Ocular Complications

14

Rick Catterwell, Emad Aziz Girgis, G. Blecher, and P. Dasgupta

Introduction

Postoperative visual loss (POVL), including blindness, is a rare but devastating complication that has been reported following a wide range of procedures including robotic urological surgery [1, 2]. While patients assume a certain risk of visual loss when undergoing ophthalmic surgery, visual loss following elective non-ocular surgery is a catastrophic event for the patient, surgeon and anaesthetist [3]. Despite being a rare entity, perioperative ocular complications in non-ophthalmic surgery have become a focus for surgical, anaesthetic and neuro-ophthalmological literature and a contentious medicolegal issue.

Postoperative ocular injuries include a broad spectrum of conditions each with distinct aetiologies, risk factors, patterns of visual loss, treatment and prognoses [4]. Procedures complicated with prolonged steep Trendelenburg positioning, significant blood loss, haemodynamic perturbations and prolonged pneumoperitoneum should be recognized as higher risk for POVL and visual assessment part of the postoperative assessment [5]. When a patient reports any visual symptoms following surgery, an urgent ophthalmologic consultation should be obtained to determine its cause [6]. Initial ophthalmological assessment focuses on identifying the location of the lesion via direct examination and if no ocular injury or central retinal artery occlusion is apparent, urgent neuroimaging with MRI is recommended [3].

Corneal abrasion is the most common ophthalmic injury in the perioperative period [7]. Robotic-assisted urological surgeries, in particular those associated with Trendelenburg positioning, have been associated with a very high risk for corneal abrasion [7]. Although

R. Catterwell, MBBS, FRACS (Urol) (✉)
Guys & St Thomas' NHS Trust, Urology Department, Great Maze Pond, London SE1 9RT, UK

King's College London, Mail via Guys & St Thomas' NHS Trust, Urology Department, Great Maze Pond, London SE1 9RT, UK

Guys & St Thomas' Trust/King's College Hospital, Department of Urology, Great Maze Pond, London SE1 9RT, UK
e-mail: rick.catterwell@gmail.com

E.A. Girgis, MBBS, FRCA
Guys & St Thomas' NHS Trust, Department of Anaesthesia, Great Maze Pond, London SE1 9RT, UK
e-mail: dremadaziz@gmail.com

G. Blecher, MBBS, FRACS (Urol)
Guys & St Thomas' NHS Trust, Urology Department, Great Maze Pond, London SE1 9RT, UK
e-mail: gidsblecher@gmail.com

P. Dasgupta, MSc, MD, FRCS (Urol), FEBU
King's College London, Mail via Guys & St Thomas' NHS Trust, Urology Department, Great Maze Pond, London SE1 9RT, UK
e-mail: prokarurol@gmail.com

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corneal abrasions generally resolve quickly with limited treatment and no long-term sequelae, they are painful and anxiety inducing for the patient [8]. In direct contrast, other causes of POVL have poor prognoses and lack of validated treatment options [9]. Although these conditions are rare, they are frequently associated with complete unilateral or bilateral visual loss with the majority of cases having permanent effects. Due to their devastating impact and lack of effective treatment, prevention of these injuries is crucial.

Causes of Ocular Injury Following Robotic Surgery

Ocular injuries following robotic surgery can be categorized into five groups. Each is associated with a degree of postoperative visual loss (POVL).

- External ocular injury (corneal abrasion)
- Retinal ischaemia
- Ischaemic optic neuropathy (ION)
- Cortical blindness
- Acute glaucoma

Each of the aetiologies will be discussed separately in relation to pathophysiology, incidence, diagnosis and management.

External Ocular Injury

Direct corneal trauma can result in irritation, abrasion or laceration. Corneal abrasion (CA) is the most common ophthalmic injury in the perioperative period. Published data report an incidence range of 0.11–4.4% [7, 10, 11]. Segal et al. reported on a retrospective series of over 78,000 patients having procedures requiring anaesthesia with 0.11% of patients suffering CA [7]. They reported the most common procedure associated with CA was robotic-assisted prostatectomy. Independent significant risk factors for CA were Trendelenburg and prone positioning, prolonged operative time, increased estimated intraoperative blood loss and general anaesthesia.

In robotic surgery corneal injury may result from direct mechanical force such as from robotic instruments or chemical interaction from gastric reflux. Trendelenburg positioning is associated with elevated intravascular, episcleral venous and intraocular pressure which may result in increased corneal thickening. Longer and more complicated surgical procedures may ultimately compromise the vitality of the corneal epithelial cells with an increased propensity for sloughing and abrasion [7, 10].

Corneal abrasion typically presents as blurred vision, tearing, redness and foreign body sensation in the eye. Diagnosis is confirmed with the aid of fluorescein staining of the cornea and examination under a cobalt blue light.

Treatment with a broad spectrum topical antibiotic is usually rapidly effective [8]. Corneal injury prevention involves taping and application of lubricants to prevent corneal dehydration and eye shields to prevent mechanical insults [12, 13].

Retinal Ischaemia: Branch and Central Retinal Artery Occlusion

Central retinal artery occlusion (CRAO) decreases the blood supply to the entire retina, whereas *branch retinal artery occlusion* (BRAO) affects supply to a portion. The majority of instances of perioperative retinal artery occlusion are unilateral and secondary to improper patient positioning resulting in external compression of the eye [4, 14]. External compression of the eye can produce sufficient intraocular pressure (IOP) to stop flow in the central retinal artery which has, in animal models, been demonstrated to result in irreversible retinal damage in 20–60 min [15, 16]. Following removal of external compression reperfusion can result in increased swelling and further increases in compartmental pressure. Orbital compartment syndrome can ensue resulting in increased retinal ischaemia and retinal cell

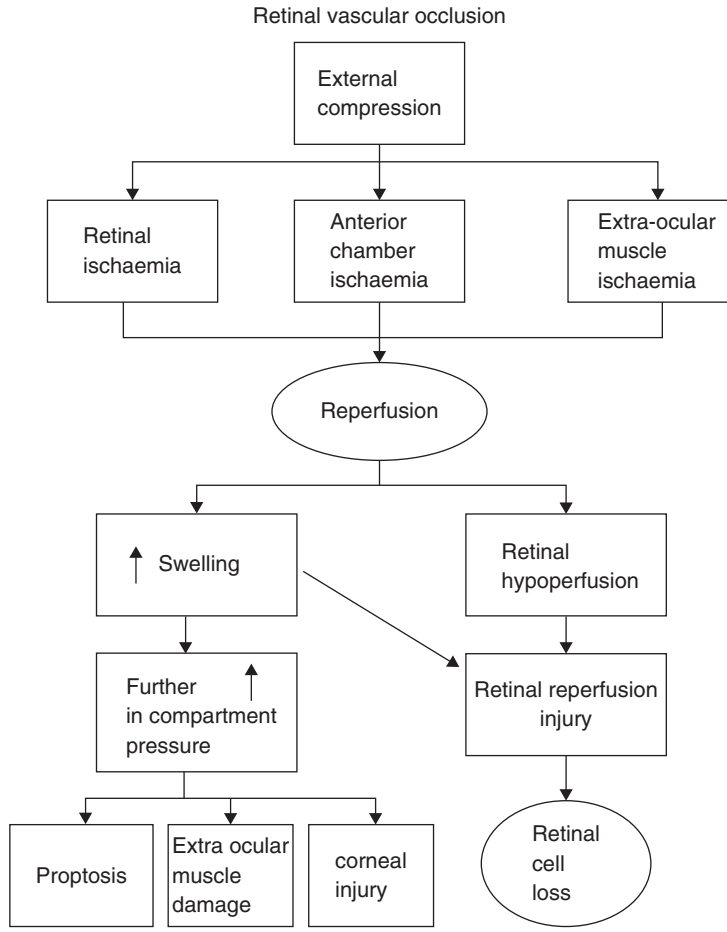

Fig. 14.1 Mechanism of injuries resulting from external ocular compression and resulting reperfusion injuries

loss [4] (Fig. 14.1). Although this cause of POVL is predominately associated with prone positioning, it can occur in any surgery where prolonged external pressure is inadvertently applied to the eye [17]. There have been no reported cases associated specifically with robotic surgery.

Other rare causes of CRAO include embolism to the retinal circulation, decreased blood flow secondary to systemic hypoperfusion, impaired venous drainage of the retina or coagulation disorder [18].

Signs and symptoms of patients with postoperative CRAO include painless unilateral visual loss, no light perception, afferent pupil defect, periorbital oedema, chemosis, proptosis, ptosis paraesthesia of the supraorbital region and corneal abrasion [19]. Diagnosis is prompted by the sudden onset of visual loss and the presence of retinal whitening with or without classical 'cherry-red' macula on fundoscopy (Fig. 14.2).

Prognosis for CRAO is generally poor and treatment inadequate. Cold compress, ocular massage and vasodilatation via induced hypercapnia have been advocated in presentations less than 90 min. Paracentesis may facilitate distal migration of the embolus limiting extent of injury. Fastidious attention to patient positioning aimed at avoiding external ocular pressure is paramount in prevention of CRAO.

Branch retinal artery occlusion (BRAO) causes permanent ischaemic retinal damage with partial visual field loss. BRAO is primarily the result of emboli. The vast majority of reported cases are associated with cardiopulmonary

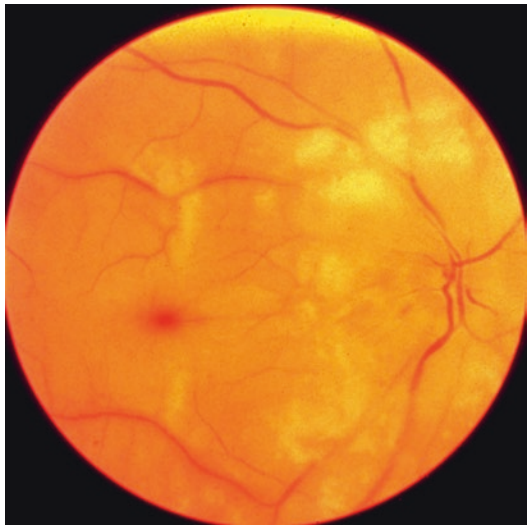

Fig. 14.2 Fundus photography of the right eye with non-arteritic CRAO demonstrating cherry-red spot and retinal opacity of the posterior fundus (Reprinted from Hayreh, Sohan Singh. Ocular Vascular Occlusive Disorders. © Springer International Publishing, Switzerland 2015. Chapter 13, Central Retinal Artery Occlusion; p. 239. With permission of Springer Nature)

bypass where circulating embolic material is implicated. Embolism passage from surgical site via the venous system and a patent foramen ovale has been reported as a cause of perioperative retinal vascular occlusion in spinal surgery [20]. BRAO is associated with painless partial visual field loss and sectoral whitening in the path of a branch retinal artery on fundoscopy.

Ischaemic Optic Neuropathy

Ischaemic optic neuropathy (ION) refers to ischaemic damage to the optic nerve itself. ION is subclassified into arteritic or non-arteritic ION. Arteritic ION is secondary to inflammation of blood vessels chiefly associated with giant cell/temporal arteritis and responds to steroid therapy. In contrast, non-arteritic ION is secondary to occlusive disease or other noninflammatory disorders. In the general population, non-arteritic ION is the leading cause of sudden visual loss in patients above 50 years of age with an annual incidence in the United States of 82 per 100,000 persons [21]. Non-arteritic ION is the overwhelming cause of POVL. It has been reported after a wide spectrum of surgical procedures, most commonly cardiothoracic surgery [18], instrumented spinal fusion [22] and head and neck surgery [23, 24]. Multiple cases following gynaecological, urological and general surgical procedures have also been reported [25].

ION is further classified by the location of the nerve ischaemia into anterior ischaemic optical neuropathy (AION) and posterior ischaemic optic neuropathy (PION). This classification is of importance due to the difference in incidences, proposed aetiologies and clinical presentations of each group. Postoperative AION predominately occurs following cardiothoracic surgeries. All reported cases of POVL secondary to ION related to robotic pelvic surgery have been PION injuries [1, 25]. Similarly the vast majority of reported ION following spinal surgery have been posterior injuries.

The exact mechanism of PION and AION is contentious and likely multifactorial. Posterior ischaemia occurs behind the globe and is probably not related to predictable increases in intraocular pressure; it may well be related to disruption of blood supply to the optic nerve from a network of very small perforating pial arteries (Fig. 14.3). In contrast AION proposed to be caused by disruption of blood supply through the posterior ciliary arteries feeding the head of the optic nerve, and this condition may be related to impaired autoregulation of flow (perfusion pressure vs intraocular pressure).

AION and PION have been reported in the setting of massive fluid replacement especially in prone-positioned patients. Excessive fluid administration could result in increased IOP or accumulation of fluid in the optic nerve or both. As the retinal vein exits out of the optic nerve, the oedematous nerve may inhibit venous outflow resulting in an internal 'compartment syndrome' [4]. Patients on the ASA Postoperative Visual Loss Registry received on average 9.7 L of crystalloids intraoperatively, suggesting that fluid replacement may play a role [26].

Key surgical factors linked to perioperative ION are prolonged prone or steep Trendelenburg positioning, prolonged overall surgical duration and massive blood loss. Possible intraoperative

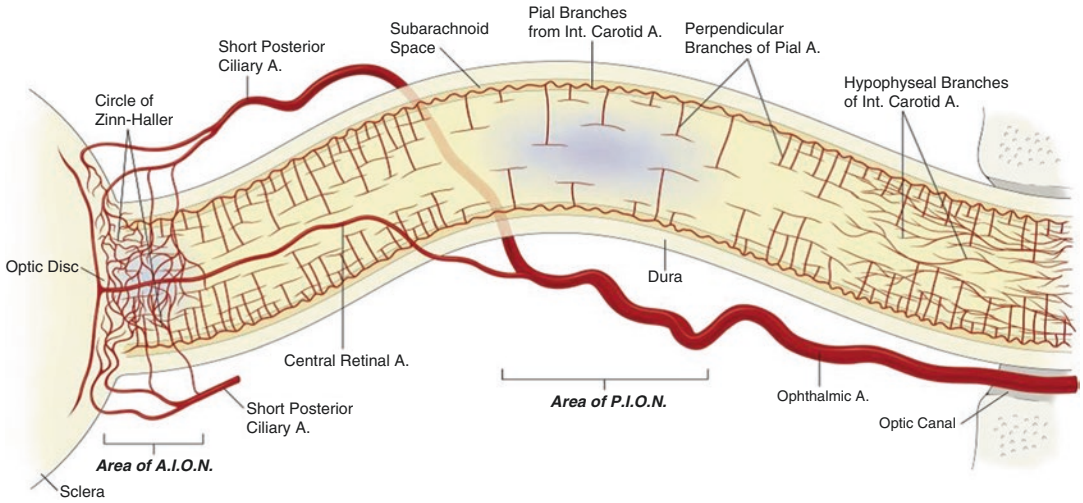

Fig. 14.3 Diagram of the orbital optic nerve and arterial supply. Areas implicated in ischaemic optic neuropathy are indicated in *blue*. The mid-orbital optical nerve has a paucity of blood supply compared to the anterior component. This area supplied by only the pial branches is the region involved in PION. The pial branches have variable density and in an unusual perpendicular T-shaped pattern, characteristic of a low pressure system. There is low density of arteriolar and capillary supply to this mid-orbital segment compared with the canalicular or retrobulbar segments of the optic nerve. Abbreviations: *A* artery, *AION* anterior ischaemic optic neuropathy, *PION* posterior ischaemic optic neuropathy

haemodynamic factors include decreased systemic blood pressure, anaemia or haemodilution, a high ratio of crystalloid to colloid fluid replacement and venous congestion. Characteristics of the optic nerve and disc may predispose to ION such as reduced flow of cerebrospinal fluid, abnormal auto regulation, anatomic variants in blood supply and small cup-to-disc ratio. Potential systemic risk factors include hypertension, diabetes, atherosclerosis, hyperlipidaemia, smoking history and hypercoagulability [4, 14, 18, 26, 27]. Minimization of these potential risk factors where possible is the basis of ION prevention.

Typically PION results in complete visual loss within 24 h postoperatively compared to AION where two thirds of cases were not evident until more than 24 h following surgery and initial symptoms more likely to be incomplete visual loss. Bilateral visual loss is more common with PION (63%) compared with AION (52%). Nearly all patients with AION have disc oedema, pallor or both on initial assessment (Fig. 14.4). In comparison PION is associated with a normal optic disc on initial fundoscopic evaluation in 92% of patients [4, 14].

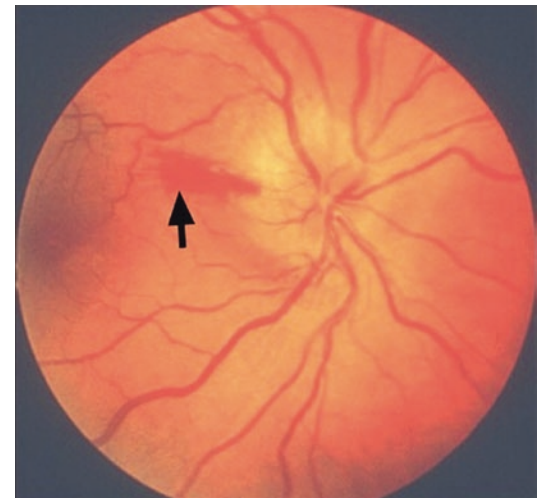

Fig. 14.4 Fundoscopy in acute non-arteritic anterior ischaemic optic neuropathy. The optic disc is oedematous and hyperaemic. Splinter haemorrhages (*Arrow*) are present

No effective treatment for ION has been proven. Only approximately 30% of patients with

either AION or PION will have any improvement. The focus of management is therefore on prevention.

Cortical Blindness

Cortical blindness is the result of decreased perfusion to the occipital cortex by tributaries of the posterior cerebral artery. The cause is either hypoperfusion or embolic phenomenon. Cortical blindness is a very rare cause of POVL that is usually associated with cardiac surgery [4, 28].

As the optic tracts and radiations are preserved, patients with cortical blindness have normal light reflexes and fundoscopic examination is normal. With unilateral involvement visual field examination demonstrates contralateral homonymous hemianopia. Bilateral involvement results in peripheral visual loss or complete blindness [28]. Diagnosis is confirmed in both unilateral and bilateral conditions via MRI with gadolinium.

Cortical blindness is usually accompanied by signs of acute stroke in the parieto-occipital region. Patients frequently demonstrate agnosia (an inability to interpret sensory stimuli) and impaired spatial perception. Focal neurological signs suggestive of stroke extension may be evident.

Treatment is aimed at preventing extension of the cerebral infarction. Most described preventative measures discuss reducing risk of embolic phenomena with cardiac surgery, but in the context of robotic surgery, prevention is via maintenance of global cerebral perfusion. Visual recovery in cortical blindness is usually prolonged and incomplete [4, 14].

Acute Glaucoma

Acute angle-closure glaucoma has been described rarely after general anaesthesia. Patients are genetically predisposed with a shallow anterior chamber and thick lens. Presentation is with a painful red eye and blurred vision usually accompanied by headache, nausea and vomiting. The pupil is mid-dilated with a pupillary block and the condition is often bilateral. It should be differentiated from corneal abrasion, which also produces pain but without papillary signs, increased IOP or headache.

Acute angle-closure glaucoma is an ophthalmological emergency as prolonged elevated intraocular pressure will result in glaucomatous damage to the optic nerve. Acute management is with topical α-agonists, β-antagonist, cholinergic agonists and steroids.

Approach to the Patient with Perioperative Visual Loss

So with all this background knowledge, the question remains…. What is the management of a patient that awakens from general anaesthesia with complains of visual loss?

Vision should be assessed early in all patients following high-risk surgery which includes robotic pelvic surgery especially if complicated prolonged steep Trendelenburg positioning, significant blood loss, transfusion or intraoperative haemodynamic instability. If there is concern regarding potential visual loss, an urgent ophthalmologic consultation should be obtained to determine its cause. If an ocular cause, such as corneal injury or central retinal artery occlusion, is not apparent, urgent neuroimaging should be obtained. Gadolinium-enhanced MRI is preferred assessing for intracranial pathology such as occipital infarction. If imaging is unremarkable, ION is the likely cause of which PION is most likely given normal fundoscopy. Additional management may include optimizing haemoglobin levels, haemodynamic status and arterial oxygenation, but little evidence exists for the efficacy of any interventions for ION.

Conclusion

Robotic urological surgery has one of the highest rates of corneal abrasion of all surgical procedures. POVL in robotic surgery is a rare but catastrophic complication. Potential causes of POVL after robotic surgery include anterior ischaemic optic neuropathy, posterior ischaemic optic

neuropathy, cortical blindness, retinal ischaemia and acute glaucoma. The vast majority of cases are related to posterior ischaemic optic neuropathy. The exact risk factors and pathophysiological mechanism of ischaemic optic neuropathy are poorly understood and likely multifactorial.

Given the complete lack of effective treatment modalities, prevention is crucial for limiting the incidence and destruction of POVL. Minimization of presumed risk factors is particularly important in robotic pelvic surgery where minimizing duration of Trendelenburg positioning, overall operative time and blood loss are likely protective.

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