

a. Non-integrated (non-Porous)—these are non-porous implants which do not integrate with the orbital tissues. If used after enucleation, the implant needs to be wrapped in natural (banked sclera, banked human dura mater, autogenous fascia lata) or synthetic materials (gelfoam, polyglactin mesh) to facilitate attachment of the extraocular muscles. If a wrapping material is not available, Myoconjunctival technique of enucleation with ball implantation should be used.[1,2]

- Polymethylmethacrylate (PMMA),
- Silicon.

b. Semi integrated—these are synthetic implants that have multiple holes in their anterior surface to facilitate the attachments of extraocular muscles, thereby giving a good ocular motility. They are made of PMMA. They have a rough anterior surface that might cause the orbital tissues to be pinched between the implant and prosthesis. These are of historical importance as their use have been discontinued due to unacceptably high exposure and extrusion rates.

- Castroviejo,
- Allen's.

c. Fully integrated implants (porous)—these are made of porous material that allow fibrovascular growth into the implant and thereby its integration into the orbital tissues that aids better vascularization. This is said to result in lower rates of extrusion and infection, spontaneous healing of exposure and better motility of prosthesis.[1,2,6]

- Natural Hydroxyapatite,
- Synthetic Hydoxyapatite,
- Aluminium oxide,
- Polyethylene,
- Porous PMMA.

d. Expandable implants—these implants expand *in situ* and thus are specifically useful in cases of children where a stimulus for orbital growth is required.[2]

- Silicon balloon expanders,
- Hydragel expanders.

2. Biogenic/aulogous implants—dermis fat graft (DFG)

DFG is an autologous implant, harvested most frequently from the gluteal region or abdominal wall (non-hair bearing areas). While harvesting the graft the epidermis is removed off the rest of the graft so that the graft contains only dermis which acts like a scaffold for the conjunctiva to grow over it once the DFG is placed in the socket.[7,8] DFG is preferred in the following scenarios:[2,7]

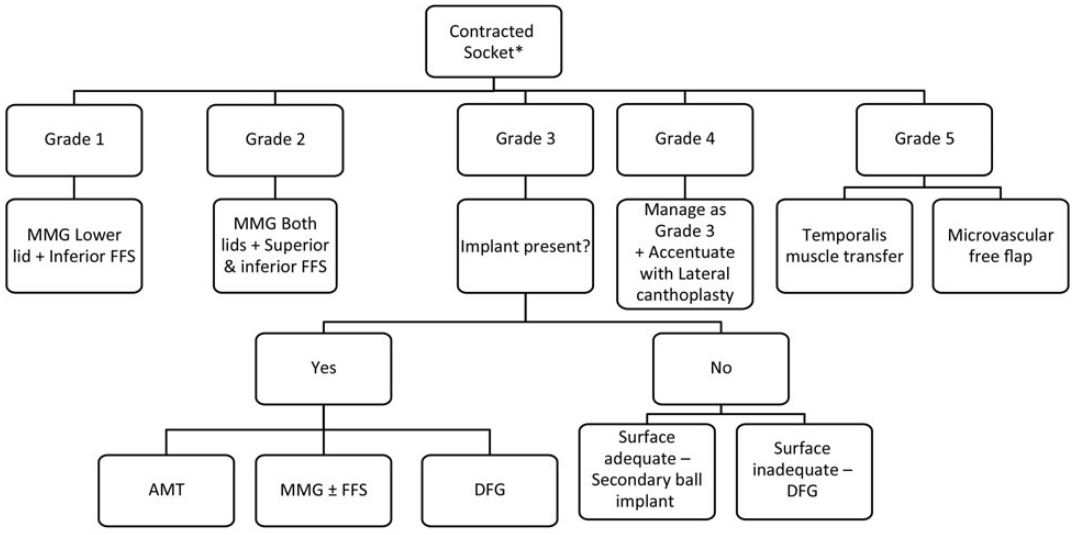
- As a secondary procedure after extrusion or exposure of ball implant.
- For reconstruction of badly disorganized sockets.
- In patients where extrusion of implant is anticipated.
- Deficient conjunctival lining along with volume deficit.
- As a primary procedure in children, since the DFG is found to grow with the growth of the child.
- As a double implant (along with ball implant) for gross volume deficit (citation).

Advantages of DFG are as follows:[2,7,8]

- Since it is an autologous material, there are no chances of extrusion, exposure or infection.
- No additional material required for wrapping, thereby reducing dependence and risk of transferring pathogens or prions.
- The extraocular muscles can be sutured to the ends of the DFG thereby resulting in acceptable implant motility and preservation of conjunctival fornices.

Disadvantages of DFG are[7,8]

- DFG undergoes variable amount of resorption over a period of time resulting in orbital volume reduction.
- Overgrowth of DFG is seen in children or adults with gain in weight.
- Occasionally can undergo necrosis.
- If extraocular muscles are not attached to the graft resultant motility of the prosthesis is compromised.



*at all stages secondary ball implantation to be considered as first step if implant not present

3. Mucous membrane graft (MMG)

MMG is used in contracted sockets where the conjunctival lining is deficient resulting in forniceal shrinkage. The mucous membrane provides additional tissue to form the fornix and a fornix forming suture can be taken to augment the procedure. MMG can also be used in conjunction with DFG if the conjunctival lining is deficient and fornices need to be deepened.[7,9,10]

MMG is harvested from the buccal or lip mucosa. The graft should be slightly oversized as it is expected to shrink postoperatively.[9]

4. Other modalities

a. Other autologous grafts—the other autologous grafts used in socket reconstruction are

- Hard palate graft[11]—for fornix reconstruction.
- Temporalis muscle transfer[12]—for poorly vascularized grade 5 contracted sockets.
- Amniotic membrane transplant[13]—for fornix reconstruction when posterior lamella is deficient, as a scaffold for the conjunctiva to grow on it.

b. Free microvascular flap—for grade 5 contracted and poorly vascular sockets, a free flap from the forearm based on radial artery is taken and its artery and vein is anastomosed to ipsilateral superficial temporal artery and vein.[14] Similarly other flaps such as forehead flap based on supratrochlear artery,[15] lateral arm flap,[16] etc. can be taken. Rest of the socket reconstruction is done few weeks later.

c. Antimetabolites—safety profile of antimetabolites in lacrimal and facial surgery has been established.[17] These can be used in grade 5 contracted sockets as adjuncts to DFG, MMG and other grafts.

Mitomycin C (MMC) is an alkylating agent and inhibits the DNA synthesis. It has a prolonged suppressive effect on fibroblastic activity and scarring. 5-Fluorouracil (5-FU) on the other hand is a short acting pyrimidine analog that promotes apoptosis in the fibroblasts during a couple of weeks after focal exposure. Thus, MMC can be given as a single dose during surgery while 5-FU can be given in repeated doses in the postoperative period along with the intraoperative dose.

Potential complications can be allergic reaction, punctual stenosis, tissue necrosis and wound dehiscence.[18]

Contracted socket leading to inability in retaining prosthesis can be distressing to the patient as well as the surgeon. Meticulous clinical assessment of each patient is imperative in deciding the appropriate plan of management. Patience on the part of the patient and clinician are essential as the management of this condition may have a long course with multiple surgical procedures and hospital visits.

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SOCKET Examination Sheet

OD/OS

COSMESIS: Good/Fair/Poor

- Color match: Good/Fair/Poor
- Movements: Good/Fair/Poor
- Lagophthalmos: ……mm
- Pseudoptosis: Present/Absent
- Enophhthalmos: Present/Absent

SOCKET: Healthy/Congested/Congestion with Papillae/Granuloma

- Volume: Adequate/Deficit
- Implant: Present/Absent
- Implant position: Central/Migrated
- Implant exposure: Present/Absent
- Superior fornix: Well-formed/Shallow/Absent
- Inferior fornix: Well-formed/Shallow/Absent
- Medial fornix: Well-formed/Shallow/Absent
- Lateral fornix: Well-formed/Shallow/Absent

SHELL

- Discoloration: Present/Absent
- Deposits: Present/Absent
- Edges: Sharp/Blunt
- Surface: Smooth/Rough/Scratches

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Eyelid Injury

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Key points

- Eyelid injuries may be associated with ocular, orbital and even intracranial injuries, management of which takes precedence over eyelid reconstruction.
- Presence of orbital fat in the wound indicates a deep wound.
- Laceration in the medial aspect of the lids is likely to involve the canalicular system.

Goals of eyelid repair

- To reestablish anatomical configuration.
- To restore physiological function.
- To provide optimum cosmetic appearance.

Timing of repair

- Eyelid injury, *per se*, is not an ophthalmic emergency, but any resultant corneal exposure is. Otherwise the repair may even be delayed for several days (ideally not more than 48 h) in case of excessive eyelid edema, infection, intoxicated or uncooperative patients.
- As mentioned already, management of intra-ocular or systemic injuries takes precedence.
- Adequate antibiotic coverage and measures to control edema (ice compress, head elevation and anti-inflammatory medications) and protection of cornea should be considered during the intervening period.

Investigations

Radiologic evaluation: indicated in suspected orbital fractures, optic nerve injury and foreign bodies.

Non-marginal superficial lid lacerations

- Superficial lacerations parallel to the relaxed skin tension lines (RSTL)
  - Tissue glue (Fibrin/Amycrylate/Dermabond®)
  - Adhesive sterile tape (Steristrip).

This method is especially helpful in young children.

- Wounds situated perpendicular to the RSTL—tendency to gape because of orbicularis contraction.
  - Suture skin and orbicularis in single layer using non-absorbable sutures like 6-0 nylon, interrupted. These sutures can be removed after 5 days.

Non-marginal deep eyelid lacerations

- Exclude intra-ocular injury.
- Explore wound.
  - Wash thoroughly with antibiotic solution (betadine).
  - Look for and meticulously remove foreign bodies, especially if organic.
  - Debride carefully. Remember, the eyelids being exquisitely vascular structure, even necrotic looking parts usually survive.
  - Assess integrity of orbital septum, and levator aponeurosis, especially if fat is seen in the wound.
  - Suture the cut edges of the levator if lacerated and reattach to the tarsal plate if it is disinserted.
  - Never suture the septum as it will lead to lagophthalmos.
  - Close in layers obliterating dead space.

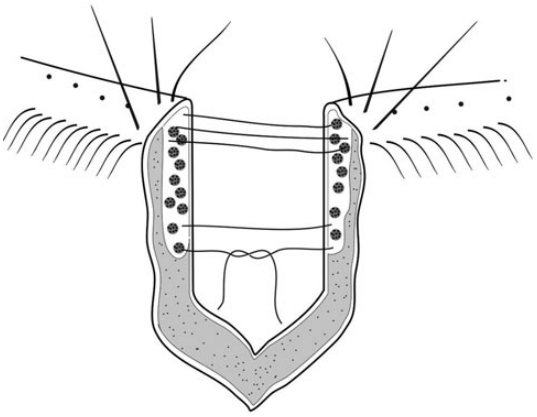
Repair of eyelid marginal laceration

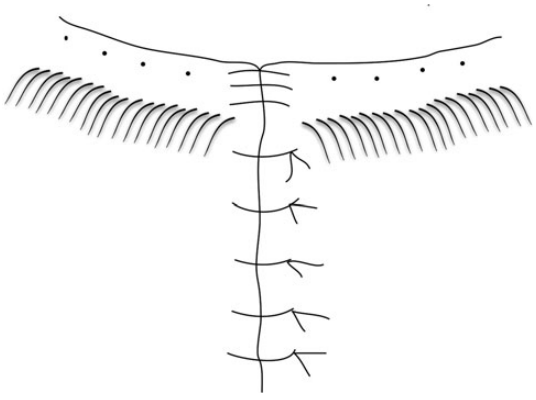
- Infiltration of local anesthetic with adrenaline at least 15 min prior to repair aids in achieving hemostasis.
- Orientation and alignment of eyelashes, meibomian gland openings and gray line help in achieving good wound apposition.
- Debridement of the wound margin must be done to achieve a pentagon-shaped defect with square edges.

Surgical steps: eyelid margin laceration (the three-suture technique)

- Re-approximate the tissue as accurately as possible.

- Marginal suture is taken through the meibomian gland orifices (1.5–2 mm from the wound edge and 1.5–2 mm in depth) with 6-0 silk or nylon.
- Two similar sutures are taken through the gray line and the lash line, tied and ends kept long.
- Tarsal plate is sutured with partial thickness 6-0 absorbable (chromic catgut or polygalactin) interrupted sutures.
- Skin is sutured with 6-0 nylon, silk or polypropylene, and the ends of marginal sutures are incorporated in the knot of skin sutures to keep them away from the cornea.
- The closure should be done with minimal tension.
- Mattress suture is used for margin apposition when there is tension in the opposing cut ends.





Eyelid margin lacerations with tissue loss

- When tissue loss is <25%, lateral canthotomy and cantholysis of the corresponding limb of the lateral canthal tendon will lead to adequate mobilization for closure.
- In case the eyelid margin defect is too large to be closed by the above-mentioned method, the following reconstructive techniques can be used:
  - Tenzel semicircular flaps.
  - Mustarde's Cheek rotation flap.
  - Hughe's Tarsoconjunctival flap with anterior lamellar reconstruction for lower lid.
  - Cutler–Beard method for upper lid.

Post-operative instructions

- Wound should be kept clean and moist.
- Topical antibiotic ointment.
- Skin sutures are usually removed after 5–7 days and lid margin sutures after 10–14 days.

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A Case of Orbital Fracture

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Introduction

The upwardly sloping orbital floor is the shortest and one of the thinnest of the four walls that frame the orbital cavity. Formed by the confluence of the zygomatic, maxillary and palatine plates and suspended above the maxillary sinus, the orbital floor support the human eye. The floor also acts a safety mechanism which protects the globe from rupturing by absorbing the energy when a blunt object, which is of equal or greater diameter than that of the orbital aperture, strikes the eye. The resultant force is transmitted throughout the orbit causing a fracture of the orbital floor.

An orbital fracture, *per se*, is not an emergent condition, with one exception (see WEBOF). Orbital fractures may be associated with intraocular injuries, like retinal detachment, even if the globe remains intact, which should be investigated first. Because just by manipulating the periorbital tissues to fix a fracture one can make the intraocular condition worse. However, failure to diagnose and treat an orbital fracture adequately may lead to severe lifelong disabilities, both functional and cosmetic, often necessitating numerous secondary attempts with suboptimal results. The difficulty level of the surgery and cost also escalate exponentially.

Most fractures are the result of accidental or homicidal trauma. There may be potential Medico-legal connotations hence proper and detailed documentation of the history of the trauma is mandatory.

Clinical features

External signs of periocular injury, like lid edema, ecchymosis, subconjunctival hemorrhage, emphysema, enophthalmos or proptosis are frequently present. Exception is seen in cases of white eyed blow out fracture (WEBOF) in pediatric age group which present with little or no signs of external injury.

Evaluation

1 Clinical
   a Systemic involvement and serious non-ocular injuries need to be ruled out first in the setting of polytrauma.
   b Comprehensive Ophthalmic Examination is required in all cases.
   c Orbit (see Evaluation of proptosis).
2 Imaging
   a CT scan: Reconstructed coronal views are most accurate and useful imaging modality in trauma patients for assessing the size, extent and configuration of the fractures. CT scan are invaluable for evaluation of entrapped soft tissues, calculation of volume for late enophthalmos, and assessment of optic canal fractures. In general, helical CT with 3-mm sections will suffice, but in situations where intraocular or intraorbital foreign bodies are suspected or fine detail is required, as in optic canal fractures, 1.25-mm thin slices or less may be desirable.
   b MRI: required in cases with associated head trauma.
3 FDT, FGT (to differentiate between paretic and restrictive motility patterns), Diplopia charting, HESS charting.
4 Photographic documentation is important for patients to appreciate an acceptable operative result as the patient or relatives may not be aware of the extent and severity of injury at the time of the trauma and may be disappointed with the results of surgery.

Tips and Tricks in clinical evaluation of a fracture patient:

Clinical sign	Denotes	Instructions	Possible complications
Epistaxis	Medial wall/nasal bone fracture	Do not blow nose; Decongestant nasal drops	Orbital emphysema
CSF rhinorrhea	Roof fracture	Neurosurgeon consult	Meningitis
Telecanthus	NOE fracture	ENT consult	Traumatic NLDO
Malocclusion/Trismus	Mandibular fracture	OFMS consult	Inability to open mouth fully
Numbness cheek/ upper teeth	Floor fracture involving infra orbital canal		Persistent paresthesia/ anesthesia

Continued

Continued

Clinical sign	Denotes	Instructions	Possible complications
Orbital margin discontinuity	Impure fracture	Rim involvement	Depressed malar prominence
Crepitus	Medial wall fracture	Do not blow nose; restrict air travel	Orbital emphysema
Enophthalmos	Large fractures	Need early repair	Late enophthalmos
Proptosis	Soft tissue swelling; orbital hemorrhage; emphysema; blow-in fractures	Careful assessment of Optic nerve function; Systemic Steroids	Optic nerve compression; globe indentation
Diplopia	EOM/orbital tissue incarceration/nerve palsy	FDT/FGT to differentiate between mechanical and neurological	Residual diplopia
Pupil involvement (check consensual reaction of contralateral eye if affected pupil not seen)	RAPD—traumatic optic neuropathy or Anisocoria —posterior floor fracture (involvement of inf divn of third nerve)	Explain visual prognosis	Visual disability or Glare
Nausea, vomiting, bradycardia	Oculo-cardiac reflex in WEBOF	Immediate surgical intervention	Ischemia of the entrapped muscle due to Compartment syndrome

NOE, naso-orbito-ethmoidal; NLDO, naso-lacrimal duct obstruction; EOM, extra ocular muscle; FDT/FGT, forced duction/generation test; RAPD, relative afferent pupillary defect; WEBOF, white eyed blow out fracture.

Management:

Primary: As mentioned, orbital fracture is not an emergency, and hence can be treated on an outpatient basis after assessment and investigations. Surgery, if required, can be done within 1–2 weeks for resolution of edema and inflammation. Meanwhile, the following steps are taken:

1 Patients instructed not to blow nose.
2 Short course of systemic steroids after ruling out any contraindication.
3 Cold compresses.

Indications of surgical intervention:

1 Symptomatic persistent diplopia in functional gazes with +ve FDT.
2 CT evidence of entrapped/herniated orbital tissue or muscle.
3 Enophthalmos of 2 mm or more which is cosmetically unacceptable to the patient.
4 Significant globe ptosis.
5 Large fractures (<50%, posterior to equator) likely to result in late enophthalmos.
6 Associated facial/comminuted fractures.
7 WEBOF.
8 Blow-in fractures compromising vision.
9 Non-resolving Oculo-cardiac reflex.

Of the above, only WEBOF and non-resolving oculo cardiac reflex (which may cause even syncope and asystole), are true emergencies. These should command immediate attention.

Fracture repair:

The aims of surgery are

a Reconstruction of normal orbital anatomy.
b Restoration of orbital volume.

The surgical steps are:

1 Exposure of the fractured area up to the posteriormost extent.
2 Release of all prolapsed/entrapped tissues and reposition in the orbital cavity.
3 Replace the missing part of orbital walls with grafts/implants.
4 Restore orbital volume, if needed, by thicker/enophthlmic implants.

The surgical entry of choice for me is a transconjunctival (swinging eyelid) approach to the floor instead of transcutaneous, as chances of ectropion are much lower. If the medial wall needs exploration, it is relatively easy to extend this into

a transcaruncular approach as well. For larger fractures, cantholysis of the inferior crus of lateral canthal tendon is required. FDT is performed before and after surgery to ensure release of any entrapped muscle and the pupil is monitored during the surgery.

Choice of implants: I generally do not prefer autografts (cartilage/bone) because of their unpredictable absorption; donor site morbidity; increased surgical time and complexity. Alloplastic implants like titanium and porous polyethylene plates are easily available. These can be molded to required shape, size and thickness and offer significant technological improvement over mesh, silicone sheets and calvarial bone.

I use preformed titanium implants (with convexity simulating that of orbital floor) that can be fixed to the orbital rim for large fractures; porous polyethylene for medium fractures with stable posterior ledge (on which the implant can be placed without fear of dislocation) and thin Polycaprolactone bioresorbable material for pediatric trapdoor fractures (as they need only temporary support and not volume replacement).

Some cases need multidisciplinary approach for optimum care. Associated mid facial fractures, lateral wall or ZMC fractures are repaired with help from Oro-facio-maxillary Surgeon; roof fractures need neurosurgeon consultation; NOE fractures may need ENT opinion. Each specialty provides a particular expertise for best result.

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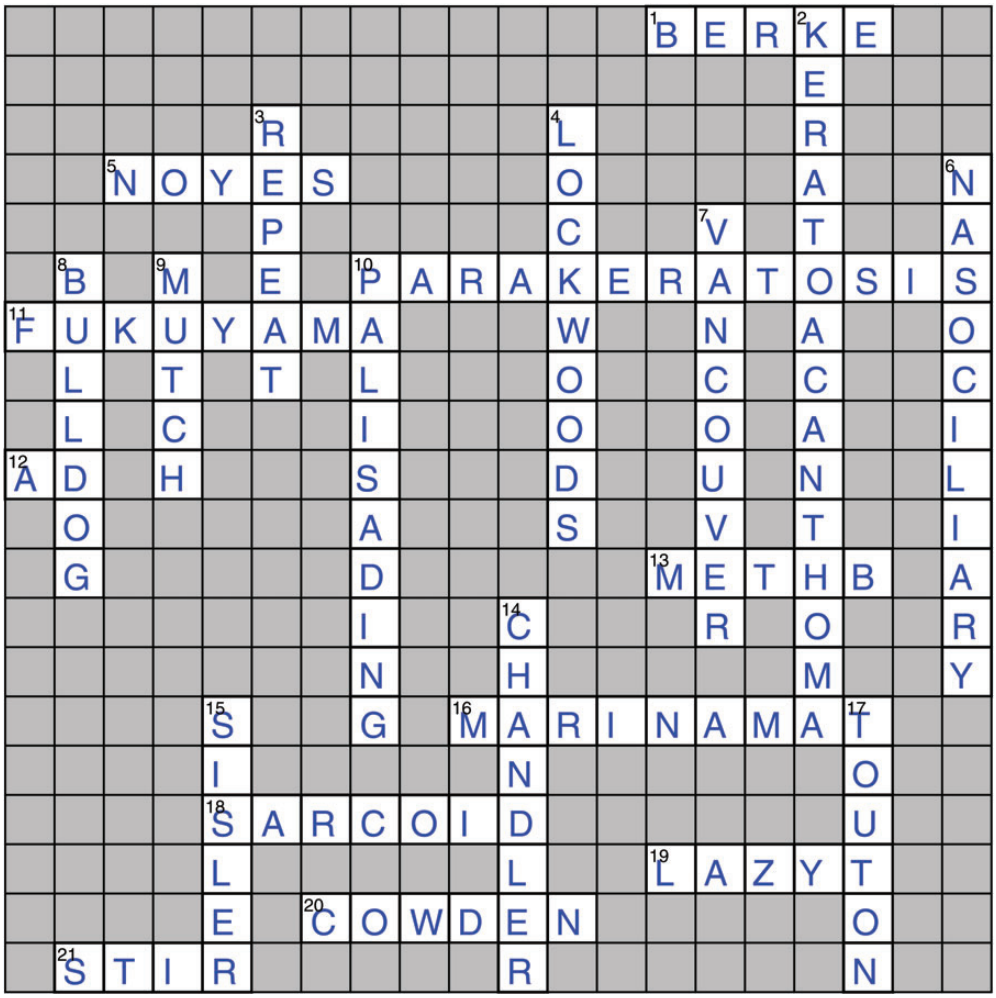
Oculoplasty Crossword – Answer



Evaluation of a Case of Proptosis

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Proptosis is defined as an abnormal protrusion of the globe. Exophthalmos denotes an active process, e.g., thyroid eye disease (TED).

History

- Age
  - Congenital:
  - Acquired:
- Onset and duration:
  - acute: traumatic, infective or malignant
  - subacute: inflammatory
  - chronic: benign neoplasm
- Clinical features:
  - Symptoms
    - Painful—inflammatory, infectious lesions, orbital hemorrhage , malignant tumors
    - Diurnal variation: symptoms are more in the morning in thyroid ophthalmopathy
  - Signs
    - Typical lid signs in TED
- Systemic history: H/o trauma, thyroid dysfunction, sinus problem, systemic malignancies, bleeding tendencies or drug use.

Beware of pseudoproptosis

- Facial asymmetry
- Ipsilateral enlargement of globe
  - High Myopia
  - Buphthalmos
- Ipsilateral lid retraction
- Contralateral Ptosis
- EOM weakness or paralysis.
- Contralateral enophthalmos

Examination

- Visual Acuity
- Refraction
- Colour Vision
- Pupils
- Extraocular Motility/Hirschberg/Cover test
- IOP
- Slit lamp examination
- Fundus
  - Optic Disc: Pallor/edema
  - Blood Vessels: Optico-ciliary shunt vessels
  - Choroidal Folds
  - Venous engorgement
- Facial asymmetry/dysmorphia
  - Neurofibromatosis
  - Fullness in temporal fossa: sphenoid wing meningioma
- Exophthalmometry
  - Hertel's Exophthalmometer: Measures position of the apex of the cornea in relation to outer orbital margin. An asymmetry of greater than 2 mm between the two eyes suggests proptosis or enophthalmos. The concavities are placed over the lateral orbital rim lightly and the patient is asked to look straight ahead. The examiner places his/her fifth finger in the patient's external auditory meatus for stabilization. The same base setting is used in each follow up of a given patient. The red line and its mirror image are superimposed, which corresponds to 18mm. the position of the apex of the cornea is read from the matching calibration.
  - Nafziger method: Examiner stands behind the patient with patients head thrown back and looking straight ahead. Examiner raises the upper lids and compares the level of apex of the cornea.
- Globe Displacement—Horizontal & Vertical (2 ruler test)
  - Horizontal distance between center of bridge of the nose to the limbus of either eye is measured and compared to find out presence of horizontal displacement.
  - A second ruler held vertically measures the distance the globe above or below the canthi for vertical displacement
- Eyelids
  - Position (retraction, inf. scleral show, lateral flare)
  - Margin-Reflex Distance (MRD1)
  - PFH (Palpebral fissure height)
  - Lagophthalmos
- Palpation
  - Orbit
    - Tenderness/warmth
    - If any mass is palpable note the size, shape, position, mobility, tenderness.
    - Orbital rims for discontinuity
  - Thyroid gland
  - Regional lymph nodes
    - Pre and post-auricular; submandibular; cervical
- Globe retropulsion (RBR)
- Pulsation/Thrill/Bruit (with the Bell of the stethoscope)
- Valsalva
- Cranial nerves examination (II, III, IV, V, VI)
- Systemic: Skin, Oral & Nasal Examination
- Imaging : USG, CT, MRI

6 Ps: Pain, Proptosis, Progression, Palpation, Pulsation, Periorbital changes

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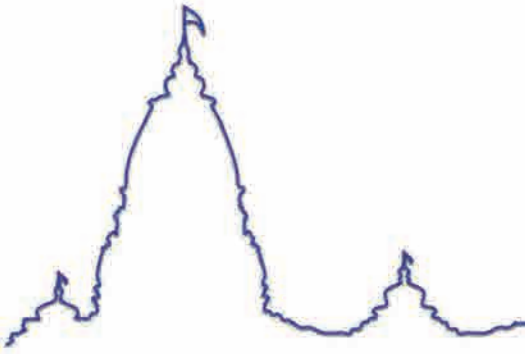

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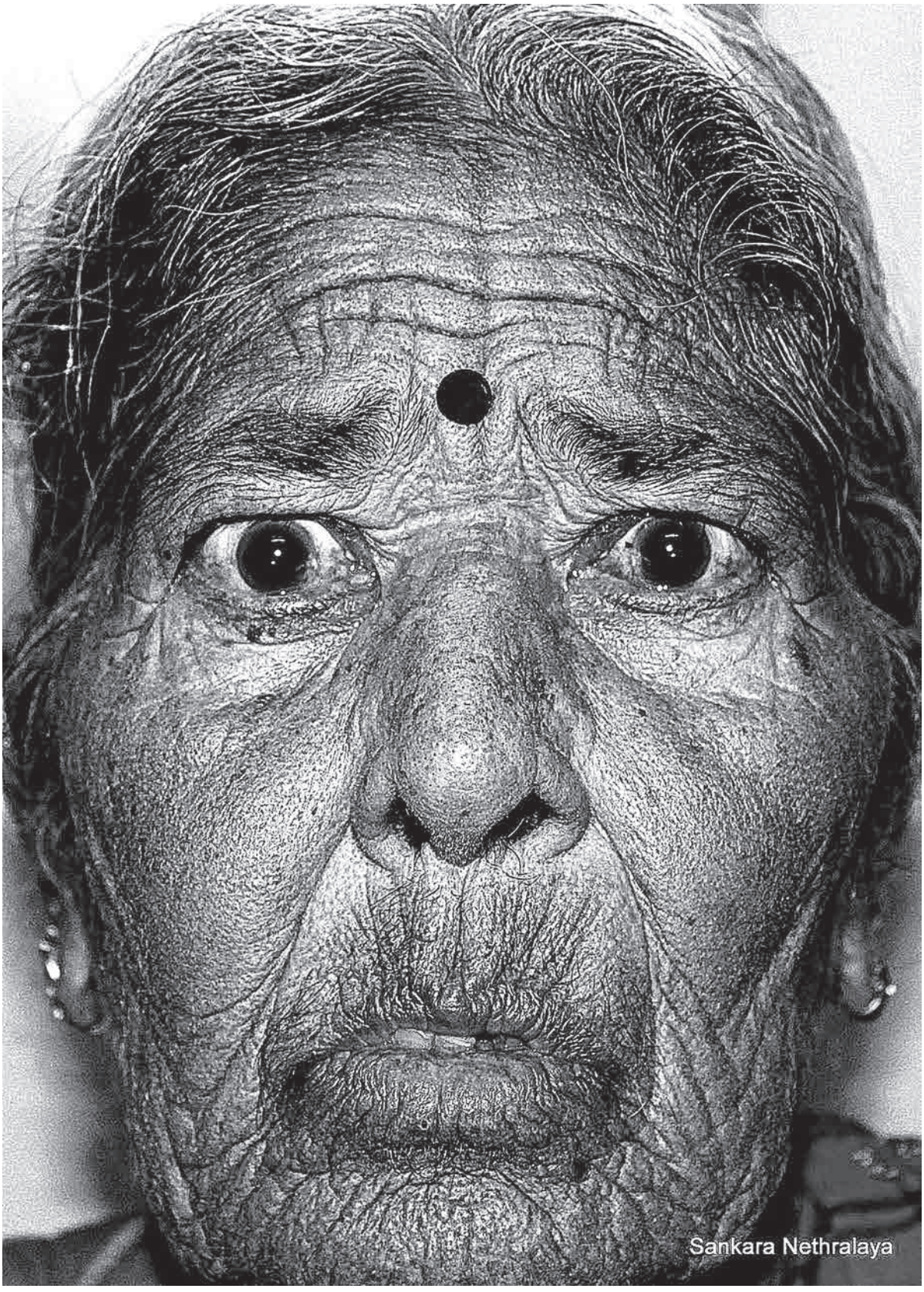
SANKARA NETHRALAYA

"The Map of Life"



The static and dynamic lines on the face can be ameliorated by injecting botulinum toxin, dermal fillers or even by editing in photoshop. But nobody can erase the stories behind these lines. Each wrinkle, each rhytid, each groove on this face has been etched by life herself. A smooth unlined face can never illustrate the character which is depicted on this black and white portrait of this 70-yr-old female patient. The map of life represents the indomitable spirit of this woman against the inexorable march of time.