"Shock Lung" Syndrome following Diabetic Ketoacidosis; Treatment with Heparin

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Following recovery from shock due to diabetic ketoacidosis, a 44-year-old man experienced respiratory distress with pulmonary infiltrates and disseminated intravascular coagulation. On two occasions heparin corrected the bleeding diathesis and led to clearing of the lungs. Death occurred from an intercurrent infection. At autopsy, the typical findings of "shock lung" were demonstrated.

In 1967, Hardaway introduced the term "shock lung" to characterize the pulmonary pathologic findings of capillary thrombi, hemorrhages, edema and atelectasis occurring in patients in whom pulmonary insufficiency occurred following recovery from shock. Shortly thereafter, Rigby and Christy reported an instance of survival following prolonged Gram-negative shock and clinical "shock lung." Christy has suggested that "shock lung" may be a manifestation of localized intravascular coagulation and has recommended treatment with dextran or heparin. There have been few reported cases of "shock lung" associated with intravascular coagulation and none prior (to the author's knowledge) documenting the effectiveness of heparin therapy in "shock lung."

CASE REPORT

A 44-year-old black merchant seaman had been in good health until five days prior to hospitalization when he noted weakness and polyuria. One day prior to admission he was found unconscious aboard ship and 24 hours later he was hospitalized. On admission, he was noted to be dehydrated and delirious. His pulse was 120/minute, blood pressure 60/0 and rectal temperature 100°F. The odor of acetone was noted on his breath. His chest was clear to percussion and auscultation. Significant laboratory findings included blood sugar 832 mg/100 ml, potassium 2.9 mEq/L, CO2 13 mM/L, acetone present in a 1:8 semidilution, blood urea nitrogen (BUN) 40 mg/100 ml, hematocrit 37 percent, arterial Po2 64 mm Hg, Pco2 29 mm Hg and pH 7.24. Sodium, chloride, glutamic-oxaloacetic transaminase (SGOT), glutamic-pyruvic transaminase (SGPT), serologic test for syphilis (VDRL) and chest roentgenogram were negative or normal. The sputum contained an occasional neutrophile and no organisms were present on Gram stain or culture. He had oliguria (less than 2 ml of urine per hour) and the urine contained protein and red cell casts. The central venous pressure was 0 cm water. Following rehydration, potassium replacement and insulin administration, the venous pressure increased to 5 cm water and the urine flow to 75 ml/hour. Forty-eight hours after admission, blood chemistry tests were normal, the serum was free of acetone and the patient was alert, oriented and afebrile.

A repeat chest roentgenogram was negative. On the fifth hospital day there was the onset of wheezing, pulmonary infiltrates on the chest roentgenogram and a rising BUN. The arterial Po2 (breathing room air) was 50 mm Hg. A tracheotomy was performed. Sputum and blood cultures were negative and a trial of antimicrobials was without benefit. On the seventh hospital day, hemorrhagic phenomena occurred, manifested by melena, hematemesis, bleeding from the tracheotomy incision and needle puncture sites, multiple ecchymoses and a fall in the hematocrit to 25 percent. Fragmented red cell forms were present in the peripheral blood smear. The plasma hemoglobin was 22 mg/100 ml, the thrombin time eight seconds (control three seconds) and the platelet count 80,000 per cu mm. Prothrombin and partial thromboplastin times were prolonged. A cryoprecipitate was present in the plasma. The F1 test for fibrin split products was negative in a 1:8 serum dilution. Protein electrophoresis, immunofluorescent antinuclear antibody titer, LE cell preparations, latex fixation, Coombs' test, G6PD screening test, Ham's test, autohemoly-
SHOCK LUNG SYNDROME

Figure 3. Thrombi in pulmonary capillaries (arrow) (hematoxylin-eosin, × 100).

sis, further blood and sputum cultures and a bone marrow aspirate were normal or negative. Following the intravenous administration of heparin (5,000 units every six hours) bleeding ceased, the chest roentgenogram showed resolution of the pulmonary infiltrates and coagulation indices improved. Following withdrawal of heparin, melena, a further fall in the hematocrit and pulmonary infiltrates with wheezing promptly recurred. Heparin was reinstituted; improvement was rapid and renal function returned to normal. By the 24th day the arterial Po2 (breathing room air) was 88 mm Hg. On the 30th hospital day he developed fever, delirium, hypotension and left and right lower lobe infiltrates. Coagulase positive Staphylococcus aureus was cultured from the sputum and blood. Despite appropriate antimicrobial treatment, he died on the 35th hospital day.

At autopsy, the right and left lungs weighed 1050 and 800 grams respectively. There was extensive consolidation. Microscopically, there was acute pneumonia with areas of abscess formation and clumps of Gram-positive cocci. Most alveoli contained edema fluid. Capillary thrombi were widely disseminated in the lungs, pancreas, adrenals, spleen and renal glomeruli (Fig 3 and 4).

Figure 4. Thrombus in splenic vessel (hematoxylin-eosin, × 100).

sequent to diabetic ketoacidosis. After correction of shock, respiratory distress with pulmonary infiltrates and a bleeding tendency occurred. Thrombocytopenia, coagulation abnormalities, a microangiopathic hemolytic anemia and the presence of “cryofibrinogen” were consistent with disseminated intravascular coagulation,13–15 and the diagnosis was confirmed at autopsy by the finding of multiple small vessel thrombi. Following the administration of heparin on two occasions, bleeding stopped, coagulation indices improved and pulmonary infiltrates cleared. Conversely, when heparin was withdrawn, bleeding and infiltrates recurred promptly. In the context of the present clinical setting, the initial pulmonary lesions of this patient are more aptly categorized as “shock lung” syndrome. However, whether due to localized intravascular coagulation or to pulmonary microembolization, this case documents the efficacy of heparin in the treatment of the “shock lung” syndrome.

REFERENCES


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Discussion

Respiratory insufficiency following major vascular reconstructive surgery is thought to be due to pulmonary microembolization consequent to diminished blood flow and stasis in the extremities. Under such conditions of “local shock,” clotting occurs on the venous side of the capillaries as the lower pH in that location favors coagulation. Upon restoration of blood flow, microemboli are flushed from these small vessels and lodge in the capillaries of the lungs. Similar pulmonary lesions have been observed in patients who have undergone cardiopulmonary bypass. A breakdown in the hemostatic balance between the underlying hypercoagulability and the associated thrombolysis may result in a hemorrhagic state. The pulmonary lesions are similar, whether associated with major vascular reconstructive surgery or with cardiopulmonary bypass or when found in patients who have died following recovery from shock of diverse etiology. The pathogenesis may be the same in all cases.

Dehydration and shock occurred in this patient con-
Pulmonary Blastoma; Longterm Survival of Juvenile Patient

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An 11-year-old boy (at the time of initial treatment) has survived eight years, free of recurrence after left lower lobectomy for pulmonary blastoma. Pulmonary blastoma is a distinct subtype of pulmonary malignancy not to be confused with carcinosarcoma. Its distinguishing features are gland-like structures lined by nonciliated epithelium with a surrounding stroma resembling mesenchyma. Three of the 19 known are living five or more years after surgical treatment.

Pulmonary blastoma is an exceedingly rare primary neoplasm of the lung. It has also been called a pulmonary embryoma because of its close histologic resemblance to fetal lung. Its distinguishing features are gland-like structures lined by nonciliated epithelium with a surrounding stroma resembling mesenchyma. The neoplasm is so rare that relatively few cases are reported, and the natural history of the lesion is not clearly defined. Therefore, an additional case of pulmonary blastoma, occurring in an 11-year-old boy who has survived eight years following surgery without evident residual or recurrent disease, is worthy of report.

CASE REPORT

An 11-year-old boy of Japanese ancestry was admitted to the Honolulu Kaiser Foundation Hospital in April, 1964. A week earlier, a brisk episode of apparent bleeding from the nasal pharynx had occurred, but careful otolaryngologic examination revealed no site of bleeding. His hemoglobin was 10 gm. Two days later, hemoptysis was suspected as the

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