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Traumatic Symptomatic Vasospasm after Mild Head Injury

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Post-traumatic vasospasm is a well-recognized complication of head injury. However, symptomatic vasospasm rarely occurs in patients with mild head injuries. A 58-year-old female patient presented with head injury and a traumatic subarachnoid hematoma along the left lateral fissure, which subsequently induced symptomatic vasospasm. Magnetic resonance imaging of her brain showed acute infarction of the left middle cerebral artery. Angiography demonstrated multiple segmental narrowing of the left middle cerebral artery near the hematoma spatially in accordance with PTSV. In conclusion, early recognition of PTSV is crucial to improving neurological outcomes.

Key words: post-traumatic vasospasm, subarachnoid hematoma, head injury, nimodipine, Glasgow Coma Scale

INTRODUCTION

Post-traumatic vasospasm (PTV) is a well-recognized complication of head injury. The estimated incidence of PTV is around 10% to more than 50% in patients with head injuries.[1] In severe head injuries, PTV often occurs between 12 hours and 5 days after the injury. The reported duration of PTV is around 12 hours to 30 days.[2-4] Symptomatic vasospasm was defined as the time course of neurological deterioration parallel to that of radiogenic vasospasm. Taneda et al. presented that the incidence of symptomatic vasospasm was 7.7% of head-injured patients with subarachnoid hemorrhage detected on computed tomography (CT), and it developed between Day 4 and 16 after the head injury.[5] Since post-traumatic symptomatic vasospasm (PTSV) is an important factor contributing to severe neurological deficits and poor prognosis,[6-8] neurologists should be aware of and recognize symptomatic vasospasm early following a head injury. Angiography is the gold standard for the diagnosis of PTV. Herein, we report the case of a minor head injury which progressed to PTSV without a drop in Glasgow Coma Scale (GCS).

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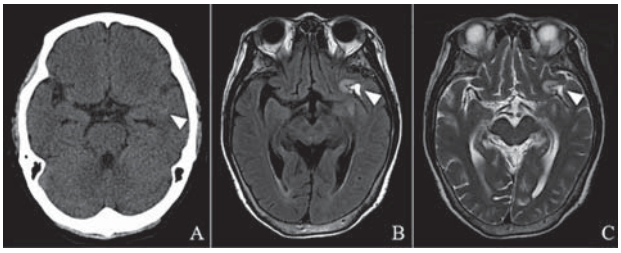

Fig. 1 (A) The white arrowhead indicates the acute subarachnoid hematoma along the left Sylvian fissure on CT of brain. (B and C) Both T1-weighted and T2-weighted MR images show high signal intensity (white arrowheads), suggestive of a subacute hematoma along the left Sylvian fissure.

CASE REPORT

A 58-year-old female suffered from headache and nausea without significant neurological deficits (GCS: E3M6V5) on arrival at our emergency room after a motorcycle accident. However, she complained of a persistent headache and dizziness on the third admission day. This prompted us to perform a CT scan of her brain, which showed a minimal subarachnoid hematoma along with a left lateral fissure (Fig. 1A). Abrupt left-side hemiplegia, dysarthria, and a central type of right-side facial palsy occurred about 10 days later. Magnetic resonance imaging of her brain (Fig. 1B and 1C) revealed a small subacute subarachnoid hematoma accumulated around the left bifurcation of the middle cerebral artery (MCA) along with the left Sylvian fissure. Moreover, there was an acute infarction involving her left centrum semiovale, corona radiata, insular ribbon and peri-fissural

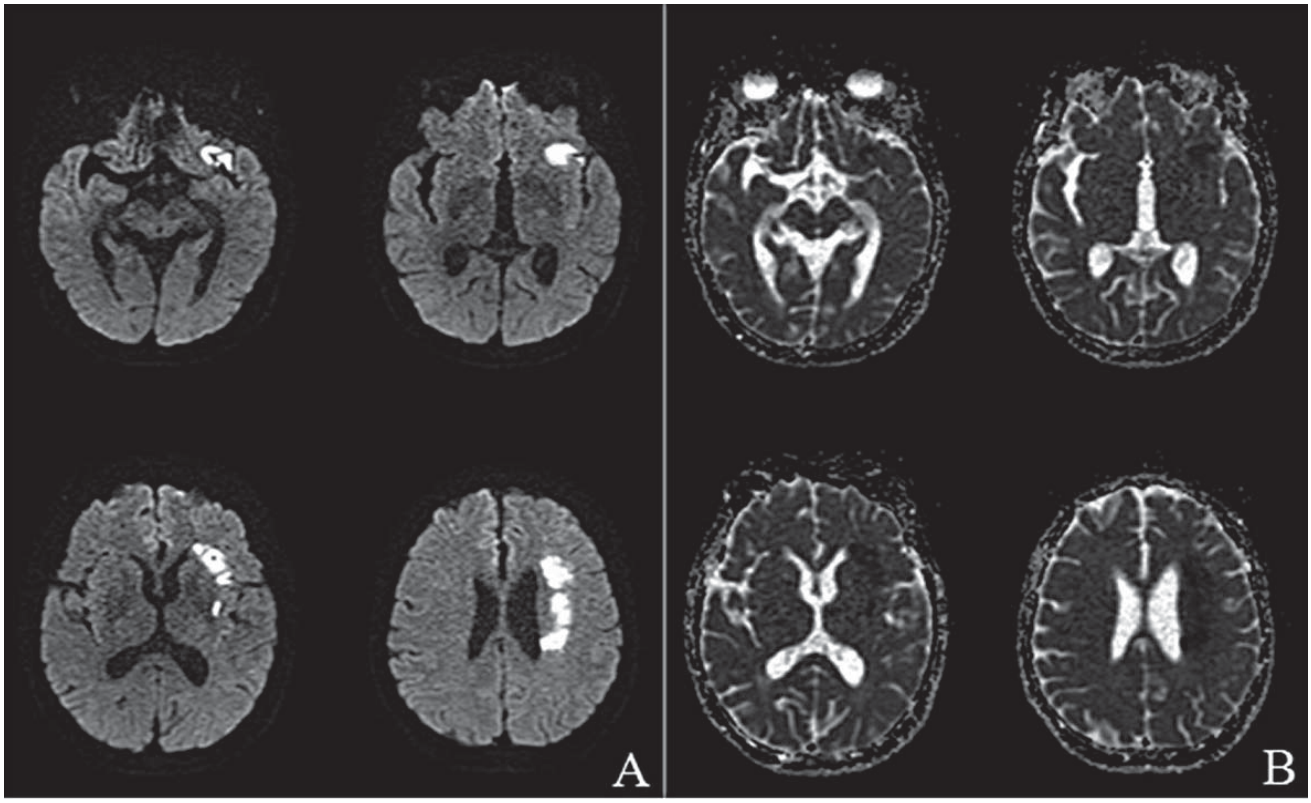

Fig. 2 Diffusion-weighted magnetic resonance images (A) show hyperintensity over the left centrum semiovale, corona radiata, insular ribbon, and peri-fissural frontal lobe, consistent with the area of hypointensity in magnetic resonance apparent diffusion coefficient maps (B). The regions mentioned above indicate acute infarction lesion.

frontal lobe (Fig. 2). Angiography of the left internal carotid artery (ICA) showed multiple segmental narrowing of the left MCA and anterior communicating artery close to the subarachnoid hematoma spatially, consistent with trauma-related vasospasm (Fig. 3A). She underwent nimodipine treatment (60 mg PO q4h) for 2 weeks, and follow-up cerebral angiography demonstrated a normal caliber without obvious stenosis of the MCA (Fig. 3B).

DISCUSSION

From a traditional viewpoint of neurocritical care, a decline in the GCS of two points or more, or a drop of one point lasting for more than 30 minutes should prompt re-evaluation of CT scan. An important finding in our case is that a minor head injury without initial neurological deficits progressed to PTSV.

Vasospasm is a potentially severe complication of head injury. The incidence of post-traumatic vasospasm reported in angiographic studies ranges from 10% to 39%. However, the diagnostic rate of vasospasm varies according to the diagnostic method used, including ultrasonography, magnetic resonance imaging, and angiography. Moreover, a much higher incidence of vasospasm exceeding 50% has been found with transcranial Doppler (TCD) ultrasonography.[1] TCD ultrasonography achieves a higher diagnostic rate because it allows for continuous assessment or repeated monitoring. The duration and time course of PTV vary among reports.

Zubkov *et al*. showed that GCS score on admission was inversely related to the development of PTV, but not in cases with a GCS score of more than 12.[3,9,10] Patients with epidural hematomas, subdural hematomas, and subarachnoid hematomas have an increased incidence of post-traumatic vasospasm. An increased volume of hematomas has also been reported to be related to PTSV.[11-14] Cerebrospinal fluid in the blood, breakdown products of blood, including oxyhemoglobin, and the coagultion-fibrinolysis system increased after head injury with subarachnoid hemorrhage and would induce either calcium-dependent and independent arterial smooth muscle contraction, as well as the metabolites of arachidonic

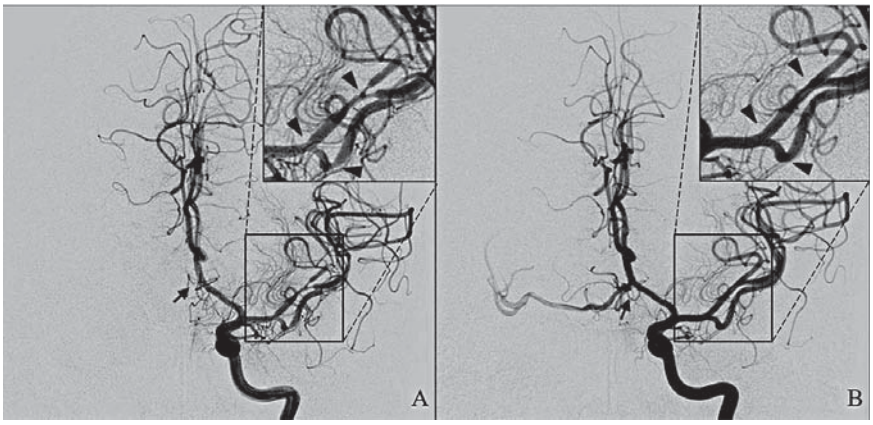

Fig. 3 (A) Anteroposterior view of left internal carotid artery angiography demonstrated multiple short-segmental narrowing from the left post-bifurcation to the insular segment of the middle cerebral artery (black arrowheads) and anterior communicating artery without the appearance of right middle cerebral artery (black arrows). (B) After two weeks of nimodipine treatment, the tapering from the left post-bifurcation to the insular segment of the middle cerebral artery was not seen (black arrowheads). Additionally, the appearance of the right middle cerebral artery was shown (black arrows).

acid, inflammatory processes and endothelium-derived substances, such as nitric oxide and endothelin.[15-19] However, the extent to which each mechanism is involved in the development of cerebral vasospasm remains unclear. Moreover, the molecular mechanism has been recognized as the alterations of calponin, a contractile protein, contributing to the migration and phosphorylation in smooth muscle membrane of vessels to enhance vasospasm. Therefore, calponin induced hypoperfusion after traumatic brain injury.[20] Although all their roles still remain to be established, pharmacological treatments of angiographic or clinical vasospasm using nimodipine, statins and endothelin receptor antagonists were proved with level-1 evidence available for the treatment of cerebral vasospasm.[21] Nimodipine, a calcium channel blocker, is more lipid soluble than other calcium channel blockers in penetrating the blood-brain barrier to provide vasodilatory effect.[22,23] Statins, HMG-CoA reductase inhibitors, have been reported to increase production of endothelial nitric oxide synthase (eNOS) which generates NO in smooth muscle cells of cerebral vasculature to attenuate cerebral vasospasm.[24] Endothelin receptor antagonists act on endothelial and smooth muscle cells of the cerebral arterioles to induce arterial relaxtion.[25,26]

Nimodipine is a dihydropyridine-derived calcium antagonist, which has been shown to be effective in preventing ischemic complications after aneurismal subarachnoid hemorrhage by dilatation of cerebral arterioles.[22,23] However, there is still much debate whether patients with traumatic subarachnoid hemorrhage should also be treated with this drug. Vergouwen et al. reviewed the association between nimodipine and head injury and found no significant difference in occurrence of poor outcome and mortality rates between patients treated with nimodipine and those treated with placebo.[27] However, some evidence suggested potential benefit in nimodipine-treated patients with traumatic head injury.[28-30]

Reversible cerebral vasoconstriction syndrome (RCVS) is a rare entity of cerebral vasospasm, which occurs spontaneously without a definite cause.[31] Patients with RCVS often present repeatedly with vasospasms on the arteries around the circle of Willis, followed by acute thunderclap headaches and transient or fluctuating neurological deficiencies.[32,33] There are parallels between the duration of cerebral vasoconstriction and thunderclap headache. Although the primary pathology remains unknown, it is thought to be related to vasoactive substances, hypertension, endocrine abnormality, and neurosurgical trauma.[33] An angiographic study is the gold-standard diagnostic tool for RCVS to demonstrate cerebral vasoconstrictions and their reversibility. Additionally, it is also essential to differentiate diagnosis of RCVS from vasospasm with other entities. Minimal cortical subarachnoid hemorrhage occurring among 25% of these patients is suspected to be related to underlying endothelial dysfunction of cerebral vasculature, but it presents with diffuse short-segmental vasoconstriction involving large- and medium-sized cerebral arteries predominantly. However, the vasospasm in aneurismal or traumatic SAH is usually long-segmental and close to the bleeding site spatially.[34] Nimodipine has been employed to treat RCVS and shown to be effective in relieving headaches.[35]

CONCLUSION

Most patients with mild head injury suffer dizziness and headache. However, CT of brain for these patients is not recommended, especially when they show no neurological abnormality. Clinicians should consider PTSV as a potential complication of head injury. Early recognition

of PTSV can significantly attenuate its severity, reduce the occurrence of neurological deficits, and improve functional neurological outcomes.

DISCLOSURE

All authors declare that this study has no conflict of interest.

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