Infective Endocarditis in Solid Organ Transplant Recipients

David L. Paterson, Edward A. Dominguez, Feng-Yee Chang,* David R. Snydman, and Nina Singh

From the Infectious Disease Section, Veterans Affairs Medical Center, and the University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania; the Section of Infectious Diseases, University of Nebraska Medical Center, Omaha, Nebraska; and the Departments of Clinical Microbiology, Pathology, and Medicine, New England Medical Center, and Tufts University School of Medicine, Boston, Massachusetts

Infective endocarditis, defined as pathologically or clinically definite by the Duke criteria, was observed in 14 transplant recipients at our institutions. In addition, we reviewed 32 previously reported cases in solid organ transplant recipients. The spectrum of organisms causing infective endocarditis was clearly different in transplant recipients than in the general population; 50% of the infections were due to *Aspergillus fumigatus* or *Staphylococcus aureus,* but only 4% were due to viridans streptococci. Fungal infections predominated early (accounting for six of 10 cases of endocarditis within 30 days of transplantation), while bacterial infections caused most cases (80%) after this time. In 80% (37) of the 46 cases in transplant recipients, there was no underlying valvular disease. Seventy-four percent (34) of the 46 cases were associated with previous hospital-acquired infection, notably venous access device and wound infections. Three patients with *S. aureus* endocarditis had had an episode of *S. aureus* bacteremia >3 weeks prior to the diagnosis of endocarditis and had received treatment for the initial bacteremia of <14 days' duration. The overall mortality rate was 57% (26 of 46 patients died), with 58% (15) of the 26 fatal cases not being suspected during life. Endocarditis is an underappreciated sequela of hospital-acquired infection in transplant recipients.

Infection remains a common cause of morbidity and mortality in recipients of solid organ transplants. Infective endocarditis is rarely described as a complication of transplantation, with few cases having been previously reported. Infective endocarditis is increasingly recognized as a nosocomial disease in many patients [1]. Transplantation surgery entails multiple invasive procedures and sometimes prolonged hospitalization and is well recognized to be associated with bacteremia [2]. A proportion of such patients with bacteremia go on to develop infective endocarditis. In addition, the immunosuppression necessary for prevention of rejection of transplanted organs predisposes patients to invasive fungal infection, particularly with *Aspergillus.* Although bacteria are widely regarded as the primary cause of infective endocarditis in most settings, fungi are a significant cause of this infection in certain populations.

In this study, we review the etiologic agents, clinical findings, treatment, and outcome of infective endocarditis in solid organ transplant recipients.

Methods

Cases of endocarditis occurring at the Veterans Affairs Medical Center in Pittsburgh, the University of Nebraska Medical Center in Omaha, and the New England Medical Center in Boston were reviewed.

A literature review of endocarditis in solid organ transplant recipients was performed by utilizing MEDLINE with the key words *endocarditis* and *transplantation.* Both English- and non-English-language articles were retrieved. The review period included 1966–1997. In addition, the references cited in the retrieved articles were reviewed to discover additional cases. Only cases of endocarditis that met the definition of definite infective endocarditis by the Duke criteria of Durack et al. [3] were included in the review. Findings or descriptions that were consistent with infective endocarditis but that fell short of the definition of definite infective endocarditis (''possible infective endocarditis'') were rejected, as were infections of intracardiac anastomoses or prosthetic material after heart transplantation and cases of viral or toxoplasma endomyocarditis.

Fifty-five cases of infective endocarditis in transplant recipients have been reported in the medical literature. Of these cases, 23 were excluded by our definitions (cytomegalovirus endomyocarditis [4], 1 case; insufficient detail to be defined by the Duke criteria as definite infective endocarditis [5–12], 12 cases; and infections of intracardiac anastomoses or prosthetic material after heart or heart-lung transplantation [13–22], 10 cases). Thus, 32 cases of definite infective endocarditis (23 that met the pathological definition and nine that met the clinical criteria for a diagnosis of definite infective endocarditis) were included in this review [23–47].

Results

Current Cases

We identified 14 cases of definite infective endocarditis at our institutions (table 1). During the time of observation, 10

Received 21 July 1997; revised 19 November 1997.

*Current affiliation: Division of Infectious Diseases, Tri-Service General Hospital, Taipei, Taiwan.

Reprints or correspondence: Dr. Nina Singh, Infectious Disease Section, Veterans Affairs Medical Center, Pittsburgh, Pennsylvania 15240.

Clinical Infectious Diseases 1998; 26:689–94

1058–4838/98/2603–0022$03.00

Table 1. Clinical features of 14 solid organ transplant recipients with infective endocarditis who were seen at our institutions.

Patient no., age (y)/sex	Transplant type	Time since transplantation	Organism	Affected area(s)	Underlying condition	Antibiotic therapy	Surgery	Outcome	Comment(s)
1, 39/M	Liver	14 d	*Aspergillus fumigatus*	Mural endocardium	None	AmB	None	Died	Not found until autopsy
2, 46/M	Liver	19 d	*A. fumigatus*	Mural endocardium	None	AmB	None	Died	Not found until autopsy
3, 40/M	Liver	30 d	*A. fumigatus*	Mural endocardium	None	AmB	None	Died	Not found until autopsy
4, 49/M	Liver	35 d	MRSA	Aortic valve	None	Vm, Gm, Rif	AVR	Alive	Splenic infarct
5, 55/M	Liver	50 d	*A. fumigatus*	Pulmonary valve	None	None	None	Died	Not found until autopsy
6, 60/F	Kidney	60 d	VR *Enterococcus faecalis*	Mitral valve	Rheumatic valve disease	Amp/Gm	None	Alive	Needed Amp desensitization prior to treatment
7, 59/M	Liver	90 d	VR *Enterococcus faecium*	Aortic valve	None	Vm, Cpfx, Rif	AVR	Alive	Early valve replacement surgery performed
8, 56/M	Liver	5 mo	MRSA	Tricuspid and aortic valves	None	Vm, Gm	None	Died	Septic pulmonary emboli; septic bursitis
9, 59/M	Liver	7 mo	*E. faecalis*	Aortic and mitral valves	None	Vm	None	Alive	Organism displayed HLR to Gm
10, 61/F	Liver	8 mo	MRSA	Mitral valve	None	Vm	MVR	Died	Cryptococcal meningitis prior to endocarditis
11, 58/M	Kidney	18 mo	*Staphylococcus aureus*	Mitral valve	Rheumatic valve disease	Naf	None	Died	TTE did not show vegetations; autopsy revealed endocarditis
12, 65/F	Kidney	3 y	*S. aureus*	Mitral valve	Prosthetic valve	Vm	None	Died	Bacteremia originated from infected iv access device
13, 55/M	Liver	6 y	*S. aureus*	Aortic valve	None	Naf/Rif/Gm	None	Alive	Presented with acute renal impairment
14, 67/M	Kidney	15 y	*S. aureus*	Aortic and mitral valves	Aortic stenosis	Naf/Rif/Gm	None	Died	Cerebral embolism

NOTE. AmB = amphotericin B; Amp = ampicillin; AVR = aortic valve replacement; Cpfx = ciprofloxacin; Gm = gentamicin; HLR = high-level resistance; MRSA = methicillin-resistant *S. aureus*; MVR = mitral valve replacement; Naf = nafcillin; Rif = rifampin; TTE = transthoracic echocardiography; Vm = vancomycin; VR = vancomycin-resistant.

cases of infective endocarditis occurred in 591 patients who received liver transplants at our institutions. Thus, the prevalence of infective endocarditis in this population was 1.7%.

Of the 14 cases, 7 were due to *Staphylococcus aureus,* 4 were due to *Aspergillus fumigatus,* and 3 were due to enterococci. Of the enterococcal isolates, two (*Enterococcus faecium* and *Enterococcus faecalis*) were vancomycin-resistant and one (*E. faecalis*), which exhibited high-level resistance to gentamicin, was vancomycin-susceptible. Two of the three enterococcal isolates were ampicillin-susceptible. All three patients with enterococcal endocarditis survived despite the antibiotic resistance profiles for their isolates. One patient infected with van-

comycin-resistant *E. faecium* was treated successfully with early valve replacement and then therapy with vancomycin, ciprofloxacin, and rifampin. Another patient with endocarditis due to *E. faecalis* resistant to vancomycin but susceptible to ampicillin and gentamicin needed desensitization to ampicillin because of a history of penicillin allergy; successful treatment of this patient's endocarditis was achieved with a 6-week course of ampicillin and gentamicin. Finally, one patient with a history of penicillin allergy who had endocarditis due to *E. faecalis* displaying high-level resistance to gentamicin was successfully treated with a prolonged course of vancomycin alone.

Among these patients, the mortality rate was high (64%; nine of 14 patients died). All four cases due to *Aspergillus* were discovered only at autopsy. Five of seven patients with *S. aureus* endocarditis died.

Combined Analysis of Previously Reported Cases and Current Cases

The clinical features and outcomes of the 32 previously reported cases and the 14 cases reported herein are detailed in table 2.

Infective endocarditis occurred in recipients of the following types of solid organ transplants: liver (16 patients), kidney (13), heart (15), heart-lung (1), and kidney-pancreas (1). No cases have been described in lung or small bowel transplant recipients. The mean age of the patients with infective endocarditis was 46 years (range, 12–67 years). Most patients were male (35 [76%] of 46).

Three patients underwent transplantation in the pre-cyclosporine era (2 developed aspergillus endocarditis, and 1 developed *S. aureus* endocarditis), 34 received cyclosporine therapy (3 developed aspergillus endocarditis, and 31 had infections due to other organisms), and 9 received tacrolimus therapy (4 developed aspergillus endocarditis, and 5 had infections due to other organisms).

Fungal infections tended to predominate as causes of infective endocarditis in the early posttransplantation period, whereas bacteria (especially *S. aureus*) tended to predominate later (figure 1). Only one of 14 kidney transplant recipients developed infective endocarditis within the first 8 weeks after transplantation, compared with nine of 16 liver transplant recipients and nine of 16 heart transplant recipients. Seventy-four percent (34) of the 46 cases of infective endocarditis were associated with a presumed hospital-acquired source, and 26% (12) of the 46 cases were community-acquired (table 3).

Endocarditis was due to bacterial infections in 72% (33) of the 46 cases and fungal infections in 28% (13) of the 46 cases. Ninety-four percent (31) of the 33 bacterial infections were left-sided (both the tricuspid and aortic valves were infected in one other case). In contrast, only six of 13 fungal infections were left-sided. Right-sided fungal infections affected the tricuspid valve (three cases) and the pulmonary valve (two cases). Except for one case in which *Aspergillus* infected the aortic valve, the remainder of fungal infections affected the mural endocardium rather than the valvular endocardium.

Eighty percent (37) of the 46 patients had no preexisting valvular disease. Prior to the development of endocarditis, 2 patients had a prosthetic valve, 1 had a bicuspid aortic valve,

Table 2. Etiology and clinical features of infective endocarditis in 46 solid organ transplant recipients whose cases were previously reported or were reported herein.

Organism	No. of patients	Transplant type (no. of patients)	Median time after surgery (range)	Outcome (no. who died/ no. infected with indicated organism)	[Reference(s)]
Staphylococcus aureus	14	Liver (6), heart (4), kidney (3), heart-lung (1)	4 mo (10 d to 15 y)	9/14	[27–30, 33, 34, 47, PR]
Aspergillus fumigatus	8	Liver (5), kidney (2), heart (1)	30 d (14 d to 11 mo)	7/8	[31, 32, 44, 46, PR]
Enterococcus species	5	Kidney (1), liver (2), heart (2)	3 mo (2 mo to 5 y)	1/5	[47, PR]
Candida species*	3	Kidney (1), kidney-pancreas (1), heart (1)	6 mo (24 d to 19 mo)	1/3	[23, 39, 47]
Gram-negative bacilli*	3	Heart (2), kidney (1)	1 mo (7 d to 6 y)	2/3	[24, 40, 47]
Coagulase-negative staphylococci	3	Heart (2), liver (1)	5 mo (23 d to 8 mo)	1/3	[25, 27, 42]
Corynebacteria	2	Kidney (1), liver (1)	(14 w to 3 y)	1/2	[26, 35]
Viridans streptococci	2	Kidney (1), heart (1)	(7 w to 9.5 y)	0/2	[23, 41]
Clostridium ramosum	1	Kidney (1)	4.5 y	1/1	[36]
Pseudallescheria boydii	1	Liver (1)	1 mo	1/1	[45]
Nocardia asteroides	1	Kidney (1)	6 mo	1/1	[37]
Polymicrobial†	3	Heart (2), kidney (1)	2 mo (34 d to 11 mo)	1/3	[38, 43, 47]

NOTE. PR = present report.

* The gram-negative bacilli included *Escherichia coli* (1 case), *Pseudomonas vesicularis* (1), and *Acinetobacter* species (1). The *Candida* species included *Candida albicans* (2 cases) and *Candida tropicalis* (1).

† Three patients had polymicrobial endocarditis (two organisms were isolated from their blood cultures or vegetation). The two organisms included *Corynebacterium* species and *Enterococcus faecalis* (1 patient), *Enterobacter cloacae* and *Lactobacillus* species (1), and *Aspergillus* species and *C. tropicalis* (1).

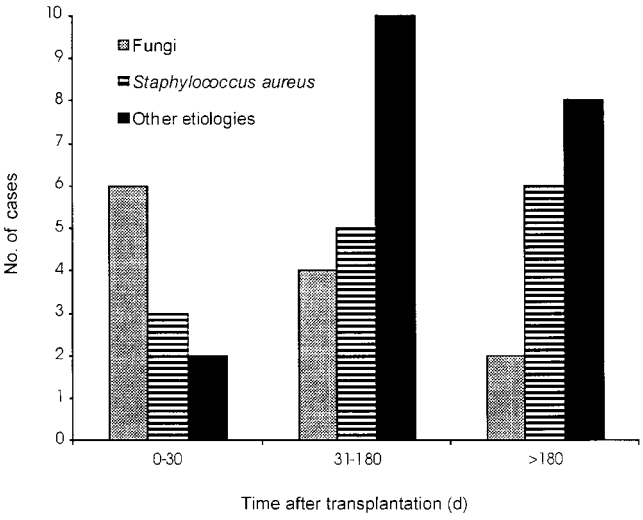

Figure 1. Timing of fungal and bacterial causes of infective endocarditis in solid organ transplant recipients in comparison with time after transplantation.

3 had aortic stenosis, and 3 had a history of rheumatic valvular disease. Nine of 33 cases of bacterial endocarditis compared with none of 13 cases of fungal endocarditis affected patients with preexisting valvular disease. Five of 14 cases of *S. aureus* endocarditis, 1 of 2 cases of viridans streptococcus endocarditis, 1 of 3 cases of coagulase-negative staphylococcus endocarditis, and 1 of 6 cases of enterococcal endocarditis affected patients with preexisting valvular disease.

Eleven patients (24%) had major embolic phenomena (embolization of cerebral arteries or large arteries of the lower limbs). The overall mortality rate was 57% (26 of 46 patients died). Fifteen cases were not suspected during life and were diagnosed at autopsy examination. Fifteen patients underwent valvular surgery; five of these patients died, compared with 20 of 31 patients who received only medical therapy. The mortality rate associated with bacterial endocarditis was 48% (16 of 33 patients died), and that associated with fungal endocarditis was 77% (10 of 13 patients died).

Discussion

Infective endocarditis is a relatively rare complication affecting solid organ transplant recipients. We found a prevalence of 1.7% among a cohort of liver transplant recipients who underwent transplantation from 1989 to 1997. In a cohort of 100 heart transplant recipients followed up for a median time of ~2 years [47], six (6%) had endocarditis. No data exist from which a prevalence of infective endocarditis can be calculated among kidney, lung, small bowel, or pancreas transplant recipients. Although previously reported cases in kidney transplant recipients are relatively numerous, no cases of infective endocarditis have been previously described in recipients of only lungs, small bowel, or pancreas.

In the early posttransplantation period, infective endocarditis in renal transplant recipients was relatively rare when compared with infective endocarditis in heart and liver transplant recipients. Patients with renal failure treated by dialysis may be less likely to have received intensive interventions that predispose to bacteremia and subsequent endocarditis, such as bronchoscopy, gastrointestinal endoscopy, or surgery and percutaneous liver biopsy [48]. Furthermore, renal transplant recipients can receive therapy to cope with graft rejection (dialysis) and may therefore receive less cumulative immunosuppressive therapy than recipients of other organs.

Table 3. Presumed source of organism in cases of infective endocarditis in 46 solid organ transplant recipients.

Source, organism	No. of patients
Presumed hospital-acquired (n = 34)	
Total no. with venous access device	11
Coagulase-negative staphylococci	2
Staphylococcus aureus	3
Corynebacterium	2
Enterococcus faecalis	2
Candida albicans	1
Acinetobacter species	1
Disseminated fungal infection	9
Aspergillus fumigatus	8
Pseudallescheria boydii	1
Wound	4
S. aureus	4
Intraabdominal infection	2
C. albicans	1
Vancomycin-resistant *Enterococcus faecium*	1
Organ donor	2
Escherichia coli	1
A. fumigatus	1
No source identified	4
S. aureus	3
Candida tropicalis	1
Urine	1
E. faecalis	1
Pericardial effusion	1
*Enterobacter**	1
*Lactobacillus**	1
Community-acquired (n = 12)†	
S. aureus	4
Viridans streptococci	2
Nocardia asteroides	1
Corynebacterium	1
Clostridium ramosum	1
Pseudomonas vesicularis	1
E. faecalis	2
Coagulase-negative staphylococci	1

* Both *Enterobacter* and *Lactobacillus* were isolated from the infected pericardial effusion in one patient.

† Both *Enterococcus* and *Corynebacterium* were isolated from one patient with community-acquired infection.

Table 4. Data on cases of previous bacteremia in solid organ transplant recipients who subsequently developed *Staphylococcus aureus* endocarditis.

Prior known source(s) of *S. aureus*	Treatment of prior *S. aureus* infection (duration in d)	Duration (d) between prior isolation and documentation of endocarditis	[Reference]
Blood, vascular catheter tip	Vm (14)	61	[PR]
Blood, vascular catheter tip	Vm (7)	60	[PR]
Blood, wound	Vm (12)	27	[27]
Blood, wound	Flucloxacillin (8)	33	[33]
Blood	NS	10	[28]

NOTE. NS = not stated; PR = present report; Vm = vancomycin.

Solid organ transplant recipients with infective endocarditis differ from other patients with infective endocarditis in many respects. Although there are limitations in extrapolating data from previously reported cases (rarer entities are more likely to be reported than those that are seemingly common), fungal endocarditis appears to be overrepresented in the transplant population. Fungi were the cause of 28% of cases of infective endocarditis in transplant recipients as compared with ~2%–4% of cases in most series outside the transplant setting [1]. Even in cases of so-called ''nosocomial'' endocarditis, aerobic bacteria predominate, and only 6% of cases of endocarditis are due to fungi [49]. In contrast, no cases of endocarditis due to viridans streptococci occurred in our institutions, and only two cases have been described in transplant recipients; it appears that transplant recipients are not at a significantly higher risk of endocarditis due to viridans streptococci than is the general population. Viridans streptococci are also comparatively rare causes of nosocomial endocarditis outside of the transplant setting [49].

Most (80%) of the solid organ transplant recipients who developed infective endocarditis had no history of valvular disease or abnormality. There may be a self-selection effect of organ transplantation for this phenomenon: patients with significant heart disease may not be considered candidates for transplantation. However, a more likely explanation is that the organism types (e.g., *S. aureus* and enterococci) causing infective endocarditis in transplant recipients are less likely to need an abnormal valve on which to lodge to produce endocarditis. In seven of the nine cases of aspergillus endocarditis, there were no valvular vegetations but there was infection of the nonvalvular endocardium (so-called mural endocarditis). Although myocardial seeding is frequently present in cases of aspergillus endocarditis, the myocardial infections were generally not contiguous with the endocardial lesions [46].

S. aureus is the most common cause of infective endocarditis in solid organ transplant recipients, accounting for 30% of cases in our series. *S. aureus* endocarditis in transplant recipients is commonly associated with previous isolation of the organism and, in some cases, inadequate treatment of the prior infection. In three cases, *S. aureus* had been isolated from blood cultures >3 weeks prior to the diagnosis of infective endocarditis, and the patients had been treated with parenteral antistaphylococcal antibiotics for <14 days (table 4). Many experts would consider that this treatment is suboptimal for staphylococcal bacteremia [50]. Although it is possible that these patients had endocarditis originally that relapsed after insufficient treatment of endocarditis, we hypothesize that in these cases suboptimal treatment of staphylococcal bacteremia allowed seeding of the valvular endothelium with subsequent development of endocarditis.

In summary, infections due to *Aspergillus* and *S. aureus* accounted for 50% of the cases of infective endocarditis in solid organ transplant recipients. *A. fumigatus* was the most common cause of infective endocarditis within 30 days after transplantation, and *S. aureus* was the most frequent pathogen after this time. Most of the cases of infective endocarditis were associated with previous hospital-acquired infections, notably venous access device and wound infections. Since most cases of endocarditis in transplant recipients occur in patients with previously normal heart valves and hence may not be readily predicted, prevention and aggressive treatment of hospital-acquired infections, particularly bacteremia, in all transplant recipients are of paramount importance. To gain a greater understanding of endocarditis in transplant recipients, we recommend that other institutions prospectively evaluate and report their experience with this disease process.

References

1. Scheld WM, Sande MA. Endocarditis and intravascular infections. In: Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas, and Bennett's principles and practice of infectious diseases. 4th ed. New York: Churchill Livingstone, **1995**:740–83.
2. Wagener MM, Yu VL. Bacteremia in transplant recipients: a prospective study of demographics, etiologic agents, risk factors and outcomes. Am J Infect Control **1992**;20:239–47.
3. Durack DT, Lukes AS, Bright DK, et al. New criteria for the diagnosis of infective endocarditis. Am J Med **1994**;96:200–9.
4. Millett R, Tomita T, Marshall HE, Cohen L, Hannah H. Cytomegalovirus endomyocarditis in a transplanted heart. Arch Pathol Lab Med **1991**; 115:511–5.
5. Rabito FJ, Pankey GA. Infections in orthotopic heart transplant patients at the Oschner Medical Institutions. Med Clin North Am **1992**;76: 1125–34.

6. Grossi P, De Maria R, Caroli A, Zaina MS, Minoli L. Infections in heart transplant recipients: the experience of the Italian Heart Transplantation Program. J Heart Lung Transplant **1992**;11:847–66.
7. Patel R, Portela D, Badley AD, et al. Risk factors of invasive *Candida* and non-*Candida* fungal infections after liver transplantation. Transplantation **1996**;62:926–34.
8. Stewart R, Sethia B, Bain WH. Cardiac surgery in patients with functional renal transplants. Thorac Cardiovasc Surg **1986**;34:86–7.
9. Venditti M, Biavasco F, Varaldo PE, et al. Catheter-related endocarditis due to glycopeptide-resistant *Enterococcus faecalis* in a transplanted heart. Clin Infect Dis **1993**;17:524–5.
10. Cooper DKC, Oliver LS, Forder AA, et al. Infectious complications after heart transplantation. Thorax **1983**;38:822–8.
11. Dresler C, Uthoff K, Wahlers T, et al. Open heart operations after renal transplantation. Ann Thorac Surg **1997**;63:143–6.
12. Laws KH, Merrill WH, Hammon JW, Prager RL, Bender HW. Cardiac surgery in patients with chronic renal disease. Ann Thorac Surg **1986**; 42:152–7.
13. Reddy SC, Katz WE, Medich GE, et al. Infective endocarditis of the pulmonary artery conduit in a recipient with a heterotopic heart transplant: diagnosis by transesophageal echocardiography. J Heart Lung Transplant **1994**;13:139–41.
14. McGiffin DC, Galbraith AJ, McCarthy JB, Tesar PJ. Mycotic false aneurysm of the aortic suture line after heart transplantation. J Heart Lung Transplant **1994**;13:926–8.
15. Adams BK, Fataar A, Boniaszczuk J, Kahn D. Equilibrium radionuclide angiocardiography in the detection of unsuspected mycotic aneurysms in cardiac transplantation. Transplantation **1992**;53:681–3.
16. Albes J, Haverich A, Freihorst J, von der Hardt H, Manthey-Stiers F. Management of mycotic rupture of the ascending aorta after heart-lung transplantation. Ann Thorac Surg **1990**;50:982–3.
17. Balaji S, Whitehead B, Elliott MJ, de Leval MR. Pseudoaneurysm of the aorta after heart-lung transplantation: diagnosis by color flow Doppler mapping. J Heart Lung Transplant **1992**;11:160–3.
18. Oaks TE, Pae WE, Pennock JL, Myers JL, Pierce WS. Aortic rupture caused by fungal aortitis: successful management after heart transplantation. J Heart Transplant **1988**;7:162–4.
19. Palac RT, Strausbaugh LJ, Antonovic R, Floten HS. An unusual complication of cardiac transplantation-infected aortic pseudoaneurysm. Ann Thorac Surg **1991**;51:479–81.
20. Thomson D, Menkis A, Pflugfelder P, Kostuk W, Ahmad D, McKenzie FN. Mycotic aortic aneurysm after heart-lung transplantation. Transplantation **1989**;47:195–7.
21. Slater AD, Ganzel BL, Keller M, Tobin GR, Gray LA. Repair of infected pseudoaneurysm with aortic arch replacement after orthotopic heart transplantation. J Heart Transplant **1990**;9:230–5.
22. Dowling RD, Baladi N, Zenati M, et al. Disruption of the aortic anastomosis after heart-lung transplantation. Ann Thorac Surg **1990**;49:118–22.
23. Bolman RM, Anderson RW, Molina JE, et al. Cardiac operations in patients with functioning renal allografts. J Thorac Cardiovasc Surg **1984**; 88:537–43.
24. Bull DA, Stahl RD, McMahan DL, et al. The high risk heart donor: potential pitfalls. J Heart Lung Transplant **1995**;14:424–8.
25. Counihan PJ, Yelland A, De Belder MA, Pepper JR. Infective endocarditis in a heart transplant recipient. J Heart Lung Transplant **1991**;10: 275–9.
26. David CA, Horowitz MD, Burke GW. Aortic valve endocarditis in a liver transplant recipient—successful management by aortic valve replacement. Transplantation **1992**;53:1366–7.
27. Egawa H, Woodley S, Keeffe EB, et al. Aortic valve endocarditis after orthotopic liver transplantation. Transplantation **1994**;58:732–4.
28. Estol CJ, Pession MS, Matinez AJ. Cerebrovascular complications after orthotopic liver transplantation: a clinicopathologic study. Neurology **1991**;41:815–9.
29. Fazia RB, Mills RM, Conti CR, Staples ED. Aortic valve endocarditis in an acutely rejecting orthotopic heart transplant recipient. Clin Cardiol **1996**;19:672–4.
30. Hasan A, Hamilton JRL, Au J, et al. Surgical management of infective endocarditis after heart-lung transplantation. J Heart Lung Transplant **1993**;12:330–2.
31. Kaplan R, Duncalf D, Cizmar S. *Aspergillus* pancarditis and cardiac arrest during anesthesia. Anesth Analg **1981**;60:440–4.
32. Keating MR, Guerrero MA, Daly RC, Walker RC, Davies SF. Transmission of invasive aspergillosis from a subclinically infected donor to three different organ transplant recipients. Chest **1996**;109:1119–24.
33. Khoo DE, Zebro TJ, English TAH. Bacterial endocarditis in a transplanted heart. Pathol Res Pract **1989**;185:445–7.
34. Losman JG, Curcio A, Barnard C. Normal cardiac function with a hybrid heart. Ann Thorac Surg **1978**;26:177–84.
35. Martinez-Vea A, Costa J, Garcia C, et al. *Corynebacterium* group JK endocarditis in a haemodialysis patient. Nephrol Dial Transplant **1993**; 8:177–9.
36. Muakkassa WF, Mohanty PK, Kipreous B, Lee HM, Goldman MH. Left ventricular mass with septic (*Clostridium ramosum*) arterial emboli in a renal allograft patient: report of a case and review of the literature. Transplant Proc **1983**;15:1715–9.
37. Niehues R, Schluter S, Kramer A, et al. Endocarditis complicating systemic *Nocardia asteroides* infection in a patient under immunosuppressive therapy. Dtsch Med Wochenschr **1996**;121:1390–5.
38. Prada JL, Villanueva JL, Torre-Cisneros J, Rodriguez F, Espinosa M, Anguita M. Endocarditis due to corynebacteriaceae in a kidney transplant patient. Nephrol Dial Transplant **1994**;9:1185–7.
39. Pruett TL, Rotstein OD, Anderson RW, Simmons RL. Tricuspid valve *Candida* endocarditis: successful treatment with valve-sparing debridement and antifungal chemotherapy in a multiorgan transplant recipient. Am J Med **1986**;80:116–8.
40. Schwarz A, Offermann G, Keller F, et al. The effect of cyclosporine on the progression of human immunodeficiency virus type 1 infection transmitted by transplantation—data on four cases and review of the literature. Transplantation **1993**;55:95–103.
41. Spes CH, Angermann CE, Mudra H, Kemkes BM, Theisen K. Infectious endocarditis following orthotopic heart transplantation. Z Kardiol **1990**; 79:66–8.
42. Stewart MJ, Huwez F, Richens D, Naik S, Wheatley DJ. Infective endocarditis of the tricuspid valve in an orthotopic heart transplant recipient. J Heart Lung Transplant **1996**;15:646–9.
43. Toporoff B, Rosado LJ, Appleton CP, Sethi GK, Copeland JG. Successful treatment of early infective endocarditis and mediastinitis in a heart transplant recipient. J Heart Lung Transplant **1994**;13:546–8.
44. Walsh TJ, Hutchins GM. *Aspergillus* mural endocarditis. Am J Clin Pathol **1979**;71:640–4.
45. Welty FK, McLeod GX, Ezratty C, Healy RW, Karchmer AW. *Pseudallescheria boydii* endocarditis of the pulmonic valve in a liver transplant recipient. Clin Infect Dis **1992**;15:858–60.
46. Woods GL, Wood RP, Shaw BW. Aspergillus endocarditis in patients without prior cardiovascular surgery: report of a case in a liver transplant recipient and review. Rev Infect Dis **1989**;11:263–72.
47. Uip DE, Neto VA, Strabelli TM, et al. Infective endocarditis in 100 patients subjected to heart transplantation. Arq Bras Cardiol **1996**;66:1–3.
48. Everett ED, Hirschmann JV. Transient bacteremia and endocarditis prophylaxis: a review. Medicine (Baltimore) **1977**;56:61–78.
49. Fang G, Keys TF, Gentry LO, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia. Ann Intern Med **1993**;119:560–7.
50. Rupp ME. Infections of intravascular catheters and vascular devices. In: Crossley KB, Archer GL, eds. The staphylococci in human disease. New York: Churchill Livingstone, **1997**:309–30.