Case Report

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In July 2007, a 70-year-old woman was transferred to our hospital from a local district general hospital for consideration of insertion of an implantable cardio-defibrillator to control intermittent non-sustained ventricular tachycardia. She had undergone a CarboMedics mechanical aortic valve replacement 5 weeks previously, which was complicated by a right ventricular puncture. Her past medical history included a Bjork–Shiley mechanical mitral valve replacement 16 years earlier, left ventricular impairment, chronic renal impairment and long-term, mild confusion.

Following transfer to our hospital, she had persistent low-grade pyrexia, a normal white cell count but an elevated C-reactive protein level of 65 mg l\(^{-1}\) and an erythrocyte sedimentation rate of 45 mm h\(^{-1}\). Four sets of blood cultures sent 5, 6 and 7 days after transfer all grew Gram-negative bacilli in the aerobic bottle. The blood cultures were incubated using the BACTEC 9240 (Becton Dickinson) and investigated following the standard laboratory operating procedure with Gram staining and subculture onto appropriate solid agar. The isolates from each of the positive blood cultures were all oxidase-positive and appeared to be identical. Further identification was performed in-house using multipoint methods (Pease et al., 1988) and the commercially available identification system API 20NE (bioMérieux). These tests identified the isolates as *Pseudomonas* species and *Pseudomonas fluorescens* (API profile 1146457), respectively. The isolates were further characterized by the reference laboratory (Colindale, UK) and subsequently confirmed to be *Pseudomonas mosselii* by gas chromatography of fatty acids and partial sequencing of the 16S rRNA gene. Breakpoint testing demonstrated the organism to be susceptible to gentamicin, piperacillin–tazobactam, ceftazidime, meropenem and ciprofloxacin.

Given the clinical history of cardiac valve replacements, a working diagnosis of prosthetic valve endocarditis was made. Prior to subculture of the positive blood cultures, the patient was initially commenced on meropenem 1 g three times daily. Following identification of the organism to genus level and susceptibility results, the meropenem was changed to piperacillin–tazobactam 4.5 g three times daily. Gentamicin 80 mg three times daily was added initially but there was delayed excretion due to mild renal impairment. It was decided that the risks of aminoglycoside therapy exceeded the possible benefits and the drug was discontinued after 3 days.

Investigations included a chest X-ray and CT scan, which showed no evidence of pneumonia; a CT scan of the abdomen and pelvis, which showed no collections or evidence of bony infection; urine cultures, which isolated enterococci; and transthoracic (TTE) and transoesophageal (TOE) echocardiograms. Both the TTE and the TOE demonstrated moderate para-prosthetic aortic regurgitation but no vegetations. Despite the lack of vegetations, prosthetic valve endocarditis remained the most likely diagnosis in view of the new valvar regurgitation, the prolonged bacteraemia and a failure to find any other focus of infection.

In view of the poor prognosis of pseudomonal prosthetic valve endocarditis (Fowler et al., 2005), a review of the valve replacement was considered. The patient’s frailty and co-morbidity, however, implied that surgery would carry an unacceptable risk of adverse outcome and management remained conservative with the continuation of intravenous antibiotics for a 6 week course. On the fifth week of antibiotic treatment, the patient developed transaminitis, which, though not proven, may have been attributable to treatment with piperacillin–tazobactam; this was therefore changed to ceftazidime 1 g three times daily to complete the final week of treatment.

Following discontinuation of the antibiotics, multiple sets of blood cultures remained negative. Further recovery was slowed by left ventricular impairment and acute-on-chronic renal failure secondary to renal hypoperfusion. The patient was eventually discharged from hospital 2 weeks after the antibiotics were stopped. She remained reasonably well, with no signs of recurrence, but was
admitted to a local hospital in early April 2008 with hypernatraemia and an elevated international normalized ratio of 8.0. She unfortunately suffered a cardiac arrest and died. There was neither systemic nor microbiological evidence of recurrence of pseudomonal bacteraemia.

Discussion

This case has a number of notable features. Pseudomonads other than *Pseudomonas aeruginosa* are only occasionally isolated from blood cultures and are a rare cause of endocarditis. A large international observational cohort study of 2671 patients with endocarditis found that only 0.4% had endocarditis caused by *P. aeruginosa* (Morpeth et al., 2007). Although there have been case reports of endocarditis caused by *Pseudomonas luteola* (Casalta et al., 2005) and *Pseudomonas mendocina* (Aragone et al., 1992), we believe that this is the first case of endocarditis due to *P. mosselii* to be reported.

*P. mosselii* was, in fact, only formally described as a novel species in 2002 by Dabboussi et al. (2002), who examined strains of *P. fluorescens*, *Pseudomonas putida* and *Pseudomonas* species using a polyphasic taxonomic approach including 16S rDNA phylogeny, numerical analysis, DNA–DNA hybridization, thermal stability of DNA–DNA hybrids and siderotyping methodology. They reported *P. mosselii* as a novel species on the basis of a low level of DNA–DNA relatedness to other *Pseudomonas* strains and that *P. mosselii* strains are phenotypically and genotypically homogeneous with characteristic phenotypic features that allow speciation. Nonetheless, the API identification system routinely used in our laboratory to characterize isolates failed to identify this organism.

Prior to the classification of *P. mosselii* as a separate species, strains would have most likely been identified as *P. fluorescens*; however, we did not find any reports of *P. fluorescens* as a cause of endocarditis. *P. fluorescens* has previously been associated with outbreaks of pseudobacteraemia due to contaminated blood bottles (Smith et al., 2002); however, there were no other temporally related isolates of similar organisms at our laboratory.

Given the relative rarity of endocarditis caused by *Pseudomonas* species, strong treatment recommendations have not been published although it is accepted that the prognosis is generally poor, with the best outcomes following treatment with high-dose antibiotics and early surgery (Elliott et al., 2004). The British Society of Antimicrobial Chemotherapy Endocarditis Treatment Guidelines (Elliott et al., 2004) suggest that combination antibiotic therapy may offer synergy, and there are case reports of successful treatment of *P. aeruginosa* endocarditis with carbapenems and tobramycin (Fichtenbaum & Smith, 1992; Gavin et al., 2003) and ceftazidime and tobramycin (Cabinian & Kaatz, 1987). It is of note that the patient described in this case appeared to respond to conservative treatment with piperacillin–tazobactam followed by ceftazidime and died due to other causes, 6 months after completing treatment, in the absence of microbiological evidence of relapse.

In conclusion, *P. mosselii* is a relatively recently described species that is not easily identified by routine laboratory methods. In contrast with previous observations that endocarditis due to non-HACEK Gram-negative bacilli usually requires surgical intervention in addition to high-dose antibiotic therapy, our patient responded well to conservative management with antibiotics alone; a finding particularly relevant for patients unfit for surgery. Finally, in 2002, Dabboussi et al. (2002) concluded that the clinical significance of *P. mosselii* was not known; however, following our experience from this case, we would propose that it should be regarded as a potential pathogen.

References


