รายงานผู้ป่วย

Fat embolism syndrome: a case report

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A case of fat embolism syndrome (FES) from multiple trauma is reported. The FES developed in an 18 year-old female 24 hours after sustaining injuries from a motor vehicle accident. Severe hypoxemia, rapid deterioration of cerebral function, petechial hemorrhage at conjunctiva and thrombocytopenia were present and established the diagnosis of FES. Management included ventilatory support with a mechanical ventilator, cardiovascular support with inotropic agents ,regulation of fluid balance and rigid fixation of the femoral fracture,. Neither heparin nor steroids were used. The patient recovered after 7 weeeks of intensive therapy and was discharged home with some impairment of cerebral function.

Key word: *Fat embolism syndrome.*

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ได้รายงานผู้ป่วยที่มี Fat embolism syndrome (FES) 1 ราย เป็นผู้ป่วยหญิงอายุ 18 ปี ได้รับอุบัติเหตุรถยนต์ มีการบาดเจ็บที่ศีรษะและช่องอก กระดูก femur ของขาซ้ายหัก ผู้ป่วยเกิดอาการของ FES หลังจากได้รับบาดเจ็บ 1 วัน การวินิจฉัย FES ของผู้ป่วยรายนี้ ได้จากการมีค่าอ๊อกซิเจนในเลือดต่ำมาก อาการทางสมองเลวลงอย่างรวดเร็ว มีจุดเลือดออกที่เยื่อบุตา และมีเกร็ดเลือดต่ำ การรักษาเป็นแบบประคับประคองเป็นส่วนใหญ่ โดยใช้เครื่องช่วยหายใจ ให้ยารักษาระดับความดันโลหิตและควบคุมมิให้ผู้ป่วยมีภาวะน้ำเกิน สำหรับกระดูก femur ที่หักนั้น ได้รับการรักษาโดยการผ่าตัดใส่เหล็ก ผู้ป่วยมีอาการดีขึ้นกลับบ้านได้ รวมเวลาที่รักษาตัวอยู่ในโรงพยาบาล 43 วัน

Fat embolism is an extremely common pathologic finding following trauma . In an autopsy series of 300 accident victims, the incidence rate ranged from 80 to 100 percent. (1) In view of the high volume of fat in long bones, it is not surprising that fat embolism is common after bony trauma. Nevertheless, the clinical fat embolism syndrome (FES) of pulmonary dysfunction, coagulopathy, and neurologic dirturbances associated with increased circulating fat globules appears to be uncommon, The overall incidence of FES is only 1-3.5 percent of patients with a fracture of the tibia or femur. (2) The FES usually follows orthopaedic injuries, but it has also been reported to occur after prosthetic joint replacement, closed chest cardiac massage, liver trauma, burns, extracorporeal circulation, rapid high-altitude decompression, bone marrow transplantation, and liposuction. The following is a case report of severe FES which was successfully treated after a period of intensive therapy.

Case presentation

An 18 year-old female patient was sent to Chulalongkorn Hospital after sustaining injuries in a motor vehicle accident. On physical examination, the blood pressure was found to be 90/60 mmHg, The pulse rate was 110/ minute and the respiratory rate was 28/minute. The patient was in a combative state with the Glasgow Coma Scale (GCS) of 7. Chest X-ray revealed bilateral pulmonary contusions with pneumohemothoraces (Figure 1). Diagnostic peritoneal lavage (DPL) was performed to exclude intraabdominal injuries. The result of the DPL was negative. She also had closed fractures of the left femur, right radius and right ulna (Figure 2A,2B). Initial management included orotracheal intubation, bilateral tube thoracostomy, fluid resuscitation and skeletal traction of the fractured femur. Cerebral CT scan was subsequently performed which revealed generalized brain swelling (Figure 3). After that she was admitted to the intensive care unit with stable vital signs. Her ventilation was supported by a volume-cycled ventilator.

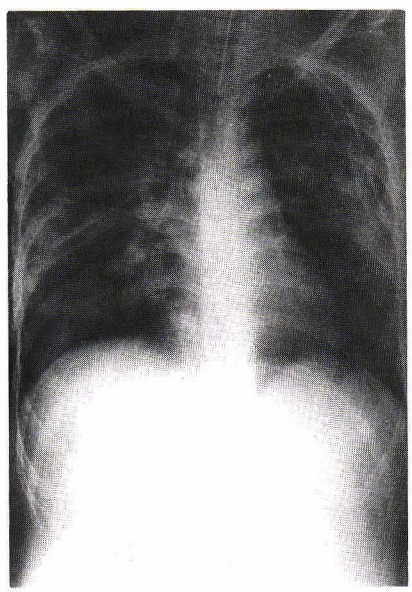

Figure 1. Bilateral pulmonary contusion with pneumohemothoraces.

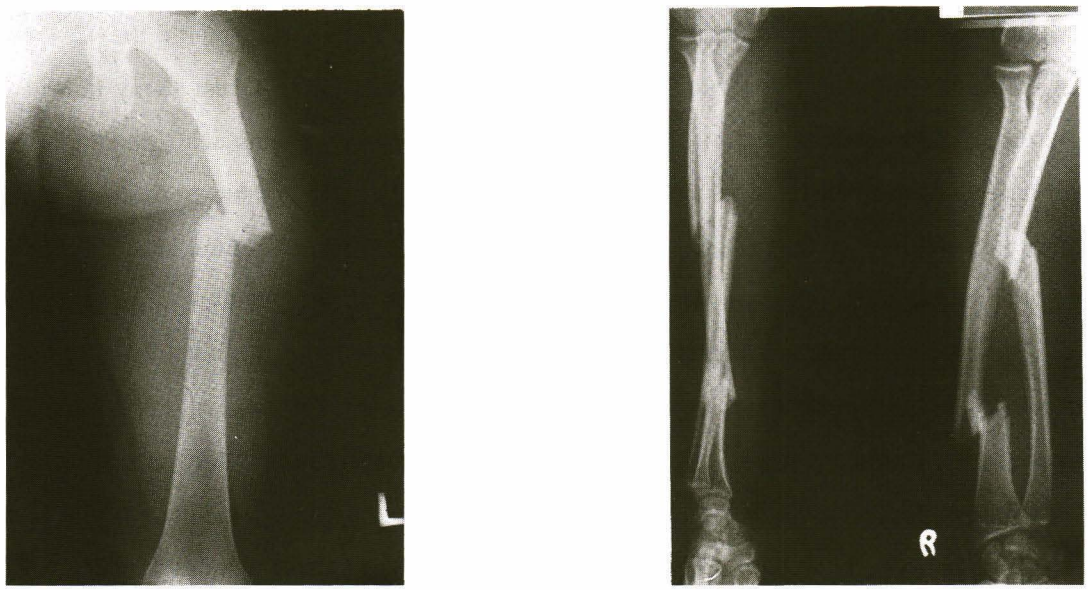

Figure 2. (A) Closed fracture of the left femur.
(B) Closed fracture of the right radius and ulnar.

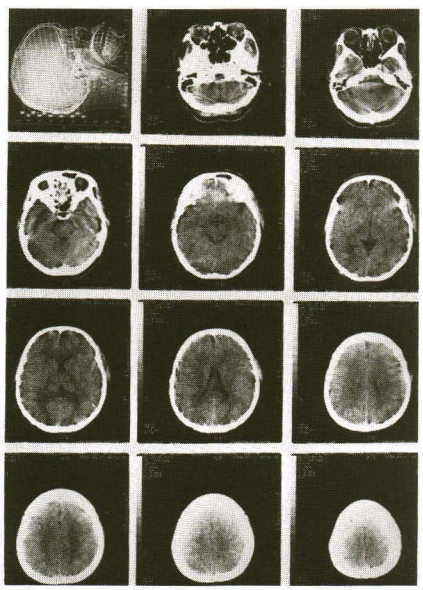

Figure 3. Generalized brain swelling.

Twenty-four hours later, her conditions deteriorated. She developed severe hypoxemia which was treated by increasing PEEP and inspired oxygen concentration. The hypoxemia persisted even though the FiO2 was increased to 1 and the PEEP was increased to 18 cmH2O (Table 1). The chest X-ray revealed haziness of both lungs (Figure 4). She was in a comatose state with the GCS of 3, but the pupils were still reactive to light. Petechial hemorrhage was observed at the anterior chest wall and at the conjunctiva of her left eye. She also had thrombocytopenia (platelet count was 64,000/mm 3) and hyperlipasemia (serum lipase was 1009 IU/L; normal value was 0-190 IU/L). At this point, the diagnosis of FES syndrome was established.

Table 1. Arterial blood gas analysis during the initial 12 hours after the diagnosis of fat embolism syndrome was made.

Time	12:45	15:00	16:45	17:15	18:20	19:15	21:45	23:30
pH	7.451	7.467	7.41	7.39	7.449	7.484	7.48	7.467
pO2	36.3	38.5	28	32.9	45.8	48.8	62.6	77.4
pCO2	37.3	35.2	40	42.4	36.8	33.8	34	36.2
HCO3	25.9	25.4	26	25.8	25.5	25.6	25.3	26.1
BE	2.9	2.9	2	1.2	2.5	3.6	3.2	3.4
O2 Sat	72.3	76.5	55	62	83.8	87.6	93.5	96.1
FiO2	1	1	1	1	1	1	1	1
TV	550	550	600	600	600	600	600	600
RR	20	20	18	18	18	18	16	16
PEEP	-	10	10	15	18	18	18	18

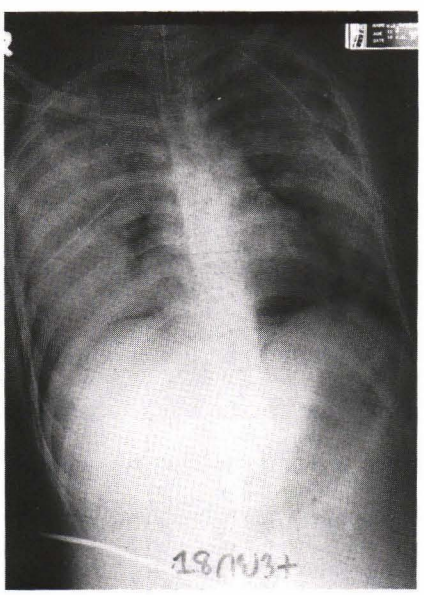

Figure 4. Twenty four hours after injury, chest X-ray revealed haziness of both lungs.

The initial treatment was directed at correction of the severe hypoxemia. Small doses of furosemide (20-40 mg) were given at six hour intervals during the first few days to decrease her lung water. Dopamine was infused to maintain the mean arterial pressure to at least 80 mmHg. Fluid balance was maintained by keeping CVP below 12 cmH20. Early enteric feeding was started on the second day of admission. The hypoxemia gradually improved with this vigorous therapy but 100% inspired oxygen and high PEEP had to be given during the first week of admission. Although her condition was complicated by bilateral pneumothoraces from barotrauma, the situation was controlled with bilateral tube thoracostomy.

A tracheostomy was performed during the second week of admission. The inspired oxygen concentration and PEEP were gradually decreased with acceptable arterial blood gas values (Table 2). Her consciousness level was improved with a GCS of 6 and she underwent open reduction and internal fixation of her left femur and right forearm with plates and screws on the tenth day of admission.

Table 2. Arterial blood gas analysis during the first week after the diagnosis of fat embolism syndrome was made.

Date	2	3	4	5	6	7	8
pH	7.488	7.486	7.534	7.4	7.43	7.5	7.506
pO2	122.3	63	249.4	133	251	230	257
pCO2	35.9	39	39.3	40	47.8	46	36.6
HCO3	27.2	30	33.1	29	31.8	36	28.9
BE	4.9	6.5	10.8	6.5	7.1	6.4	6.7
02 Sat	98.7	93	99.7	98	99.6	99	99.7
FiO2	1	1	0.8	0.7	0.8	0.8	0.8
TV	600	600	600	600	600	600	600
RR	16	16	20	16	16	20	20
PEEP	18	18	18	18	18	18	15

During the third and fourth week of admission she developed fever. The chest X-ray revealed bilateral alveolar infiltration (Figure 5). The sputum culture grew Pseudomonas aeruginosa and the diagosis of Pseudomonas pneumonia was made. Antimicrobial therapy with ceftazidime and amikacin was started and her condition improved. At the end of the fourth week, obvious improvements of her cerebral function was observed. The GCS was 9. She was subsequently removed from the ventilator and was able to breathe spontaneously in room air.

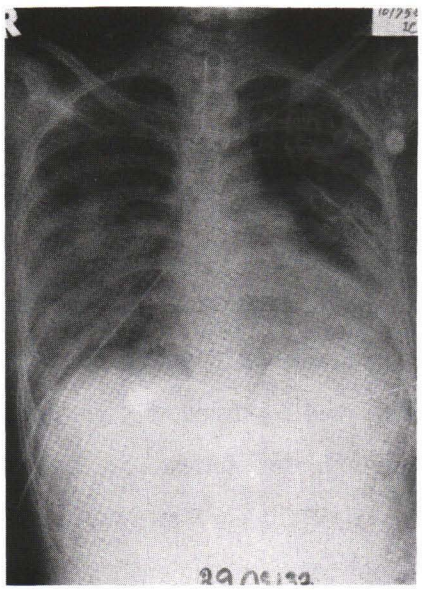

Figure 5. Pseudomonas Pneumonia.

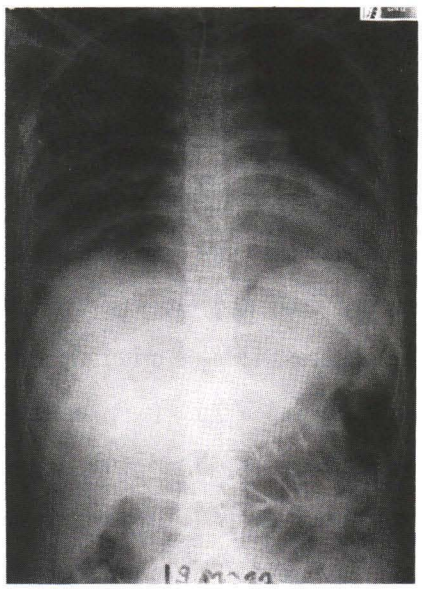

Figure 6. Pneumonia was improved.

She was eventually discharged with some residual impairment of her cerebral functions. The GCS before returning home was 12. The hospital stay was 43 days. Three months later, she was seen at the out-patient department with no impairment of cerebral function.

Discussion

For more than a century FES has been of interest to the traumatologist. Certain puzzling features have stimulated many experimental and clinical studies . The present incidence of trauma, especially in regard to autoaccidents, has enhanced the importance of this complication. Its relationship to skeletal and soft tissue injury is well recognized , and many cases have been reported.

The experimental production of fat in the veins was attempted in the seventeenth cenjury. Lower of Oxford reported his observations in 1669. Magendie in 1842 described the symptoms from the intravenous injection of olive oil. Zenker in 1862 described fat emboli in the pulmonary capillaries of a railroad worker who sustained a fatal thoracoabdominal crush injury. Wagner in 1865 reported 48 cases of fat emboli found in the lungs at autopsy. In 1862 he had demonstrated fat in the pulmonary capillaries of three patients who had osteomyelitis and in two patients with pneumonia. Von Bergmann in 1873 was the first physician to recognize fat embolic clinically in a patient with a comminuted fracture of the femur. Postmortem examination revealed the presence of a large amount of pulmonary fat. [3]

The concept that all the embolic fat takes origin from traumatized depots has been seriously challenged. The mechanical theory does not provide a satisfactory explanation for fat embolism found in association with nontraumatic conditions. The physiochemical theory regarding the source of embolic fat postulates that an alteration of the normal plasma colloidal dispersion of chylomicrons occurs, after trauma, which coalescence

into larger fat droplets. The physical state of the blood lipids is altered by tissue injury. The lipid - mobilizing hormone is activated after trauma, and blood lipid concentration in increased.[3]

Gurd's criteria for the diagnosis of FES are commonly used and are divided into major and minor features. Major criterion are axillary or subconjunctival petechia, hypoxemia (PaO2 < 60 mmHg: Fio2 < 0.4), central nervous system depression disproportionate to hypoxemia, and pulmonary edema. Minor criterion are tachycardia (more than 110 beats/minute), pyrexia (temperature higher than 38.5^{o} C),emboli present in retina on fundoscopic examination, fat present in urine, a sudden unexplainable drop in hematocrit or platelet values, increasing erythrocyte sedimentation rate, and fat globules present in the sputum. The diagnosis of FES requires at least one sign from the major criteria and at least four signs from the minor criteria categories.[2] In this case, all four major criterion are present. Typically,the full clinical syndrome of fat embolism develops 1 to 3 days after trauma.[4] The reasons for this delay are unclear, but it has been suggested that they can be explained by (1) continuing embolization from the site of injury. (2) the conversion of neutral triglycerides to unsaturated fatty acids, and (3) imbalance between coagulation and fibrinolysis, leading to deposition of fibrin in pulmonary vessels and from the pulmonary circulation, small fat droplets pass into the general systemic circulation and form emboli in many organs, notably the brain, kidneys, and skin. The toxic effects of fat embolization were thought to be the result of chemical irritation from the hydrolysis of neutral fats to fatty acid. Our patient developped the clinical syndrome 24 hours after injury, the optimum time for the occurrence of FES.

Although hypoxemia, thrombocytopenia and hyperlipasemia are common laboratory findings in FES, they are frequently present in other situation of multiple injuries. So the test is too sensitive to be of clinical value and the diagnosis of FES should be made cautiously with other supporting clinical features.[5]

Definitive fracture management, aggressive pulmonary care, and effective treatment of shock are the cornerstones of current management of FES. Many agents have been proposed to treat FES over the years. Heparin has potential benefits by increasing lipase activity and promoting clearance of circulating fat globules. However clinical data are conflicting and the use of heparin is debatable. (1) Steroids have been used on the premise that they decrease capillary leakage by stabilizing lysosomal and capillary membranes, also decreasing the inflammatory reaction caused by the free fatty acid on the lung. Patients with FES treated with steroids were first described in 1966; subsequently, corticosteroids have become widely accepted. Some investigators, however, have suggested that patients fare as well with only supportive care and corticosteroid may even have contributed to the development of fat emboli in some cases. Still, several prospective randomized studies have suggested that prophylactic corticosteroids benefit high-risk patients.[2,6-9] In

our patient,neither heparin nor steroids had been used. Intensive supportive care was the mainstay of treatment.

It has been observed that inadequate stabilization of fractures have caused FES many weeks after the initial injury. The incidence of fat embolism syndrome is also higher when operative fixation is delayed.[2] In our patient, operative fixation was done ten days after injury because we felt that the operation would have been harmful in patient with severe hypoxemia during the early phase of her illness.

The prognosis for recovery in patients with FES is poor in patients with pulmonary insufficiency and coma.[3,6] The mortality is high in this type of patient. Mild cases often go undetected, and mortality is low in patients wihtout severe pulmonary insufficiency and cerebral manifestations. Although satisfactory recovery was obtained in our patient, residual brain damage was present for a period of time. This is probably resulted from primary brain damage after head injury which was aggravated by FES. However, after three months follow-up, no impairment was observed.

References

1. Hiyama DT,Zinner MJ. Surgical complications. In: Schwartz SL, Shires GT, Spencer FC, Husser WC, eds. Principles of Surgery. Vol 1. 6 th ed. New York: McGraw. Hill, 1994: 455-87
2. Levy D. The fat embolism syndrome: a review. Clin Orthop 1990 Dec; 261: 281-6
3. Evarts CM. The fat embolism syndrome: a review. Surg Clin North Am 1970 Apr; 50(2): 493-507
4. Fraser RG, Peter Pare JA, Pare PD, Fraser RS, Genereux GP. Diagnosis of Diseases of the Chest. Vol 3. 3rd ed. Philadelphia: W.B.Saunders, 1990: 1782-6
5. Peltier LF. Fat embolism. A perspective. Clin Orthop 1988 Jul; 232: 263-70
6. Peltier LF, Collins JA, Evarts CM, Sevitt S. A panel by correspondence. Fat embolism. Arch Surg 1974 Jul; 109(1): 12-6
7. Alho A, Saikku K, Eerola P, Koskinen M,Hamalainen M. Corticosteroids in patients with a high risk of fat embolism syndrome. Surg Gynecol Obstet 1978 Sep; 147(3): 358-62
8. Moylan JA, Birnbaum M, Katz A, Everson MA. Fat emboli syndrome. J Trauma 1976 May; 16(5): 314-7
9. Lahiri B, ZuWallack R. The early diagnosis and treatment of fat embolism syndrome: a prelimanary report. J Trauma 1977 Dec; 17(12): 956-9