Case Report

Retrobulbar Hemorrhage: A Critique

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Abstract

Introduction: Retrobulbar hemorrhage is a rare, progressive, sight-threatening emergency. Standardized treatment algorithms for it exist in the literature; however, their efficiency is uninterpretable in most non-ophthalmic settings. This paper reviews retrobulbar bleeding, the pathophysiology of vision loss, and the therapeutic modalities and their efficacy in addressing such visual damage.

Case Report: We present the unfortunate case of an 82-year-old male with coronary artery disease on anticoagulants who presented for a vitrectomy for a vitreous hemorrhage secondary to eccentric disciform degeneration under general anesthetic with an accompanying retrobulbar block. He subsequently developed a retrobulbar hemorrhage at some point post-operatively. He presented to his surgeon 18-hours after the operation with no light perception on ophthalmologic examination as well as facial ecchymosis and proptosis. Efforts to reduce his intraocular pressure were successful, but there was no return of vision.

Conclusion: Retrobulbar hemorrhage can be a devastating visual event. Standardized treatment algorithms have been described, however, the effectiveness or need of such treatments can only be evaluated by an ophthalmologist and are often performed too late to provide meaningful recovery.

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Introduction

Retrobulbar hemorrhage is a rare, progressive, sight-threatening emergency that results in the accumulation of blood in the retrobulbar space. Although the incidence of retrobulbar hemorrhage is low, its association with blindness is significant with estimates nearing as high as around 48% [1].

Since the etiology of a retrobulbar hemorrhage is multiple including trauma, a sequela of ophthalmic surgery, retrobulbar injections, orbital vascular abnormalities, or the use of anticoagulant medication it can present to multiple different practitioners including emergency room doctors, otorhinolaryngologists, plastic surgeons, nurse practitioners as well as ophthalmologists. Since the latter have the ability to adequately monitor the visual and intraocular effects of the hemorrhage and the results of treatment methods, current standardized treatment algorithms recommended in many articles may not be necessary and their efficiency uninterpretable in most non-ophthalmic settings.

In order to understand the pathophysiology of retrobulbar hemorrhage and the rationale for certain treatments and their effectiveness, it is imperative to understand the anatomy of the retrobulbar space. The retrobulbar compartment is located behind the eye globe [2]. It is bounded by the orbital septum anteriorly, which is a fibrous sheath originating from the orbital rims. This septum is attached to the lateral canal tendon and the lateral orbital rim. Medially, laterally, and posteriorly, the space is bordered by the bones of the orbit. The optic nerve and the blood vessels that supply the eye travel through this space. Due to the compactness of this compartment and its inability to expand, the accumulation of blood and the subsequent pressure that results can be hazardous. Essentially, a retrobulbar hemorrhage results in a compartment syndrome which can lead to obstruction of the ocular venous drainage system, compression and ischemia to the optic nerve, and eventual occlusion of the arterial system leading to visual loss.

Patients with a significant retrobulbar hemorrhage may present with pain, periocular ecchymosis, hemorrhagic chemosis, proptosis, nausea and vomiting, and visual loss. The latter being obviously the most major concern of a retrobulbar hemorrhage. Since it is such a small space...
around the globe, compartment syndrome can develop quickly and vision loss can be rapid, thus prompt intervention within the first 90 minutes is paramount to prevent permanent damage [3].

However, not all peri-orbital hemorrhages result in compartment compression and its associated damaging sequelae. Hemorrhages in the pre-septal space, which may often be mistaken for retrobulbar hemorrhages by non-ophthalmic practitioners, typically require no intervention at all and true retrobulbar hemorrhages are more often than not clinically inconsequential [2]. Owing to the rare and emergent nature of retrobulbar hemorrhage, there are no clinical trials to guide therapy. As such, many of the recommended treatment algorithms may not be needed. This is complicated by the fact that, as previously stated, such patients may present to non-ophthalmic practitioners who are forced to deal with a possible emergent situation that is outside of their usual scope of practice. This leads to the inability to accurately judge ocular manifestations and subsequently the efficiency of treatment regiments can often not be accurately determined. It is unrealistic, however, to expect an ophthalmologist to appear within the needed time limit in a true emergent situation so the advised treatments are carried out whether needed or not.

Typical treatment is directed at lowering intraorbital or intraocular pressure to protect the optic nerve from damage. In patients with visual loss, proptosis, limited extraocular movements, afferent pupillary defect or sign of elevated IOP, medical management would include IV acetazolamide 500mg, or IM/IV hydrocortisone 100mg. Surgical treatment includes lateral canthotomy/cantholysis and septolysis an effective way of reducing intraocular pressure is used to monitor the intraorbital pressure. There are recommended as immediate treatment regimens, but in many scenarios are not needed. Lowering intraocular pressure is also attempted, yet many feel that it does little to address intraorbital pressure although experiments show there is a direct correlation between the two [5]. Tonometric pressure measurement of IOP can be a helpful indicator in guiding management [6]. Unfortunately, this may not be readily available.

Ultimately, the effectiveness or need of such treatments can only best be evaluated by an ophthalmologist. As stated previously, preseptal hemorrhage, although alarming in appearance, has little if any visual consequences and can be monitored. However, this is often quite difficult for a non-ophthalmologist to determine. Visual acuity, intraocular pressure, and status of intraocular arterial perfusion would be the best criteria for choosing a certain therapy and measuring its success. Unfortunately, the availability of this evaluation is unlikely. Thus, due to the exigency of the situation there is a necessity for the recommended treatments, regardless of their effectiveness or need. Most unfortunate is the fact that due to the limited time available to prevent visual loss unless the event occurs in the immediate setting to allow treatment, such intervention usually is too late.

Conflicts of interest

The authors report no conflicts of interest

REFERENCES