ISSN: 0975-833X

Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 11, Issue, 07, pp.5692-5694, July, 2019

DOI: https://doi.org/10.24941/ijcr.33757.07.2019

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

CASE REPORT

TRAUMATIC "TERSON SYNDROME PLUS": PNEUMOCEPHALOCELE WITH OPTIC ATROPHY

***Dr. Sanjoy Chowdhury, Dr. Madhumita Srivastava and Dr. Nilanjan Chowdhury**

Bokaro General Hospital, Bokaro steel city, Jharkhand, India

ARTICLE INFO

Article History:
Received 18th April, 2019
Received in revised form
14th May, 2019
Accepted 19th June, 2019
Published online 31st July, 2019

Key Words:

Terson syndrome plus,
Subarachnoid hemorrhage,
Subhyaloid,
Sub retinal,
Optic atrophy.

****Corresponding author:***
Dr. Sanjoy Chowdhury

ABSTRACT

Terson Syndrome is subarachnoid hemorrhage (SAH) with sub retinal hemorrhage flowing through channel. Reduced vision in such fresh case is due to hemorrhage itself, blocking macula / other photo receptors in the long run macular cellophane retinopathy which causes profound visual loss. SAH causes neurological problems which can become a risk factor for evacuating blood from vitreous. Hypertension is commonest cause to cause Terson Syndrome, but trauma is also devastating cause as it can lead to irreversible visual consequences like total loss of perception of light or blindness. Here we describe a case of Terson Syndrome plus disease features SAH in frontal lobe. When there is traumatic pneumocephalocele, it gives space to blood to imbibe towards bony optic canal and form hematoma around nerve sheath which causes compression around the same and leads to optic atrophy. Optic nerve can be injured by direct traumatic dissection during RTA, but even without that blood may accumulate around optic nerve and in turn leads to formation of hematoma and subsequently pressure induced optic atrophy. Moreover blood can slowly travel to subhyaloid space / sub retinal space with gliosis covering typical boat shaped blood. Gliosis may resolve through three injections of Tricort in the orbital floor near apex, but optic atrophy snatches vision. Thus diagnosis of Terson syndrome plus disease was established by addressing all features on CT scan and MRI. Plus features include pneumocephalous, optic nerve sheath hematoma, optic atrophy and gliosis over subhyaloid hemorrhage, typical boat shaped.

Citation: Dr. Sanjoy Chowdhury, Dr. Madhumita Srivastava and Dr. Nilanjan Chowdhury, **2019.** "Traumatic "Terson syndrome plus": Pneumocephalocele with optic atrophy", *International Journal of Current Research*, 11, (07), 5692-5694.

INTRODUCTION

When vitreous hemorrhage occurs in association with subarachnoid hemorrhage is known as Terson syndrome. Intraocular hemorrhages of any type, call it retinal, subhyaloid, or vitreous all of these have been documented in 10–40% of individuals with subarachnoid hemorrhage. It can also occur in association with intracranial hemorrhage and elevated intracranial pressure. Diagnosis of Terson syndrome is clinically important, as it is associated with significantly higher mortality. Up to 77% of these cases are overlooked on daily reports. Timely ophthalmologic intervention to prevent long term visual loss is of utmost importance.

CASE REPORT

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 subhyaloid haemorrhage involving macula with disc pallor. Extra ocular movements were free and full in both eyes. CT head showed contusion involving left frontal region with perilesional edema with subarachnoid haemorrhage in basal cisterna (Illustration 3). MRI brain showed normal study except thickening of optic nerve in left side and haemorrhage around optic nerve (Illustration 4). On follow up fundus photography the haemorrhage resolved but the patient's visual acuity did not improve.

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 (Czorlich *et al.,* 2014; Skevas *et al.,* 2014). Terson syndrome is now recognized as intraocular haemorrhage associated with subarachnoid haemorrhage (SAH), intra-cerebral haemorrhage, or traumatic

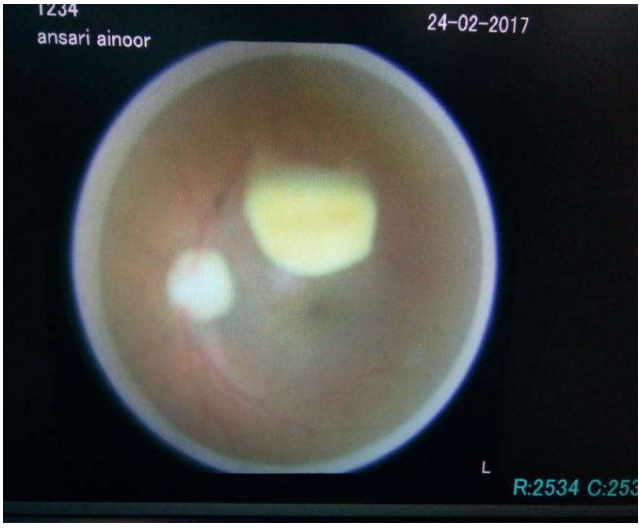


Illustration 1. Initial presentation showing optic atrophy with perimacular gliosis

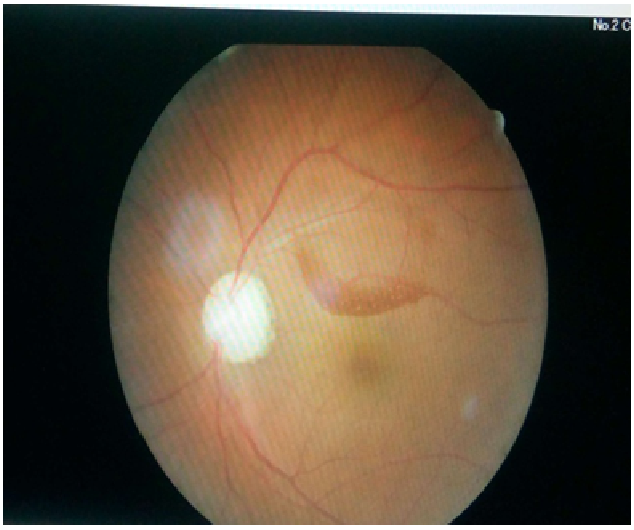
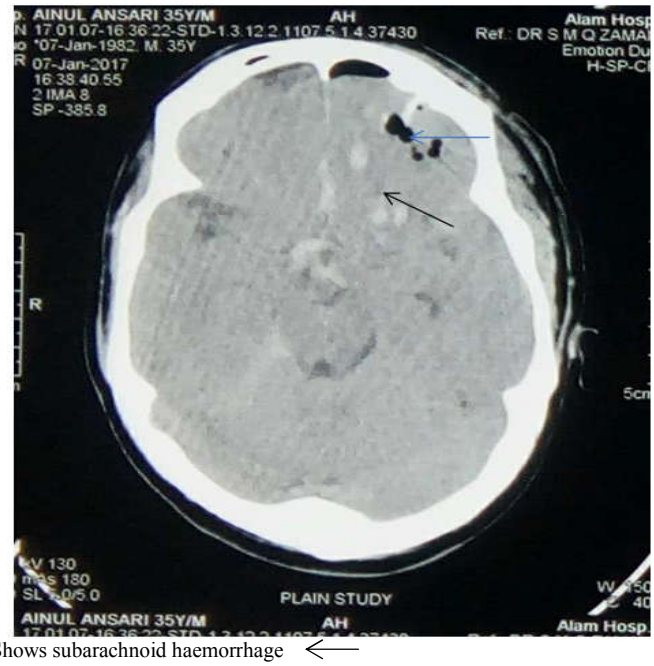
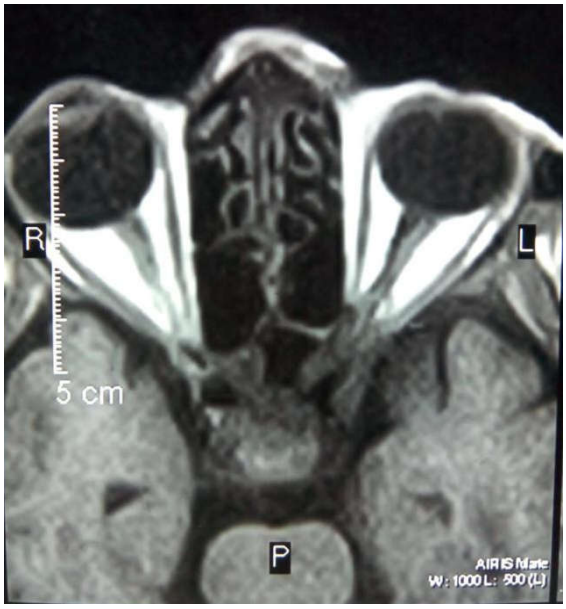


Illustration 2. Treatment with peri ocular steroid resulting in disappearance of gliosis exposing retinal haemorrhage



Shows subarachnoid haemorrhage ←
Shows pneumocephalous in frontal lobe ←

Illustration 3. CT SCAN of Brain showing subarachnoid haemorrhage and pneumocephalous in frontal lobe



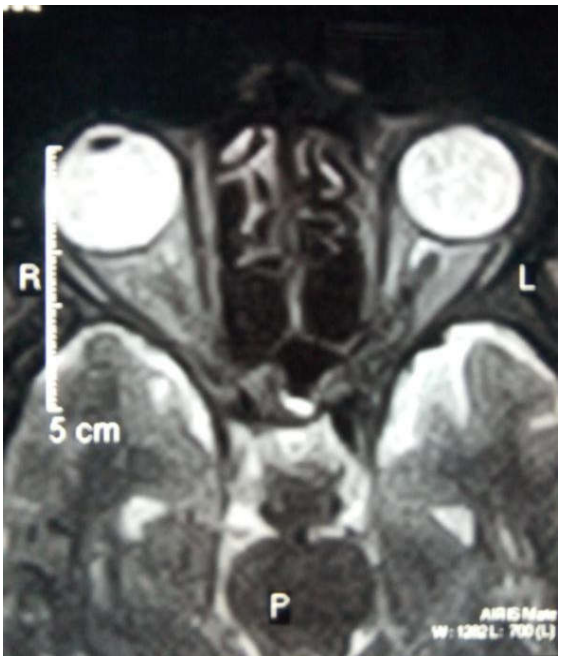


Illustration 4. T1 and T2 weighted images showing hematoma near optic nerve in left eye. (Blue and black arrows)

brain injury (Czorlich *et al.,* 2014). Haemorrhage may be present in the vitreous, subhyaloid, or intraretinal /sub-internal limiting membrane. Several possible pathophysiologic mechanisms for Terson syndrome are known. Blood present in subarachnoid may be directly transmitted forward through the optic nerve sheath (Czorlich *et al.,* 2014; Iuliano *et al.,* 2014). 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 (Gress *et al.,* 2013). Fluorescein angiography has demonstrated a leakage site at the disc margin in a patient with Terson syndrome with vitreous haemorrhage. This theorizes potential damage to the peripapillary retina induced by increased intracranial pressure transmitted through the optic nerve sheath (Gress *et al.,* 2013). Terson syndrome can present with dome-shaped haemorrhages in the macula (Friedman *et*

al., 1997). 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 (Srinivasan and Kyle, 2006). Even if intraocular haemorrhages most frequently develop in the first hour after SAH (Manschot, 1954), Terson syndrome can have a delayed onset, with reports of intraocular haemorrhage occurring up to 47 days after SAH (Czorlich *et al.,* 2014; Vanderlinden and Chisholm, 1974). Low Glasgow coma scale, high Hunt and Hesse grade, and high Fisher grade are associated with a higher incidence of Terson syndrome (Czorlich *et al.,* 2014). 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 (Fountas *et al.,* 2008; Stienen *et al.,* 2012; Pfausler *et al.,* 1996). Swallow investigated the use of orbital CT to indentify intraocular haemorrhage in patients with Terson syndrome. There was presence of retinal crescentic hyper densities and retinal nodularity in CT in two-thirds of patients with Terson syndrome (Shaw and Landers, 1975; Kim *et al.,* 2012; Swallow *et al.,* 1998). 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% (Sharma *et al.,* 2012; Rubowitz and Desai, 2006; Schultz *et al.,* 1991; Yokoi *et al.,* 1997). Vitreous blood may cause ERMs by inducing glial proliferation and disruption of the ILM (Takkar *et al.,* 2013). In 20% of patients with Terson syndrome retinal folds/perimacular folds occur. Also occurrence of retinal detachment in 9% and ghost cell glaucoma in around 4% cases has been seen reported (Takkar *et al.,* 2013). Proliferative vitreoretinopathy and preretinal fibrosis have also been reported after Terson syndrome. Studies have shown no significant 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 (Schultz *et al.,* 1991).

Conclusion

Traumatic Terson Syndrome is one of a very old known entity. Several possible pathophysiologic mechanisms for Terson syndrome are well known, but it is always good to assess any disease from different perspective, as minute finding can be vital for new outcome. The case of Terson Syndrome plus discussed here describe SAH in frontal lobe. Diagnosis of "Terson syndrome plus" disease was established by addressing all features on CT scan and MRI.

REFERENCES

Czorlich P, Skevas C, Knospe V, *et al.* 2014. Terson syndrome in subarachnoid haemorrhage, intracerebral haemorrhage, and traumatic brain injury. *Neurosurg Rev. Epub.,* 2014 Aug 31.

Fountas KN, Kapsalaki EZ, and Lee GP, *et al.* 2008. Terson hemorrhage in patients suffering aneurysmal subarachnoid hemorrhage: predisposing factors and prognostic significance. *J Neurosurg.,* 109(3):439-44.

Friedman S.M. and Margo C.E. 1997. Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. *Am J Ophthalmol.,* 124: pp. 850-851.

Gress DR, Wintermark M, Gean AD. 2013. A case of Terson syndrome and its mechanism of bleeding. *J Neuroradiol.,* 40(4):312-4.

Iuliano L, Fogliato G, Codenotti M. 2014. Intrasurgical imaging of subinternal limiting membrane blood diffusion in terson syndrome. *Case Rep Ophthalmol Med.,* 689-793.

Kim HS, Lee SW, Sung SK, Seo EK. 2012. Terson syndrome caused by intraventricular hemorrhage associated with moyamoya disease. *J Korean Neurosurg Soc.,* 51(6):367-9.

Manschot WA. 1954. Subarachnoid hemorrhage. Intraocular symptoms and their pathogenesis. *Am J Ophthalmol.,* 38:501-505.

Pfausler B, Belcl R, Metzler R, Mohsenipour I, Schmutzhard E. 1996. Terson's syndrome in spontaneous subarachnoid hemorrhage: a prospective study in 60 consecutive patients. *J Neurosurg.,* 85(3):392-4.

Rubowitz A, Desai U. 2006. Nontraumatic macular holes associated with Terson syndrome. *Retina.,* 26(2):230-2.

Schultz PN, Sobol WM, Weingeist TA. 1991. Long-term visual outcome in Terson syndrome. *Ophthalmology*, 98(12):1814-9.

Sharma T, Gopal L, Biswas J, Shanmugam MP, Bhende PS, Agrawal R, Shetty NS, Sanduja N. 2002. Results of vitrectomy in Terson syndrome. *Ophthalmic Surg Lasers.,* 33(3):195-9.

Shaw HE Jr and Landers MB III. 1975. Vitreous hemorrhage after intracranial hemorrhage. *Am J Ophthalmol.,* 80: 207–213.

Skevas C, Czorlich P, Knospe V, *et al.* 2014. Terson's syndrome--rate and surgical approach in patients with subarachnoid hemorrhage: a prospective interdisciplinary study. *Ophthalmology*, 121(8):1628-33.

Srinivasan S, Kyle G. 2006. Subinternal limiting membrane and subhyaloid haemorrhage in Terson syndrome: the macular 'double ring' sign. Eye (Lond)., 20(9):1099-101.

Stienen MN, Lücke S, Gautschi OP, Harders A. 2012. Terson haemorrhage in patients suffering aneurysmal subarachnoid haemorrhage: a prospective analysis of 60 consecutive patients. *Clin Neurol Neurosurg.,* 114(6):535-8.

Swallow CE, Tsuruda JS, Digre KB, Glaser MJ, Davidson HC, Harnsberger HR. 1998. Terson syndrome: CT evaluation in 12 patients. *AJNR Am J Neuroradiol.,* 19(4):743-7.

Takkar A, Kesav P, Lal V, Gupta A. 2013. Teaching NeuroImages: Terson syndrome in cortical venous sinus thrombosis. *Neurology*, 6;81(6):e40-1.

Vanderlinden RG. and Chisholm LD. 1974. Vitreous hemorrhages and sudden increased intracranial pressure. *J Neurosurg.,* 41: 167–176.

Yokoi M, Kase M, Hyodo T, Horimoto M, Kitagawa F, Nagata R. 1997. Epiretinal membrane formation in Terson syndrome. *Jpn J Ophthalmol.,* 41(3):168-73.

****