

# Diffusion Weighted MRI May be a Life Saving Tool in Cerebral Fat Embolism: Report of a Case

## Difüzyon MRG Serebral Yağ Embolisinde Hayat Kurtarıcı Araçtır: Olgu Sunumu

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Cerebral fat embolism syndrome is a rare, but potentially lethal, complication of long bone fractures. Neurological symptoms are variable, the clinical diagnosis is difficult and occurs in only 0.9-2.2% of these cases. A 19 year-old male with no head injury suffered 3 epileptic seizures 17 hours after left femoral shaft and left tibia fractures. He had hypoxia. Chest x-ray and thoracic computed tomography (CT) were normal. T2 and diffusion weighted magnetic resonance imaging (MRI) showed multiple hyperintensity within the bilateral basal ganglia. We thought that this indicated areas with multiple microemboli. He was intubated after clinical deterioration. Heparin, acetylsalicylic acid, mannitol and corticosteroids were administered, he was operated on next morning for left femoral shaft and left tibia fractures and he showed neurological improvement after 24 hours. Diffusion weighted MRI of the brain should become the first step in the diagnostic algorithm of cerebral fat embolism and patients have neurological improvement with early diagnosis and appropriate management.

**Key Words:** Diffusion MRI, fat embolism, epilepsy, bone fractures

Serebral Yağ Emboli Sendromu; uzun kemik kırıklarına bağlı ortaya çıkan nadir ve öldürücü bir komplikasyondur. Nörolojik semptomlar değişken olup klinik tanısı zor ve hastaların %0,9-2,2 sinde görülür. Burada 19 yaşında kafa travması olmaksızın sol femoral shaft ve tibia kırıkları olan, travma sonrası 17. saatte 3 kez epileptik nöbeti olan erkek hasta sunulmuştur. Hastanın hipokisi mevcuttu. Çekirilen PA-AC grafisi ve AC tomografisi normaldi. T2 ve Difüzyon MRG'lerde multipl bilateral bazal ganglionlarda hiperintens lezyonlar saptandı. Bu görünümün multipl mikroembolilere bağlı olduğunu düşündük. Hasta klinik kötüleşme sonrası entübe edildi. Hastaya heparin, asetilsalisilik asit, mannitol ve kortikosteroid uygulandı. Ertesi gün sol femoral shaft kırığı ve tibia kırığı için opere edilen hastada klinik düzelmeye görüldü. Difüzyon MRG Serebral Yağ Embolisi tanı algoritminde ilk basamakta düşünülmelidir. Bu tür hastalar erken tanı ve uygun tedavi ile nörolojik düzelmeye göstermektedir.

**Anahtar Kelimeler:** Difüzyon MRG, yağ embolisi, epilepsi, kemik kırıkları

### Introduction

Fat embolism syndrome (FES) is characterized with pulmonary insufficiency and hypoxemia, neurologic dysfunction and petechia, which develop after trauma, especially with fractures of long bones (1). FES typically manifests within 24 to 72 hours after trauma (2). Incidence of cerebral fat embolism may be as low as 0.9% to 2.2% in long bone fractures (3). Mortality associated with fat embolism has been reported to be 13%-87%; however, this rate has decreased with recent progresses in respiratory and intensive care (4). Magnetic Resonance Imaging (MRI) has been shown to be the most sensitive means of diagnosing cerebral fat embolism and the typical findings of multiple punctate hyper intensities on T2-weighted and diffusion-weighted imaging (5).

### Case Report

A 19-year-old male was brought to the Nevşehir State Hospital, Emergency Department after a motor vehicle accident. He had sustained left femoral shaft and left tibia fractures (Figure 1a, b). He did not lose consciousness and had no head trauma. On arrival at the hospital, his blood pressure was 110/60 mmHg and he was conscious with a Glasgow Coma Score (GCS) of 15.

Low molecular-weight heparin was initiated for thromboembolism prophylaxis. At the 17<sup>th</sup> hour of his hospitalization, he had tonic clonic seizures. In the following two hours, he had three epileptic seizures. He was disoriented and sleepy. The arterial blood gas analysis while breathing 0.40 oxygen by a face mask showed a pH of 7.50, partial pressure of carbon dioxide (PCO<sub>2</sub>) 21.50 mmHg, partial pressure of oxygen (PO<sub>2</sub>) 57.90 mmHg, saturation of oxygen 91%. He was tachypneic and tachycardic. He was intubated and connected to a mechanical ventilator. His blood glucose, urea, nitrogen, serum electrolytes and results of liver functions tests were within normal limits. There was no petechia on his torso. Heparin and acetylsalicylic acid was administered to treat a possible pulmonary embolism. Chest x-ray and computed tomography of the brain and thorax was obtained because of hypoxemia and seizures. These imaging tests came back as normal transthoracic echocardiography with bubble study showed no cardiac abnormalities and revealed no embolic sources.

Cranial MRI and diffusion MRI were performed for differential diagnosis. Cranial T2 weighted MRI and diffusion MRI showed multiple hyperintense punctuate lesions within the bilateral basal ganglia (Figure 2).

The diagnosis of cerebral fat embolism was confirmed with MRI, and mannitol and glucocorticoids were started. The next morning, the patient was transferred to the operating room for open reduction and internal fixation of the fractures of left femoral shaft and left tibia. By the first postoperative day, there was evidence of neurologic improvement. He could open his eyes spontaneously and move his limbs on command. He was weaned from the ventilator and the endotracheal tube was removed. There were no neurological deficit but he had amnesia. The control diffusion MRI after 27 days, showed resolution of hyper intense foci (Figure 3)

## Discussion

Cerebral fat embolism syndrome consists of the triad of acute respiratory distress with hypoxemia, cutaneous petechial hemorrhages and variable neurologic dysfunction. Minor criteria are tachycardia, pyrexia, thrombocytopenia and anemia (4,5). Deterioration of neurological status can change from confusion to encephalopathy with coma and seizures (3). The patient had neurological dysfunction after 3 attacks of epileptic seizures and had hypoxemia. His PaCO<sub>2</sub> was 21.50 mm Hg, PaO<sub>2</sub> was 57.90 mm Hg, and saturation was 91% while breathing 0.4 oxygen by a facemask. He also had tachy-

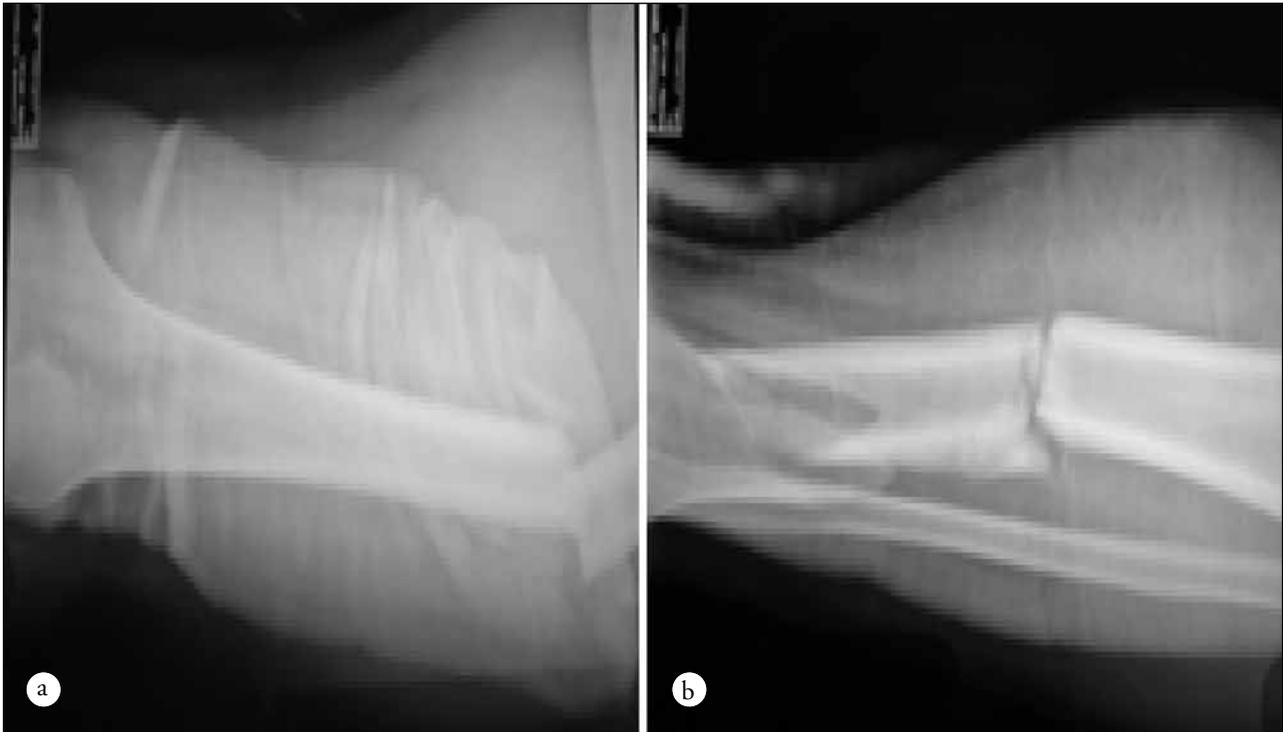


Figure 1a, b. Left femoral shaft fracture and left tibia fracture

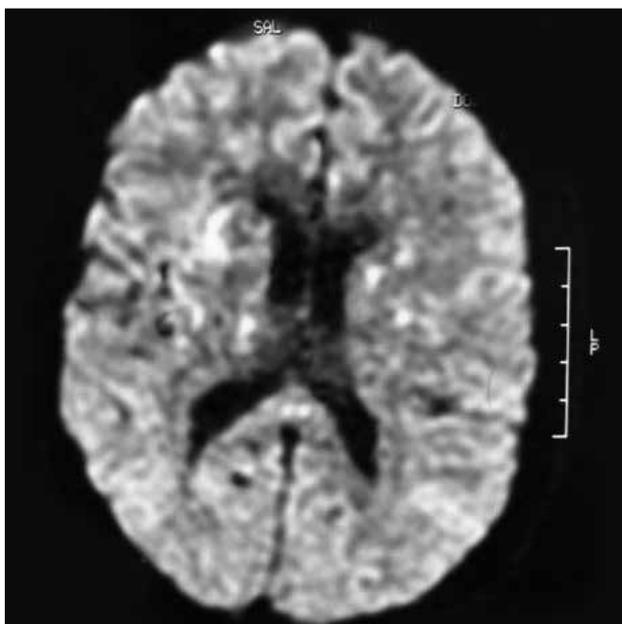


Figure 2. Diffusion MRI showed multiple bilateral hyper intense punctate lesions within the bilateral basal ganglia

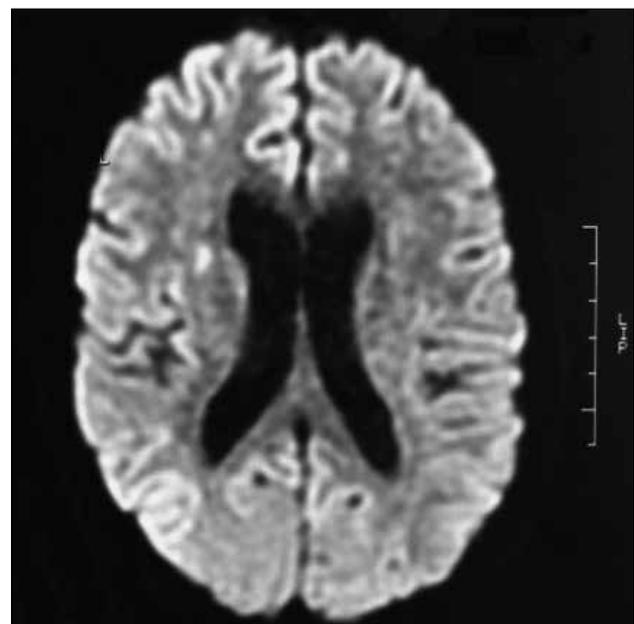


Figure 3. Control diffusion MRI showed resolution of hyper intensity within the bilateral basal ganglia

pnea and tachycardia. On the other hand, he had no petechial hemorrhages or pyrexia and all laboratory tests were normal.

There are two theories explaining the pathogenesis of fat embolism syndrome. According to the mechanical theory, free fat particles from the bone marrow enter vein sinusoids at the site of the fracture and reach the pulmonary arterioles. The chemical theory suggests that fat emboli arise from plasma fat through some kind of systemic stimulus associated with trauma and other medical conditions. As a result of this stimulus, chylomicrons coalesce and fuse each other to form larger fat globules (1, 5- 7). The mechanical theory suggests that intracardiac shunts such as patent foramen ovale, ventricular septal defects, pulmonary arteriovascular malformations and persistent truncus arteriosus are responsible for the passage of fat particles to the cerebral circulation, resulting in neurological symptoms (7). The manifestation of this patient is compatible with the chemical theory because his transthoracic echocardiography with bubble study showed no cardiac abnormalities and revealed no embolic sources. The patient was hypoxemic after epileptic seizure. His hypoxemia could be due to pulmonary micro embolism, but chest X-ray and computed tomography of the thorax showed no signs of fat embolism syndrome.

Radiological findings are useful for diagnosis. Chest X-ray shows diffuse bilateral infiltrates in fat embolism syndrome (8,9). Cranial CT shows normal findings in most cases. Cranial MRI is more sensitive than CT imaging. T2 weighted and diffusion weighted MRI shows small hyper intense punctate lesions in the cerebral white matter, cerebellum and brain stem (3, 5-7). In this patient, cranial CT and cranial MRI studies were performed. Cranial CT was also normal but, T2 weighted and diffusion weighted MRI showed lesions compatible with FES in bilateral basal ganglia.

The differential diagnosis of disseminated hyperintense lesions on T2-weighted scans includes diffuse axonal injury, areas of vasogenic edema, micro infarcts, and foci of gliosis, dilated perivascular Virchow-Robin spaces and demyelinating disease (10). Some authors reported that the lesions in diffusion MRI disappear by the end of the subsequent week (3-5). Patient's cranial CT showed no signs of hematoma and contusion. For differential diagnosis, we planned a cranial MRI and diffusion MRI. T2 weighted and diffusion MRI showed multiple hyperintense spots within the bilateral basal ganglia. According to the clinical and radiological findings, we decided that cerebral fat embolism syndrome was the final diagnosis for the present clinical picture. In this patient, control diffusion MRI, which was performed after 27 days, revealed resolution of the previous hyperintense lesions.

Treatment of fat embolism syndrome consists of preserving good arterial oxygenation. Mechanical ventilation and positive end expiratory pressure can be used to maintain oxygenation. Also, intravascular volume is important in the prevention of lung injury and albumin is used generally. It restores blood volume and protects the lung by binding with fatty acids. This patient was also intubated and connected to a mechanical ventilator (11).

Kim et al. (7) reported that the use of a tourniquet can be effective in the prophylaxis of fat embolism. Rigid immobilization or early

surgical intervention for fractures can reduce the risk of fat embolism syndrome. Pharmacologic agents have also been used for fat embolism syndrome treatment. Heparin has lipolytic effects, dextrans have a salutary effect on the microcirculation, and corticosteroids and acetylsalicylic acid have an anti-inflammatory action (1). Although low molecular-weight heparin was applied as prophylactic treatment, we did not use a tourniquet in this patient, open reduction and internal fixation could not be performed earlier due to the operating room's schedule. In this patient, cerebral fat embolism syndrome developed after 17 hours of hospitalization, and he was intubated after clinical deterioration. Heparin, acetylsalicylic acid, mannitol and corticosteroids were administered, and he was operated on the following morning for left femoral shaft and left tibia fractures. He showed neurological improvement after 24 hours.

## Conclusion

Cerebral fat embolism should be considered in non-head injured patients who are initially lucid and develop acute mental status deterioration. Diffusion MRI should be included in the diagnostic algorithm. Heparin, dextran, corticosteroids and acetylsalicylic acid can be used in the treatment of cerebral fat embolism syndrome. Prophylaxis using a tourniquet, and low molecular-weight heparin may be used to reduce cerebral fat embolism in patients with long bone fractures.

## Conflict of Interest

No conflict of interest was declared by the authors.

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