TRAUMATIC RETINAL ANGIOPATHY AND SEAT BELTS: PATHOGENESIS OF WHIPLASH INJURY

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SUMMARY

Three cases of traumatic retinal angiopathy associated with whiplash injury are presented. The pathogenesis of the fundal appearances is discussed. Local microcirculatory disturbances are postulated as the cause of the retinopathy as opposed to the systemic disturbance associated with Purtscher's retinopathy. This condition may be underdiagnosed as there may be few abnormal signs on funduscopy. Fluorescein angiography may be very helpful. The incidence may be increasing as a result of legislation concerning the wearing of seat belts, and the condition has medico-legal significance. Increasing awareness may increase diagnosis.

The classification and nomenclature of the manifestations of injury to the retina following indirect trauma are confused and imprecise. We have had the opportunity of studying three cases of whiplash injury with ocular symptoms and signs and in the light of these reconsider the pathophysiology of this injury. Understanding the mechanism is important not only in patient management but in circumstances of medico-legal significance.

CASE REPORTS

Case 1

A 52-year-old man was involved in a road traffic accident while wearing a seat belt. He suffered a whiplash injury but no other injuries. In particular he did not sustain a head injury or any fractures. Shortly after the accident he noticed blurred vision and a paracentral scotoma in his right eye and presented himself to the Ophthalmology Department 2 weeks later.

At presentation the patient's visual acuities were 6/5 right and left. The paracentral scotoma was confirmed with an Amsler grid. Funduscopy revealed a para-macular dilated capillary network with a blot haemorrhage superotemporally. Fluorescein angiography confirmed the microcirculatory disturbance (Fig. 1).

The patient made a complete recovery and was discharged from follow-up 6 months later.

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Case 2

A 58-year-old man presented to the Casualty Department complaining of blurred vision after being involved in a road traffic accident. He was wearing a seat belt. He banged his head on the steering wheel, but remained fully conscious. He did not suffer any bony injury.

The patient's visual acuities were 6/12 right, 6/6 left. A central scotoma in the right eye was confirmed with an Amsler grid. Funduscopy revealed pigment dispersion at the macula, but no haemorrhages. Fluorescein angiography revealed an area of vessel leakage close to the fovea (Figs. 2).

Over the next 6 months the visual acuity improved to 6/5, although a paracentral scotoma persisted.

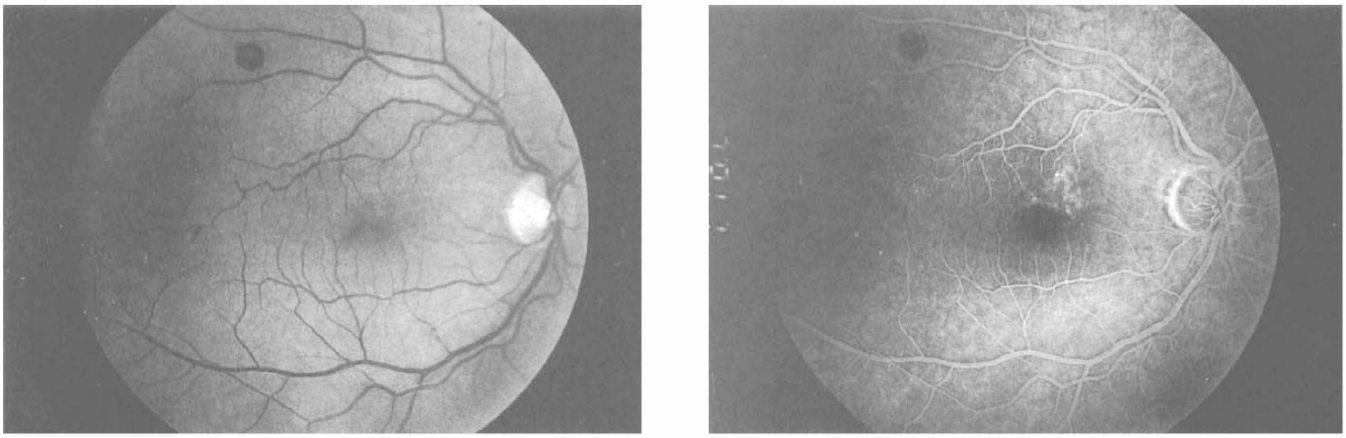
Case 3

A 42-year-old woman presented 1 year following a very severe whiplash injury. At the time she was seen by the ophthalmologist she was still wearing a soft collar. She suffered no bony injury at the time of the accident.

The patient complained of difficulty reading and her vision was recorded at 6/6, N8. She said her vision was much worse for a few weeks after the accident, but had gradually improved. Amsler chart testing showed a defect in the left eye and fluorescein angiography revealed perifoveal vascular disruption (Fig. 3).

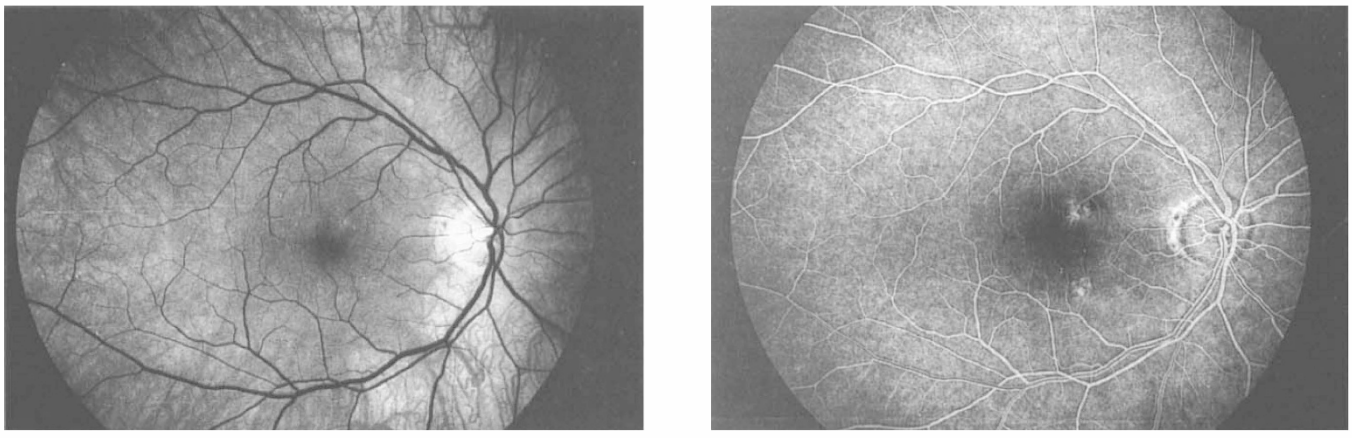
DISCUSSION

The effects of indirect trauma on the retina are variable, as is the terminology used to describe them. This is due to the different pathogenesis of the various lesions described. Purtscher in 1910 described a retinopathy consisting of cotton wool spots and haemorrhages developing within 1–2 days of extraocular trauma.[1] The retinopathy characteristically involves the macula and peripapillary area, spreading to the mid-periphery. The theoretical basis of the clinical findings has been attributed to the microembolisation of retinal arterioles.[2] While the clinical appearance and pathogenesis of Purtscher's retinopathy are well described[1,2] the causes of the clinical retinopathy are varied: fat embolism syndrome, pancreatitis.[3] On the



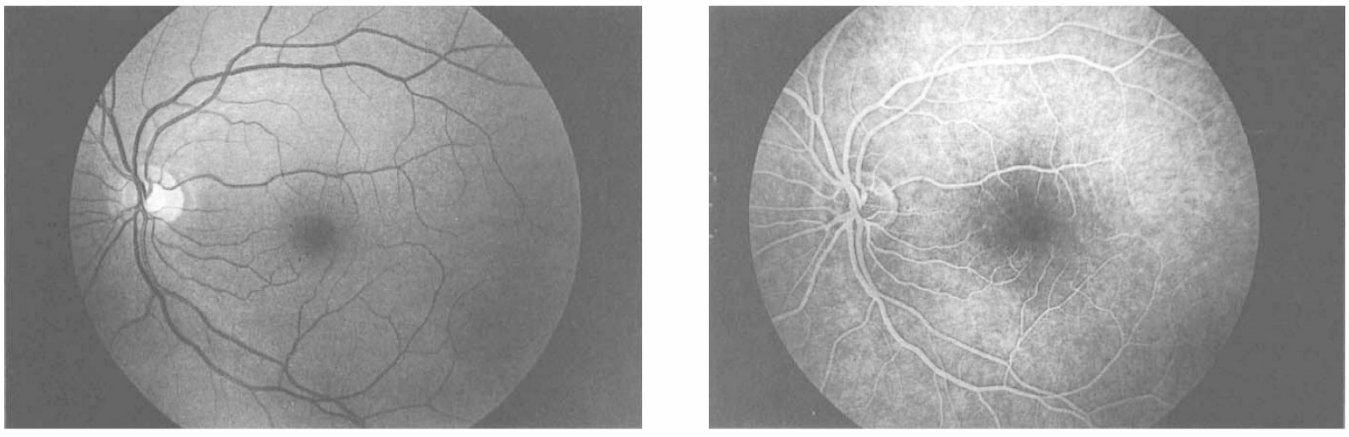
(a) *(b)*

Fig. 1. *Case 1. Red-free (a) and fluorescein (b) photographs taken 2 weeks after the accident.*



(a) *(b)*

Fig. 2. *Case 2. Red-free (a) and fluorescein (b) photographs taken 4 days after the accident.*



(a) *(b)*

Fig. 3. *Case 3. Red-free (a) and fluorescein (b) photographs taken 1 year after the accident.*

other hand, various different names have been used to describe a spectrum of clinical entities: traumatic retinal angiopathy, whiplash retinopathy, Valsalva retinopathy, shaken baby syndrome, Purtscher-like retinopathy, fat embolism syndrome.[4] The multiplicity of names causes some confusion and an understanding of the pathogenesis may clarify the situation. There are three main processes affecting the retina as a result of indirect trauma:

1. A retinopathy associated with *localised* microcirculatory insufficiency. Raised intraluminal pressure (secondary to seat belt compression, whiplash or Valsalva manoeuvre) may result in localised endothelial damage, complement and coagulation activation and microcirculatory insufficiency. This clinical entity has been described as traumatic retinal angiopathy.[5] We classify our three cases in this group.
2. A retinopathy associated with a *systemic* coagulopathy. This is the pathogenesis in fat embolism syndrome, pancreatitis and eclampsia. The underlying cause is a generalised activation of complement and coagulation pathways, leading to disseminated intravascular coagulation.
3. A maculopathy, previously described as whiplash maculopathy, occurring as a result of traumatic posterior vitreous detachment at the macula, leading to photoreceptor damage and foveal changes.[6] No microcirculatory defects are seen on fluorescein angiography in these cases.

In some patients, a combination of all three processes may be present.

In our cases it is not possible to say whether it is thoracic compression by the seatbelt or the whiplash which results in the retinopathy, as the two are concurrent.

CONCLUSION

Our cases were all unilateral. Similar cases have been reported.[7]

While our patients have made good recoveries in terms of visual acuity, their scotomata have persisted in two cases. This sequence of events agrees with previous reports.[5,8,9] Several months after the original injury the patient may be complaining of symptoms, while funduscopy is almost normal. A patient who presents to an ophthalmologist complaining of visual symptoms following a road traffic accident with a dearth of physical signs may be suspected of malingering, particularly if legal proceedings are pending. Some of these patients may have suffered traumatic retinal angiopathy. Fluorescein angiography may reveal persistent defects months or years after the original trauma and is therefore very useful in making the diagnosis. Awareness may increase diagnosis. Furthermore, this syndrome may be on the increase as a result of increasingly stringent seat belt laws.

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Key words: Pathogenesis, Purtscher's retinopathy, Retina, Retinopathy, Seatbelt, Whiplash.

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