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(Citation)

The Kobe journal of the medical sciences, 47(3):113-121

(Issue Date)

2001-06

(Resource Type)

departmental bulletin paper

(Version)

Version of Record

(JaLCOI)

<https://doi.org/10.24546/00082674>

(URL)

<https://hdl.handle.net/20.500.14094/00082674>



POSTTRAUMATIC ISOLATED INFARCTION IN THE TERRITORY OF HEUBNER'S AND LENTICULOSTRIATE ARTERIES: CASE REPORT

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KEY WORDS

cerebral infarction; head injury; lenticulostriate artery; recurrent artery of Heubner

ABSTRACT

A 12 year old male had a secondarily generalized epileptic seizure and a subsequent right hemiparesis with fasciobrachial predominance after a closed head injury. His seizures responded to antiepileptic drug therapy immediately. Computerized tomographic scanning and magnetic resonance imaging showed an acute infarct of the head of the left caudate nucleus, indicating the isolated occlusion of the left recurrent artery of Heubner and lateral lenticulostriate arteries. Pathologies leading to vasculitis and embolism were also looked for, but no finding of associated systemic disease could be disclosed. We present this case since posttraumatic infarction in the territory of the deep perforators such as recurrent artery of Heubner and lateral lenticulostriate arteries are exceptionally rare conditions especially in this age group.

INTRODUCTION

Trauma is known to cause a number of vascular complications in and about the head. Some of them are well known, such as occlusion of the extracranial vertebral and carotid arteries.⁵⁾ Also, occlusion of the main intracranial arteries due to nonpenetrating head injuries has been frequently observed in patients with minor head injuries, often without loss of consciousness.^{10, 15)} Spasm, emboli from the cervical portion of the internal carotid artery, dissecting aneurysm and thrombus formation have all been suggested as causes.^{11, 15, 16)} But we know very little about posttraumatic

isolated occlusion of the perforating branches We present a young patient suffering from hemiparesis due to a posttraumatic infarction confined to the territory of the recurrent artery of Heubner (RAH) and lateral lenticulostriate arteries.

CASE REPORT

A 12 year old male was admitted to our neurosurgery department with complaints of epileptic seizures and weakness of the right-sided extremities. Since he fell down from the fences surrounding his school and hit his head slightly on the ground, he was allowed to return home for resting. After he told his parents that he fell down at school and did not feel good, he had a secondarily generalized convulsions that had begun from the right side of his face and right arm. On the way to hospital, he vomited and still had convulsions in his right arm while entering the emergency unit. The seizure immediately responded antiepileptics. He was conscious and cooperating, but desorientated. A right hemiparesis with fasciobrachial predominance and a positive Babinsky sign on the right side was observed during his first neurological examination.

Although the computerized tomography (CT) on the second hour failed to disclose any intracranial abnormalities (Figure 1A), he was hospitalized for close follow-up and further investigations. Doppler ultrasonographic examination of the extracranial carotid arteries and echocardiography were performed in order rule out any source for a possible intraarterial embolism. But both failed to show any pathology regarding the extracranial carotid arteries and heart (like carotid dissection and cardiac thrombus, respectively). Transcranial Doppler ultrasonographic evaluation was also performed. Neither increase in the mean flow velocities nor abnormality in the pulsatility or resistance indices of the basal cerebral arteries were registered. The next day, Todd's paralysis were subsided with the persistence of hemiparesis. Electroencephalography revealed a left-sided subcortical focus with spike and wave activity and administration of carbamazepine with a daily dose of 10mg/kg was maintained for seizure prophylaxis

A magnetic resonance imaging (MRI) was performed on the 36th hour (Figure 2A and B) and signs of ischemia was found on the head of the left caudate nucleus, and anterior part of nucleus lentiformis which led to the diagnosis of an acute infarction on the territory supplied by the RAH (dominantly from anterior cerebral artery), and lateral lenticulostriate artery (a branch of middle cerebral artery). Then, an angiography was proposed to his parents. But they refused it because of an unexpected complication which had been experienced in one of their relatives during a coronary angiography procedure, A repeat CT scan was also performed on the fourth day (Figure 2A) in order to evaluate any possible consequence of ischemia but findings other than as that of an established infarction could have not been demonstrated.

Since such an isolated perforating artery infarct was uncommon after head injuries, rheumatologic processes that could lead to vasculitis were searched in detail by pediatricians for differential diagnosis. Beyond the rare occurrence of buccal aphtous lesions and a recent upper tract respiratory infection, neither history nor physical examination of the patient

avored the existence of systemic lupus erythematosus, Behcet's disease, or postinfectious vasculitis.

Laboratory findings were as follows; hemoglobin: 14.8gr/dl, hematocrit: 39.4%, white cell count: 14.300, Platelet count: 417.000, eritrocyte sedimentation rate: 15 mm/h, antistreptolisin-O antibody: 2911U/ml, c-reactive protein: <3mg/l, rheumatoid factor: negative, anticardiolipin IgG and IgM: negative, IgE: 571U/ml, antineutrophilic cytoplasmic antibody: negative and parasite in stool: negative. Ophthalmologic and dermatologic examinations with a negative pathergy test also did not support any finding of Behcet's disease.

He did not develop any new ischemic or epileptic attack during his hospitalization. Physical therapy and carbamazepine was given when he was discharged at the end of the second week. Latest control neurological examination was performed 9 months later and he was still suffering from a slight weakness of the right arm. Thus, Babinsky sign was positive on the right side. However, his school performance was acceptable and no seizure has developed.

DISCUSSION

Acute CT/MRI findings of moderate to severe traumatic brain injury of children are skull fractures, shearing injury, hemorrhages (subdural, interhemispheric, convexity, intraparenchymal, multiple extraaxial), and infarct/edema.^{3,9)} Posttraumatic cerebral infarction is a relatively rare consequence of head injury and its frequency, cause, and influence on mortality are not well defined.¹⁸⁾ It can usually be differentiated from edema resulting from mechanical trauma by the fact that it conforms to the distribution of a particular cerebral artery and shows evolution in a distribution atypical for traumatic contusion.^{1,10)} In most cases, major cerebral arteries are occluded and spasm, emboli from the cervical portion of the carotid and vertebral arteries, dissecting aneurysm, and thrombus formation have all been accused for occurrence of infarction.^{5, 15)} The compression and distortion of the brain secondary to cerebral herniation and increased intracranial pressure may also induce focal areas of ischemic necrosis. primarily by impairing cerebral blood flow locally, regionally, or globally.¹²⁾ However, a posttraumatic brain herniation did not develop in our patient. Besides, the ambient and quadrigeminal cisterns were intact on the initial CT images and we easily ruled out this possibility. In addition, hyperprexia and epileptic seizure activity may elevate metabolic demand, inducing a relative state of ischemia.¹²⁾ Posttraumatic cerebral infarction is demonstrated in 1.9% of patients who require cranial CT for trauma and posterior cerebral artery, as well as anterior and middle cerebral arteries are the most frequently involved vessels.¹⁸⁾ But our knowledge about posttraumatic cerebral infarction due to occlusion of the perforating arteries is very limited. The lenticulostriate, thalamoperforating, or choroideal arteries have been found to be occluded against the skull base, resulting in basal ganglionic infarction after head injuries.¹⁹⁾

Classification of subcortical infarcts in the carotid system include a-deep perforator territory, b-perforating medullary branches (from the superficial middle cerebral artery branches), c-junctional (territory between a and b), and d-combined territories.⁶⁾ The term of deep

perforators from the carotid system refers to the anterior choroidal artery, and posterior communicating artery, the medial and lateral lenticulostriate arteries that originate from middle cerebral artery and to medial lenticulostriate arteries, and the RAH that originate from anterior cerebral artery.^{13,14,20,22)}

Because branches of the lenticulostriate arteries are usually occluded, many of the subsequent radiological appearances are misinterpreted as edema or resorption of a contusion. Contrary to small penetrating arteries, selective occlusion of the main trunk of either the RAH or lenticulostriate arteries may result either from spasm or thromboembolism after trauma.

Although relatively small area is affected, contralateral hemiparesis with fasciobrachial predominance is unavoidable. Thus, associated findings of dysarthria, epilepsy, athetosis, and cognate and behavioral abnormalities are reported.^{7,17)} Most of the patients may suffer minor head injuries, often without loss of consciousness.¹⁵⁾ But a more careful differential diagnostic evaluation is necessary in such cases. Because the relatively more frequent causes of cerebral infarcts like atherosclerosis, ischemic heart disease and hypertension could have contributed to the occurrence of infarction.²⁰⁾ Moreover, it may not be easy to decide whether cerebral infarct is due to head trauma in young patients, or vice versa.

In our patient, we could initially not explain the occurrence of such an unusual infarction only with minor head injury. Apart from the common causes of infarction belonging to elderly patients like hypertension-related arteriosclerosis, we looked for autoimmune disorders leading to small vessel disease in our patient. Because 4 per cent of cases with cerebral infarct were attributed to the intrinsic diseases of intracranial vessels.⁴⁾ However, we could not find any evidence of vasculitis due to a systemic disorder that would lead to cerebral infarction.

Rather than vasculitis or juvenile ischemic strokes, delayed cerebral arterial spasm that is an important secondary posttraumatic insult and frequent complication of closed head injury, might have been the reason for occlusion of the deep perforators in our patient. A cerebral angiography would be helpful in the differentiation of an arterial dissection, if it was performed. Besides, the occurrence of arterial dissection in such minor head injuries had previously been reported.²⁾ But its necessity for our case was questionable, because CT and transcranial Doppler ultrasonographic findings were inappropriate for an arterial dissection. Since noncontusion-related posttraumatic infarction rate was reported considerably high in cases with no increase of mean flow velocities accompanied with normal or altered cerebral perfusion pressures,⁸⁾ such an isolated infarction might have been developed in our patient. The involvement of two groups of perforators, originating from two discrete basal cerebral arteries were not supporting a cerebral arterial dissection. Thus, an extracranial carotid dissection had also been ruled out with the carotid Doppler ultrasonography.

We concluded that epileptic seizures together with the delayed arterial spasm secondary to the stretching of the deep perforators during the movement of the hemispheres on a plane perpendicular to that of the perforators, were the reasons of the posttraumatic infarction in our patient.

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FIGURES LEGENDS

Fig. 1. Initial CT scan that was performed on the second hour (A) demonstrated no pathology in the basal ganglia while latest CT scan on the fourth day (B) showed two discrete isolated and well-demarcated foci of hypodensities at the head of the left nucleus caudatus and the anterior part of the nucleus lentiformis.

Fig. 2. MRI was performed on the 36th hour and demonstrated acute ischemic areas characterized with hyperintense signals on T₂ Weighted (TSE T₂ ax. TR= 3500 msec, TE= 93 msec and 5 mm of slice thickness) images (A) and hypointense signals on T₁ weighted (SE T₁ cor. TR= 580 msec, TE= 14 msec, and 5 mm of slice thickness) images (B) at the head of the left nucleus caudatus and the anterior part of the nucleus lentiformis. Thus, no features of hemorrhage was detected

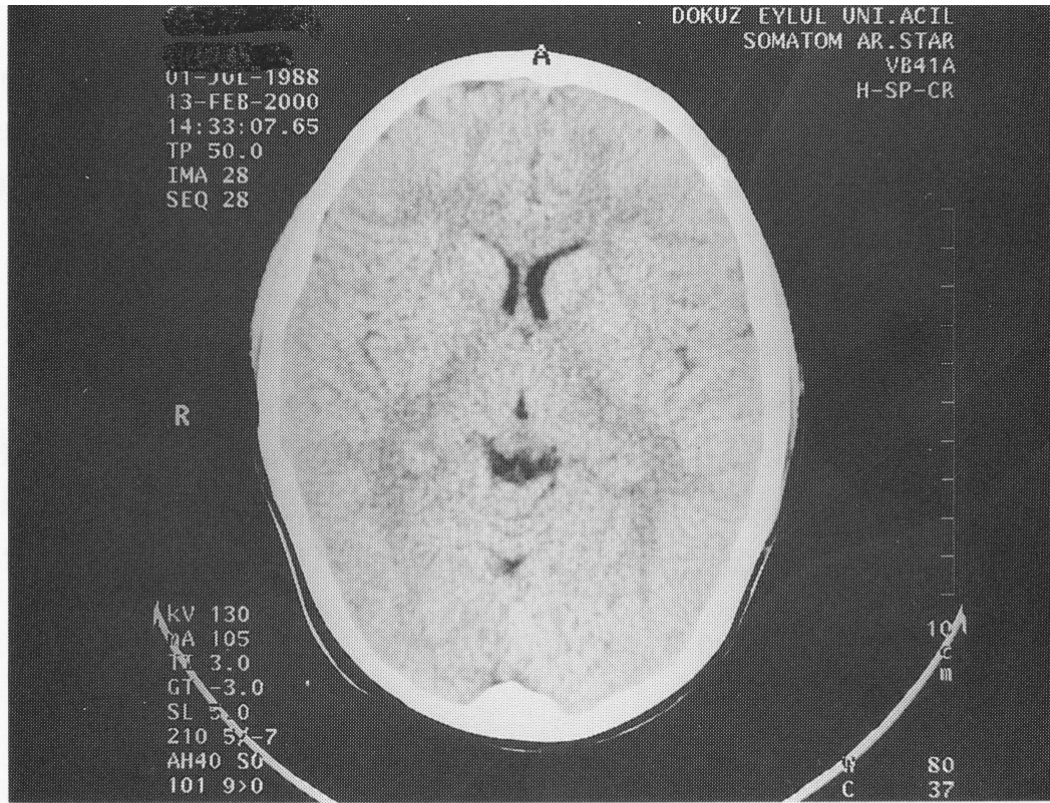


Figure 1 A.

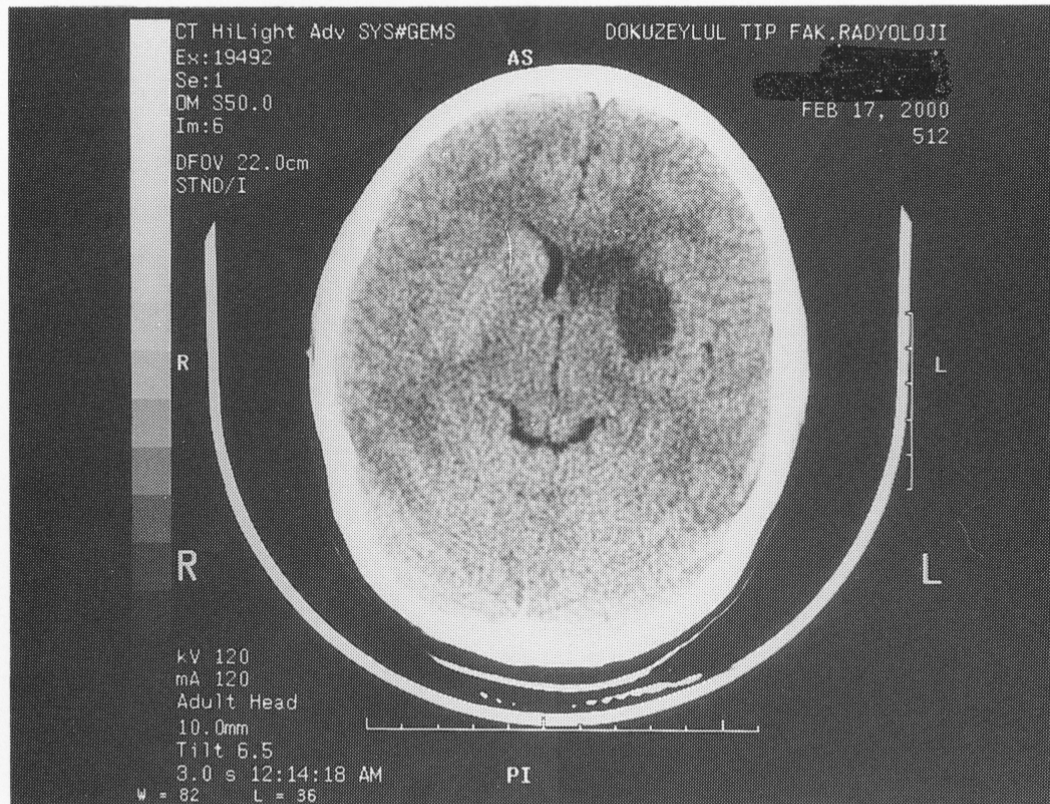


Figure 1 B.

TRAUMA AND DEEP PERFORATORY INFARCT

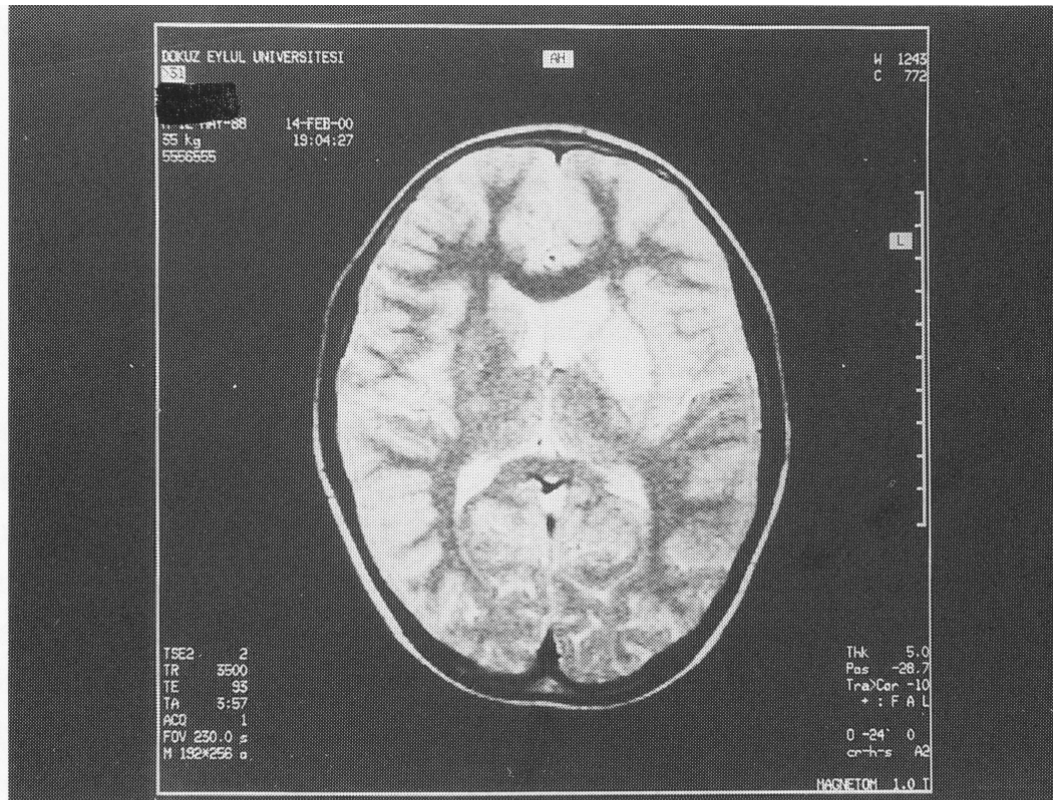


Figure 2 A.

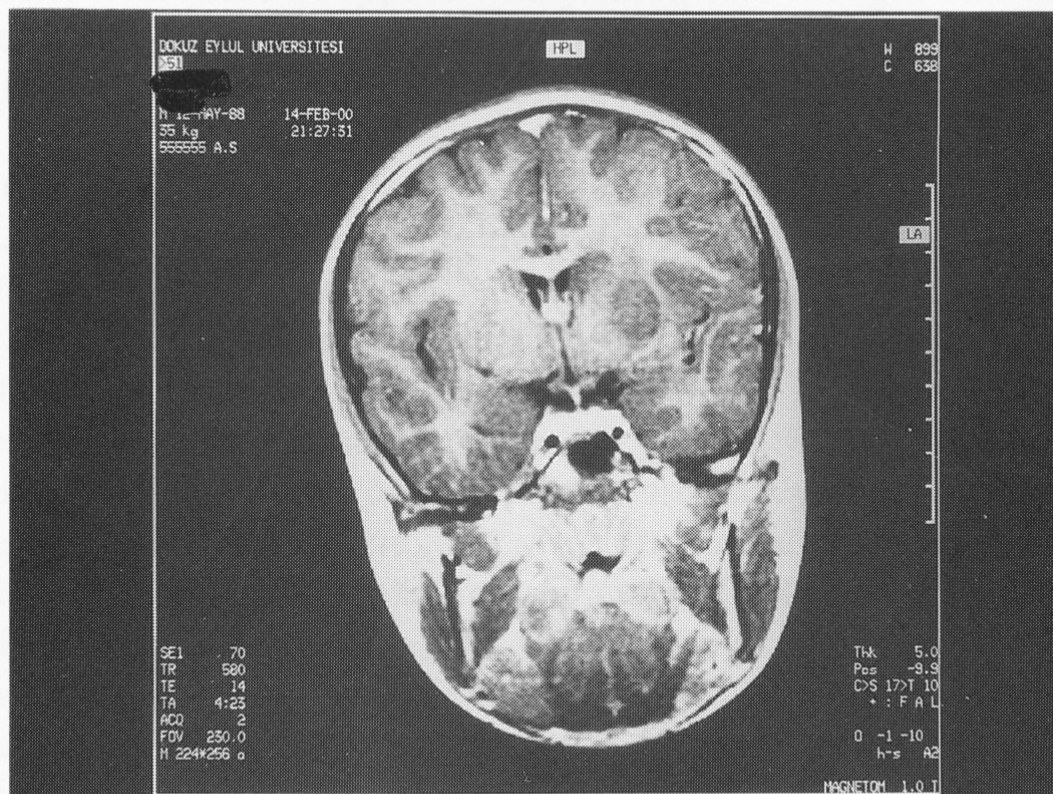


Figure 2 B.