Post-Traumatic Late Onset Cerebral Ischemia

Post-Travmatik Geç Başlangıçlı Serebral İskemi

Post-Travmatik İskemi / Post-Traumatic Ischemia

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Özet

Travma sonrası iskeminin en sık nedeni çoğunlukla diseksiyon nedeniyle ortaya çıkan arterden artere emboli veya kraniyoservikal arterlerin tıkanmasıdır. 29 yaşındaki erkek hasta kafa travmasının olmadığı sert paraşütle inişinden sonra 45 dakika süren bilinç kaybı nedeni ile 3 gün önce başka bir hastaneye başvurmuş ve nörolojik muayenesinin ve beyin BT incelemesinin normal olması üzerine 24 saatlik gözlem sonrası taburcu edilmiş. Taburcu olduktan 2 gün sonra epileptik nöbet geçiren hasta hastanemize başvurdu. Nörolojik muayenede sol hemianopi saptanan hastanın çekilen beyin MR incelemesinde sağ oksipital bölgede subakut iskemi saptandı. Romatolojik, kardiyolojik ve vasküler patolojiler dışlandıktan sonra hastaya geç başlangıçlı serebral iskemi tanısı konarak antiödem ve antiepileptik tedavi başlandı. Hasta sol hemianopi ve hafi kognitif bozukluk ile taburcu edildi. Travmayı takiben bilinç kaybı olan hastaların en az 72 saat gözlem altında tutulmasının mantıklı olacağını düşünmekteyiz.

Anahtar Kelimeler

Post-Travmatik Serebral İskemi; Post-Travmatik Epilepsi; Geç Başlangıçlı Epilepsi; Kafa Travması

Abstract

Artery-to-artery emboli or occlusion of craniocervical arteries mostly due to dissection are the most common causes of ischemia after trauma. A 29 year-old male had been admitted to another hospital with loss of consciousness lasting for about 45 minutes after a hard parachute landing without head trauma three days ago. As his neurological examination and brain CT were normal, he had been discharged after 24 hours of observation. Two days after his discharge, he was admitted to our department with epileptic seizure. His neurological examination revealed left hemianopia. After observing occipital subacute ischemia at right side in brain magnetic resonance imaging (MRI), we performed cerebral angiography and no dissection was observed. Excluding the rheumatologic, cardiologic and vascular events, our final diagnosis was late onset cerebral ischemia. Anti-edema and antiepileptic treatment was initiated. He was discharged with left hemianopia and mild cognitive deficit. We suggest that it will be wise to hospitalize patients for at least 72 hours who has a history of unconsciousness following trauma.

Keywords

Post-Traumatic Cerebral Ischemia; Post-Traumatic Epilepsy; Late Onset Epilepsy; Head Trauma

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Introduction

Traumatic brain damages occur primarily due to hemorrhage, cranium fractures and parenchyma damage caused by mechanical effects of acute trauma. Post-traumatic hemorrhage or bone fracture may be determined on computerized tomography (CT) but detecting parenchyma damage due to concussion known as "whiplash injury" may not be possible in the early periods. Injury due to concussion is a result of pressure change of parenchyma towards cranium because of rapid speed changes like in traffic accidents. Neuronal injury may cause late onset ischemia in hours, days or weeks [1, 2]. Thus, patients with concussion injury must be observed even though their first cerebral images are normal.

Post-traumatic cerebral infarction is a known complication of craniocerebral trauma, with a frequency ranging from 1.9% to 10.4% [3,4]. It was suggested that cerebral vasospasm, direct vascular compression by mass effects, vascular injury, embolization, and systemic hypoperfusion play a role in the etiology [4].

We present a case with post-traumatic late onset cerebral ischemia due to hard landing during parachute training (without any head trauma) who presented with epileptic seizures 72 hours after trauma.

Case Report

A 29 year-old male, army paratrooper sergeant, was admitted to our department after an epileptic seizure. He was admitted to a local hospital with loss of consciousness lasting for about 45 minutes after a hard parachute landing without head trauma three days ago. On his admission his neurological examination and brain CT was in normal limits (Figure 1A). He

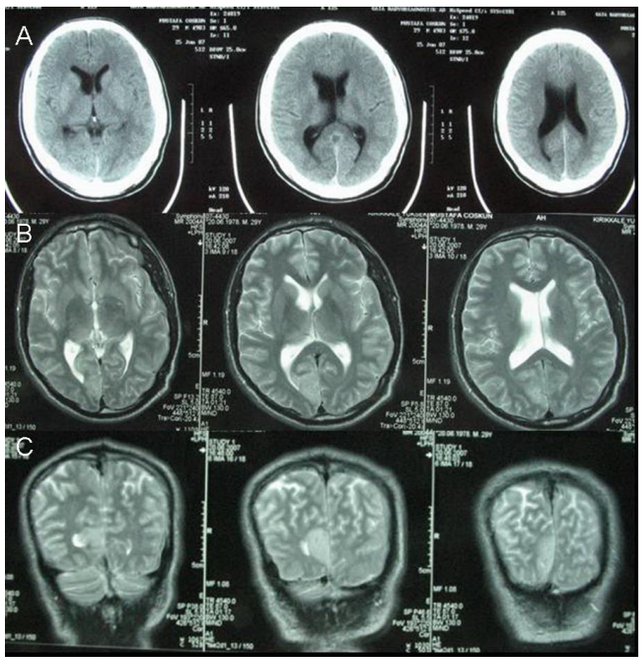


Figure 1. Normal brain CT scan, performed in a local hospital in the first 24 hours of trauma(A). Axial and coronal T2 weighed brain MR images revealing right occipital ischemia (B,C).

was discharged after 24 hours of observation. Two days after his discharge, he had generalized tonic-clonic seizure and was admitted to our department. His neurological examination revealed left hemianopia. After observing occipital subacute ischemia at right side in brain magnetic resonance imaging (MRI), we performed cerebral angiography to detect possible craniocervical artery dissection (Figure 1B-1C). Main cervical and cerebral arteries were normal and no dissection was observed. We performed rheumatologic, cardiologic, vascular screening tests for explaining a coincidental cerebral ischemia due to other causes but all resulted in normal limits. Our final diagnosis was late onset cerebral ischemia due to whiplash injury. Anti-edema (IV mannitol %20) and antiepileptic (carbamazepine 800 mg/day PO) treatment was initiated. He was discharged with left hemianopia and mild cognitive deficit (Mini mental state examination: 27/30 points).

Discussion

Traumatic brain damage causes important mortality and morbidity rates at all ages [5]. Artery-to-artery emboli or occlusion of craniocervical arteries mostly due to dissection are the most common causes of ischemia after trauma. In our case cerebral angiography revealed no dissection or occlusion.

Infarction of the occipital lobe after compression of the PCA against the rigid edge of the tentorium by the herniating medial temporal lobe is the most well recognized mechanism leading to cerebral infarction. In a study evaluating the neuroimaging findings, etiology and outcome of patients with post-traumatic cerebral infarction, it has been shown that focal mass effect and acquired intracranial herniations accounted for infarction in 81.2% of cases, with the PCA territory as the most common location. This study revealed the mortality of patients with craniocerebral trauma complicated by post-traumatic cerebral infarction was 43.8%, and suggested that post-traumatic cerebral infarction is an indication of a poor clinical outcome [4]. As post-traumatic cerebral infarction may lead to life-threating problems, all patients with cerebral trauma should be monitorized closely.

Posttraumatic epilepsy is an another issue and the most common known complication of traumatic brain damage. Although seizures are frequent, prophylactic antiepileptic treatment is not recommended in post-traumatic patients routinely. The treatment must be initiated after an epileptic seizure [6]. Post-traumatic brain damage may not develop clinical or imaging signs in early periods of trauma. After trauma, ischemia and microvascular circulation disorders due to membrane dysfunction and edema (vasogenic and cytotoxic) may be seen. Gowda et al. showed that 70% of the patients had regional hypo-perfusion in single photon emission tomography scanning although having normal brain CT scans after 48-72 hours of trauma [2]. Preferred first line therapy is anti-edema medication to overcome vasogenic and cytotoxic edema. In previous case reports, mannitol and dextran was applied. We also initiated mannitol and carbamazepine therapy in our patient.

As a result we must keep in mind that post-traumatic brain damage may be caused not only by direct head trauma but also by concussion. Ischaemic changes may not be shown in brain CT in early periods but regional hypo-perfusion due to membrane dysfunction may develop in late terms of trauma. We suggest that it will be wise to hospitalize patients for at least 72 hours who has a history of unconsciousness following trauma as opposed to our classical knowledge which 24 hours of observation is sufficient.

Competing interests

The authors declare that they have no competing interests.

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