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Case Report

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Left posterior cerebral artery occlusion following minor head trauma: a case report

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ABSTRACT

A 17-year-old male patient who received a punch behind the left ear 2 days ago, was admitted to the emergency room with a headache and dizziness. His neurological examination was normal. Brain computed tomography (CT) images demonstrated diffuse hypodense areas and sulcus deletion in the left occipital lobe. On magnetic resonances (MR) images, diffusion restriction for acute ischemia in the left posterior cerebral artery (PCA) watershed area was determined, and hospitalization was recommended. However, he and his relatives left the hospital. Through the electronic information system called "e-nabız", it was learned that the patient was hospitalized in an external center and treated with antiplatelet and anticoagulant regimens for ten days due to an acute thrombus at the left PCA P1 distal-P2 level observed on the brain CT and MR angiography images. In our clinic, his neurological examination was completely normal thirty days later. Still, a visual field examination revealed a total quadrianopsy in the upper temporal pole on the right and left eyes. An encephalomalacia in the left PCA watershed area was observed on CT images. The findings of this case showed that minor head trauma in children can cause large vessel occlusion and therefore close follow-up of these children for a certain time would be appropriate.

Keywords: Posterior cerebral artery, stroke, occlusion, minor head trauma

INTRODUCTION

It has been reported in the literature that dissection-occlusion may occur in intracranial vessels after minor head trauma.[1] Different publications show that 12% of children followed up with acute ischemic stroke (AIS) have a history of trauma, which is independent of energy, up to an average of 12 weeks ago.[2,3] While intracranial hemorrhages may be seen in high-energy traumas, it has been noticed that vascular occlusions may occur after low-energy traumas. Due to this ischemia, a wide variety of permanent or temporary sequelae may appear depending on the affected area of the brain.[1]

In this case report, the patient who developed posterior cerebral artery (PCA) occlusion after minor head trauma was discussed.

CASE

A 17-year-old male patient presented to the emergency department of our hospital with a headache and dizziness. It was learned from his anamnesis that he was punched behind the left ear two days ago during a fight with his friend and was discharged from the hospital after no abnormal findings were detected in computed tomography (CT) images of the brain taken at an external center. During the examination performed at our center, he was conscious, cooperative, and oriented, and had a Glasgow Coma Scale (GCS) score of 15/15. Cranial nerve and cerebellar examination findings were found to be normal. No deficit or pathological reflex was detected in the motor examination. In addition, it was learned from the patient's medical history that he had no known comorbidities and no medication use. Based on the patient's clinical findings, it was suspected that there might be an intracranial lesion and a brain CT was performed. CT images showed diffuse hypodense areas and sulcus effacement in the left occipital lobe (Figure 1). Brain magnetic resonances (MR) images revealed diffusion restriction in the anterior part of the left thalamus and the left PCA watershed area, and hospitalization was recommended; However, patients and their relatives left the hospital voluntarily (Figure 2). Through the electronic

information system (called "e-nabiz"), it was learned that the patient was admitted to an external center and his treatment started on the same day. Brain CT-angiography and MR-angiography images taken in this external center revealed an acute thrombus in the lumen of the left PCA P1 distal-P2 level and there was no filling in the distal part (Figure 2). It was learned that the patient was discharged with full recovery under "acetylsalicylic acid" and "enoxaparin sodium" regimens after a ten-day treatment period at the relevant external center.

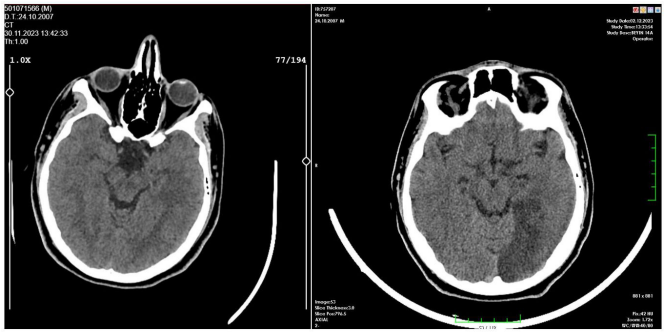

Figure 1. The patient's early CT scans at an external center were normal (left). A hypodense area (ischemia?) located in the left occipital lobe was detected in CT images taken in our hospital two days after the trauma (right)

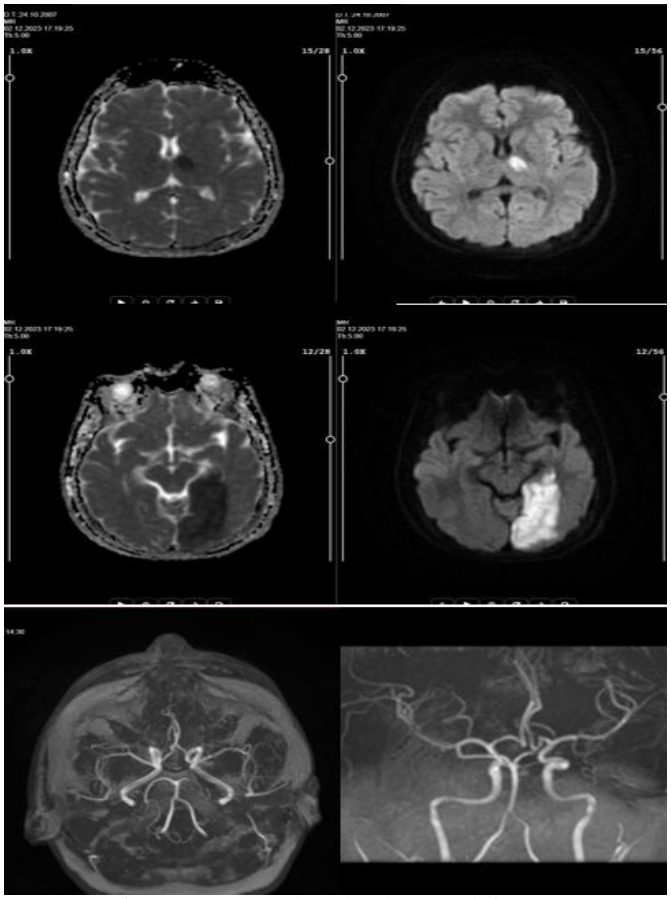

Figure 2. Diffusion MR images show that there was diffusion restriction in favor of acute ischemia in the anterior part of the left thalamus and the left PCA irrigation area. Brain MR-angiography images showed that there was no filling in the patient's left posterior cerebral artery after the P1 segment

One month later, the patient applied to our outpatient clinic for a check-up. The neurological examination was found to be normal. A loss of density consistent with chronic ischemia was observed in the control brain CT images taken of the patient (Figure 3). Following the eye examination, the direct and indirect light reflexes of the right and left eyes were normal, and the visual acuity value was found 20/20, the biomicroscope examination of the right and left eyes showed natural findings, the fundus, and optic disc, and macula findings of the right and left eyes were normal, the pupils of the eyes were normal. It was learned that there was no tear, hole, or detachment in the retina after dilatation with the applied pharmacological agent. In the visual field examination, total quadrianopsia was detected in the upper pole of the temporal field on the right, and partial hemianopia in the upper pole of the temporal field on the left. Examination performed by Pediatric Hematology revealed no coagulation disorder/bleeding diathesis. Blood biochemistry and coagulation parameters were performed. In the results, there were seen that D-dimer was under 0.19 mg/L (reference range < 0.55mg/L), fibrinogen was 1.97g/L (reference range 1.7-4.2 g/L), activated partial thromboplastin time (aPTT) was 26.2 seconds (reference range 21-32 seconds), prothrombin time (PT) was 12.6 seconds (reference range 9.8-14 seconds), prothrombin activity was 68.7% (reference range 70-130%), international normalized ratio (INR) was 1.14 INR (reference range 0.8-1.2 INR), c-reactive protein (CRP) was 0.9 mg/L (reference range 0-5 mg/L), collagen and epinephrine induced bleeding time was more than 300 seconds (reference range 82-150 seconds), collagen and adenosine diphosphate (ADP) induced bleeding time was 80 seconds (reference range 60-100 seconds), troponin-I H was under 0.0025 ng/mL (reference range <0.045ng/mL). In addition, no thrombus or anomaly was detected in the heart cavities, heart valves, and main heart vessels by examination of Pediatric Cardiology. Finally, the carotid artery and vertebral artery Doppler ultrasonography revealed no thrombus, dissection, plaque, aneurysm, or filling defect in the relevant vessels.

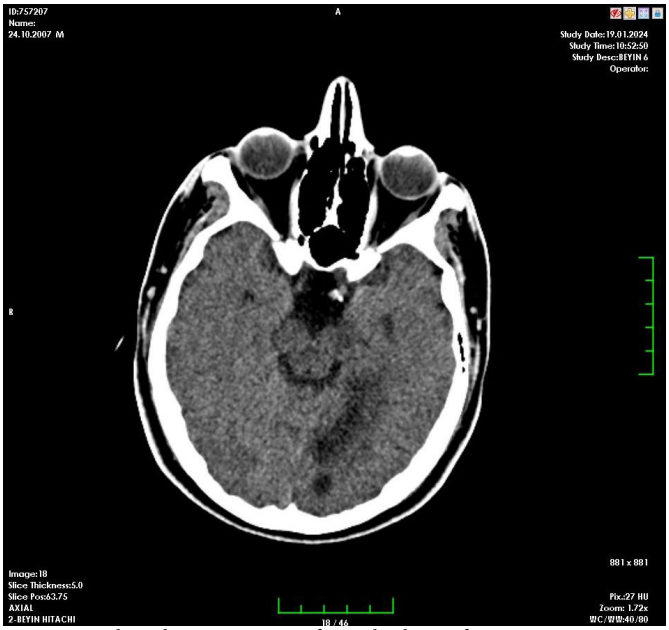

Figure 3. When the patient came for a check-up after receiving inpatient treatment, areas of chronic ischemia were seen on the CT images taken in our hospital

DISCUSSION

Headache is the third most common reason why pediatric patients visit the emergency department.[4] It is known that headaches are mostly caused by upper respiratory tract infections or sinusitis.[5] It was learned that the patient

received a fist blow behind the left ear two days ago and had no complaints other than headache and dizziness, which continued to increase within forty-eight hours. At the time of admission, it was observed that blood pressure was within the normal range, there was no fever, and respiratory and pulse rates were normal and rhythmic. The physical examination was normal. According to the anamnesis, it was learned that there was no acquired or hereditary disease. Considering that the patient presented with complaints that increased after trauma, CT scans were taken due to the possibility of intracranial disorders (subarachnoid hemorrhage, epidural hemorrhage, etc.), and an infarct area in the PCA watershed on CT images was seen. Therefore, it was thought that the patient might have a pediatric ischemic stroke.

It has been reported in the literature that pediatric acute ischemic stroke (pAIS) syndrome occurs mostly in male children (60%) and that even if treated, two-thirds of the patients may have sequelae of various degrees.[2] One of the most common causes of pAIS is cardiac problems (heart valve diseases, large vessel diseases, cardiac anomalies, rheumatic diseases, etc.).[6,7] Another important factor is arteriopathies, and the most common arteriopathy is a focal occlusive disease of the terminal internal carotid or proximal middle/anterior cerebral artery, and these arteriopathies may be triggered by infection and inflammation.[1,2,6-8] In our patient, infection was not considered based on his history, clinical findings, and blood biochemistry test results at the time of admission. In addition, echocardiography revealed neither thrombus nor anomalies in the heart cavities, heart valves, and main heart vessels. Finally, the carotid and vertebral artery Doppler ultrasonography examinations showed no filling defect in the carotid and vertebral arteries. With these findings, it was thought that the patient's PCA occlusion could not be of cardiac or main vasculature origin.

Additionally, it is known that hematological disorders (bleeding diatheses, coagulation disorders, etc.) may cause pAIS. Although it has been reported that factors such as protein S, antithrombin III, plasminogen and plasminogen activator inhibitor (PAI) deficiencies, lipoprotein (a) elevation, antiphospholipid syndrome, and hyperlipidemia may also cause pAIS, in terms of prothrombic factors, Factor V (Leiden) mutation, protein C deficiency, and prothrombin mutation are the most common causes.[1]

In the present case, the Pediatric Hematology consultation revealed no hematological disease. On the other hand, no visual field defect could be detected in the patient with confrontation, the Ophthalmology Department revealed no pathological findings originating from the orbit. However, in the automatic perimetry examination, total quadrianopsy was detected in the upper pole of the temporal field on the right eye, and partial hemianopsia in the upper pole of the temporal field on the left eye. With these findings, although the neurological examination findings were found to be normal, it was concluded that objective examinations should support the findings.

Additionally, it has been reported that pAIS can occur as a result of minor or major head trauma.1 This is thought to be due to the anatomical location of the brain vessels of trauma in pediatric patients. It is known that brain parenchymal vessels in children are present at a steeper angle than in adults. For this reason, the vessels can be stretched and ruptured due to the tension force and high inertia that occur after trauma. If the damage occurs with high energy, it causes bleeding due to complete dissection. On the other hand, trauma with lower energy causes damage to the vascular endothelium due to partial dissection. The inflammatory process secondary to this damage to the vascular endothelium leads to traumatic endothelial intimal lesions, followed by fibrin accumulation, leukocyte reaction, and white thrombus formation, resulting in lumen obstruction. This occlusion causes cerebral parenchymal ischemia with clinical findings after a symptom-free latent period.[1,3] After the patient's history, consultations, radiological imaging, and biochemical research results, it was thought that the acute-subacute infarct area in the patient's left occipital lobe might be caused by minor trauma behind the patient's left ear.

Moreover, it has been reported that the time to emergence of neurological disorders (such as headache, dizziness, nausea, hemiparesis, drowsiness, and confusion) may be shorter in children with pAIS. For this reason, it has been reported that waiting for a focal deficit that continues for 24 hours, as in adults, may cause a delay in the correct diagnosis and treatment process.[6] In the literature, recombinant tissue plasminogen activator (r-tPA) and/or heparin infusion are recommended in adults if PCA or other main vessel occlusions are detected within the first four hours of their occurrence. If this period is exceeded, heparin, anticoagulant, and antiplatelet treatments can be applied. However, considering the severity of hemorrhagic complications in pediatric patients, r-tPA or heparin infusion is not recommended, instead acetylsalicylic acid (ASA), low molecular weight heparin, or coumadin is preferred. While very long-term treatment is recommended in pAIS of cardiac origin, it is recommended to keep the treatment duration shorter in post-traumatic pAIS.[6,9,10] Since the patient's vital signs were stable, there was no significant neurological loss, and the diagnosis of PCA occlusion could be made forty-eight hours after the minor trauma, it was observed that the patient was treated at an external center with acetylsalicylic acid as an antiplatelet and low molecular weight heparin (enoxaparin sodium) as an anticoagulant. The fact that no progression was detected in the patient's radiological imaging after 30 days suggested that the treatment given was effective.

In conclusion, it is recommended that pediatric patients with asymptomatic minor head trauma and no abnormal findings detected in the first brain CT images taken should be followed closely and that patients who are symptomatic in the future should be subjected to detailed radiological, hematological, and cardiological and laboratory examinations in terms of complications that may occur secondary to trauma and it was argued that families should be informed in detail in advance about possible symptoms and complications.

ETHICAL DECLARATIONS

Informed Consent

The signed written informed consent form was obtained from the patient's parents.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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