Review

Endocarditis caused by *Propionibacterium* species: a report of three cases and a review of clinical features and diagnostic difficulties

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Propionibacterium species are members of the normal flora of skin and the mouth but their pathogenic potential is often overlooked. Three fatal cases of endocarditis caused by *Propionibacterium* species over an 8-year period are reported, and a review is presented of a further 33 cases from the world literature. In most cases, infection was protracted, with minimal signs in the early stages. Fourteen cases (42.4%) involved native valves, 16 (48.5%) involved prosthetic valves and three (9.1%) were associated with other intracardiac prosthetic material. Intracardiac abscesses were commonly encountered, with *Propionibacterium* endocarditis occurring in 28.6% of native valve infections and 52.9% of prosthetic valve infections. A very high proportion of all of the cases (70.6%) required surgical intervention. Several factors appeared to delay institution of appropriate therapy and may have contributed to abscess formation, including an indolent clinical course, negative or delayed culture results, and the tendency to consider this organism as a blood-culture contaminant. The authors recommend careful clinical evaluation before disregarding a blood-culture isolate of *Propionibacterium* spp. as a skin contaminant, and consideration of this bacterium as a potential cause of apparently culture-negative endocarditis.

Overview

Propionibacterium species are members of the normal microbial flora of human skin and the mouth. A role for *Propionibacterium acnes* in the pathogenesis of acne has been debated for decades, but never adequately proven (Bojar & Holland, 2004). Serious infections due to *P. acnes* are rarely reported, but this bacterium is increasingly recognized as a cause of serious infections, such as endocarditis, prosthetic joint infection, endophthalmitis, osteomyelitis and central nervous system infections (Bruggemann, 2005). In such cases, an indwelling medical device such as a prosthetic heart valve, cerebrospinal fluid shunt or intraocular lens is often involved. Here, we review the clinical features of endocarditis caused by *Propionibacterium* species and the diagnostic difficulties frequently encountered with this pathogen. We illustrate some of these issues with our own experience of this condition and highlight an apparent predisposition of this low-grade pathogen to cause intracardiac abscesses and prosthetic valve dehiscence.

Review methodology

A prospectively collected database of all endocarditis cases seen at the General Infirmary at Leeds has been maintained since 1998, and this was used to identify patients with *Propionibacterium* endocarditis seen between January 1998

and December 2005. A literature search was carried out using the Pubmed and Medline databases with the search terms '*Propionibacterium*', '*propionibacteria*', 'endocarditis', 'infective endocarditis', '*P. acnes*' and '*P. granulosum*'. Further references were identified from the reference lists of papers identified. Any papers describing one or more cases of *Propionibacterium* endocarditis within the last 25 years with sufficient information to categorize the episode using modified 'Duke criteria' were included (Li *et al.*, 2000).

Review-derived data

A review of the world literature identified 28 papers describing 33 cases in sufficient detail. Clinical details are summarized in Table 1. The median age of affected patients was 48 years (range 15–78) and 27 (81.8%) of the patients were male. Twenty-six cases (78.8%) were definite by Duke criteria and seven (21.2%) were possible. Fever was the commonest presenting complaint, reported in 27 cases (81.8%); lethargy and malaise were reported in 14 (42.4%) and sweats or chills in 9 (27.2%). Examination findings were limited: a murmur was reported in 15 (45.4%), but in only four cases (12.1%) were 'classical' stigmata of infective endocarditis seen (e.g. Osler nodes, Janeway lesions, splinter haemorrhages, macular haemorrhage, etc.). Fourteen cases (42.4%) involved native valves, 16 (48.5%) involved prosthetic valves and three (9.1%) were associated with other

Table 1. Selected clinical details of cases of infective endocarditis caused by *Propionibacterium* species

Abbreviations. Valve: N, native; P, prosthetic; A, aortic; M, mitral; T, tricuspid; NK, not known. Duke criteria: D, definite; P, possible. Surgery, Abscess: Y, yes; N, no. Outcome: S, survived; D, died.

Reference	Age	Sex	Valve		Duke criteria	Days to positive culture	Blood culture	Valve culture	Surgery	Abscess	Outcome
			Type of valve	Location							
Lewis & Abramson (1980)	35	M	N	A	D		<i>P. acnes</i> (<i>S. epidermidis</i>)	<i>P. acnes</i>	Y	N	S
Fornaciari <i>et al.</i> (1985)	63	M	N	A	P		<i>P. acnes</i>	—	N	N	S
Branger <i>et al.</i> (1987)	38	F	N	NK	P	10	<i>P. granulosum</i>	—	N	N	S
Hernandez <i>et al.</i> (1988)	42	F	N	?A/M	D		<i>P. acnes</i>	—	N	N	S
O'Neill <i>et al.</i> (1988)	41	M	P	M	D	14	<i>P. acnes</i>	—	N	N	S
Lazar & Schulman (1992)	63	M	P	A	D		—	<i>P. acnes</i>	Y	N	S
Abramczuk <i>et al.</i> (1992)	40	F	N	M	D		—	<i>P. acnes/Neisseria</i> sp.	Y	N	S
Abramczuk <i>et al.</i> (1992)	63	F	N	M	P		<i>P. acnes</i>	—	N	N	S
Abramczuk <i>et al.</i> (1992)	50	M	P	NK	D		<i>P. acnes</i> (<i>S. epidermidis</i>)	—	N	N	D
Abramczuk <i>et al.</i> (1992)	48	M	P	?M/T	P		<i>P. acnes</i>	—	N	N	S
Abramczuk <i>et al.</i> (1992)	62	F	P	?A/M	P		<i>P. acnes</i>	—	N	N	S
Horner <i>et al.</i> (1992)	34	M	P	A	D	6	<i>P. acnes</i>	Not cultured	Y	Y	S
Scheel <i>et al.</i> (1992)	59	F	P	A	D		<i>P. acnes</i>	—	N	N	S
Lee <i>et al.</i> (1993)	57	F	P	M	D		<i>P. acnes</i>	Negative	Y	Y	S
Gunthard <i>et al.</i> (1994)	73	M	P	A	D	7	<i>P. acnes</i>	<i>P. acnes</i>	Y	N	S
Huynh <i>et al.</i> (1995)	46	M	P	A	D	7	<i>P. acnes</i>	<i>P. acnes</i>	Y	Y	D
Armstrong & Wuerflein (1996)	52	M	N	A	D	5	—	<i>P. granulosum</i>	Y	Y	S
Caballero Gueto <i>et al.</i> (1997)	23	M	N	A	D	10	<i>P. acnes</i>	Not cultured	Y	Y	S
Chua <i>et al.</i> (1998)	78	M	Pacemaker		D		<i>P. acnes</i>	<i>P. acnes</i>	*	N	S
Durupt <i>et al.</i> (1998)	47	M	P	A	D		<i>P. acnes</i>	Negative	Y	Y	D
Durupt <i>et al.</i> (1998)	61	M	P	A	D		<i>P. acnes</i>	<i>P. acnes</i>	Y	Y	S
Praderio <i>et al.</i> (1998)	35	M	N	NK	P	10	<i>P. acnes</i>	—	N	N	S
Jimenez-Navarro <i>et al.</i> (1999)	70	M	N	A	D		—	<i>P. acnes</i>	Y	Y	S
Chaudhry <i>et al.</i> (2000)	38	F	N	M	D	7	<i>P. granulosum</i>	<i>P. granulosum</i>	Y	N	S
Clarke & Banning (2000)	67	M	P	A	D		<i>Propionibacterium</i> sp.	Not cultured	Y	Y	S
Moreira <i>et al.</i> (2000)	64	M	N	A + M	D		—	<i>P. acnes</i>	Y	N	S
Mohsen <i>et al.</i> (2001)	36	M	N	A	D	6	<i>P. acnes</i>	Negative	Y	Y	S
Vandenbos <i>et al.</i> (2001)	61	M	Teflon patch		P		<i>P. acnes</i>	Not cultured	N	N	S
Chakour <i>et al.</i> (2002)	48	M	P	M	D		<i>P. acnes</i>	<i>P. acnes</i>	Y	Y	S
Zedtwitz-Liebenstein <i>et al.</i> (2003)	31	M	Pacemaker		D		<i>P. acnes</i>	—	N	N	S
Koya <i>et al.</i> (2004)	24	M	N	T	D		<i>P. acnes</i>	Not cultured	Y	N	S
Vanagt <i>et al.</i> (2004)	15	M	P	M	D		—	<i>P. acnes</i>	Y	N	S
Pan <i>et al.</i> (2005)	58	M	P	A	D		<i>P. acnes</i>	Negative	Y	Y	S
This study	43	M	P	A	D	8	<i>P. acnes</i>	Gram-positive bacilli	Y	Y	D
This study	56	F	N	M	D		—	<i>P. acnes</i>	Y	N	D
This study	67	M	N	M	D	5	<i>Propionibacterium</i> sp.	—	N	N	D

*Removal of pacing leads and generator.

intracardiac prosthetic material (pacing wires in two cases and a Teflon patch in the third). Ten of the 14 (71.4%) patients with native valve infection had an underlying cardiac factor predisposing to infection. In this group, the valves most commonly affected were the mitral and aortic valves, while those with prosthetic valves were more likely to have aortic valve involvement than mitral valve involvement, partly due to the pattern of the valves replaced. The infecting organism was *P. acnes* in 29 cases, *Propionibacterium granulosum* in three cases and an unspecified *Propionibacterium* species in the one remaining case. Intracardiac abscesses were commonly encountered, with *Propionibacterium* endocarditis occurring in 28.6% of native valve infections and 52.9% of prosthetic valve infections. Valve dehiscence was also a common finding in prosthetic valve infections, occurring in 52.9% of cases. Valve replacement surgery was undertaken in 70.6% of all prosthetic infections and 64.3% of native valve infections.

Cases from Leeds endocarditis service database

The Leeds endocarditis service database contained details of three further cases, which are described below. All of these cases were defined as definite by modified Duke criteria.

Case 1

A 43-year-old male underwent aortic valve replacement with a 25 mm mechanical prosthesis (St Jude Med) and mitral valve replacement with a 31 mm St Jude mechanical prosthesis following valve damage due to infective endocarditis. Two years later, further aortic valve surgery was required to repair an aortic paravalvular leak, but the aortic prosthesis was not replaced.

Four years after the original surgery he was admitted to hospital with a 10-day history of worsening malaise, lethargy and night sweats. His temperature was 37.1 °C. There were no clinical stigmata of infective endocarditis and a transthoracic echocardiogram (TTE) showed no evidence of prosthetic valve endocarditis. A total of seven samples were collected for blood culture. All were sterile after 7 days incubation. The patient made a gradual improvement without specific therapy and was discharged home after 10 days.

Three weeks later the patient was readmitted with similar symptoms. On admission, his temperature was 37.4 °C. He had macular lesions scattered over the forearms and lower legs and splinter haemorrhages under the fingernails of both hands. He had an early diastolic murmur suggestive of recurrent paravalvular leak but there was no clinical evidence of heart failure. TTE revealed an aortic paraprosthesis leak and normal function of the aortic prosthesis. The mitral prosthesis was functioning normally. Transesophageal echocardiogram (TEE) confirmed these findings. Laboratory investigations showed a haemoglobin of 10.0 g dl⁻¹, white cell count of 8.1×10^9 l⁻¹, platelet count of 249×10^9 l⁻¹ and C-reactive protein (CRP) was 42 mg l⁻¹. A provisional diagnosis of prosthetic valve endocarditis

(PVE) was made and a total of eight samples were collected for blood culture. He was commenced on intravenous vancomycin 1 g 12-hourly and oral rifampicin 600 mg 12-hourly.

Eight days after admission, one blood culture collected on admission revealed a Gram-positive bacillus. This was identified as *P. acnes* and was susceptible to vancomycin. Six out of eight blood cultures subsequently grew the same organism. Treatment with vancomycin was continued. The patient remained clinically stable but serial CRP measurements remained essentially unchanged. Three weeks following admission, follow-up TEE revealed a large anterior aortic root abscess with significant paraprosthesis aortic regurgitation but no visible vegetations. Due to sudden haemodynamic deterioration, urgent surgery was performed. Destruction of the aortic root was found, involving both coronary ostia and the inter-ventricular septum, and the destructive process extended into the right ventricular outflow tract. Aortic root replacement with a 25 mm cryopreserved homograft was performed. Unfortunately the patient could not be weaned off cardiopulmonary bypass. Gram-positive bacilli with an appearance consistent with *P. acnes* were seen on microscopy of pus removed from the aortic root abscess but failed to grow on culture.

Case 2

A 56-year-old female with rheumatic mitral valve disease presented with worsening breathlessness, oedema and orthopnoea over a 6-month period. She had been working full time until 9 months previously. She had suffered 'flu-like' symptoms and was tired and lethargic before becoming increasingly short of breath. She was dyspnoeic on minimal exertion and slept upright at night. On examination, she was obese and markedly oedematous. She was consistently afebrile. There was an audible murmur consistent with mitral regurgitation and atrial fibrillation with a rate of 110 beats per minute (b.p.m.). Her normal medications included warfarin, digoxin, bendrofluazide and furosemide. Angiography showed moderate left ventricular (LV) impairment, mitral regurgitation and pulmonary hypertension. Coronary vasculature was normal. She was transferred to our facility for an elective mitral valve replacement. Infection was not suspected so neither microbiological investigations nor markers of inflammation were performed pre-operatively.

At operation, both leaflets of the mitral valve were fibrotic with fusion of the subvalvular apparatus. A 25 mm mechanical valve (Sorin Biomedica) was inserted. Postoperative transthoracic echocardiography showed good LV and mitral valve function. Culture of the resected valve showed *P. acnes*. She was commenced on intravenous benzylpenicillin 1.8 g every 4 hours for 5 weeks and concurrently received gentamicin 80 mg b.d. for 3 weeks followed by rifampicin 600 mg o.d. for 2 weeks.

The early postoperative course was complicated by wound breakdown and sternal osteomyelitis caused by

Staphylococcus aureus, treated with intravenous flucloxacillin 1 g q.d.s. and rifampicin 600 mg b.d. for 6 weeks. The sternum was rewired and the wound was debrided 9 days after the initial surgery. Eleven weeks after admission, her condition deteriorated with increased pulmonary oedema and worsening renal function. She was transferred to the Intensive Care Unit, but subsequently died.

Case 3

A 67-year-old man was admitted with a 1-week history of increasing breathlessness, a sore throat and weight loss. He was a lifelong smoker with chronic obstructive airways disease, ischaemic heart disease and peripheral vascular disease. He had previously had two myocardial infarctions and had undergone a right-sided femoral-popliteal bypass 9 years previously. An allergy to penicillin was noted.

On admission, he had a temperature of 38.7 °C, although was otherwise stable. A grade 3 apical pansystolic murmur was audible, radiating to the axilla, although this had been noted for some years. Auscultation of the chest revealed right-sided basal crepitations. There were no stigmata of endocarditis, but peripheral oedema was present.

Blood tests revealed a normocytic anaemia with a haemoglobin of 10.7 g dl⁻¹ (12.5 g dl⁻¹ 4 months previously) and a white cell count of 12.9 × 10⁹ l⁻¹ with a neutrophilia of 10.9 × 10⁹ l⁻¹. CRP was 67 mg l⁻¹. Urea was 10.2 mmol l⁻¹ and creatinine 147 µmol l⁻¹. He was hypoxic, with a PaO₂ of 7.9 kPa. A diagnosis of right basal pneumonia was initially made, and he received erythromycin 500 mg q.d.s. for 7 days. Two sets of blood cultures, both taken after commencing antibiotics, were negative.

A subsequent plain chest radiograph showed enlargement of the heart compared to 4 months previously, plethoric lungs but no focal consolidation. The diagnosis was revised to severe refractory cardiac failure. Transthoracic echocardiography showed a dilated left ventricle and moderate-severe mitral regurgitation with thickened calcified mitral leaflets, one of which had an unusual appearance, thought to be a vegetation. An atrial septal defect was also seen. A TEE was planned, but delayed.

Some improvement was initially noted with diuretic treatment, fluid restriction and angiotensin-converting enzyme (ACE) inhibitors, but his oedema worsened despite medical management. Renal function also deteriorated. The possibility of endocarditis was reconsidered and a further four sets of blood cultures were sent 10 days after admission. Empirical therapy was also started: intravenous vancomycin 1 g daily, gentamicin 60 mg daily and oral rifampicin 300 mg b.d. Hepatic function deteriorated the next day, so the rifampicin was stopped. Two weeks after admission, he was transferred to the Coronary Care Unit in view of worsening renal failure, hyponatraemia and hypotension. Inotropic support was given; however, the patient's haemodynamic condition rapidly deteriorated, he developed ventricular tachycardia, and could not be resuscitated.

The day after his death, the anaerobic bottle of one set of blood cultures became positive after 5 days and grew Gram-positive bacilli, identified as *Propionibacterium* spp.

Propionibacterium species and endocarditis

Propionibacterium species are an underrecognized cause of endocarditis, but they have been associated with infection of both native and prosthetic valves. The incidence of *Propionibacterium* species endocarditis is not known, but was approximately 0.3 cases per year in a recent report from another UK tertiary referral centre (Wallace *et al.*, 2002), and 1.4 cases per year from a French cardiothoracic referral centre (Fol *et al.*, 2003). In Leeds, we have seen 0.4 cases per year over the past 8 years, in a hospital serving a population of approximately 700 000. The true incidence of *Propionibacterium* endocarditis may be underestimated because of insufficient culture time and a lack of appreciation of the true pathogenicity of this organism, as outlined below.

Diagnosis

Propionibacterium endocarditis is usually associated with a relatively long history with minimal clinical signs of infection at initial presentation. This is typified in the cases presented, especially case 2, in which infection was not suspected prior to surgery. CRP, commonly used to monitor inflammatory response, may not show the significantly raised levels seen in other infections. In published cases, the median CRP at presentation was 51 mg l⁻¹, (range 3–262 mg l⁻¹).

These cases highlight the potential severity of the condition, but also the difficulty in isolating *Propionibacterium* spp. It is a slow-growing bacterium that typically requires between 7 and 14 days for isolation from clinical samples. In this review, where time to positivity was reported, the median incubation period was 7 days (range 5–14 days). Many laboratories routinely incubate blood cultures for 5 days; this may be insufficient for the growth of this organism in many circumstances. Prolonged aerobic and anaerobic culture for up to 2 weeks may be required in order to isolate the organism from blood. In case 1, 8 days were required before growth of *Propionibacterium* from blood cultures, in this case, 4 weeks after the patient's initial presentation. Five days were required for growth of the organism in case 3, 3 weeks after presentation. In both cases, previous blood-culture sets were returned negative. Twenty-seven of the 33 cases (81.8%) reviewed in Table 1 had growth of *Propionibacterium* spp. in the blood cultures, 24 with *P. acnes*, two with *P. granulosum*, and one with an unspecified species. In two patients, *Staphylococcus epidermidis* was also cultured; this was felt most likely to represent contamination, given that there were multiple sets of cultures positive with *P. acnes* in each patient and just one set growing *S. epidermidis*.

Although generally considered to be a bacterium of low virulence, the pathogenic potential of *Propionibacterium*

species is becoming increasingly apparent. In a survey of anaerobic bacteraemia in a Finnish hospital, 25 % of blood cultures positive for propionibacteria were found to be clinically significant (Salonen *et al.*, 1998). The converse, that 75 % of blood cultures positive for propionibacteria were considered to be skin contaminants, highlights the problem of interpreting positive blood cultures. To this end, it is common practice to send multiple serial sets of blood cultures when investigating for infective endocarditis. Indeed, to fulfil a major criterion in the Duke classification, there is a requirement to have multiple positive blood-culture sets. However, there appears to be marked heterogeneity in investigations for bacteraemia in this review. In 13 cases it is clear how many blood-culture sets were investigated; for each of these patients a median of seven sets were sent for culture (range 2–42 sets per patient). Of the 25 patients with positive blood cultures, there was a median of three sets of positive cultures (range 2–13). Where both of the above are reported together (in 12 cases), the positivity rate for blood cultures averages 50·1 %. This reinforces the importance of sending multiple sets of cultures. When there is a paucity of positive microbiological results, as in case 3 presented here, doubt can be cast upon the validity of the diagnosis. In this case, there were sufficient criteria to rank this as a definite diagnosis by Duke criteria (valvular vegetation, an at-risk cardiac lesion, a temperature $>38^{\circ}\text{C}$ and a single positive blood culture), but one cannot be certain that in an instance where many sets were taken, that contamination of one set of the cultures did not occur.

The cases reviewed here emphasize the need to carefully evaluate risk factors and clinical evidence of endocarditis in any patient in whom *Propionibacterium* species is isolated from blood cultures. Repeated isolation of *Propionibacterium* species should not be considered as contamination in a systemic infection associated with implants such as artificial heart valves or indwelling prostheses. Until molecular diagnostic techniques are sufficiently refined, diagnosis of endocarditis caused by slow-growing or fastidious bacteria remains constrained by the growth characteristics of the organism.

Medical management

The majority of propionibacteria are susceptible to a number of antibiotics. Oprica & Nord (2005) investigated 304 isolates of *P. acnes* from invasive infections in Europe and found 100 % susceptibility to penicillin and vancomycin. Of these isolates, 15·1 % were resistant to clindamycin. While treatment guidelines exist for endocarditis caused by more commonly isolated organisms, there is no current consensus on how to manage endocarditis caused by *Propionibacterium* species. Review of the literature shows much diversity in management of this phenomenon (Table 2).

Of the 33 cases reported, 12 were predominantly managed with penicillins alone (including penicillin, amoxycillin, ampicillin, imipenem, flucloxacillin, nafcillin and cloxacillin, as well as various beta-lactam/beta-lactamase inhibitor combinations) while a further 11 were treated with combinations of a penicillin and an aminoglycoside (including gentamicin, netilmicin and streptomycin). Three received vancomycin or teicoplanin with an aminoglycoside or rifampicin. Other options included cephalosporins, lincomycin or clindamycin. Duration of treatment was reported in 23 cases and was found to be variable. Every case received a minimum of 4 weeks therapy; median duration of therapy was 6 weeks. Eight of these cases received more than 6 weeks therapy. Nine cases were discharged after conversion to oral therapy, most commonly a penicillin, tetracycline or clindamycin. Of the three cases with definite infective endocarditis who improved without the requirement for surgery, all three received a penicillin/aminoglycoside combination; one of these patients relapsed however, but responded when retreated. Of the patients, 72·7 % required surgical therapy in addition to antibiotics, indicating that in this infection, consideration should always be given to the requirement for surgery. However, given the small number of known cases and heterogeneity of management, it is difficult to draw clear conclusions as to what represents best practice.

Table 2. Mainstay of antimicrobial therapy

Antibiotic regimen	Number of cases	Cured with antibiotics alone	Mortality
Penicillins	12	3 (25·0 %)	1 (8·3 %)
Penicillins + aminoglycoside	11	4 (36·4 %)	0
Cephalosporins alone	3	1 (33·3 %)	1 (33·3 %)
Cephalosporins + aminoglycoside	1	0	0
Glycopeptides + aminoglycoside	2	0	0
Glycopeptides + rifampicin	1	0	0
Lincomycin	1	0	1 (100 %)
Clindamycin	1	1 (100 %)	0
Unknown	1	0	0

Surgical management

Case 1 illustrates how *P. acnes* infection can cause irreversible and severe damage to a prosthetic valve and surrounding structures resulting in death. In previous cases from the literature, 64.7 % of those with prosthetic valve infections had either dehiscence of the valve or an associated abscess, all of whom required surgery. A very high proportion of all of the cases (70.6 %) required surgical intervention. This compares unfavourably with published figures of a requirement for surgery in 30 % of cases with acute endocarditis caused by any organism (Horstkotte *et al.*, 2004). It appears that abscess formation is common in endocarditis caused by *Propionibacterium* species. The incidence of abscess formation seen in endocarditis caused by all organisms is variable, but has been reported as 6.2 % (Ako *et al.*, 2003) and 11.4 % (Jaffe *et al.*, 1990). The rates seen in this review are much greater, perhaps due to the insidious nature of the infection. It must be noted that as there are reasonably few cases reported in the literature, it is possible that reporting bias may distort the true incidence of abscess formation and the requirement for surgery in this condition. It is also of note that in native valve infections, abscesses were only seen in association with aortic valve infection.

Where surgery was performed, endocardial tissue was cultured in 79.2 % of cases. *P. acnes* was grown in the majority of cases, but *P. granulosum* was seen in two native valves. One patient was found to have a mixed growth of *P. acnes* and a *Neisseria* sp., and failed therapy with a cephalosporin alone, yet responded to vancomycin monotherapy, suggesting the role of *P. acnes* in this infection. In this review, there was a 22.2 % negative rate for valve culture where performed. Despite this, valve culture is useful for making a clear diagnosis in cases where blood cultures remain negative, as was the case in six patients in this review. In the first case presented here, microscopy of the valve was of benefit as Gram-positive bacilli were observed. Newer technologies such as PCR may be of use to increase the diagnostic yield in cases where culture fails.

Infections with pacing leads *in situ*

Two cases from the literature refer to infection of intracardiac pacing leads. In one case, these leads were removed and the patient was treated with a penicillin/aminoglycoside combination (Chua *et al.*, 1998). In the other case, the patient responded to a penicillin without removal of the leads (Zedtwitz-Liebenstein *et al.*, 2003).

Outcome of infective endocarditis due to propionibacteria

Endocarditis caused by this bacterium has a mortality previously estimated at 21–46 % (Mohsen *et al.*, 2001); this review revealed an overall mortality of 9.1 % (0 % in native valve infections and 18.8 % in prosthetic valve infections), although it is possible that these figures are affected by reporting bias. Fol *et al.* (2003) report a mortality of 27.3 % in their series of 11 patients. Three known cases in our

institution, two native valve infections and one prosthetic valve infection, all died. This compares unfavourably to other cases of endocarditis in our hospital, where the mortality is approximately 20 %, in line with other recent reports (Wallace *et al.*, 2002).

Propionibacterium species endocarditis is associated with intracardiac abscess formation in a high proportion of reported cases. Several factors evident from the current literature may contribute to delayed institution of appropriate therapy and to abscess formation: an indolent clinical course without 'classical' stigmata of endocarditis; the prolonged incubation frequently required to culture this bacterium; and the tendency to consider this organism as a blood-culture contaminant. It is of vital importance that the microbiology laboratory is aware of a clinical suspicion of endocarditis, the presence of a prosthetic heart valve and the need for prolonged culture to detect *P. acnes*. Furthermore, a delayed response to appropriate antimicrobial therapy should raise the possibility of an intracardiac abscess.

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