

Native Double-Valve Endocarditis by *Mycobacterium fortuitum* following Percutaneous Coronary Intervention

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Infective endocarditis caused by atypical mycobacteria, especially *Mycobacterium fortuitum*, is rare. Most reported cases involve diseased valves or prosthetic heart valves. Only four cases of native valve endocarditis caused by this organism have been previously reported. Herein is reported a case of native valve

Infective endocarditis caused by atypical mycobacteria, especially *Mycobacterium fortuitum*, is rare (1), with only four cases of native valve endocarditis caused by this organism having been reported previously (2). Herein is reported a case of native valve endocarditis caused by *M. fortuitum*; the epidemiology and management of this rare cause of culture-negative endocarditis are also highlighted.

Case report

A 50-year-old male (body weight 75 kg) presented to the present authors' institution with a seven-week history of fever and chills, and non-progressive dyspnea on exertion of four weeks' duration. The fever was high grade, intermittent with one or two spikes per day, but the patient had no other significant complaints. He had sustained an anterior wall myocardial infarction eight weeks prior to admission, and had undergone (at another institution) a drug-eluting stent insertion to the left anterior descending and obtuse marginal arteries. The fever had started two days after this procedure.

On examination, the patient appeared unwell, but was hemodynamically stable and maintaining oxygen saturation. The liver and spleen were enlarged, but the results of routine laboratory tests were unremarkable. Transesophageal echocardiography revealed multiple

endocarditis caused by *M. fortuitum*; the epidemiology and management of this rare cause of culture-negative endocarditis is discussed.

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freely moving flagellar masses attached to the anterior mitral leaflet, without mitral regurgitation, and smaller masses on the aortic side of the non-coronary cusp of the aortic valve. Routine cultures (including multiple blood cultures) were taken, and the patient was commenced on intravenous vancomycin and gentamicin; the valve lesions were followed up by serial echocardiography. Serial blood, sputum, urine and fungal cultures each failed to grow any organism, however, and the patient remained febrile. After 12 days, and in consultation with the institution's microbiologist, the antibiotic regimen was changed to linezolid and ciprofloxacin (fluorinated quinolone); this was administered for another two weeks, but without relief of fever. During this time, serial echocardiography showed that the valve lesions had not progressed.

Subsequent serial cultures remained negative, and special cultures for HACEK organisms were sterile. Fortunately, mycobacterial blood cultures were also performed on Lowenstein-Johnson medium and using the BACTEC 460 radiometric system (Becton Dickinson SA). These cultures grew mycobacterium at two weeks; these microorganisms grew rapidly in the presence of *p*-nitro-acetylamine-hydroxypropopnone (NAP test), indicating the presence of *M. fortuitum*. Antibiotic susceptibility revealed sensitivities to amikacin, ciprofloxacin, and imipenam. Quantitative antibiotic susceptibility tests were not conducted.

The patient was commenced on intravenous imipenam (1 g, q.d.) and intravenous ciprofloxacin (400 mg, b.d.). On the sixth day of this regimen the patient became afebrile, but echocardiography conducted at this time revealed increasingly severe mitral and aortic regurgitation. The patient was taken for sur-

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Table I: Summary of cases of native valve endocarditis caused by *M. fortuitum*.

Authors (Reference)	Patient age (years)	Valve affected	Previous procedure	Outcome
Galil et al. (7)	66	Tricuspid	Hemodialysis	Death
Singh et al. (8)	54	Aortic	Hemodialysis	Death
Spell et al. (9)	47	Aortic	Urethral dilatation	Death
Kuruville et al. (10)	20	Mitral	Balloon mitral valvotomy	Death

gery 18 days after starting appropriate antibiotics, at which time a double-valve replacement was performed, using mechanical valves. The operative findings included large flagellar masses on the anterior mitral leaflet and the non-coronary aortic cusp, cuspal perforation of the non-coronary cusp, and no evidence of local spread of infection. On day 1 postoperatively, the patient developed generalized tonic-clonic convulsions. Fundoscopy, cerebrospinal fluid analysis and serial computed tomography scans of the brain were normal, and the convulsions subsided with a reduction in the imipenam and ciprofloxacin doses. Cultures of both excised valves were sterile.

The patient made an uneventful recovery and was discharged on day 15, with the plan to continue intravenous imipenam and ciprofloxacin for a further six weeks.

Discussion

Mycobacterium fortuitum is a member of group IV of Runyon's classification of atypical mycobacteria. The organism can cause a spectrum of lesions in different organs (3), and is also a rare (4) but recognized cause of various infections in the post-cardiac surgical patient (5). Endocarditis caused by *M. fortuitum* is a rare occurrence (2), with only about 20 cases having been reported to date.

In approximately 80% of these cases, the infection occurs on prosthetic valves (either mechanical or biological), and leads to fulminant endocarditis requiring valve explantation. Only rarely is conservative management successful (6). Of note, 85% of patients with prosthetic valve endocarditis due to *M. fortuitum* have died in spite of diagnosis, antibiotic therapy, and valve excision. Native valve endocarditis caused by this organism is even more rare, with only four cases having been reported before the present case (7-10) (Table I). To date, only one patient has survived the infection; this patient had isolated tricuspid endocarditis as a part of disseminated atypical mycobacterial infection that did not require valve replacement.

A nosocomial origin for prosthetic valve infections is

supported by reports of the isolation of *M. fortuitum* in cardioplegia solutions (5), operating room water (3), and valve preservatives (11). A water-based origin of the pathogen in cases of native valve involvement is possible, as all cases have either followed hemodialysis, chronic intravenous access, urethral dilatation or balloon mitral valvotomy. In the present patient, culture of the excised valves did not grow any organism. In general, cultures of valves excised due to active endocarditis will grow organisms if appropriate microbiological techniques are employed (12); this currently includes the use of specific polymerase chain reactions (PCR) to detect bacterial antigens (13). The present patient was operated on 18 days after starting specific antibiotics against *M. fortuitum*, and this may have been the reason for the negative valve culture obtained. PCR techniques used to detect bacteria on the excised valves were not performed due to their non-availability in the authors' country. Hence, it might be questioned as to whether or not *M. fortuitum* was in fact the causative organism in this case.

In the past, infection with *M. fortuitum* has been treated with anti-tuberculous drugs (1), although drug resistance was problematic. More recently, a spectrum of antibiotics has been recommended (14), namely, amikacin, imipenam, cefoxitin, clarithromycin and fluorinated quinolones, and those patients who have survived have in general received these antibiotics (2).

In conclusion, *M. fortuitum* must be considered as a cause of culture-negative endocarditis, both after valve replacement and in cases of endocarditis following other hospital procedures. Although the current literature indicates a grim prognosis with this infection, it is probable that with awareness and earlier diagnosis, valve debridement with or without valve replacement, and the use of appropriate antibiotics, the mortality rate of this infection can be reduced. Since it is poor aseptic technique which allows *M. fortuitum* to colonize fluids and gain access to the patient, ultimately causing endocarditis, this disease should be preventable.

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