Cerebral infarction following acute subdural hematoma

The occurrence of hypodensity lesions on computed tomography (CT) following acute subdural hematoma (ASDH) in infants and young children, termed cerebral infarction following ASDH (CIASDH) has been well recognized, which was also known as “big black brain” and “diffuse brain swelling”.

**Etiology**

In CIASDH, cerebral infarction occurs from the day of the ASDH onset to several days after the onset, which is mainly caused by child abuse or accidental fall. In many cases, subdural hematomas are small-to-moderate size, which do not show a severe mass effect. The pattern of hypodensity lesions in the cerebral hemispheres on CT of CIASDH is distinctive to spare the deep gray structures and hindbrain.

**Pathophysiology**

Although both histopathological examination and magnetic resonance (MR) image/diffusion weighted image (DWI) studies showed that the hypodensity lesion was cerebral infarction, the pathophysiology of this cerebral infarction and its relationship to the ASDH are poorly understood.

Insights from both clinical observation and experimental studies have helped to clarify the probable causes of this injury pattern, which appears to require a combination of stressors during a particular period of maturation.

**Diagnosis**

The study of Ichord et al. is the first to characterize hypoxic ischemic injury (HII) using Diffusion weighted magnetic resonance imaging (DWI-MRI) in young children, comparing inflicted trauma (IT) to accidental trauma (AT). The higher rate of HII on DWI-MRI in IT than in AT is likely multifactorial, involving respiratory insufficiency, seizures, and intracranial mass-occupying lesions requiring neurosurgical intervention. HII predicted need for in-patient rehabilitation in a large majority of children.

**Case series**

Momose et al. retrospectively examined consecutive children 6 years of age or younger, who were diagnosed with acute subdural hematoma (ASDH) and were admitted to the hospital between 2000 and 2014. In 57 consecutive children with ASDH, 12 (21.1%) developed CIASDH. The multivariate analysis revealed five predictors for CIASDH:

- Presence of seizure
- Consciousness disturbance at admission
- Absence of skull fracture
Hematoma thickness ≥ 5 mm on computed tomography (CT)
Midline shift ≥ 3 mm on CT (p < 0.05).

In three of six patients (50%) undergoing magnetic resonance (MR) imaging/fluid-attenuated inversion recovery (FLAIR) within 5 days of admission, serpentine hyperintensities in the subarachnoid space (FLAIR vessel hyperintensities) were demonstrated. MR angiography showed neither occlusion nor stenosis of the cerebral arteries. Single photon emission CT performed at admission in one patient showed a cerebral blood flow reduction in the ASDH side. All the children with CIASDH showed unfavorable outcomes at discharge. Children showing multiple predictors at admission should be carefully observed for development of CIASDH. Evaluation of the imaging studies suggested that a blood flow disturbance in the level of peripheral arteries to microcirculation was one candidate for possible mechanisms to induce the CIASDH.

Case reports

Emergency medical service (EMS) brought a 4-year-old girl involved in a car accident to the emergency room. She had had seizure controlled by diazepam. She was unconscious and her Glasgow coma scale (GCS) score was eight. Early vital signs were stable. Her first brain CT scan showed a subdural hematoma (SDH). One day after admission to ICU, her GCS decreased to five; hence, a control brain CT was performed. The brain CT scan showed a brain infarction. Six days after admission, her status worsened and her GCS dropped to three and her pupils became dilated bilaterally and unresponsive to light; she was pronounced dead.

It was an uncommon case of posttraumatic brain infarction and synchronous SDH.
