Iatrogenic Visual Loss After Orbital and Eyelid Surgery

21

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Introduction

Perioperative visual loss (PVL) has been described in many types of surgery. The risk of PVL after any surgery in general was estimated to be about 0.002 % [1]. Not surprisingly, the risk of PVL is highest among patients undergoing cardiac, spinal or ophthalmic/neurologic surgeries, risk estimated to be about 0.086 %, 0.03 % and 0.05 %, respectively [1–3].

For most non-ophthalmic procedures, the face-down positioning (especially for spinal and neurosurgical procedures) has long been blamed as one of major causes for the visual loss, partly explained by the unintentional pressure over the periorbital region by the headrest in some cases, while some believe that compromise of orbital circulation due to raise in intra-orbital pressure caused by prone positioning itself may be a more important factor [4].

Visual loss after non-ocular ophthalmic procedures encompasses mainly orbital and eyelid surgeries. Though rare, it can result in major sequelae to both the patients and surgeons. A few studies have attempted to elucidate the incidence, aetiology, clinical course and optimal management of this devastating complication [4–7]. However, due to its rarity, different inclusion criteria and definition of visual loss used in various studies, this condition still remains not fully understood and unfamiliar to some practitioners.

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Visual Loss After Orbital Surgery

The incidence of visual loss after orbital surgery in general was estimated to be about 0.44–0.6 % [4, 5]. In the study by Bonavolonta, which was one of the early studies on the incidence of visual loss after orbital surgery, all patients with preoperative visual acuity of <20/40 were excluded [5]. This may theoretically excluded those higher risk cases for visual loss, which resulted in a lower estimated risk of 0.44 % [5]. Moreover, the definition of visual loss was not standardized in various studies, which made comparison between studies not meaningful.

On the other hand, different procedures on different locations carry different risk. Orbital decompression was found to carry a lower risk of visual loss comparing to orbital exploration for tumour removal or biopsy [4]. In the study by Rose published in 2007, there was no case of visual loss after 1350 orbital decompression, whereas there were 14 cases of visual loss in 1150 orbital exploration (1.2 %) [4].

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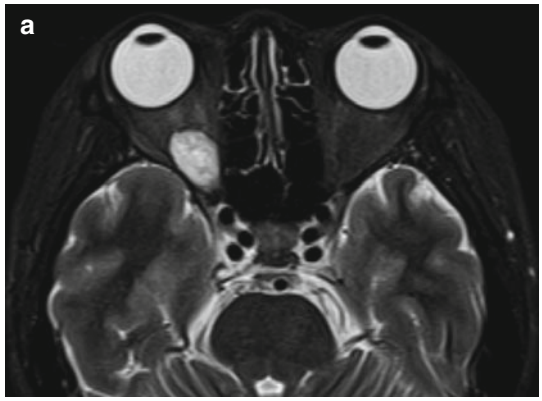

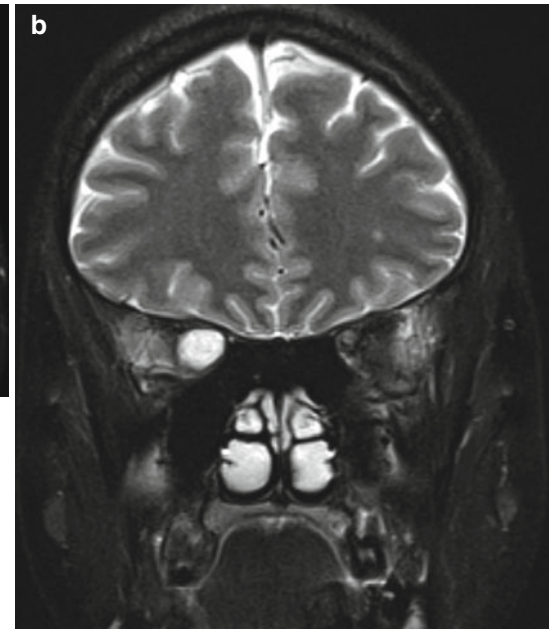

Fig. 21.1 (**a**) MRI T2-weighted axial scan of patient with right optic apex cavernous venous malformation (cavernous haemangioma): axial scan. (**b**) MRI T2-weighted axial scan of patient with right optic apex cavernous venous malformation (cavernous haemangioma): coronal scan

Two major mechanisms were postulated to be the major causes of the post-operative visual loss after orbital surgery, namely, ischaemic optic neuropathy and retinal artery occlusion due to vasospasm [4, 5]. Manipulation around the posterior part of intra-orbital optic nerve is of particular risk since this part of the optic nerve has a sole blood supply from the perforating dural vessels [4]. Interruption of the blood supply due to vasospasm may cause irreversible ischaemic optic neuropathy, depending of the duration of ischaemia.

With the knowledge of the possible pathophysiology for visual loss after orbital surgery, it is not surprising that certain disease condition and patient characteristics carry a higher theoretical risk of post-operative visual loss.

Risk Factors

It is thought that larger lesions, intraconal lesions, especially located around the orbital apex, and lesions that are in contact with/alongside the optic nerve or even displacing the middle and/or posterior third of the optic nerve are of higher risk [4]. The highest risk lesions are those "peanut" lesions wedged in the orbital apex and massive vascular malformations [4] (Fig. 21.1a, b). This may be related to the need for additional surgical manipulation around the optic nerve, which may induce reversible vasospasm of the perforating dural vessels or even transecting the vessels. Excessive bleeding (with free blood and inflammatory mediators) and use of bipolar cautery may also induce the vasospasm [4]. The former may also lead to an intraoperative "steal syndrome" compromising the blood supply to the optic nerve [4].

For patients with preoperative gaze-evoked visual obscuration, it is thought that the risk of post-operative visual loss due to cilio-retinal artery occlusion may be higher [4]. Rose postulated that the transient visual obscuration was due to a critical impairment of optic nerve

head perfusion and this predisposed to post-operative vascular occlusion [4].

Delayed ischaemic optic neuropathy may also occur due to post-operative orbital swelling or haemorrhage compromising the blood supply or accumulation of inflammatory mediators which may induce arterial spasm [4, 5].

Other factors that might conjecturally increase the risk of visual loss include preoperative dehydration, intraoperative hypotension, atherosclerotic disease or other vascular risk factors including migraine [4, 5].

Management

There is currently no universally accepted treatment for postorbital surgery visual loss. Treatments are mainly anecdotal and unproven.

For patients with visual loss due to retinal artery occlusion, traditional treatment for vasodilation including rebreathing bag, systemic acetazolamide or paracentesis may be considered, though all are not of any proven benefit. High-dose systemic steroid, one of the commonly anecdotal treatment prescribed, is also not of proven benefit [4, 5].

Several measures have been suggested to reduce the occurrence of post-operative visual loss. Avoiding prolonged globe and orbital retraction, prolonged surgery and extensive use of bipolar diathermy may reduce post-operative inflammatory debris and hence the risk of vasospasm; high-dose systemic steroid during and after surgery may also theoretically reduce the post-operative inflammation; placement of a vacuum drain to clear fluid from the operative site and positioning patient in a semi-recumbent position from immediately after surgery may help to reduce the intra-orbital venous pressure [4]. The use of lignocaine with adrenaline should be avoided for orbital surgeries as the adrenaline component may cause vasospasm of the central retinal artery potentially leading to retinal ischaemia. Checking of visual function and intraocular pressure should be done in the early post-operative for early detection of visual problem. Further workup including imaging with CT scan should be initiated if necessary.

Visual Loss After Eyelid Surgery

Visual loss after eyelid surgery is a rare and even more unexpected complication as perceived by the patient. The most commonly described eyelid surgery with this complication in the literature is cosmetic blepharoplasty [8]. It is estimated to occur in about 0.0052 % blepharoplasty and permanent visual loss in about 0.0019 % [7].

The most common cause of the visual loss is retrobulbar haemorrhage. It is not surprising that this occurred mainly at two peaks: intraoperative to one post-operative hour and the second peak between 6 and 12 post-operative hour due to secondary haemorrhage [7]. Risk factors for retrobulbar haemorrhage include the use of anti-platelet or anticoagulating agents, use of certain herbal supplements (gingko biloba, ginseng, garlic), suboptimal controlled hypertension and suboptimal haemostasis during surgery [7]. Rarely, visual loss can be due to inadvertent corneal perforation during lid anaesthesia [6].

The most common presenting symptoms are pain, pressure sensation and proptosis [7]. If the patient is suspected to have retrobulbar haemorrhage and impending visual loss, treatment should be initiated immediately so as to prevent irreversible visual loss. Lateral canthotomy and inferior cantholysis to relieve the intra-orbital pressure should be considered. Orbital decompression can also be attempted in selected case. As in postorbital surgery visual loss, high-dose steroid is also used by some, but its use is again anecdotal and not well proven.

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