

Posttraumatic Infarction in the Basal Ganglia after a Minor Head Injury in a Child: Case Report

Hafif Kafa Travmalı bir Çocukta Posttravmatik Bazal Gangliyon Enfarktı: Olgu Sunumu

ABSTRACT

We present a case of posttraumatic infarction in the territory supplied by the lateral lenticulostriate artery after a minor head injury in a child. A 2.5-year-old child was admitted to our emergency room after a head-on fall from a height of 50cm. He developed a right hemiparesis and he could not speak properly for about half an hour. An initial computerized tomography of the head taken two hours after the accident was normal. A follow-up CT obtained two days later revealed a hypodense lesion at the left basal ganglia and a diffusion-weighted magnetic resonance imaging disclosed an area of infarction. The patient was conservatively medicated and full recovery was made in three weeks. Hospital admission, careful observation and early diffusion-weighted MR examination should be considered for patients with persistent neurological deficits.

KEY WORDS: Brain hemorrhage, Traumatic, Basal ganglia, Infarction, Hemiparesis, Children

ÖZ

Hafif kafa travması sonrası lateral lentikülostriat arter tarafından kanlanan alanda infarkt gelişen bir çocuk olguyu sunuyoruz. İki-buçuk yaşında erkek hastada 50 cm yüksekten düşme sonrası sağ tarafta kuvvetsizlik ve konuşma bozukluğu gelişmiş. Acil servis muayenesinde sağ kol ve bacakta 4/5 parezi vardı. Olgunun kazadan iki saat sonra çekilen bilgisayarlı beyin tomografisi normaldi. İki gün sonra çekilen kontrol BBT'sinde sol bazal ganglion bölgesinde hipodens alan saptandı. Difüzyon ağırlıklı manyetik rezonans görüntüleme (MRG) tanısı infarkt olarak bildirildi. Olgu tıbbi olarak tedavi edildi üç haftada tamamen iyileşti. Hafif kafa travmalı olgularda nörolojik kayıp uzun sürüyorsa, olguların hastanede yakın izlem altına alınmaları ve erken dönem özellikle difüzyon ağırlıklı MRG incelemelerinin yapılması düşünülmelidir.

ANAHTAR SÖZCÜKLER: Beyin kanaması, Travmatik, Bazal gangliyon, İnfarkt, Hemiparezi, Çocuklar

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INTRODUCTION

Minor head injuries are common accidents in childhood. They often present with normal neurological findings. Stroke in the basal ganglia is a rarely described event after a minor head trauma (1,5,7,9,12,13,15). We present a childhood case of an infarction in the left lenticulostriate artery territory after a mild head injury.

CASE REPORT

A 2.5-year-old child was admitted to our emergency room after a head-on fall from a height of 50cm. The child did not lose consciousness nor have a history of a seizure. He developed a right hemiparesis and he could not speak properly for about half an hour. Upon admittance there was a muscle strength of -4/5 in right upper extremity and +4/5 in right lower extremity. Other neurological findings were normal. He and his parents were right handed according to his parents. Computed tomography (CT) taken two hours later did not show any abnormalities (Figure 1A). The right hemiparesis was found to be resolved on follow-up examination two days later. However despite his normal muscle strength, the child preferred his left upper extremity to grasp objects which were handed to him, to point out objects he wanted to show and to hold his parents' hands. When his left arm was held still, he used his right upper extremity with a normal muscle strength but his arm and hand movements were clumsy. Follow-up CT revealed a hypodense lesion at the left lentiform nucleus (Figure 1B). Magnetic resonance imaging (MRI) revealed subacute infarction at the left posterior putamen and body of caudate nucleus on fluid-attenuated inversion recovery (FLAIR), diffusion-weighted and diffusion-weighted ADC mapping images (Figure 2 A,B,C,D). MR angiography (MRA) of the intracranial and cervical vessels failed to demonstrate either obliteration or stenosis of the large cerebral arteries. (Figure 3) A hematological work-up was performed to exclude the possibility of a genetic predisposition to thrombosis including Factor 8,9,11,13 and homocystein A and lipoprotein A levels were within normal limits. Factor 5 Leiden and MTHFR mutations were heterozygotic. Prothrombin 20210 A mutation was normal. Protein C (77%), protein S (69%), and D-dimer (221 ng/ml) levels were normal. Anti-nuclear antibody (0.22), and anti-Ds DNA (0.01) were negative. Additional tests for increased

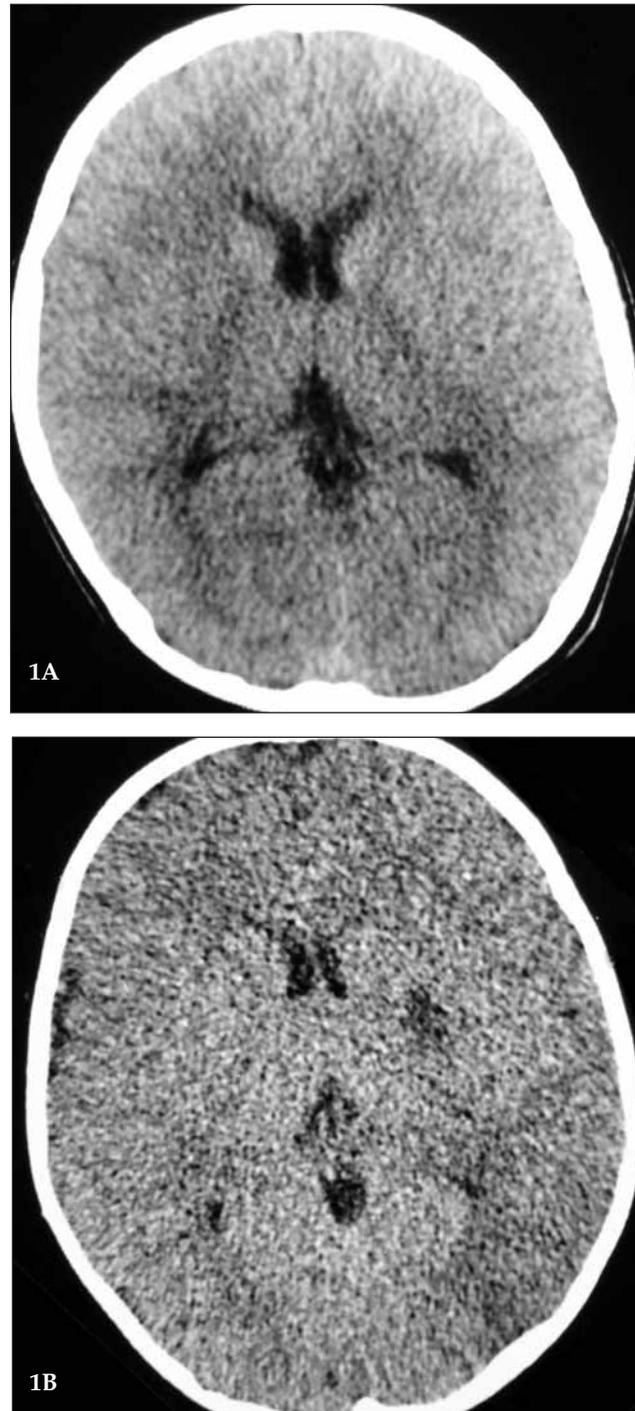


Figure 1: A- Initial CT taken two hours after head trauma. There are no pathological findings. B- CT taken two days after the accident. A hypodense area at the left basal ganglia is seen.

vascular infarction including anti-cardiolipin IgM and IgG, anti-phospholipid IgM and IgG were also negative. His T3, T4 and TSH levels were also normal. Acetyl salicylic acid was started at a dose of 50mg po/d for one week. A full recovery was made in three weeks.

Follow-up MRI examination in two months revealed persistent hyperintense lesions at the putamen and body of caudate nucleus on the left hemisphere (Figure 4A and B).

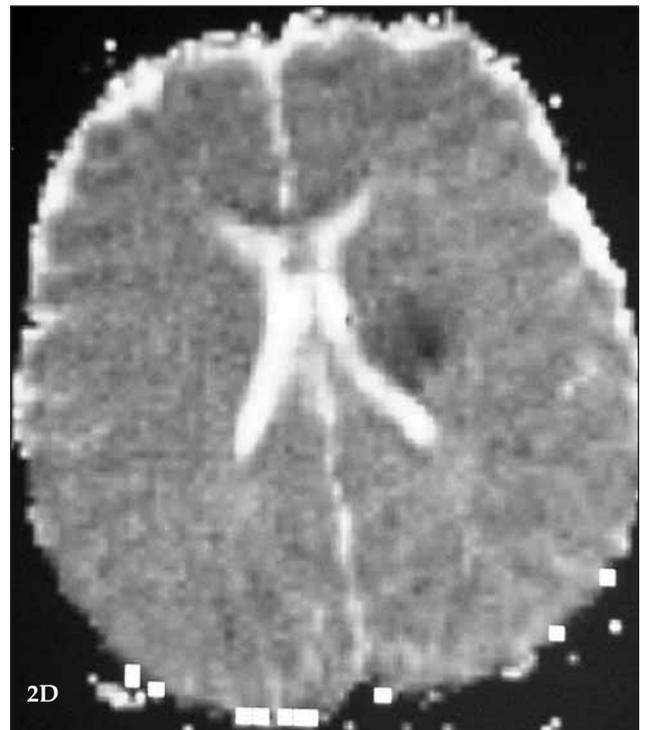
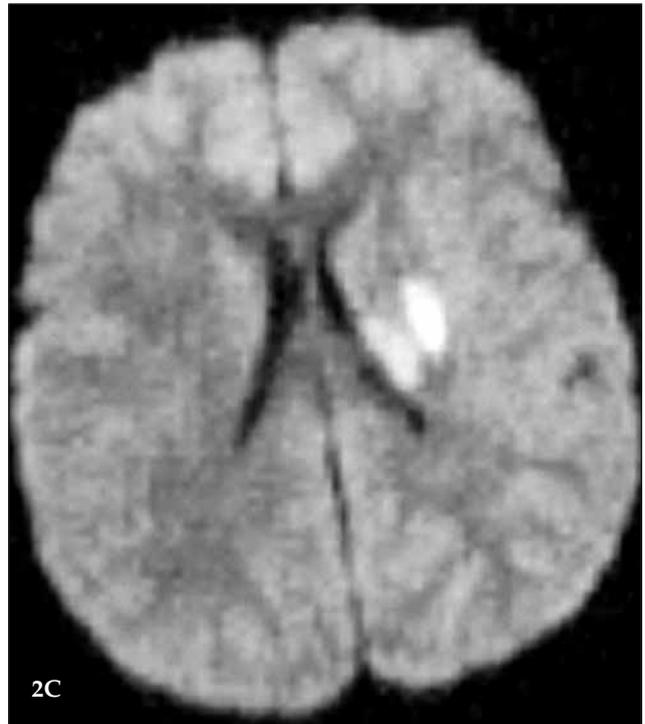
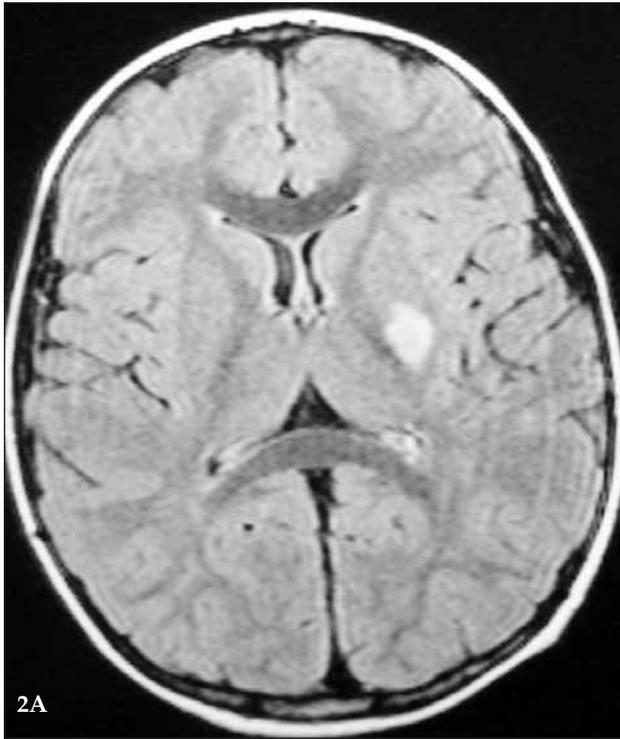


Figure 2: A- Fluid-attenuated inversion recovery MR showing a hyperintense lesion suggestive of a diffuse axonal injury or subacute infarction at the left basal ganglia. B- An MRI on DW imaging taken two days after the accident. There is a hyperintense lesion suggestive of a subacute infarction at the left posterior putamen and C- the body of the caudate nucleus. D- An apparent diffusion coefficient (ADC) map of a DW weighted axial image reveals a hypointense lesion suggestive of diffusion limitation.



Figure 3: Cranial MRA revealed neither occlusion nor stenosis of the large cerebral arteries.

DISCUSSION

Cerebral infarction after a minor head injury is an unusual mechanism of stroke in children. Other possible causes should systematically be excluded before a diagnosis is made (15). It may be difficult to rule out the possibility of an insult preceding and precipitating a head injury. In our case, the patient had a history of neurological signs after a fall witnessed by his parents. Furthermore, a genetic or an acquired metabolic cause which might lead to vessel thrombosis was ruled out by an extensive hematological work-up.

Head CT is the common diagnostic work-up in minor head trauma. Arterial dissection may account for up to 20% of cases of stroke in children and adolescents (14). Therefore, this possibility must be ruled out before a definite diagnosis is made. We obtained an MRA to evaluate the major cervical and intracerebral arteries in our case. Although the gold-standard is an angiography, we suggest that an initial MRA may be sufficient to exclude dissection.

In the previously reported cases, the lenticulostriate, thalamoperforating, or choroidal arteries have been found to be occluded (5,6,11,14). Several pathological mechanisms including occlusion, vasospasm, arterial emboli, dissection, thrombus formation and vascular compression secondary to increased intracranial pressure have been suggested (1,4,6,9). The basal ganglia and internal capsule are supplied by the lenticulostriate

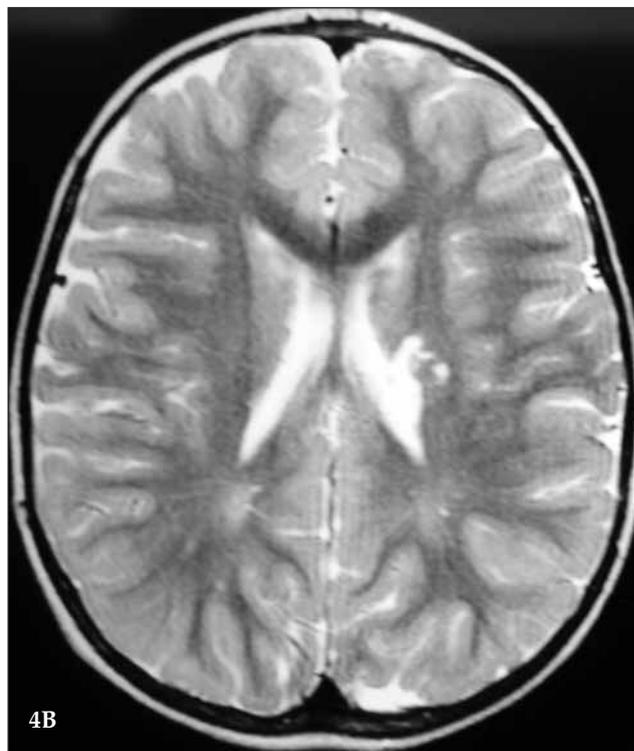
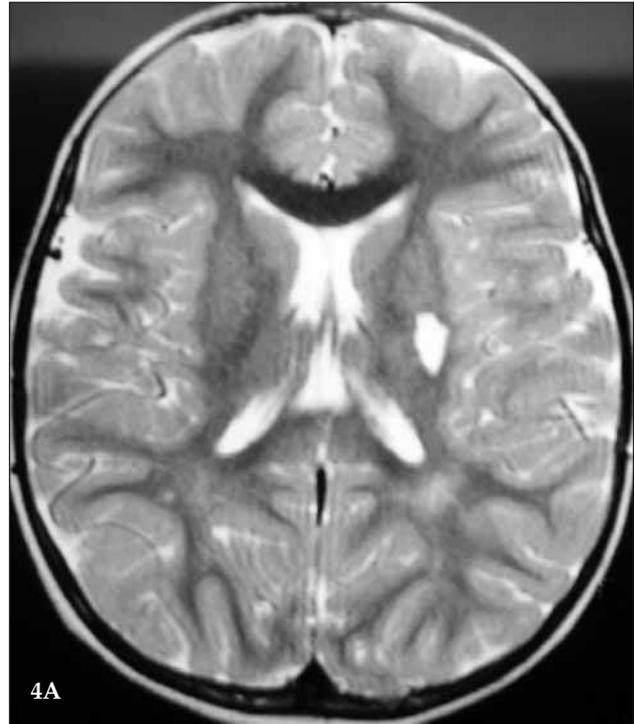


Figure 4: Follow-up T2-weighted MRI taken 2 months after the incident showing persistent hyperintense lesions at the putamen (A) and body of caudate nucleus (B) suggestive of an infarction area.

branches of the middle cerebral artery. The angle between the trunk of the middle cerebral artery and the lenticulostriate arteries is more acute and these

follow a recurrent course before penetrating the anterior perforated substance (17). These arteries are functional end arteries. Stretching and distorting the angle of perforating branches during trauma were suggested to lead to damage to the vessel and cause a decrease in the blood flow (5). The exact nature of this damage remains unsolved although vasospasm or intimal trauma and subsequent thrombosis were suggested (10,16). In the present case, the early onset and rapid resolution of neurological signs would be consistent with cerebral vasospasm.

The affected area is small in these patients. A contralateral hemiparesis with fasciobrachial predominance, as seen in the present case, is unavoidable (1). The immediate weakness occurs more frequently in the upper extremity than the lower extremity. Our case experienced a transient dysarthria, a symptom which has been reported in epilepsy, athetosis and cognitive and behavioral abnormalities among others (3). Most children have been reported to recover completely following conservative therapy (5,9,13). Rapid reversal or attenuation of neurological symptoms may be attributed to the resolution of vasospasm. However, the healing process despite an infarction may be the result of the childhood neuronal plasticity, a theory which was supported by research reports (2,8).

CONCLUSION

We present a case of posttraumatic infarction in the territory supplied by the lateral lenticulostriate artery after a minor head injury in a child. Minor head injuries may cause cerebral infarction at the internal capsule or basal ganglia in children with minor head injury whose initial CT scans are normal. The underlying mechanism may be cerebral arterial vasospasm or thromboembolism. Hospital admission, careful observation and early diffusion-weighted MR examination should be considered for patients with persistent neurological deficits.

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