These two cases underline the potentially pathogenic role of *Staphylococcus capitis* particularly in patients with predisposing factors such as cardiac valve disease.

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References


A Case of Fatal Septicemia due to *Mycobacterium kansasii*

The Ostrava-Karviná region in the Czech Republic is an endemic area for *Mycobacterium kansasii* infection with an increased incidence since 1968 (1, 2). Like other endemic areas in the world, the region is characterized by a concentration of heavy industry and a high population density. Until 1990, the disease had been diagnosed in more than 1000 patients in the Ostrava-Karviná region, presenting in most cases as pulmonary involvement. We report an uncommon case of extrapulmonary *Mycobacterium kansasii* infection in a young man from a district in the endemic area which led to the patient’s death.

The 20-year-old man with primary myelodysplastic syndrome was admitted to the Department of Internal Medicine of the University Hospital in Ostrava in January 1991 with a diagnosis of cryptogenic septicemia. He reported a high temperature of about 40 °C in the previous four weeks and diarrhea without mucous or blood in the feces. Six months before admission the patient had been treated for knee and wrist arthritis with anti-rheumatic drugs and low doses of glucocorticoids (10 mg of prednisone per day). Immediately before admission, the patient had been treated with high doses of glucocorticoids (80 mg of prednisone per day) due to anemia, leukopenia and thrombocytopenia in connection with a primary myelodysplastic syndrome. At the same time he was treated with antibiotics (ampicillin, lincomycin) due to septicemia.

In spite of the intensive treatment with antibiotics after admission, high fever (about 39 °C) persisted. He was treated with ketoconazole, cefazoline and gentamicin, thereafter combined with cefotaxime, vancomycin and imipenem, and eventually with a combination of miconazole and rifampicin. The marked anemia, leukopenia and throm-
bocytopenia were treated with repeated blood transfusions. The patient died after six weeks in hospital.

Four weeks before death splenomegaly occurred. The patient had lost 9 kg in weight, had severe anemia (hemoglobin concentration 50-100 g/l), a leukocyte count of $1.8 \times 10^9$-6.0 $\times 10^9$, (at death 0.7 $\times 10^9$--0.8 $\times 10^9$), thrombocytopenia (7.0 $\times 10^9$--35.0 $\times 10^9$) and a significant shift to the left in the differential blood count (78--85 %). The erythrocyte sedimentation rate varied in the range of 28--78 mm. Biochemical tests showed serious malnutrition and hypercatabolism. Ultrasonography and computerized tomography showed hepatosplenomegaly, small abscess foci in the liver and spleen preterminally, and a minor degree of ascites. Heart and chest X-rays were repeatedly negative. Autopsy was carried out 15 hours after death. Most of the changes were detected in the abdominal cavity organs, especially in the liver and spleen. The spleen was conspicuously enlarged (weight 950 g, size 22 x 16 x 5 cm) and had a smooth capsule. The liver was also markedly enlarged. There were no pathological changes in other organs with the exception of anemia and local bleeding, especially in the kidneys.

Histological examination of the liver and spleen showed numerous abscess cavities containing many polymorphonuclear cells and the presence of a great number of acid-fast bacilli. The findings in bone marrow were compatible with the diagnosis of myelodysplastic syndrome.

The blood cultures were repeatedly negative. Immunological investigations showed significantly decreased immune responses and signs of inflammation. Tests for anti-HIV antibodies one week before death were negative. The first investigations for mycobacteria were performed on sternal bone marrow specimens taken one day prior to the patient's death. Smears examined by the fluorescence method were negative. Culture on three media inoculated without decontamination (liquid Šula medium, solid Loewenstein-Jensen and Ogawa media) yielded heavy growth (> 100 colonies) suspected of being Mycobacterium kansasii after ten days. After the patient's death, autopsy samples of the liver and spleen were homogenized and decontaminated by the Petroff method, a swab from the bone marrow was decontaminated by the HCl and NaOH method, and the blood sample was inoculated without decontamination. Smears examined by the fluorescence and Ziehl-Neelsen staining techniques revealed a large number of acid-fast organisms, and cultures were positive (> 100 colonies) after 7 to 10 days of incubation at 37 °C. All strains isolated were identified according to the standard Czechoslovakian methods (3) as Mycobacterium kansasii. The strains were sensitive to streptomycin (10 mg/l), pyrazinamide (400 mg/l), ethambutol (2 mg/l), rifampicin (5 mg/l), ofloxacin (MIC 0.25 mg/l), gentamicin (MIC 2 mg/l) and amikacin (MIC 0.5 mg/l), and resistant to isoniazid (0.2 mg/l).

Diagnosis of intraabdominal tuberculosis is usually difficult because in less than 50 % of cases pulmonary tuberculosis is present (4, 5). In our patient the chest X-rays were repeatedly negative. The possibility of a tuberculous etiology was considered only after unsuccessful treatment with antibiotics and other chemotherapeutic agents in the last eight weeks before the patient died. Because direct microscopic examination of the sternal bone marrow before the patient's death was negative, the suspected mycobacterial infection was confirmed only after death by culture of blood and sternal bone marrow obtained while the patient was alive. The cause of death was Mycobacterium kansasii septicemia in this patient with myelodysplastic syndrome. The strain showed good susceptibility to all antimicrobial agents tested in vitro, with the exception of isoniazid. In spite of the fact that two of the agents tested were administered to the patient in combination with other agents, therapy failed, probably due to the short period of treatment and the combination used.

The source of infection in our patient was not found. He worked as an electrician in a factory and there were no other cases of Mycobacterium kansasii infection in his working collective. The development of infection was probably facilitated in this case by the decreased cellular immunity as a consequence of the primary myelodysplastic syndrome and the long-term treatment with high doses of glucocorticoids (6, 7).

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