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Invited Review

Orbital Compartment Syndrome

Burak Turgut,[1] Feyza Calis Karanfil,[2] Fatos Altun Turgut[2]

[1]Department of Ophthalmology, Çanakkale Onsekiz Mart University, Canakkale, Turkey
[2]Department of Ophthalmology, Yuksek Ihtisas University, Ankara, Turkey
[3]Department of Nuclear Medicine, Elazig Training and Research Hospital, Elazig, Turkey

Abstract

Orbital compartment syndrome (OCS) is a true ophthalmological emergency. It develops as a result of an acute rise in intra-orbital pressure, and if not treated immediately, damage to the optic disc and retina will lead to irreversible vision loss. Thus, immediate diagnosis and management are vital to preserve vision. Presented here is a brief summary of OCS in order to call attention to this condition.

Keywords: Cantholysis, canthotomy, ocular compression, orbital compartment, true emergency.

Introduction

True ophthalmic emergencies include acute central retinal artery occlusion (CRAO), endophthalmitis, arteritic anterior ischemic optic neuropathy, acute retinal detachment, severe orbital cellulitis, acute angle closure glaucoma, alkali chemical ocular injury, infectious keratitis, penetrating globe trauma, ocular perforation due to severe corneal thinning or descemetocele, acute third cranial nerve paralysis, and orbital compartment syndrome (OCS) (1–4).

Although OCS is an uncommon ophthalmic emergency, learning the findings, signs, and the emergency management of OCS is essential for all ophthalmologists and emergency physicians in order to prevent severe and irreversible vision loss. OCS is characterized by an acute or subacute rise in intra-orbital pressure (INOP) and if it is not treated immediately, permanent loss of vision may develop due to damage to ocular and other intra-orbital structures (1, 2, 5). Thus, early diagnosis of OCS should be performed clinically without the delay of obtaining orbital imaging. A lateral canthotomy and inferior cantholysis (LCIC) should be performed to provide emergent orbital decompression. Decompression surgery to the bone or an orbital septum incision may be necessary in unresponsive cases (5).

Causes of OCS: OCS primarily occurs due to hemorrhage, abscess, tumor, orbital edema or emphysema, orbital cellulitis, retrobulbar injection, or preexisting medical disorders (5). However, it may also result from acute orbital inflammation or an allergic reaction following any peribulbar drug injection, prolonged hypoxemia with a capillary leak, an intra-orbital foreign body, extravasation of contrast dye used in the catheterization of the middle meningeal artery in the orbit, the injection of a hydraulic solution, or retained foreign material, such as bacitracin ointment or oxidized regenerated cellulose, especially in cases of sinus surgery (5, 6).

Acute or subacute orbital/retrobulbar hemorrhage (quickly compromising ocular perfusion) may by caused by various conditions, including direct facial trauma or indirect orbital trauma due to severe sneezing, coughing, or the Valsalva maneuver; maternal labor; barotrauma; surgery related to the orbita using orbital, retrobulbar, or peribulbar injections; periocular surgery; extra-ophthalmic surgery, such as sinus surgery, facial trauma surgery, orthognathic, or neurosurgical surgery of the anterior or middle cranial fossae; or previous diseases, such as an aneurysm of the ophthalmic artery, venous and lymphatic anomalies, orbital myositis, or chronic sinusitis (7, 8). Hematological disorders,

Address for correspondence: Feyza Calis Karanfil, MD. Yuksek Ihtisas Universitesi, Oftalmoloji Anabilim Dali, Ankara, Turkey
Phone: +90 530 433 30 71 **E-mail:** feyzacalis@gmail.com

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anticoagulant medications (e.g., aspirin), nonsteroidal anti-inflammatory agents, warfarin, and clopidogrel may predispose the patient to retrobulbar hemorrhage and eventually OCS (8–10). The expanding tissues seen in Graves' disease and rapidly enlarging tumors involving the orbit may also lead to subacute OCS. Excessive fluid accumulation following an eyelid burn injury or excessive blood loss may also be a predisposing factor for OCS. However, a posttraumatic or postsurgical retrobulbar hemorrhage is the most common cause of OCS (5, 8).

The main risk factors for OCS are trauma; coagulopathy; Graves' disease; the use of nonsteroidal anti-inflammatory drugs, antiplatelet medications, anticoagulant drugs, thrombolytic drugs, or corticosteroids; and excessive intravenous fluids or blood products (5–10).

Clinical diagnosis of OCS: Resistance to retropulsion of the globe, a tight orbit, and tense eyelids are the most important indicators of increased INOP (11). If a patient has either acute or onset decreased vision, diplopia, ocular pain, periorbital edema, proptosis, limited ocular movements, a fixed dilated pupil or an afferent pupillary defect, increasing intraocular pressure (IOP), ocular tenderness, ecchymosis, or subconjunctival hemorrhage, OCS should be suspected, especially in patients with previous sinusitis or related surgery, trauma, or retrobulbar injection (5, 11). If ophthalmoscopy can be performed, it may reveal optic disc and/or retinal edema, retinal venous congestion, pulsation in the central retinal artery or CRAO. Additionally, the presence of periorbital crepitus should provoke the suspicion of orbital emphysema, which is a traumatic cause of OCS (5).

Pathophysiological mechanisms of OCS: The orbit has limited capability to expand, as it is an enclosed space restricted by the orbital septum, orbital rim, eyelids, and 4 walls formed by bone. The normal orbital volume is about 30 mL and normal INOP is <20 mmHg (usually 3-6 mmHg) (4, 5). Small but acute elevations in INOP are compensated for with partial forward movement of the globe. When the INOP exceeds the pressure in the central retinal artery and ophthalmic artery, blood flow in the vessels stops, causing ischemia of the retina, optic disc, and other ocular tissues, and eventually irreversible vision loss. It has been demonstrated that increased INOP lasting 60 to 100 minutes can cause permanent vision loss. If the IOP increases to >40 mmHg, an urgent LCIC should be performed (4, 5, 11–13). In OCS, vision loss may occur due to ischemia from one or more causes, including CRAO, direct traumatic or compressive optic neuropathy, compression of the optic nerve vasculature and vasa vasorum, or ischemic optic neuropathy (11–13).

Emergency management of OCS: The initial and emergency management of OCS includes an LCIC procedure. LCIC should be performed immediately to provide orbital decompression, even in a bedside procedure, if necessary. The main indication for urgent LCIC is retrobulbar hemorrhage with consequential symptoms, such as proptosis, elevated IOP, decreased visual acuity, an afferent pupillary defect, or restricted extraocular movements (14).

The anatomical junctional ligaments of the upper and lower eyelids in both the medial and lateral terminals of the palpebral fissure form the canthi. The superior and inferior tarsal plates extend to the lateral terminal branch and are secured by the lateral canthal tendon (LCT). Immediately posterior to the orbital bone rim, the LCT enters Whitnall's tubercle and separates the inferior tarsal plate from the lateral bony orbit (4, 14).

Lateral canthotomy: Although this surgical intervention does not provide for a decrease in INOP, it exposes the LCT and enables the application of an inferior cantholysis. Due to its distant and lateral location from the lacrimal system, the LCT is a safe anatomical area for urgent orbital decompression. To perform a lateral canthotomy, 1 to 2 cc of a local anesthetic (1% lidocaine with 1/100.000 adrenaline) is injected into the LCT (Fig. 1A). A tissue clamp is then applied to the region for 30 seconds to 1 minute to achieve devascularization and hemostasis (Fig. 1B) (4, 5, 14). The area around the lateral canthus is irrigated with saline or chlorhexidine, and then draped. Sterile scissors are carefully inserted in the lateral palpebral artery along the internal face of the lateral canthus. The incision of the skin and underlying eyelid tissue should be approximately 1 cm in length and extend to the lateral bony orbital rim (Fig. 1C). Maximum care and attention must be given while directing the scissors laterally and superficially to avoid iatrogenic injury to the globe. A lateral canthotomy achieves separation of the skin, fascial septum, orbicularis oculi muscle, and conjunctiva, and exposes orbital fat tissue. Although the LCT can be easily identified, a lateral canthotomy cannot accomplish a significant increase in the laxity of the eyelid (4, 14).

A mnemonic "one is the number" has been proposed by some authors to highlight the important steps in a lateral canthotomy since it is an uncommon emergent procedure:

- 1 cc of 1% lidocaine with epinephrine for local anesthesia
- 1 minute hemostasis time
- 1 cm incision length (14).

Inferior cantholysis: In this surgical step of an LCIC, following the exposure of the orbital rim with a lateral canthotomy, the LCT is identified with palpation around the point of inferior insertion. The use of a metal instrument may make the identification of the crus of the LCT easier. The inferior crus of the LCT is isolated with inferior retraction of the lower lid. The tips of the scissors should be directed away from the globe. Anterior traction is placed on the free lateral edge of the lower lid, and the inferior crus of

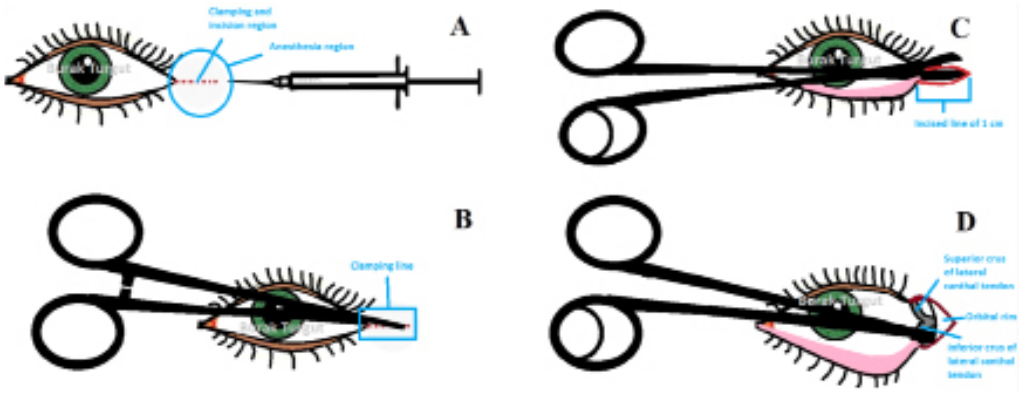

Figure 1. (a) The stages of the lateral canthotomy and inferior cantholysis procedure. Local anesthesia, **(b)** clamping and hemostasis, **(c)** incision of the lateral canthus, **(d)** cutting the inferior crus of the lateral canthal tendon.

the LCT can be identified as a taut band. Next, the inferior crus of the LCT is cut (Fig. 1D). The fibrous tarsal plate of the lower lid relaxes. This step allows for a significant decrease in intra-orbital volume and eventually decreases the INOP. The result is a completely mobile lower lid. Usually, the incision site will heal spontaneously and there is no need for suturing. However, if an oculoplastic or cosmetic deformity occurs in the lower lid, further surgical repair may be needed (14, 15) (Fig. 1).

The efficacy of LCIC: Significant relief of symptoms and a reduction of IOP typically occurs within several minutes of a successful LCIC; there is improvement in proptosis and movement of the extraocular muscles. The afferent pupillary defect should disappear due to the normalization of ocular circulation. A lateral canthotomy alone can achieve an INOP reduction of approximately 14 mmHg, while an inferior cantholysis alone can reduce pressure by approximately 19 mmHg. The LCIC procedure can attain a pressure reduction of about 30 mmHg (4, 5, 14, 15).

Failure of an LCIC: In the event of incomplete dissection and inferior cantholysis, the surgeon should examine the conditions and may elect to approach the inferior crus again. Even in cases of a completed inferior cantholysis, if there is no improvement in the OCS, the superior limb/crus of the LCT should be cut from the superior and lateral direction while again taking care to keep the instrument away from the globe and the lacrimal gland. Successful severing of the superior crus should provide significant laxity in both eyelids, as well as ocular mobility (14, 15). Both eyelids are disconnected from their lateral attachments to the bony orbit, and maximum decompression of the orbit is achieved (5, 14). If there is no relief in the elevated INOP despite all these interventions, urgent referral to an orbital surgeon for extensive orbital bone decompression is needed (4, 14, 15).

The importance of radiological findings: Although further investigation is not essential for the diagnosis of OCS, computed tomography (CT) and magnetic resonance imaging are often performed to confirm the diagnosis or to follow up. The radiological finding of globe tenting is the most common sign associated with acute proptosis, as seen in OCS. The size may contribute to the estimation of visual prognosis. It has been demonstrated that a posterior globe angle of fewer than 120° with acute proptosis observed on a CT scan requires urgent orbital decompression and is associated with a poorer prognosis and a greater risk of permanent vision loss (4, 5).

Medical management of OCS: Medical treatment of the underlying cause of OCS should be initiated during the emergent LCIC. Osmotic agents, carbonic anhydrase inhibitors, and aqueous suppressants may be used to assist in the reduction of INOP. Systemic antibiotics should be administered if an infective etiology is considered. The effectiveness of systemic corticosteroids in OCS is controversial. Intravenous or oral corticosteroids may be used to provide neuroprotection if an inflammatory cause is responsible (4, 5, 15).

OCS patients should be warned to avoid coughing/straining or taking antitussives, antiemetics, and laxatives. The head of the bed should be elevated to 45°. Cold/ice compression may reduce periorbital/orbital edema. Blood pressure and coagulopathies should be normalized. All cases of OCS should be closely monitored for progression or recurrence and patients should be advised to seek care at a hospital immediately if severe pain, proptosis, or blurry vision reoccur (2–5, 15).

Conclusion

OCS is an uncommon but vision-threating ophthalmic emergency. Knowing the signs and symptoms of OCS and the appropriate management is necessary to prevent permanent vision loss. All ophthalmologists and emergency physicians should keep OCS in mind with patients who have risk factors for this entity and should know the LCIC technique,

as this procedure may need to be performed rapidly in the emergency department or at bedside.

Disclosures

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References

1. Tarff A, Behrens A. Ocular Emergencies: Red Eye. Med Clin North Am 2017;101:615–39. [CrossRef]
2. Khare GD, Symons RC, Do DV. Common ophthalmic emergencies. Int J Clin Pract 2008;62:1776–84. [CrossRef]
3. Hodge C, Lawless M. Ocular emergencies. Aust Fam Physician 2008;37:506–9.
4. Cheung CA, Rogers-Martel M, Golas L, Chepurny A, Martel JB, Martel JR. Hospital-based ocular emergencies: epidemiology, treatment, and visual outcomes. Am J Emerg Med 2014;32:221–4.
5. Lima V, Burt B, Leibovitch I, Prabhakaran V, Goldberg RA, Selva D. Orbital compartment syndrome: the ophthalmic surgical emergency. Surv Ophthalmol 2009;54:441–9. [CrossRef]
6. Tiong KI, Aziz S, Hazlita I. Orbital compartment syndrome in idiopathic orbital inflammatory disease: A case report. Med J Malaysia 2015;70:316–7.
7. Sun MT, Chan WO, Selva D. Traumatic orbital compartment syndrome: importance of the lateral canthomy and cantholysis. Emerg Med Australas 2014;26:274–8. [CrossRef]
8. Kloss BT, Patel R. Orbital compartment syndrome from retrobulbar hemorrhage. Int J Emerg Med 2010;3:521–2. [CrossRef]
9. Leibovitch I, Casson R, Laforest C, Selva D. Ischemic orbital compartment syndrome as a complication of spinal surgery in the prone position. Ophthalmology. 2006;113:105–8. [CrossRef]
10. Sokol JA, Baron E, Lantos G, Kazim M. Orbital compression syndrome in sickle cell disease. Ophthalmic Plast Reconstr Surg 2008;24:181–4. [CrossRef]
11. Dalley RW, Robertson WD, Rootman J. Globe tenting: a sign of increased orbital tension. Am J Neuroradiol 1989;10:181–6.
12. Hayreh SS, Weingeist TA. Experimental occlusion of the central artery of the retina. IV: Retinal tolerance time to acute ischaemia. Br J Ophthalmol 1980;64:818–25. [CrossRef]
13. Hayreh SS, Kolder WE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology 1980;87:75–8.
14. Rowh AD, Ufberg JW, Chan TC, Vilke GM, Harrigan RA. Lateral canthotomy and cantholysis: emergency management of orbital compartment syndrome. J Emerg Med 2015;48:325–30.
15. Samples JR, Hedges JR. Ophthalmologic procedures. In: Roberts JR, Hedges JR, editors. Clinical procedures in emergency medicine. 3rd ed. Philadelphia: W.B. Saunders Co; 1998. p. 1089–119.