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Septic shock caused by *Mycobacterium tuberculosis* in a non-HIV patient

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Sir: Septic shock due to *Mycobacterium tuberculosis* is rarely reported. Most cases concern patients with proven HIV infection or who are at risk of it but have unknown serologic status [1–5]. We report a case of refractory septic shock caused by *M. tuberculosis* in a non-HIV patient leading to multiple organ failure and death.

A 44-year-old male was admitted to our intensive care unit with a combination of profound asthenia, anorexia, weight loss, acute respiratory failure, and septic shock. His past medical history included inferior myocardial infarction, tobacco and alcohol abuse, and cancer of the right tonsil considered cured 2 years previously. On admission, the physical examination revealed the following vital signs: temperature 39.5 °C, respiratory rate 22 breaths/min, blood pressure 80/45 mm Hg, and pulse 100 beats/min, after volume expansion and under dopamine (8 μg/kg per min) and dobutamine (5 μg/kg per min) infusion. Crepitant rales were found bilaterally. The patient was alert and oriented. Chest radiograph showed diffuse bilateral pulmonary infiltrates consistent with ARDS. Arterial blood gases obtained under oxygen therapy were pH 7.46, arterial carbon dioxide tension 6.3 kPa, arterial oxygen tension 7 kPa, and oxygen saturation 87 %. Lactic acid was 3.2 mmol/l. His white blood cell count was 2400/mm^3, hemoglobin 9.9 g/dl, and platelet count 96 000/mm^3. Cardiac tissue enzymes were normal. The enzyme-linked immunosorbent assay test for HIV 1 and 2 was negative. The patient was intubated, and mechanical ventilation was promptly instituted because of refractory hypoxemia. A two-dimensional Doppler transthoracic echocardiography showed left ventricular depressed systolic function without any indication for a preload increase, consistent with severe septic shock. Then the dobutamine dose was increased to 20 μg/kg per min and norepinephrine infusion was started due to persistent hypotension. Specimens of urine, blood, and bronchial secretions were sent for culture, and empirical antibiotic treatment consisting of ceftriaxone, ofloxacin, and antituberculosis drugs (rifampin, isoniazid, and ethambutol) was begun. Direct examination of bronchoalveolar lavage revealed numerous acid-fast bacilli, subsequently identified as *M. tuberculosis* susceptible to the drugs given. Routine blood and urine cultures were negative. Despite antituberculous therapy, mechanical ventilation, and maximum use of catecholamine combination therapy, his condition continued to worsen and he died on day 3 in multiple organ failure. No autopsy was performed.

To date, nine well-documented cases of septic shock due to *M. tuberculosis* have been reported in the English literature. Tumor necrosis factor production from mycobacterial products is assumed to be responsible for septic shock hemodynamic alteration [2]. Of these nine cases, seven were HIV-infected patients [1, 3–5], and two were considered to be at high risk for HIV disease, although testing had not been performed [2]. It is well known that adults infected by HIV have increased susceptibility to *M. tuberculosis* and progress more rapidly to disease. Conversely, the case presented herein is one of the first reported of hemodynamic septic shock due to *M. tuberculosis* in a patient without HIV. Because two-dimensional echocardiography is considered a reliable technique to evaluate left ventricle preload and contractility in septic shock [6], we did not used pulmonary artery catheterization to monitor our patient's hemodynamic profile. For this patient, with a previous history of cancer, several weeks of weight loss, anorexia, asthenia, combined with respiratory symptoms and chest radiographic aspects on admission led to the diagnosis. However, despite initiation of prompt and appropriate empirical antibiotic therapy and maximum supportive therapy, the infection proved refractory and the patient died.

We conclude that *M. tuberculosis* should be considered in the differential diagnosis of septic shock, even in non-HIV patients, especially in patients with other causes of immunosuppression such as cancer or transplantation.

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