The Shock Lung - Tonio J. Bugeja

This is a summary of a paper read by Tonio J. Bugeja at the Intensive Care Conference at St. Luke's Hospital, Saginaw, Michigan, on August 2nd, 1972.

The pulmonary lesion which occurs with trauma and shock has been variously known as "traumatic wet lung", "congestive atelectasis" or "haemorrhagic pulmonary oedema". Recently the designation "shock lung" has been used. It has now been established that in patients who die of shock and trauma, the lungs are involved more frequently than any other organ.

Pathology

Often on gross examination the lung tissue exhibits evidence of congestion and oedema as well as thromboembolism and bronchopneumonia. The tissue is often of a dark purple colour. Occassionally pulmonary haemorrhage, haemothorax and hyaline membrane formation are encountered.[1] If these pathological changes are extensive the lungs may appear solid.

On microscopical examination there is marked interstitial oedema[1]; the alveolar spaces in the involved parts of the lung are filled with fluid and red and white blood cells. Oedematous thickening of the interalveolar septa and marked capillary congestion are additional features[2]. Fat embolism is often met with while hyaline membrane formation occurs occassionally. In cases where the shock is severe, large areas of all lobes may be involved.[1,2]

Aetiology

The mode of production of shock lung is as yet theoretical. **Eaton, Czebrinski** and **Smith**[3] suggested that pulmonary capillary hypoxia caused by the blood loss and tissue trauma lead to an increase in capillary permeability and hence alveolar oedema. **Blaisdell** *et al*[4] suggested that the primary consequence of blood loss and tissue trauma is pulmonary microembolism which then leads to a significant diminution of clotting factors. The intravascular coagulation is held responsible for the parenchymal damage in the lung. Other workers[5,6] consider a loss of pulmonary surfactant and therefore a rise in the surface tension as the major cause of alveolar collapse and transudation into septal tissues.

Clinical Features

Shock lung is clinically characterized by progressive respiratory failure in the shocked patient, falling PO_2 levels increasing tachypnoea, dyspnoea and cough, cyanosis, respiratory acidosis and physical signs of pulmonary congestion or pneumonia.[4] The arterial hypoxaemia is uncorrected by oxygen breathing;[7] a low central venous oxygen saturation[8] has also been noted. Some of these features are accounted for by abnormal diffusing capacities,[9] redistribution of pulmonary blood flow[10] and ventilation perfusion defects.[11] Often respiratory failure sets in days after the surgical repair of the traumatized tissue as well as the rise of the blood pressure to quasi normal values; it may however appear with and in the presence of shock.[11]

Treatment

This includes the correction of shock and the hypovolaemic state. The traumatized tissue must be repaired as soon as possible.[12] In the case of cardiogenic shock, cardiac failure as well as any arrhythmias must be dealt with. Digitalis and isoproterenol are to be used to improve the intrinsic efficiency of the heart. Vasoconstrictors may be useful. If thromboembolism is thought to be present heparin is also administered. Good ventilatory theory is a major part of the treatment. This includes clearing the airways of excess secretions, ensuring adequate oxygen administration and appropriate antibiotic cover. Continuous positive pressure breathing may be of basic importance in the management of these patients.[11]

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