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Fatal septic shock due to *Gemella morbillorum* in two HIV-positive patients

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Bacterial infections have been appreciated as frequent and important causes of morbidity and mortality in people with HIV infection. *Gemella morbillorum* is a component of the oropharyngeal commensal flora, rarely responsible for clinically important infections similar to those due to viridans streptococci. Endocarditis and bacteremia are most frequently seen, but septic arthritis and meningitis have also been observed [1,2]. We report two cases of infection, eventually fatal, caused by *G. morbillorum* in HIV-positive patients.

Case 1: A 35-year-old man, an intravenous drug abuser with AIDS (previous *Pneumocystis carinii* pneumonia – PCP), was admitted to our hospital because of fever, cough and dyspnea. The patient was not receiving anti-HIV treatment but only chemoprophylaxis with trimethoprim-sulfamethoxazole (TMP-SMZ). On admission, his temperature was 38.8 °C, blood pressure 105/60 mmHg, pulse 136/min and respiratory rate 28/min. The level of consciousness was reduced, but there was no neck stiffness. Laboratory investigations showed raised ESR, anaemia (hemoglobin = 9.1 g/dL) and leukocytosis (WBC = 18×10^9/L). Chest radiography demonstrated an infiltrate of variable opacity in the right lower lobe. Arterial blood gases revealed hypoxemia (PaO_2 = 57 mmHg). Bronchoscopy was not performed, because of the poor condition of the patient. Three sets of blood cultures and urine and sputum specimens were obtained for microbiological examination. Blood cultures were collected within a 24-h period using BACTEC Plus (Becton Dickinson, Sparks, Maryland, USA) and Isolator lysis-centrifugation system (du Pont de Nemours & Co, Wilmington, Delaware, USA). Because few squamous epithelial cells (< 25 per low-power field) were present in the specimens of sputum, the smear was examined under oil immersion (×1000) and the specimen was cultured on blood agar. Empirical antibiotic therapy was started with intravenous piperacillin-tazobactam (4–0.5 g IV t.i.d.). After 24 h, blood and sputum cultures on blood agar yielded slow-growing Gram-positive bacteria that were identified as *G. morbillorum* by the API 20 Strep system (Bio-Mérieux, Lyon, France) and by the nitrite test (the isolates transformed nitrites with production of gas (nitrogen)). The isolate was susceptible in vitro to penicillin G, ampicillin, ureidopenicillins, chloramphenicol, trimethoprim-sulfamethoxazole, erythromycin, rifampin, aminoglycosides and tetracyclines. No other possible causative organism was revealed by blood, urine and sputum cultures. Despite therapy, the patient worsened rapidly and died after 8 days because of septic shock. Autopsy examination showed a massive necrotizing pneumonia of the lower and middle right lobes associated with a fibrino-haemorrhagic pleuritis. Multiple, small abscesses were also found in the left lung. Microscopy disclosed the presence of a large number of Gram-positive bacteria. Microbiological tests on suppurative material removed at autopsy revealed the presence of *G. morbillorum*. No other opportunistic agent was found.

Case 2: A 31-year-old man with AIDS (previous esophageal candidiasis) was hospitalized because of fever, productive cough and cachexia. One week before admission a chest X-ray had revealed a pneumonia of the right lower lobe. Viridans streptococci, subsequently identified as *G. morbillorum*, had been isolated from serial sputum cultures. No other organism was identified. At that time the patient had been receiving zidovudine treatment for 6 months and chemoprophylaxis for PCP with TMP-SMZ. Oral erythromycin therapy had been started, but the patient's clinical conditions had worsened. On admission, the patient was afebrile, his blood pressure was 90/50 mmHg, pulse rate 130/min and respiratory rate 30/min. He was confused and delirious, but without any signs of meningism or focal neurologic findings. Laboratory tests revealed marked leukocytosis

(WBC = 19.7×10^9/L) and severe hypoxemia (PaO_2 = 38 mmHg). A chest X-ray showed an extensive density in the right lung associated with pleural effusion. A small infiltrate was also present in the juxta-diaphragmatic left pulmonary region. Blood cultures obtained on admission grew Gram-positive bacteria, identified as *G. morbillorum* by the same methods indicated above for the first case, with a pattern of in vitro susceptibility similar to the previous patient. Notwithstanding an aggressive therapy including fluid replacement, oxygen, vasopressors and antibiotics (ampicillin and netilmicin), the patient died 5 h later with the clinical picture of septic shock. Autopsy showed massive suppurative lesions involving both lungs, and microscopy demonstrated a large number of Gram-positive bacteria. The microbiological examination of purulent material removed from the lungs identified the presence of *G. morbillorum*. Also in this case no other microorganism was present.

Among HIV-positive patients, the most frequently isolated bacteria are *Streptococcus pneumoniae, Haemophilus influenzae, Staphylococcus aureus* and *Pseudomonas aeruginosa* [3,4]. These common pathogens often cause diseases which may present in unusual and particularly severe ways and sometimes require diagnostic and therapeutic approaches different from those used in patients not infected with HIV.

In 1988, *Streptococcus morbillorum* was reclassified as *Gemella morbillorum* joining *Gemella haemolysans* as the second member of this genus [5]. It has already been demonstrated that *G. morbillorum* can cause severe infections in the immunocompromised host. In fact, this microorganism has been identified as the etiologic agent in 13% of cases of septicemia among cancer patients [6].

To our knowledge, no previous reports are available on infection due to *G. morbillorum* in HIV-positive patients. In the cases described here, the isolation of *G. morbillorum* from blood and sputum indicates that the microorganism was the main pathogen in this infection, responsible for the severe lung disease and the subsequent septic shock. The immune deficiency associated with HIV infection could represent a condition predisposing to increased mortality in *G. morbillorum* infection because, as far as we are aware, only one lethal case of this disease has been reported [2].

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Postoperative *Cryptococcus neoformans* endocarditis

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Although fungal endocarditis is known to occur in patients with underlying heart disease [1,2], reports of mycotic cardiac infections have increased with the development of cardiovascular surgery [3]. Fungal endocarditis accounts for 6% [4] to 20% [5] of secondary infections following open-heart operations. However, only two of eight *Cryptococcus neoformans* endocarditis previously reported were postoperative [6,7]. We report a case of postoperative *C. neoformans* endocarditis, successfully managed by reoperation and combined amphotericin B/fluconazole therapy.

Case report: A 12-year-old male Vietnamese HIV-negative child presented to Ho-Chi-Min-Ville Institut du Coeur with severe malnutrition (22 kg, 135 cm) and terminal cardiac failure (NYHA 4), due to mitral and tricuspid valvulitis secondary to rheumatic heart disease without vegetations. Immediate mitral annuloplasty and tricuspid conservative plasty were performed. Four months later, fever (38 to 39 °C), conjunctival and cutaneous petechiae, hepatomegaly and severe anemia occurred (there was no lymphopenia and lymphocyte subsets were not differentiated). Echocardiography (EC) revealed a vegetation on the mitral valve. There was no obvious portal of entry: chest X-ray was normal and repeated blood cultures were negative. In spite of antibiotics, the patient remained febrile and 19 days later cerebral thromboembolism occurred. EC demonstrated three polypoid, mobile vegetations on the mitral valve (Figure 1). After a further week, two additional