Terson’s syndrome in subarachnoid hemorrhage and severe brain injury accompanied by acutely raised intracranial pressure

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Object. The syndrome of retinal or vitreous hemorrhage in association with subarachnoid hemorrhage (SAH) is known as Terson’s syndrome. The authors’ purpose was to determine whether intraocular hemorrhage occurs with similar incidence when caused by severe brain injury accompanied by acutely raised intracranial pressure (ICP).

Methods. Prospective ophthalmological examination was performed in 22 consecutive patients with SAH or severe brain injury and elevated ICP. Thirteen patients were admitted for SAH (World Federation of Neurological Surgeons Grades II–IV) and nine for severe brain injury (Glasgow Coma Scale scores 3–10). Monitoring of ICP was performed at the time of admission via a ventricular catheter. Initial ICP exceeded 20 mm Hg in all patients. Indirect ophthalmoscopy without induced mydriasis was performed within the 1st week after the acute event. Retinal or vitreous hemorrhage was seen in six (46%) of 13 patients with SAH and in four (44%) of nine patients with severe brain injury. Ocular bleeding was found bilaterally in three patients with SAH and in one patient with severe brain injury (18%). Six of the 10 patients with Terson’s syndrome died as a result of their acute event.

Conclusions. The present results indicate that Terson’s syndrome may be related to acute elevation of ICP, independent of its causes, and may occur with similar incidence in patients with severe brain injury and those with SAH. Because recognition and treatment of Terson’s syndrome may prevent visual impairment and associated secondary damage to the eye, increased awareness of this entity in all patients with acute raised intracranial hypertension is recommended.

KEY WORDS • Terson’s syndrome • subarachnoid hemorrhage • severe brain injury • intracranial pressure
in two surviving patients with Terson’s syndrome; the other two surviving patients were lost to follow up.

Statistical Analysis

All values are given as means ± standard deviation. Statistical analysis was performed using the Whitney-Mann-Wilcoxon test. A significant difference was assumed with an error probability of less than 0.05.

Results

There was no significant difference in maximum, initial, and average ICP within the first 72 hours in patients with SAH (mean maximum ICP 27 ± 9.6 mm Hg; mean initial ICP 17 ± 8.3 mm Hg; and mean average ICP 14 ± 9.4 mm Hg) compared with patients with severe brain injury (mean maximum ICP 26.5 ± 23.9 mm Hg; mean initial ICP 19 ± 16.6 mm Hg; and mean average ICP 14 ± 11.4 mm Hg).

Terson’s syndrome (Fig. 1) was observed in 10 (45%) of 22 patients. Six (46%) of 13 patients with SAH and four (44%) of nine patients with severe brain injury (44%) had retinal or vitreous hemorrhage. Ocular bleeding was found bilaterally in three cases of SAH and in one case of severe brain injury (18%). There was no correlation between the severity of the intracerebral pathological conditions graded on CT scans and the amount of intraocular hemorrhage.

With one exception, initial coma was observed in all patients with intraocular hemorrhage. Seven patients with ocular findings showed extremely raised ICP (> 30 mm Hg) during ventricle catheter placement. The initial ICP
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values measured in the ICU and the maximum measured ICP values were significantly higher in patients with Terson’s syndrome (mean initial ICP 26 ± 6.9 mm Hg in patients with intraocular hemorrhage compared with 15 ± 5.8 mm Hg in others; mean maximum ICP 32 ± 15.5 mm Hg in patients with intraocular hemorrhage compared with 28 ± 11.5 mm Hg in others; mean average ICP 16.5 ± 11.3 mm Hg in patients with intraocular hemorrhage compared with 13 ± 7.3 mm Hg in others; Fig. 2).

The outcome of patients with intraocular bleeding was poor. Six of 10 patients died within the 3-month follow-up period. One surviving patient with SAH still had not regained vision in one eye at that time because of severe nonclearing vitreous hemorrhage. This patient later underwent pars plana vitrectomy and vision improved to 0.8 within the following weeks. The patient with brain injury who was available for follow-up review showed no visual deterioration.

Discussion

The occurrence of bleeding in posterior eye compartments is a known complication in patients surviving SAH. The reported frequency of Terson’s syndrome in such patients ranges between 20% and 50%, which is in accordance with our results. However, this phenomenon has only rarely been described in association with subdural and epidural hematomas or traumatic SAH. Only case reports are found in the literature. Billotte, et al., described Terson’s syndrome in an infant with severe brain injury. Another patient was reported to have intraocular hemorrhage caused by coagulopathy as a consequence of acute promyelocytic leukemia after retinoic acid treatment. Our results suggest that intraocular hemorrhage occurs with similar incidence when caused by severe brain injury compared with SAH and may be responsible for vision impairment in such patients.

Coincident injury to the optic nerve and optic pathways can be found in many cases of severe brain injury. Thus, we cannot rule out that intraocular hemorrhage in these patients may have been a condition merely associated with more profound damage to the optic system. Unfortunately, afferent pupillary testing, which might have provided the respective information, was not performed in our study. On the other hand, the available literature on Terson’s syndrome does not provide evidence for injury to afferent pathways in cases of severe SAH. Although literature on the topic is sparse, Karel and Gergelyova reported on seven patients with SAH and Terson’s syndrome. All underwent pars plana vitrectomy. In six of their patients, vision recovered to values between 0.7 and 1. In the only patient with severe brain injury who was available for follow-up review in our study, no impairment of vision was noted. In another patient with SAH, vision was only restored after pars plana vitrectomy had been performed.

Initial attempts at explaining Terson’s syndrome suggested that, in SAH, blood traverses the subarachnoid space into its continuation within the optic nerve sheath. It was thought that blood penetrates the sclera in the porous region where the optic nerve enters the globe, and finally appears in the vitreous space within the eye. Many textbooks still attribute Terson’s syndrome to this mechanism, despite evidence that there is no connection between the optic nerve sheath subarachnoid space and the vitreous body. Castren suggested that rapid increases in ICP result in Terson’s syndrome, which is caused by venous congestion due to impairment of venous drainage to the cavernous sinus. Retinchoroidal connections and the central retinal vein could be compressed by pressure-induced dilation of the optic nerve–sheath subarachnoid space.

In the initial phase of SAH, dramatic increases in ICP are noted for several minutes. Such a pressure pattern can also be observed in severe brain injury initially caused by hypercarbia in the unconscious patient. Furthermore, posttraumatic Terson’s syndrome may result from plateau waves, which are well described in the early posttraumatic phase. Other patterns of intracranial hypertension without extreme ICP peaks, which are observed, for instance, in hydrocephalus, cavernous sinus thrombosis, or carotid cavernous sinus fistula, do not result in Terson’s syndrome. A typical ophthalmological finding in such patients is papilledema but not intraocular hemorrhage.

The outcome of patients with Terson’s syndrome has been reported to be poor. In many series the overall mortality rate is significantly higher than that for patients without Terson’s syndrome. Garfinkle and colleagues reported a 36.3% mortality rate in patients with intraocular hemorrhage; Pfaueler, et al., reported death in nine of 10 patients. In our study we observed a mortality rate of 60%.

The prognosis for vision recovery in surviving patients with SAH has been reported to be good. Most vitreous body bleedings clear spontaneously within months. Conversely, the diagnosis of Terson’s syndrome may be important with regard to management because severe non-
clearing vitreous hemorrhage may result in blindness.\textsuperscript{7,13,25} In cases in which there is no tendency for blood resorption, the method of choice is a pars plana vitrectomy.\textsuperscript{18} Most authors recommend a period of 6 months after the acute event for the timing of surgery.\textsuperscript{19} If vitrectomy is performed, complete recovery of vision can be expected in many cases.\textsuperscript{6,10,26,31,37} Alternatively, intravitreous injections of anti-Rh serum have been reported to improve visual acuity within 5 to 6 weeks in cases of vitreous hemorrhage without proliferative vitreoretinopathy.\textsuperscript{29} 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.\textsuperscript{9,26}

With regard to our own observations, multiple intracranial pathological conditions accompanied by acutely raised ICP may result in intraocular hemorrhage. The frequencies were similar in patients who had severe brain injury compared with those who had SAH. For surviving patients, close ophthalmological evaluation and treatment are recommended.

References


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