Pneumococcal Sepsis with Hemolytic-Uremic Syndrome in the Adult

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Dear Sir,

Hemolytic-uremic syndrome comprises a triad of findings: acute microangiopathic hemolytic anemia, thrombocytopenia, and acute renal insufficiency [1]. In most cases of hemolytic-uremic syndrome in the adult the etiology is unknown, but a few cases have been reported as complications of typhoid fever [2], E. coli and Pseudo-monas septicemia [3, 4], and mumps [3]. We report an adult patient with the syndrome who had a fulminant pneumococcal infection. A 35-year-old man had undergone splenectomy at the age of 14 and had been in good health until the present disorder. On admission he was intermittently confused and soporose and had multiple petechiae on the anterior trunk. His temperature was 39.6 °C rectally and his initial blood pressure was normal. Laboratory analyses included the following values: hemoglobin 8.9 mmol/l (range of reference values 8–11); white blood count 7.1 × 10⁹ cells/l (3–10); thrombocytes 25 × 10¹⁰/l (135–400); prothrombin-proconvertin test 0.43 (> 0.70); serum fibrinogen 4.1 g/l (2–8); partial thromboplastin time 37 ( < 35), and creatinine 465 µmol/l ( < 120). Blood film showed schistocytes. Streptococcus pneumoniae type 8 was cultured from his blood and cerebrospinal fluid. Shortly after admission he was given high dosaged penicillin and transfusion of fresh plasma (fig. 1). On the second day diplococci were found on examination of a blood smear. Serum fibrinogen was normal. He developed unmeasurable blood pressure, respiratory insufficiency, and anuria, and was treated with mechanical ventilation, repeated hemodialysis, and transfusions of plasma and blood (fig. 1). Successively his confusion regressed, the purpura disappeared, and the hemoglobin, thrombocyte and creatinine levels normalized (fig. 1). On the 50th day of observation he was discharged in general well being.
In our patient the diagnosis of hemolytic-uremic syndrome was based on the typical triad of findings [1]. On admission, before he became hypotensive, his creatinine level was considerably increased and he had severe oliguria. Therefore acute tubulointerstitial nephropathy due to hypotension was improbable.

The decreased thrombocyte count and prothrombin-proconvertin test, and the initially increased fibrinogen-fibrin degradation products and partial thromboplastin time indicate that in the beginning of the clinical course he could have had disseminated intravascular coagulation. However, the prothrombin-proconvertin test was only moderately decreased, his serum fibrinogen level was normal, and a renal fibrinolysis as a response to the hemolytic-uremic syndrome might have contributed to the initially raised fibrinogen-fibrin split products. So disseminated intravascular coagulation contributed little to his severe renal impairment.

Splenectomized patients with pneumococcal sepsis have a poor prognosis and so have adults with hemolytic-uremic syndrome (80% mortality in one series) [2]. Our patient became critically ill but nevertheless he recovered due to early administration of high dosaged penicillin and transfusions of fresh plasma. Patients with hemolytic-uremic syndrome lack a plasma factor that inhibits platelet aggregation, and transfusions of fresh plasma provide the patient with this factor [5]. The reversion of the hemolytic-uremic syndrome after adequate treatment of the pneumococci indicates that the bacteria or products from the bacteria were essential for the hemolytic-uremic syndrome of our patient. He recovered without antiaggregatory drugs, long-term corticoid treatment, or plasmapheresis being used.

We recommend that a bacterial infection be searched for in all adult patients with hemolytic-uremic syndrome – especially intensively in those with fever or other signs of inflammation. Early and intense antibiotic treatment as well as transfusions of fresh plasma should be undertaken in splenectomized and other patients with fulminant bacterial infections and hemolytic-uremic syndrome.

References