ISSN Print: 2394-7500
ISSN Online: 2394-5869
IJAR 2015; 1(5): 193-199
www.allresearchjournal.com
Received: 13-03-2015
Accepted: 15-04-2015

Suresh Kumar Behera
Department of Cardiology, IMS and SUM Hospital, Siksha O Anusandhan University, K 8, Kalinga Nagar, Bhubaneswar, Odisha, India

Rajesh Kumar Lenka
Department of Microbiology, IMS and SUM Hospital, Siksha O Anusandhan University, K 8, Kalinga Nagar, Bhubaneswar, Odisha, India

Correspondence:
Rajesh Kumar Lenka
Department of Microbiology, IMS and SUM Hospital, Siksha O Anusandhan University, K 8, Kalinga Nagar, Bhubaneswar, Odisha, India

Pseudomonas aeruginosa infective endocarditis in patients who do not use intravenous drugs: Our experience

Suresh Kumar Behera and Rajesh Kumar Lenka

Abstract
Aim and objectives: Infective endocarditis (IE) because of *Pseudomonas aeruginosa* is uncommon and represents just about 3% of all patients with this malady. Most contaminations are related with the utilization of intravenous medications. Patients with *P. aeruginosa*-related IE who don't utilize intravenous drugs are incredibly uncommon. We did an audit of the writing to recognize the nature what's more, hazard elements of this malady.
Methods: Patients with IE reported between 1993 and 2013 were reviewed by searching the Medline database using the keywords "endocarditis" and "*Pseudomonas aeruginosa*". All of the patients included met the definition of the modified Duke criteria.
Results: Twenty-seven patients in 22 reports were looked into. IE connected with social insurance represented 20 patients (74%). The mean age of the patients was 53.4 years and there was a transcendence of men (81.5%). Local valve endocarditis was found in 20 (74.1%) patients. Medical procedure for contamination control was performed in 15 (55.6%) patients and the death rate in patients who experienced medical procedure was 33.3% (five patients). A backslide of IE after sufficient treatment was found in nine (33.3%) patients. The death rate in each of the 27 patients was 28.6% (2/7) for those with network obtained IE and 40% (8/20) for those with IE connected with medicinal services. Univariate examination demonstrated a higher death rate in patients matured >60 years and in those whose wellspring of endocarditis was identified with a prosthetic gadget.
Conclusion: *P. aeruginosa* endocarditis has substantial morbidity and mortality. It is characterized by easy relapse and is highly associated with prosthetic devices.

Keywords: infective endocarditis; *Pseudomonas aeruginosa*; risk factors

Introduction
Infective endocarditis (IE) brought about by Pseudomonas aeruginosa is inconsistent, representing around 3% of all patients with IE.1 About 90% of patients with IE identified with *P. aeruginosa* utilize intravenous drugs. 2 Patients with IE identified with *P. aeruginosa* who don't utilize intravenous medications are incredibly uncommon and just a couple of patient reports and brief case arrangement have been distributed. A large portion of these reports uncover that IE brought about by *P. aeruginosa* might be extremely forceful and has a high rate of mortality. To give more bits of knowledge into the clinical introductions of this infection and to assess the prognostic factors, a writing audit covering the period 1993 to 2013 was led, including one late patient treated in IMA and SUM medical clinic, Bhubaneswar, India.

Materials and Methods
A precise Medline audit covering the period from January 1, 1993 to June 30, 2013 was led utilizing the pursuit terms "endocarditis" and "Pseudomonas aeruginosa". The hunt concentrated just on articles distributed in English. Two clinicians read the articles freely and chose which articles ought to be incorporated into the audit. The articles were incorporated if the patient announced met the changed Duke criteria of IE with disengages of *P. aeruginosa*. 3 These articles contained explicit data on clinical, helpful, and result factors. For every patient announced, the accompanying factors were recorded: statistic information (Age and sex), clinical information (Site and kind of valves influenced, echocardiography discoveries, passage site of the contamination, any past history of intrusive strategies, and a backslide of the IE after culmination of treatment), sort of treatment (Antimicrobial treatment with or without medical

procedure), and result (demise or recuperation). An ongoing intrusive methodology was characterized as an intravascular devicerelated technique inside 1 year of the scene of *P. aeruginosa* IE, for example, prosthetic valve substitution, focal venous catheter inclusion, an arterio-venous unite task, cardiovascular catheterization, pacemaker addition, and significant medical procedure (i.e., open heart medical procedure), renal transplantation, biliary prosthesis implantation, and intrusive urinary techniques, for example, cystoscopy. IE connected with social insurance was characterized as either nosocomial disease or non-nosocomial contamination related with wellbeing care.4 Nosocomial disease was characterized as IE creating in a patient hospitalized for >48 hours before the beginning of the side effects and indications of IE. Non-nosocomial disease related with human services was characterized as IE analyzed inside 48 hours of affirmation as an outpatient with broad social insurance contact including any of the accompanying criteria: [1] the patient had gotten intravenous treatment, wound consideration, or specific nursing care at home inside 30 days preceding the beginning of *P. aeruginosa* IE; [2] the patient had gone to a medical clinic or hemodialysis center or got intravenous chemotherapy inside 30 days preceding the beginning of *P. aeruginosa* IE; [3] the patient was hospitalized in an intense consideration emergency clinic for _2 days in the 90 days preceding the beginning of *P. aeruginosa* IE; or [4] the patient dwelled in a nursing home or long haul care facility.4,5 Community-procured IE that was not identified with intravenous medication use was characterized as IE analyzed at the season of confirmation (or inside 48 hours of affirmation) in a patient not satisfying the criteria for disease related with human services and with no history of utilizing intravenous medications. Immunocompromised patients were characterized as patients who were accepting long haul immunosuppressive treatment, or who had hematological ailment because of chemotherapy. A backslide of IE after sufficient treatment is characterized as another scene brought about by *P. aeruginosa* inside a half year of the primary scene of *P. aeruginosa* IE. Factual investigation SPSS variant 17.0 (SPSS Inc., Chicago, IL, USA) was utilized for every measurable examination. The mean, standard deviation, middle, and range were determined for persistent factors. The c2 test and Fisher's careful test were utilized to decide noteworthy contrasts between gatherings with unmitigated factors. Univariate investigations were utilized to decide the relationship between potential hazard variables and mortality. Factual hugeness was set at $p<0.05$.

Results

In this extensive audit of the writing from 1993 to 2013, 27 patients with *P. aeruginosa* IE were recognized from 22 articles.6e27 All 27 patients met the changed Duke criteria and none of the patients utilized intravenous medications.

Network gained *P. aeruginosa* IE represented seven patients (25.9%) and *P. aeruginosa* IE connected with social insurance represented 20 patients (74.1%). The clinical attributes of the announced patients are given in Table 1. The patients with network gained IE had a mean time of 50.9 years and those with IE connected with medicinal services had a mean time of 54.4 years. Five (71.4%) patients with network gained IE were men and 17 (85%) patients with IE connected with human services were men. Seventeen (85%) patients with IE connected with human services had a known entryway of section, contrasted and just two (28.6%) patients with network obtained IE. Comorbidities happened in six (85.7%) patients with network gained IE and in 14 (70%) patients with IE connected with medicinal services. Nine (45%) patients with IE connected with social insurance had a past filled with an ongoing intrusive methodology, which was just found in one (14.3%) understanding with network procured IE. Contamination of the local valve happened in five (71.4%) patients with community acquired IE and in 15 (75%) patients with IE connected with human services. Left-sided IE happened in four (57.1%) patients with network gained IE and 13 (65%) of patients with IE connected with medicinal services. Of the 27 patients who got echocardiography, 25 (92.6%) had noticeable bacterial development. Medical procedure for disease control was required in four (57.1%) patients with network procured IE and in 11 (55%) patients with IE connected with human services. A backslide of IE after satisfactory treatment happened in two (28.6%) patients with network procured IE and in seven (35%) patients with IE connected with social insurance. The general mortality of *P. aeruginosa* IE was 28.6% in network gained IE and 40% in IE connected with medicinal services. Contrasted and network obtained *P. aeruginosa* IE, just the known site of passage of the contamination was fundamentally higher in IE connected with medicinal services in the univariate examination. The mortalities and survival of patients with *P. aeruginosa* IE were looked at (Table 2). In univariate examination, age >60 years and a prosthetic gadget as the wellspring of IE were factors that had noteworthy relationship with mortality. Patients who backslid and the individuals who did not backslide with *P. aeruginosa* IE were thought about (Table 3). In univariate investigation, just mortality was essentially higher in patients who backslid.

Table 1: Clinical characteristics of 27 patients with *Pseudomonas aeruginosa* infective endocarditis

Variable	N (%) (n Z 27)		N (%) with community-acquired infection (n Z 7)		N (%) with infection associated with health care (n Z 20)	p
Age(y)	53.4 16.4	(216.3)	50.9 15.7 (21e66)		54.4 16.9 (21e83)	0.64
Age 60 y	11	(40.7)	(42.9)	8	(40.0)	>0.99
Male sex	22	(81.5)	(71.4)	17	(85.0)	0.58
History of congenital heart disease	3	(11.1)	(28.6)	1	(5.0)	0.16
Known site of entry of infection	19	(70.4)	(28.6)	17	(85.0)	0.01 *
Diabetes mellitus	5	(18.5)	(28.6)	3	(15.0)	0.58
Hemodialysis	3	(11.1)	(0)	3	(15.0)	0.55
Immunocompromised patient	5	(18.5)	(0)	5	(25.0)	0.28
Location						
Left-side	17	(63.0)	(57.1)	13	(65.0)	d
Mitral valve	8	(29.6)	(42.9)	5	(25.0)	d
Aortic valve	8	(29.6)	(0)	8	(40.0)	d

Both	1	(3.7)	(14.3)	0	(0)	d
Right-side	7	(25.9)	(28.6)	5	(25.0)	d
Tricuspid valve	1	(3.7)	(0)	1	(5.0)	d
Pulmonary valve	3	(11.1)	(14.3)	2	(10.0)	d
Both valves	3	(11.1)	(14.3)	2	(10.0)	d
Pacemaker	2	(7.4)	(0)	2	(10.0)	d
Patent foramen ovule	1	(3.7)	(14.3)	0	(0)	d
Bacterial growth seen on echocardiography	25	(92.6)	(100.0)	18	(90.0)	>0.99
Echocardiography						

Table 2: Comparisons between patients who died and surviving patients with *Pseudomonas aeruginosa* infective endocarditis

Variable	Total no. of patients N (%) of patients who died			OR (95% CI)		p
Age 60 y	11	7	(63.6)	7.58	(1.31.53.92)	0.04 *
Male sex	22	9	(40.9)	0.36	(0.03.3.79)	0.62
IE associated with health care	7	2	(28.6)	0.60	(0.09.6.89)	0.68
Diabetes mellitus	5	2	(40.0)	1.17	(0.16e8.53)	>0.99
Hemodialysis	3	0	(0)	d		d
Congenital heart disease	3	1	(33.3)	0.83	(0.07.80.55)	>0.99
Immunocompromised patient	5	3	(60.0)	3.21	(0.43e23.79)	0.33
Surgery for infection control	15	5	(33.3)	0.70	(0.15.7.37)	0.71
Prosthetic valve	6	4	(66.7)	4.67	(0.67.62.74)	0.16
Bacterial growth seen on echocardiography	25	9	(36.0)	0.56	(0.03.50.11)	>0.99
Right-sided vegetation	7	2	(28.6)	0.60	(0.09.6.17)	>0.99
Prosthetic device-related infection	16	9	(56.3)	12.88 (1.312.25.78)		0.02 *
Previous open heart surgery	9	4	(44.4)	1.60	(0.31.6.25)	0.68
History of recent invasive procedure	10	4	(40.0)	1.22	(0.24e6.11)	>0.99
Complications	19	9	(47.3)	6.30	(0.64e61.63)	0.19
Congestive heart failure	10	6	(60.0)	4.88	(0.90e26.42)	0.10
Intracardiac abscess	4	3	(75.0)	6.86	(0.60e77.98)	0.13
Embolization	7	2	(28.6)	0.60	(0.09e0.89)	0.68

Table 3: Comparisons between relapse and non-relapse patients of *Pseudomonas aeruginosa* infective endocarditis

Variable	N (%) of patients relapsing		OR (95% CI)		p
Age 60 (y)	5	(45.5)	2.50	(0.49.52.89)	0.41
Male	8	(36.4)	0.44	(0.41.4.62)	0.64
Health care-associated	7	(35.0)	0.74	(0.11.7.87)	>0.99
Diabetes mellitus	2	(40.0)	1.43	(0.19.40.57)	>0.99
Hemodialysis	0	(0)	d		d
Congenital heart disease	2	(66.7)	4.68	(0.38.62.63)	0.25
Immunocompromised patient	1	(20.0)	0.43	(0.04.4.62)	0.64
Prosthetic valve	3	(50.0)	2.33	(0.36.35.05)	0.63
Bacterial growth on echocardiography	8	(32.0)	0.47	(0.03.4.52)	>0.99
Right-sided vegetation	3	(42.9)	1.38	(0.23.3.30)	>0.99
Prosthetic device-related	5	(45.5)	2.50	(0.49.52.89)	0.41
Previous open heart surgery	5	(55.6)	4.38	(0.78.34.47)	0.11
Recent invasive procedure history	3	(30.0)	0.79	(0.15.7.21)	>0.99
Surgery for infection control	5	(33.3)	1.00	(0.20.0.00)	>0.99
Complication	8	(42.1)	5.09	(0.52.00.00)	0.20
Congestive heart failure	5	(50.0)	3.25	(0.61.27.28)	0.22
Intracardiac abscess	3	(60.0)	4.00	(0.53.00.16)	0.29
Embolization	3	(42.9)	1.75	(0.30.7.00)	0.65
Mortality	7	(70.0)	17.5	(2.377.29.51)	<0.05 *

Discussion

P. aeruginosa IE was first detailed in 1899 by Reyes *et al*. [28] t has a place with the gathering of non-HACEK gram-negative microorganisms (species other than Haemophilus species, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella corrodens, or Kingella spp.) that reason IE. About 90% of patients with *P. aeruginosa* IE utilize intravenous drugs 2; in any case, social insurance related contamination might be another critical hazard factor in *P. aeruginosa* IE. The investigation of Morpeth *et al*. [29] stressed the significance of the relationship between social insurance contact and non-HACEK gram-negative bacillus IE. In their investigation, the greater part of the patients with non-HACEK gram-negative bacillus IE had disease related with social insurance (57%) and embedded endovascular gadgets were as often as possible related with non-HACEK gram-negative bacillus IE (p< 0.05). In this examination, contamination related with social insurance in patients with *P. aeruginosa* IE who did not utilize intravenous medications represented 20 (74.1%) of 27 patients.

In this investigation, patients with *P. aeruginosa* IE with a past filled with a past intrusive system represented 15 (55.6%) of 27 patients. IE identified with a prosthetic gadget represented [11] (40.7%) patients. In an investigation of 1779 patients, an assumed intravascular gadget source accounted 28.4% of patients with Staphylococcus aureus IE.4 Patients with IE identified with a prosthetic gadget had a higher death rate in this audit (p< 0.05). Therefore gadget related contamination is

an imperative hazard factor for mortality in *P. aeruginosa* IE. Despite the fact that staphylococci represent most of devicerelated contaminations and either coagulase-negative staphylococci or S. aureus is the most widely recognized pathogen distinguished, 30 this audit proposes that *P. aeruginosa* should even now be considered in gadget related IE. In an accomplice contemplate with 1874 patients, rehash IE happened in 4.8%. Among these, 81% had another contamination and 19% were attempted to be backslid patients. S. aureus was the real reason for rehashed IE (41%); the backslide rate of S. aureus in this associate investigation was 2.9% (15/519).31 In the present survey, 33% (9/27) of patients had a backslide of IE and had a noteworthy contrast in mortality ($p< 0.05$). These propose that *P. aeruginosa* IE may have a higher rate of backslide than different microorganisms and a higher mortality if patients have an assumed backslide. Past investigations have distinguished hazard factors for rehashed IE, for example, intravenous medication use, inborn and rheumatic coronary illness, earlier episode (s) of endocarditis, endless dialysis, male sex, and more seasoned age (>65 years).31 In the present survey, examination of the hazard components and result among backslid and non-backslid patients indicated just a single noteworthy contrast in mortality (Table 3). In the present audit, *P. aeruginosa* IE in patients who did not utilize intravenous medications normally created in male patients with a period of around 50 years. Most patients gained the contamination through an intrusive system (14.3% in network procured IE; 45% in IE connected with human services), which is like that of S. aureus IE, the main current reason for IE. In the examination by Fowler *et al*, [4] an ongoing obtrusive technique represented 9.6% of community acquired S. aureus IE in patients who did not utilize intravenous medications and 48.6% of patients with S. aureus IE connected with human services. In the present survey, *P. aeruginosa* all the more often attacked the left-sided valves, with mitral or aortic valve contribution, than the right-sided valves (55% versus 33%). The greater part of diseases included the local valve (76.9%). In this examination, 55.6% (15/27) of patients experienced valve substitution or other open heart medical procedure for contamination control. Four arrangement demonstrated that rates of valve substitution in IE brought about by all pathogens went from 15% to 45%.1,32e34 The rate of medical procedure in patients with S. aureus IE in an investigation of 1779 patients was 37.8%,4 demonstrating that *P. aeruginosa* IE may have a higher rate of medical procedure. Albeit heart valve medical procedure was suggested for non-HACEK gram-negative bacillus IE, which included *P. aeruginosa* IE, [35] the death rate in this survey did not uncover any critical distinction among careful and non-careful treatment. Inborn coronary illness (CHD) is the main hazard factor for pediatric IE in creating nations after the decay of rheumatic heart disease [36] In an extensive survey article, the main microorganism causing CHD-related IE was Streptococcus spp. (44%), Staphylococcus spp. (27%), and Gramnegative HACEK bunch microbes (16%). [36] *P. aeruginosa* is an uncommon pathogen for CHD-related IE. In this audit, three patients had a past filled with CHD and none of the three experienced open heart medical procedure for CHD redress. One was effectively treated with antimicrobial treatment alone. The other two patients experienced heart medical procedure for IE control and one of them kicked the bucket because of a recurrent scene of IE. The general mortality in CHD-related IE had diminished to 10% during the 1990s because of enhancements in determination, antimicrobial treatment, cardiovascular medical procedure, and interventional catheter treatment [36]. In an investigation with 216 patients, careful mediation was as yet a prescient factor for a lower rate of in-emergency clinic mortality in CHD-related IE.37 However, most arrangement had couple of patients of CHD-related *P. aeruginosa* IE, in this way justifying further investigation and examination as far as careful sign and results. Confusions of *P. aeruginosa* IE incorporate heart disappointment and blood vessel emboli including the mind, spleen, and kidneys. 38 Cerebritis, mind abscesses, and mycotic aneurysms may likewise occur [39]. In this audit, one patient had meningitis26 and another had endophthalmitis [15]. The rate of complexities was 85.7% (6/7 patients) in network obtained IE and 65% (13/20 patients) in IE connected with human services, which are moderately higher than that of S. aureus IE (run 12.7e8.9%) [4]. Patients with entanglements likewise have higher death rates (47% versus 12.5%). In this investigation, the death rate related with *P. aeruginosa* IE was 28.6% in network obtained IE and 40% in IE connected with medicinal services. In a past arrangement, the general mortality ran from 18.0% to 24.6%.1, 32e34 In an investigation of S. aureus IE, the mortality_ENREF_35 rate was 21.1% in IE not related with intravenous medication use and 29.4% in IE connected with wellbeing care.4 Compared with these outcomes, the mortality of patients with *P. aeruginosa* IE was comparative in essential network gained IE and somewhat higher in IE connected with human services. Another arrangement with few patients showed that *P. aeruginosa* IE connected with intravenous medication use had a death rate of 10% (1/10). 40 In the present survey, the mortality of network procured *P. aeruginosa* IE not related with intravenous medication use (28.6%) had all the earmarks of being like that of *P. aeruginosa* IE connected with intravenous medication use. Be that as it may, further investigation ought to be performed to check this.

Conclusions

There are a few constraints in this investigation. The fundamental wellspring of data was patients detailed in the writing, which may have critical choice inclination for a higher seriousness, progressively convoluted course, and more unfortunate result. An individual specialist's report can't be institutionalized for the patient factors and a predisposition may likewise exist. A few patients who might be viewed as contender for medical procedure may have been prohibited from careful choices because of their poor condition. Certain subgroups of few patients may create type II blunders. There are missing information in these writing reports, incorporate the seriousness of infection, sufficiency of the anti-toxin routine, satisfactory dose and span of the anti-toxin routine, size of bacterial development, sign for medical procedure, and powerlessness information of *P. aeruginosa*. The patient numbers in this examination are too little to even consider drawing a noteworthy conclu particle. All in all, *P. aeruginosa* IE not identified with intravenous medication use is uncommon, yet is a forceful illness. It has the normal for simple backslide and is exceptionally connected with the utilization of a prosthetic gadget. Later on, if more arrangement reports are distributed, there ought to be adequate patients for an increasingly nitty gritty investigation.

References

1. Sandre RM, Shafran SD. Infective endocarditis: review of 135 cases over 9 years. Clin Infect Dis. 1996; 22:276e86.
2. Wieland M, Lederman MM, Kline-King C, Keys TF, Lerner PI, Bass SN *et al*. Left-sided endocarditis due to

Pseudomonas aeruginosa. A report of 10 cases and review of the literature. Medicine (Baltimore) 1986; 65:180e9.
3. Li JS, Sexton DJ, Mick N, Nettles R, Fowler Jr VG, Ryan T *et al*. Proposed modifications to the Duke criteria for the diagnosis of infective endocarditis. Clin Infect Dis. 2000; 30:633e8.
4. Fowler Jr VG, Miro JM, Hoen B, Cabell CH, Abrutyn E, Rubinstein E *et al*. Staphylococcus aureus endocarditis: a consequence of medical progress. JAMA. 2005; 293:3012e21.
5. Friedman ND, Kaye KS, Stout JE, McGarry SA, Trivette SL, Briggs JP *et al*. Health care-associated bloodstream infections in adults: a reason to change the accepted definition of community-acquired infections. Ann Intern Med 2002; 137:791e7.
6. Aggarwal A, Ritter N, Reddy L, Lingutla D, Nasar F, El-Daher N *et al*. Recurrent Pseudomonas aortic root abscess complicating mitral valve endocarditis. Heart Lung. 2012; 41:181e3.
7. Apple J, Hunt JL, Wait M, Purdue G. Delayed presentations of aortic valve endocarditis in patients with thermal injury. J Trauma. 2002; 52:406e9.
8. Bicanic TA, Eykyn SJ. Hospital-acquired, native valve endocarditis caused by *Pseudomonas aeruginosa*. J Infect 2002; 44:137e9.
9. Branco L, Pitta ML, Galrinho A, Real HC, da Cruz AG, Conceicao J *et al*. A rare cause of late infective endocarditis after heart surgery. J Am Soc Echocardiogr 1997; 10:371e4.
10. Chacko ST, Chandy ST, Abraham OC, Swaminathan S, Varghese GM, Priscilla R *et al*. Pacemaker endocarditis caused by *Pseudomonas aeruginosa* treated successfully. J Assoc Physicians India 2003; 51:1021e2.
11. Chikazawa G, Nakajima M, Hirayama T, Misumi H, Ideta I, Tomioka H *et al*. A case of radical operation for right-sided infective endocarditis due to *Pseudomonas aeruginosa* concurrent with VSD in the elderly person. Kyobu Geka 1997; 50:483e6 [in Japanese].
12. Dawson NL, Brumble LM, Pritt BS, Yao JD, Echols JD, Alvarez S. Left-sided *Pseudomonas aeruginosa* endocarditis in patients without injection drug use. Medicine (Baltimore) 2011; 90:250e5.
13. Gavin PJ, Suseno MT, Cook FV, Peterson LR, Thomson Jr RB. Left-sided endocarditis caused by *Pseudomonas aeruginosa*: successful treatment with meropenem and tobramycin. Diagn Microbiol Infect Dis 2003; 47:427e30.
14. Hassan KS, Al-Riyami D. Infective endocarditis of the aortic valve caused by *Pseudomonas aeruginosa* and treated medically in a patient on haemodialysis. Sultan Qaboos Univ Med J 2012; 12:120e3.
15. Hsu KH, Ben RJ, Shiang JC, Feng NH. *Pseudomonas aeruginosa* endocarditis associated with endophthalmitis caused by arteriovenous fistula and graft infection. J Chin Med Assoc. 2003; 66:617e20.
16. Barshay J, Nemets A, Ducach A, Lugassy G. *Pseudomonas aeruginosa* endocarditis in acute myeloid leukemia: a rare complication. Int J Biomed Sci. 2008; 4:330e2.
17. Kato Y, Ohashi H, Tsutsumi Y, Murakami T, Takahashi Y. Prosthetic valve endocarditis caused by metallo-beta-lactamaseproducing *Pseudomonas aeruginosa*. J Card Surg 2009; 24:347e9.
18. Laguno M, Miro O, Font C, de la Sierra A. Pacemaker-related endocarditis. Report of 7 cases and review of the literature. Cardiology 1998; 90:244e8.
19. Nasim A, Baqi S, Akhtar SF. *Pseudomonas aeruginosa* endocarditis in renal transplant recipients. Transpl Infect Dis. 2012; 14:180e3.
20. Reinsch N, Plicht B, Lind A, Janosi RA, Buck T, Kamler M *et al*. Recurrent infective endocarditis with uncommon Gramnegative Pasteurella multocida and *Pseudomonas aeruginosa*: a case report. J Heart Valve Dis 2008; 17:710e3.
21. Sa MI, Moco R, Cabral S, Reis AH, Pereira LS, Torres S *et al*. Isolated pulmonary valve endocarditis due to *Pseudomonas aeruginosa*. Rev Port Cardiol 2007; 26:43e8.
22. Saraiva RM, Camillis LF, Francisco RM, Gomes MV. Isolated pulmonary valve *Pseudomonas aeruginosa* endocarditis related to catheter embolism. Int J Cardiol 2002; 83:83e4.
23. Stollberger C, Bastovansky A, Finsterer J. Fatal septicemia in a patient with cerebral lymphoma and an Amplatzer septal occluder: a case report. J Med Case Rep 2011; 5:554.
24. Tembe AG, Kharbanda P, Dalal JJ, Vaishnav G, Joshi VR. Infective endocarditis e a tale of two cases and the lessons (re) learned. J Assoc Physicians India 2010; 58:319e22.
25. Todaro J, Bollmann PW, Nussbacher A, Camargo LF, Santos BF, Alvarenga D *et al*. Multiple myeloma complicated with pseudomonas endocartiditis. Einstein (Sao Paulo). 2012; 10:498e501.
26. Venkatesan A, Spalding C, Speedie A, Sinha G, Rumbaugh JA. *Pseudomonas aeruginosa* infective endocarditis presenting as bacterial meningitis. J Infect. 2005; 51:e199e202.
27. Yilmaz M, Sunar H, Mert A. Community-acquired left-sided *Pseudomonas aeruginosa* endocarditis in a patient without intravenous drug use. Infection. 2013; 41:243e5.
28. Reyes MP, Palutke WA, Wylin RF. Pseudomonas endocarditis in the Detroit Medical Center. 1969e1972. Medicine (Baltimore). 1973; 52:173e94.
29. Morpeth S, Murdoch D, Cabell CH, Karchmer AW, Pappas P, Levine D *et al*. Non-HACEK gram-negative bacillus endocarditis. Ann Intern Med. 2007; 147:829e35.
30. Baddour LM, Bettmann MA, Bolger AF, Epstein AE, Ferrieri P, Gerber MA *et al*. Nonvalvular cardiovascular device-related infections. Circulation. 2003; 108:2015e31.
31. Alagna L, Park LP, Nicholson B, Keiger A, Strahilevitz J, Morris A *et al*. Repeat endocarditis: analysis of risk factors based on the International Collaboration on Endocarditis e Prospective Cohort Study (ICE-PCS). Clin Microbiol Infect. 2013; 20:566e75.
32. Chu J, Wilkins G, Williams M. Review of 65 cases of infective endocarditis in Dunedin Public Hospital. N Z Med J. 2004; 117:U1021.
33. Ferreiros E, Nacinovich F, Casabe JH, Modenesi JC, Swieszkowski S, Cortes C *et al*. Epidemiologic, clinical, and microbiologic profile of infective endocarditis in Argentina: a national survey. The Endocarditis Infecciosa en la Republica Argentina-2 (EIRA-2) Study. Am Heart J 2006; 151:545e52.
34. Krcmery V, Gogova M, Ondrusova A, Buckova E, Doczeova A, Mrazova M *et al*. Etiology and risk factors of 339 cases of infective endocarditis: report froma 10-year national prospective survey in the Slovak Republic. J Chemother 2003; 15:579e83.

35. Baddour LM, Wilson WR, Bayer AS, Fowler Jr VG, Bolger AF, Levison ME *et al*. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. Circulation. 2005; 111:e394e434.
36. Knirsch W, Nadal D. Infective endocarditis in congenital heart disease. Eur J Pediatr 2011; 170:1111e27.
37. Yoshinaga M, Niwa K, Niwa A, Ishiwada N, Takahashi H, Echigo S *et al*. Risk factors for in-hospital mortality during infective endocarditis in patients with congenital heart disease. Am J Cardiol. 2008; 101:114e8.
38. Fowler Jr VG, Bayer AS. Endocarditis and intravascular infections. In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. 7th ed. Philadelphia,
39. PA: Churchill Livingstone/Elsevier. 2010, 1067e112.
40. Reyes MP, Lerner AM. Current problems in the treatment of infective endocarditis due to *Pseudomonas aeruginosa*. Rev Infect Dis 1983; 5:314e21.
41. Reyes MP, Ali A, Mendes RE, Biedenbach DJ. Resurgence of Pseudomonas endocarditis in Detroit, 2006e2008. Medicine (Baltimore). 2009; 88:294e301.