Pearls & Oy-sters:

Delayed traumatic intracerebral hemorrhage caused by cerebral venous sinus thrombosis

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PEARLS

1. Cerebral venous sinus thrombosis is a possible etiology of delayed traumatic intracerebral hemorrhage.
2. Skull base fracture can result in cerebral venous sinus thrombosis. Careful examination of bony defects in any patient with head injury is critical.

OY-STERS

1. Intracerebral hemorrhage can follow an asymptomatic interval after a trauma and may be due to local or systemic coagulation abnormalities, necrosis of blood vessels in areas of brain injury, transient cerebral dysautoregulation, reduction of intracranial pressure either medically or surgically, or cerebral venous sinus thrombosis.
2. Cerebral venous sinus thrombosis may be easily overlooked by routine noncontrast head CT. Further vascular imaging should be considered in the context of unusual intracerebral hemorrhage, such as delayed hemorrhage in patients without predisposing factors.

CASE REPORT A 14-year-old boy who was sitting on the backseat of a motorcycle without wearing a helmet was injured in a crash. His head hit the ground, resulting in a transient loss of consciousness. He was brought to a local hospital and no neurologic deficit was noted initially. However, he had frequent vomiting on the third day after the crash, and he became increasingly confused. Head CT at that time showed neither hemorrhage nor obvious skull fracture, but a focal hyperdensity with few tiny air bubbles close to the left temporal bone (figure, A and B). The next day, the patient was transferred to a tertiary medical center. On examination, he was drowsy, had impaired verbal fluency, and had mild right limbs weakness. Head CT with contrast showed an acute intracerebral hemorrhage (ICH) in the left frontal subcortical area (figure, C) and tapering occlusion of the left sigmoid sinus, which was close to a temporal bone fracture with a small amount of epidural air present and

diastasis of the left lambdoid suture (figure, D). Digital subtraction angiography performed 1 week after the crash confirmed occlusion of the left sigmoid sinus (figure, E and F). We prescribed intermittent IV bolus of mannitol 100 mL (20%, 0.4 g/kg) every 8 hours the fourth day after the crash and then increased the frequency to every 6 hours due to more drowsiness the next day. The dose was kept for 4 days and was tapered off thereafter. The patient improved gradually with conservative management. A neuropsychological examination 6 months later revealed mild impairment of attention, memory, and language functions.

DISCUSSION Delayed traumatic ICH was first described by Bollinger¹ in 1891, who proposed diagnostic criteria including a definite traumatic history, an asymptomatic period after trauma, a delayed event of apoplexy, and absence of previous vascular disease. A variety of possible pathogeneses of delayed traumatic ICH have been proposed and include local or systemic coagulation abnormalities, necrosis of blood vessels in areas of brain injury, transient cerebral dysautoregulation, and reduction of the intracranial pressure either medically or surgically.^{2,3} Our patient presented with a clear symptom-free interval and the initial head CT did not show any type of hemorrhage. He did not have coagulation disorders, use of an antithrombotic agent, or previous vascular abnormalities. Therefore, his condition should be compatible with delayed traumatic ICH.¹ Importantly, the finding of focal hyperdensity with air bubbles inside on the initial CT hints at the existence of cerebral venous sinus thrombosis (CVST) and adjacent temporal bone fracture.

Although a causal relationship between the occurrence of delayed ICH and traumatic CVST is not certain, the impairment of venous return originally through the superior sagittal sinus and vein of Labbé may theoretically increase regional intracranial pressure and contribute to delayed ICH. One study showed that the superior sagittal sinus is the most commonly involved head injury-related CVST.⁴

Head CT without contrast on the third day after the crash did not reveal any hemorrhage (A), but a focal hyperdensity with 2 tiny air bubbles close to the left temporal bone (arrow) (B). Head CT with contrast on the fourth day after the crash shows delayed hemorrhage at the left frontal subcortical area (C) and tapering occlusion of the left sigmoid sinus (arrowhead), close to a temporal bone fracture with small epidural air (arrow) and diastasis of the left lambdoid suture (D). Digital subtraction angiography 1 week after the crash shows occlusion of the left sigmoid sinus (arrows) (E, F).

Other cerebral venous sinus, such as sigmoid sinus in our proposed case, is less common, although the association between basilar skull fracture caused by head trauma and transverse/sigmoid venous thrombosis has been mentioned.^{4,5}

The finding of CVST in our case could have been overlooked if no further neurovascular imaging had been performed.⁶ The concurrent existence of delayed ICH and traumatic CVST is uncommon in head injury patients; another possible explanation for the coexistence of these 2 conditions being rarely reported is the difficulty in identifying CVST on routine noncontrast head CT.⁶ Vascular imaging such as CT angiography/venography or magnetic resonance angiography/venography should be considered for a head injury patient with unusual presentations such as delayed ICH.^{7,8}

So far, there is no consensus on the treatment of delayed traumatic ICH and CVST. Because the use of anticoagulant in a traumatic patient may increase risk for bleeding, some suggest that the management of patients with traumatic CVST can start with hyperosmolar therapies for intracranial pressure control. There is a paucity of literature regarding choice, dose, concentration, duration, and monitoring parameters of hyperosmolar therapy. Selection of hyperosmolar therapy should be individualized to the patient's condition. Generally, mannitol is dosed 0.25–1.0 g/kg,

with low dose in renal impairment. Commercially available hypertonic saline ranges from 3% to 23.4%. Bolus dosing with 250 mL of 3%, 2 mL/kg of 7.5%, or 30 mL of 23.4% hypertonic saline has been advocated.⁹ If further deterioration of neurologic signs happens despite adequate control of increased intracranial pressure, systemic anticoagulation, surgical decompression, and endovascular thrombolysis may be considered.^{4,7,10}

AUTHOR CONTRIBUTIONS

Dr. Hsu was responsible for the concept and drafting of the manuscript. Dr. Lee was responsible for analysis of the radiologic data and revision of the manuscript. Dr. Tang was responsible for revision and final approval of the manuscript. Jiann-Shing Jeng was responsible for critical revision of the manuscript.

STUDY FUNDING

No targeted funding reported.

DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

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Neurology 2014;83:e135-e137

DOI 10.1212/WNL.0000000000000836

This information is current as of September 29, 2014

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