Principles of Protection of the Eye and Vision in Orbital Surgery

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Abstract

Orbital surgery can result in damage to ocular and orbital structures, leading to a range of structural and visual sequelae, including corneal abrasions, globe malposition, diplopia, and blindness. Vision loss in particular is the most feared and devastating complication, occurs with an overall incidence of 0.84%, and can occur secondary to direct injury, optic nerve compression, or ischemic events. Different types of orbital surgery and surgical approaches carry their own hazards, and it is important to be mindful of these risks in addition to having a thorough understanding of individual risk factors and anatomical variations for each patient. Although universal guidelines for preserving vision in orbital surgery do not yet exist, there are concrete steps that every surgeon can take at the preoperative, intraoperative, and postoperative stages to minimize the risk of injury and maximize the likelihood of preserving the eye and visual function.

Keywords

► orbital surgery
► orbit
► postoperative blindness
► postoperative diplopia

Introduction

Visual function requires proper functioning of the eye itself as well as the ocular adnexa including the eyelids, extraocular muscles, nerves, and vasculature. Damage to any of these structures can occur during orbital surgery, leading to deficits ranging in severity from temporary corneal dryness to irreversible blindness. Here, we review common causes of both blinding and nonblinding ocular complications in orbital surgery and outline basic strategies and precautions that surgeons can take preoperatively, intraoperatively, and postoperatively to minimize the risk of structural and visual complications.

Vision Loss

Blindness is the most feared and devastating potential complication of orbital surgery and can occur in a wide variety of procedures. Case reports have described vision loss after decompression for thyroid eye disease, removal of retrobulbar tumors, orbital fracture repair, and optic canal decompression, among others.1–3 A 2018 retrospective case study by Jacobs et al estimated the overall incidence of postoperative blindness in orbital surgery to be 0.84%, with a higher risk in intracranial orbital roof and apex surgery (18%), endoscopic trans sinus orbital apex surgery (10%), and a combined approach to orbital floor fracture repair (6%).4 In 2019, Kansakar and Sundar reported specifically on the incidence of blindness after orbital tumor excisions (4.7%), posttraumatic orbital reconstruction (2.08%), and orbital decompressions for thyroid orbitopathy (0.15%).5

Mechanisms of injury leading to vision loss in orbital surgery include direct ocular and optic nerve injury, optic nerve compression, and optic nerve ischemia. Direct optic nerve injury can occur intraoperatively from mechanical, thermal, and electrical sources.5 Additionally, the globes themselves may be penetrated by instrumentation or even inadvertently enucleated during functional endoscopic sinus surgery.6 Optic nerve compression can occur secondary to retrobulbar hemorrhage, particularly in patients who are on anticoagulants, or in the setting of prolonged and aggressive retraction, malpositioned implants, or other malpositioned surgical materials. Vision loss from ischemic causes can occur secondary to anterior ischemic optic neuropathy, posterior ischemic optic

published online August 7, 2020

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ISSN 2193-6331.
Pearls and Tips

- Pre-operative planning including management of anti-coagulation and careful choice of surgical approach are critical to preventing injury.
- Intraoperatively, protection of the various structures of the eye and visual apparatus must be constantly checked: lubrication of the cornea, traction on the optic nerve, pupillary response, proptosis, and hemorrhage.
- Following surgery, monitoring for vision loss and orbital hemorrhage should be performed by patient and surgeon immediately after surgery, and for the subsequent week by performing vision checks and having a rapid line of communication.

Neuropathy, central retinal artery occlusion, and ophthalmic artery occlusion. In Jacobs et al's 2018 review, 14 patients developed blindness from various causes: retrobulbar hemorrhage in four (29%) patients, malpositioned implant in two (14%) patients, ischemic events in two (14%) patients, optic nerve compression from seroma (7%), optic nerve compression from expansion of hemostatic packing agent (7%), and direct optic nerve injury (7%). In three of the patients with postoperative blindness, the etiology was indeterminate.4

In surgeries for orbital lesion removal, the risk of vision loss may vary based on specific lesion characteristics. For instance, removal of larger lesions results in a smaller working space making traumatic dissection, greater distortion of tissue, and aggressive prolonged retraction more likely.5 Lesions in contact with the optic nerve or invading the optic canal and orbital apex lesions in the superomedial vascular quadrant also carry a higher risk of removal.5 The nature of the lesion also affects prognosis: vascular, poorly encapsulated, cystic, and infiltrative lesions carry a higher risk, as do recurrent tumors.5 In general, age and preoperative visual status correlate with the risk of vision loss. Younger patients are better able to compensate for deprivation of blood flow, and a longer preoperative duration of vision loss is more likely to result in a poor postoperative visual outcome.5

Other Ocular Injuries

Nonblinding complications of orbital surgery include a wide variety of sequelae with varying degrees of seriousness. Potential complications include corneal dryness secondary to intraoperative exposure, direct orbital injury leading to corneal abrasions or perforations, eyelid malposition including ectropion, entropion, or retraction, globe malposition such as enophthalmos or hypoglobus, infection or exposure of implanted synthetic materials, postoperative diplopia, and postoperative hyposthesia.7,8 The incidence and type of complication vary depending on the type of orbital procedure performed. In a retrospective review of 93 orbits undergoing orbital decompression for thyroid eye disease, Jefferis et al found the most common complications to be temporary postoperative hyposthesia (29% of lateral decompressions, 17% of others) and new postoperative diplopia (9% of lateral decompressions, 39% of others).2

Postoperative diplopia can occur after any orbital surgery secondary to extraocular muscle transection or contusion, cranial nerve impairment, muscle or fat incarceration, or fascial scarring.9 New-onset diplopia with orbital decompression can occur due to the altered shape of the orbit and secondary effect on the course of the extraocular muscles. During medial orbital decompression specifically, blunt instrumentation and gentle handling of tissues limit iatrogenic damage.

In cases of functional endoscopic sinus surgery, the risk of orbital injury is higher with surgery of the ethmoid sinus given that the lamina papyracea is thin or even dehiscent, meaning that a well-defined surgical landmark delineating the boundary to the orbital space does not exist.10 Right-sided maxillary antrostomy is also associated with higher rates of orbital complications, which is attributed to difficulty in visualization and instrument manipulation of this particular area for a right-handed surgeon.9 Other variables that may increase the risk include anatomical variants of the sinuses or performing sinus debridement with mechanized systems.10,11

In general, studies do not appear to correlate the surgical approach (i.e., lateral versus anterior orbitotomy) with the risk of vision loss.4,12 However, the risk of certain types of nonblinding complications does appear to correlate with surgical approach. For example, lateral orbitotomy appears to confer a higher risk of scarring than an anterior approach due to the use of powered instruments for the lateral approach and an inherently more posterior or larger pathology necessitating bone removal.13 Jefferis et al noted in their study that lateral decompression for thyroid eye disease was more likely to lead to postoperative numbness and diplopia than other approaches.7

Pearls and Tips

Preoperative

There are several strategies and precautions that providers can take to minimize the risk of eye injury, vision loss, and structural complications in orbital surgery. The importance of patient selection, preoperative evaluation, and defining a threshold for surgical intervention coupled with patient counseling should not be underestimated. Risk factors such as anticoagulant use and vasculopathic comorbidities should be elicited from the history and a thorough preoperative ophthalmic examination should be performed. Discontinuation of nonessential anticoagulants should be discussed with the patient’s primary medical providers. In patients with a history of a bleeding disorder, appropriate laboratory workup should be performed, including prothrombin time, partial thromboplastin time, bleeding time, and platelet count.14

Appropriate imaging can guide preoperative planning and inform surgical approach as well as choosing the appropriate surgical team, which may comprise representatives from multiple specialties. A better understanding of the size,
location, and nature of the lesion will indicate possible outcomes of surgical intervention and expected recovery course. The choice of surgical approach should be tailored to each patient with consideration of possible postoperative complications more likely to arise with certain techniques based on adjacent structures, individual anatomical variation, or existing comorbidities. Minimizing the risk of orbital complications in functional endoscopic sinus surgery, in particular, can be achieved through a thorough understanding of the patient's individual anatomy. A comprehensive and detailed preoperative assessment in these patients should include CT (computed tomography) imaging to assess the extent of disease and detect any existing anatomical variations. Intraoperative image guidance, when coupled with experienced knowledge of anatomical landmarks, can help with orientation during surgery.

Selecting the appropriate timing for intervention is also important and requires a comprehensive understanding of the individual patient’s clinical case. In cases of retrobulbar hemorrhage, immediate intervention is crucial to minimize visual sequelae. In cases of compressive optic neuropathy due to thyroid eye disease or orbital tumors, the pressure should be alleviated as soon as possible.

**Intraoperative**

Topical ocular lubrication and a corneal shield should be applied over the cornea for physical protection of the eye. Intermittent removal of the corneal shields should be performed as needed for intraoperative pupillary assessment. Early identification of important anatomical landmarks, such as the lamina papyracea in functional endoscopic sinus surgery, is crucial in avoiding iatrogenic damage. To help identify defects in the lamina papyracea, the eye may be pushed gently into the orbit by the surgical team while observing the lateral ethmoid wall. Optic nerve and retinal ischemia can be prevented by avoiding protracted arterial hypotension and external compression of the orbit. The risk of iatrogenic optic nerve damage should be minimized through the use of appropriate surgical magnification and illumination during direct visualization, judicious use of bipolar rather than monopolar cautery within the orbit, delicate handling of tissues, and gentle periodic retraction with malleable retractors. The degree of wound closure is dependent on the degree of hemostasis and expected postsurgical swelling. A drain may be placed to allow evacuation of orbital bleeding and serous fluid.

Intraoperative monitoring of pupillary responses is beneficial in assessing the real-time health of the pupillary fibers. Thus, avoiding presurgical dilating drops or ocular contact with epinephrine from anesthetic injections is vital, and arranging the drapes to allow visualization of both eyes can be beneficial in pupillary assessment. The visualization of both eyes during surgery can also aid in functional endoscopic sinus surgery such that any movement resulting from traction on orbital fat can be quickly identified. Importantly, direct optic nerve injury does not cause pupil dilation. Rather, pupil shape and size differences are representative of traction on or damage to the pupillomotor nerve. The presence of a new relative afferent pupillary defect (which can only be determined by comparing the pupil response to light of both eyes) may denote optic nerve or severe retinal damage.

In addition to being able to check pupillary responses accurately, all surgeons who operate in the orbit should be well-versed in the signs of orbital compartment syndrome (OCS): rapid, tense orbital swelling, proptosis, globe immobility, elevated intraocular pressure, and later signs of hemorrhagic chemosis and a dilated pupil. Orbital surgeons should also know how to manage OCS and be well-equipped to perform an urgent lateral canthotomy and cantholysis if warranted. In some cases, lateral canthotomy and cantholysis is not sufficient to treat OCS and incision into the eyelid crease with hematoma evacuation, extended fasciotomy (via incision of the orbital septum), or orbital decompression may be required. In some circumstances, it may be prudent for orbital surgeons who are not trained in ophthalmology to work in close conjunction with an ophthalmologist in the intraoperative and postoperative periods.

The risk of retrobulbar hemorrhage can be diminished intraoperatively through meticulous hemostasis, intermittent orbital tamponade, and bipolar cautery of bleeding vessels. To further minimize the risks of postoperative retrobulbar hemorrhage, a “deep” extubation should be performed to reduce coughing during anesthesia reversal. In addition, head elevation should be maintained, pain and blood pressure should be meticulously controlled, and postoperative nausea and vomiting should be minimized.

**Postoperative**

Patients who have had orbital surgeries with higher risk of vision loss or who have other high-risk features (i.e., orbitotomy resulting from orbital fracture, comorbid facial fractures, anticoagulant use) should have frequent serial monitoring postoperatively, and thus every orbital surgeon should be able to reliably and consistently conduct a vision and pupillary assessment. Reports of the timing of postoperative retrobulbar hemorrhage range from the immediate postoperative period to up to 7 days after surgery, with most episodes occurring in the first 10 hours. This supports keeping high-risk patients in the hospital for inpatient observation given that rapid identification and treatment of optic nerve compromise (in 60 minutes or less) can be crucial in preventing permanent vision loss. Obtaining postoperative orbital imaging is recommended if there were intraoperative features or postoperative examination findings that suggest causes of vision loss potentially visible on imaging. When patients are discharged, they, along with family members or caretakers, should be trained in doing vision checks at home, with instructions to contact the care team urgently if problems arise.

Patients who develop diplopia after functional endoscopic sinus surgery should be imaged to identify and characterize possible extraocular muscle injury, and findings should be correlated with a thorough ophthalmological examination. In cases of suspected acute extraocular muscle damage such as muscle transection, tissue incarceration, or embedded bone fragments, early surgical intervention should be
performed before fibrosis occurs. Expedient referral to an orbital and/or strabismus surgeon is essential for timely evaluation and treatment. Other forms of impairment such as muscle contusion or neurovascular injury may be treated more conservatively with careful observation.

**Conclusion**

Overall, there are significant and varied potential ocular and visual risks inherent in orbital surgery that all orbital surgeons should be aware of. Staying mindful of potential complications that can occur with specific procedures or surgical approaches as well as understanding the individual risk factors and anatomical variations that exist in each patient is imperative in preoperative counseling and surgical planning. Appropriate use of ophthalmology consultation and collaboration across medical and surgical fields, in general, is also crucial. There are numerous steps at the preoperative, intraoperative, and postoperative stages that surgeons can take to minimize complications and maximize the chances of preservation of ocular and visual function.

**Conflict of Interest**

None.

**References**