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Respiratorische Insuffizienz bei Mehrfachverletzten

Respiratory insufficiency in patients with multiple injuries

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LUNG PATHOLOGY IN NONTHORACIC TRAUMA

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Summary

The sequential pathological changes of the respiratory distress syndrome have been studied in patients with nonthoracic trauma by percutaneous lung biopsies.

The initial ultramicroscopic changes consisted of accumulation and aggregation of platelets and leukocytes in pulmonary vessels. These were followed by marked interstitial edema and later by changes in alveolar lumen. In some patients fat embolism is the predominant lesion. Pathogenesis of these changes is discussed.

The respiratory distress syndrome (RDS) is recognized as a major complication which may follow nonthoracic trauma. Its initial manifestation is tachypnea with increased respiratory effort. Laboratory studies show a fall in arterial blood

Fig. 1 Percutaneous lung biopsy. A pulmonary capillary is filled with degranulated platelets and leucocytes. Some granules are free in the lumen. The endothelium shows focal cytoplasmic rarefaction. Alveolar edema.

oxygen tension with evidence of arterio-venous shunting, while chest X-ray films show a gradual development of reticular infiltrates which may progress to a dense consolidation.

The pathological anatomical findings in the lung of patients died with posttraumatic respiratory insufficiency are variable: they depend on the time that has elapsed since the clinical event and vary also with the instituted therapy. Moreover lung specimens obtained from autopsies show post-mortem changes and can not be utilized for ultrastructural observations. This study reports on an attempt to investigate in vivo sequential changes in lung morphology by a disposable biopsy needle.

Materials and Methods

Ten percutaneous lung biopsies were performed in 8 patients with posttraumatic (nonthoracic) pulmonary insufficiency. The disposable assembly (Travenol Lab.) used consists of a 6 inch cannula hub and an obturator hub with a 20 mm specimen notch. Its inner diameter is 1,4 mm. The needle was introduced in the 5th intercostal space along the middle axillary line. Specimens were immediately fixed in 1 per cent osmium tetroxyde in phosphate buffer, dehydrated in alcohol and embedded in Epon 812. Ultrathin sections cut by Reichert Om U2 ultramicrotome and a diamond knife were stained with uranyl acetate and lead citrate and examined using a Philips E M 200 electron microscope. Thick plastic sections for light microscopy were stained according to Rüdeberg's technique.

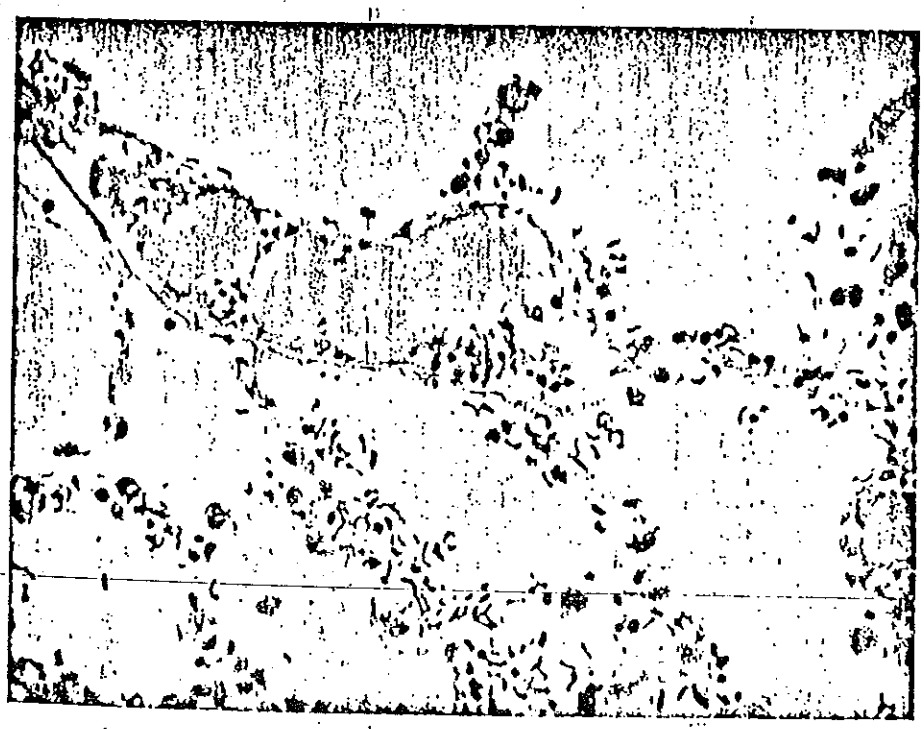

Fig. 2 Percutaneous lung biopsy. Fat emboli are seen in pulmonary vessels. Alveolar hemorrhage.

Results

The study of biopsies reveals many morphological changes in the lung.

Microcirculatory alterations

Marked capillary congestion is the first alteration observed. Abundant polymorphonuclear leukocytes, scattered strands of fibrin and platelets are in the capillary lumen (fig. 1). A decrease in lysosomes granules is noted within the cytoplasm of the leukocytes. Many mitochondria and ribosomes, but only few secretory granules are observed in platelets. In fact it is possible to observe granules just released into the capillary lumen. The platelets and leukocytes form microemboli. Many fat emboli, which measure from 50 to 1'000 micron, are in the vascular lumen (fig. 2). Capillary endothelium is attenuated but intact.

Interstitial space alterations

In the interstitium there is marked edema. Separation of the connective tissue elements by electron-lucent spaces is striking and often extensive (fig. 3). These spaces do not contain the electron-dense granularity of plasma proteins. The separation of connective tissue elements is often extended into the contiguous thin alveolar septa for a variable distance but never into the thinnest portion of the air-blood barrier which consists of a type I alveolar cell, a capillary endothelial cell and a basement membrane. This leads to a marked widening of the alveolar wall. There are numerous fibroblasts and histiocytes. The recognizable lymphatics are distended. Some of the endothelial cells of these lymphatics are partially swollen.

Alveolar space alterations

Type I alveolar cells do not show alterations. Type II alveolar cells contain few lamellar bodies, mitochondria and ribosomes.

Electron-dense granularity of plasma proteins fills the alveolar lumen. There are numerous clusters of surfactant, red cells, macrophages and leukocytes. Hyaline membranes, the last alveolar alteration, line the alveolar wall (fig. 4).

Discussion

In considering the pathogenesis of these changes, the early pronounced pulmonary alterations in microcirculation provide a useful starting point. Many reports have pointed to microemboli as one of the etiologic factors in the RDS. Experimentally, pulmonary platelet trapping has been shown to appear in the circulation following acute trauma (4,5). Platelet aggregates can have a peripheral origin, and therefore come to the lung as emboli, or they can form in the lung itself. The problem is now whether the accumulation and the aggregation of the platelets in the lung is causing the rise in pulmonary resistance and the hypoxia by blocking the pulmonary circulation mechanically or whether any biochemical factors are involved. Several experimental investigations indicate that accumulation of platelets in the lung per se does not have any harmful effects. It must be the release of substances from the platelets which causes the damage. As a matter of fact we know that granules taken from platelets contain histamine, serotonin, prostaglandins (E_2, F_{2a}), and catecholamines. All of these substances are

Fig. 3 Percutaneous lung biopsy. In the interstitium there is marked edema. Separation of the connective tissue elements by electron-lucent spaces is striking and often extensive. A = alveolar space, I = interstitium.

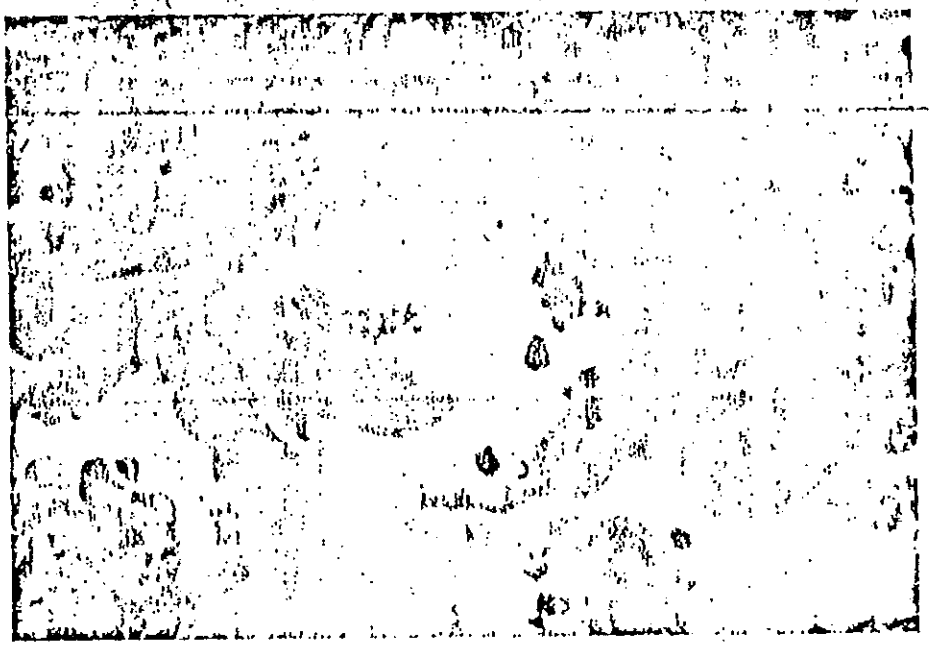

Fig. 4 Percutaneous lung biopsy. Hyaline membranes line the alveolar wall.

released during platelet aggregation and act on the vessels. Serotonin, prostaglandins and catecholamines provoke an intense venous constriction and thus a stasis in the capillary district. The stasis causes alteration of the equilibrium of forces which regulate the liquid and solute exchanges through the alveolar-capillary membrane by increasing the capillary hydrostatic pressure, thus causing edema in the interstitial tissues. Dysproteinemia and infusion of large volumes of cristalloids would be a facilitating factor. Moreover, changes in vascular permeability appear to be important for the formation of edema.

Histamine, serotonin, and lysosomal enzymes deriving from granulocytes would alter capillary permeability by acting on interendothelial junctions as recently documented with electron microscope by using a stroma free Hb solution as tracer (2).

The effect of these vascular changes on pulmonary surfactant may now be considered. This substance is inactivated by an outflow of edema fluid into the alveolar space and further amounts are therefore required. Its synthesis however is impaired by the reduced perfusion of the lung, as is shown by the defective incorporation of surfactant precursors (3). Inactivation and deficient production of surfactant leads to increased surface tension which causes not only collapse but also further exudation of fluid into the alveolar lumen.

The eventual consequence of all those events is the formation of hyaline membranes in the alveolar lumen.

Fat embolism is another striking feature on our morphological findings. The source of fat globules in the pulmonary circulation is assumed to be bone marrow or soft tissue fat (6). Recent studies point to toxicity from free fatty acids as the cause of pulmonary manifestations of the fat embolism syndrome (1). Two sources for free fatty acids have been demonstrated. Neutral fat mobilized from injured bone or soft tissue and trapped in pulmonary capillaries is hydrolyzed to fatty acids by a specific lipase produced in the lungs. The other source is stated to be free fatty acids mobilized as a response to stress. Accumulation of fatty acids in the pulmonary circulation results in damage to capillary endothelium and to alveolar surfactant.

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