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Invited Review

Orbital Compartment Syndrome

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Abstract

Orbital compartment syndrome (OCS) is one of a few true ophthalmologic emergencies. It develops due to acute intra-orbital pressure rising and if not immediately treated, the irreversible visual loss is unavoidable due to the damage to optic disc and retina. Thus, the immediate diagnosis and management are vital for vision. In this review, we aimed to summarize the OCS and to call attention to it.

Keywords: Canthotomy, cantholysis, orbital compartment, ocular compression, 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 finally orbital compartment syndrome (OCS) (1-4).

Although OCS is an uncommon ophthalmic emergency, learning the findings, signs, and the emergency management of the OCS is essential and critical for the prevention of severe and irreversible vision loss for all ophthalmologists and emergency physicians. OCS is characterized by acute or subacute intra-orbital pressure (INOP) rising and if it is not treated immediately, the irreversible visual loss may be developed due to damage to ocular and other intra-orbital structures (1, 2, 5). Thus, the early diagnosis of the OCS should be performed clinically without any orbital imaging, and emergent orbital decompression with the procedure of lateral canthotomy and inferior cantholysis (LCIC) should be created. However, the decompression surgery to the bones or orbital septum incision may be necessary in unresponsive cases (5).

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

Acute or subacute orbital/retrobulbar hemorrhage (fast compromising the ocular perfusion) may be derived from various conditions including direct facial trauma or indirect orbital trauma due to severe sneezing, coughing, Valsalva maneuver, labor, and barotrauma, surgery related to orbita or orbital, retrobulbar or, peribulbar injections, periocular surgeries, extra-ophthalmic surgeries such as sinus surgery, facial trauma surgery, orthognathic, neurosurgical surgery of the anterior or middle cranial fossae, previous diseases such as

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aneurysm of ophthalmic artery, venous and lymphatic anomalies, orbital myositis and chronic sinusitis (7, 8). Hematologic disorders, anticoagulant medications (e.g., Aspirin), nonsteroidal anti-inflammatory agents, warfarin, and clopidogrel may predispose the retrobulbar hemorrhage and eventually OCS (8-10). On the other hand, the expanding tissues as seen in Graves and rapid enlarging tumors involving the orbit may be the reason of the subacute OCS. Eyelid burns may also be predisposing the OCS through excessive fluid accumulation following burn injury or excessive blood loss. However, a posttraumatic or postsurgical retrobulbar hemorrhage is the most common cause of the OCS (5, 8).

The main risk factors for OCS include trauma, coagulopathy, Grave's disease, the usage of NSAIDs, antiplatelet medications, anticoagulant drugs, thrombolytic drugs, and corticosteroids, excessive intravenous (iv) fluids or blood products (5-10).

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

Pathophysiologic mechanisms of the OCS: The orbit has limited expanding capability as it is an enclosed space limited by the orbital septum, orbital rim, eyelids and four walls formed by bones. The normal orbital volume is about 30 mL and normal INOP is under 20 mmHg (usually between 3-6 mm Hg) (4, 5). The acute but small elevations in INOP is compensated by partially forward movement of the globe. When INOP exceeds the pressure in the central retinal artery and ophthalmic artery, blood flow in vessels stops, causing the ischemia of retina, optic disc and other ocular tissues, and eventually irreversible vision loss. It has been demonstrated that the increased INOP lasting 60-100 minutes causes the permanent visual loss. If IOP increases over 40 mmHg, urgent LCIC should be performed. (4, 5, 11-13). In OCS, visual loss occurs due to ischemia from one or more causes including CRAO, direct traumatic or compressive optic neuropathy, the compression of optic nerve vasculature and vasa vasorum and ischemic optic neuropathy (11-13).

Emergency management of OCS: The initial and emergency management of OCS include LCIC procedure. LCIC should be applied immediately to orbital decompression even if the patient is in the bedside. The main indication for urgent LCIC is the retrobulbar hemorrhage including consequential symptoms like proptosis, elevated IOP, decreased visual acuity, an afferent pupillary defect, and restricted extraocular movements (14).

The anatomic junctional ligaments of the upper and lower eyelids in both medial and lateral terminals of the palpebral fissure creates both canthi. The superior and inferior tarsal plates accompany to the lateral terminal and create the lateral canthal tendon (LCT). LCT joins immediate posterior to the orbital bone rim at the region of Whitnall's tubercle and separates the inferior tarsal plate from the lateral bony orbit (4, 14).

Lateral canthotomy: Although this surgical intervention itself does not allow to decrease INOP, it exposes the LCT and enables to inferior cantholysis. Due to its distant and lateral location from lacrimal system, LCT is a safe anatomic area for performing urgent orbital decompression. When performing the lateral canthotomy, amount in 1-2 cc of a local anesthetic (1% lidocaine with 1/100.000 adrenaline) into the skin on LCT is injected (Fig. 1a). Then, a tissue clamp is applied on this region for a half to one minute to crush providing the hemostatis of the area (Fig. 1b) (4, 5, 14). The area around the lateral canthus is cleaned using the irrigation with saline or chlorhexidine, and then draped. Sterile scissors are inserted carefully in the lateral palpebral terminal along the internal face of the lateral canthus. The incision on the skin and underlying eyelid tissue should be approximately 1 cm long and be extended to the lateral bony orbital rim (Fig. 1c). However, a maxımum attention must be given while directing the scissors laterally and superficially to avoid iatrogenic injury to globe. Lateral canthotomy provides dividing of skin, fascial septum, orbicularis oculi muscle, and conjunctiva, presenting orbital fat tissue. Although LCT can be easily identified, lateral canthotomy cannot achieve a significant increase in the laxity of the eyelid (4,14).

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

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

Inferior cantholysis: In this surgical step of LCIC, following the exposure of orbital rim with lateral canthotomy, LCT is identified with palpation around its inferior insertion. The identification of the cruse of LCT may be easier with the usage of a metal instrument. The inferior crus of the LCT is isolated with inferior retraction of the lower lid and

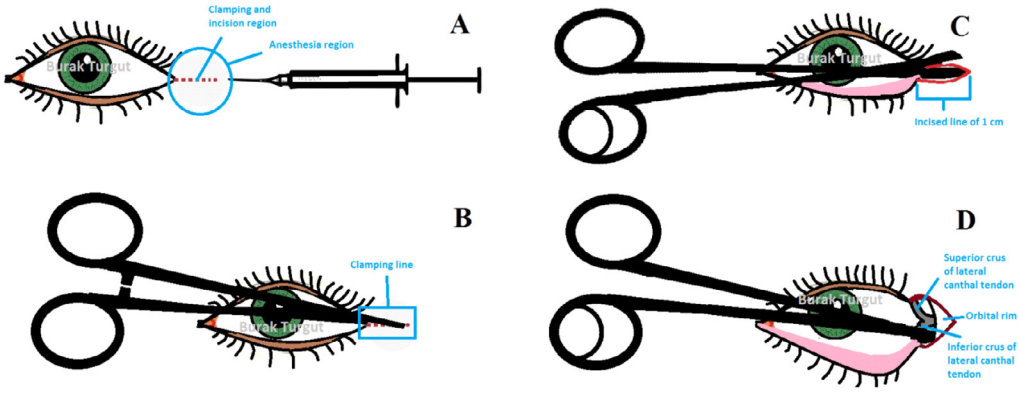

Figure 1. (a) The stages of Lateral canthotomy and inferior cantholysis procedure. Local anesthesia. **(b)** Clamping and hemostasis. **(c)** Incision on lateral canthus. **(d)** Cutting inferior crus of lateral canthal tendon.

attention is given to the direction of the tips of the scissors away from the globe. Anterior traction is placed on the free lateral edge of the lower lid and the inferior cruse of LCT can be identified as a taut band. Then, inferior crus of the LCT is cut with sharp scissors (Fig. 1d). At this moment, the fibrous tarsal plate of the lower lid relaxes. This step allows the significant decrease in the intra-orbital volume and eventually decreases INOP. This results in a completely mobile lower lid. As the incision site will usually heal spontaneously, there is no need to the suturing. However, if an oculoplastic or cosmetic deformity occurs in lower lid, further surgical repair may be needed (14, 15) (Fig. 1).

The efficacy of the LCIC: Although a fast and significant relief of the symptoms and quick reduction of IOP occurs within several minutes but the improvements of proptosis and the movements of extraocular muscles is less after a successful LCIC. However, the afferent pupillary defect should disappear due to the normalization of the ocular circulation. Single lateral canthotomy alone allows a pressure reduction of approximately 14 mmHg in INOP while as the inferior cantholysis alone reduces pressure by approximately 19 mmHg. LCIC procedure approves a pressure reduction of about 30 mmHg (4, 5, 14, 15).

The failure of the LCIC: In case of incomplete dissection and inferior cantholysis, the surgeon should question the condition and should approach the inferior crus again. Even the completed inferior cantholysis, if there is no improvement in OCS, the superior limb/crus of the LCT should be cut through the superior and lateral direction of the instrument with keeping away the globe again and additionally the lacrimal gland because of its proximity. Successful cutting of the superior crus should provide a significant laxity of both eyelids and ocular mobility (14, 15). Both eyelids are disconnected from their lateral attachments to the bony orbit, and the maximum decompression of the orbit is achieved (5, 14). If there is no relief in elevated INOP despite all these interventions, urgent referral to an orbital surgeon for the surgery of extensive orbital bone decompression is needed (4, 14, 15).

The importance of the radiological finding: Although a further investigation is not essential for the diagnosis of OCS, CT and MRI are often performed to confirm the diagnosis or to follow up. Additionally, the radiological finding of "the globe tenting" is the most common sign associated with the acute proptosis as seen in OCS, and its amount may detect the prediction of the visual prognosis. It has been previously demonstrated that a posterior globe angle of fewer than 120 degrees with acute proptosis on CT scan is associated with a poorer prognosis with the higher risk of permanent vision loss requiring urgent orbital decompression (4, 5).

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

The patients should be warned about avoiding coughing/straining or taking antitussives, antiemetics, and laxatives. The head of the bedside should be elevated to 45 degrees. Cold/ice compression may reduce periorbital/orbital edema. Patients should be advised to avoid coughing or straining. Blood pressure and coagulopathies should be normalized. All cases of OCS should be closely monitored for progression or recurrence and should be warned about admitting to hospital immediately if severe pain, proptosis and blurry vision re-occur (2-5, 15).

Conclusion

The OCS is an uncommon but vision-threating ophthalmic emergency: Knowing the signs and symptoms

of OCS and its management is necessary for prevention of vision loss. Thus, all ophthalmologists and emergency physicians should keep in mind OCS in patients having risk factors for this entity and know the technique of LCIC as this procedure may be performed rapidly at the emergency department or bedside.

Disclosures

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