Terson Syndrome after Subarachnoid Hemorrhage Occurred by Thrombolysis and Mechanical Thrombectomy to Treat Acute Ischemic Stroke — A Case Report —

Ha Young Byun, M.D., Hoyeon Jung, M.D., Hye Jung Choi, M.D., Joong Hoon Lee, M.D., Min-Kyun Oh, M.D., Chul Ho Yoon, M.D., Heesuk Shin, M.D. and Eun Shin Lee, M.D.

Department of Rehabilitation Medicine, Gyeongsang National University Graduate School of Medicine

Terson syndrome is a vitreous hemorrhage associated with subarachnoid hemorrhage. This can be caused by spontaneous, aneurysmal rupture or traumatic subarachnoid hemorrhage, but never has been reported as a consequence of hemorrhage due to thrombolysis and thrombectomy treatments of acute ischemic stroke patient. A 48-year-old man presented with left sided weakness was diagnosed as cerebral infarction on right middle cerebral artery territory due to complete occlusion of right distal internal carotid, middle cerebral, and anterior cerebral artery. He underwent thrombolysis and mechanical thrombectomy, and subarachnoid hemorrhage developed. Later, visual disturbance on right eye occurred so he was consulted to ophthalmology. Vitreous hemorrhage was found and surgery was recommended after two weeks of observation. After pars planar vitrectomy, visual acuity improved, along with functional ability. Therefore, possibilities of Terson syndrome in patients with subarachnoid hemorrhage have to be kept in mind to improve not only visual acuity but also rehabilitation outcome. (Brain & NeuroRehabilitation 2014; 7: 136-142)

Key Words: vitreous hemorrhage, stroke, subarachnoid hemorrhage

Introduction

Vitreous hemorrhage secondary to the subarachnoid hemorrhage is known as Terson syndrome, which called after the French ophthalmologist Albert Terson, who reported this association in 1990. Visual disturbance of the affected eye is the most common symptom of the Terson syndrome, and it is known to occur in 10~50% of patients with subarachnoid hemorrhage. Although visual disturbance is reversible, it can cause delayed physical and functional recovery.

Case Report

A 48-year-old male with known history of arrhythmia was admitted to emergency department due to suddenly developed left side weak-
ness, which started an hour ago. He was on warfarin medication for arrhythmia and did not have any other medical problems, such as diabetes mellitus, hypertension, or head trauma. On the neurological examination, his mental status was alert. Manual muscle test (MMT) revealed that his motor power was grade 1/5 on left upper and lower extremities. He also had mild dysarthria, left sided facial palsy and severe sensory decrease on his left side of body and extremities. Babinski sign was found on left side.

Magnetic resonance image (MRI) which was taken immediately after the admission indicated an acute infarction in right middle cerebral artery (MCA) territory (Fig. 1), and magnetic resonance angiography (MRA) showed complete occlusion of right distal internal carotid artery (ICA), right MCA and right anterior cerebral artery (ACA) (Fig. 2). He was decided to undergo intravenous (IV) thrombolysis with mechanical thrombectomy. The 7 mg of alteplase (Actilyse inj, Boehringer Ingelheim GmbH, Korea) was loaded and 63 mg of alteplase was infused during 60 minutes. A 6-Fr guiding catheter (Shuttle Select™, Cook Medical, Inc. Bloomington, IN, USA) and the Penumbra Reperfusion Catheter (Penumbra System™, Penumbra, Inc. Alameda, CA, USA) were used to mechanically push the thrombus with assistance of Prowler Select Plus microcatheter (Cordis Neurovascular, Miami, FL, USA) and Traxcess® microguidewire (MicroVention, Inc. Tustin, CA, USA). Partial recanalization of MCA was achieved 4 hours after onset, but the clot in the lower trunk of right MCA M2 division could not be dissolved. Brain computed tomography (CT) taken immediately after the mechanical thrombolysis showed SAH (Fig. 3), which was resolved on follow up CT taken 10 hours later (Fig. 4). His left sided motor weakness persisted, so he was transferred to department of rehabilitation three weeks after the admission.

Fig. 1. Axial images of brain magnetic resonance imaging obtained at the emergency department visit. Note the acute infarction in right basal ganglia and periventricular white matter.
Fig. 1. Axial images of brain computed tomography image taken immediately after the mechanical thrombolysis shows SAH with extravasated contrast media in right basal ganglia and periventricular white matter.

Fig. 2. MR angiography with Gadolinium demonstrates complete occlusion of right distal ICA, right MCA and right ACA.

Fig. 3. Axial images of brain computed tomography image taken immediately after the mechanical thrombolysis shows SAH with extravasated contrast media in right basal ganglia and periventricular white matter.

When transferred, he had mild cognitive impairment with a Korean version of the Mini-Mental Status Examination (K-MMSE) score of 23. MMT showed grade 1/5 on left upper and distal lower extremities, while proximal lower extremity improved to grade 2/5. He had no spasticity, and deep tendon reflex was exaggerated on left upper extremity. His superficial sensory responses were decreased on left side compared to the normal right side, and proprioception was impaired. Modified Barthel Index (MBI) score was 43. Functionally he could roll, but could neither sit nor stand up. The patient was dependent during bed mobilization and transfer. Occupational therapy evaluations revealed him requiring assistances in most activities of daily living.

Two weeks after transfer, he complained visual disturbance on right eye. He reported that visual disturbance started a few weeks ago, but could not remember the exact onset time. Although not clear, he recalled that the visual disturbance seemed to neither aggravate nor improve since the first time he recognized. The ophthalmology service was consulted. On the first examination, the best-corrected visual acuity (BCVA) was very poor on right eye and relatively normal on left eye (counting fingers at 1 meter in the right eye and 20/20 in the left eye). There was no ocular pain, discharge or injection, and intraocular pressure was in normal range for both eyes. Slit lamp examination showed clear conjunctiva, cornea and anterior chamber. Fundoscopic examination revealed vitreous hemorrhage on right eye.
Fig. 4. Ten hours later, hemorrhage resolved and extravasated contrast media disappeared.

(Fig. 5A), which was consistent with Terson syndrome. He was recommended to observe with regular consultations. His BCVA on right eye slightly improved but not to relatively normal range until four weeks later (to 20/200 and 20/50 at two weeks and four weeks later, respectively). At this moment, surgery was recommended since the BCVA in right eye did not improve afterward. However the patient refused. On seventh week of observation, patient decided to pursue pars planar vitrectomy. After vitrectomy, the patient took bed rest without any physical treatment for a week. His right eye BCVA improved a month after the surgery from poor to relatively normal range (20/50 to 20/20). Funduscopie examination also showed disappeared vitreous hemorrhage four weeks after the surgery (Fig. 5B).

He continued inpatient rehabilitation program and warfarinization for three months, and his MBI score improved to 52. At the end of
the inpatient rehabilitation program, he was able to ambulate with cane. Occupational therapy evaluations revealed that he could feed himself, dress up, and go to toilet without assistance.

Discussion

Acute ischemic stroke is one of the leading sources of morbidity and mortality, especially in the industrialized countries. Its outcome depends on the time since onset to revascularization, reperfusion and the presence of the hemorrhage. Although patients with successful recanalization after stroke increased the chance of independence 4.4 times more than the patients without recanalization, IA thrombolysis followed by thrombectomy had a risk of SAH in 18.6% of patients.

SAH is frequently associated after thrombectomy by vessel rupture or mechanical stretch during stent retrieval. Serial non-enhanced CT is useful to investigate subarachnoid hyperdense lesions that can be SAH or SAH with contrast extravasation. When a hyperdense lesion appears from post-therapeutic CT scans, it can be defined as SAH when maximum Hounsfield unit is less than 90, and as SAH with contrast extravasation when maximum Hounsfield is same or greater than 90. In this case, the CT image taken immediately after the thrombectomy seemed more likely to be the SAH, but there is still a possibility of extravasation of contrast.

During clinical course of the patient, there was uncertainty about the onset of the visual disturbance. The patient reported that the visual disturbance seemed to be started a few weeks before he complained. However, it is not clear whether the hemorrhage started when SAH developed after the thrombectomy treatment or other time of point, since neurologic exam including visual portion was not conducted at that time. SAH after thrombectomy treatment is often asymptomatic and clinically insignificant, so the physicians may be easy to miss complications without detailed neurologic examinations. Although the patient was taking warfarin, the risk of ocular hemorrhage in patients with warfarin medication is reported to be small when the patient has no preexisting ocular disease. So the vitreous hemorrhage in this patient has little possibility of being spontaneous hemorrhage caused by warfarin.

Terson syndrome, which usually defined as the vitreous hemorrhage occurring in association with subarachnoid hemorrhage, is hard to distinguish with other types of intraocular hemorrhages such as retinal and subhyaloid hemorrhages. Few mechanisms known about these hemorrhages may contribute to the hard distinction. There was one study suggesting that different types of intraocular hemorrhage have different prognosis. In their article, authors suggested that mild retinal hemorrhage has better prognosis than large preretinal or vitreous hemorrhages. Also, vitreous hemorrhage has been known to be a factor of poor prognosis in patients with subarachnoid hemorrhage in general.

Pathogenic mechanism of Terson syndrome is controversial. Most widely accepted model suggests that rapidly increased intracranial pressure is transmitted to the subarachnoid space of the optic nerves through effusion of cerebrospinal fluid or blood via the optic nerve sheath. This compresses the central retinal vein and choroidal anastomosis by high pressure. Venous obstruction and stasis occur as a consequence, and result in distension and rupture of retinal capillaries and veins.

The mechanism supports the idea that Terson syndrome is related to poor prognosis in patients with SAH. Patients suffering from SAH are more likely to have vitreous hemorrhage, thus Terson syndrome, when they have low Glasgow coma score, high Fisher grade, or high Hunt and Hess grade. These patients may tend to have higher intracranial pressure. Higher intracranial pressure of patients may results in large amount of hemorrhage. When the hemorrhage is cleared, blood products are usually cleared by macrophages. Large hemorrhages of patients with high intracranial pressure may require more time for clearing, which result in more toxic effect of iron related to the damage of retina.

Most of decreased visual acuity from Terson syndrome gradually improves since the hemorrhage spontaneously resolves in months to years. Therefore, conservative treatment has been considered as the treatment of choice. Nonetheless, surgical intervention is required in some cases, and delayed surgery in them can lead to complications such as retinal detachment, proliferative retinopathy, epiretinal membrane formation and cataracts. Also, factors such as age, occupation, visual needs, psychological and neurologic status with family background need to be considered when treatment modality is decided.

Visual ability is one of the important factors of successful rehabilitation. If visual impairment is suspected or complained, it can hinder the physical therapy or rehabilitation. Thus physicians should consider the possibilities of Terson syndrome in patients with SAH. Early recognition of this condition may allow time to determinate appropriate treatment principle, and delayed diagnosis can result
in ocular complication with delayed physical and functional recovery. Therefore, timely consultation to ophthalmology is mandatory, especially if the patient with SAH has been comatose or had low Glasgow coma score, high Fisher grade, or high Hunt and Hess grade at admission. In addition, rehabilitation program should include the strategy to compensate the visual disturbance for active participation, rapid recovery and safety. Early diagnosis of Terson syndrome may help to allow integrating these elements in planning of rehabilitative measures.

In summary, we experienced a case of SAH occurred after thrombolysis and mechanical thrombectomy, who complained visual disturbance caused by vitreous hemorrhage, which is the Terson syndrome. This is the first case reporting the occurrence of visual disturbance caused by Terson syndrome, after thrombectomy and IA thrombolysis treatment of acute ischemic stroke. The visual disturbance was reversible when acute diagnosis and proper treatment was conducted. Intraocular hemorrhage caused by thrombolysis has been previously reported after treatment of acute myocardial infarction and ophthalmological problems. Also, there was one case reporting permanent visual loss as a consequence of thrombolysis in patient with acute ischemic stroke. However, this case is different from previous reports. Firstly, the patient in this case showed reversible visual disturbance after vitrectomy, compared to previous permanent visual loss case. This stresses the importance of accurate diagnosis and treatment, and different nature of the intraocular hemorrhage of two cases. Also the patient from previous case had underlying ophthalmologic problems, while this patient had no previous underlying disease on eye. Visual disturbance by Terson syndrome is reversible but can cause complications if not treated properly. Therefore, physiatrists should be aware of its clinical presentation and possibilities in patients who develop decreased visual acuity after SAH. Also, rehabilitation plan need to coordinate strategies that can compensate the visual loss for effective and rapid physical and functional improvements.

References


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