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Diseases of the Eye Orbit: A Narrative Review of Orbital Anatomy, Disorders, and Developmental Anomalies

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ABSTRACT

The orbit is a complex anatomical structure that houses the eyeball and its associated neurovascular and muscular components. Orbital diseases comprise a diverse group of disorders that may be inflammatory, infectious, vascular, endocrine, neoplastic, traumatic, or developmental in origin. Because of the close relationship of the orbit with the cranial cavity, paranasal sinuses, and visual pathways, orbital disorders may result in significant ocular morbidity and, in severe cases, life-threatening complications.

This narrative review provides an overview of the anatomy of the eye orbit and discusses important orbital diseases including proptosis, orbital cellulitis, enophthalmos, endocrine enophthalmos, and cavernous sinus thrombosis. The review further highlights developmental anomalies of the orbit such as anophthalmos, microphthalmos, orbital hypertelorism, hypotelorism, craniosynostosis, orbital coloboma, dermoid cysts, encephalocele, meningocele, and congenital orbital cysts. Emphasis is placed on aetiology, clinical features, diagnostic investigations, and current management strategies.

Timely diagnosis and appropriate multidisciplinary management are essential for preventing visual impairment, cosmetic deformities, and intracranial complications. Advances in diagnostic imaging, medical therapy, and reconstructive surgical techniques have significantly improved the prognosis and quality of life of patients with orbital diseases.

KEYWORDS

Orbit, Proptosis; Orbital Cellulitis; Enophthalmos; Cavernous Sinus Thrombosis; Developmental Anomalies; Orbital Tumors, Ophthalmology

INTRODUCTION

The orbit is a protective bony cavity that houses the eyeball and its associated structures, including the extraocular muscles, nerves, blood vessels, lacrimal gland, and orbital fat. The protective bony margins surrounding the eye are commonly referred to as the

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orbital socket. Anatomically, the two bony orbits resemble quadrangular truncated pyramids and are located between the anterior cranial fossa superiorly and the maxillary sinus inferiorly [1].

The orbit plays a vital role in protecting the globe and supporting normal ocular function. Because of its close relationship with the paranasal sinuses, cranial cavity, and important neurovascular structures, orbital diseases may lead to significant ocular and systemic complications. Disorders affecting the orbit can impair vision, ocular motility, facial symmetry, and, in severe infectious or intracranial conditions, may even become life-threatening [2].

Orbital diseases comprise a broad spectrum of conditions that may be inflammatory, infective, vascular, endocrine, neoplastic, traumatic, or developmental in origin. Common orbital disorders include proptosis, orbital cellulitis, enophthalmos, cavernous sinus thrombosis, and various congenital anomalies such as microphthalmos, hypertelorism, dermoid cysts, and craniosynostosis. Early recognition and timely management of these conditions are essential to prevent visual loss and long-term complications [3].

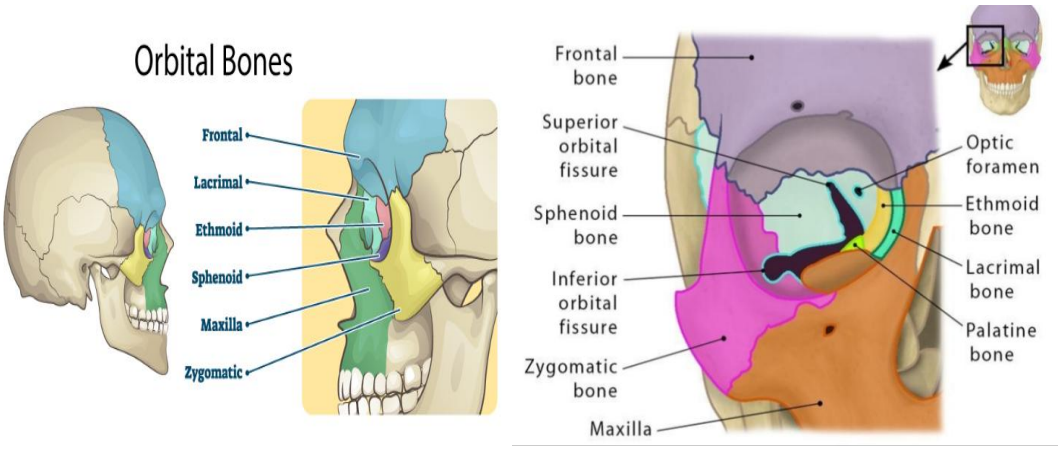
A sound understanding of orbital anatomy and pathology is therefore fundamental for ophthalmologists and healthcare professionals involved in the diagnosis and management of orbital diseases.

ANATOMY OF THE EYE ORBIT

The orbit is a complex bony cavity that accommodates the eyeball and its supporting structures, including extraocular muscles, nerves, blood vessels, orbital fat, and the lacrimal gland. Each orbit is composed of seven bones: the frontal, ethmoid, lacrimal, palatine, maxillary, zygomatic, and sphenoid bones [1].

The medial walls of the two orbits are parallel to one another and are closely related to the ethmoidal and sphenoidal sinuses, which separate the orbit from the nasal cavity. In contrast, the lateral walls are positioned at an angle of approximately 45° to the medial walls, while the lateral walls of both orbits form nearly 90° with each other. This arrangement contributes to binocular vision and protection of the ocular structures [2].

The average orbital depth measures approximately 42 mm along the medial wall and 50 mm along the lateral wall. The orbital base is about 40 mm wide and 35 mm high.



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Figure 1: Anatomy of the Orbit showing the bony orbital cavity and surrounding structures. (Source: Standard orbital anatomy diagram, *Khurana A. K., Comprehensive Ophthalmology*)

Figure 1 Illustration showing the important bones of the orbit in different views, demonstrating the contribution of the frontal, ethmoid, lacrimal, maxilla, zygomatic, sphenoid, and palatine bones in forming the orbital cavity.

Walls of the Orbit

The orbit consists of four walls: the medial wall, floor (inferior wall), lateral wall, and roof.

Medial Wall

The medial wall is quadrilateral in shape and is formed by the frontal process of the maxilla, lacrimal bone, orbital plate of the ethmoid bone, and body of the sphenoid bone. Anteriorly, the wall contains the lacrimal sac fossa, which continues inferiorly as the nasolacrimal canal [3].

Floor of the Orbit

The orbital floor is triangular and forms the shortest wall of the orbit. It is mainly composed of the orbital surface of the maxilla, with contributions from the zygomatic and palatine bones. Because of its thin structure, the orbital floor is particularly vulnerable to blowout fractures following trauma.

Lateral Wall

The lateral wall is triangular and considered the strongest wall of the orbit. It is formed anteriorly by the zygomatic bone and posteriorly by the greater wing of the sphenoid bone.

Roof of the Orbit

The roof is formed mainly by the orbital plate of the frontal bone and posteriorly by the lesser wing of the sphenoid bone. The anterolateral portion of the roof contains the lacrimal gland fossa.

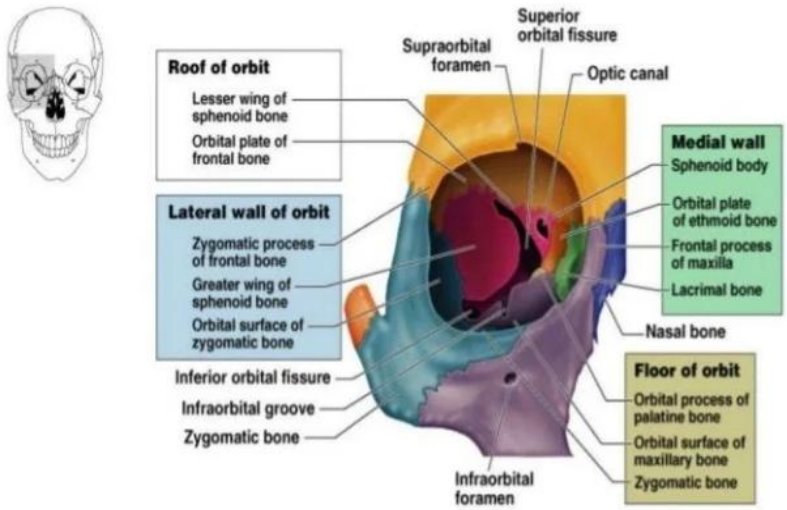


Figure 2: Orbital walls showing medial, lateral, roof, and floor with bony components. (Source: Orbital wall anatomy illustration, *Kanski J. J., Clinical Ophthalmology*)

Figure 2 illustrates the anatomical walls of the orbit, including the medial wall, lateral wall, roof, and floor, for better understanding of orbital structure and its clinical significance.

Base and Apex of the Orbit

The anterior opening of the orbit is referred to as the orbital base and is bounded by the orbital margins. The posterior end is known as the orbital apex, where all four orbital walls converge. The orbital apex contains two important openings: the optic canal and the superior orbital fissure, both of which transmit essential neurovascular structures [2].

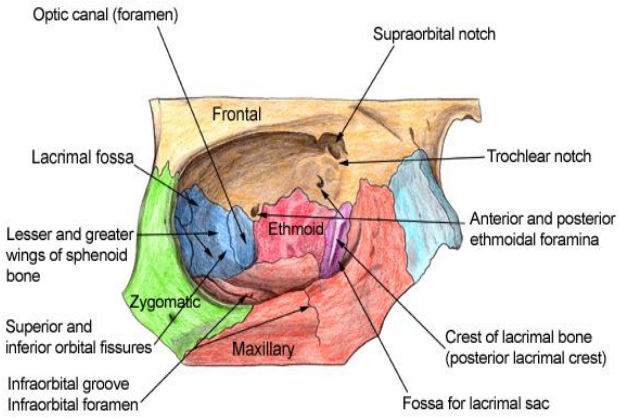


Figure 3: Base and apex of the orbit demonstrating orbital rim and optic canal/superior orbital fissure. (Source: Anatomical diagram of orbital apex and base, *Parsons' Diseases of the Eye*)

Figure 3 show anatomical representation of the base and apex of the orbit showing the orbital margins anteriorly and the optic canal and superior orbital fissure at the orbital apex.

DISEASES OF THE ORBIT

PROPTOSIS

Proptosis is defined as forward displacement of the eyeball caused by a space-occupying lesion within the orbit. The term exophthalmos is commonly used specifically for protrusion associated with thyroid eye disease [1]. Proptosis may be classified into the following types Unilateral proptosis, Bilateral proptosis, Intermittent proptosis and Pulsating proptosis



Figure 4: Proptosis showing anterior displacement of the eyeball. (Source: Clinical orbital proptosis illustration, *Kanski's Clinical Ophthalmology*)

Figure 4 show clinical representation of proptosis showing forward displacement of the eyeball due to an orbital space-occupying lesion.

AETIOLOGY OF PROPTOSIS

1. Unilateral Proptosis

Common causes of unilateral proptosis include inflammatory lesions such as orbital cellulitis and orbital abscess, vascular abnormalities including orbital haemorrhage and vascular malformations, and orbital tumors such as dermoid cysts, optic nerve glioma, and meningioma. Systemic conditions like leukemia and Graves disease may also contribute. Paralysis of extraocular muscles, as seen in ophthalmoplegia, can additionally produce apparent proptosis.

2. Bilateral Proptosis

Bilateral proptosis is most commonly associated with thyroid eye disease. Other important causes include craniofacial anomalies such as oxycephaly, cavernous sinus thrombosis, lymphoma, and orbital pseudotumor [3].

3. Intermittent Proptosis

Intermittent proptosis is usually caused by orbital varices and tends to become more prominent on bending forward or looking downward.

4. Pulsating Proptosis

Pulsating proptosis generally occurs due to carotico-cavernous fistula following trauma, resulting in abnormal communication between the carotid artery and cavernous sinus.

Clinical Features of Proptosis

Patients with proptosis commonly present with symptoms such as foreign body sensation, watering, ocular pain, diplopia, and diminished vision. Clinical examination may reveal axial or non-axial displacement of the globe, restricted ocular movements, lid retraction, conjunctival chemosis, corneal exposure, papilledema, or optic atrophy [1].

Investigations

Important investigations in the evaluation of proptosis include:

i. Hertel exophthalmometry
ii. Visual acuity assessment
iii. CT scan of the orbit
iv. MRI of the orbit and brain
v. Thyroid function tests
vi. Orbital ultrasonography
vii. Orbital biopsy when indicated

These investigations help identify the underlying cause and guide appropriate management.

Management of Proptosis

Management depends on the underlying aetiology.

Medical Treatment

Medical therapy may include corticosteroids for inflammatory orbital diseases, broad-spectrum intravenous antibiotics for orbital cellulitis, immunosuppressive agents in selected autoimmune conditions, and appropriate thyroid control in Graves orbitopathy.

Surgical Treatment

Surgical intervention may involve orbital decompression, drainage of orbital abscess, or excision of orbital tumors. Supportive eye care is essential and includes lubricating eye drops, ointments, and protective measures to prevent exposure keratopathy.

ORBITAL CELLULITIS

Orbital cellulitis is an acute bacterial infection involving tissues posterior to the orbital septum. It is considered both a sight-threatening and life-threatening emergency because of its potential for intracranial spread [1].

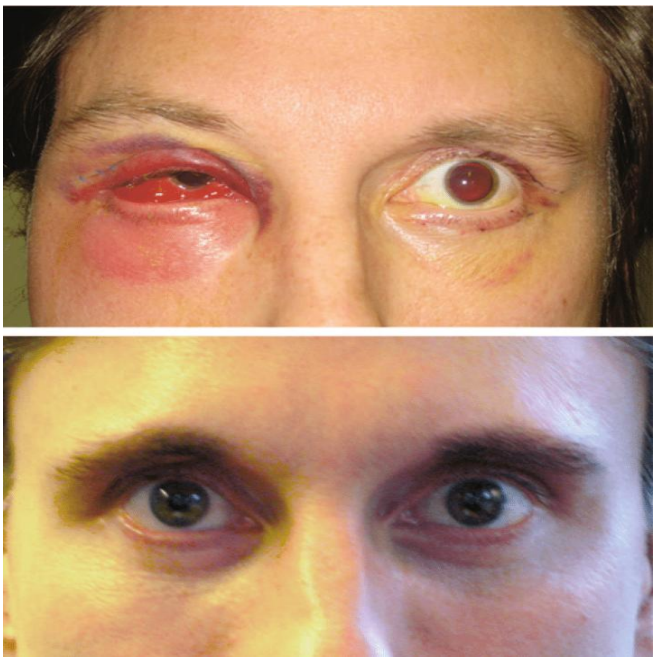


Figure 5: Orbital cellulitis showing eyelid edema, erythema, and proptosis. (Source: Clinical image of orbital infection, *Khurana A. K., Comprehensive Ophthalmology*)

Figure 5 illustrates Clinical presentation of orbital cellulitis showing painful eyelid swelling, erythema, proptosis, and restricted ocular movements due to acute post-septal orbital infection.

Aetiology

The infection most commonly spreads from adjacent sinusitis, particularly ethmoidal sinusitis. Other causes include trauma, orbital surgery, and spread from surrounding skin infections. Common causative organisms include, *Staphylococcus aureus*, *Streptococcus* species and *Haemophilus influenzae*.

Clinical Features of Orbital Cellulitis

Patients with orbital cellulitis usually present with painful swelling of the eyelids, fever, proptosis, restricted ocular movements, pain on eye movement, and reduced vision. In severe cases, optic nerve involvement and intracranial complications may occur [3].

Diagnosis and Treatment of Orbital Cellulitis

CT imaging of the orbit and paranasal sinuses plays a crucial role in confirming the diagnosis of orbital cellulitis and identifying associated complications such as subperiosteal or orbital abscess formation (Khurana, 2020). Early diagnosis is essential because orbital cellulitis is considered an ophthalmic emergency.

Management requires urgent hospitalization and prompt initiation of broad-spectrum intravenous antibiotics targeting common causative organisms. Surgical drainage is indicated when an orbital abscess develops or when there is inadequate response to medical therapy. Delay in treatment may result in serious complications including meningitis, brain abscess, cavernous sinus thrombosis, and permanent visual loss [2].

ENOPHTHALMOS

Enophthalmos is defined as posterior displacement of the eyeball within the orbit, producing a characteristic sunken appearance of the eye. It is essentially the opposite of proptosis and may result from abnormalities affecting the bony orbit or orbital soft tissues [3].

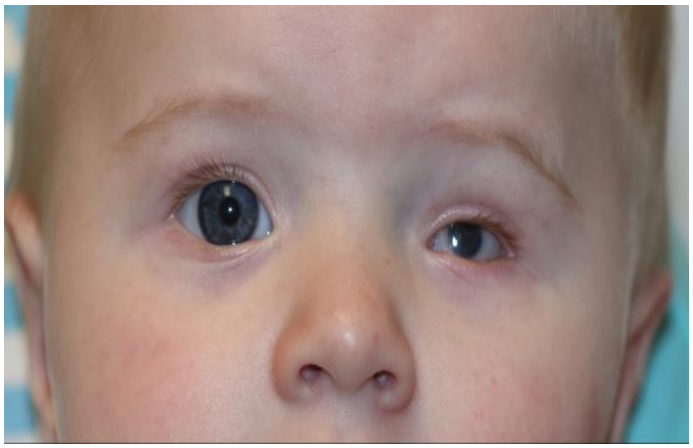


Figure 6: Enophthalmos showing posterior displacement of the globe. (Source: Orbital trauma and enophthalmos diagram, *Kanski's Clinical Ophthalmology*)

Figure 6 shows Clinical appearance of enophthalmos showing posterior displacement of the eyeball within the orbit, resulting in a sunken eye appearance.

Causes

Common causes of enophthalmos include:

A. Orbital blowout fractures
B. Loss of orbital fat
C. Horner syndrome
D. Chronic sinus disease
E. Orbital tumors
F. Congenital abnormalities

Management

Management depends on the underlying cause. Treatment may involve repair of orbital fractures, orbital reconstruction, and restoration of orbital volume using implants or fat grafting procedures. Appropriate management of associated pathology is essential to achieve both functional and cosmetic improvement.

ENDOCRINE ENOPHTHALMOS

Endocrine enophthalmos most commonly occurs in the late fibrotic stage of Graves disease. Progressive fibrosis of the extraocular muscles and atrophy of orbital fat lead to reduction in orbital volume and posterior displacement of the globe [1].



Figure 7: Endocrine enophthalmos in late Graves orbitopathy with sunken eye appearance. (Source: Thyroid eye disease orbital changes, *Parsons' Diseases of the Eye*)

Figure 7 shows clinical representation of endocrine enophthalmos showing posterior displacement of the globe associated with Graves orbitopathy in its fibrotic stage, with deep superior sulcus and sunken eye appearance. Patients may present with:

A. Sunken eyeballs
B. Deep superior sulcus
C. Diplopia
D. Restricted ocular motility

Management involves proper control of thyroid dysfunction along with orbital reconstructive procedures in cosmetically significant cases. Surgical correction may include orbital implants or fat grafting to restore orbital volume [2].

CAVERNOUS SINUS THROMBOSIS

Cavernous sinus thrombosis is a rare but potentially life-threatening condition caused by thrombosis within the cavernous sinus, usually secondary to infections of the face, orbit, or paranasal sinuses [3].

Clinical Features

Patients commonly present with:

a) Fever
b) Severe headache
c) Proptosis
d) Ophthalmoplegia
e) Cranial nerve palsies
f) Vision loss

MRI with MR venography is considered the preferred diagnostic investigation because it clearly demonstrates venous thrombosis and associated intracranial involvement. Management requires immediate intravenous broad-spectrum antibiotics and treatment of the primary source of infection. Early intervention is critical to reduce morbidity and mortality.

ANOPHTHALMOS

Anophthalmos refers to complete absence of the eyeball within the orbit. It may be classified as primary, secondary, or degenerative depending on the stage of embryological developmental failure [2]. Management includes:

1. Ocular prosthesis
2. Orbital implants
3. Socket reconstruction
4. Multidisciplinary rehabilitation and psychological support



Figure 8: Anophthalmos showing complete absence of the eyeball and underdeveloped orbit. (Source: Congenital orbital anomaly illustration, *Kanski J. J., Clinical Ophthalmology*)

Figure 8 illustrates Clinical representation of anophthalmos showing complete absence of the eyeball within the orbit, with associated underdeveloped orbital socket and facial asymmetry.

MICROPHTHALMOS

Microphthalmos is a congenital anomaly characterized by an abnormally small eyeball. It may occur as isolated simple microphthalmos or in association with structural abnormalities such as coloboma [1].



Figure 9: Microphthalmos showing abnormally small globe within the orbit. (Source: Congenital microphthalmos diagram, *Khurana A. K., Comprehensive Ophthalmology*)

Figure 9 illustrate clinical representation of microphthalmos showing an abnormally small eyeball within the orbit, often associated with reduced visual function and possible facial asymmetry.

Clinical manifestations include:

1. Poor vision
2. Small eye
3. Facial asymmetry
4. High hypermetropia

Management involves refractive correction, amblyopia therapy, treatment of associated ocular anomalies, and cosmetic rehabilitation when necessary.

ORBITAL HYPERTELORISM

Orbital hypertelorism is characterized by increased distance between the two orbits, resulting in widely spaced eyes. It is frequently associated with craniofacial syndromes such as Crouzon syndrome and Apert syndrome [3]. Severe cases may require surgical orbital repositioning to improve both cosmetic appearance and orbital alignment.



Figure 10: Orbital hypertelorism showing increased interorbital distance. (Source: Craniofacial developmental anomaly illustration, *Parsons' Diseases of the Eye*)

Figure 10 illustrate clinical representation of orbital hypertelorism showing increased interorbital distance with widely spaced eyes due to congenital craniofacial developmental anomaly.

HYPOTELORISM

Hypotelorism refers to decreased interorbital distance resulting in closely spaced eyes. It is commonly associated with holoprosencephaly and chromosomal abnormalities [2]. Management is mainly supportive and directed toward correction of associated craniofacial and neurological abnormalities.



Figure 11: Hypotelorism showing decreased interorbital distance. (Source: Craniofacial midline defect diagram, *Kanski's Clinical Ophthalmology*)

Figure 11 illustrates clinical representation of hypotelorism showing decreased interorbital distance with closely spaced eyes, typically associated with congenital midline craniofacial developmental anomalies.

CRANIOSYNOSTOSIS

Craniosynostosis involves premature fusion of cranial sutures leading to abnormal skull and orbital development. Shallow orbits may produce proptosis and optic nerve compression, potentially resulting in visual impairment [1]. Early surgical correction remains the cornerstone of treatment to prevent raised intracranial pressure and progressive craniofacial deformity.

Orbital Coloboma

Orbital coloboma is a congenital defect caused by failure of closure of the embryonic fissure. Eyelid coloboma is the most common type and may lead to exposure keratitis and corneal ulceration [3]. Surgical reconstruction is usually required to protect the ocular surface and improve eyelid function.

Dermoid Cyst

Dermoid cyst is a benign congenital choristoma resulting from sequestration of ectodermal tissue along embryonic suture lines. It commonly presents as a painless superotemporal orbital swelling [2]. Complete surgical excision without rupture is considered the treatment of choice.



Figure 12: Dermoid cyst showing superotemporal orbital swelling due to congenital choristoma. (Source: Orbital dermoid cyst illustration, *Khurana A. K., Comprehensive Ophthalmology*)

Figure 12 shows clinical representation of a dermoid cyst showing a painless, slow-growing, well-defined superotemporal orbital swelling due to a congenital choristoma arising from ectodermal tissue sequestration.

ENCEPHALOCELE AND MENINGOCELE

Encephalocele and meningocele involve herniation of brain tissue or meninges through congenital skull defects into the orbit. Patients may present with orbital swelling, proptosis, and associated neurological abnormalities [1]. Management requires neurosurgical repair and multidisciplinary care.



Figure 13: Encephalocele and meningocele with orbital extension through skull defect. (Source: Neuro-orbital developmental anomaly diagram, *Parsons' Diseases of the Eye*)

Figure 13 shows clinical representation of encephalocele and meningocele showing a congenital orbital mass due to herniation of brain tissue (encephalocele) or meninges (meningocele) through a skull defect, often associated with pulsation and progressive proptosis.

CONGENITAL ORBITAL CYSTS

Congenital orbital cysts include dermoid cysts, epidermoid cysts, conjunctival cysts, and microphthalmos-associated cysts. Patients commonly present with painless orbital swelling and proptosis. Imaging studies such as CT scan and MRI are important for determining lesion extent and detecting associated intracranial involvement. Treatment depends on the type and severity of the cystic lesion [3].



Figure 14: Congenital orbital cysts showing cystic orbital mass present since birth. (Source: Orbital cyst classification illustration, *Kanski J. J., Clinical Ophthalmology*)

Figure 14 shows clinical representation of congenital orbital cysts showing a cystic orbital swelling present since birth, which may be associated with proptosis and restricted ocular movements depending on size and location of the lesion.

CRANIOSYNOSTOSIS

Craniosynostosis refers to the premature fusion of one or more cranial sutures, resulting in abnormal skull development and altered orbital morphology, with possible restriction of brain growth. Craniosynostosis may be:

a) Primary (idiopathic)
b) Secondary, associated with systemic or metabolic disorders

Syndromic forms affecting the orbit include:

A. Crouzon syndrome
B. Apert syndrome
C. Pfeiffer syndrome

Premature fusion of cranial sutures leads to restricted skull growth perpendicular to the fused suture. This results in compensatory overgrowth at remaining patent sutures, producing abnormal skull shape and orbital deformity. In severe cases, raised intracranial pressure may develop. Premature suture fusion:

i. Restricted cranial growth
ii. Compensatory skull expansion elsewhere
iii. Abnormal skull shape and orbital changes
iv. Possible increased intracranial pressure (in severe cases)

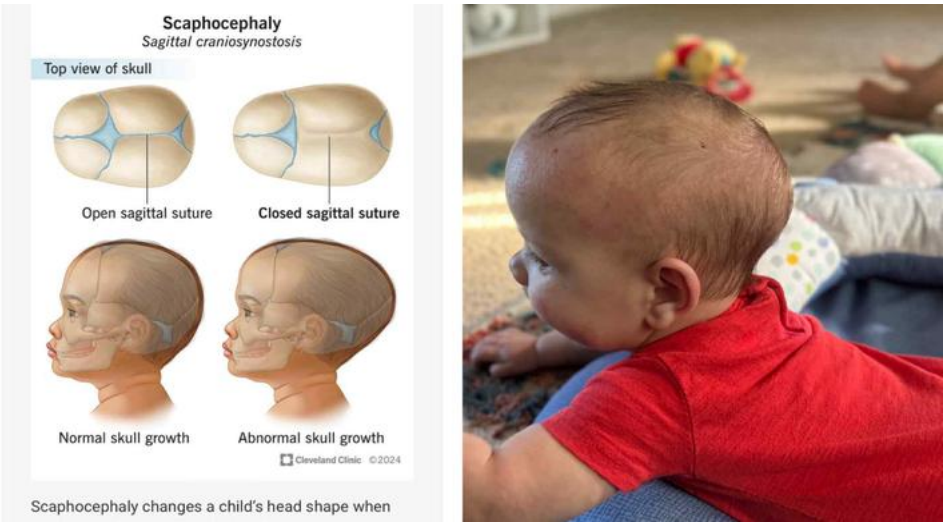


Figure 15: Craniosynostosis showing abnormal skull shape with orbital involvement. (Source: Craniofacial syndrome diagram, *Khurana A. K., Comprehensive Ophthalmology*)

Figure 15 shows clinical representation of craniosynostosis showing premature fusion of cranial sutures leading to abnormal skull shape, shallow orbits, and associated orbital deformities such as proptosis or hypertelorism depending on the type of suture involved.

CONCLUSION

Orbital diseases encompass a wide and diverse spectrum of disorders ranging from inflammatory and infectious conditions to endocrine, vascular, neoplastic, traumatic, and congenital abnormalities. Because the orbit contains critical ocular and neurovascular structures, even minor pathology can significantly affect vision, ocular motility, and facial appearance. In severe cases, orbital diseases may extend intracranially and become life-threatening, making early recognition and timely intervention essential.

Recent advances in orbital imaging modalities, medical therapies, and reconstructive surgical techniques have greatly improved diagnostic accuracy and treatment outcomes. However, optimal management still relies on a careful clinical evaluation supported by appropriate investigations and individualized treatment planning.

A multidisciplinary approach involving ophthalmologists, radiologists, neurologists, endocrinologists, and neurosurgeons is often required to ensure comprehensive care. Such coordinated management is crucial for preserving vision, preventing complications, and achieving the best functional and cosmetic outcomes in patients with orbital disease.

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