Case Report / Olgu Sunumu

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# Post-traumatic Carotid Artery Dissection and Infarction

## Travma Sonrası Karotis Arter Diseksiyonu ve Enfarktüsü

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## Abstract

Post-traumatic internal carotid artery dissection is a very rare condition in children that occurs after blunt force trauma to the head or neck. A 15-year-old male patient who presented to the emergency room with weakness after a trauma to his neck sustained during a physical altercation is presented.

**Keywords:** Pediatric trauma, internal carotid artery, dissection, ischemic stroke

## Öz

Çocuklarda baş boyuna künt travma sonrası internal karotid arter diseksiyonu çok nadir bir durumdur. Kavga esnasında boyun travmasına maruz kalan ve sonrasında acil servise halsizlik, bilinç bulanıklığı şikayetleri ile başvuran 15 yaşında erkek hasta sunulmaktadır.

**Anahtar Kelimeler:** Pediyatrik travma, internal karotid arter, diseksiyon, iskemik inme

## Introduction

Post-traumatic carotid artery dissection (PTCAD) describes mechanical compression of the entire carotid artery wall caused by a subintimal hematoma. The main lumen may be narrowed by the pseudo-lumen, leading to stenosis.[1] In addition, thrombus in the pseudo-lumen may cause intracranial embolism. Arterial ischemic stroke (AIS) can cause morbidity in both children and adults. Early diagnosis and appropriate treatment are important to prevent or limit the damage caused by brain ischemia.[2] The pathophysiology of pediatric and adult carotid artery dissection (CAD) differs according to location and clinical presentation.[3] Most pediatric cases of PTCAD result from direct blunt or penetrating trauma of the internal carotid artery (ICA), acute hypertension, sudden hyperextension, or excessive rotation of the neck. Since children show craniocervical instability due to weak neck muscles, adherence to ligament structure rather than bone structure, a high head-neck ratio, and underdeveloped protective reflexes, their risk of PTCAD is higher than that of adults.[4]

PTCAD typically affects the distal cervical segment of the ICA, and the degree of vascular mobility of the ICA suddenly changes before it enters the carotid canal at the base of the skull in children.[5] During both hyperextension due to rapid deceleration or rotation of the head, the ICA stretches over the upper cervical vertebra, and rupture of the internal wall of the vessel can occur.[6]

## Case Report

A 15-year-old male patient involved in a physical altercation with a friend the day before presentation to the emergency department, and who was punched in the neck, presented with weakness and altered mental status. The vitals signs were as follows: Temperature, 36.7 °C; heart rate, 84 beats/min; blood pressure, 125/80 mm Hg; respiratory rate, 18 breaths/min; and oxygen saturation, 97%. On physical examination, besides altered mental status, no patient orientation or cooperation was present. The response to the painful stimulus was weak. The Babinski sign was positive on the right side, but not on the left side. The deep tendon reflex

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was normal in the right patella. No anisocoria or abnormal pupil reaction was seen, and all other physical examination findings were normal.

Cranial computed tomography (CT) revealed brain edema and a mild hypodense area in the left globus pallidus (Figure 1). Cranial and diffusion magnetic resonance imaging (MRI) showed dissection of the left ICA and acute ischemia in the left lentiform nucleus, respectively (Figure 2). CT angiography (CTA) revealed marked thinning in the calibration, compatible with the PTCAD evident at the level of the skull base and starting from the distal cervical segment of the left ICA (Figure 3).

Surgical and interventional procedures were not required by department of neurosurgery and neuroradiology. The patient was transferred to the intensive care unit, and 1 mg/kg enoxaparin was started. No features other than bradycardia was detected on echocardiography and electrocardiography. A slow wave was observed in the left hemisphere in electroencephalography. During follow-up, the patient regained consciousness and started to respond to questions. Muscle strength improved to 4/5. Enoxaparin maintenance therapy was stopped, and clopidogrel 75 mg/day was started.

The patient was discharged without sequelae, and MRI, MR angiography (MRA), and carotid color Doppler ultrasound findings were normal 3 months after discharge.

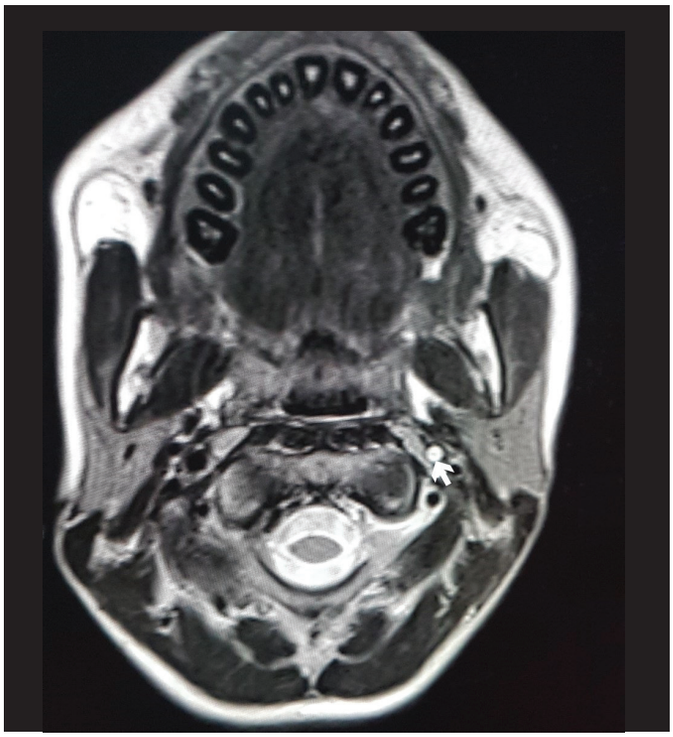

**Figure 1.** Non-contrast CT showing ventricular compression compatible with brain edema, and a suspicious left-sided hypodense area

CT: Computed tomograhpy

## Discussion

The carotid artery dissection accounts for about 2% of all strokes. Carotid artery dissection is most common among young people. Spontaneous or traumatic CAD can affect the intracranial or extracranial segment of the carotid artery. Penetrating or blunt trauma can cause CAD. The carotid artery dissection accounts for about 20% of pediatric cases

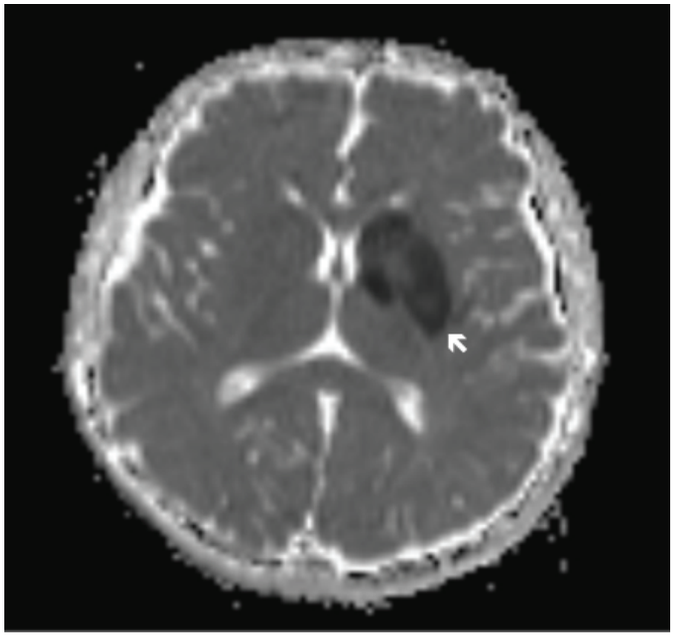

**Figure 2.** Diffusion MRI showing diffusion restriction, consistent with the ischemic area in the left lentiform nucleus

MRI: Magnetic resonance imaging

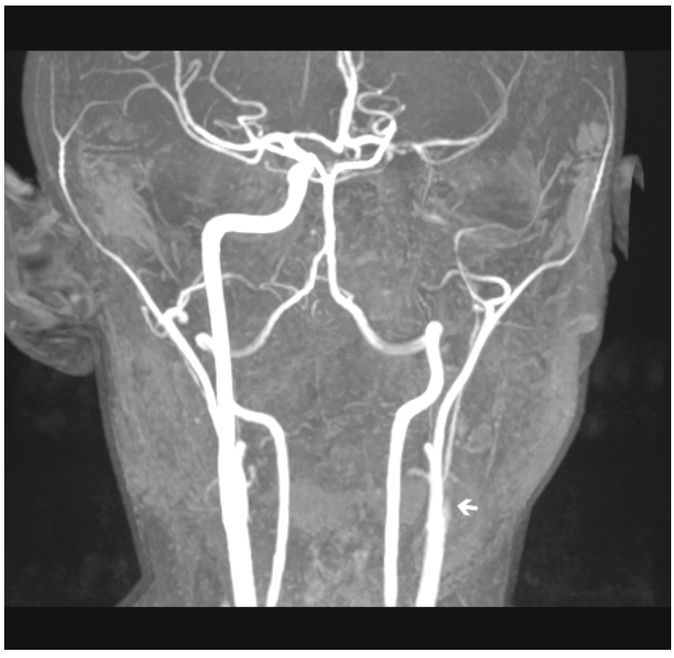

**Figure 3.** CT angiography showing total occlusion of the internal carotid artery

CT: Computed tomograhpy

of AIS.[7] The long-term outcome depends on the size of the brain area affected. Serious neurological sequelae may occur, such as severe hemiparesis, hemiplegia, aphasia, and epileptic seizures.[8] Ischemia-induced damage increases with the size of the affected area. Prodromal symptoms of CAD can vary markedly among pediatric patients. Symptoms such as epileptic-like seizures and coma may delay diagnosis. Early diagnosis and treatment can reduce morbidity.[9]

Many genetic and environmental factors are associated with CAD in children, including upper respiratory tract infections, congenital heart diseases, connective tissue diseases, homocystinuria, and head and neck trauma.[10]

The distal segment of the ICA, before it enters the carotid canal at the base of the skull, is the segment most vulnerable to PTCAD. During hyperextension and neck rotation, the ICA is stretched over the upper cervical vertebrae, which may cause intimal tears.[11]

CT can reveal skull base fractures typically associated with PTCAD in children. In our case, non-contrast CT was performed during the initial assessment, and brain edema and apparent parenchymal damage were detected.[12] Although it has not been used extensively, CTA has been reported to be as sensitive as MRA for CAD detection.[13]

Conventional angiography is the gold standard for diagnosis of PTCAD; however, it is not suitable for all pediatric patients.[14] MRI findings of PTCAD include the absence of a normal flow cavity and narrowing of the arterial lumen caused by hematoma within the arterial wall. MRA may reveal conical narrowing or occlusion of the dissected vessel. The main advantage of MRI over conventional angiography is that it can be used to estimate the time of occurrence of CAD-associated thrombosis. Anticoagulants may be indicated to prevent thromboembolism from affecting the brain. However, evidence regarding their effects in CAD is scarce.[15]

## Conclusion

PTCAD is one of the most common causes of pediatric AIS. Emergency physicians should be aware of the increased risk of PTCAD in children with head and neck trauma, arising from the characteristics of the pediatric cranio-cervical junction and the mobility of the neck. PTCAD should be excluded in patients with a history of blunt or penetrating injury, sudden acceleration/deceleration of the head, or excessive rotation of the head and neck region. Early diagnosis and treatment of PTCAD, before the onset of neurological symptoms, are important for good long-term outcomes.

### Ethics

**Informed Consent:** The consent of the patient's parents was obtained for the publication of this case report.

**Peer-review:** Externally peer-reviewed.

### Authorship Contributions

Concept: Y.S., Y.E.D., Design: Y.S., Y.E.D., Analysis or Interpretation: Y.S., Y.E.D., Writing: Y.S., Y.E.D.

**Conflict of Interest:** No conflict of interest was declared by the authors.

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