Case Report : Intraocular hemorrhage after blunt head trauma
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Conclusion
This case reviews a bilateral intraocular hemorrhage, causing visual field defects and diplopia in a patient with a history of blunt head trauma. The patient had a history of hypertension and diabetes mellitus. The hemorrhage was confirmed by ultrasonography and computed tomography. The patient was treated with bevacizumab and underwent pars plana vitrectomy with membrane peeling and endolaser. The patient's visual acuity improved from 20/200 to 20/25 in the right eye and from 20/30 to 20/25 in the left eye.

Reviewed and approved by
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Intraocular hemorrhage after blunt head trauma

Abstract

Objectives
To report about a patient with intraocular hemorrhage after blunt head trauma

Case Report
A 30 years old woman came to Cicendo Eye Hospital (CEH) with the chief complaint decreased vision in both eyes since 1.5 months ago. Two months ago the patient experienced a traffic accident while riding a motorcycle with head trauma, and underwent craniotomy surgery. The posterior segment of both eyes revealed hazy media. The patient was diagnosed vitreous opacities on both eyes. Ultrasonography examination suggested by the results of Vitreous Opacities on both eyes caused by vitreous hemorrhage DD vitreous fibrosis, complete posterior vitreous detachment on both eyes and subretina hemorrhage on the left eye. The patient was suggested to do Pars Plana Vitrectomy and endolaser for the right eye and then for the left eye in general anestesia.

Conclusion
This case resume is a bilateral vitreous hemorrhage caused by blunt head trauma assume as Terson's syndrome

I. Introduction

Intraocular hemorrhages usually originate from either the retina or the choroid. Hemorrhages occur in vitreous, subhyaloid, sub-internal limiting membrane (ILM), intraretinal, and subretinal space. Normal retinal vessels can be damaged by an acute process (e.g, ocular trauma, retinal detachment), or by chronic disorders (e.g, retinal vascular diseases, hematologic and inflammatory diseases).

Nonmechanical globe injuries may be caused by mechanical impact elsewhere in the body. Traumatic event not directly related to the eye rarely lead to ocular lesions, if they do, there are via a sudden increase of the intravascular pressure.

In 1990 Terson first described vitreous hemorrhage as a result of a sudden increase of the intracranial pressure (ICP). Usually the increase of ICP is due to spontaneous subarachnoid hemorrhage (SAH), secondary to aneurismal rupture.
Terson's syndrome has however also been described in traumatic intracranial hemorrhages. Retinal and preretinal hemorrhages occur in 20-40% of adult patients and in almost 70% of children with SAH.\(^3\)

II. Case Report

A 30 years old woman came to Cicendo Eye Hospital (CEH) on November 2\(^{nd}\) 2011 with the chief complaint decreased vision in both eyes since 1.5 months ago. Two months ago the patient experienced a traffic accident while riding a motorcycle with head trauma, after the incident the patient was unconscious and taken to General Hospital Cianjur then transferred to the General Hospital Sukabumi and examination a computed tomography scan revealed intracranial hemorrhage underwent craniotomy surgery. Two weeks after the operation the patient became conscious and began to feel decreased vision on both eyes. Patient went to the local Ophthalmologist and was given an eye drop two times daily and oral medication one time daily for one month. The patient was advised to control one month later and was consulted to the Cicendo Eye Hospital. Before the accident the patient had normal vision without glasses, no history of floaters and photopsia, no history of headache and pain on eye movement, no history of hypertension and diabetes mellitus.

General examination was within normal limits. Ophthalmological examination revealed the uncorrected visual acuity (UCVA) on right eye was finger counting at two meters and left eye was hand movement. Ocular motility was full to all direction. Intraocular pressure was 17.3 mmHg in both eyes using a tonometer schiotz. Anterior segment examination on both eyes were within normal limits. The posterior segment of the right eye (RE) revealed hazy media, round optic disc and the border of optic disc was difficult to measure, cup disc ratio was difficult to measure, artery vein ratio was difficult to measure, retina seemed flat. The posterior segment of the left eye (LE) revealed hazy media detail difficult to assess.
The patient was diagnosed by vitreous opacities caused by vitreous hemorrhage on both eyes related to blunt head trauma.

![Fundus drawing on both eyes](image1)

**Fig 2.1** Fundus drawing on both eyes

The patient was suggested to the Ultrasonography examination and the results were Vitreous Opacities on both eyes caused by vitreous hemorrhage DD vitreous fibrosis, complete posterior vitreous detachment on both eyes and sub retinal hemorrhage on left eye.

![Ultrasonography on both eyes](image2)

**Fig 2.2** Ultrasonography on both eyes
The patient was suggested to do Pars Plana Vitrectomy and endolaser for the right eye and then for the left eye in general anesthesia.

Fig 2.3 Computed tomography scan

III. Discussion

Vitreous hemorrhage is a significant cause of sudden vision loss. Vitreous hemorrhage can be due to a wide variety of causes including traumatic, proliferative diabetic retinopathy, posterior vitreous detachment (PVD) with or without retinal tear or retinal detachment, and other retinal vascular, hereditary, and inflammatory diseases.4

At the turn of the century, the French ophthalmologist Albert Terson described the finding of vitreous hemorrhage associated with an acute subarachnoid hemorrhage. The term Terson’s syndrome is now used to refer to any intraocular hemorrhage present after either spontaneous or trauma induced intracranial bleeding.5
Terson syndrome now encompasses any intraocular hemorrhage associated with intracranial hemorrhage and elevated intracranial pressures. Intraocular hemorrhage includes the development of subretinal, retinal, preretinal, subhyaloidal, or vitreal blood.6

Some degree of intraocular hemorrhage is seen in approximately 20 to 40 % of patients with various types of acute intracranial bleeds. A smaller percentage of patients, at around 3 to 5%, present with frank vitreous hemorrhage.5 The intracranial hemorrhage is usually subarachnoid in location, and the most common reported cause is the spontaneous rupture of in anterior communicating artery aneurysm and internal carotid artery.5,7

At the presentation, the most common ocular manifestation of Terson’s syndrome is multiple retinal hemorrhage. The hemorrhage are almost always bilateral and tend to be concentrated in the posterior pole. The amount of intraocular hemorrhage is often but not consistently proportional to be amount of intracranial hemorrhage. Patients have varying degrees of decreased visual acuity, typically related to the extent of ocular hemorrhage. Sometimes, however, hemorrhage that is sparse involves the fovea, and this causes a significant loss of acuity. Although the hemorrhages are occasionally subretinal, they are usually more superficial, being either just under the internal limiting membrane (ILM) or preretinal (subhyaloid) in location. Significant vitreous hemorrhage also occurs and is thought to result from blood breaking through the ILM or posterior hyaloids face into the vitreous gel.5,1 Intraretinal or subretinal hemorrhages have been reported but are less frequent. Preretinal hemorrhage can develop into vitreous hemorrhage weeks after the initial inciting event.6 This breakthrough bleeding can occur at any point in the clinical course and may be a cause for further worsening of the visual acuity during follow up.5
It was reported that the patient underwent blunt head trauma indicated by intracranial bleeding, noticed at the result CT Scan. At the examination posterior segment of both eye revealed hazy media and the result of the USG showed vitreous hemorrhage DD vitreous fibrosis, complete posterior vitreous detachment on both eyes and sub retinal hemorrhage on left eye.

The exact pathogenesis of Terson’s syndrome is unknown. One report suggested that it was caused by the dissection of blood from the subarachnoid space through the optic nerve sheath and into the eye. However, there is no direct communication between the subarachnoid space and the vitreous cavity in the normal eye, and hemorrhage that is not contiguous with the optic disc can be seen in this condition. A more plausible explanation suggests that an acute rise in intracranial pressure, such as occurs with intracranial hemorrhage, is transmitted down the intravaginal space of the optic nerve, causing venous stasis via compression and stretching of the intraorbital veins. This in turn, causes a rapid rise in intraocular venous pressure, distention, and rupture of the papillary and retinal capillaries and subsequent vitreous hemorrhage.8

In most causes of Terson’s syndrome, the blood gradually clears and vision returns to normal. Therefore observation is usually all that is needed. Vision however, may remain decreased from persistent vitreous hemorrhage or epiretinal membrane formation with surface wrinkling. Vitrectomy surgery is indicated in such cases to help restore or improve vision. Early intervention should be considered in any case with significant bilateral involvement or in young children with either one or both eyes visually affected the prevent ambyopia. Surgery without delay with also necessary should be an associated retinal detachment. Even in cases without there significant complications, visual acuity loss may sometimes persist to some degree because of permanent pigmentary disruption or outer retinal damage from subretinal blood in the macula.5
Kuhn and associates used 3 months in adults as a reasonable period of observation to determine whether spontaneous vitreous clearing can be expected. In counseling patients regarding observation versus surgery, several factors need to be considered such as bilaterality, extent of vitreous opacity, and signs of spontaneous resorption, as well as the patient’s age, occupation, visual needs, psychologic and neurologic status. In patients opting for observation, repeated visual testing and ultrasonography are recommended for early detection of retinal detachment. All patients with intracranial hemorrhage should undergo a dilated funduscopic examination, since the presence of vitreous hemorrhage has implications for the patient’s general condition.

The patient complained decreasing vision caused by vitreous hemorrhage which was then suggested to do pars plana vitrectomy. This intervention was taken as significant bilateral, extent vitreous opacities, to the patient who was still productive.

Reported complications of Terson’s syndrome include visual loss, macular holes, epiretinal membrane formation, retinal folds, proliferative vitreoretinopathy, and retinal detachment. It has been suggested that these complications are the result of proliferation of glial and retinal pigment epithelial elements capable of causing retinal distortion and fibrotic adhesions. The most common complication is the formation of epiretinal membranes, observed in 27-78% of patients with Terson’s syndrome.

Terson’s syndrome has been correlated significantly in several studies with elevated morbidity and mortality when compared with subarachnoid hemorrhage without intraocular bleeding. Vitreous hemorrhage is associated with a 3-9 fold higher rate of mortality in comparison to other sites of intraocular bleeding in Terson’s syndrome.
Patients able to survive the neurologic complications usually have a favorable visual prognosis. The prognosis for vision recovery in surviving patients with SAH has been reported to be good. Most vitreous body bleedings clear spontaneously within months. If vitrectomy is performed, complete recovery of vision can be expected in many cases. In a study of 30 patients with Terson's syndrome, over 83% of patients achieved a long-term visual acuity better than 20/50 following observation or a vitrectomy.

The vital prognosis of these cases are good. The functional prognosis is dubia.

IV. Conclusion

This case resume is a bilateral vitreous hemorrhage caused by blunt head trauma assume as Terson's syndrome.
REFERENCES


