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REVIEW ARTICLE

RETROBULBAR HEMORRHAGE: CURRENT CONCEPTS IN PATHOPHYSIOLOGY, EARLY RECOGNITION, AND EMERGENCY MANAGEMENT

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ABSTRACT

Background: Retrobulbar hemorrhage (RBH) is an uncommon yet potentially vision-threatening orbital emergency characterised by bleeding within the retrobulbar space, resulting in a rapid rise in intraorbital pressure and risk of optic nerve ischemia.

Objective: This review aims to summarise the current evidence regarding the aetiology, clinical presentation, diagnosis and management strategies of RBH, with a focus on timely intervention and improved outcomes.

Methods: A narrative review of the literature was conducted, focusing on the aetiology, pathophysiology, clinical features, and therapeutic approaches associated with RBH in trauma, surgical, and non-traumatic settings.

Results: Retrobulbar hemorrhage (RBH) most commonly follows facial trauma, orbital or maxillofacial surgery, and local anaesthetic injections, although vascular abnormalities and coagulopathies may also contribute. Clinically, it presents with proptosis, severe orbital pain, reduced visual acuity, ophthalmoplegia, and relative afferent pupillary defect. Prompt recognition is essential to prevent irreversible vision loss. Management includes medical reduction of intraocular and intraorbital pressure; however, emergency lateral canthotomy and cantholysis remain the definitive vision-saving interventions when optic nerve perfusion is compromised.

Conclusion: RBH requires immediate diagnosis and management to prevent permanent vision loss. Increased awareness, perioperative vigilance, and standardised emergency protocols are essential for enhancing clinical outcomes and minimising associated morbidity.

Keywords: Canthotomy, Cantholysis; Compartment syndrome; Retrobulbar hemorrhage (RBH).

INTRODUCTION

Retrobulbar hemorrhage (RBH) is an uncommon but potentially devastating orbital emergency that demands immediate recognition and intervention to prevent irreversible visual impairment. RBH leads to a rapid elevation of intraorbital pressure due to the rigid confines of the bony orbit. Even small increases in orbital volume can precipitate orbital compartment syndrome (OCS), compromising optic nerve perfusion and retinal circulation. If left unaddressed, ischemic injury may occur within minutes, leading to visual impairment or permanent loss of vision [1]. The aetiology of RBH is diverse. Trauma remains the most common cause, particularly in association with midfacial injuries, zygomaticomaxillary complex fractures, and orbital blowout fractures. Surgical interventions involving the orbit, eyelids, sinuses, and maxillofacial region also represent significant risk factors. Retrobulbar injections administered during ophthalmic anaesthesia, cosmetic procedures such as

blepharoplasty, and endoscopic sinus surgery have all been linked to iatrogenic RBH [2]. Additionally, systemic conditions, including hypertension, coagulopathies, anticoagulant therapy, and vascular malformations, can predispose patients to spontaneous hemorrhage within the orbital compartment [19]. For oral and maxillofacial surgeons, the relevance of RBH is significant. Many maxillofacial trauma cases and surgical procedures place the orbital contents at risk, and failure to recognize RBH can lead to preventable blindness. Therefore, a thorough understanding of the anatomical considerations, mechanisms of injury, clinical presentation, and emergency management protocols is essential. Despite being rare, RBH carries disproportionately high morbidity relative to its incidence, and published literature continues to emphasize the importance of rapid, informed decision-making to optimize visual outcomes.

ANATOMY OF RETROBULBAR SPACE

The retrobulbar space, also referred to as the intraconal space, is the compartment located posterior to the globe and enclosed by the four rectus muscles that converge posteriorly at the annulus of Zinn. This space contains critical neurovascular and muscular structures, including the optic nerve, ophthalmic artery, ciliary nerves, ciliary ganglion, and adipose tissue that cushions and stabilizes the globe. The extraocular muscles form a conical boundary, with the optic nerve occupying the central axis as it travels posteriorly from the globe to the optic canal. Laterally, the space is bordered by the lateral rectus muscle and the lacrimal gland fossa; medially by the medial rectus muscle and ethmoid air cells; superiorly by the superior rectus–levator palpebrae superioris complex; and inferiorly by the inferior rectus muscle. The orbital walls provide rigid containment, such that even small increases in volume, such as those caused by bleeding, can rapidly elevate intraorbital pressure. This tightly confined arrangement makes the retrobulbar space particularly vulnerable to compressive pathology, especially acute hemorrhage, which can threaten optic nerve perfusion. The adipose tissue within the intraconal compartment facilitates smooth ocular movements, while the neurovascular bundles traverse the space to supply the globe and extraocular muscles. Because the orbital septum restricts anterior expansion, pathological processes within the retrobulbar space typically expand posteriorly or exert forward pressure on the globe. Understanding the anatomical layout of this space is essential for diagnosing and managing conditions such as retrobulbar hemorrhage, optic neuropathies, and surgical complications.

ETIOLOGY

Retrobulbar hemorrhage can be caused by surgical reduction of orbital and mid-face fractures, cosmetic or reconstructive eyelid operations, endoscopic sinus procedures, retrobulbar injections, blunt trauma, and thrombolytic agents [4]. A retrospective study on orbital fractures reveals that the incidence of RBH is approximately 1.3%. The overall incidence of retrobulbar hemorrhage in maxillofacial trauma is roughly 1–4%, depending on the nature and severity of the fractures and the study population [5-6]. Local anaesthesia in the retrobulbar space is a traditional method for providing akinesia and analgesia to the eye during surgical procedures. However, this treatment is rarely complicated by the occurrence of RBH (incidence: 0.75%), which poses a potential risk to vision loss [4]. RBH occurs when bleeding develops within the limited retrobulbar or intraconal space, most typically caused by disruption of the arterial or venous network in the orbit.

RBH likely occurs due to 3 major causes **(Fig. 1)**

1) Trauma is the most frequent etiological cause, especially blunt periorbital injuries, zygomaticomaxillary fractures, and penetrating orbital trauma, which may disrupt the ophthalmic artery, ciliary arteries, or orbital veins.
2) Non-traumatic causes include vascular malformations, spontaneous bleeding from hypertension, coagulopathies, anticoagulant or antiplatelet medication, and systemic disorders like hemophilia or liver dysfunction that impede haemostasis.
3) Iatrogenic causes include periocular surgeries such as blepharoplasty, endoscopic sinus surgery, and retrobulbar or peribulbar anaesthetic injections, when unintentional vascular perforation can result in rapid blood accumulation [4,7].

Postoperative factors such as uncontrolled hypertension, coughing, Valsalva maneuvers, and anticoagulant use may precipitate delayed bleeding in vulnerable patients. Because the orbit is a non-expandable compartment, even minimal bleeding during maxillofacial procedures can rapidly elevate intraorbital pressure, underscoring the importance of meticulous surgical technique, thorough understanding of orbital anatomy, and prompt recognition of visual symptoms [3].

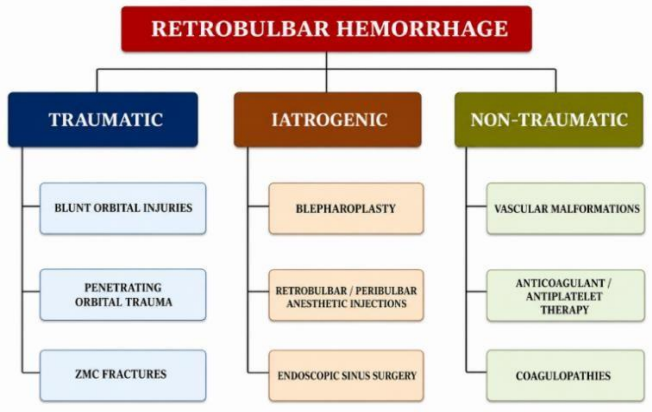

Figure 1. Etiology of Retrobulbar Hemorrhage (RBH)

PATHOPHYSIOLOGY

The orbit is a rigid, non-distensible bony cavity containing the globe, extraocular muscles, optic nerve, orbital fat, and a dense vascular network. Consequently, even a small orbital hemorrhage can significantly increase intraorbital pressure, causing anterior displacement of the globe [7]. Elevated pressure compresses the optic nerve, central retinal artery, and short posterior ciliary arteries, resulting in retinal and optic nerve ischemia. Progressive pressure further impairs venous outflow through the superior and inferior ophthalmic veins, leading to venous congestion, worsening proptosis, and expansion of hematoma [6]. The orbital septum restricts anterior decompression, directing hemorrhagic expansion posteriorly toward the optic nerve. When intraorbital pressure exceeds central retinal artery pressure, retinal perfusion ceases, potentially causing irreversible vision loss **(Fig. 2).** Concurrent compression of the extraocular muscles and stretching of the optic nerve contribute to ophthalmoplegia and impaired axonal transport.

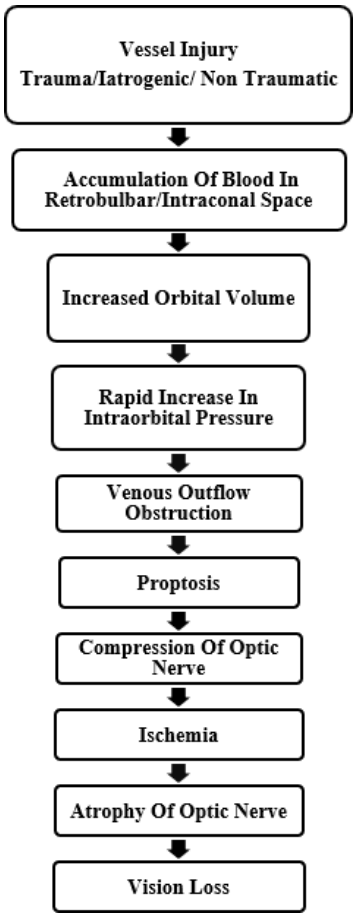

Figure 2 Pathophysiological sequence of Retrobulbar hemorrhage (RBH)

CLINICAL FEATURES

All patients presenting with orbital injuries or midfacial fractures should undergo a complete ophthalmologic examination[10]. The most common signs and symptoms include pain, proptosis, chemosis, diplopia, subconjunctival hemorrhage, increased intraocular pressure, a tense globe, decreased visual acuity, loss of the direct pupillary light reflex, or ophthalmoplegia [4,7-8]. The appearance & size of the hemorrhage were graded on a four-point scale: (1-spot ecchymosis), (2-lid ecchymosis involving half of the lid surface area or less), (3-lid ecchymosis all around the eye with no increase in intraocular pressure), (4-retrobulbar hemorrhage with elevated intraocular pressure) [4,10]. If these signs are not recognized early, the outcome could result in complete loss of vision in the affected eye. Prevention, recognition, and intervention are therefore crucial to prevent those devastating conditions [7].

MANAGEMENT:

The management of retrobulbar hemorrhage (RBH) is a time-critical emergency aimed at rapidly decompressing the orbit, preserving optic nerve perfusion, and preventing irreversible visual loss. Under physiological conditions, normal intraorbital pressure ranges from 3 to 6 mmHg [11]. It can rise to many times the normal value in the event of RBH, producing a vision-threatening compartment syndrome. In animal studies, it has been shown that ischaemia, due to transient central retinal artery occlusion, is well tolerated up to 100 min; however, irreparable damage can occur after ischaemia of 105 min [12]. After a diagnosis of retrobulbar hemorrhage, Prompt orbital decompression is essential to preserve optic nerve function. It has been stated that if decompression is carried out within 2 hours of the onset of symptoms, it is likely to be successful in avoiding permanent damage According to Erickson et al., emergent orbital decompression is indicated when relative proptosis, significant eyelid tightness resistant to digital opening, and a relative afferent pupillary defect are present concurrently. Collectively, these findings strongly indicate optic nerve ischemia secondary to orbital compartment syndrome and warrant immediate surgical management to prevent permanent visual loss.[8]. If proptosis or tight lids are present after orbital trauma, clinicians should promptly check for RAPD rather than other vital signs, such as visual acuity, IOP, or extraocular movements, which can lead to unnecessary delay in vision-saving procedures. Patients presenting with proptosis, marked eyelid tension in the absence of voluntary squeezing, or a substantial preseptal haematoma should undergo urgent orbital decompression. In contrast, the presence of a relative afferent pupillary defect (RAPD) without associated proptosis or eyelid tightness is more suggestive of optic neuropathy resulting from a pressure-independent mechanism. In such cases, lateral canthotomy and cantholysis are unlikely to provide therapeutic benefit. Regular eye examinations are required after surgical reduction of zygomatic/orbital fractures to assess the

RBH in early stages. Hayter and Sugar created an orbital observation chart, which is currently widely used in many centers to record observations of orbital pain, visual acuity, proptosis, direct light reflex, and pupil size [13]. Once the clinical diagnosis of retrobulbar hemorrhage is made, treatment should begin immediately and involves both medical and surgical procedures. Additional investigations, such as CT or ultrasound scans, should not delay treatment once a clinical diagnosis has been established. Management of RBH encompasses both medical and surgical methods, as reported in the literature, and is outlined in **(Fig. 3).**

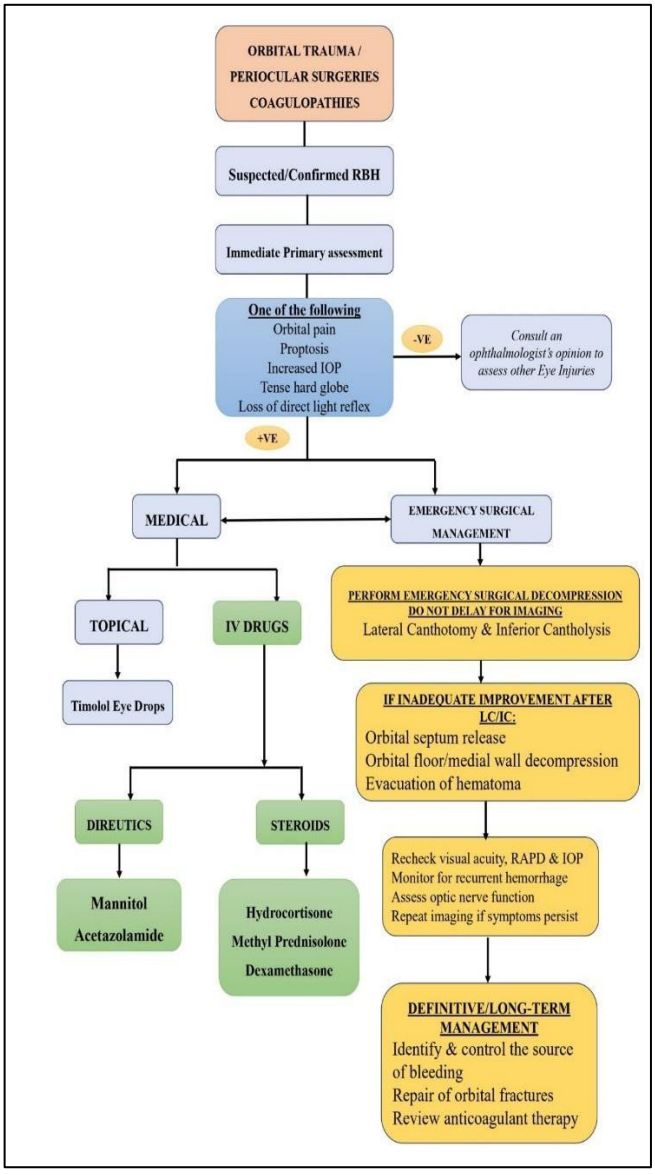

Figure 3. Clinical management algorithm for the diagnosis and emergency treatment of Retrobulbar hemorrhage. (RBH)

Medical management may be used as an adjunct to surgical decompression or in selected mild cases.It includes Diuretics (mannitol, acetazolamide), corticosteroids (Methylprednisolone, Dexamethasone, or Hydrocortisone,) and topical beta blockers like Timolol eye drops are used.

Intravenous administration of Acetazolamide 500 mg or hydrocortisone 100 mg can be given. A rapid infusion of 20% mannitol can also be used [14]. If vision does not improve within 30 to 45 minutes, surgical intervention is necessary.

Carbonic Anhydrase inhibitors, such as acetazolamide, lower intraocular pressure by inhibiting carbonic anhydrase, which significantly reduces aqueous humor output. Hyperosmotic agents, such as mannitol, shrink the vitreous and reduce the volume of tissue in the orbit [4]. Corticosteroids such as dexamethasone can reduce inflammation and stabilize cell membranes against ischemic damage. Wood et al. reported a case in which intravenous mannitol, acetazolamide, and hydrocortisone were administered for the management of retrobulbar hemorrhage, resulting in significant improvement in visual acuity within six hours without the need for surgical decompression .[7]

Conservative treatment should be continued for five to seven days if there is a decrease in local signs & symptoms and an improvement in vision [15].

Other nonsurgical therapeutic approaches are recommended, including the use of timolol maleate eye drops, 0.25% topical solution, 1 to 2 drops daily, which functions by decreasing the production of aqueous humor. Additional conservative strategies that may be overlooked include elevating the head, applying topical ice packs, and avoiding dressings that can be tight and potentially elevate intraocular pressure [18].

Lateral canthotomy with inferior cantholysis is the most widely accepted surgical decompression technique for retrobulbar hemorrhage. Goodall et al. demonstrated that combining pharmacological therapy with surgical decompression improves visual outcomes and reduces the risk of permanent blindness. Once acute RBH is diagnosed, immediate lateral canthotomy and inferior cantholysis should be performed, as the procedure is simple, effective, and can be carried out under local anaesthesia in an outpatient or emergency setting [17].

Lateral Canthotomy & Inferior Cantholysis: The inferior and superior crus of the lateral canthal tendon attach to the inner part of the lateral orbital rim, known as Whitnall's tubercle. The lateral canthotomy involves placing an artery clip between the upper and lower lids, moving towards the orbital rim, and crushing the tissues. With the sharp dissection scissors, the lateral canthal tendon is incised, followed by inferior cantholysis.

The lateral canthal tendon is separated from the skin anteriorly and the conjunctiva posteriorly. The inferior crus of the lateral canthal tendon is entirely disinserted, allowing the lower lid to move freely to evacuate the hematoma [4,17,19].

PREVENTION:

Preventing retrobulbar hemorrhage requires careful planning preoperatively, intraoperatively, and postoperatively for orbital/maxillofacial procedures and

ophthalmic surgeries. Pre-operatively, identifying high-risk patients, such as those with hypertension, coagulation disorders, or on anticoagulant therapy, is essential, along with optimizing systemic conditions and reviewing orbital anatomy. Intra-operatively, prevention focuses on gentle dissection, avoiding deep or blind instrumentation, and achieving meticulous hemostasis while maintaining stable blood pressure and minimising Valsalva or coughing. Post-operatively, prevention includes avoiding tight dressings, elevating the head to reduce venous pressure, and monitoring early warning signs like pain, proptosis, or visual changes. Educating patients to prevent straining, heavy lifting, and forceful nose blowing further reduces their risk. These combined measures significantly lower the likelihood of retrobulbar hemorrhage and its vision-threatening complications **(Fig.4)**

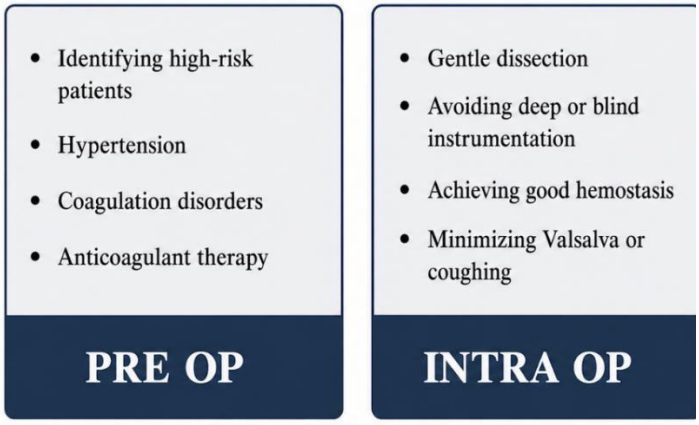

Figure4. Preoperative, intraoperative, and postoperative strategies for the prevention of Retrobulbar hemorrhage (RBH).

CONCLUSION

Retrobulbar hemorrhage (RBH) remains a rare but potentially vision-threatening complication encountered in orbital, ophthalmic, maxillofacial, and trauma-related procedures. Early recognition, rapid intervention, and adherence to well-defined preventive strategies are essential to minimize morbidity. A thorough understanding of orbital anatomy, meticulous surgical technique, and vigilant perioperative monitoring significantly reduce the risk of RBH and its complications.

DECLARATIONS

Acknowledgment

Declared none.

Conflict of Interest

The author declare no conflict of interest.

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Ethical Approval

Ethical approval was not required as this is a narrative review of published literature.

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