Intraoperative diagnosis of mitral valve endocarditis secondary to *Paenibacillus provencensis*

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ABSTRACT

We report the first case of infective endocarditis caused by *Paenibacillus provencensis*. A mitral valve vegetation was incidentally discovered by intraoperative transoesophageal echocardiography in a 70-year-old woman undergoing aortic valve replacement. The precise identification of the causative agent was by means of genotypic characterisation with 16S rDNA gene sequencing. The patient was successfully treated with a 6-week course of antibiotics postoperatively, following debridement of the valve vegetation.

KEYWORDS

Mitral valve – Bacterial endocarditis – Cardiac surgery – *Paenibacillus provencensis*

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Introduction

We report the first case of native valve endocarditis caused by *Paenibacillus provencensis*, a very rare Gram-negative spore-forming rod.¹ Despite recent advances in diagnostic and therapeutic strategies, infective endocarditis remains challenging to treat and is thus associated with poor prognosis in terms of morbidity and mortality, particularly when uncommon microorganisms are involved.²

Case history

A 70-year-old woman was admitted with non-ST segment elevation myocardial infarction on a background of long-standing aortic stenosis. Preoperative echocardiography confirmed severe aortic stenosis and mild mitral regurgitation with a severely calcified mitral annulus (Fig 1) and coronary angiography ruled out significant disease. She developed a post-angiogram right femoral pseudoaneurysm on the access site, which required ultrasound-guided compression prior to surgery. The operation was postponed because of raised inflammatory markers, which were attributed to cellulitis on the femoral access site, which did not, however, respond to antibiotics.

Intraoperative transoesophageal echocardiography showed a 1.5-cm mobile mass adherent to the mitral valve, suggestive of a vegetation. After debridement of the mitral valve, an uneventful aortic valve replacement was

performed and empirical treatment with teicoplanin and gentamicin was started. Real-time polymerase chain reaction (PCR) with 16S rDNA sequencing and culture of the tissue specimen identified the organism as *P. provencensis*, sensitive to vancomycin and daptomycin. The patient completed a 4-week course of intravenous vancomycin and meropenem, followed by a further 2 weeks of daptomycin because vancomycin-resistant enterococci were identified in a rectal swab. A postoperative transthoracic echocardiogram confirmed no sequelae from endocarditis (Fig 2). When last seen in clinic, the patient had completely recovered.

Discussion

To the best of our knowledge, this is the first case of infective endocarditis caused by *P. provencensis*. This organism was only discovered in 2008, following identification in the cerebrospinal fluid of a patient with meningitis caused by *Tropheryma whippelii*, when it was considered to be a contaminant.¹ To exclude contamination, we repeated 16S rDNA sequencing and obtained concordant results in both tests. The genotypic test was also corroborated by the positive cultures and by the fact that no other microorganisms could be identified in the mitral valve specimen.

Paenibacillus species are common saprophytic bacteria³ but they have rarely been considered to be pathogenic for man. However, different human infections have been

Figure 1 Preoperative echocardiogram illustrating severe aortic stenosis, heavily calcified mitral annulus and a 'vegetation' adherent to the atrial aspect of the mitral valve (arrow)

Figure 2 Postoperative echocardiogram demonstrating an intact mitral valve after debridement of the 'vegetation' seen on the preoperative scan

attributed to several *Paenibacillus* species, including a single case of cardiac device-related endocarditis caused by *P. glucanolyticus*.⁴ The latter case, together with the absence of other likely infectious sources, raised the suspicion that the infection in our patient might have been secondary to the formation of a femoral pseudoaneurysm as complication of angiography. In fact, that pseudoaneurysm developed infectious signs that motivated antibiotic treatment for what was assumed to be cellulitis. In hindsight, those infectious signs might have been a missed beacon to the endocarditis that was slowly developing in the mitral valve.

The precise identification of these bacteria is challenging, owing to the absence of clear discriminating phenotypical

features, as well as the fact that many proposals of new species have been made on the basis of the characterisation of single isolates.¹ In addition, these organisms are particularly difficult to grow in culture and no biochemical tests is yet available. Thus, their exact identification often relies on modern molecular biology methods, such as 16S rRNA gene sequencing.¹

Our case is also unique in that the patient had no cardiac device or other prosthetic implant prior to undergoing aortic valve replacement. However, retrospective analysis of the preoperative transthoracic echocardiogram revealed some echogenic material adherent to the atrial aspect of the mitral valve, which appeared to be a precursor of the fully fledged vegetation subsequently found in the intraoperative transoesophageal echocardiogram (Fig 1). Indeed, it is well established that transthoracic echocardiography is not adequate for diagnosing endocarditis, which ideally requires transoesophageal echocardiography.² This probably explains why the incipient endocarditis was missed on the preoperative scan and only discovered later in theatre. With hindsight, the persistently elevated inflammatory markers were probably secondary to endocarditis and not to vascular access site cellulitis, as attributed at that time, which justifies the absence of response to flucloxacillin.

In our case, the heavily calcified mitral annulus may have acted as a 'foreign body' which facilitated formation of a bacterial biofilm. However, even in this setting, *Paenibacillus* species would not be expected as the causative agent.²

Conclusion

This is, to the best of our knowledge, the first report showing that *P. provencensis* can be a pathogen in humans and that a member of the genus *Paenibacillus* may be involved in native valve endocarditis. This case highlights the importance of transoesophageal echocardiography for detecting mitral valve endocarditis, as subtle lesions can be easily missed by transthoracic echocardiography. Diagnosis of infective endocarditis is therefore difficult unless there is a high degree of suspicion and appropriate investigations are undertaken in a timely manner. It also emphasises the importance of 16S rDNA gene sequencing by real-time PCR as a rapid and accurate method of identifying microorganisms such as *Paenibacillus*, which can be missed by conventional microbiological investigations.

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