(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].

Cristina Gervasoni, Annalisa Ridolfo,
Spinello Antinori, Teresa Bini, Roberto Esposito
Clinic of Infectious Diseases,
University of Milan
L. Sacco Hospital, Via G.B. Grassi 74
20157 - Milan - Italy.

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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
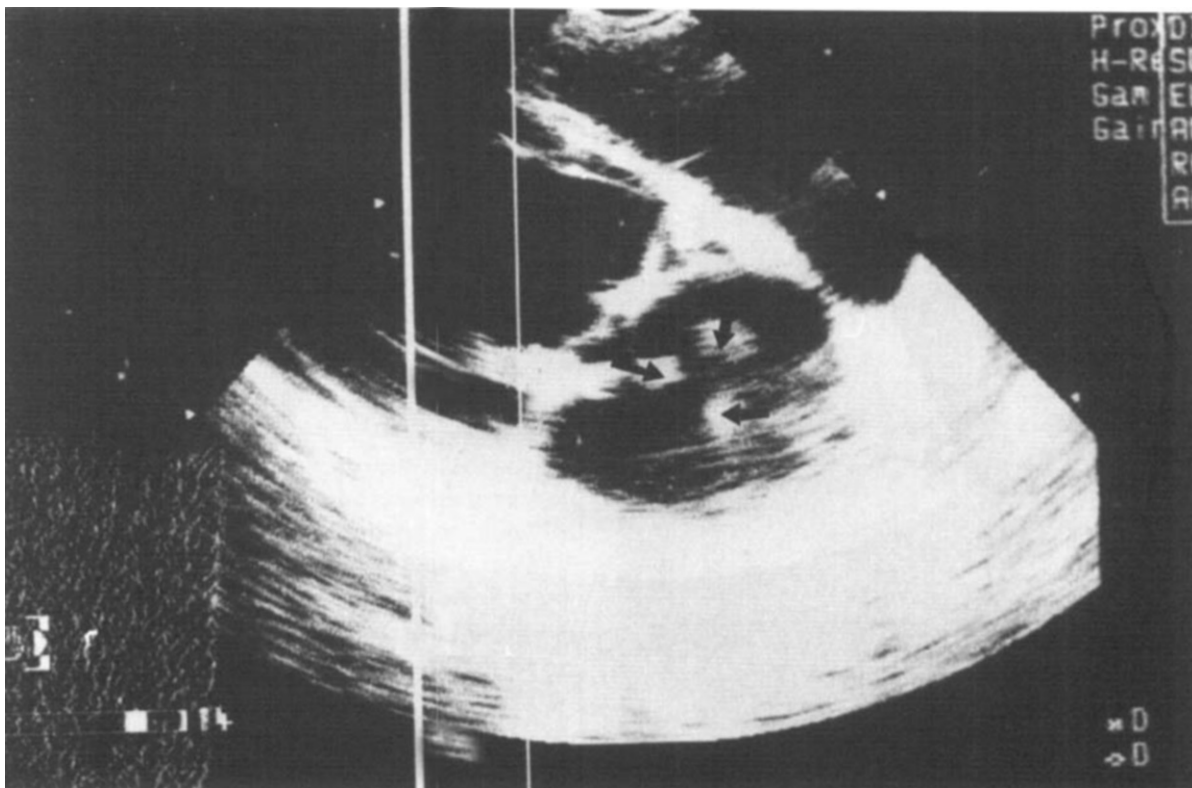
Figure 1 Second echocardiography showing three polypoid vegetations of the mitral valve, extending onto the left atrium (arrow)

vegetations appeared. Fifty blood cultures on usual bacteriologic media were performed between the two EC examinations; only three of these later grew yeasts. Lipidic infusions (Intralipid®, Pharmacia) of amphotericin B 1.5 mg/kg/day were started and the original mitral valve was replaced 72 h later with a 27-mm mechanical allocarbon valvular prosthesis. Gram- and May-Grunwald-Giemsa-stained smears from the removed valve and blood cultures disclosed encapsulated single budding yeasts. Cultures yielded *C. neoformans* serotype A. The isolate was sensitive in vitro to amphotericin B, flucytosine (Sanofi diagnostic Pasteur disks on solid media Casitone and YNB), and fluconazole (RPMI broth medium). Despite amphotericin B therapy, the patient remained febrile, and 1 month later neurologic signs were noted. Post-contrast computed tomographic (CT) scan of the brain showed edema and two enhanced areas of suppurative encephalitis suggestive of cryptococcal abcesses (Figure 2). A serum cryptococcal antigen test (SCA) (Pastorex Cryptococcus, Sanofi diagnostic Pasteur) was positive at 1:10. After a further month, the SCA was still positive at 1:10 and cerebral CT scan showed extended lesions. Fluconazole 400 mg/day was added to

amphotericin B, leading to apyrexia within 7 days and regression of cerebral lesions on a new CT. The SCA decreased to 1:4. Over a total period of 3 months, 3.960 mg of amphotericin B was given; this was discontinued when the patient remained afebrile and blood cultures remained sterile. Fluconazole was maintained for a further 6 months. Despite high dosage of fluconazole no side effects were observed. One year after termination of antifungal therapy, SCA was undetectable in cerebrospinal fluid and serum and there had been no recurrence of infection.

While the incidence of cryptococcosis has markedly increased in recent years because of its frequent occurrence in patients with AIDS, the infection may also occur in other patients with impaired cell-mediated immunity, such as patients with lymphoma or leukemia, or patients receiving immunosuppressive therapy [8]. Moreover, as observed in 50% of cases before the AIDS epidemic, cryptococcosis may occur in patients with no obvious immune deficiency, although malnutrition, as observed in our patient, may be considered as a subtle defect in the ability to mount an immune response to cryptococcal antigens [8]. Myocardial cryptococcal abscesses have been reported
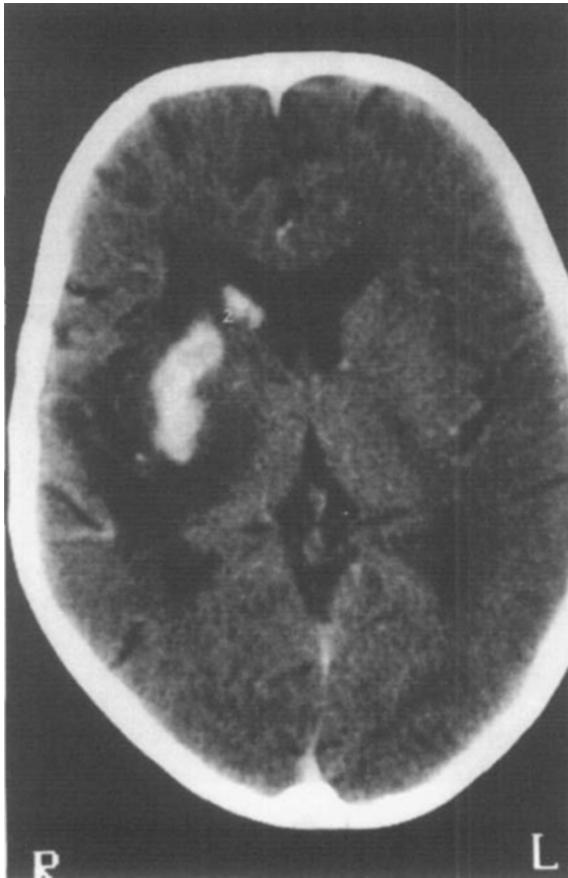
Figure 2 First post-contrast CT scan of the brain showing two enhanced areas of suppurative encephalitis.

fungal etiology, but symptomatology did not permit etiologic differen-tiation from bacterial endocarditis. Since numerous blood cultures were sterile, diagnosis and treatment were delayed. Early surgical reintervention is necessary for eradication of fungal cardiac infections after valve operation [7,16]. When fungal endocarditis had been diagnosed in our patient, amphotericin B was started immediately and he underwent reoperation 72 h later. Nevertheless, systemic dissemination occurred, with probable cerebral involvement, as evidenced by the CT scan. Recovery was obtained with the addition of fluconazole, a triazole agent that is now recognized as an effective alternative to amphotericin B as treatment of cryptococcal meningitis [17]. Such treatment of *C. neoformans* endocarditis complicating valve operations has not been reported previously.

This unusual case confirms the difficulties of diagnosis in fungal cardiac infections and the value of reoperation and agressive medical therapy in order to salvage patients with postoperative fungal endocarditis.

Véronique Blanc[a], *Véronique Lavarde*[a],
Nguyen Thi Thanh[b], *Ho Huynh Quang Tri*[b],
Romain Guillemain[c], *Catherine Amrein*[c]
and Alain Carpentier[c]
[a]Service de Microbiologie, Hôpital Broussais,
96 rue Didot, 75674 Paris Cedex 14, France;
[b]Institut du Coeur, Ho-Chi-Minh Ville, Vietnam;
[c]Service de Chirurgie Cardiaque,
Hôpital Broussais, Paris, France

in AIDS [9] and non-AIDS patients [2,10,11]. Cryptococcal endocarditis is extremely rare, only eight cases having been reported previously [1,6,7,12–15]. Two of these cases complicated open-heart surgery [6,7]. One patient developed fatal *C. neoformans* endocarditis more than 2 years after aortic valve replacement. Fungal endocarditis was diagnosed ante mortem but amphotericin B alone was insufficient to cure the patient [6]. In the other case, cryptococcal pancarditis was diagnosed 10 days after aortic replacement and was successfully treated with combined amphotericin B/flucytosine therapy and reoperation [7]. In the present report, as has previously been described, some of the usual features of infective endocarditis were missing, such as splenomegaly, cardiac enlargement, new heart murmur, hematuria or leukocytosis [1,2]. Prior cardiac surgery was the main predisposing factor in the patient's history that alerted physicians to the possibility of infective endocarditis. The 4-month delay between open-heart surgery and onset of clinical manifestations was compatible with a

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