Fat embolism syndrome after lower extremity replantation associated with tibia fracture: case report

Tibia kırığı ile birlikte olan alt ekstremite replantasyonu sonrası saptanan yağ embolisi sendromu: Olgu sunumu

Burçak TÜMERDEM,[1] Defne ÖNEL,[1]
Murat TOPALAN,[1] Şefika KÖRPINAR,[2] Şamil AKTAŞ[2]

We report a patient who was diagnosed as fat embolism syndrome after replantation surgery of left amputated foot. This diagnosis was based on the presence of a long bone fracture with an amputation of a major extremity, supported by the signs of pulmonary and cerebral dysfunction and confirmed by the demonstration of arterial hypoxemia in the absence of other disorders.

Key Words: Fat embolism; lower extremity; replantation.

Ampute sol ayak için gerçekleştirilen replantasyon ameliyatı sonrası yağ embolisi sendromu düşünülen olgu sunuldu. Ameliyat sonrası pulmoner ve serebral fonksiyon bozuklukları gelişen, majör ekstremite amputasyonuyla birlikte tibia kırığı olan olguda arteriyel hipoksemiyi destekleyecek herhangi bir patoloji saptanmaması bu tanımızı güçlendirmektedir.

Anahtar Sözcükler: Alt ekstremite; replantasyon; yağ embolisi.

The number of lower limb amputation cases is increasing, due to high-energy trauma in road accidents and work-related injuries. Lower limb salvage procedures may cause general or local complications such as necrosis, infections, nonunions, and they may need secondary lengthening, or other reconstructive procedure.[1] Traumatic fat embolism syndrome occurs most often following fractures of long bones.

We report a case of foot replantation with tibia fracture who was diagnosed as fat embolism syndrome postoperatively.

CASE REPORT

A twenty-eight-year-old male patient referred to emergency department as his right foot was amputated at the ankle level due to work accident. While he was working at a construction area, the wire of a machine which was wrapped around his right ankle amputated his foot. He was taken to the operation four hours after the accident. The radiologic view of the injured extremity showed tibia fracture (Fig. 1). An external fixator was applied for the tibia fracture. Two K-wires were used for the fixation of the amputate to the leg. The foot was replantated successfully. The operation lasted for 10 hours. Following extubation persistent unconsciousness developed. The neurological examination and cranial BT were normal. The ECG showed sinus tachycardia of 120 beats/min. He developed a fever of 38.5°C and had tachypnea. His blood count was normal except a mild anemia. A diagnosis of pulmonary emboli was considered. Arterial blood gas readings and a chest radiograph were obtained.

Departments of [1]Plastic and Reconstructive Surgery and [2]Underwater and Hyperbaric Medicine, Istanbul University, Istanbul Faculty of Medicine, Istanbul, Turkey.

İstanbul Üniversitesi İstanbul Tıp Fakültesi, [1]Plastik ve Rekonstrüktif Cerrahi Anabilim Dalı, [2]Deniz, Sualtı ve Hiberbarik Anabilim Dalı, İstanbul.

Correspondence *(İletişim):* Burçak Tümerdem, M.D. Ataköy 9. kısım B1 blok, D: 56, 34750 Ataköy, İstanbul, Turkey.
Tel: +90 - 216 - 399 97 50 / 2145 Fax *(Faks)*: +90 - 216 - 345 89 32 e-mail *(e-posta):* burcaktumerdem@yahoo.com

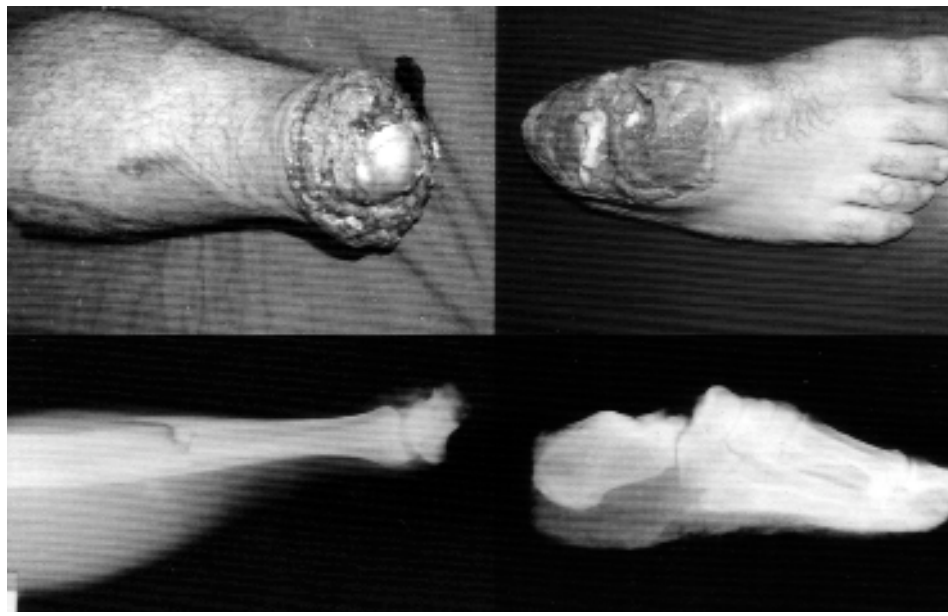

Fig. 1. The view of right foot amputated at the ankle level and the radiologic view of the lower extremity showing tibia fracture.

Chest radiograph showed indefinite perihilar fullness without infiltration. Arterial blood gas findings were pH 7.45, carbondioxide 40 mmHg and oxygen 84 mmHg with 90% saturation. Serial arterial blood gas measurements revealed persistent hypoxemia.

The patient was admitted to the intensive care and was supported with oxygen therapy and ventilation. He was given heparin. Pulmonary and cerebral symptoms resolved in 6 hours. He required a transfusion of 3 U of packed red blood cells. His high-grade temperature resolved and his blood pressure remained stable. The oxygen saturations improved. The general health status of the patient made a good progress.

On the dorsolateral side of the foot a 5 x 8 cm of skin necrosis developed. After the debridement of the skin necrosis on the 7th day, hyperbaric oxygen (HBO) therapy was started. The edema of the replanted foot subsided and the skin defect became granulated after 10 sessions. A split thickness skin graft was applied to the defect successfully (Fig. 2). HBO therapy continued as a pin-tract infection and osteomyelitis at the fracture site occured in the following days. Infection was under control with appropriate antibiotherapy, HBO therapy and removal of the external fixator. The patient is still under follow-up of orthopaedics department.

DISCUSSION

Fat embolism syndrome remains a rare, but potentially life threatening complication of long bone fractures. The true incidence is difficult to assess as many cases remain undiagnosed. Cerebral involvement varies from confusion to encephalopathy with coma and seizures.[2] Fat embolism is a clinical entity and its diagnosis can be made on the basis of the symptom complex. Hypoxia, anemia, disturbances of consciousness and hyperpyrexia are

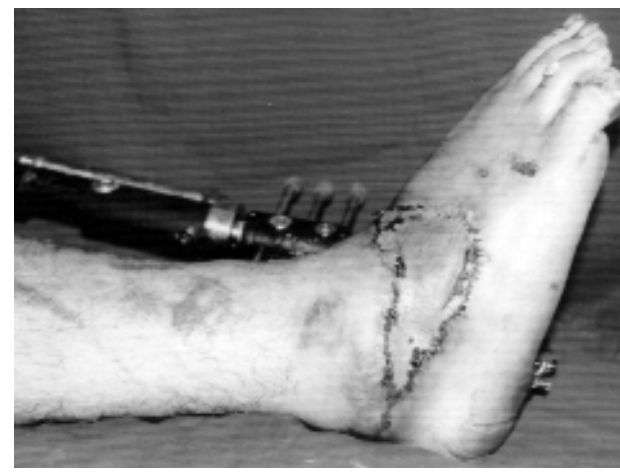

Fig. 2. Postoperative view of the lower extremity after successful replantation and skin grafting of the skin defect on the dorsolateral side of the foot.

constant signs.[3-5] Clinical diagnosis is essential, as laboratory and radiographic findings are nonspecific.[6] Treatment consists of general supportive measures including splinting, maintenance of fluid and electrolyte, the administration of oxygen. Endotracheal intubation can be indicated. The role of corticosteroids remains controversial.[7]

A review of our patient's symptoms indicated that he met three of Gurd and Wilson's criteria for fat embolism syndrome, including hypoxemia, tachycardia and pyrexia.[8] Therefore the patient was diagnosed as fat embolism syndrome. This diagnosis was based on the presence of a long bone fracture with an amputation of a major extremity, supported by the signs of pulmonary and cerebral dysfunction and confirmed by the demonstration of arterial hypoxemia in the absence of other disorders.

In literature there is not a case of fat embolism syndrome associated with major limb extremity amputation. It is mostly described after orthopaedic surgeries. Our case also had tibia fracture with the amputation of the foot. In this report, we would like to take attention to "fat embolism syndrome" which should be considered in differential diagnosis of respiratory and cerebral symptoms that occurs after a lower extremity replantation of a long bone fracture.

REFERENCES

1. Battiston B, Tos P, Pontini I, Ferrero S. Lower limb replantations: indications and a new scoring system. Microsurgery 2002;22:187-92.
2. Gregorakos L, Sakayianni K, Hroni D, Harizopoulou V, Markou N, Georgiadou F, et al. Prolonged coma due to cerebral fat embolism: report of two cases. J Accid Emerg Med 2000;17:144-6.
3. Mellor A, Soni N. Fat embolism. Anaesthesia 2001;56:145-54.
4. Lepistö P, Alho A. Diagnostic features of the fat embolism syndrome. Acta Chir Scand 1975;141:245-50.
5. Glazer JL, Onion DK. Fat embolism syndrome in a surgical patient. J Am Board Fam Pract 2001;14:310-3.
6. Parisi DM, Koval K, Egol K. Fat embolism syndrome. Am J Orthop 2002;31:507-12.
7. Richards RR. Fat embolism syndrome. Can J Surg 1997;40:334-9.
8. Gurd AR, Wilson RI. The fat embolism syndrome. J Bone Joint Surg [Br] 1974;56:408-16.