Terson's Syndrome – A Report of Two Cases

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SUMMARY

Introduction Vitreous or retinal hemorrhage occurring in association with subarachnoid hemorrhage is known as Terson's syndrome. In Terson's syndrome, intracranial hemorrhages are followed by intraocular hemorrhage, classically in the subhyaloid space, but may also include subretinal, retinal, preretinal, and vitreal collections. Vitreous hemorrhage recovery is usually spontaneous in six to 12 months, otherwise vitrectomy is considered.

Outline of Cases We report of two cases of Terson's syndrome. The first was in a hypertensive middle-aged female, following anterior communicating artery aneurismal subarachnoid hemorrhage, after post-neurosurgical interventions. The second case report was of a young male who suffered from the bilateral vitreous hemorrhage after a severe traumatic brain injury.

Conclusion Terson's syndrome should be considered in patients who had previous cerebral hemorrhage and are referred to eye specialist because of loss of vision. However, this phenomenon has only rarely been described in association with subdural and epidural hematomas or traumatic subarachnoid hemorrhage.

Keywords: cerebral hemorrhage; rupture of the aneurysm; traumatic brain injury; Terson syndrome; vitreous hemorrhage

INTRODUCTION

Vitreous or retinal hemorrhage occurring in association with subarachnoid hemorrhage (SAH) is known as Terson's syndrome (TS) [1]. A French ophthalmologist, Albert Terson, is credited with discovering this clinical sign in a patient with SAH in 1900 [2], although in 1881 Litten [3] described vitreous bleeding occurring in association with SAH. Although it is a well described entity in the ophthalmological literature, it has been only rarely commented upon in the neurosurgical discussion of SAH [4]. The etiopathology of TS has been controversial since its inception. Early investigators believed that the resulting intraocular hemorrhage (IOH) emanated from the direct dissection of SAH down the optic nerve sheath. However, poor anatomical communication between the subarachnoid space of the optic nerve and the vitreous humor makes this mechanism unlikely. Currently, sudden spiking of intracranial pressure occurring at the time of an intracranial bleed is recognized as the primary event that precipitates the intraocular bleeding [5]. In TS, intracranial hemorrhages are followed by IOH, classically in the subhyaloid space, but may also include subretinal, retinal, preretinal, and vitreal collections [6]. Vitreous hemorrhage recovery is usually spontaneous in six to 12 months, otherwise vitrectomy is considered, although currently there is research claiming that the vitrectomy should be done earlier [7]. Incidence of TS has been quoted by various studies to be in the range of 10–50% following SAH [8]. We report of two cases of TS. The first in a hypertensive middle-aged female, following the anterior communicating artery aneurysmal SAH, after post-neurosurgical interventions and one year of follow up; and the second one, the young male who suffered from the bilateral vitreous hemorrhage after a severe traumatic brain injury (TBI) and two years of follow-up. We also summarize the relevant literature, as well as reference sections of selected articles.

CASE REPORTS

Case report 1

We report data of a 45-year-old Caucasian female who was referred to University Eye Clinic, Clinical Center Serbia, Belgrade, because of visual loss on the left eye after neurosurgical procedure. The patient was admitted to the Neurosurgical Clinic, Clinical Center of Serbia in Belgrade, as an emergency case because of sudden headache accompanied by nausea, vomiting and depression of awareness. An urgent computed tomography (CT) scan of the endocranium was performed, where existence of spontaneous rupture of the aneurysm with SAH was proven. After performed CT angiography of cerebral blood vessels, which pointed to the existence of spontaneous rupture of an aneurysm of the communicating artery, neurosurgical operative treatment was indicated. Thus, neck clipping was performed on the same day. Although her consciousness level gradually improved after operation, she complained of left vision disturbance immediately after operation. Postoperative control CT scan was done,

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Figure 1. Ocular fundus image from a patient with Terson's syndrome six months after onset of visual symptoms – persistent vitreous hemorrhage

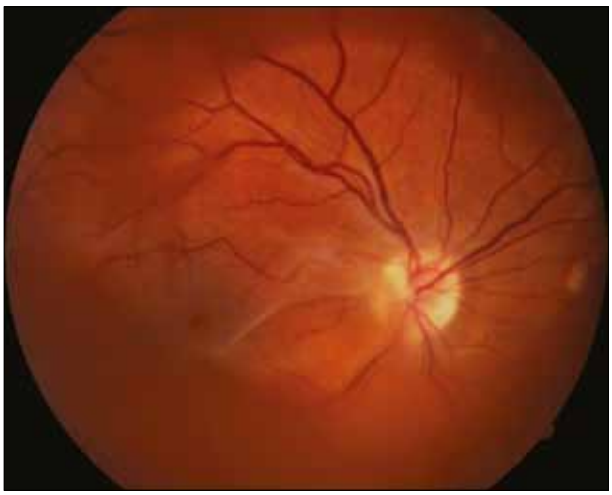

Figure 2. Fundus photography of the right eye – preretinal fibrosis starting from the optic disc and extending along posterior pole, and persistent vitreous hemorrhage

which pointed to the state after clipping aneurysm, without the existence of hydrocephalus and hematoma in the lodge. The patient was discharged from the neurosurgical clinic in normal neurological condition eight days after surgery. She was referred to our clinic. On admission, ophthalmological evaluation revealed normal visual acuity in the right eye (1.0), and reduced in the left eye (1/60), bilaterally normal anterior segments, normal pupils (bilaterally 3 mm, round, regular, and exhibiting both direct and consensual light reflexes), and high intraocular pressures in the left eye (32 mmHg) – ghost cell glaucoma. It was precisely examined using fundoscopic examination and B-mode ultrasonography making the diagnosis of left vitreous hemorrhage (TS) caused by SAH. Ultrasonography of the affected eye confirmed the massive vitreous hemorrhage and ruled out retinal detachment, retinal tear, epiretinal membrane, or other abnormality of retina, choroid or sclera. The right eye was unremarkable. We decided to follow the patient periodically to monitor for clearing of the vitreous hemorrhage with conservative treatment of raised intraocular pressure. During the second postoperative month, her vision in the left eye began to improve. At the tree-month follow-up, best corrected visual acuity on her left eye was 0.4. Results of the ophthalmic examination disclosed persistent vitreous hemorrhage, but the posterior pole and upper retinal periphery were visible and showed no abnormality (Figure 1). Ultrasonography showed less blood in the vitreous than in the initial stage. After six months of follow-up, spontaneous clearing was sufficient to make vitrectomy unnecessary. Visual acuity was 1.0 on the right eye, 0.6 on the left eye. Intraocular pressure was within range in both eyes.

Case report 2

We present a case report of a 22-year-old male who presented with TS on both eyes with subdural cerebral hemorrhage secondary to TBI. The patient had fallen from the second floor of a building, following which he developed a right front-parietal contusion. He was hospitalized in the Neurosurgery Clinic, Clinical Center of Serbia in Belgrade. A CT scan of the patient showed subdural hematoma and cerebral edema, which was managed with conservative medical treatment. Ophthalmoscope clearly demonstrated vitreous hemorrhage in both eye globes. Visual acuity and other function tests could not be performed because the patient was in a coma for a day. The patient's condition improved slowly, with progressive reabsorption of the hematoma and normalization of ventricular morphology of the brain as shown by CT scan. At the time of discharge, one month subsequent to the TBI, the patient was conscious with no motor deficit and the front-parietal contusion had mostly resolved. The only problem the patient had was low vision in both eyes, more pronounced in the right eye. The patient was referred to the University Eye Clinic of the Clinical Center of Serbia in Belgrade. Visual acuity in the right eye was determined by hand movements at one meter, and it was determined to be 0.8 in the left eye. Intraocular pressure was within range in both eyes. The patient's ophthalmic signs were as follows: vitreous hemorrhage in both eyes, denser in the right eye, preretinal hemorrhage in the left eye nasally from the papilla, macula lutea and optic papilla in both eyes with no abnormality. We followed this patient monthly, performing visual acuity assessment, fundus examination, color retinography and B-scan ultrasonography. His vision in the right eye began slowly to improve. After four months of follow-up, best corrected visual acuity of his right eye was 0.2, and of his left eye 1.0. Results of the ophthalmic examination disclosed persistent vitreous hemorrhage in the right eye, but the posterior pole and upper retinal periphery were visible and showed no abnormality. Due to gravity the blood mostly collects in the inferior half of vitreous, from where it slowly gets absorbed. In the left eye there was no previously described preretinal hemorrhage. After one year of follow-up, retinal examination of the right eye revealed preretinal fibrosis starting from the optic disc and extending along posterior pole, as well as persistent vitreous hemorrhage (Figure 2). Optical coherence tomography of the right eye showed preretinal membrane with traction near macula lutea (Figure 3). As

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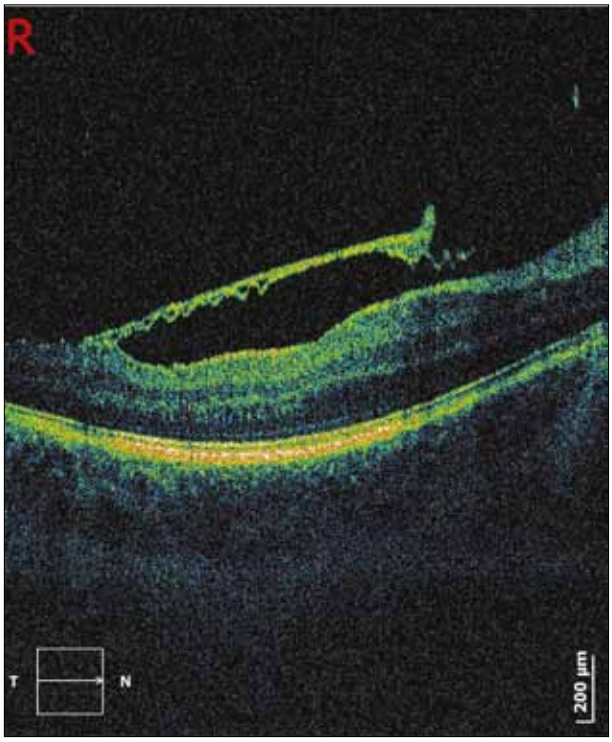

Figure 3. Optical coherence tomography of the right eye showing preretinal membrane, macula lutea and optic papilla traction

his visual acuity of the right eye was 0.9, we abstained from vitrectomy. Our patient was to be monitored on a four-month basis as long as no new symptoms and signs occur, i.e. visual deterioration or retinal detachment.

DISCUSSION

Terson's syndrome is defined as vitreous or retinal hemorrhage associated with SAH and is thought most likely to result from sudden large increase in intracranial pressure [9]. The pathogenesis of the ocular findings is thought to be related to disturbed circulation in the retinal vessels consequent to the increased intracranial pressure, which leads to retinal venous hypertension and, eventually, to hemorrhage [10]. It is seen in 10–50% of patients with spontaneous or traumatic SAH [8]. Generally, it begins as bleeding between the internal limiting membrane and the retina. The blood may remain confined in this space and appear as a mound on the retina, or may partially or completely decompress through the hyaloid membrane and seep into the vitreous [10]. It is most commonly seen after anterior circulation aneurismal rupture, especially the anterior communicating artery or internal carotid artery [4], such as in our first case. Rare causes include subdural hematoma, traumatic SAH, severe brain injury [11] and posterior circulation or vertebral artery aneurismal rupture [12]. In our second case, it is hypothesized that the cause of TS may have been a rapid elevation in ICP at the time of TBI, which affected peripapillary structures through the intervaginal space of the optic nerve sheath [13]. The clinical course of this IOH is variable. While in some patients the hemorrhage clears spontaneously, many will incur vision loss, chronic hematoma, or epiretinal membranes requiring ophthalmologic care, including vitrectomy [10]. Some of the previous studies did not distinguish vitreous hemorrhage from other types of IOH. However, the current findings suggest that vitreous hemorrhage on its own is an indicator of poor prognosis in patients with SAH. In one study there was also a suggestion that different types of IOH had different prognostic significance, in that mild retinal hemorrhages were more strongly associated with better prognosis than large preretinal hemorrhages or vitreous hemorrhages [1]. In another study, no difference in mortality was found between patients with and patients without IOH [4]. Therapeutic management of TS depends on the position of the IOH. For vitreous or subhyaloid hemorrhage the treatment can be conservative, i.e. based on periodic observation awaiting spontaneous resorption, or more aggressive, i.e. pars plana vitrectomy. In several cases, spontaneous resorption is observed within a few months from the acute event. Head-end elevation with bed rest and avoidance of anticoagulation medications may benefit patients. Immediate vitrectomy for IOH is not recommended, except in cases of submacular hemorrhage, monocular with severe visual loss, or in pediatric patients at risk of amblyopia [6]. Therefore, according to some authors, vitrectomy should be delayed and considered only in severe or bilateral cases that do not show signs of recovery [14]. Most authors recommend a period of six months after the acute event for the timing of surgery [12]. A surgical approach is not possible in the presence of intraretinal hemorrhage [14]. Nevertheless, all patients should be closely monitored for sequelae of intraocular bleeding. These include the development of intraocular hypertension and retinal membrane formation with resulting retinal detachment [12].

The presence of bleeding in posterior eye compartments is a common complication associated with cerebral hemorrhage. In TS the bleeding typically clears completely within several months. In some cases, severe non clearing vitreous hemorrhage or epiretinal membrane formation may result in a significant decrease of vision. In such cases, vitrectomy can improve the visual outcome. Close ophthalmological and radiological evaluation is required in all patients with cerebral hemorrhages, as early diagnosis and treatment of TS may prevent visual loss and associated complications.

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Терсонов синдром – приказ два болесника

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КРАТАК САДРЖАЈ

Увод Витреално и ретинално крварење настало након субарахноидалног крварења је познато као Терсонов (*Terson*) синдром. Код овог синдрома интракранијално крварење доводи до интраокуларног крварења, које је обично локализовано у субхијалоидном простору, мада може да се јави и као субретинална, преретинална и витреална накупина крви. Најчешће се јавља након руптуре анеуризме предње комуникантне артерије и унутрашње каротидне артерије, док се ређе јавља као последица субдуралног хематома и трауматске субарахноидалне хеморагије.

Приказ болесника Представљамо два случаја Терсоновог синдрома. Први случај је једнострано интравитреално крварење настало након субарахноидалног крварења због руптуре предње комуникантне артерије код средовечне жене с хипертензијом, док је други случај 22-годишњег момка с обостраним витреалним крварењем насталим као последица субдуралног церебралног хематома након тешке трауматске повреде мозга.

Закључак На Терсонов синдром треба помислити код особа које су доживеле моћдано крварење и које су упућене код очног лекара због губитка вида. Овај синдром се ретко доводи у везу са субдуралним и епидуралним хематомом, као и трауматском субарахноидалном хеморагијом. Лечење витреалне и субхијалоидне хеморагије може да буде конзервативно или хируршко код тешких и обостраних случајева без знакова повлачења крварења.

Кључне речи: церебрално крварење; руптуре анеуризме; трауматска повреда мозга; Терсонов синдром; витреално крварење

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