



TERSON SYNDROME WITHOUT CEREBRAL HAEMORRHAGE: A RARE PRESENTATION

Sunil Verma^{1*}, Rakesh Verma², Latika Pandey³, Reena Lamba⁴, Dixit Soni⁵, Aakash Sharma⁶

¹Sunil Verma, Assistant Professor, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.

²Rakesh Verma, Resident, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.

³Latika Pandey, Resident, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.

⁴Reena Lamba, Resident, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.


⁵Dikshit Soni, Resident, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.

⁶Aakash Sharma, Resident, Department of Ophthalmology, RIO (PGIMS) Rohtak, Haryana, India.

ABSTRACT

We are reporting here a rare case of 33 years old male who presented in ophthalmology department with sudden loss of vision after a traumatic brain injury (TBI) due to road side accident. Computed tomography scan of the patient, who had sustained an impact injury to the right occipital region, showed no cerebral lesion (hemorrhage). Ophthalmoscopy clearly demonstrated vitreous hemorrhage in both eyes. In this case, the visual disturbance was attributed to Terson syndrome secondary to TBI. Therefore, close ophthalmological and radiological evaluation is required in patients with TBI, in order to enable the diagnosis and management of Terson syndrome.

Key words: Cerebral hemorrhage, intracranial pressure, traumatic brain injury, Terson syndrome, vitreous hemorrhage.

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INTRODUCTION

Terson syndrome which occurs in 10–20% of patients is represented by a vitreous, retrohyaloid, retinal, or subretinal hemorrhage [1] may be caused by a severe cerebral hemorrhage induced by a head injury or by a subdural or epidural hematoma. It was first described by the French ophthalmologist Albert Terson in the beginning of the 1900's [2]. The pathogenesis of Terson's Syndrome has been controversial, but there are 2 main accepted mechanisms [3]. One of them states that elevated intracranial pressure has a crucial role, causing the rise of

intraocular venous pressure and the rupture of the superficial vessels, hence the hemorrhage. The other onestates that the accumulated blood form the subarachnoid space enters the eye along the optic nerve and retinal vessels space, producing a vitreous or retrohyaloid hemorrhage. However, to the best of our knowledge, there is no literature on Terson syndrome without cerebral hemorrhage following traumatic brain injury (TBI) [4]. This case is being reported here for its rare presentation.

Corresponding Author

Sunil Verma

Email: -verma.sunil4june@gmail.com

Case report

33 years old male patient referred to Ophthalmology clinic from emergency department with sudden loss of vision after a traumatic brain injury (TBI)

due to road side accident. Computed tomography showed no cerebral lesion. On ophthalmological examination, the patient had positive perception of light with accurate projection of rays in both eyes. Intraocular pressure was raised (30 mmhg) in both eyes with no other significant finding. Fundoscopic examination revealed diffuse vitreous hemorrhage in both eyes with retrohyaloid hemorrhage in the macular area and hematoma under the

internal limiting membrane as shown in figure 1.

Taking into account the fact that both eyes have been affected by vitreous hemorrhage, we decided to perform 23G Pars plana posterior vitrectomy after controlling intraocular pressure in both eyes and postoperatively outcomes was favourable in both eyes.

Fig 1. Fundus showing retrohyaloidhemorrhage in macular area in both eyes.



DISCUSSION

Terson syndrome is usually described in correlation with ruptured cerebral vessel aneurysms, mainly in three locations: in the internal carotid artery, the middle cerebral artery bifurcation, and in the upper part of the basal artery [5]. The sudden elevation of the intracranial pressure has a crucial role in Terson's syndrome. It causes the rise of intraocular venous pressure and the rupture of the superficial vessels, hence the hemorrhage. Also, the pressure is transmitted along the optic nerve sheath and retinal vessels space, occluding the retinal and choroidal anastomoses at the lamina cribrosa. Approximately 20% of the patients diagnosed with subarachnoid hemorrhage present with Terson's syndrome. This association has a negative influence on the mortality rate. Patients diagnosed with Terson syndrome have a 40-60% mortality rate, 3 to 9 times higher as compared to the patients who only present with subarachnoid hemorrhage unaccompanied by ocular manifestations [6]. Most often, the patient is neurologically impaired and the visual acuity cannot be tested, but the degree of vision loss is usually related to the extent of the intraocular hemorrhage. It can range from 20/20 to light perception as seen in our case. Also, the amount of intraocular hemorrhage is influenced by the speed of accumulation and magnitude of the intracranial pressure elevation [7]. Usually, the intraocular hemorrhage is bilateral and intraretinal or subretinal. Vitreous hemorrhage incidence is lower: 3-13% [8]. GlattșiMachemer have demonstrated that blood has a toxic effect over the retina's photoreceptors, especially in the

first 7 days after the hemorrhage [9]. The iron from the hemoglobin catalyses the conversion of hydrogen peroxide into hydroxyl radical which causes peroxidation of lipids, breaking of DNA chains and biomolecular degradation. Since the main function of the retinal pigment epithelium (RPE) is to phagocyte the photoreceptors external segments, which are rich in lipids, the retina, and RPE are prone to oxidative damage [10]. The visual prognosis of the patients who survive a subarachnoid hemorrhage is favorable. Most of the vitreous hemorrhages spontaneously clear up. Only 40% of the cases need a vitrectomy and only half of these also need a peeling of the internal limiting membrane. Vitrectomy is indicated in the cases showing persistent or bilateral vitreous or macular hemorrhages. Recent studies suggest that an early vitrectomy may help with a fast restoration of vision, thus reducing the incidence of complications that can occur, such as proliferative vitreoretinopathy and glaucoma. Kuhn et al. have described the accumulation of blood underneath the internal limiting membrane in Terson's syndrome and reported a 39% incidence of macular hemorrhages. In 1991, Lewis has introduced the tissue plasminogen activator (tPA), a protease that transforms plasminogen in plasmin which, subsequently, breaks the fibrin clot. It can be used as a subretinal injection during the vitrectomy or it can be injected intravitreally along with the pneumatic displacement of the clot. Taking into account the young age of our patient and the fact that the vitreous hemorrhage was persistent and bilateral, with macular involvement in the left eye, we decided that vitrectomy was necessary in

this case in order to prevent the occurrence of further complications and to improve the quality of life. Both eyes regained a good visual acuity (6/36P) after surgery, suggesting that the persistence of blood in the macular area influenced the functional prognosis.

CONCLUSION

A prompt diagnosis of Terson syndrome is mandatory in patients with TBI for intraocular hemorrhage as non-clearing severe vitreous hemorrhage may result in visual loss. An early vitrectomy can achieve favourable outcomes

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