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Ruptured Anterior Communicating Artery Aneurysm with Terson's Syndrome

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Spontaneous subarachnoid hemorrhage (SAH) is a disease commonly encountered in daily neurosurgical practice. Patients usually suffer acute symptoms with various manifestations including severe headache, conscious disturbance, or even deep coma. SAH is caused by a ruptured cerebral aneurysm in about 80% of cases. In the acute stage, the comparatively minor symptom of blurred vision is usually neglected by neurosurgeons because of the devastating nature of the disease. Here, we present a 47-year-old male with spontaneous SAH that was found to be caused by a ruptured anterior communicating artery aneurysm. After securing the ruptured aneurysm with an endovascular coiling procedure, the patient recovered well with return of consciousness; however, he complained of blurred vision. He received an ophthalmic examination and intraocular vitreous hemorrhage was found, which was consistent with the diagnosis of Terson's syndrome. Vitreous hemorrhage after SAH is a characteristic syndrome reported to occur in 10-40% of patients. However, clinicians have not paid much attention to this syndrome and it is usually misdiagnosed because a routine check is only conducted on comatose patients and clinical physicians overlook using a fundoscope to check patients with SAH. We describe our case to illustrate this syndrome and remind physicians of this diagnosis. Clinical features, diagnosis, and treatment are reviewed.

Key words: Terson's syndrome, subarachnoid hemorrhage, ruptured cerebral aneurysm, vitreous hemorrhage

INTRODUCTION

Vitreous hemorrhage was first reported by Albert Terson in 1900[1]. It was then grouped as a clinical entity with subarachnoid hemorrhage and was named after Albert Terson in 1926[2]. The definition of Terson's syndrome (TS) was later expanded to include retinal hemorrhage[3]. In neurosurgical practice, the syndrome is usually unrecognized because patients with TS are in a very critical period of their life. Spontaneous SAH usually implies that serious clinical hazards of a ruptured cerebral aneurysm have occurred. Patients with SAH subsequently undergo many life-threatening episodes, including rebleeding, acute hydrocephalus, angiography-related complications, neurosurgical or endovascular risks, and ischemic stroke due to vasospasms, all of which occur despite the securing of the aneurysm. As a result, minor ocular conditions, such as vitreous or retinal hemorrhage, which do not present the immediate risk of causing death, are usually neglected and misdiagnosed by neurosurgeons. It is often difficult to obtain symptoms or signs of disturbances in visual acuity for these conditions because of the impaired neurological status of the patient and because the main focus is on strategies to treat the ruptured cerebral aneurysm and raised intracranial pressure. Here, we present a patient with this syndrome to highlight the diagnosis of TS to physicians, especially neurosurgeons and ophthalmologists, to achieve early diagnosis and treatment of TS and reduce the morbidity of vision impairment.

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CASE REPORT

A 47-year-old businessman from Taiwan who had been working in Mainland China presented to the local hospital in Guangzhou City 2 months ago with sudden disturbance in consciousness; after shouting that he had an explosive headache, his condition progressively deteriorated into a semicoma. Before this episode, he had been in good health, except for occasional hypertension that was noted but neglected. His personal history revealed a habit of heavy smoking, consuming more than two packets of cigarettes per day. His initial score for the Glasgow Coma Scale (GCS) was 11 (E3M5V3). His pupils were isocoric and showed a sluggish response to light. Positive findings of

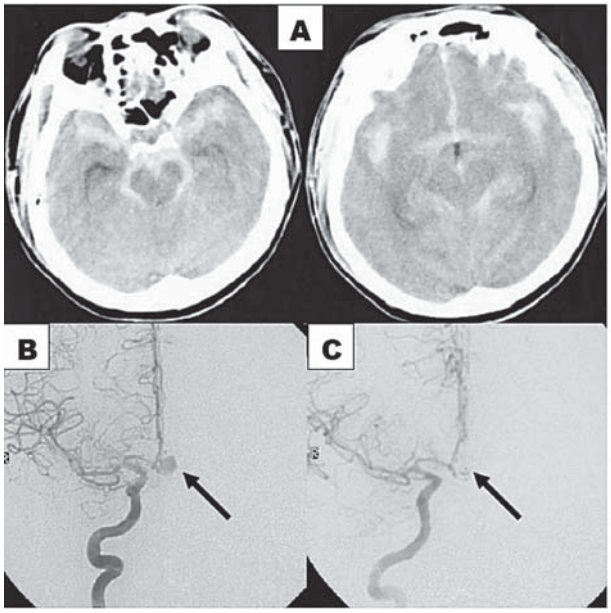

Fig. 1 (A) A nonenhanced CT scan of the brain revealed SAH and hydrocephalus. (B) A digital subtraction angiography of the right internal carotid artery showed a saccular aneurysm (arrow) arising from the anterior communicating artery, which measured 7.1×8.2 mm. (C) A postcoiling angiography showed disappearance of the aneurysm.

Brudzinski's sign and Kernig's sign were noted in his medical record. A nonenhanced computed tomography (CT) scan of the brain revealed subarachnoid hemorrhage and hydrocephalus (Fig. 1A). The patient was kept under close clinical observation for 2 weeks. Conventional digital subtraction angiography was later conducted and revealed a saccular aneurysm arising from the anterior communication artery (Fig. 1B). An endovascular approach with coil embolization was performed (Fig. 1C). The patient recovered consciousness and was sent back to Taiwan by the International SOS service. On admission, he was fully conscious and complained of headache and blurred vision. His vital signs were stable and his GCS score was 15 (E4M6V5). No remarkable neurological deficits or cranial nerve palsies were detected. We made an ophthalmic consultation to evaluate his visual condition. The visual acuity was 1/60 in the right eye and was reduced to counting fingers in the left eye. Fundoscopic examinations disclosed papilledemas and vitreous hemorrhages (Fig. 2A). TS was diagnosed and he received periodic follow-up thereafter to decide whether further surgical intervention would be necessary. Complete resolution of the vitreous hemorrhage was noted in a follow-up ophthalmoscopic evaluation 3 months later (Fig. 2B). The patient

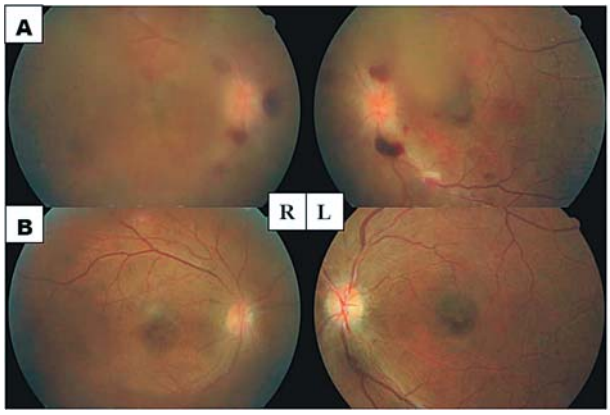

Fig. 2 Ophthalmoscopic photographs. (A) Initial images (27 days after subarachnoid hemorrhage) showed intraretinal, subhyaloidal, and vitreous hemorrhages in the right (R) and left (L) eyes. Papilledemas were also noted. Visual acuity was 1/60 in the right eye and reduced to counting fingers in the left eye. (B) Pictures of the fundus taken 3 months later. Note that the hemorrhages and papilledemas resolved spontaneously in both eyes. The visual acuity was 6/12 in the right eye and 6/15 in the left eye.

recovered well and his vision improved: visual acuity was 6/12 in the right eye and 6/15 in the left eye.

DISCUSSION

Spontaneous SAH is commonly encountered in daily neurosurgical practice. It mainly results from a ruptured cerebral aneurysm and can cause significant mortality and morbidity. In patients who survive a ruptured cerebral aneurysm, classic TS is a phenomenon known to occur in 17-27% of cases[4-6]. Several studies have reported that most patients with TS were in a state of comatose consciousness on initial presentation[4,7,8]. We speculate that this could be why TS is seldom diagnosed and usually neglected by most neurosurgeons in the acute setting. Most patients in this situation cannot talk and express their complaints. In patients with aneurysmal SAH and histories of transient or prolonged coma, ophthalmological screening is highly recommended to identify this syndrome[6].

The pathogenic mechanisms of TS remain somewhat controversial. Two theories have been proposed to explain the pathogenesis of vitreous hemorrhage after SAH. The first theory is that subarachnoid blood is pressed through the optic nerve sheath into the interior of the eyeball when a bleeding episode occurs[9]. The second theory, which has been mostly accepted, suggests that vitreous hemorrhage is the result of venous hypertension and rupture of retinal

veins secondary to abruptly raised intracranial pressure[3,4,10,11]. The second theory could be underscored by clinical findings of intraocular hemorrhage and significant papilledema occurring in the eye contralateral to the side on which the ruptured aneurysm occurs. The distribution of ruptured aneurysms and whether they occur on the right or left side have no correlation with vitreous hemorrhage[4]. Vitreous hemorrhage could still occur in patients in whom a ruptured aneurysm is located in the posterior circulation, such as the basilar artery. The hemorrhagic patterns revealed in fundoscopic examinations show that blood extends from the region of the optic disc into the retina, preretinal space, and vitreous portion, which also supports the second theory. Besides SAH, TS could also occur in patients with severe head injury and intracranial hemorrhage. Medele et al. found that 46% of patients with SAH and 44% of patients with severe head injury (GCS=3 to 10) were associated with the syndrome[8].

Radiological imaging plays a smaller role than fundoscopy in diagnosing TS and few studies address this issue. However, nonenhanced CT scans of the brains of patients with SAH have been published and reveal subtle findings[12]. Focusing on the orbital region of a CT scan of the head, retinal nodularity and crescentic hyperdensities were evident in the posterior eyeball in 62.5% (5 out of 8) of patients with SAH with TS. A positive result on a CT image warrants a detailed fundoscopic evaluation to disclose the vitreous or retinal hemorrhage. In our patient, no significant nodularity or hyperdensity was found in the orbital region of the brain CT scan. Only one case report, by Arakawa et al., addresses this syndrome with magnetic resonance imaging (MRI)[13]. This may be because fundoscopy is a simple, available, and low-cost tool with which to evaluate these critical patients. We advocate the use of routine fundoscopic examinations in patients with SAH as a fundamental way of disclosing this syndrome, by ophthalmologists and other clinical physicians.

The course of progression of vitreous hemorrhage and its management vary in each patient. Vitreous hemorrhage could happen again on any rebleeding event following initial SAH. The involvement of both eyes has been observed in 60% of patients with TS[5]. Sometimes the hemorrhage clears by itself, whereas some patients incur visual loss, chronic hematoma, or epiretinal membrane formation, which require ophthalmic surgery[14,15]. Rapid diagnosis of intraocular hemorrhage is important because a nonclearing vitreous hemorrhage may result in permanent visual loss[5,16]. Periodic ophthalmic follow-up is the key to detecting a number of consequences. Timely surgery with pars plana vitrectomy is necessary to obtain good recovery of vision in some patients. Furthermore, in patients undergoing neurosurgery, the diagnosis of TS usually implies a poor clinical course, frequently heralding further episodes of bleeding. In a prospective study by Pfausler et al.[4], the overall mortality of patients with SAH was 23% (14 of 60), whereas 90% of patients with TS expired.

In conclusion, TS is usually overlooked in the acute stage of patients with SAH. The patient cannot complain of the comparatively minor symptoms of blurred vision, intractable headache, or poor consciousness when unconscious and these cannot be detected by physicians. Therefore, TS rarely prompts an ophthalmic workup on the ocular fundus and underdiagnosis of this syndrome is common. Routine fundoscopic examination in patients with SAH is an important diagnostic tool for ophthalmologists and clinical physicians to use that leads to the diagnosis of TS. Without early discovery of TS, some patients might lose their vision. We report this case to highlight this syndrome as a differential diagnosis in these critical patients with SAH. Early diagnosis could save the vision of these patients and lessen morbidity.

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