Spontaneous orbital haemorrhage

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SUMMARY A 57-year-old housewife, with controlled hypertension, presented with acute right proptosis and visual loss with external ophthalmoplegia due to spontaneous orbital haemorrhage. Vision and ocular motility were returned to normal by the rapid surgical evacuation of the blood. Acute orbital haemorrhage is an ocular emergency requiring urgent treatment to prevent the usual previously reported outcome of permanent visual loss.

Spontaneous orbital haemorrhage is the uncommon condition of haemorrhage within the orbit not caused by local trauma and not referable, so far as can be ascertained, to any constitutional causative condition.¹ The visual prognosis is excellent except in the elderly, who often suffer permanent visual loss associated with a secondary vascular occlusion or an arterial bleed.²

This case is reported as a reminder that rapid diagnosis of the cause of the visual loss and urgent surgical drainage of the blood are required to prevent irreversible visual loss.

Case report

A 57-year-old housewife was awakened by severe right orbital pain associated with proptosis of the right eye and with ptosis, followed by nausea and vomiting. For the previous 18 months she had required Viskin (pindolol) 5 mg twice daily for control of hypertension, Brufen (ibuprofen) 400 mg three times daily for arthritis, and Serepax (oxazepam) 30 mg each morning for 'nerves.' She had previously undergone the uneventful repair of a hiatus hernia, hysterectomy, stripping of varicose veins, and biopsy of a benign breast lump.

Six hours after the onset of symptoms she was acutely distressed, with severe right orbital pain and vomiting. Her vital signs were normal, with a blood pressure of 110/70 mmHg. The corrected visual acuities were right 6/12, left 6/6. There was a complete right ptosis, ecchymosis of the eyelids, conjunctival chemosis, and irreducible right proptosis of 15 mm compared with the left eye by Hertel measurements.

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There was neither pulsation nor thrill nor bruit over the globe or orbit. The pupils were equal and reactive, and there was an almost complete external ophthalmoplegia of the right eye with residual abduction and elevation of only 5° (Fig. 1). Corneal sensation and all branches of the fifth cranial nerve were intact. Intraocular pressures by Goldmann applanation tonometry were right 48 mmHg, left 18 mmHg. Fundal examination was normal; there was neither papilloedema nor striae.

She was admitted to the Royal Victorian Eye and Ear Hospital, where she received Diamox (acetazolamide) 500 mg intravenously. Urgent skull x-ray and computerized tomography were performed.

Computerized tomography (Fig. 2) showed a large, dense (CT number 83), well rounded lesion in the right orbit extending from the lateral wall, displacing the globe forwards and downwards and flattening the posterior margin of the globe. The optic nerve was displaced medially. More posteriorly the lesion almost completely filled the whole of the posterior part of the orbit. The changes were consistent with acute haemorrhage into the right orbit.

Investigations which were normal included skull x-ray, full blood examination (haemoglobin 13.9 g/dl, white cell count $5.4 \times 10^9/l$, platelet count $213 \times 10^9/l$), bleeding and clotting times, prothrombin ratio, partial thromboplastin time, thrombin time, clot appearance and retraction, erythrocyte sedimentation rate (16 mm in one hour), urea, and electrolytes.

By 12 hours after the onset of symptoms she had deteriorated further. The orbit was so tense that it was very difficult to raise her ptotic right eyelid. The best corrected visual acuity was: right, counting fingers at one metre; left 6/6. There was right proptosis of 18 mm compared with the left eye, and the globe

Fig. 1 Ocular movements at time of presentation.

Fig. 2 Computerised tomogram. Blood clot indicated by arrow.

was deviated laterally 1 cm. The right pupil was slightly dilated and reactive. There was a complete external ophthalmoplegia. The eye felt very hard.

The patient underwent immediate surgical exploration of the orbit in the operating theatre. An incision was made superolaterally at the orbital margin and the periosteum incised laterally over a mass. 7 ml of old blood was drained, with a resultant marked reduction in the proptosis. A red rubber drain tube was inserted and the wound closed in layers.

Twelve hours later proptosis, pain, and vomiting recurred. The best corrected visual acuity in the right eye was 6/36. There was 7 mm proptosis of the right eye compared with the left, the right pupil was dilated with only a sluggish reaction to light, and the only residual movement was less than 5° abduction and elevation. The fundus appeared normal. Hyalase (hyaluronidase) 1500 units was irrigated down the drain tube in an unsuccessful attempt to lyse the clot.

The visual acuity continued to deteriorate, and 16 hours after the initial surgery re-exploration was carried out. Transconjunctival incision with disinsertion of the superior rectus failed to show the mass, and reopening of the old wound did not release any blood. A lateral orbitotomy was then performed. After the lateral wall of the orbit was removed the periosteum was incised and the orbit entered. A large

old blood clot was delivered from the inferotemporal quadrant of the orbit and was followed by copious pulsatile bleeding of bright blood. The haemorrhage was controlled by packing, and the wound was then closed in layers. A temporary tarsorrhaphy was performed, and because of the possibility of an aneurysm she was transferred to the neurosurgery unit at St Vincent's Hospital.

The postoperative course was uneventful. Cerebral angiography with subtraction failed to demonstrate an intracerebral or ophthalmic artery aneurysm or a haemangioma. By two months after surgery her best corrected visual acuity in each eye was 6/4, the ptosis had resolved, her pupils were equal and reactive, and she had regained a full range of ocular movements with only residual diplopia on extreme lateral gaze to the right.

Discussion

True spontaneous orbital haemorrhage is rare.¹⁻⁵ Trauma remains the most common cause of orbital haemorrhage² and should never be forgotten. Most of the reported cases of spontaneous orbital haemorrhage appear to have a cause to which the haemorrhage could be referred. In one series² venous anomalies were responsible for most of the orbital haemorrhages, with arterial haemorrhage occurring in a few elderly patients with advanced atherosclerosis. Lymphangiomas occur infrequently in the orbit^{6,7} and were not responsible for any of the haemorrhages in Krohel and Wright's series.² Blood disorders associated with leukaemia,⁸ haemophilia,^{9,10} uraemia,¹¹ scurvy,¹² and sickle cell disease and malaria¹³ are less common. Carotid cavernous fistula¹⁴ and aneurysm of the ophthalmic artery either at its origin¹⁵ or intraorbital^{16,17} and haemangioma^{18,19} have also been reported. Straining has also been associated with orbital haemorrhage; the orbital veins lack valves and the pressure is transmitted directly to them.²⁰ Hypertension has also been reported as a cause of spontaneous orbital haemorrhage.²¹

The symptoms of sudden pain, vomiting, and proptosis with limitation of motility occur in most patients with orbital haemorrhage. A marked initial decrease in visual acuity is more common in the elderly, in whom the visual loss often persists if there is a delay in evacuation of the blood. In Krohel and Wright's series² over half the elderly patients had severe and permanent visual loss. However, in children chronic, slowly developing blood cysts may arise from orbital haemorrhages and simulate a neoplasm. Lateral orbitotomy with bone removal was used in our case, but should be avoided in children, as

this can result in delayed bone growth and later facial deformity.²

Proper evacuation and drainage of the blood must be maintained for 24 hours or it may reaccumulate, as in our case, necessitating further surgery with the risk of further visual loss.

In our case, although an aneurysm was suspected, angiography with subtraction studies²² failed to demonstrate either an aneurysm or haemangioma. It is concluded that this was an arteriosclerotic spontaneous orbital haemorrhage with underlying hypertensive vascular disease. The favourable outcome was achieved by the necessarily rapid evacuation of the clot.

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