Hidden Dense Middle Cerebral Artery Sign in a 4-Year-Old Boy With Traumatic Subarachnoid Hemorrhage

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Abstract

A 4-year-old boy was admitted with acute onset of hemiplegia of the right side that was secondary to a traffic accident. Initial computed tomography revealed a traumatic subarachnoid hemorrhage, and follow-up computed tomography showed a more localized hematoma of the left sylvian cistern. After a few days of conservative treatment, magnetic resonance imaging (MRI) revealed a cerebral infarction of the left lenticulostriate territory, even though magnetic resonance angiography showed preserved middle cerebral artery flow. Thus, we realized that the hematoma of the sylvian cistern was the so-called dense middle cerebral artery sign. This case of posttraumatic infarction suggested the importance of meticulous investigations and clinical correlations of imaging studies in pediatric patients with head injuries.

Keywords

posttraumatic infarction, traumatic subarachnoid hemorrhage, lenticulostriate infarct, striatocapsular infarct, dense middle cerebral artery sign

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In rare cases, cerebral infarction can occur in pediatric patients after head trauma. Previous reports have suggested that traumatic infarcts in children usually involve the basal ganglia.¹ Although there are some existing hypotheses, the exact mechanisms of basal ganglia infarctions are still not clear.

In this case, the initial imaging analysis of the hemiparetic pediatric patient revealed a traumatic subarachnoid hemorrhage of the left sylvian cistern. However, the follow-up study revealed an infarction of the basal ganglia and periventricular white mater. To our knowledge, cerebral infarctions after traumatic subarachnoid hematomas are extremely rare in pediatric patients. There is only 1 report about a child with middle cerebral artery cortical infarction caused by vasospasm.²

We describe the case of a 4-year-old boy who experienced right hemiparesis and motor aphasia due to a striatocapsular infarction after a traumatic subarachnoid hemorrhage.

Case Report

A 4-year-old boy visited the emergency room for treatment after a pedestrian traffic accident. At the neurologic examination, he had right hemiplegia and a right-side facial palsy (House-Brackmann grade 2) that had been present since immediately after the trauma. He showed drowsy consciousness and a Glasgow Coma Scale score of 9. Brain computed tomography revealed a fractured right temporal bone and subarachnoid hemorrhage of the basal cistern, especially around the left middle cerebral artery (Figure 1A). However, there was no obvious traumatic contusion or abnormal low density in the brain parenchyma, which could explain his hemiplegia. Brain magnetic resonance imaging (MRI) could not be performed because of the child's poor cooperation. Instead of magnetic resonance imaging, a 4-hour follow-up brain computed tomography was performed. The follow-up computed tomography showed a reduction in hematoma and still there was no parenchymal abnormality (Figure 1B). The next day, transcranial Doppler was performed at the bedside and the flow velocity was within the normal range. On posttrauma day 5, brain magnetic resonance imaging and magnetic resonance angiography were obtained. Fluid-attenuated inversion recovery imaging revealed a hyperintense left lentiform nucleus and periventricular white matter (Figure 2A and B). Magnetic resonance

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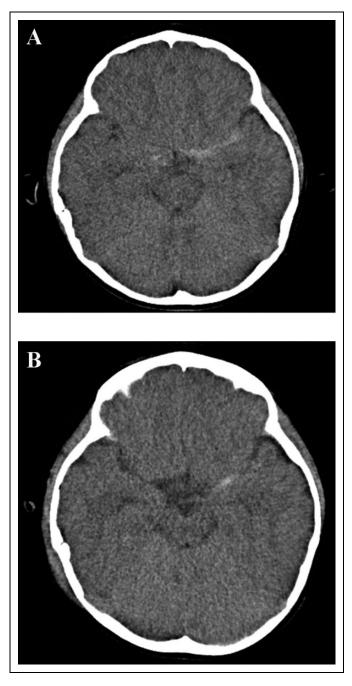


Figure 1. The initial computed tomographic images of the brain shows traumatic subarachnoid hematomas in the basal cistern and in the left sylvian cistern (A). In the follow-up computed tomography image, the hematoma was somewhat resolved and restricted to the left sylvian cistern (B).

angiography showed preserved middle cerebral artery flow (Figure 2C). The child was treated conservatively. After rehabilitation, he was able to walk and was discharged.

Discussion

In the present case, initial computed tomography of the hemiparetic child showed a traumatic subarachnoid hemorrhage of the left sylvian cistern and a right-sided temporal bone fracture (Figure 1A). Follow-up computed tomography revealed that the subarachnoid hematoma had somewhat resolved and showed the presence of a hyperdense left sylvian hematoma (Figure 1B).

We reviewed the literature to evaluate the mechanism of stroke in our patient. Although it is unusual, sometimes patients develop stroke after head injuries. The suggested pathogeneses are vasospasm, dissection, and trauma-related thromboembo-lism.^{1,3-7} Posttraumatic stroke may develop in pediatric cases.⁸ Interestingly, in such cases, the infarctions tend to occur in the basal ganglia or in the lenticulostriate territory.^{1,9-12}

In the present patient, the precise extent of the infarction spanned the lentiform nucleus and the periventricular white matter, which is the striatocapsular territory (Figure 2A and B). Why do most pediatric strokes tend to occur in the lenticulostriate territory after head injuries? Although the mechanisms have not yet been confirmed, some authors have suggested that this is due to anatomic differences in pediatric cases. The pediatric lenticulostriate arteries are more tensely stretched and they are arranged at a more acute angle with the middle cerebral artery than those in adults.³ Other authors^{1,13} have noted the possibility of predisposing vasculopathy of the lenticulostriate artery, which was observed as calcification of the basal ganglia. In the present case, there was no calcification observed on computed tomography and, thus, this case cannot be explained by vasculopathy.

We thought that the traumatic subarachnoid hemorrhage was related to the mechanisms of infarction in some way. According to the findings of the Fung et al series,⁷ traumatic subarachnoid hematomas that originate from a large vessel may result in vasospasms similar to those of aneurysmal subarachnoid hematomas. Moreover, in the O'Brien et al series,⁴ more than one-third of children suffered from middle cerebral artery vasospasm after traumatic brain injury. Owing to the existence of subarachnoid hematoma, we assumed that a vasospasm occurred. However, transcranial Doppler showed normal flow velocity. According to previous studies, the sensitivity of transcranial Doppler for detecting middle cerebral artery vasospasm ranges from 39% to 94%.¹⁴

Another possible mechanism for traumatic subarachnoid hemorrhage and subsequent stroke is dissection. Kondoh et al⁶ have described a case of striatocapsular infarction that was caused by proximal middle cerebral artery dissection. In our patient, magnetic resonance angiography showed no evidence of dissection (Figure 2C).

We realized that we previously overlooked the hyperdense attenuation of the left sylvian cistern on the follow-up computed tomography. After the surrounding subarachnoid hematomas somewhat resolved, the left sylvian cistern showed a more localized clot (Figure 1B). It resembled the so-called dense middle cerebral artery sign, which suggested thrombosis of the proximal middle cerebral artery. De Caro et al⁵ have reported thrombosis that is caused by tearing of the vessel wall of the middle cerebral artery. We supposed that the partially injured intima of the left proximal middle cerebral artery resulted in thromboembolic occlusion rather than dissection.

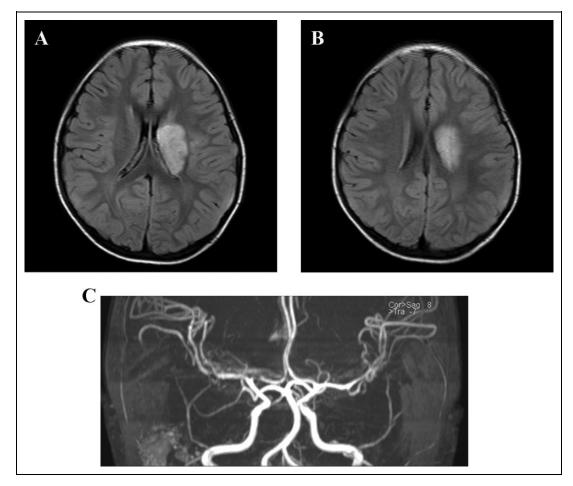


Figure 2. Magnetic resonance images (MRIs) acquired on posttrauma day 5. Axial fluid-attenuated inversion recovery images reveal an acute cerebral infarction of the left lentiform nucleus (A) and periventricular white matter (B). Magnetic resonance angiography shows favorable flow of the left middle cerebral artery (C).

Though transcranial Doppler is known to be as effective as cerebral angiography for detecting middle cerebral artery occlusion,¹⁴ it was not helpful in this case. Thus, we assume that the normal transcranial Doppler and preserved flow on magnetic resonance angiography of our patient suggest recanalization of the middle cerebral artery. The thrombus might have undergone rapid lysis and the distal middle cerebral artery territory could probably be saved because of sufficient collaterals. However, as mentioned above, the vulnerable pediatric lenticulostriate artery was sacrificed. Van Overbeek et al¹⁵ described that striatocapsular infarction is caused by transient occlusion in the proximal middle cerebral artery.

The findings of this study suggest one of the possible mechanisms of traumatic basal ganglia infarctions in pediatric cases. In many patients with trauma, especially pediatric patients, magnetic resonance imaging is hard to perform. Computed tomography, as an alternative, has the following advantages: it is rapid, it can be monitored, and it has the ability to detect fractures and hematomas. However, as in our case, computed tomography has poor sensitivity for the detection of stroke. If patients have unexplained neurologic defects, potential stroke should be considered, and meticulous reviewing of the images is imperative for the clinician to determine the appropriate treatment. In addition, if there is a hematoma that is restricted to the sylvian cistern, the hidden dense middle cerebral artery sign should be considered because it may provide a clue for early diagnosis.

Author Contributions

B-JK wrote the first draft of the manuscript. J-IC, S-KH, D-JL, and S-DK participated in the revision of the manuscript.

Declaration of Conflicting Interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical Approval

Parental consent was obtained for the patient described in this case report.

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