Orbital haemorrhage and prolonged blindness: a treatable posterior optic neuropathy

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SUMMARY Two patients developed traumatic orbital haemorrhage resulting in prolonged blindness. Orbital decompression successfully reversed the visual loss, though both eyes had no light perception for at least 3½ hours. The clinical findings suggest damage to the posterior optic nerve as cause of the visual loss. The optic nerve tolerance time to ischaemic injury may be longer than previously thought.

Visual loss secondary to orbital haemorrhage has been appreciated by clinicians in the fields of ophthalmology and otolaryngology as well as those in plastic, dental, and neurological surgery.¹⁻¹⁷ Most frequently it is seen following trauma.¹⁸ Visual loss secondary to increased orbital contents from an infectious process has been seen in cellulitis and sinusitis,¹⁹⁻²² with orbital foreign body and abscess formation,²³ and as a sequela of delayed treatment of a dental abscess.²⁴ 'Spontaneous' orbital haemorrhage has been recorded with local disease^{18,25-27} and associated with concurrent systemic disease.^{18,28-31}

Suggested explanations for this visual loss include central retinal artery or vein occlusion, acute glaucoma, anterior ischaemic optic neuropathy, reflex vasospasm of the retinal vasculature, toxic or metabolic ischaemia, optic nerve sheath haemorrhage, optic nerve avulsion, and excessive tugging by orbital fat on nutrient vessels to the optic nerve.

We recently treated 2 patients with total loss of vision from traumatic orbital haemorrhage who had return of vision after prolonged periods of blindness. The purpose of this report is to review these 2 cases and to discuss the likely pathophysiology and appropriate therapy.

Case reports

CASE 1

While working with a screwdriver a 37-year-old male accidentally slipped, causing a puncture injury to his

left orbit superonasally. Immediately after the accident the vision was normal and there was minimal external bleeding. Within 30 minutes he was aware of loss of vision, with periorbital pain and swelling. An examination was performed by an ophthalmologist approximately one hour after the accident. Visual acuity was 20/20 OD, no light perception OS. The left orbit was tense, the left pupil in mid position and unreactive, and ductions of the left eye were restricted to all fields of gaze. Anterior chamber paracentesis was performed without return of vision.

The patient was seen at the University of Arizona for further evaluation 4 hours after the accident and some 3½ hours after the onset of his total blindness OS. Visual acuity was 20/20 OD, no light perception OS. A 6 mm laceration was noted above the left upper eyelid. The left globe was proptotic and there were extensive periorbital ecchymoses. The left pupil had no direct but normal consensual response. The left cornea was clear, but the anterior chamber was shallowed. Examination of the fundus showed what appeared to be normal retinal vasculature and choroidal blood flow bilaterally, with no signs suggestive of oedema or intraretinal haemorrhage.

The patient was treated with 100 g of mannitol intravenously, and a lateral canthotomy was performed. During the canthotomy the patient reported return of vision, and acuity was recorded as hand movements. The wound was then explored and the orbit decompressed through a transconjunctival approach. No obvious clots or localised areas of haemorrhage were identified. The anterior chamber spontaneously reformed during the procedure, and indirect ophthalmoscopy again revealed a normal

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Fig. 1 Case 1. Left eye. Optic disc and peripapillary retina 17 days after trauma.

posterior pole with apparently normal vasculature and blood flow.

On the first postoperative day visual acuity had improved to counting fingers at 3 feet (90 cm). Proptosis had decreased, the left pupil showed a mild afferent defect, and ocular motility had returned to normal. The cornea was clear and the anterior chamber deep. The left fundus showed normal retinal

Fig. 2 Case 1. Left eye. Optic disc and peripapillary retina 6 weeks after trauma, illustrating optic atrophy and loss of nerve fibre layer.

and choroidal vasculature, without evidence of retinal oedema. Visual field testing on a Goldmann perimeter revealed a large centrocoecal scotoma breaking out to the periphery, associated with an altitudinal defect seen only with small isoptres.

Seventeen days after his initial accident visual acuity had returned to 20/60, while his afferent pupillary defect remained. Fundus examination showed the disc with minimal temporal pallor (Fig. 1). Visual field testing showed a paracentral scotoma and persisting altitudinal defect. Six weeks after the accident his visual acuity had returned to 20/40, though his pupillary reaction and visual field remained unchanged. The fundus showed diffuse loss of nerve fibres, with temporal pallor of the disc (Fig. 2).

CASE 2

A 65-year-old woman fell to the ground, injuring her left periorbital region. The skin was not broken and vision was normal. Within 5 minutes of the injury she noted pain and swelling about the left orbit and total visual loss in her left eye.

On presenting to the Ophthalmology Clinic 3 hours after injury her visual acuity was measured at no light perception OS. The globe was proptotic, there were periorbital oedema and ecchymoses, and the pupil was fixed and in mid position. The cornea was clear and the anterior chamber was formed. Intraocular pressure by applanation was 57 mmHg. Faint pulsations were observed in the central retinal artery. Immediate surgical decompression of the orbit by lateral canthotomy and cantholysis was performed. Within 30 minutes of this treatment, which occurred some 4½ hours after her injury, the patient's vision returned to light perception and her intraocular pressure fell to 31 mmHg. Over the next hour the visual

Fig. 3 Case 2. External anatomy, one day following orbital decompression.

Fig. 4 Case 2. Left eye. Optic disc and peripapillary retina one day after trauma.

acuity improved to 20/80, and intraocular tension decreased to 21 mmHg.

By the next day her visual acuity had improved to 20/30 OS, and proptosis had decreased (Fig. 3). An afferent pupillary defect was noted. Ocular motility was full, though the patient complained of pain on upgaze. The posterior pole was normal to direct and indirect ophthalmoscopy (Fig. 4). Visual field testing to confrontation was normal. Fluorescein angiography showed normal retinal and choroidal circulations, without signs of retinal or disc oedema (Fig. 5). X-rays showed the presence of an orbital floor fracture on that side.

Fig. 5 Case 2. Left eye. Fluorescein angiography one day after trauma, illustrating normal pattern of retinal and choroidal circulation during early A-V phase.

Discussion

Visual loss from orbital haemorrhage is related to the surgical anatomy of the orbital cavity. The orbit is a poorly expandable space comparable to the cranial cavity. It is bounded posteriorly and on its sides by rigid bony structures. Anteriorly it is limited by the globe and the orbital septum. Any demand for expansion of this orbital space can be met only by the anterior displacement of the globe and septum. There is some limited capacity for forward displacement by stretching of the septum, but once this limit of proptosis has been reached the globe acts as a plug in a fully distended palpebral fissure. The orbit is then 'sealed' by its anterior 'wall'. That the orbit is sealed posteriorly as well is shown by the fact that orbital haemorrhages do not spontaneously decompress themselves by dissecting intracranially.

When orbital haemorrhage occurs, the globe moves forward until it reaches the orbit's anatomical boundary. As further bleeding occurs into the orbital space, which is now a closed cavity, there can be little increase in volume to accommodate any increase in pressure, which is distributed equally throughout the entire orbital contents. It is this increased orbital pressure acting on the vascular supply of the globe and optic nerve which probably accounts for reversible visual loss from orbital haemorrhage.

Three possible separate circulations may be affected: the retinal circulation, the choroidal circulation, or the blood supply to the optic nerve itself. The normal appearance of blood flow in our patients' retinal and choroidal circulations as observed by ophthalmoscopy and shown by fluorescein angiography speaks for the integrity of the retinal and choroidal vasculature. The presence of an intact consensual response of the ipsilateral pupil (case 1) demonstrates some integrity of the iris sphincter, and therefore the anterior ciliary circulation. The lack of return of vision after anterior chamber paracentesis (case 1) makes it unlikely that high intraocular pressure (with choroidal or retinal ischemia) is causative.

The visual loss we observed was not associated with signs of retinal, choroidal, or disc oedema. It was characterised by return of central acuity, though accompanied by an afferent pupillary defect, diffuse loss of nerve fibres, descending optic atrophy, and an altitudinal field defect (case 1). These signs carry the signature of optic nerve dysfunction, and are best explained by a posterior injury to the optic nerve. A recent report of a case of visual loss from orbital haemorrhage in which the visual evoked response (VER) was unrecordable at a time when the ERG was normal^{1,2} lends electrophysiological evidence to this entity's being an optic neuropathy.

Whether the optic nerve dysfunction is due to ischaemia as a primary event or secondary to compression cannot be ascertained. Precisely where in its intraorbital or intracanalicular course the optic nerve is compromised remains conjectural, as do the effects of ischaemia and compression on optic nerve axonal transport. The clinical courses of both our patients, however, discount the pathophysiological mechanisms of central retinal artery occlusion, central retinal vein occlusion, anterior ischaemic optic neuropathy, or acutely raised intraocular pressure as being the cause of visual loss seen with retrobulbar haemorrhage.

Whatever the actual pathophysiology may be, the appropriate therapeutic manoeuvre is immediate orbital decompression. Attempts to lower intraocular pressure are not appropriate, since intraocular circulations are not the limiting physiological factors. Medical therapy with the use of osmotic diuretics to decrease oedema helps to decompress the orbit, but prompt surgical intervention in the form of immediate canthotomy and/or cantholysis, in the clinic or in the emergency room, is the treatment of choice.

We believe these cases represent the longest documented interval (4 hours) of reversible total blindness due to orbital haematoma. They suggest that the optic nerve is more resilient when rendered ischaemic than the retina or choroid, and offer observations to support recent demonstrations that axons can resist ischaemia longer than nerve cell bodies.³² Both patients had no perception of light for at least 3½ hours, yet they recovered to 20/40 and 20/20 acuity respectively. Experimental studies suggest that the retina tolerates temporary ischaemia up to 100 minutes before potential for recovery of vision is lost.^{33–35} The optic nerve tolerance time to acute ischaemia has not been established experimentally, but if ischaemia were the injury in our cases, these patients' courses suggest it may be 240 minutes or longer.

Our cases illustrate that the optic nerve, and possibly its vascular supply, may become compromised and account for visual loss seen in the setting of orbital haemorrhage, and that decompression of a tense, tight orbit, when done in time, can successfully reverse a mechanical posterior optic neuropathy even in the setting of prolonged blindness.

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