

Acute retrobulbar haemorrhage: An ophthalmologic emergency for the emergency physician

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ABSTRACT

Acute retrobulbar haemorrhage (ARBH) is a rare ophthalmic emergency observed following blunt eye trauma. Multiple trauma and loss of consciousness can hide symptoms of ARBH. Rapid diagnosis and immediate lateral canthotomy and cantholysis must be performed to prevent permanent visual loss in patients. Medical treatment can be added to surgical therapy. Lateral canthotomy and cantholysis are simple procedures that can be performed by emergency physicians. In this report, it was aimed to present a case with post-traumatic ARBH and provide general knowledge about the diagnosis, follow-up and treatment of ARBH.

Key words: Maxillofacial trauma; orbital compartment syndrome; retrobulbar haemorrhage.

INTRODUCTION

Acute retrobulbar haemorrhage (ARBH) is a rare complication observed following blunt orbital trauma. ARBH concerns both emergency physicians and ophthalmologists. ARBH may result in loss of vision at various levels that may progress to permanent visual loss unless early treatment is provided. Loss of vision can be prevented through early diagnosis and treatment.^[1] Therefore, follow-up examinations of vision should be performed in emergency patients who develop proptosis after craniofacial trauma. In addition, visual loss can develop in patients with facial trauma for various reasons. In one study, in 67% of 727 patients with facial fracture, ocular damage was observed at different levels and it was reported that 3% of these damages resulted in complete loss of vision.^[2] Therefore, all patients with periorbital trauma should be evaluated in terms of potential globe damage. Dutton have reported the importance of measuring and recording the visual acuity of all patients referring to the emergency with facial trauma.^[3] As immediate and right approach is essential in the treatment

of ARBH, medical personnel should recognize the signs of ARBH, treat it and have a sufficient level of training in patient care for patients with facial trauma.

In this article, we aimed to present a patient who developed ARBH, and provide general review regarding ARBH.

CASE REPORT

A 45-year-old male patient exposed to blunt orbital trauma referred to the emergency with complaints of pain and loss of vision in the left eye. He stated that one hour prior, his left eye was exposed to an impact by a solid object. On his first examination, visual acuity was normal in the right eye and counting fingers from a 1-meter distance in the left eye. Ocular movements were limited to every direction in the left eye. Afferent pupillary defect was detected; the patient also had left periorbital edema, ecchymosis and proptosis. After blunt trauma, he got a non-penetrating laceration on his upper left eyelid on the region consistent with the orbital rim line. Furthermore, the patient stated that he had nasal congestion in the left side of his nose. In biomicroscopic examination, his right eye was normal; whereas there was corneal edema, subconjunctival hemorrhage and chemosis in the left eye. In the fundus examination, the right eye was normal; whereas the posterior segment in the left eye could not be clearly evaluated because of corneal edema. Intraocular pressure (IOP) was measured as 60 mmHg. The patient referred to our clinic following blunt orbital trauma was diagnosed with ARBH and the patient's condition was conveyed to the radi-

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ology unit. Axial and coronal orbital computerized tomography (CT) was performed immediately. Upon assessment by CT, fracture of the left medial orbital wall, haemorrhage within the retrobulbar space, nasal fracture and suspicious fracture at the level of infraorbital canal were detected (Fig. 1). The patient was laid down with head up at an angle of 45°. Intravenous (IV) mannitol and topical dorzolamide-timolol were administered to the patient and lateral canthotomy and cantholysis were performed. The left lower eyelid was completely released with lateral canthotomy and cantholysis (LCC) (Fig. 2). Skin laceration on the left upper eyelid was left to primary healing. Soon after LCC, IOP decreased to 30 mmHg and the patient's visual acuity improved to 4/10. The eye was not closed for follow-up examination and pressure dressing was not used. Oral prophylactic antibiotic treatment was initiated. IOP decreased to 18 mmHg after 8 hours. In visual examination, the patient's vision was detected to be at the level of 8/10 and globe movements were normal. The patient had complaints of nasal congestion but was told not to blow his nose in order to prevent the development of orbital emphysema. In his evaluation on day 10, the patient had no visual loss. In this follow-up, lower eyelid reconstruction with lateral canthal sling was performed due to lower eyelid ectropion (Fig. 3).

ARBH is not a commonly encountered condition. It may develop after orbital surgery, endoscopic sinus surgery, retrobulbar and peribulbar injection, spontaneously with facial and craniofacial traumas or trauma surgery.^[4-12] As a complication

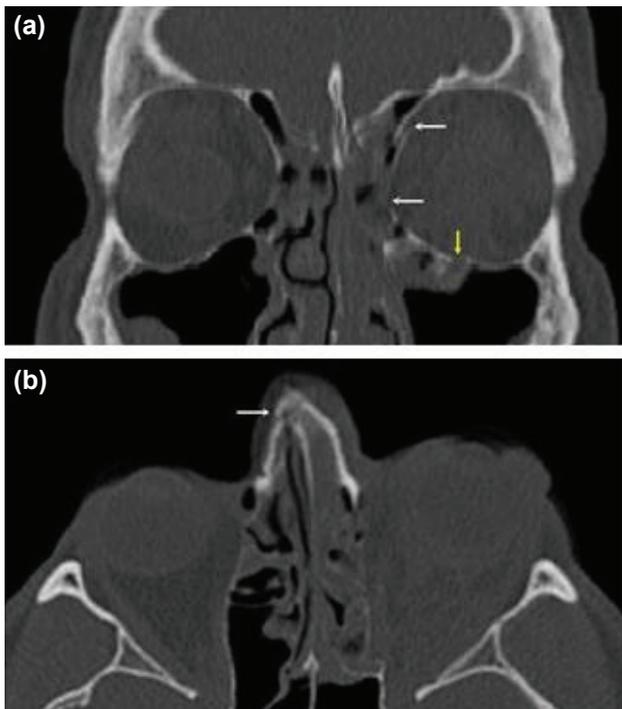


Figure 1. (a) Retrobulbar haemorrhage is observed in coronal orbital CT; fracture in the left medial orbital wall (small arrows), suspicious fracture at the level of infraorbital canal (big arrow). (b) Fracture in the nasal bone in axial orbital CT (arrow).



Figure 2. After canthotomy and cantholysis: Frontal view in the figure above, lateral view in the figure below. The release of the lower eyelid is clearly observed in the picture below.



Figure 3. Patient's appearance in the follow-up examination on day 10.

of orbital or periorbital surgery, orbital hemorrhage may develop particularly when the orbital septum is passed.^[13,14]

DISCUSSION

The rate of development of ARBH after retrobulbar and peribulbar injection is less than 2%.^[6,7] The incidence of orbital haemorrhage development with blepharoplasty has been reported as 0.055% (1:2,000) and after zygomatic fracture repair as 0.3%.^[12,14]

The orbital space is comprised of three compartments, namely subperiosteal space, intraconal space and extraconal space. In case haemorrhage is observed in one of these compartments, the orbital space does not comply with the

increase in volume. Therefore, a forward protrusion of the globe is observed leading to clinical appearance of proptosis. The orbital bone structure is open only at the front. However, as the eyelids are attached to the orbital rim by the canthal tendons in this segment, essentially it is also limited at the front. Even though minor volumetric increases in the orbit are compensated, rapid elevations in volume increases intraorbital pressure and the globe displaces forwardly.^[15,16] As the intraorbital pressure increases, interstitial pressure also increases and perfusion pressure decreases. The globe, pushed backwards by the eyelids at the front, is simultaneously pushed forward from behind with the effect of increasing orbital pressure. IOP increases, and the perfusion of the eye deteriorates. As in our case, IOP may increase to 60 mmHg or further. Normal IOP value is 8–21 mmHg.^[17] The orbit acts as a closed box; as in the other compartments of the body, there is a similar pressure-volume relationship in orbital fissures and foramina. Therefore, there is the risk of development of compartment syndrome. Compartment syndrome develops in the orbital area, and as a result of the increasing tissue pressure, perfusion pressure decreases.^[18] In the elevation of orbital pressure, the posterior ciliary artery is affected more than the central retinal artery (CRA) is. The CRA can be protected due to being covered by the optical nerve. High systolic pressure is also one of the mechanisms protecting the CRA. There is no similar protection in other blood vessels inside the muscular cone or in the ones entering the eye around the optical nerve. Hence, complete visual loss has been reported due to the CRA occlusion as well as anterior ischemic optic neuropathy.^[18]

Venous pooling is observed due to increasing intraorbital pressure after retrobulbar haemorrhage leading to a further increase in the orbital pressure. As the pressure on the tissues increases, arterial flow will stop.^[19,20] If the vasa nervorum is affected, optical nerve involvement is expected as well. When the orbital pressure exceeds the CRA pressure, retina begins to be affected. Basically, with an increase in the orbital pressure and decrease in perfusion, damage starts and pathology establishes. Further, the optical nerve may be damaged due to compression and tension.^[21] Visual loss generally develops as a result of the CRA occlusion, direct compression to the optical nerve or to the blood vessels feeding the optical nerve.^[8] It should also be kept in mind that visual loss may also develop after blunt ocular trauma due to retinal detachment, hyphema, rupture of the globe and vitreous haemorrhage. Although the mechanism of visual loss is not clear, recovery of vision after decompression suggests that ischemia and reperfusion lie under the pathogenesis of this condition.^[22,23] Vision is the most valuable criterion in diagnosis and follow-up. Level of vision is correlated with optical nerve and retinal perfusion. In our case, improvement in vision from the level of counting fingers from a 1-meter to the 4/10 level after lateral canthotomy and inferior cantholysis supports this argument. When retrobulbar pressure increases to a critical level, irreversible optical nerve and retinal ischemia develop in 60

minutes and resistant visual loss in 1.5 to 2 hours.^[1,24–27] It was reported that healing in retinal ischemia lasting no longer than 90 minutes was good; whereas the response to ischemia exceeding 105 minutes was poor; therefore, it was emphasized that surgery should be performed in the first 90 minutes.^[26] Our case had referred to our clinic in 1 hour. The patient's diagnosis was made immediately and his condition was conveyed to radiology. Axial and coronal CT scan of the orbit was performed immediately. As the diagnosis was confirmed by the CT scan, lateral canthotomy and inferior cantholysis were performed under emergency room conditions. The time to lateral canthotomy and inferior cantholysis was approximately 30 minutes. Total time, including this, was 90 minutes after the trauma. The level of vision was 4/10 post-surgery and 8/10 after 8 hours. In cases when imaging cannot be performed immediately, if clinical diagnosis of retrobulbar haemorrhage is made, performing lateral canthotomy and inferior cantholysis, which are simple and safe methods, without imaging would be the right approach.

When diagnosing a patient with orbital trauma, visual acuity and frontal segment examination should be performed and reactions to light should be carefully evaluated. Evaluation of the patient's reactions to light is of major importance regarding optical nerve function. Pupil dilation and fundus examination should be reserved after light reflex examination. In case of optical nerve pathology or cranial pathology are suspected, fundus examination may be delayed. During the assessment of light reactions, when one eye is exposed to light, bilateral pupils constrict, indicating that afferent system (signal to the brain) and efferent system (signal from the brain) are not damaged. If there is afferent pupillary defect, when the damaged eye is exposed to light both pupils stay dilated. Constriction of both pupils indicates that the optical nerve in the eye exposed to trauma is not damaged.

It is sometimes difficult to make a complete evaluation in traumatic patients. The patients are generally confused, agitated or unresponsive. Proptosis is the first marked finding in cases when visual acuity assessment cannot be performed. The main clinical features of ARBH are decrease in visual acuity, pain, proptosis and ophthalmoplegia. In unresponsive and agitated patients, it may not be easy to assess pain and ophthalmoplegia. In unresponsive and unconscious patients, globe movements can be evaluated by forced duction test. The evidence of elevated orbital pressure is tight lids, resistance to retropulsion and tight orbit. The course of intraocular pressure in ARBH is high.^[17] Chemosis and subconjunctival haemorrhage may occur in orbital haemorrhage. Periorbital crepitus may be present in orbital emphysema.^[28,29] In case of fundus examination can be performed, optical disc edema, retinal venous congestion, CRA pulsation or occlusion and retinal edema may be observed.^[30]

Even though orbital tissue pressure can be measured directly by manometer, clinically, it is not used very commonly. Nor-

mal orbital pressure is 3–6 mmHg.^[15,16] In a patient exposed to periorbital trauma, the intraocular pressure bears an alerting character. Elevated IOP is the most substantial finding that will indicate compartment syndrome if the patient is unconscious or non-cooperating. The normal value of IOP ranges between 8–21 mmHg.^[17] In a patient with increased IOP, complaints such as pain in the eye, decrease in vision and changes in perceptions of colour are observed. If IOP is ≥ 40 mmHg in the proptotic eye, this is an important indication concerning ischemia and requires immediate treatment. As our patient's eyes could be partially opened and his cooperation was good, intraocular pressure could be measured. In patients with facial trauma, measuring intraocular pressure may not always be possible, so increase in intraocular pressure can be examined by palpation of the globe. It may sometimes be difficult to decide whether the globe is solid or not under emergency conditions. Palpation is a subjective method and may prove dangerous in cases with suspected perforation. Whatever the IOP is, visual loss and relative afferent pupil defects indicate the need for immediate treatment.

ARBH diagnosis can easily be made in the presence of proptosis, abnormally reactive pupils and swelling in the disc. Proptosis is the most prominent sign in a traumatic patient. It sometimes develops due to a secondary edema in the retrobulbar tissues and compartment syndrome may be a part of the phenomenon. In some cases, it is difficult to make a diagnosis of proptosis because edema in the periorbital region may conceal proptosis. An objective diagnosis of proptosis can be made by an exophthalmometer.

Within 1–3 hours following ARBH, the probability of irreversible exposition to damage in retina increases.^[1,6,7,24–27,29] As ARBH may be observed after an orbital surgery; orbital pain, visual acuity, proptosis, direct light reaction and pupil size should be evaluated and recorded in the postoperative follow-up. If pathology is detected, immediate action should be taken for diagnosis and treatment. Caution should be taken regarding retrobulbar haemorrhage in a patient with tense and proptotic eye with complete visual loss and non-reactive pupillary response after facial trauma. In one study, it was emphasized that the most common reason of visual loss after facial fracture repair was the increase in the orbital pressure developing after the haemorrhage.^[10] This condition also requires immediate surgery. Performing immediate surgical decompression shall prevent visual loss.

Orbital examination of patients referring for head and face trauma should not be ignored. Rupture in globe, optical nerve damage, intraorbital haemorrhage, foreign objects, periorbital and orbital apex fractures can easily be differentiated. Imaging methods are useful in showing the severity, cause, location and pathology of the condition. The connection between the presence of orbital fracture and hematoma should be assessed by CT scan. Diagnosis can also be confirmed by ultrasonography (USG).^[31,32] Magnetic resonance imaging is

often performed later. In the USG scan, retrobulbar hematoma can be observed in front of the orbital bone and behind the globe as a hypoechoic lesion. Tension in the optical nerve after proptosis can be viewed by CT scan. Further, in the CT scan, distortion in the posterior segment of the globe can be observed to some extent due to retrobulbar hemorrhage.^[31]

Patients referring to the emergency with orbital trauma should be told that due to sino-orbital connections, exhaling from their noses forcibly will carry the risk of orbital haemorrhage and emphysema due to valsalva and cough.^[28] The patient with orbital trauma should be laid down with an angle of 45°. In these patients, eye dressings and pressure dressing should not be used; pressure dressing can increase intraorbital pressure, and also covering can delay early diagnosis.^[32] Our patient's eye was not covered. Our patient with medial orbital wall fracture on the left had nasal congestion as well. Therefore, the patient was told not to blow his nose to prevent the development of orbital emphysema.

Medical treatment has a role before and/or after the surgical treatment of ARBH. Medical treatment has a supplementary contribution to surgical decompression. Osmotic agents, carbonic anhydrase inhibitors, high-dose steroids and β -blockers are used in medical treatment.^[1,32] Using osmotic agents such as mannitol increases the osmolarity of blood and creates osmotic gradient thereby providing a reduction in IOP. Further, using carbonic anhydrase inhibitors such as acetazolamide, helps decrease IOP by reducing aqueous humor production. Osmotic agents decrease IOP by reducing the water content of the vitreous. Mannitol is a rapid acting agent; however, mannitol and acetazolamide should not be used in hypovolemic shock. Anti-inflammatory and anti-oxidant activities of corticosteroids are utilized. It has been shown that high dose corticosteroids have a protective effect on traumatic optic neuropathy.^[32] Steroids stabilize the cell membrane against ischemic damage by reducing inflammation.^[33] Timolol maleate also reduces the production of aqueous humor.^[32] Hence, it has a role in medical treatment. In the medical treatment of ARBH, i.v. 20% mannitol 2 g/kg q.i.d., i.v. acetazolamide 250 mg q.i.d., i.v. methylprednisolone 250 mg q.i.d. and topical timolol maleate one drop b.i.d. are recommended.^[1,32] Certain amount of time should pass in order for the medical treatment to reduce orbital pressure. Further, we think that medical treatment has a limited effect on reducing the orbital pressure. Therefore, we think that it may only be used as an adjuvant therapy. There are publications contrary to this opinion. A case with complete recovery of vision has been reported without the need of surgery after the medical treatment.^[34] There is also a publication recommending surgical intervention in case the vision is not recovered in 30–45 minutes.^[11] However, we think that as a certain amount of time will pass from the moment of the incident to the patient's arrival to the emergency room and also time is required for the medical treatment to show effect and additionally since we think that medical treatment has a limited effect in reducing

the orbital pressure, we recommend that medical treatment and LCC should be performed concurrently. LCC will reduce the pressure exerted on the orbit by the eyelids which limit the orbit in the front. Firstly, a sudden intraorbital pressure reduction will occur in consequence of the removal of the limiting effect of the eyelid on the orbit. Then, a slow orbital pressure reduction will occur as a result of the reduction in pressure in the foramina and veins, decrease in venous congestion and resolution of edema. Intraocular pressure measured 60 mmHg in our patient, decreased to 30 mmHg postoperatively and to 18 mmHg in 8 hours. There are also authors adopting surgery as the first choice resembling our approach. Goodhall et al.^[4] reported that when they performed LCC alone, it was effective in improving visual acuity and preventing permanent visual loss and that they had performed surgery concurrent with intravenous acetazolamide in two cases.

LCC as surgical treatment is a well-documented approach in the treatment of ARBH.^[8] Lateral canthotomy and inferior cantholysis were recommended in many studies.^[4,8,32] Canthotomy and inferior cantholysis can easily be performed under the conditions of a simple outpatient clinic or emergency room under local anesthesia.^[4,8] It is favourable that decompression surgery is performed by an ophthalmologist. However, under emergency conditions if an ophthalmologist cannot be reached, LCC should be performed immediately by a trained medical doctor.^[35,36] Suner et al.^[36] have defined a training model of lateral canthotomy and cantholysis for emergency physicians. Lateral canthotomy is the procedure of separating lateral canthal tendon in two by cutting it up to the orbital rim. Afterwards, inferior cantholysis is performed by releasing the lower leg of the canthal tendon. Following inferior cantholysis, the lower leg of the lateral canthal tendon is entirely separated, consequently the lower eyelid is completely mobile (Fig. 2). Even though canthotomy can be performed alone or with cantholysis, some authors recommend performing cantholysis with lateral canthotomy.^[4,8] Inferior cantholysis is more effective than canthotomy in decreasing the IOP.^[37] However, concurrent lateral canthotomy and cantholysis reduce orbital pressure more effectively. In a study where orbital pressure was increased by administering saline solution to a sheep's eye, concurrent canthotomy and cantholysis (30.4 mmHg) provided more reduction in the intraocular pressure compared to canthotomy (14.2 mmHg) and cantholysis (19.2 mmHg) alone.^[37] In our experience, lateral canthotomy and cantholysis technique, when combined with intravenous corticosteroids mannitol and acetazolamide, is a much more successful method. In another case who had retrobulbar haemorrhage symptoms after retrobulbar anesthesia, after decompression with lateral canthotomy and inferior cantholysis IOP decreased from 50 mmHg to 30 mmHg.^[38] The efficacy of the technique is also observed in our case.

LCC releases the lateral canthal tendon and allows mobility of the lower eyelid. After LCC, the skin can stay uncov-

ered for a few days, and soon after the swellings are resolved, it can be repaired or be left to heal spontaneously. If IOP is still elevated after the LCC, hematoma should be discharged with decompression surgery. Should lateral canthotomy and inferior cantholysis fail, the upper leg of the lateral canthal tendon can be released. With medial, lateral and basal surgery, a space can be formed where the volume can expand in the orbital area. Burkat has described inferolateral anterior orbitotomy for the discharge of hematoma or compressed air.^[6]

The potential complications of LCC are ectropion, lacrimal gland damage and post-operative infection. Lateral canthal incision then can be sutured or more often left open to heal with granulation.^[4,18] In our case, eyelid repair with lateral canthal sling was performed due to ectropion in the lower eyelid.

As a conclusion, ARBH may cause permanent visual defect. LCC should be performed immediately in patients diagnosed with ARBH in the presence of elevated intraocular pressure, proptosis and decrease in visual acuity. Further, we think that starting medical treatment shall provide a supplemental contribution to reducing orbital pressure.

Conflict of interest: None declared.

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OLGU SUNUMU - ÖZET

Acil hekimlerinin oftalmik acili: Akut retrobulber hemoraji

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Akut retrobulber hemoraji (ARBH) күnt göz travması sonrası görülen nadir bir oftalmik acildir. Hastanın çoklu travmatik hasarı ve bilinç kaybı varsa semptomlar gizlenebilir. Hastada kalıcı görme kaybını önlemek için hızlı tanı konup zaman kaybetmeden lateral kantotomi, kantoliz yapılmalıdır. Medikal tedavi cerrahi tedaviye ek olarak başlanabilir. Lateral kantotomi ve kantoliz acil hekimleri tarafından uygulanabilecek kolay bir prosedürdür. Bu makalede travma sonrası retrobulber hemoraji gelişen bir olgumuzu sunarken, retrobulber hemorajinin tanısı, tedavisi ve takibi hakkında genel bilgi verilmiştir.

Anahtar sözcükler: Maksillofasial travma; orbital kompartman sendromu; retrobulber hemoraji.

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