Purtscher's retinopathy after a rally-driving accident

Introduction

Loss of vision following motor accidents has several potential causes. This article presents a case of Purtscher's retinopathy (PR) and reviews clinical aspects and possible mechanisms.

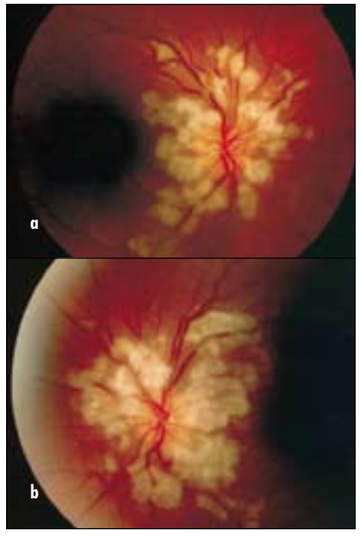
Discussion

Road traffic accidents can cause facial and orbital injuries and blunt or penetrating ocular trauma. Anecdotally before seat belt legislation, bilateral perforating eye injuries were not uncommon as people were thrown through a breaking windscreen. Blunt trauma can cause loss of vision through a variety of means. Anterior segment signs of injury include hyphaema (which usually clears but can cause glaucoma or corneal staining), lens subluxation or dislocation, and angle recession with the consequent long-term risk of glaucoma. Posterior segment findings can include vitreous haemorrhage (which can obscure signs of optic nerve avulsion), retinal breaks (such as a retinal tear, peripheral retinal dialysis or macular hole), and choroidal rupture which tends to occur in the macula. These injuries can each cause irreversible loss of vision and some, such as optic nerve avulsion and choroidal rupture, are not amenable to treatment.

This case is a dramatic example of PR. PR was first described by Purtscher at the 1910 Congress of Ophthalmology in Heidelberg (Marr and Marr, 1962). In PR the vision may be severely or mildly impaired, visual field defects vary and signs may be unilateral or bilateral (Buckley and James, 1996). However, in all cases cotton wool spots (microinfarctions of the retinal nerve fibre layer) occur in the posterior pole, along with retinal oedema and retinal haemorrhages. Serous detachment of the posterior retina and optic disc swelling can also occur.

The retinopathy can result from head trauma or chest trauma. The degree of chest trauma is not related to the risk of developing PR as it has been described following minor chest compression from a seat belt injury. Purtscher's-like retinopathy can also occur as a result of acute pancreatitis, fat embolization following long bone fracture, amniotic fluid embolization, renal failure, barotrauma, retrobulbar anaesthesia and systemic vasculitides such as systemic lupus erythematosus (SLE) (Marr and Marr, 1962; Lemagne et al, 1990; Stoumbos et al, 1991).

Figure 1. a. Right and (b) left fundi on presentation, 6 days after accident.



Case Report

A 31-year-old man was a driver in a rally when his car hit a concrete block at approximately 100 km/h. He lost consciousness for an unknown number of minutes. His helmet was split. He had no external signs of injury; his Glasgow Coma Scale was 15 and computed tomography of the brain was normal. His vision gradually deteriorated over the following 6 days. He had no history of ophthalmic problems, including ambylopia. His best corrected visual acuities were hand movements in each eye. There was no relative afferent pupillary defect but dilated fundal examination revealed multiple, almost confluent, cotton wool spots around both optic discs with some slivers of retinal haemorrhage (*Figure 1*). Purtscher's retinopathy was diagnosed and the patient was counselled regarding the uncertain visual prognosis. His vision improved; 18 days later his visual acuities were 6/9 unaided in each eye, and the cotton wool spots were reducing (*Figure 2*).

Although the use of steroids for PR has been investigated there is no useful treatment (Gibson et al, 2000). Identification and management of systemic injuries should be performed in all cases. The clinical course is variable, with final outcome ranging from complete recovery to optic atrophy (Gibson et al, 2000). Diffuse macular pigmentary disturbance may develop and paracentral scotomata often persist.

Different theories have been postulated as the mechanism. Purtscher attributed the signs to a sudden increase in intracranial pressure (ICP) transmitted down the optic nerve, causing extravasation of lymph from retinal vessels. Vogt (1923) proposed that increased ICP could compress the optic nerve. Marr and Marr (1962) suggested that central venous pressure is increased by chest compression, the gravitational force of deceleration and an anticipatory Valsalva just before the trauma.

Figure 2. a. Right and (b) left fundi 24 days after accident.

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As well as venous back pressure, arterial pressure is thought to increase as blood is ejected from the chest by compression. This may damage retinal endothelial cells making occlusion of small vessels more likely (Gibson et al, 2000). These theories do not explain the PR-like picture seen where there is no trauma. Arteriolar occlusion is suggested by fluorescein angiography. Air emboli have been implicated. However, fat emboli from long bone fracture, or amniotic fluid emboli, would not be able to pass through the pulmonary vasculature to the retinal circulation, except in rare cases of patent foramen ovale. Hypercoagulability leading to fibrin clot formation has been postulated (Behrens-Baumann et al, 1992). Disseminated intravascular coagulopathy, however, causes choriocapillaris occlusion and serous detachment of the retina; a different picture to that seen in PR.

There is increasing evidence for complement activation. This causes leucocyte aggregation and microemboli formation. Trauma, acute pancreatitis and connective tissue disease – all scenarios in which Purtscher's-like retinopathy has been described – induce complement activation. Injection of complement-induced leucocyte aggregates in the pig induced a PR-like picture experimentally (Scheurer et al, 1992). Amniotic fluid emboli activating complement may cause PR following childbirth (Blodi et al, 1990). Complement activation would cause retinal ischaemia, but leucocytes clumps may spontaneously disaggregate, allowing recovery of function (Buckley and James, 1996), as seen in this case.

Conclusions

Loss of vision following motor accidents has several potential causes, and PR should be considered even in the absence of significant systemic injuries. This case shows the potential recovery in vision that can occur despite the lack of treatment available. BJHM

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An oily argument

In 1 week in March, a pair of contradictory medical stories made the news. First we heard that oily fish was good for prostate cancer, slowing the progression of established disease. There was a smattering of simplistic feel-good coverage. The local nadir was 3 minutes of regional BBC television news spent wandering around a supermarket with a celebrity sufferer, who selected mackerel from the fish counter. At the end of the item, the anchorman grinned and said nothing useful at all before moving on.

The euphoria lasted until the *BMJ* appeared on the Friday. Now, a meta-analysis told us that oily fish had none of its previously supposed health-giving properties. This story – it was the *BMJ* after all – achieved blanket coverage.

In the same way that word processor programs have templates for common types of document, do newspapers have templates for stock replies to medical stories? It seemed that way from letters in the following day's *Independent*. What infuriates is how often these stock replies are factually wrong, or based on false logic that takes moments to disprove.

The first letter, from Tom Green, suggested that the credibility of the whole medical establishment had been damaged, because one certainty – that fish oils were good for us – had been replaced by another – that they are not. He failed to see that the certainty in these stories comes not from the medical establishment but in their reporting. One shouldn't shoot the messenger, but here the messenger is complicit in misrepresenting the original story and transferring blame for the misrepresentation. Mr Green wrote of the Northwick Park drug trial, in which six young men became critically ill, that it had been conducted with 'the same naively unshakeable faith' in science.

Tom Green fervently misunderstands how medicine and science work, and the newspaper should not have published the letter. It doesn't matter that many people think the same; it is an opinion with no value. This was illustrated by the next letter, from Peter Easton, cautioning that any single study is not the truth, but merely one more step towards it. This is something that not enough people realise, and this letter needed to be published. As Mr Easton wrote, the moderating comments made by the study authors had been 'insufficient to offset the impact of the headline'.

Then Christopher McDouall weighed in with how omega 3 cured his angina. He's been taking it ever since and has been symptom-free for 12 years. Not just that but a chiropractor cured his recurrent backache. Would a letter editors counter the evidence linking smoking and lung cancer with a letter from someone who had smoked 40 a day and lived to 90? The public need to learn the fallibility of personal experience in matters of science: anecdote is more likely to confound than confirm.

The final letter, from Jane Dawson, was short and sweet: omega 3 must be good for you if a study shows no benefit. If in doubt, do the opposite of what the doctors say. Written without Tom Green's vitriol, or Peter Easton's erudition, it highlighted perfectly that we don't know as much as we think we know. BJHM

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