CASE REPORT

Terson’s Syndrome Successfully Treated with Combined Therapy

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Purpose: To present a case of Terson’s syndrome consequent to ruptured intracranial aneurism treated successfully with a combined therapeutic approach. 

Methods: Case report. A 37 year old man was urgently referred to the UCC Tuzla with sudden headache, vomiting and unconsciousness. CT scan showed ruptured aneurism of anterior communicant artery with signs of retinal thickening. Later ocular examination showed the massive bilateral vitreous haemorrhage.

Results: Patient was treated with medicamentous therapy for 7 months with no significant improvement. 7 months after aneurism rupture patient agreed to a surgical treatment. Right eye was treated surgically and left eye continued with conservative therapy. Visual acuity after vitrectomy improved in right eye to 1,0 and 0,5 in left eye.

Conclusions: Terson’s syndrome is relatively common complication of subarchnoid haemorrhage. Vitrectomy is safe and efficient approach to treat patients with Terson’s syndrome. 

Key words: Terson’s syndrome, SAH, vitrectomy

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1. INTRODUCTION

Terson’s syndrome is defined as intracocular haemorrhage in association with any type of intracranial haemorrhage (1). Vitreous bleeding occurring with subarchnoid haemorrhage (SAH) was first described by German ophthalmologist Moritz Litten in 1881 (2). Definition is later extended to any vitreous haemorrhage occurring in conjunction with any form of intracranial bleeding and it is named Terson’s syndrome in honour of French ophthalmologist Albert Terson (3, 4). It occurs most commonly due to ruptured intracranial aneurism, raised intracranial pressure or after serious brain injury. The reported incidence of vitreous haemorrhage in patients with subarchnoid haemorrhage is 13% in prospective studies (5).

Here we present a case of Terson’s syndrome in young patient where we decided to combine surgical and conservative ophthalmic therapy for both eyes.

2. CASE REPORT

A 37 year old man was urgently referred to the Neurology Clinic University Clinical Centre Tuzla with sudden headache, vomiting and consciousness. No ocular manifestations were present at that time. An urgent Computer Tomography (CT) angiography revealed aneurism of anterior communicant artery and CT scan of head showed massive subarchnoid haemorrhage (Fisher grade 3) with signs of retinal thickening and initial vitreous haemorrhage adjacent to optic nerve (Figure 1). No signs of haemorrhage in optic nerve were found.

Patient was shifted to Neurosurgery Clinic UCC Tuzla where urgent craniotomy with clipping of the anterior communicant artery was done. In early postoperative rehabilitation visual impairment in both eyes was noticed due to the vitreous haemorrhage. Patient had slow postoperative recovery and we were not able to pursue further ophthalmic diagnostic.

Three months after the first attack, visual acuity was finger counting in both eyes. Ophthalmic ultrasound showed massive vitreous haemorrhage with complete vitreous detachment (Figure 2). Vitrectomy was suggested at that time but patient was not motivated for another operation either in general or local anaesthesia. Patient was treated with intermittent conservative ophthalmic therapy consisting of etamsylate, trypsin-chymotrypsin and vitamin C.

Figure 1. CT scan obtained on the day of the intracranial haemorrhage showing hyperdense retinal thickening and initial vitreous haemorrhage adjacent to optic nerve (arrow)
but no significant improvement in visual acuity was achieved at that time.

Seven months after initial attack, upon finishing psychiatric and physiatrist rehabilitation, patient agreed for operation of right eye to clear the haemorrhage. Visual acuity before operation was light perception in right eye and 0.1 in left eye. Preoperative ophthalmic ultrasound showed massive organised vitreous haemorrhage. On the other hand CT and MRI showed no signs of vitreous or optic nerve changes. (Figure 3).

Pars plana vitrectomy, peeling of epiretinal membrane and endolaser barrage was performed in right eye. Operative finding showed massive organised haemorrhage attached to optic nerve with diffuse epiretinal membrane and peripheral retinal breaks with no signs of intra or subretinal bleeding. Early postoperative rehabilitation was successful and visual acuity at the last follow up (3 months after the operation) improved to 1.0 in right-operated eye and 0.5 partially in left eye. Patient still has massive intravitreal changes in the left eye (Figure 4) but has no intention of undergoing another operation. Patient’s general condition is good and significant psychiatric improvement is noticed.

3. DISCUSSION

Vitreous haemorrhage is relatively common complication in patients with intracranial haemorrhages. Its incidence ranges between 8 to 46% (5, 6, 7, 8, 9, 10, 11, 12, 13). Recent studies show incidence of Terson’s syndrome to be approximately 13% and prospective studies identify more patients with vitreous haemorrhage than retrospective studies (5). Terson’s syndrome occurs often with SAH but it has been described in association with subdural or epidural haematomas and traumatic SAH (6, 13, 14, 15, 16, 17). There are reports of iatrogenic Terson’s syndrome as postoperative complication after neurosurgical interventions (18). Another patient was reported to have intracranial haemorrhage caused by coagulopathy as consequence of acute promyelocytic leukaemia after retinoid acid treatment (19).

Terson’s syndrome was noted in infants with severe brain injury and trauma (20) and it is also associated with shaken baby syndrome (21).

Most of patients (89%) suffering with subarachnoid haemorrhages have history of unconsciousness (9). Visual loss is usually noticed after the surgery or when patient regain consciousness. Vitreous haemorrhage can cause a considerable visual handicap but there is delay in appearance visual symptoms which is reported to be between 24 hours and 47 days (22, 23). Damage to the visual pathway correlates with the severity of SAH and timing of aneurismatic surgery (24). Despite severity of symptoms, reports in show that mean interval between visual symptoms and referral to an ophthalmologist is up to 5 months (25).

Intraocular haemorrhages occur in vitreous, subhyaloid, in various layers and locations of the retina, particularly the macula, sub-internal limiting membrane (ILM), intraretinal, and subretinal spaces, in association with macular holes, retinal detachments, and optic neuropathy (26, 27, 28, 29, 30). In some cases blood occurs along optic nerves, within nerve sheaths and in the subdural and subarachnoid spaces (26, 27). The pathophysiology of Terson’s syndrome is still controversial. One theory suggest that rapid increase in intracranial pressure can result in influx of cerebrospinal fluid into the subarachnoid space of optic nerve sheath, causing dilatation and compression of blood vessels, resulting in stasis of venous drainage of retina and result with intraocular haemorrhages (7, 8, 10, 31, 32). Some other studies suggest that blood from subarachnoid space enter directly the vitreous space through intervaginal space around the optic nerve (33, 34, 35). In general first mechanism is widely accepted although argument is not finished yet.

Presence of Terson’s syndrome has been reported to be predictive factor for poor clinical outcome. Studies show that overall mortality rate is higher in patients with Terson’s syndrome be-
between 10% and 60% (5, 7, 8, 10, 36, 37). Studies show that 18-24% of patients with subarachnoid haemorrhage die in first 24 hours upon symptoms onset and do not reach hospital to be clinically accessed (38). This means that many patient die before intraocular haemorrhage develop, which can be limiting assessment factor.

Several authors reported that vitreous haemorrhage in Terson’s syndrome can resolve spontaneously in period between 6 and 29 months (23, 37, 39, 40). On the other Timberlake reported that they never observed a complete resolution of vitreous haemorrhage in any of their patients. Recent studies suggest that surgical treatment namely pars plana vitrectomy is successful and safe method to treat ocular complications in Terson’s syndrome with successful rate up to 98% (8, 13, 23, 25, 42, 43, 44, 45, 46). Some authors argue these results indicating high incidence of severe postoperative complications (46) Reported late and postoperative complications are cataract (27-36%) (25, 44), retinal detachment, secondary glaucoma and retinal changes with proliferative vitreoretinopathy (13, 25). Results of surgical treatment depend on operation timing and many suggest that vitrectomy should be performed in those cases where no spontaneous resolution happen in 6 months (8, 23, 25) and some suggest that it should be made earlier even before neurosurgical treatment (13). Generally patients with operative delay less than 3-6 months and patients younger than 45 years of age experience better functional recovery.

We present a first case operative treatment of patient with Terson’s syndrome in our clinic. We successfully combined operative and conservative therapy in both eyes of one young patient. No evidence on mechanism of ocular haemorrhage is found. Operative treatment was successful with no any complication mentioned in literature. Organised haemorrhage in vitreous attached to optic nerve, epiretinal membrane with retinal breaks was found during the operation. No other characteristic findings have been found. Conservative treatment in left eye was also partially successful but haemorrhage was much smaller and patient still has risk of late complications such as proliferative vitreoretinopathy. We suggested operation in left eye too, but patient was not motivated even though we explained possible late complications. Due to significant visual improvement patients life quality improved resulting with physical and psychiatric improvement.

Terson’s syndrome is underreported complication in our clinic. Careful and serial ophthalmic examination of all patients with intracranial haemorrhages should be performed for detection of ophthalmalic complications such as Terson’s syndrome which can be used as important prognostic factor. Terson’s syndrome can be treated successfully both with operative and conservative therapy.

REFERENCES