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Miliary tuberculosis and disseminated intravascular coagulation
A case report

Mohsin Alam, MD,* and George Bower, MD**

A case of miliary tuberculosis complicated by respiratory insufficiency and disseminated intravascular coagulation was treated successfully with antituberculosis drugs, ventilatory support, and heparin, packed red cells, platelet transfusions and plasma. Tuberculosis is a rare cause of disseminated intravascular coagulation.

A variety of hematologic abnormalities has been described in association with tuberculosis, including anemia, leukopenia, leukemoid reactions, and thrombocytopenia. We report here a rarely described complication: disseminated intravascular coagulation.

Case report.

A 62-year-old black man was admitted to Henry Ford Hospital on October 3, 1973, with a two-week history of fever, chills, cough, anorexia, nausea, vomiting and 20 lb weight loss. He had no history of previous lung disease and had not used alcohol to excess. On admission he was acutely ill and toxic. Temperature was 40°C, pulse rate 110/min, and respiratory rate 26/min. Funduscopic examination gave normal results. Coarse rales were heard in the lower lung fields. The liver span measured 15 cm by percussion. No lymphadenopathy or splenomegaly were detected. Results of the rest of the examination, including neurologic evaluation, were unremarkable.

Initial laboratory studies revealed a white blood cell count of 4,200/mm³ with 88% polymorphonuclear leukocytes, hemoglobin 15.8 gm%, hematocrit value 48%, and normal electrolytes, creatinine, blood sugar, prothrombin time, platelet count, creatinine phosphokinase and serum protein electrophoresis. Alkaline phosphatase was 23.1 (n = 1.5-4.0 Bodansky units), lactic dehydrogenase was 977 and ranged up to 1400 units during hospitalization (n = 140-280 units) and the serum glutamic oxalacetic transaminase was 78 (normal up to 30). Urine analysis revealed 3+ proteinuria. From the initial chest x-ray films (Figure 1), a fine reticular pattern in the midlung fields was interpreted as pneumonia. After blood, sputum and urine samples were obtained for culture, intravenous ampicillin

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therapy was begun. Because of progressive deterioration in the patient's condition it was discontinued two days later. Clindamycin and gentamicin were substituted but the patient remained febrile and his respiratory distress increased. On October 13, the tenth hospital day, his PaO₂ was 57 mm Hg, saturation 88%, PaCO₂ 38 and pH 7.36 while on 6 1/min nasal oxygen. Negative studies had included antinuclear factor, alpha fetoprotein, Australian antigen, LE preps, cold and febrile agglutinins and complement fixation tests for histoplasmosis and coccidiodomycosis. Sputum smear was negative for acid fast bacilli. Blood, sputum and urine cultures were negative and neutralizing antibody titres were not raised against Echo and Coxsackie viruses, PPD-T and B and mumps skin tests were negative on two occasions. A technicium 99 liver scan showed liver enlargement but no defects. An intravenous pyelogram gave normal results. Bone marrow examination revealed hyperplasia, increased iron stores and sideroblasts but no granulomas. CSF examination showed no abnormality. Smears and cultures for bacteria and acid-fast bacilli were negative.

On October 14, 11 days after admission, the chest x-ray films showed a bilateral consolidation pattern (Figure 2). Disseminated tuberculosis was suspected and isoniazid 300 mg and streptomycin 1 gram IM daily were added to the regimen. Over the next five days the man's condition became worse and he became obtunded, disoriented and oliguric. Severe hypoxemia and respiratory acid-
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osis developed (PaO₂ 35 mm Hg, PaCO₂ 57, pH 7.30 on a nonbreathing oxygen mask). On the 19th, he was intubated and placed on a volume respirator with an FjO₂ of 0.6. On that day the first positive sputum smear for AFB was obtained. The same day bloody tracheal aspirates and hematomas at sites of IM injections were noted, the hemoglobin level fell to 9.0 gm, and the peripheral blood smear showed decreased platelets, moderate poikilocytosis and anisocytosis, target cells and a few burr cells. Other laboratory data confirmed the diagnosis of consumption coagulopathy (Table I). Further treatment included a short course of continuous IV heparin 20,000 units/day, packed red cell and platelet transfusions and fresh frozen plasma.

During the next few days he made a remarkable recovery, his sensorium cleared, respiratory status improved. He became afebrile on the 22nd and was extubated on the 25th. No further bleeding occurred. By November 7 the chest x-ray showed marked improvement. Five sputum cultures and one urine culture were subsequently positive for M. tuberculosis.

When the patient was discharged in January, 1974, hematologic parameters were normal except for anemia attributed to his infection. The chest x-ray films continued to show a fine reticular pattern. No further problems occurred during two years of treatment with isoniazid, rifampin and ethambutol.

Figure 2
Bilateral consolidation pattern on 10/14/73 at the height of the illness.
TABLE I
HEMATOLOGIC DATA

<table>
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<tr>
<th>Date</th>
<th>Hb  (gms %)</th>
<th>Hct (Vol %)</th>
<th>Partial Thrombo-</th>
<th>Platelet Count</th>
<th>Blood Fibrinogin</th>
<th>Fibrin Split Products</th>
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<tr>
<td>10/3/73</td>
<td>15.8</td>
<td>48.2</td>
<td>11.5</td>
<td>—</td>
<td>172,500</td>
<td>—</td>
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<tr>
<td>10/20/73</td>
<td>9.7</td>
<td>29.9</td>
<td>17</td>
<td>37</td>
<td>—</td>
<td>—</td>
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<tr>
<td>10/21/73</td>
<td>9.0</td>
<td>28</td>
<td>3 min.</td>
<td>114</td>
<td>19.6</td>
<td>640</td>
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<tr>
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<td>32</td>
<td>14</td>
<td>107</td>
<td>36,000</td>
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<td>10/23/73</td>
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<td>29.7</td>
<td>13</td>
<td>79</td>
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<td>29.3</td>
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<td>68</td>
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<td>135</td>
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<td>27</td>
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<td>11.5</td>
<td>—</td>
<td>255,000</td>
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Discussion

Disseminated intravascular coagulation is a condition characterized by the formation of fibrin thrombi, consumption of specific plasma proteins, loss of platelets and activation of the fibrinolytic system. Clinically there may be evidence of a hemorrhagic diathesis often associated with ischemia or necrosis of vital tissues due to generalized fibrin thrombi formation. The condition has been observed in a variety of disorders such as abruptio placenta, neoplastic disease, hematologic disorders including acute leukemia and intravascular hemolysis, infections due to bacteria, viruses, fungi, parasites and rickettsiae, tissue damage occurring with burns or trauma, surgery, profound acidosis, and in shock states that can accompany massive pulmonary embolism, cardiac arrest, anaphylaxis or hemorrhage. Extensive reviews of the subject have been published. The criteria for the diagnosis include a proper clinical setting and features of hemorrhagic-thrombotic diathesis and laboratory confirmation of anemia, thrombocytopenia, reduced fibrinogen and plasma plasminogen, and increased prothrombin time and partial thromboplastin time and abnormally high levels of fibrin split products.

Disseminated intravascular coagulation secondary to miliary or other forms of tuberculosis appears to be rare, only seven cases having been reported to date in the English language literature. In recent reviews of miliary tuberculosis, no examples of DIC were mentioned among the hematologic abnormalities. In our patient DIC occurred shortly after the clinical onset of miliary tuberculosis and disappeared during the early stage of specific antituberculosis treatment together with administration of
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heparin, blood and platelet transfusions, plasma, ventilatory assistance and oxygen. The known association of disseminated intravascular coagulation with infection favors the view that, in this instance, it was initiated by infection with *M. tuberculosis*. A contributory role of respiratory insufficiency, hypoxemia and acidosis is possible. Perhaps the respiratory insufficiency could be secondary to disseminated intravascular coagulation where embolisation of fibrin and platelet thrombi to the lungs play a role. Bacterial endotoxins have been implicated in the pathogenesis of the DIC syndrome. This is particularly true of infection with meningococci, clostridia species, and gram negative bacilli. The fundamental lesion in these disorders is probably vascular injury which triggers the hemostatic mechanism. Tubercle bacilli produce neither endotoxin nor exotoxin but, in experimental animals with tuberculosis, massive thrombosis of small vessels has been demonstrated surrounding the developing tubercles.

Goldfine et al postulated that extensive fibrin thrombi form in vessels adjacent to miliary tubercles and in some cases consumption of coagulation factors might occur to a degree severe enough to cause the clinical picture of disseminated intravascular coagulation. Recently, three patients were reported with miliary tuberculosis and concomitant adult respiratory distress syndrome and disseminated intravascular coagulation. It was postulated that respiratory insufficiency was probably due to injury to alveolar capillary membrane, with loss of protein-rich fluid.
References


