Purtscher’s Retinopathy an Eye on Head Injury

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Case Report

Abstract: We report a case of 45 year old man following a motor vehicle accident noted decreased vision on 5\textsuperscript{th} day following the trauma. Vision in right eye was hand movement close to face and left eye was counting finger at one meter. Multiple cotton wool spots, pre-retinal and retinal hemorrhages were present in both eyes with macular oedema on basis of which diagnosis of Purtscher’s retinopathy was made and patient was treated with iv methylprednisolone and at end of 6 weeks, vision in right eye was counting finger at one meter and in left eye 6/12. Prognostic factor for poor outcome in right eye due to corneal opacity and irregular corneal surface and hypermetric (childhood amblyopia; +5.5D)

Key words: Purtscher’s retinopathy, trauma, methylprednisolone

Introduction

Purtscher’s retinopathy (a.k.a. angiopathia retinae traumatica) was first described by Otman Purtscher in 1910 in a patient with severe head trauma\textsuperscript{(1)}. This retinopathy is characterized by bilateral retinal hemorrhage (pre-retinal or flame), multiple ischemic infracts (cotton wool spots or purtscherflecken), optic disc swelling. Reduced vision is the primary symptom of this condition which can be minimal or marked. Other causes include acute pancreatitis, fat embolism syndrome, renal failure, and connective tissue disorder. The exact pathogenesis is not known, intermediate size emboli from various sources appear to be capable of producing features of Purtscher’s retinopathy. Complement activation induced leukocyte aggregation is also found in cases with Purtscher’s retinopathy\textsuperscript{(2)}. There is no well-defined treatment for Purtscher’s retinopathy.

Case Report

A 45 year old male patient met with a road traffic accident due to which he had blunt trauma to chest, contused lacerated wound over scalp (tempero-occipital region on left side measuring 7cm x 3cm x 1cm), abrasion over shoulder and forearm and contused lacerated wound over both eyelid near medial canthus(measuring 1cm x 0.5cm). Ct-scan of head was done and showed no intracranial pathology, but x-ray shoulder showed incomplete fracture of acromion process of scapula. Patient was immediately taken up for debridement and suturing of contused lacerated wound over scalp; eyelid suturing was also done at same setting. Ophthalmology consultation was sought for diminution of vision in both eyes which was 5 days after trauma. Patient gives history of low vision in right eye since childhood. On ocular examination vision in right eye was hand movement close to face and left eye was counting finger at 1 meter. Both eyes showed mild periorbital oedema with ecchymosis and sutures were present over lower eyelid and wound was healthy. Subconjunctival haemorrhage was present in both eyes posterior border of which cannot be defined and there was old macular corneal opacity with irregularity of corneal surface in right eye. Rest anterior segment was within normal limits in both eyes with no RAPD.

On fundoscopy (direct and indirect with 20D and 78D lens) showed scattered areas of cotton wool spots(ischemic infracts) in both eyes with superficial retinal hemorrhage(flame shaped) predominantly in posterior pole region with both eyes macular oedema and left eyes showed one disc dioptre pre-retinal hemorrhage in superotemporal nasal region.
Right eye fundus photograph

Left eye fundus photograph

On basis of these findings diagnosis of Purtscher's retinopathy was made and treated with IV methylprednisolone 1 gm. for 3 days and then in tapering doses with napalact eye drops locally three times per day. 2 weeks after treatment with steroid and topical nevanac eye drops, there was significant reduction in macular oedema vision in right eye improved to counting finger 1 meter and left eye improved to 6/12.

Discussion

Purtscher's retinopathy is a rare condition which maybe unilateral or bilateral and leads to severe diminution of vision which make this an important entity. Though the exact pathophysiology of Purtscher's retinopathy is not known with certainty, there are four main proposed mechanisms:

1. The classic mechanism proposes that head injury or chest compression generates an intravascular hydrostatic "shock wave" that is transmitted to the retinal vasculature resulting in endothelial damage. This can affect retinal veins, macular capillaries, and radial peripapillary capillaries resulting in the clinical picture seen.
2. It is proposed that emboli of fat, air, or amniotic fluid can cause the clinical appearance of Purtscher's.
3. Complement mediated hypothesis. Complement C5a is known to be associated with trauma, acute pancreatitis, and systemic vasculitic diseases, and has been proposed to play a role in the development of Purtscher's. By this theory, component C5a initiates leukocyte aggregation and embolization. Leukoembolism in concert with other factors initiates intravascular coagulation of platelets.
4. A recent paper modeling shear stress in the retina suggests that Purtscher retinopathy may be a rheological event at a retinal posterior pole foci of vascular endothelial dysregulation, followed by downstream endothelin-induced vasculopathy.

Due to the peculiar anatomy of the blood supply of the peri-papillary retina and macular area, the retinal changes are usually confined to these areas and this was very evident in our case. The arterioles and capillaries in these areas are more susceptible to embolic occlusion as a result of fewer arteriolar feeders and fewer anastomoses. There is no well-defined treatment for Purtscher’s retinopathy. Papaverine HCL, a peripheral vasodilator, has been used in a case of Purtscher’s retinopathy based on the theoretical rationale of dilating retinal arteriole to increase oxygen supply. However, the therapeutic value of papaverine on ocular circulation is uncertain. It has been proposed that the use of megadose steroid may stabilize the damaged cellular membrane in neuronal tissue, and thereby enable a degree of recovery from tissue insult. Steroid treatment also can block the formation of complement-activated leukocyte aggregation and inhibit the production of oxygen-free radical.

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Conclusion

We believe that patient who is diagnosed to have Purtscher’s retinopathy should be treated with IV. Methylprednisolone considering the general condition of patient weighing potential benefits against risk involved in treating patient with high dose of steroid.

References