# FAT EMBOLISM SYNDROME

(Case Report and Review of Current Concepts)

O. Güven\*

\* Assistant Professor, Department of Orthopedics and Traumatology, Faculty of Medicine, Marmara University, İstanbul, Turkey

## SUMMARY

The fat embolism syndrome is a relatively rare complication of musculoskeletal trauma, in contrast to subclinical fat embolization which occurs after nearly all of the long bone fractures. In this report two patients with fat embolism syndrome have been presented.

Key Words: Fat. embolism.

### INTRODUCTION

Fat embolism syndrome (FES) may be defined as a complex alteration of homeostasis which occurs as an infrequent complication of fractures of the pelvis and long bones and manifests itself clinically as acute respiratory insufficiency.

The clinical syndrome is evident in 0.5 % to 2 % of long bone fractures and approaches to a 5 - 10 % incidence in multiple fractures associated with pelvic injuries (1, 2).

Subclinical fat embolization occurs after almost all long bone fractures and it can be recognized by arterial blood gas analysis. The major end organs involved are the lungs and the nervous system. The kidneys may also be involved infrequently (1, 3, 4). Fat macroglobules impair small vessel perfusion and endothelial damage in the pulmonary capillary leads to ventilation/perfusion imbalance. Vascular congestion, interstitial hemorrhage, alveolar wall damage and airway collapse occur subsequently. Pulmonary gas exchange is impaired as a result of these changes (3, 5, 6).

Nontraumatic fat embolization can be seen infrequently (7, 8, 9, 10).

#### CASE REPORT

251 patients with bone fractures were admitted to Alanya State Hospital in two years period between 1984 - 1986. 132 of them were treated surgically. 35 of them had multiple fractures and two of these patients experienced FES clinically.

CASE I- A 27-year-old man was admitted to hospital after a traffic accident. He had a femoral and open tibial fracture on left side. (Fig. 1 - 2) Debridement and irrigation were applied to the open fracture of the tibia and it was immobilized with cast. Skeletal traction was applied from the tibial tubercle to the left leg. He was in hypovolemic shock when first seen and recovered by i.v. fluid infusion and blood transfusion. He suddenly got worse 24 hours later. Deterioration, tachypnea, tachycardia, restlessness and pyrexia (39,4 °C) arised subsequently. After a few hours, he became unconscious and urinary incontinence occured. Nearly 30 hours later petechiae appeared at the both conjuctivas and sclerae. (Fig. 3) Arterial blood pressure was 220/110 mm Hg and daily urine out put was 600 cc.



Fig. 1-A femoral fracture on left side

Laboratory findings: Hb, 11g %; Leucocytes, 10800/mm<sup>3</sup>; BUN, 28 mg/dl and platelet count was 150000/mm<sup>3</sup>. Fat globules could not be demonstrated in the urine. Arterial  $PO_2$  level dropped significantly to 50 mm Hg suggesting FES.

Oxygen inhalation and i.v. methyl prednisolon were used for the treatment of FES and the patient recovered rapidly. Antibiotics were given intravenously. This serious state lasted for 10 days. Open reduction and internal fixation was performed at the end of the 4 th week and the patient was discharged uneventfully.



Fig. 2- An open tibial fracture on left side.

CASE 2 - A 23-year-old man was admitted to hospital after a traffic accident. He had bilateral femoral fractures and open fractures of the right radius and ulna. Bilateral skeletal traction was performed from the both proximal tibial tubercles. Debridement was performed to the open fractures of the right forearm which was immobilized with cast subsequently. Tachypnea, tachycardia, restlessness and pyrexia were seen at the 14th hour.

Laboratory findings: Hb, 13 g%; Hct, 39 %; BUN, 49 g/dl; and the platelet count was  $160000/mm^3$ . Microscopic hematuria was also detected.

Supportive therapy was applied. Nasal oxygen, i.v. fluids (Rheomacrodex, isotonic....),heparin and i.v. methylprednisolon were given. This state went on for one week and the patient recovered consequently. Open reduction and internal fixation was performed to the both femoral fractures three weeks later.



Fig. 3- Mucous membranes can be affected in FES. Petechiae are seen along the mucosal margin of the left lower eyelid and sclera.

# DISCUSSION

Zenker (1862) described the features of the fat embolism in the pulmonary tissues of persons dying following injury. Bergmann (1873) made the first description of clinical findings following long bone fractures such as confusion, dyspnea and petechiae.

Fenger and Salisbury (1879) presented the first American experience of the pathological process. The management of fat embolism was first described byHermann (1932) using a solution of 5 % ethyl alcohol and 5 % dextrose.

Although the condition has been documented for over a century, the pathogenesis of fat embolism still remains obscure.

Typical symptoms are evident in the first 24 - 72 hours in FES. Positive laboratory findings may be suggestive earlier. (1, 3, 11, 12) Gurd's (1970)(13) criteria for a positive diagnosis are in common usage and are grouped under major and minor criteria. The major features are respiratory insufficiency, cerebral involvement and petechial rash. The minor features are pyrexia, tachycardia, retinal changes, jaundice and renal changes.

Central nervous system findings were evident in the first case while respiratory system findings were evident in the second case. Kidney functions were deteriorated slightly in both cases, with an increase in BUN in conjunction with a drop in the daily urine output which were thought to be due to hypovolemic shock. This prerenal insufficiency improved after appropriate administration of fluids and blood.

The most promising work has been done with steroids in the treatment of FES as inspired by the two succesful case reports of Ashbugh and Petty in 1966 (14). Methylprednisolone has a significant effect in preventing the development of the clinical and subclinical FES (15, 16). Methylprednisolone offers advantages but it is not a "wonder drug".

The modern technology in respiratory assistance i.e., volume ventilators and positive end expiratory pressure (PEEP) has improved our ability to care for FES patients (17).

Fat embolism is a self resolving illness. The mainstay of treatment concerns respiratory support. Early immobilization of fractures and delay of internal fixation until 4 days after trauma have been advocated to lessen the likelihood of developing the syndrome. Although historically 5% ethyl alcohol, heparin and low molecular-weight dextran have been advocated (1, 3, 4), only corticostreoids used prophy lactically have been shown to reduce the incidence of fat embolism in patients with long bone fractures.

#### REFERENCES

- 1. Gossling HR, Pellegrini VD. Fat embolism syndrome: A review of the pathophysiology and physiological basis of treatment. Clin Orthop 1982; 165: 68 - 82.
- 2. Gardezi SAR, Chaudary AM, Ahmad AKS, Yousaf A. Fat embolism. JPMA 1984; 34: 122 - 6.
- 3. Epps CH. Complications in orthopedic surgery. Philadelphia: JB Lipincott Co. 1986.

- 4. Jacobson DM, Terrence CE, Reinmuth OM. The neurologic manifestations of fat embolism. Neurology. 1986; 847 - 51.
- 5. Williams AG, Mettler FA, Christie JH, Gordon RE. Fat embolism syndrome. Clin Nucl Med. 1986; 11: 495 - 7.
- 6. Park HM, Ducret PR, Brindley DC. Pulmonary imaging in fat embolism syndrome. Clin Nucl Med. 1986; 11: 521 - 2.
- Suzuki T, Ikeda N, Umetsu K, Kashimura S. Pulmonary fat embolism as an ante-mortem reaction in traumatic immediate death. Med.Sci Law. 1984; 24: 175 - 8.
- 8. Rosen JM, Braman SS, Hasan FM, Teplitz C. Nontraumatic fat embolization. Am Rev Respir Dis.1986; 134: 805 - 8.
- 9. Jones JP. Fatembolism and osteonecrosis. Orthop Clin. NA 1985; 16: 595 - 633.
- 10. Stanley ML. Renal fat emboli: Maltese, crosses and heart surgery. JAMA. 1986; 256: 40.
- 11. Balk R, Bone RL. The adult respiratory distress syndrome. Med Clin NA. 1983; 67: 685 - 8.
- 12. Kaplan RP, Grant JN, Kaufman AJ. Dermatologic features of the fat embolism syndrome. Cutis. 1986; 23: 52 - 5.
- 13. Gurd AR. Fat embolism: An aid to diagnosis. J Bone Joint Surg. 1970; 52 - B: 732 - 7.
- Asbaugh DG, Petty TL. The use of corticosteroids in the treatment of respiratory failure associated with massive fat embolism. Surg GynecolObstet. 1966; 123: 493 - 7.
- Lindeque BGP, Schoeman HS, Domisse GF, Boeyens MC, Vlok AL. Fatembolism and the fatembolism syndrome. J Bone Joint Surg. 1987; 69 -B: 129 - 31.
- Schonfeld SA, Yongyudh P, Diliso R, et al. Fat embolism prophylaxis with corticosteroids: A prospective study in high risk patients. Ann Intern Med. 1983; 99: 438 - 43.
- 17. Gang H. Positive pressure ventilation in adult respiratory distress syndrome. Clin Chest Med. 1982; 3: 69 - 72.