PURTSCHER'S RETINOPATHY, A CASE REPORT

Dr. Saumendranath Ghose, Dr. Suvabrata Parida,
Dr. Indrani Rath, Prof. Madhumati Mishra
Dept. of Ophthalmology, SCB Medical College, Cuttack, Orissa

INTRODUCTION

Purtscher's retinopathy is a rare form of hemorrhagic & vaso-occlusive retinopathy which was first described as syndrome of sudden blindness with severe head trauma. Since its original description it has been associated with many traumatic as well as many non traumatic diseases. We report this case of Purtscher retinopathy which was associated with fracture of forearm & multiple superficial injuries as a result of road traffic accident.

CASE REPORT

A 21 year old male patient Mr. Prahas Kumar Mulia presented at our OPD 5 days after a motor vehicle accident with the complain of decrease of vision in his both the eyes; LE more than RE.

According to the patient when he was driving his motorcycle fell on the hydrant beside the road in order to avoid a collision from an incoming auto rickshaw. He was taken to the casualty where it was found that he suffered a fracture in his left rorearm, several back contusions & few bruises over the face. Few hours after leaving the casualty he noticed vision loss in his both the eyes.

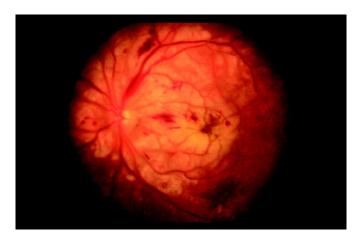


Figure -1:-Showing multiple confluent cotton wool spots at macula with multiple intra retinal haemorrhages

He did not have a relevant past ocular or medical history & his family & social history was noncontributory.

On ocular examination his VA was 6/12 in RE & counting finger at 1 foot the LE, both of which did not improve with pinhole. External & anterior segment was within normal limit but ther was a Grade 2 RAPD in the LE.

On examination of the fundus of the LE... there was multiple confluent white cotton wool spots in the macular region & few intra-retinal hemorrhages (Fig-I). In the RE the fundus picture wes less dramatic with few cotton wool spots at macula with out any hemorrhages (Fig-II). The patient was diagnosed clinically as a case of Purtscher's retionpathy & treated with high dose oral sterods for 1 week & then gradually tapered . At his first follow up after 4 weeks the patients vision improved to 6/6 in right eye and counting finger at 3 feet in left eye. Fundus examination revealed reduction of number of cotton wool spots in macula of both eyes, more marked in left.

DISCUSSION

Purtscher retinopathy is a hemorrhagic &

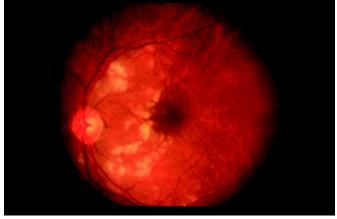


Figure -2: Showing few cotton wool spots at macula without any retinal haemorrhage

vasoocclusive vasculopathy first described by Dr. O. Purtscher on 1912 in the patients of severe head trauma¹. He proposed a mechanism of lymph stasis secondary to increased cerebral spinal fluid (CSF) pressures and named the condition **angiopathia retinae traumatica**. Later this clinical picture was found in several other conditions like comressive chest injury, long bone fractures, acute pancreatitis, fat or amniotic fluid embolism, connective tissue disorder & systemic vasculitis.

Patient with Purtscher retinopathy typically present with gross dimness of vision immediately or within 48 hours of injury. One eye or both the eyes may involve. Severity of trauma may not correlate with the severity or retinopathy.

The most common retinal finding is multiple large white cotton wool spots around the optic nerve associated with superficial peripapillary hemorrhages. Less common reported findings are mild serous detachment, preretinal hemorrhage, dilated vessels & optic disc edema. Confluent cotton wool spots at central macula some time may produce cherry red spots & can mimic CRAO. In FFA of early diseases there may be capillary leakage & staining of the retinal arteries. In severe diseases there may be capillary non-perfusion, venous dilatation & perivenous staining.

The exact pathophysiology of Purtscher retinopathy is not known. Three main hypotheses have been propsed. The classic mechanism describes during a chest or head trauma, an intravascular hydrostatic "shock wave" is generated which is then transmitted to retinal vasculature resulting in endothelial injury. However it fails to describe the non traumatic cases. A second mechanism of embolism is proposed in which emboli of fat, air or amniotic fluid is responsible for the clinical pictures. Lastly complement

mediated damage to the endothelium has been proposed, in which complement C5a act as the initiating factor leading to endothelial injury, leukocyte aggregation & embolization.²

There is no proven therapy for this condition although some claims mega dose corticosterids may improve the visual out come³. Controlling the underlying systemic diseases with medication is indicated & may be useful in visual recovery.

The prognosis is variable with about 50% of the patient having permanent central visual loss, thought the initial decreased vision may improve to certain extent over period of few months.⁴

CONCLUSION

Although Purtscher retinoipathy is a rare diseases but owing to the guarded visual prognosis & relevant medicolegal complications which may be associated with it, all patients of blunt trauma must undergo ophthalmoscopic examination.

REFERENCE

- Purtscher O. Noch unbekannte Befunde nach Schadeltrauma. Berl Dtsch Ophthal Ges. 1910;36:294-301
- Jacob HS, Goldstein IM, Shapiro I, Craddock PR, Hammerschmidt DE, Weissmann G, et al. Sudden blindness in acute pancreatitis. Possible role of complement-induced retinal leukoembolization. *Arch Intern. Med.* Jan. 1981;141 (1):134-6.
- 3. Atabay C, et al, Late visual recovery after intravenous methylprednisolone treatment of Purtscher's retinopathy. Ann Ophthalmol. 1993; 25 (9):330-333
- 4. Agrawal A, McKibbin MA. Purtscher's and Purtscher-like retinopathies: a review. *Surv Ophthalmol*. Mar-Apr. 2006; 51 (2): 129-36
