

Neuroimaging Highlight

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Terson's Syndrome

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

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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,

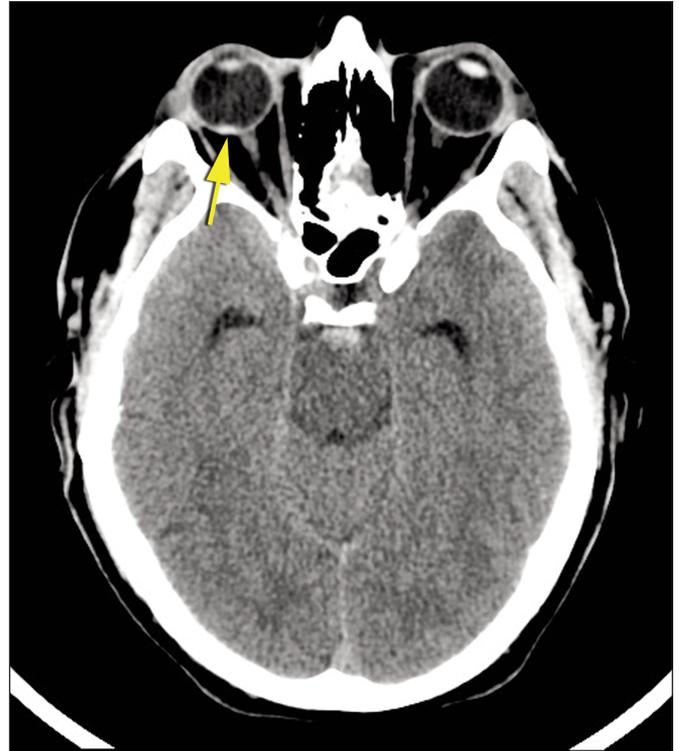


Figure 1: CT scan demonstrating SAH with an associated hyperdense crescent at the level of the retina of the right eye, corresponding to an intra-ocular hemorrhage.

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.

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

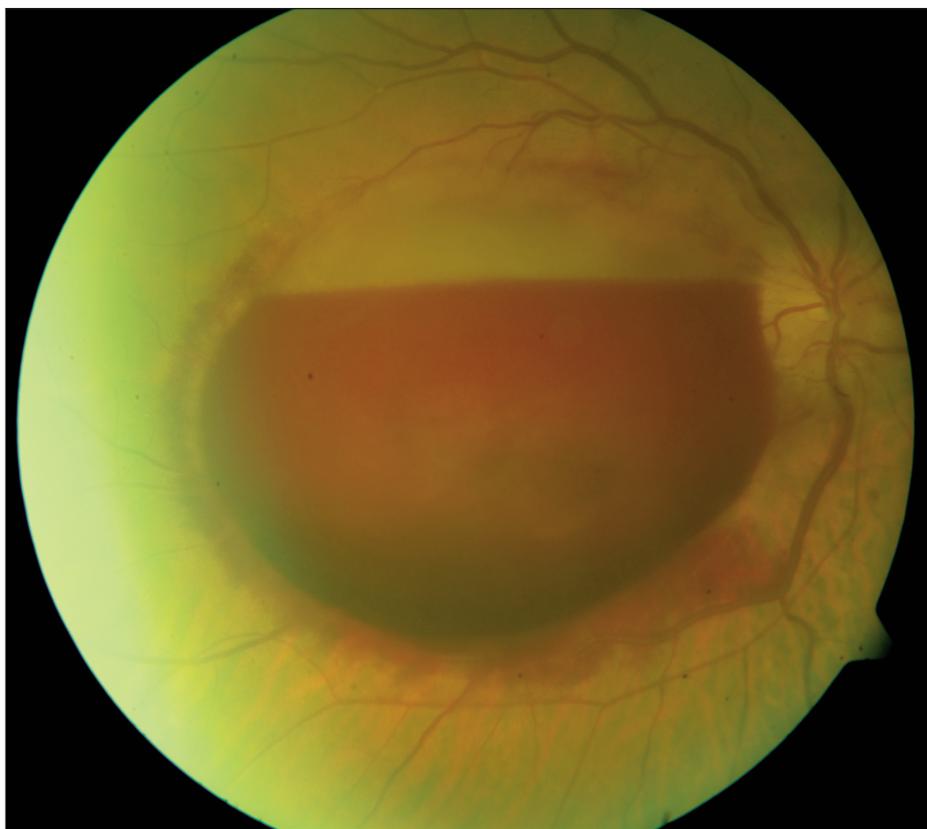


Figure 2: Fundoscopy in Terson's syndrome demonstrating subhyaloid hemorrhage.

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.

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