Early fundus fluorescein angiographic findings and sequelae in traumatic retinopathy: case report

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SUMMARY A 44-year-old man involved in a minor road traffic accident developed traumatic retinopathy in both eyes. We present the unusual early angiographic findings and the late sequelae of persistent ring scotomata. The previous work on the retinal pathophysiology is reviewed, and we suggest that the total clinical picture in traumatic maculopathy may often be a combination of the 'angiopathic retinal trauma' of Purtscher and the 'commotio retinae' of Berlin, that is, a combination of haemodynamic forces leading to vascular damage in the nerve fibre layer and of direct mechanical forces leading to photoreceptor destruction.

A 44-year-old man was involved in a minor traffic accident. His car was travelling at about 10 miles (16 km) per hour and collided with another vehicle. The patient was wearing a seat belt. He felt that his head had undergone a minor whiplash action but it had caused him no discomfort. He did not strike his head nor did he feel that his chest had suffered any significant crushing force. He had no bruising of the

chest wall. Approximately 5 minutes after the accident he noticed his visual acuity suddenly deteriorate. One hour later his vision was 6/90 in the right eye and 6/120 in the left eye. Anterior segments, pupils, and intraocular pressures were normal. Fundal examination showed marked retinal oedema at the posterior pole with apparent macular cyst formation. There were no haemorrhages or cotton-wool spots. The Bjerrum visual field showed a dense central scotoma (Fig. 1).

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The left fundus fluorescein angiogram performed

Fig. 1 Visual fields 2 hours after injury, performed on Bjerrum screen at 1 m with 10 mm white target.

within 2 hours of the accident showed slight early masking of choroidal fluorescence between the disc and macula (Fig. 2A). Subsequently arteriolar leakage developed and became more diffuse in the late phase (Figs. 2B,C,D). No venous or capillary leakage appeared. The right fundus showed a similar though less marked appearance.

A week later the visual acuity had returned to 6/7.5 right, 6/6 left, but the patient complained of a ring field defect in both eyes. Absolute ring scotomata in both eyes were confirmed on Bjerrum screen field testing (Fig. 3). The scotomata encroached to 1° from fixation on the right and 2° on the left and extended out to around 7° from fixation on both sides. The

Fig. 2A

Fig. 2B

Fig. 2C

Fig. 2D

Figs. 2A,B,C,D Fundus fluorescein angiogram 2 hours after injury. Progressive leakage developing after the early venous phase.

Fig. 3 Visual fields 1 week after injury, performed on Bjerrum screen at 1 m with 5 mm white target.

repeat fluorescein angiogram was entirely normal, as was the appearance of both retinæ. Over the subsequent 6 months the area of central vision extended out slightly in both eyes (Fig. 4) but has remained constant since. A fine pigmentary macular change with retinal thinning has developed.

Discussion

In 1912 Purtscher described the 'angiopathic retinal trauma' which now bears his name.¹ The clinical manifestations are well described (2,3). Following the injury marked generalised retinal oedema with

Fig. 4 Visual fields 6 months after injury, performed on Bjerrum screen at 1 m with 5 mm white target.

macular involvement rapidly develops. Retinal cotton-wool spots, usually related to the major temporal vessels, and haemorrhages are found. Disc oedema is often present. Initially a dense central scotoma is nearly always detected, with visual acuity reduced to 6/60 or less. Recovery of visual acuity is variable. The oedema, cotton-wool spots, and haemorrhages resolve. A degree of optic atrophy usually results, and diffuse macular pigmentary disturbance may develop. Paracentral scotomata often persist, and occasionally a central scotoma remains.

Reports of fundus fluorescein angiographic studies in Purtscher's retinopathy are scarce but the findings are: (a) blocked choroidal fluorescence; (b) occluded retinal arterioles; (c) areas of capillary nonperfusion; (d) late perivenous staining; and (e) optic disc oedema.^{3,4} Only one case is reported in which the fundus fluorescein angiography was performed within hours of the injury.² In this case the choroidal filling pattern was normal, but leakage of the small arterioles in the mid-venous phase which spread in the late venous phase was noted. Leakage gradually developed from the capillary or near-capillary size vessels on both arterial and venous sides of the posterior pole circulation. The fluorescein angiographic abnormalities disappeared with time.

Purtscher's retinopathy has been reported following cases of severe head trauma;¹ post-traumatic fat embolism;^{5,6} compression cyanosis;⁷ minor chest compression from seat belt injury;^{2,8} acute pancreatitis with fat embolism;⁹ and in the ophthalmological hydrostatic pressure syndrome.¹⁰ The variety of aetiologies leading to the common clinical finding has led to a search for a common pathophysiological cause. It has been suggested that arteriolar occlusion due to embolisation with air or fat may be the underlying mechanism.³ Experimental work with fibrin clot,¹¹ air,¹² and glass microsphere^{13,14} embolisation of animal retinæ can produce the fundal appearances of Purtscher's retinopathy as can acute pancreatitis with fat embolism in the absence of physical trauma.

In the present case the injury would appear to be too minor to have been caused by either fat or air emboli. Indeed no evidence of occlusion of retinal arterioles was found on fundus fluorescein angiography, nor did the retinal fibre layer infarcts that would be expected to follow retinal vascular occlusion and appear as cotton-wool spots subsequently occur.

Two other alternative hypotheses based on haemodynamic considerations have been put forward to explain the pathogenesis of Purtscher's retinopathy. Firstly, it has been suggested that the increased intra-venous hydrostatic pressure caused by chest

compression or the gravitational force of deceleration leads to vessel wall trauma. This seems compatible with the marked venous changes seen in fluorescein angiography performed after several days have elapsed, but would be unlikely to cause the kind of arteriolar changes seen in our case or that reported by Kelley.² Secondly, it may be that in the traumatic cases the victim, anticipating the accident, performs an involuntary Valsalva manoeuvre. This leads to an increase in venous pressure in the head and hence in the retinal circulation. When the chest is suddenly compressed, blood is ejected from the chest into the arterial system, and the rise in arterial pressure is developed against the increased venous back pressure. This leads to arteriolar endothelial damage and swelling. This course of events could certainly explain the arteriolar leakage of fluorescein found in our patient.

In the present case the apparent injury was very slight. The fundus fluorescein angiogram, however, showed dramatic arteriolar leakage, though no cotton-wool spots developed and the angiographic changes had resolved entirely one week after the accident. A persistent ring scotoma has not previously been described, though central scotomata and small and large paracentral scotomata have been reported,³ and indeed in Hoare's case,⁸ in which trauma was also rather mild, the left eye showed a partial ring-shaped scotoma. The persistent scotomata present in our patient are unlikely to have resulted from the transient arteriolar damage, and indeed a macular ring scotoma is not compatible with nerve fibre layer infarction and suggests possible photoreceptor damage. Recently, Sipperley *et al.*¹⁵ have produced a model of commotio retinae in owl monkeys with blunt trauma to the eye. Their histological findings showed the damage to the retina to be centred on the photoreceptors, which, once disrupted, were then phagocytosed by the pigment epithelial cells, which subsequently migrated more superficially, leading to clinically observable pigmentary changes. Our patient has developed a fine pigmentary mottling at the macula, which is common following commotio retinae.^{15,16} It is therefore suggested that damage to the photoreceptor and pigment epithelial layers has accounted for the persistent visual field defect.

It is clear that in cases of retinopathy due to acute pancreatitis the angiopathy and sequelae cannot be attributed to trauma. However, in most cases of Purtscher's type retinopathy considerable trauma involving crushing, deceleration, and blows to the head is sustained by the patient. Theoretically even relatively minor blows to the head or deceleration forces should impart considerable internal stress and oscillatory movement to the vitreous, and hence

whiplash injury might be expected to cause very large shearing forces between the attached vitreous and the retina, or severe concussive injury where the vitreous is detached and free to strike against the retina.

Our case is unusual in that the immediate angiopathic effects were mild and transient though the sequelae were quite severe. This has allowed us to separate the 2 pathophysiological mechanisms involved. We postulate that haemodynamic forces may cause arteriolar and often simultaneous venous trauma, which may lead to superficial retinal damage. In addition often superimposed on this is a mechanical disruption of the photoreceptor layer which contributes to the pigmentary maculopathy, residual scotomata, and visual impairment.

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