
Basic Concepts in Orbital Examination

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Abstract

Orbital disease usually arises within the orbit or spreads from adjacent structures. The aetiology can be inflammatory, vascular or neoplastic, either benign or malignant. To reach a correct diagnosis, a thorough history is essential, followed by a proper clinical examination, supported by investigations to evaluate the pathophysiology changes, and imaging techniques to define the lesions, allowing for the correct diagnosis and treatment of diseases that in some cases can be life- or sight-threatening.

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Orbital disease usually arises within the orbit from the bone, muscles, nerves, blood vessels or connective tissue, or spreads from adjacent structures like the paranasal sinuses. The aetiology can be inflammatory due to vascular anomalies or neoplastic lesions, and either benign or malignant primary or metastatic tumours. Thus, orbital disease may be due to serious and sometimes life-threatening entities.

Evaluation of a patient with orbital disease should seek to glean information regarding pathophysiology and location. In such cases, we have to deal with disorders than cannot be readily visualised using either biomicroscopy or fundoscopy. Therefore, to reach a correct diagnosis, a thorough history is essential, followed by proper clinical examination, and finally validated by investigations that evaluate pathophysiology changes and imaging techniques that can define the lesions in terms of location, characteristics and relationship to adjacent structures.

History taking and physical examination provide an opportunity to define whether the disease process is characterised by features of inflammation, infiltration, mass



Fig. 1. Right eye proptosis, basal view. As the orbital walls are not flexible, any increase in the orbital volume behind the globe (intraconal space) is compensated by an anterior displacement of the eyeball. This forward displacement of the eye is known as proptosis. The term 'exophthalmos' is also used to describe the proptosis and bulging eyes so frequent in patients with thyroid-associated orbitopathy.

effect or vascular change, and it should define the location of the disorder. The changes should be validated psychophysical, motor and sensory tests, and then by imaging techniques.

History Taking

Taking a patient's medical history helps elucidate the dynamics of the disease; patients should be questioned closely on the nature, intensity, location, radiation and duration of the symptoms. Factors that relieve or exacerbate them should be sought.

Symptoms

The symptoms of orbital disease can be structural, sensory, motor, psychophysical and vascular in nature.

With structural symptoms, the treating physician should be aware of globe displacement, lid fullness or the presence of masses, and should look for proptosis, pseudoproptosis, pulsating proptosis and intermittent proptosis lid malposition.

- *Proptosis* is the hallmark of orbital disease and implies an axial protrusion of the globe from the socket (fig. 1). It is helpful to determine if the globe is displaced in another direction because a lesion presenting in one quadrant of the orbit will frequently displace the globe into the opposite quadrant, as well as produce a degree of proptosis. Swelling or a mass within the muscle cone displaces the globe directly anteriorly (axial proptosis), whereas a mass outside the muscle cone will also cause sideways or vertical displacement (non-axial proptosis; fig. 2). Proptosis does not impair vision unless there are corneal changes due to exposure.



Fig. 2. Left eye hypoglobo. The eye can not only be displaced anteriorly, but also vertically and horizontally in the presence of a mass outside the muscle cone or in the presence of an orbital floor defect. The line between the two pupillary reflexes is tilted, showing a lower location of the reflex on the left affected eye demonstrating the hypoglobo.



Fig. 3. Right eye pseudoproptosis. Pathological myopia is a common cause of pseudoproptosis. In these patients, the prominence of the eye from the bony orbit is not the result of an increased orbital content. Nonetheless, a thorough study should be carried out to rule out the presence of a mass before setting this diagnosis.

- *Pseudoproptosis* is apparent proptosis in the absence of orbital disease. It may be due to high myopia (fig. 3), buphthalmos or lid retraction, and can occur if the pathologic opposite side is enophthalmic (fig. 4), which may cause confusion. The most frequent cause of enophthalmos is a blowout fracture, but metastatic breast carcinoma can also cause enophthalmos.

Fig. 4. Left enophthalmos due to orbital fracture. **a** Clinical photograph of a male patient 2 months after blunt trauma to his left eye with a ball. The decreased orbital volume is evident with the deep superior sulcus and the secondary ptosis. **b** Hertel exophthalmometry measured 8 mm of enophthalmos in the left eye, and the basal view corresponds with it. **c** CT scan showing a fracture and displacement of both the inferior and medial orbital walls with the secondary expansion of the bony orbit responsible for the enophthalmos.

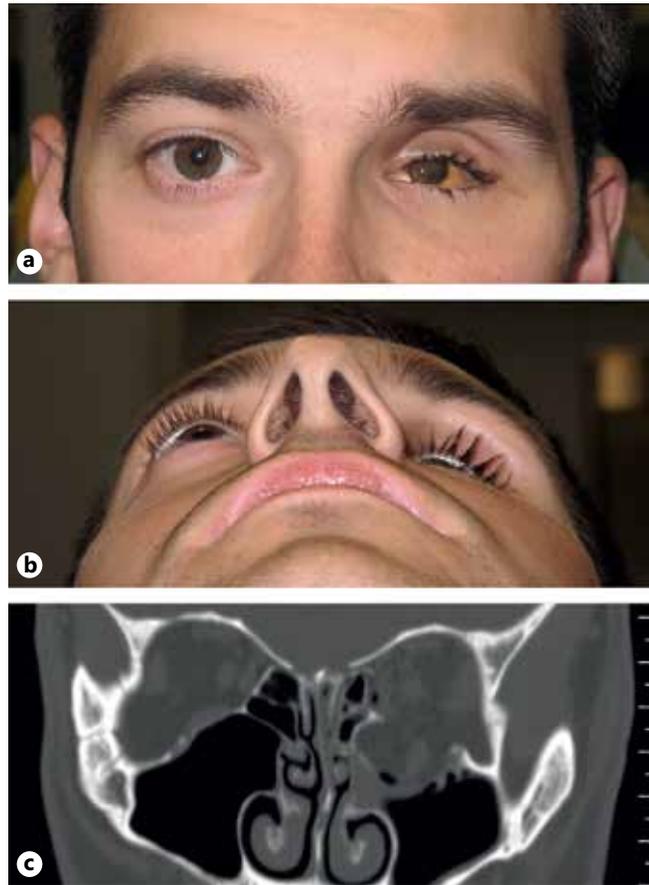


Fig. 5. Clinical photographs of a patient with left carotid-cavernous fistula: frontal view (**a**) and basal view (**b**). Typical signs include unilateral red eye with dilatation of the episcleral veins (Medusa head) and proptosis, associated with diplopia, decreased vision and pain. In acute traumatic fistulas, the proptosis can be pulsating.



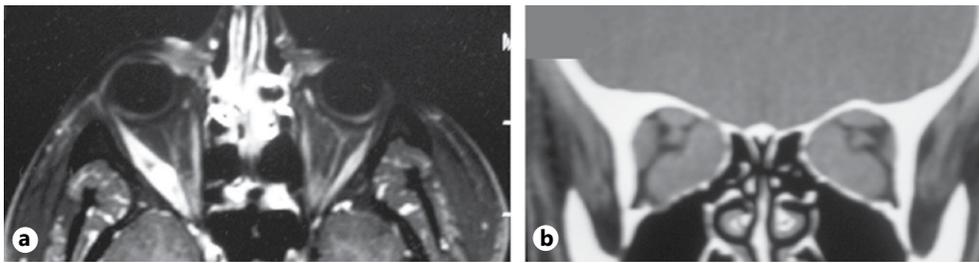


Fig. 6. Causes of diplopia. A well-delimited mass within the muscle (a) or diffuse extraocular muscle enlargement due to thyroid-related orbitopathy (b) can cause diplopia due to the muscle function restriction.

- *Pulsating proptosis* may be due to carotid-cavernous fistula (fig. 5), arterial orbital vascular malformation or transmission of cerebral pulsations due to a defect of the superior orbital roof.
- *Intermittent proptosis* or a proptosis that increases on bending the head forward or with Valsalva's manoeuvre is a sign of orbital vascular malformations (orbital varices, carotid-cavernous fistulae and lymphangiomas) or may be the result of a sinus mucocele or meningoencephalocele.
- *Lid malposition* may be associated with orbital masses in the anterior extraconal space, such as lacrimal gland masses causing lateral ptosis of the upper eyelid. Superior orbital tumours may produce upper eyelid ptosis due to interference with the normal function of the levator muscle. Lid retraction of the upper and lower eyelids is one hallmark of Graves' orbital disease.

Sensory symptoms involve *pain and/or modification in sensation*. Patients with orbital disorders frequently complain of pain or discomfort on the affected side, which may range from very mild to very severe. The quality of the pain is an important characteristic. Burning, scratching and foreign body sensation are frequently described in patients presenting with Graves' disease, and are often related to the desiccation of the cornea and conjunctiva. Patients with inflammatory lesions, whether benign or malignant, frequently complain of an aching, throbbing or boring pain that resides behind the eye and may radiate into the forehead, cheek or temporal areas. Dysesthesias, in the distribution of the supraorbital nerve or the infraorbital nerve, are encountered following orbital trauma, especially during the recovery period while the initial post-traumatic hypoesthesia is resolving. Pain may occur as a result of rapid expansion, inflammation or infiltration of sensory nerves.

Motor symptoms include diplopia, pain and tightness. *Diplopia* is a common symptom in orbital disorders, related to a paralysis of the extraocular muscles (lesions residing in the cavernous sinus or posterior orbit) or a restriction of ocular movement (lesions immediately adjacent to the extraocular muscles or diseases that involve the muscle tissue; fig. 6). More anterior orbital lesions typically produce

Fig. 7. Patient presenting with all of the vascular signs on her right eye: eyelid swelling, redness, hyperaemia and epibulbar vascular dilatation.



diplopia by means of a mechanical effect. *Pain*, especially gaze evoked, is also associated with motor symptoms. Blowout fractures and myositis are often associated with pain on ocular movement, and *tightness* might reflect a cicatricial or restrictive component.

Psychophysical symptoms involve a *decrease in visual acuity*. Loss of visual acuity usually implies involvement of the optic nerve, either by compression, infiltration, vascular compromise or inflammation, but it can also be explained by choroidal folds, keratopathy or secondary glaucoma. Additionally, signs of *optic nerve dysfunction*, *blurred vision*, *loss of colour vision* and *visual field cuts* may be identified.

Finally, vascular symptoms include swelling, redness, hyperaemia and epibulbar vascular dilatation (fig. 7).

Onset and Chronicity: 'When Did It Start?'

The time of onset of the orbital condition is important. While some patients may be aware of the displacement related to acute inflammation or a painful process, in some only relatives or friends will have noted the displacement, mostly if the onset of the proptosis was very gradual when related to slow-growing benign lesions. Old photographs may be helpful in establishing the duration of displacement.

In the case of pain or diplopia, patients are usually accurate and specific about the time of onset. The practitioner should try to get a sense of whether the orbital symptom has been progressive or stable since its onset.

Intermittency

Symptoms like pain or diplopia can be permanent or have an intermittent pattern. Asking about what makes the pain worse is especially useful in patients presenting with double vision, as diplopia is often worse in specific fields of gaze. The Valsalva manoeuvre may worsen the proptosis in vascular lesions (fig. 8). Lymphangiomas and orbital varix typically enlarge during upper respiratory tract infections.