Cryoglobulinemia After Intravesical Administration of Bacille Calmette-Guérin

BCG, an attenuated strain of Mycobacterium bovis, is commonly administered intravesically for the treatment of superficial bladder tumors. Reports of disseminated infection are rare [1]. We describe, to our knowledge, the first case of cryoglobulinemia associated with a systemic reaction after intravesical BCG therapy.

A 51-year-old man was admitted to the hospital on 4 November 1996 because of persistent fever, anorexia, and a 6-kg weight loss. He had received weekly intravesical BCG immunotherapy since September, 1 month after undergoing transurethral resection of a superficial carcinoma of the bladder. During the initial three inoculations, a transient fever was noted. The fourth attempt at catheterization of the bladder was difficult, and 30 minutes after the administration of BCG, the patient developed a fever (temperature, >40°C) with chills, diarrhea, vomiting, and hypotension. Streptococcal infection and gram-negative sepsis following catheterization were excluded. These symptoms subsided after treatment with ceftriaxone and pefloxacin for 7 days, but they recurred after administration of two additional courses of BCG therapy.

On admission to the hospital, the patient was acutely ill with a temperature of 40°C. His liver and spleen were enlarged. A rectal examination disclosed blood and a rectal ulcer. A purpuric rash was noted on both legs. Funduscopic examination revealed a left cotton wool spot. A tuberculin test was positive. Laboratory studies revealed the following values: erythrocyte sedimentation rate, 20 mm/h; C-reactive protein, 35 mg/L; ferritin, 1,984 µg/L; hemoglobin, 114 g/L; WBCs, 1,99 × 10^9/L (55% neutrophils and 39% lymphocytes); platelet count, 97 × 10^9/L; alanine aminotransferase, 152 IU/L; aspartate aminotransferase, 172 IU/L; alkaline phosphatase, 986 IU/L; α-glutamyl transpeptidase, 462 IU/L; and serum creatinine, 137 µmol/L. The prothrombin time was normal, the activated partial thromboplastin time was prolonged, the fibrinogen level was 2.6 g/L, and fibrinogen degradation products were weakly positive (40 µg/mL; normal, <20 µg/mL). IgG antipolyclonal antibody level was 64 GPL units (normal, <18 GPL units). A test for lupus anticoagulant was negative. Serology for circulating immune complexes was positive at 9.4 mg/L (normal, <7 mg/L). Serum C3 and C4 complement factors were within normal limits. The CH50 value was 58 U CH50 (normal range, 60–100 U CH50), but the C1q component was increased to 0.66 g/L (normal, 0.09–0.27 g/L), suggestive of complement consumption. Monoclonal gammopathy IgM κ and type II cryoglobulinemia were detected. Serum immunoglobulin levels were as follows: IgA, 2.9 g/L (normal level, 1-3.3 g/L); IgG, 8.9 g/L (normal level, 6.4–12 g/L); and IgM, 7.2 g/L (normal level, 0.5–1.5 g/L). Serological tests for hepatitis A, B, and C, for antibodies to HIV, and for systemic autoimmune diseases were negative.

An abdominal CT scan showed hepatosplenomegaly with splenic infarction. Colonoscopy disclosed a 4-cm rectal ulcer. Purpuric le-
Splenic Abscess and Empyema Due to Lactobacillus Species in an Immunocompetent Host

Lactobacilli are non-spore-forming, catalase-negative, gram-positive rods that are strictly or facultatively anaerobic and are part of the normal flora of the human oral cavity, vagina, and gastrointestinal tract [1]. Although generally innocuous, Lactobacillus species have been isolated in an increasing number of serious infections including endocarditis [2], gastrointestinal abscesses, and necrotizing esophagitis. A few reports of pneumonia or pleural infections due to lactobacilli exist [3, 4]; these infections were also associated with immunodeficiency. In a review of the world literature, we found only one case of lactobacillus splenic abscess, also in an immunocompromised patient [5]. We report, to our knowledge, the first case of splenic abscess and empyema due to Lactobacillus species in an immunocompetent patient.

A 69-year-old male was referred to our hospital for evaluation of left pleural effusion. He reported the presence of left pleural pain as well as night sweats for 20 days before admission. He had had a tooth extracted 7 days before presentation. His medical history was otherwise unremarkable, and there was no evidence of immunosuppression.

Physical examination revealed that the patient was in a good state of health; he did not have fever or lymphadenopathy, but he had multiple carious teeth. Findings on cardiac auscultation were normal. Lung auscultation revealed the absence of left lower lung field sounds. Other physical findings were unremarkable. Laboratory examinations showed the following data: erythrocyte sedimentation rate, 60 mm/h; hemoglobin, 13.3 g/dL; hematocrit, 41.2%; WBCs, 106/μL; and lactate dehydrogenase, 585 U/L (normal level, <160 U/L). Levels of serum urea, glucose, creatinine, amylase, transaminases, and electrolytes were normal.

Urinalysis