

## Terson Syndrome with Traumatic Optic Atrophy

Sanjoy Chowdhury<sup>1\*</sup>, Sneha Kumari<sup>2</sup>, Priti<sup>2</sup> and Priyanka<sup>2</sup>

<sup>1</sup>Chief Ophthalmologist, Bokaro General Hospital, Bokaro Steel City, Jharkhand, India

<sup>2</sup>DNB Resident, Bokaro General Hospital, Bokaro Steel City, Jharkhand, India

**\*Corresponding author**

Dr. Sanjoy Chowdhury, Chief Ophthalmologist, Bokaro General Hospital, Bokaro Steel City, Jharkhand, India, Tel: 08986872847; E-mail: drsanjoy@hotmail.com, drsanjoybokaro@rediffmail.com

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**Case Summary**

A 38 year old male came to our hospital with history of RTA 4 months back, he sustained head injury and was treated outside for brachial plexopathy. He now presented with total loss of vision in left eye which was sudden in onset and non-progressive.

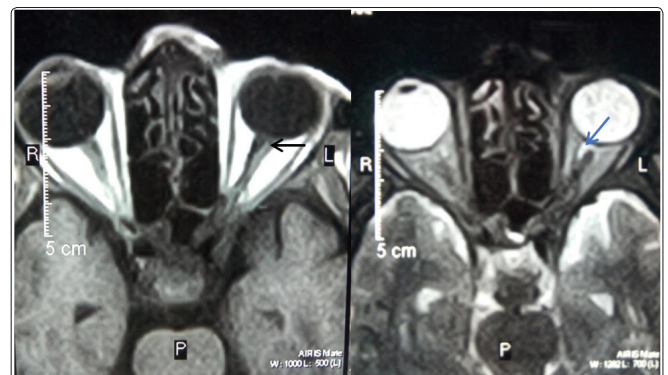
On examination his BCVA was 6/6 in right eye and PL negative in left eye. Anterior segment was within normal limits. RAPD was present in left eye. Fundus examination showed subhyloid hemorrhage involving macula with disc pallor. Extraocular movements were free and full in both eyes (Figure 1 and 2).

CT head showed contusion involving left frontal region with perilesional edema with Subarchnoid hemorrhage in basal cisterna (Figure 3).



**Figure 3:** CT SCAN of Brain showing subarachnoid haemorrhage and pneumocephalus in frontal lobe

MRI brain showed normal study except thickening of optic nerve in left side and hemorrhage around optic nerve (Figure 4).



**Figure 4:** T1 and T2 weighted images showing hematoma near optic nerve in left eye (Blue and black arrows)

On follow up fundus photography the hemorrhage resolved but the patient's visual acuity did not improve.



**Figure 1:** Initial presentation showing optic atrophy with perimacular gliosis



**Figure 2:** Treatment with periocular steroid resulting in disappearance of gliosis exposing retinal haemorrhage

## Speciality of the Case

1. Frontal lobe haemorrhage leading to haematoma near optic nerve
2. Retinal Haemorrhage with gliosis
3. Gliosis resolved with periocular steroid
4. No mortality even with SAH and Terson syndrome
5. Optic nerve atrophy due to pressure of optic nerve haematoma leading to high ocular morbidity

## Discussion

First described by Litten in 1881 and then in 1900 by French ophthalmologist Albert Terson [1,2]. Terson syndrome is now recognized as intraocular haemorrhage associated with SAH, intracerebral haemorrhage, or traumatic brain injury [1]. Haemorrhage may be present in the vitreous, sub-hyaloid, or intraretinal/sub-internal limiting membrane.

There are several possible pathophysiologic mechanisms for Terson syndrome. Subarachnoid blood may be directly transmitted forward through the optic nerve sheath [1,3]. More commonly, a sudden increase in intracranial pressure leads to rapid effusion of CSF into the optic nerve sheath which causes dilatation of the retro bulbar optic nerve mechanically compressing central retinal vein and ensuing venous hypertension results in rupture of thin retinal vessels. This mechanism is consistent with the fact that Terson syndrome can be seen in patients without intracranial haemorrhage [4].

Fluorescein angiography has demonstrated a leakage site at the disc margin in a patient with Terson syndrome with vitreous haemorrhage. This suggests potential damage to the peripapillary retina induced by increased intracranial pressure transmitted through the optic nerve sheath [4]. Terson syndrome can present with dome-shaped haemorrhages in the macula [5]. A macular “double ring” sign may be seen with the inner ring caused sub-ILM haemorrhage and the outer ring caused by sub-hyaloid haemorrhage [6].

Although intraocular hemorrhages most frequently develop in the first hour after SAH, Terson syndrome can have a delayed onset, with reports of intraocular haemorrhage occurring up to 47 days after SAH [1,7,8]. Low Glasgow coma scale, high Hunt and Hesse grade and high Fisher grade are associated with a higher incidence of Terson syndrome [1].

Neurological outcomes and mortality rate are worse in patients with SAH and Terson syndrome than patients with SAH alone [1]. In a study by Pfausler, mortality was 90% in patients with SAH and Terson syndrome and 10% in those with SAH without Terson syndrome [9]. Swallow investigated the use of orbital CT to identify intraocular haemorrhage in patients with Terson syndrome. Retinal crescentic hyperdensities and retinal nodularity were seen in CT in two-thirds of patients with Terson syndrome [4]. Thus CT may be useful to identify possible Terson syndrome prior to an eye exam.

Multiple complications have been reported after Terson syndrome. Epiretinal membrane is the most common sequel of Terson syndrome, with an incidence of 15-78% [10-13]. Vitreous blood may cause ERMs by inducing glial proliferation and disruption of the ILM [14]. Retinal folds/perimacular folds occur in 20% of patients with Terson syndrome, retinal detachment occurs in 9%, and ghost cell glaucoma occurs in around 4% [14].

Proliferative vitreoretinopathy and preretinal fibrosis have also been reported after Terson syndrome. Studies have shown no difference in final visual acuity between patients who were conservatively managed and those who underwent PPV. However, visual recovery was more rapid in the vitrectomy group despite these patients having denser vitreous haemorrhage [12,15-19].

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