General Problems Encountered In Orthopaedic Surgical Practice

An Essay Submitted for partial fulfillment of Master Degree in Orthopaedic Surgery

By

Dr. Waleed El Sayed Aly Heikel
M.B.B.CH.

Under Supervision of

Prof. . Dr. Mamdouh Zaki Saad
Prof. . of Orthopaedic Surgery

Ass. Prof. Dr. Ahmed Kamel
Ass. Prof. of Orthopaedic Surgery

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Ain Shams University
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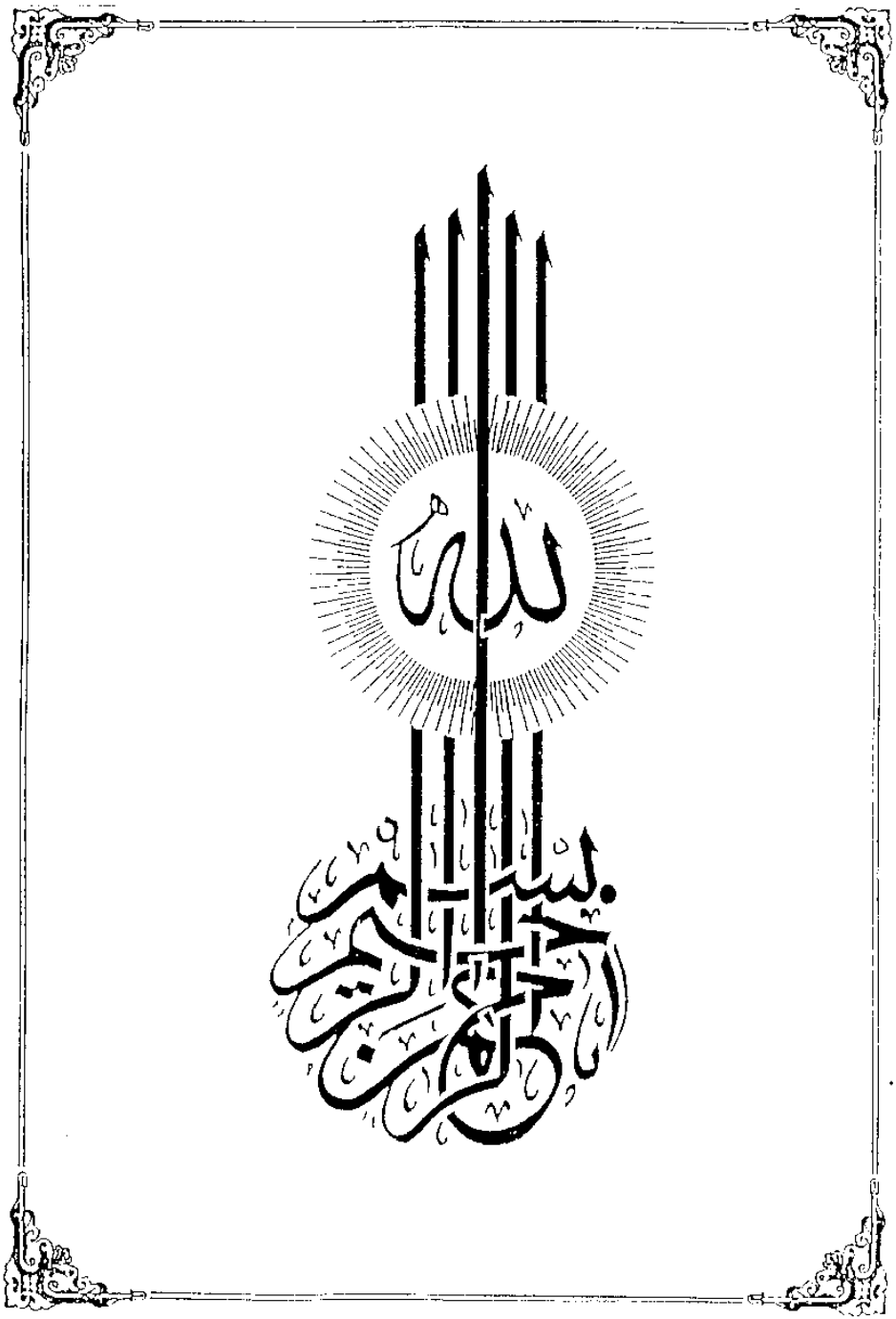

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This is a comprehensive study dealing with some of the general problems that we face in our surgical practice .
The evolution of the diagnostic procedures and the increased incidence of trauma together with the advances in orthopaedic surgery as the use of metal implants and artificial prosthesis have pointed to these complications as major problems facing orthopaedic surgeons in this era .
In this study we dealt with four of these problems which are :

(a) The fat embolism syndrome and adult respiratory distress syndrome (ARDS) and their relation to polytraumatized patient with emphasis on the role of early fracture stabilization .

(b) Thromboembolism and pulmonary embolism , stressing on their incidence following arthroplasties & recent methods for prophylaxis and treatment .

(c) Musculoskeletal infection & the role of antibiotics and infections following implants .

(d) Hypovolemic shock and the compensatory mechanisms of the body to overcome organ failure .

In this study, we tried to find the link between these complications and polytrauma as well as their relations to each other (for example shock predisposes to infection and both complicates polytrauma) aiming at directing the attention of the reader to these problems and to be a review for these serious complications in a polytraumatized patient.

CHAPTER 1

Fat Embolism & Adult Respiratory Distress Syndrome (ARDS)

Fat Embolism Syndrome

Post traumatic Fat embolization , more commonly referred as "Fat embolism syndrome" (FES) has been a source of interest and misconception *since 1861 when Zenker* first described its occurrence after a thoracoabdominal crush injury. (Quoted from Gossling and Pelligrini, 1982).

Definition:

Fat embolism syndrome 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 clinically as acute respiratory insufficiency.

Incidence:

The clinical syndrome is evident in 0.5% to 2% of long bone fractures and approaches 5% to 10% incidence in multiple fractures associated with pelvic injuries. (Gossling and Pelligrini, 1982)

Fat embolization as a subclinical event , however , occurs with all fractures of long bones , and its clinical effect is most readily quantitated by monitoring the arterial blood gas. (Gossling and Pelligrini, 1982)

Leading factors:

Similarly, Fat embolization can occur with many other traumatic and non traumatic (metabolic) conditions including hemoglobinopathy, collagen disease, diabetes ,burns , severe infections , inhalation anesthesia , metabolic disorders, neoplasms, osteomyelitis, blood transfusion and cardiopulmonary bypass, but is usually a postmortem finding rather than a clinical syndrome per se.

The clinical syndrome manifests as the adult respiratory distress syndrome (ARDS) in association with musculoskeletal trauma and a consumptive coagulopathy.

The major end organ response appears in the lungs and central nervous system. Fat macroglobules impair small vessel perfusion and endothelial damage in the pulmonary capillaries leads to ventilation/perfusion mismatch, vascular congestion , interstitial hemorrhage , alveolar wall damage, and airway collapse. The resulting clinical manifestation is life threatening impairment of pulmonary gas exchange. (Gossling and Pellegrini 1982)

It is no longer possible to consider fat embolism an isolated, unique phenomenon. It must be regarded as one of the important pulmonary complications of trauma, and it shares many features with other such complications including the respiratory distress syndrome, micropulmonary embolism syndrome, congestive atelectasis, shock lung , postperfusion lung and hypoperfusion syndrome. (Peltier, 1969)

Pathophysiology:
Aspects of lipid metabolism.

The circulating plasma lipids consist of triglycerides ,phospholipids , cholesterol, and free fatty acids (FFA) complexed with protein to enhance their solubility.

The enzyme lipoprotein lipase hydrolyzes the plasma triglycerides to FFA for transport across cell membrane. Lipoprotein lipase is released after intravenous injection of heparin and is referred to as the clearing factor.
It is this heparin effect that has been employed unsuccessfully as a treatment modality in fat embolism.

A significant rise in serum lipids can be demonstrated in humans after trauma, particularly in the FFA and triglyceride fractions.
This increase is considered to be significant in the pathophysiology of the fat embolism syndrome.

Peltier (1969) has shown direct FFA toxicity to lung parenchyma cells, capillary endothelium and surfactant resulting in disruption of the alveolocapillary membrane, interstitial hemorrhage and edema.

Riseborough and Herndon (1976) in a prospective study of 118 patients with lower extremity fractures ,demonstrated a significant rise in FFA preceded by an increase in serum lipase activity in more than half of the study group.
These changes were associated notably with a fall in PaO2 concentration, increased platelet adhesiveness, thrombocytopenia and elevated fibrinogen degradation products.

Nixon and Brock-Utne (1978) demonstrated a statistically significant correlation between arterial hypoxemia and a rise in serum FFA levels in fracture patients.

Kerstell, et al in (1969) in their studies showed that fatty acid profiles of pulmonary embolic fat most closely resembled those of depot marrow fat rather than albumin-bound FFA, circulating lipoproteins and chylomicra, or adipose tissue stores.

Additional work with C14- labeled fatty acids injected into marrow cavities before fracture also supported a bone marrow origin of embolic fat. (Gossling and Pelligrini, 1982)

Autopsy material in 208 cases studied within one hour of death due to skeletal trauma revealed congealed fat, connective tissue and marrow cells in venous circulation from the injured extremity to the pulmonary arterioles.

Observations by Meek et al (1972) on filtered arterial and venous blood samples taken from patients with isolated femoral fractures, localize the origin of fat macroglobules to the injured extremity.

Further study of a similar patient population has correlated quantitative levels of fat macroglobules in the femoral vein of the injured extremity with the appearance of signs and symptoms of fat embolism syndrome. (Gossling and Pelligrini, 1982)

In Other studies, observations of marrow and methacrylate emboli to the lungs of total hip replacement patients have demonstrated the easy access of marrow contents to the fragile medullary venous sinuses . (Breed, 1974)

Although it is attractive to hypothesize the agglutination of circulating fat, the conflicting research data and the paucity of clinical evidence doesn't support this as the usual pathophysiology in the fat embolism syndrome .
Accordingly at this stage of understanding it is reasonable to assume that embolic fat is derived from the marrow depots of the body, and that this embolic phenomenon plays a central role in the syndrome of post fracture respiratory failure. (Gossling and Pellegrini , 1982)

Coagulation and Intravascular Fat

There is increasing clinical and experimental support for the relationship of hyperlipidemia and alterations of coagulation.
Most of these studies have made several observations suggestive of a significant role of coagulation abnormalities in the fat embolism syndrome:

1. A rapid fall in hematocrit value.
2. Erythrocyte aggregation.
3. Increased platelet adhesiveness.
4. Relative thrombocytopenia.
5. Elevation in fibrin degradation products.
6. Prolonged prothrombin and partial thromboplastin times.
7. Increase in haptoglobin (alpha- 2- macroglobulin) .
   and alpha-1- antitrypsin (antifibrinolysin activators) .

(Riseborough, et al , 1976)

The fibrinolytic system may function to remove incidental fibrin deposits in animals and man through the action of activated plasmin.
It has been postulated that a failure in this system after trauma results in a rise in plasma fibrinogen and fibrin initiating intravascular clotting. (Rennie, et al , 1974)

In experimental animals there has been observed a phase of increased fibrinolytic activity followed by a prolonged fall in fibrinolysis and an associated rise in plasma fibrinogen.
Surgical trauma has been further demonstrated to produce a rise in antiplasmin activity and the serum inhibitors of plasminogen activation. (Fahmy, et al , 1981)

Observations made in patients after lower extremity fractures have shown an initial post trauma depression of fibrinogen levels followed in three to five days by a rebound hyper fibrinogenemia.
The time course of the rise in fibrinogen coincides with the disappearance of fat macroglobules from the blood stream and the presence of clinical respiratory failure, mental obtundation, petechiae and lipuria. (Riseborough, et al , 1976)

Postmortem study of the pulmonary vessels reveals platelet and fibrin clumps closely associated with areas of microscopically - evident fat embolism.
Examination of peripheral blood smears from these patients demonstrated many activated platlet "spread forms" which temporarily correspond with the

appearance of thrombocytopenia and arterial hypoxia in lower extremity fracture victims. (Riseborough, et al , 1976)

Additionally , oscillating prolongations in prothrombin and partial thromboplastin times with depressed levels of factor V and VIII have been cited as evidence of periodic episodes of disseminated intravascular coagulation.

In view of the available information it seems plausible to believe that neutral fats & tissue thromboplastin released from the fracture site activate the clotting cascades and platlet aggregation.

Suppression of the fibrinolytic system in the injured patient then serves to aggravate an ongoing accumulation of aggregates of fat macroglobules, platelets, erythrocytes, leucocytes and fibrin which are passively concentrated in the lung by virtue of its filtering action on venous blood before it is recycled to the systemic circulation.

The Lung as a filter

The Lung's filtering efficiency is not absolute.
The physical properties of embolic material and their surface interaction with endothelial cells affect transport through the system.
Once the fat emboli become lodged in the pulmonary bed, there ensues a biphasic pattern of clinical pathology.
The pulmonary circulation is normally a low-pressure low-resistance system. Observations of pulmonary emboli following deep venous thrombosis have demonstrated that 80% of the pulmonary circulation must be obstructed to have a significant hemodynamic effect. (Gossling and Pellegrini , 1982)

In elaborate investigations following neutral fat injection in dogs, Tornabene et al (1979) noted an acute shift of 40% of the effective alveolar ventilation to segments with a ventilation / perfusion ratio (V/ Q) between 5 and 100. There was no increase in shunt component or ventilation to unperfused segments.
Cardiac output was halved and pulmonary artery pressure increased by 50% with respect to controls, while arterial PO2 fell 10- 80 mm Hg.
These changes are readily explained by mechanical pulmonary vascular obstruction.

Gas exchange returned to normal in a matter of hours by an autoregulatory distribution of ventilation effected by local bronchoconstriction in areas of diminished blood flow.
These acute phase changes in their most fulminant form , result in death from acute cor pulmonale, a very rare clinical manifestation of fat embolization in man. (Tornabene, et al , 1979)

The subacute or delayed clinical manifestation of fat embolization are those most frequently seen in humans with full- blown (FES).
The primary defect is in arterial oxygenation with severe hypoxemia resulting from a large ventilation / perfusion ratio (V/Q).

Peltier hypothesized that a facultative increase in lipase produced by pneumocytes in response to embolic neutral fat results in liberation of FFA that are directly toxic to the lung. (Peltier, 1969)

Observations in both Laboratory animals and fracture patients reveal an elevation in serum Lipase activity preceding a simultaneous rise in FFA and fall in arterial oxygenation. (Riseborough , et al , 1976)

The rise in FFA , however , remains questionably attributed to the catecholamine response to trauma.
Moylan et al (1976) contend that the catabolic response to trauma results in lowered serum albumin levels and an increase in the amount of unbound FFA which is toxic to the pneumocytes. Electron micrographs of lung tissue from such patients show vesiculation and thickening of capillary and alveolar walls.

Lung surfactant.

Type II pneumocytes produce the lung surfactant which lines the alveoli as an extracellular layer of fluid that consists of a layer of phospholipid material lying on top of a base of protein material.
The lung surfactant stabilizes the alveoli as they expand and contract and protects them against an inherent tendency for collapse and atelactasis.
Pulmonary fat embolism may affect lung surfactant activity as a result of either local hypoperfusion of the pulmonary capillaries (Mechanical blockage), or direct contact between the neutral fat and its hydrolysis product, free fatty acids and lung surfactant (Chemical effect).