

Posttraumatic infarction in the territory supplied by the lateral lenticulostriate artery after minor head injury

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Abstract

Background Occlusion of the intracranial arteries due to blunt head traumas has been less frequently observed in patients with minor head injuries.

Case report A 4-year-old boy presented with speech disturbance 2 h after minor head injury. An initial computed tomography (CT) scan showed a questionable finding of a focal punctate high density in the left basal ganglia. Hemiparesis developed on the right limbs 8 h post-injury, and a subsequent CT scan revealed a discrete low-density change around the focal high density. Diffusion-weighted images revealed a clearly demarcated high-signal intensity lesion in similar area on T2-weighted and fluid-attenuated inversion recovery sequences images, compatible with infarcted tissues on the territory supplied by the lateral lenticulostriate artery. His hemiparesis improved gradually, and by post-trauma day 10 he was able to walk briefly without assistance. He was discharged on foot at post-trauma day 14.

Discussion and conclusion Children with minor head trauma who have normal findings on initial CT scan may rarely have basal ganglionic infarction resulting from arterial spasm or thromboembolism of the perforating

arteries. Hospital admission and careful observation should be considered for patients with minor head injury and persistent neurologic deficits despite normal CT findings. Magnetic resonance study is valuable for the evaluation of posttraumatic infarction, differentiating from hemorrhagic diffuse axonal injuries.

Keywords Minor head injury · Stroke · Basal ganglia · Lenticulostriate artery

Introduction

Stroke after minor head trauma, especially in the basal ganglia, is a rare event. They are usually hemorrhagic lesions, either small or large, and are usually a consequence of shearing forces in moderate to severe head injury [1]. Occlusion of the main intracranial arteries due to blunt head traumas has also been less frequently observed in patients with minor head injuries, often without loss of consciousness [2–7]. Spasms, emboli from the proximal major artery, dissecting aneurysm, and thrombus formation have all been suggested as causes [2, 7, 8]. But our knowledge about posttraumatic cerebral infarction due to occlusion of the perforating artery is very limited. We recently encountered a pediatric patient presenting with unilateral basal ganglionic infarction confined to the territory of the lateral lenticulostriate artery after a minor head injury. The clinical and radiological features are delineated and the literature is reviewed.

Case report

A 4-year-old boy fell to the ground while playing seesaw and incurred bruising to the head. Although the boy's sensorium was clear immediately after the impact, speech

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disturbance was noted approximately 2 h later. On admission, he could open his eyes, but his speech was indistinct and he could not answer questions. There were no focal neurological signs including that of motor power of the extremities and cranial nerve functions.

Plain skull X-ray results showed no fracture line. Computed tomography (CT) scans 2 h after injury revealed a focal punctate high density in the left basal ganglia, a not clearly delineated and questionable finding (Fig. 1a). The patient was admitted to the hospital for close observation. Hemiparesis developed on the right limbs (upper, grade II; lower, grade IV), so repeat CT scans were performed 8 h after injury. Compared with the previous CT scans, discrete low-density change is noted around the focal high density (Fig. 1b). Magnetic resonance (MR) studies were performed as a matter of differentiation tool of hemorrhagic or ischemic lesion. Fluid-attenuated inversion recovery (FLAIR) sequences images demonstrated a focal high-signal intensity area in the central portion of the left basal ganglia and overlying centrum semiovale (Fig. 2a). T2-weighted gradient echo (GRE) sequences images showed no definite hypointense lesion corresponding with high-signal lesion on T2-weighted image (Fig. 2b). Diffusion-weighted images (DWI) revealed a clearly demarcated high-signal intensity lesion in similar area on T2-weighted and FLAIR sequences images, compatible with infarcted tissues on the territory supplied by the lateral lenticulostriate artery (Fig. 2c). MR angiography showed no gross abnormalities including that of the left middle cerebral artery and internal carotid artery (Fig. 3). To exclude the possibility of a genetic predisposition to thrombosis, platelet count with peripheral blood smear examination, erythrocyte sedimentation rate, liver function test, lipid profile, and plasma homocysteine levels were evaluated, all of which were normal. To exclude the embolic cause of the infarct, echocardiography and carotid Doppler examination were also performed, all of which were normal.

The patient was treated conservatively with osmotic diuretic agent and dextran. His hemiparesis improved

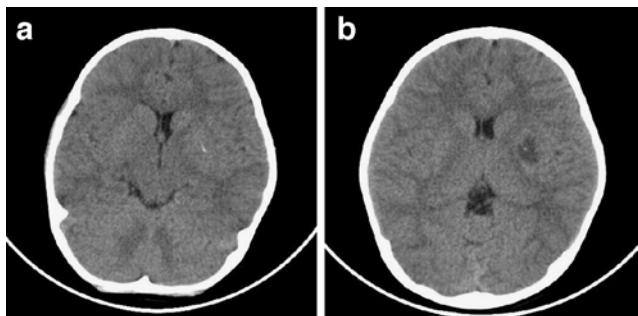


Fig. 1 **a** An initial CT scan 2 h after injury reveals a focal punctate high density in the left basal ganglia, not clearly delineated and questionable finding. **b** In repeat CT scan 8 h after injury, discrete low-density change is noted around the focal high density

gradually, and by post-trauma day 10 he was able to walk briefly without assistance and to grasp a colored pencil. Follow-up CT scans showed low-density lesion with minimal mass effect. He was discharged on foot by post-trauma day 14. A follow-up examination 2 months after discharge documented complete resolution of the neurologic deficits. One year after discharge, his neurological status remains stable.

Discussion

Posttraumatic cerebral infarction is a relatively rare complication of head trauma, but its frequency, cause, and influence on mortality are not well defined [5]. Mirvis et al. [5] reported that posttraumatic infarction was demonstrated in 25 (1.9%) of 1,332 patients who required brain CT for trauma and posterior cerebral artery and that the anterior and middle cerebral arteries were the most frequently involved vessels. But our knowledge about posttraumatic infarction due to occlusion of the perforating arteries is very limited. The lenticulostriate, thalamoperforating, or choroidal arteries have been found to be occluded against the skull base, resulting in infarction on basal ganglia after head traumas [5, 9–11]. Vascular lesions in the basal ganglia may be disclosed in patients with shearing injury due to angular acceleration forces as in our case. In most major arteries occlusion, spasm, emboli from the proximal major arteries, dissection, and thrombus formation have all been accused for occurrence of infarction [2, 7, 8]. The compression and distortion of the brain secondary to brain herniation and increased intracranial pressure may also induce focal areas of ischemic necrosis [10, 12]. However, the injury mechanism of the perforating arteries has not been demonstrated perfectly. The suggestive mechanisms are spasm or thromboembolism. The lateral perforating branches of the middle cerebral artery follow a recurrent course to reach their points of penetration in the anterior perforating space; their angle of origin is very acute. Furthermore, the more lateral the origin the more acute the angle. Dharker et al. [9] thought that stretching and distorting the angle of perforating branches lead to damage to the vessel, e.g., by ‘spasm’, with a consequent decrease in local blood flow. In our case, we could identify the punctate high-density lesion in initial CT scans, which is suggestive of thrombus in the lenticulostriate artery. The possible mechanism of the thrombus formation could be postulated such that the differential motion between the brain and the skull can lead to acute acceleration and deceleration at the time of a head injury, which compresses the arterial wall and sets in motion the thrombotic process.

Most of the patients may suffer minor head injuries, often without loss of consciousness [9]. Although a

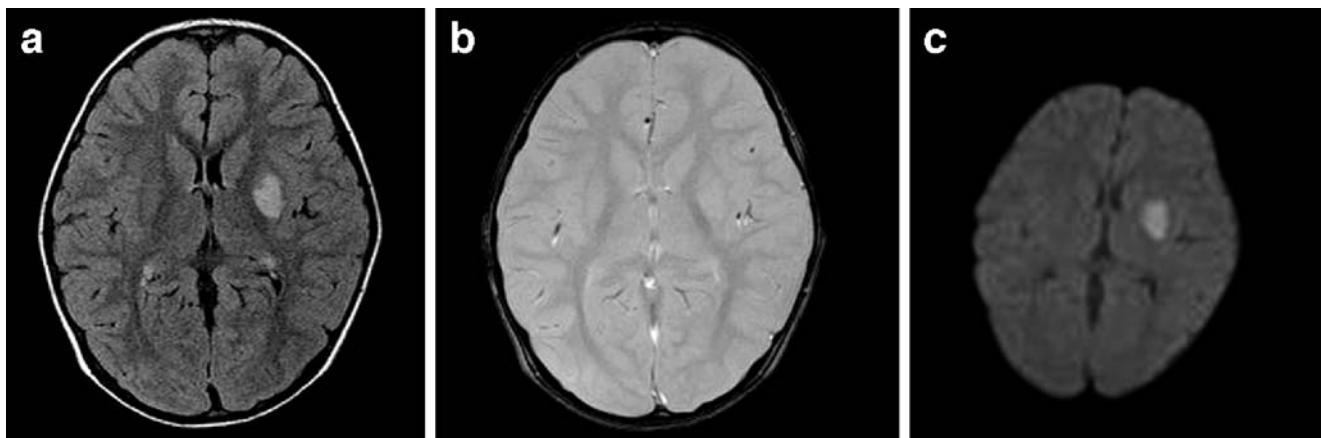


Fig. 2 **a** Fluid-attenuated inversion recovery sequences axial image demonstrates a focal high-signal intensity area in the central portion of the left basal ganglia and overlying centrum semiovale. **b** T2-weighted gradient echo sequences image shows no definite hypointense lesion corresponding with high-signal lesion on T2-weighted image.

c Diffusion-weighted images reveal a clearly demarcated high-signal intensity lesion in similar area on T2-weighted and FLAIR sequences images, compatible with infarcted tissues on the territory supplied by the lateral lenticulostriate artery

relatively small area is affected, contralateral hemiparesis with fasciobrachial predominance is unavoidable. The distinctive features include the immediate weakness that occurred more frequently in an upper limb than a lower limb after minor injury as in the present case. The associated findings of dysarthria, epilepsy, athetosis, and cognate and behavioral abnormalities are reported [13]. Recovery is usually not rapid or complete, but neurological recovery is observed within a week in most patients [9]. Subsequent recanalization in almost all perforators effectively limited the infarcted areas to pallidus, caudate nucleus, anterior limb of the internal capsule, and centrum semiovale. Most patients experienced complete recovery within 4 months as our patient.

Exclusion of a treatable lesion such as hemorrhagic lesions is important but is often difficult in practice. In our case, neither unconsciousness nor definite neurological

signs were present immediately after the trauma, and hypodense lesions in the cerebral parenchyma did not appear in the initial CT images. Delayed CT images could visualize low-attenuation regions. Differential diagnoses included cerebral infarction, edema, contusion, or demyelination. Studies using MRI indicate that low-signal intensity regions in T1-weighted images and high-signal intensity lesions in T2-weighted images are often seen in well-defined arterial distributions. For the differentiation from hemorrhagic diffuse axonal injury (DAI) lesions in the chronic stage, GRE sequences are conversely useful because they can detect susceptibility effects of hemoglobin degradation. In our patient, GRE sequences images showed no definite hypointense lesion corresponding with high-signal lesion on a T2-weighted image. These findings mean that it is an infarcted lesion as confirmed with DWI/MRI, not a hemorrhagic DAI lesion. DWI is more sensitive than T1- and T2-weighted images for the evaluation of acute infarction [14, 15].

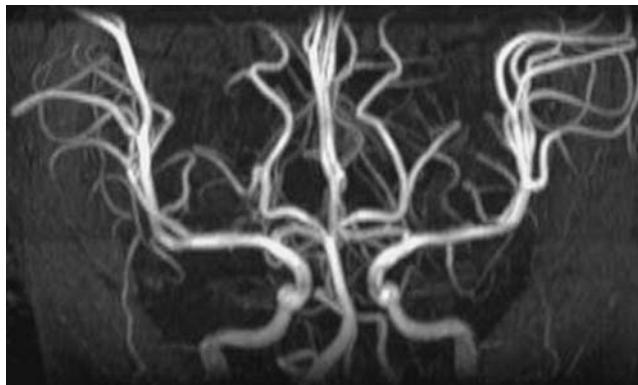


Fig. 3 MR angiography result shows no gross abnormalities including that of the left middle cerebral artery and internal carotid artery

Conclusions

We present a case of posttraumatic infarction in the territory supplied by the lateral lenticulostriate artery after a minor head injury in a child. Children with minor head trauma who have normal findings on initial CT scan may rarely have basal ganglionic infarction resulting from arterial spasm or thromboembolism of the perforating arteries. Hospital admission and careful observation should be considered for patients with minor head injury and persistent neurologic deficits (despite normal CT findings).

MR study is valuable for the evaluation of posttraumatic infarction, differentiating from hemorrhagic DAI lesions.

References

1. Macpherson P, Teasdale E, Dhaker S, Allardyce G, Galbraith S (1986) The significance of traumatic haematoma in the region of the basal ganglia. *J Neurol Neurosurg Psychiatry* 49:29–34
2. De Caro R, Munari PF, Parenti A (1998) Middle cerebral artery thrombosis following blunt head trauma. *Clin Neuropathol* 17:1–5
3. Debehne DJ, Singer JI (1991) Vertebrobasilar occlusion following minor trauma in an 8-year-old boy. *Am J Emerg Med* 9:49–51
4. Kelkar AS, Karande S, Chaudhary V, Kulkarni MV (2002) Traumatic posterior cerebral artery occlusion in a 14-month-old child. *Pediatr Neurol* 27:147–149
5. Mirvis SE, Wolf AL, Numaguchi Y, Corradino G, Joslyn JN (1990) Posttraumatic cerebral infarction diagnosed by CT: prevalence, origin, and outcome. *AJNR Am J Neuroradiol* 11:355–360
6. Oshiro S, Ohnishi H, Ohta M, Tsuchimochi H (2003) Pediatric blunt carotid injury—case report. *Neurol Med Chir (Tokyo)* 43:134–137
7. Fujioka M, Maeda Y, Okuchi K, Kagoshima T, Taoka T (1999) Secondary change in the substantia nigra induced by incomplete infarct and minor hemorrhage in the basal ganglia due to traumatic middle cerebral arterial dissection. *Stroke* 30:1975–1977
8. Martin NA, Doberstein C, Zane C, Caron MJ, Thomas K, Becker DP (1992) Posttraumatic cerebral arterial spasm: transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings. *J Neurosurg* 77:575–583
9. Dharker SR, Mittal RS, Bhargava N (1993) Ischemic lesions in basal ganglia in children after minor head injury. *Neurosurgery* 33:863–865
10. Endo M, Ichikawa F, Miyasaka Y, Yada K, Ohwada T (1991) Capsular and thalamic infarction caused by tentorial herniation subsequent to head trauma. *Neuroradiology* 33:296–299
11. Okuno T, Takao T, Ito M, Konishi Y, Mikawa H, Nakano Y (1980) Infarction of the internal capsule in children. *J Comput Assist Tomogr* 4:770–774
12. Kuroiwa T, Tanabe H, Takatsuka H, Arai M, Ozaki T, Nagasawa S, Ohta T (1994) Paramedian thalamic infarction following blunt head injury—case report. *Neurol Med Chir (Tokyo)* 34: 20–22
13. Caplan LR, Schmahmann JD, Kase CS, Feldmann E, Baquis G, Greenberg JP, Gorelick PB, Helgason C, Hier DB (1990) Caudate infarcts. *Arch Neurol* 47:133–143
14. Perkins CJ, Kahya E, Roque CT, Roche PE, Newman GC (2001) Fluid-attenuated inversion recovery and diffusion- and perfusion-weighted MRI abnormalities in 117 consecutive patients with stroke symptoms. *Stroke* 32:2774–2781
15. Ritzl A, Meisel S, Wittsack HJ, Fink GR, Siebler M, Modder U, Seitz RJ (2004) Development of brain infarct volume as assessed by magnetic resonance imaging (MRI): follow-up of diffusion-weighted MRI lesions. *J Magn Reson Imaging* 20:201–207