

Images in Clinical Medicine

Delayed spasticity in four limbs and ataxia after chronic subdural hematoma surgery

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Delayed bilateral internal capsule lesions after chronic subdural hematoma (CSDH) in a patient presenting with progressive quadriplegia and spasticity is extremely rare.

A 40-year-old woman had been healthy until 2 years previously when she had a minor head injury after a traffic accident. Initially, she had a mild headache. Three months after the event, she fell and magnetic resonance imaging (MRI) showed a left frontotemporo-parietal (FTP) CSDH (Fig. 1). Burr-hole hematoma drainage was performed. She recovered without any neurologic deficit, but progressive spasticity in the four limbs, weakness, and ataxia occurred 1 year, 9 months after the first head injury.

Physical examination revealed that the patient had noticeable unsteadiness in gait. There was no family history of any similar neurologic disorder. The neurologic examination did not reveal any cranial nerve deficits. Limb muscle strength was Grade 4 for the right limbs but only Grade 3 for the left limbs. The deep tendon reflexes were 4+ throughout, including the biceps, triceps, brachioradialis, and patellar reflexes. Spasticity in all four limbs was Grade 2 using the modified Ashworth scale. We administered baclofen and performed imaging studies.

MRI of the whole spine showed no abnormalities. MRI of the brain after 20 months revealed symmetric hyperintensities within the corticospinal tracts bilaterally on T2-weighted images, which were not evident from the previous MRI study. Focal hyperintensities were also delineated within the internal capsules

bilaterally from the level of the basal ganglia to the cerebral peduncles (Fig. 2). A minimal residual subdural hematoma was also noted in the bifrontal areas without an apparent mass effect. Magnetic resonance angiography showed patency of the great vessels without apparent abnormal focal parenchymal enhancement. Given the lack of underlying medical disease and the history of posttraumatic CSDH treated with burr-hole evacuation, we postulated that the bilateral internal capsule lesions resulted from a midline shift of the CSDH that subsequently led to the clinical presentation of limb spasticity. The internal capsule is supplied by the lenticulostriate branches of the middle cerebral artery. Bilateral internal capsule lesions have been associated with postanoxia or hypoglycemic encephalopathy [1,2]. In terms of head injury, previous reports of delayed pyramidal or extrapyramidal tract disorders were almost always unilateral in the pediatric population with vulnerable small end arterial vessels susceptible to a shearing force or mechanical vasospasm [3,4]. A CSDH leading to a brain parenchyma midline shift with herniation could also account for, to some extent, compromise of ipsilateral small vessels and could result in a delayed ipsilateral capsular lesion [5]. Although wallerian degeneration might contribute to delayed manifestation of traumatic white matter injury, most patients experience diffuse axonal injury during the initial major head injury [6]. To our knowledge, this is the first patient with delayed, bilateral capsular lesions after a minor head injury and CSDH surgery.

Progressive bilateral pyramidal tracts symptoms might develop up to 2 years after a minor head injury or with a midline shift from CSDH. Follow-up brain MRI is necessary for delayed posttraumatic neurological deterioration and may illustrate these delayed and irreversible internal capsule lesions.

Conflicts of interest: none.

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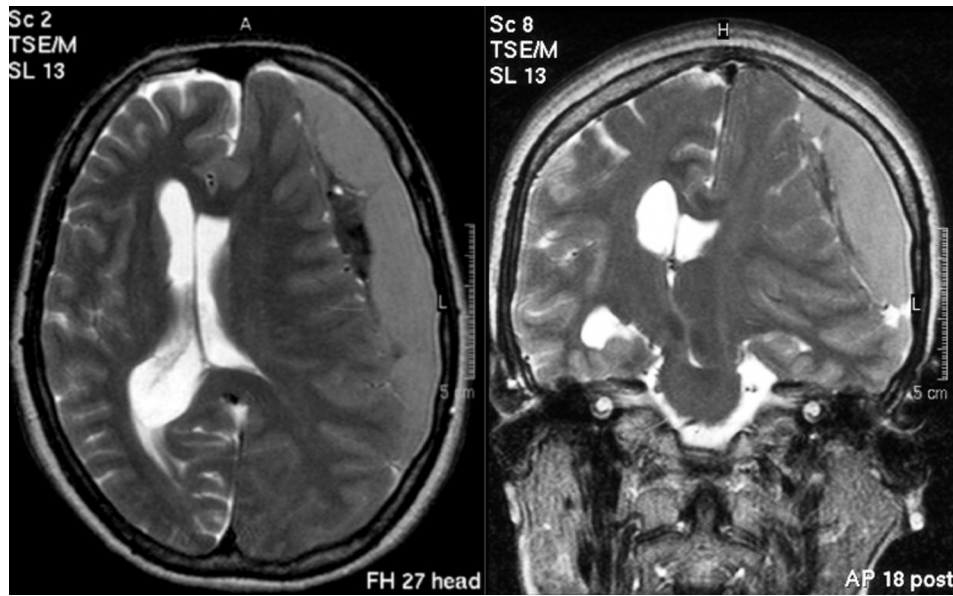


Fig. 1. Brain magnetic resonance imaging performed 3 months after the initial head injury. A T2-weighted image section shows a moderate left frontotemporoparietal chronic subdural hematoma with mass effect.

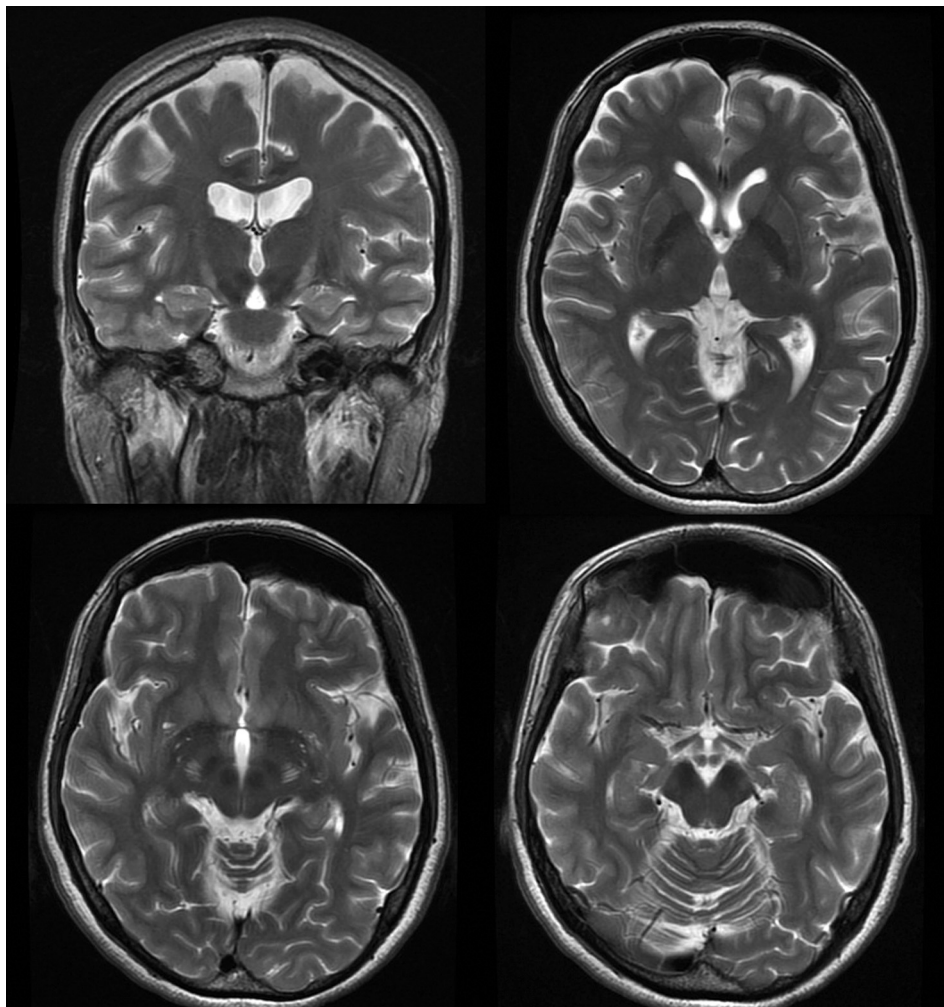


Fig. 2. Brain magnetic resonance imaging (MRI) performed 20 months after the first MRI examination. T2-weighted image coronal and serial axial sections show developed contiguous hypersignal intensity lesions from the level of the basal ganglia to the cerebral peduncles.

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