CASE REPORT

Motor Aphasia as a Rare Presentation of Fat Embolism Syndrome; A Case Report

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Received: 16 August 2014 *Accepted: 4 December 2014*

Abstract

Fat embolism syndrome is a clinical diagnosis, and diagnostic procedures are not specific. In every trauma patient, Fat embolism syndrome has to be considered as a possibility and supportive treatment should begin as soon as possible. The authors reported a rare case of Fat embolism syndrome whose only neurological symptom was motor aphasia. A young man sustained comminuted femoral shaft fracture following an accident presented dyspnea, motor aphasia and petechial rash. The Po2 and O2 Saturation were 53 and 91.1%. The body temperature was 38.5 °C. The hemoglobin decreased from 12.9 to 8.7 and platelet from 121000 to 84000 mg/dl. The pulse rate was 120 bpm. The CT scan and MRI were normal. Fat embolism syndrome was diagnosed according to both Gurd and Schonfeld criteria ruling out other possible causes. Patient recovered completely. Although rare, focal neurological symptoms and motor aphasia should be kept in mind as a part of diagnostic criteria.

Key words: Broca Aphasia, Fat embolism, Femoral fractures

Introduction

Fat embolism syndrome (FES) is a consequence of fat emboli in general circulation, which may complicate long bone and pelvic fractures. Almost all patients with long bone fractures present with transient hypoxemia in pulse oximetry but not with a symptomatic fat embolism syndrome (FES) (1). The incidence of FES has been reported to be 0.9% to 11%. With supportive treatment and early operative fixation, mortality rate after FES is 7% to 10% (2). Pulmonary system is affected more often than other organs and neurological symptoms occur most commonly after pulmonary symptoms; FES with focal neurological symptoms alone is an uncommon event (3-6).

We present a case of FES following femoral shaft fracture in a young adult in whom the only neurologic symptom was motor aphasia. To our knowledge, this is the third report of motor aphasia presenting as a neurologic symptom of FES after femoral shaft fracture. The purpose of this study is to emphasize that although rare, awareness of FES in the trauma patients can limit potential mortality with early supportive treatment.

Case report

The patient was a 24 year-old male having a traffic accident and was referred to Shahid Rahnemoun Hospital -- an educational hospital affiliated to Shahid Sadoughi University of Medical Sciences, Yazd, Iran -- four hours after the accident. His vital signs were stable at the time of admission and his Glasgow coma scale (GCS) was 15. He had head trauma and transient amnesia without nausea or vomiting. The history was negative for drug abuse. He had tenderness and deformity in his left femur and also tenderness in the right upper quadrant of his abdomen. X-rays revealed a comminuted left femur fracture. Routine laboratory tests were normal except urine analysis showing blood inside. Abdominopelvic sonography was normal. Skeletal traction was applied. The patient had dyspnea and sweating in the second day. He was conscious but unable to talk. He could reply only by his body movement and was agitated. He had a body temperature of 38.5 degrees Celcius, pulse rate of 120, and respiratory rate of 30. In the arterial blood gas (ABG) test Po2 was 53 and O2 Saturation was 91%. The patient was transferred to the ICU. After O2 therapy with 30 degrees tilt up of the head, O2 saturation raised to 98%. Complete blood count (CBC) showed a decrease in hemoglobin level from 13 to 8.7 mg/dl and platelet from 121,000 to 84,000 mg/dl. Neurologic exams including cranial motor and sensory examinations were

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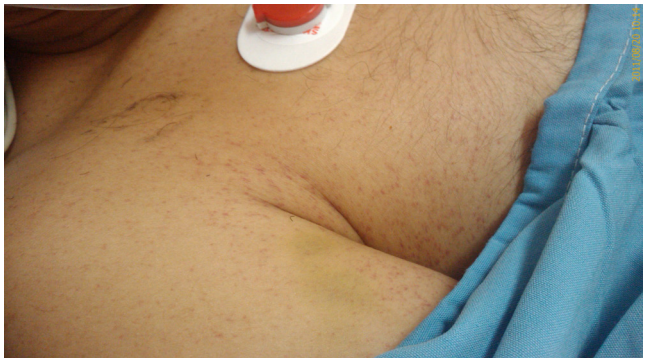

Figure 1. Petechial Rash over the axillary region.

normal except for motor aphasia. Chest x-rays and brain CT-scan were normal. Red petechial skin rashes over the chest and neck appeared gradually in the next few hours [Figure 1]. FES was diagnosed according to Gurd and Schonfeld criteria after ruling out for other possible diagnoses (7, 8). Supportive treatment was continued. The patient received packed cells, enoxaparin, cefazolin, and morphine and midazolam. We had a speech therapist working with the patient. Aphasia and tachypnea improved and disappeared on day seven. The patient underwent bone fixation five days after recovery from aphasia and was discharged 3 days after surgery. Brain MRI was normal on day 15.

Discussion

This patient had a rare presentation of FES following femur fracture. Motor aphasia with normal consciousness was the only neurological presentation along with respiratory symptoms and characteristic petechial rash. This patient had transient post-traumatic amnesia; head trauma was ruled out with further normal brain CT-scan and neurological exam. The patient was diagnosed with FES based on both Schonfeld and Gurd criteria ruling out other possible causes (7, 8).

Zenker published the first report of FES in a patient after an accident (9). Fat globules entering into multiple organs can cause specific symptoms. Patients with FES usually present with pulmonary symptoms, neurologic dysfunction, and petechial rashes. Anemia, coagulopathy, lipiduria, retinopathy, tachycardia, and pyrexia can accompany with FES (7). Pulmonary symptoms vary from hypoxia to acute respiratory distress syndrome (ARDS). Neurologic symptoms of FES may range from dementia and confusion to coma. Focal neurological symptoms subsequent to fat emboli including anisocoria, apraxia, hemiplegia, tetraplegia, aphasia, and partial seizure have been reported as well (3-5).

In case of presenting with focal symptoms, scanning of the brain seems necessary although it is usually normal in FES (3). Magnetic resonance imaging (MRI) with diffusion-weighted sequences is the most sensitive technique for the detection of FES. It shows multiple high intensity and non confluent injuries in the white and gray matter (10). MRI in FES may show hyperintense areas in the first four hours of fat emboli that will last for two weeks as the symptoms disappear. However, normal MRI does not rule out previous injury or FES (11). High density signals on T2 MRI are signs of neurological symptoms (12). Ishihara et al and Gupta et al also reported patients with motor aphasia after fracture with normal consciousness (6, 13). Both patients' MRI showed high density signals on T2 weighted images; but the MRI in our patient was negative. Nevertheless, we performed the MRI after aphasia had been disappeared.

Finally, the diagnosis of FES is based on ruling out any other possible diagnoses according to the Gurd criteria - having two major criteria or one major plus four minor criteria - or Schonfeld criteria with at least five scores (7, 8).

Early oxygen therapy decreases the effect of hypoxemia (1). Drugs including steroids, heparin, and dextran are used, but the evidence is not clear to have a specific effect. The prognosis is usually good, and most patients fully recover with no remaining sequel (2). In our patient, hypoxemia improved with supportive treatment, and motor aphasia improved gradually in one week. However, our investigation was limited due to the lack of adequate MRI facilities in the hospital and also due to the instability of the patient's femur fracture.

On summary, when we are confronting FES, focal neurological symptoms including motor aphasia although rare, should be considered as the neurologic part of diagnostic criteria.

Acknowledgement

The authors would like to acknowledge Mr. Michal Kamionko, for his assistance in editing the manuscript.

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