Cerebral Fat Embolism Diagnosed by Cognitive Disorder

Kognitif Bozuklukla Prezente Olan Serebral Yağ Embolisi

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Abstract

Fat embolism syndrome is a rarely seen complication of skeletal trauma, and it is seen at a rate of 2-5% after fractures of the long bones of the lower extremities. Its classic triad consists of hypoxemia, petechial bleedings on the skin and neurological findings. These neurological findings are highly variable and non-specific, and they can present with lethargy, irritability, delirium, stupor, or coma. In this report, a male case is presented who was diagnosed with cerebral embolism due to acute cognitive disorder after a segmental tibial fracture.

Key Words: Cerebral fat embolism, tibial fracture, acute cognitive disorder, diffusion-weighted MRI

Özet


Anahtar Kelimeler: Serebral yağ embolisi, tibiyal fraktür, akut kognitif bozukluk, difüzyon ağırlıklı MRG

Introduction

Neurological involvement in fat embolism syndrome is called cerebral fat embolism. Cerebral fat embolism is an uncommon but serious complication of long-bone fractures. These neurological findings have been postulated to be due to several mechanisms, such as the occlusion of brain blood vessels by a fat embolism, impairment of the blood-brain barrier due to free fatty acids, or the obstruction induced by the dissolution of fat in the blood secondary to the fracture, etc. [1, 2]. For the diagnosis of cerebral embolism, the presence of multiple, punctate hyperintensities is visualized in diffusion-weighted magnetic resonans imaging (MRI) [3, 4]. In this report, a male case is described who was diagnosed with a cerebral embolism due to acute cognitive disorder after segmental tibial fracture.

Case Report

A 60 year-old male patient was hospitalized in the orthopedics clinic with a tibial fracture. He consulted with us after complaints of inability to recognize his relatives, mental confusion, and unease that developed within 12 hours after the fracture. In his neurological examination, there was a slight somnolence, limited cooperation, disorientation in terms of person and place, and bilateral sole skin extensor response. The cranial system and fundus oculi examinations were normal, there was no neck stiffness, and painful stimulation was localized in each of the four extremities. A suspected potential brain contussio was excluded with a normal cranial tomography (CT) (Figure 1), and then an urgent cranial and diffusion MRI was planned. In the cranial MRI, multiple hyperintense areas were detected in diffused T2-weighted and fluid attenuated inversion recovery (FLAIR) images in the bilateral centrum semiovale, both frontal and occipital lobes, left cerebellar hemisphere, and deep and subcortical white matter (Figure 2a-c). Diffusion MRI apparent diffusion coefficient (ADC) and B1000 sections were compatible with acute phase infarct (Figure 3a-c). There were no other clinical symptoms or findings associated with fat embolism syndrome. The case was diagnosed with cerebral fat embolism based upon the finding that there was no latent period between the neurological findings and trauma development and there was no head trauma or typical temporary neuroimaging findings. The case was followed by our clinic during the post-operative period with anti-aggregate and low molecular weight hepa-
A neurological examination and mini-mental test were normal during the control at the polyclinic after two months. In this case, acute recovery was characterized.

**Discussion**

A fat embolism typically emerges within 12-72 hours following a trauma. In our case, the cognitive disorder occurred after 12 hours. Neurological symptoms can range from subclinical presentation to confusion, coma and seizures, and may rarely lead to death. Although neurological symptoms are typically accompanied by respiratory failure and skin eruptions, isolated cerebral fat embolism cases have been reported in several instances [1, 2, 5]. In our case, a detailed respiratory system and skin examination were normal. The findings matched with an isolated cerebral fat embolism. An isolated cerebral fat embolism may create diagnostic dif-

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**Figure 1.** Normal CT.

**Figure 2.** a-c. Multiple hyperintense areas shown in diffused T2-weighted and T2-Flair images in cranial MRI sections.

**Figure 3.** a-c. Diffusion MRI ADC and B1000 sections showed acute phase infarct.
Difficulty, and the use of magnetic resonance imaging findings for the brain may contribute to the diagnosis [6-8].

In our case, there was limited diffusion on the ADC map in the diffusion weighted imaging (DWI) that we performed when mental confusion developed at the beginning of the post-operative period. We ruled in favor of cerebral fat embolism as the case had no neurological findings at emergency service, but had an occurrence of mental confusion after the tibial fracture. Hyperintense punctate lesions in the DWI in MRI are most distinctively monitored at the level of the centrum semiovale. This was a rare case that presented with an acute cognitive disorder that we found worthwhile to report.

**Informed Consent:** Written informed consent was obtained from the patient who participated in this case.

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


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**References**