Neuroimaging Highlight

Editors: Richard Farb, David Pelz

Terson's Syndrome

Submitted by: Francois Paquette, Tim E. Darsaut, Mikael Sebag, Alain Weill

Can. J. Neurol. Sci. 2010; 37: 861-862

CASE REPORT

A 51-year-old male was admitted following subarachnoid hemorrhage (SAH) (Figure 1). His Glasgow coma score was 15. Other than headache and a defect in his right visual field, his neurological exam was normal. Cerebral angiography demonstrated a 3 mm anterior communicating artery aneurysm which was successfully excluded with endovascular coiling. Due to persistent visual complaints, an ophthalmology consult was obtained, which revealed a scotoma of the right eye. Fundoscopy demonstrated a large subhyaloid hemorrhage of the right eye (Figure 2), and a small retinal hemorrhage with papilledema of the left eye. Intraocular pressure was slightly higher than normal, 31 mmHg, in both eyes. The diagnosis of Terson's syndrome (TS) was made, and the decision was made to follow him conservatively for at least six months.

DISCUSSION

The pathogenesis of TS has been much debated. Initial theories suggested that blood in the subarachnoid space could track directly into the sclera and vitreous space through the lamina cribrosa¹⁻³. However, anatomic studies have failed to demonstrate a connection joining the subarachnoid space of the optic nerve sheath to the vitreous space^{1,4}. Moreover, vitreous hemorrhage has been documented to occur with diseases other than SAH⁵. The most widely accepted current theory suggests that a sudden acute increase in intracranial pressure (ICP) leads to effusion of cerebrospinal fluid within the optic nerve sheath, leading to compression of the central retinal vein and the retinochoroidal anastomotic vessels, with an increase in venous pressure accounting for the intraocular hemorrhage^{1,2,6,7}.

Terson's syndrome is reported to occur in 2.6 to 27% of patients following SAH, depending on how carefully the diagnosis is sought⁸. Patients with subhyaloid hemorrhage generally complain of scotoma as compared to those with vitreous hemorrhage, who report blurred vision and floaters. Typically, patients with TS have a worse clinical SAH grade on admission^{6,8}, likely in part due to the sudden increase in ICP required to cause intraocular hemorrhage. The acute increase in ICP immediately following SAH may last only a few minutes⁹,

Figure 1: CT scan demonstrating SAH with an associated hyperdense crescent at the level of the retina of the right eye, corresponding to an intra-ocular hemorrhage.

and the anterior communicating artery location adjacent to the optic nerve sheath may allow for a more focal increase in ICP without generalized cerebral injury. In this case, our patient suffered a significant intraocular hemorrhage, with visual loss his sole presenting complaint other than headache.

Most vitreous hemorrhages due to TS resolve spontaneously, with the majority of patients making good recovery of vision^{8,10}.

From the Department of Radiology, Centre hospitalier de l'Université de Montréal (CHUM), Hôpital Notre-Dame, Montréal, Québec, Canada.

RECEIVED APRIL 30, 2010. FINAL REVISIONS SUBMITTED MAY 27, 2010.

Correspondence to: Alain Weill, CHUM Notre-Dame Hospital, Department of Radiology, 1560 Sherbrooke East, Pavillon Lachapelle, Suite B-1036, Montreal, Quebec, H2L 4M1, Canada.

Figure 2: Fundoscopy in Terson's syndrome demonstrating subhyaloid hemorrhage.

However, delayed clot resorption may be associated with complications such as epiretinal membrane formation and retinal detachment and permanent visual loss¹¹. After a six month trial of conservative management, surgical drainage of the hematoma is generally recommended, with good outcomes in the majority of cases^{6,8}.

CONCLUSION

Intraocular hemorrhage following SAH is thought to result from a rapid increase in ICP, usually in association with profound neurological injury. It is uncommon for a patient with a large intraocular hemorrhage following SAH to be in such good clinical condition. Physicians managing SAH should be aware of the typically benign clinical course of the visual loss associated with Terson's syndrome.

REFERENCES

- Walsh FB, Hedges TR Jr. Optic nerve sheath hemorrhage. *Am J Ophthalmol*. 1951 Apr ;34(4):509-27.
- Manschot WA. Subarachnoid hemorrhage; intraocular symptoms and their pathogenesis. *Am J Ophthalmol*. 1954 Oct;38(4): 501-5.
- Doubler FH, Marlow SB. A case of hemorrhage into the optic nerve sheath as a direct extension from diffuse intra-meningeal hemorrhage caused by rupture of aneurysm of cerebral artery. *Arch Ophthalmol*. 1917;46:233-6.
- Anderson DR. Ultrastructure of the optic nerve head. *Arch Ophthalmol*. 1970 Jan;83(1):63-73.
- Hoving EW, Rahmani M, Los LI, Renardel de Lavalette VW. Bilateral retinal hemorrhage after endoscopic third ventriculostomy: iatrogenic Terson syndrome. *J Neurosurg*. 2009 May;110(5):858-60.
- Garfinkle AM, Danys IR, Nicolle DA, Colohan AR, Brem S. Terson's syndrome: a reversible cause of blindness following subarachnoid hemorrhage. *J Neurosurg*. 1992 May;76(5): 766-71.
- Muller PJ, Deck JH. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg*. Aug 1974;41(2):160-6.
- Fountas KN, Kapsalaki EZ, Lee GP, et al. Terson hemorrhage in patients suffering aneurysmal subarachnoid hemorrhage: predisposing factors and prognostic significance. *J Neurosurg*. 2008 Sep;109(3):439-44.
- Nornes H, Magnaes B. Intracranial pressure in patients with ruptured saccular aneurysm. *J Neurosurg*. 1972 May;36(5): 537-47.
- Vanderlinden RG, Chisholm LD. Vitreous hemorrhages and sudden increased intracranial pressure. *J Neurosurg*. 1974 August;41: 167-76.
- Oyakawa RT, Michels RG, Blase WP. Vitrectomy for nondiabetic vitreous hemorrhage. *Am J Ophthalmol*. 1983 Oct;96(4):517-25.