Native Valve Endocarditis Due to Mycobacterium fortuitum biovar fortuitum: Case Report and Review

Rapidly growing mycobacteria such as Mycobacterium fortuitum and Mycobacterium chelonae are well-known causes of skin and soft tissue infections. Endocarditis caused by these organisms is rare and usually involves prosthetic valve infections with M. chelonae. We describe an HIV-infected injection drug user who developed native valve endocarditis due to M. fortuitum biovar fortuitum and review the literature on all published cases of M fortuitum endocarditis. To our knowledge, this is the first case of native-valve endocarditis caused by M. fortuitum biovar fortuitum.

A 47-year-old man who used injection drugs was admitted to our hospital for evaluation of dysphagia, odynophagia, fever, and chills of 1 week’s duration. Initial examination revealed a temperature of 38.9°C and oral thrush. Laboratory evaluation showed the following values: WBCs, 3.7 × 10⁹/L; hemoglobin, 9.9 g/dL; and platelets, 67 × 10⁹/L. The patient was treated with fluconazole for presumed candidal esophagitis, and his condition subjectively improved within 4 days.

Further evaluation revealed HIV infection and a CD4 cell count of 60/mm³. Two sets of cultures of blood obtained on admission yielded acid-fast organisms on hospital day 6. Empiric therapy for Mycobacterium avium complex infection was initiated with clarithromycin, ethambutol, and rifabutin. Two days later, a new, 2/6 diastolic murmur was auscultated. Transcatheter echocardiography showed mild aortic insufficiency with no evidence of vegetations. The acid-fast organism isolated from the blood was then identified as Mycobacterium fortuitum, and the antibiotic regimen was changed to amikacin, cefoxitin, and ciprofloxacin. Three weeks later, repeated transsthoracic echocardiography displayed peduncular vegetations on the left coronary, noncoronary, and right coronary cusps of the aortic valve.

Although the patient was offered valve replacement, he preferred medical management. His fever and chills began to abate, and cultures of blood obtained after 4 weeks of therapy were sterile. Therapy with amikacin, cefoxitin, and ciprofloxacin was continued for a total of 6 weeks. The patient was discharged and continued to receive oral ciprofloxacin and trimethoprim-sulfamethoxazole (TMP-SMZ). Despite complying with his medication regimen, the patient died 12 weeks after his initial clinical presentation. Permission for autopsy was not granted. A total of 7 sets of cultures of blood obtained on 3 separate

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days yielded isolates that were identified as *M. fortuitum*. The Centers for Disease Control and Prevention (Atlanta, GA) confirmed this identification. The isolate proved susceptible to amikacin, cefoxitin, ciprofloxacin, imipenem, and TMP-SMZ, as well as resistant to clarithromycin, doxycycline, erythromycin, and tobramycin. The University of Texas Health Center at Dallas yielded isolates that were identified as *M. fortuitum*.

Endocarditis caused by *M. fortuitum* is exceedingly rare. The literature records only 7 cases of endocarditis due to *M. fortuitum* [1–6], and 6 of the 7 cases describe infections of a prosthetic valve. The onset of symptoms in these 6 patients occurred 4 weeks to 11 months after valve replacement surgery. All 6 patients died, despite antimicrobial therapy. Five of the 6 patients also underwent another valve replacement [1–5].

The other case of *M. fortuitum* endocarditis was reported by Singh et al. in 1992 [6]. They describe a 54-year-old woman with aortic stenosis who was receiving chronic hemodialysis. The patient’s blood cultures yielded *M. fortuitum*, but no vegetations were noted on echocardiography. Despite treatment with multiple antibiotics, the patient died a few weeks later. Autopsy revealed calcified aortic valve vegetations that contained acid-fast organisms identified as *M. fortuitum*.

Our patient’s case is the first reported case of native valve endocarditis due to *M. fortuitum* biovar fortuitum. It is also the first reported case of native valve *M. fortuitum* endocarditis in a patient with no previous history of abnormal valves or heart murmur. The only other report of native valve *M. fortuitum* endocarditis describes a patient with aortic valve vegetations and a history of aortic stenosis [6].

Our report of *M. fortuitum* endocarditis is also the first with visible vegetations on echocardiography. Four of the previous 7 cases were reported before the routine use of echocardiography and do not mention echocardiographic findings [1–3]. Two of the 7 cases mention that there were no visible vegetations on echocardiography [4, 6]. Chow et al. describe an aortic root abscess due to *M. fortuitum* that was diagnosed with echocardiography, but vegetations were not seen until autopsy [5].

Our case also represents the first report of *M. fortuitum* endocarditis in a patient infected with HIV. Sack [7] reasons that humoral factors may play a more significant role in response to infection with *M. fortuitum*, since B cells may be stimulated directly by large mycobacterial antigens, and so initiate an antibody response without a T cell signal. Intact direct B cell response may explain why endocarditis due to *M. fortuitum* is so uncommon in injection drug users infected with HIV, despite repeated skin injections and a T helper cell deficiency.

Although our patient died, he survived for 12 weeks after his initial presentation, the longest reported survival of any patient diagnosed with *M. fortuitum* endocarditis. This grave prognosis reinforces the importance of considering mycobacteria such as *M. fortuitum* in the evaluation of endocarditis and also considering endocarditis in the evaluation of *M. fortuitum* bacteremia.

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**References**


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**Isolation of Capnocytophaga granulosa from an Abscess in an Immunocompetent Adolescent**

The genus *Capnocytophaga* includes 7 species (*Capnocytophaga ochracea, Capnocytophaga gingivalis, Capnocytophaga sputigena, Capnocytophaga haemolytica, Capnocytophaga granulosa, Capnocytophaga canimorsus*, and *Capnocytophaga cynodegmi*) characterized as gram-negative, fastidious, cappophilic rods with fusiform morphology and gliding motility. *C. ochracea, C. sputigena, C. gingivalis*, and, recently, *C. haemolytica* and also *C. granulosa* have been isolated from the human oral cavity and therefore may be considered members of the normal flora [1–3]. *Capnocytophaga* species other than *C. granulosa* have been reported as a cause of bacteremia and sepsis, as well as localized infections, in immunocompromised patients. In immunocompetent hosts, these *Capnocytophaga* species are oc-