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CASE REPORT

An unusual etiology of infective endocarditis: *Enterobacter cloacae*

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Abstract Gram-negative microorganisms are rarely implicated in causing infective endocarditis (IE). Although the traditionally identified risk factor for Gram-negative endocarditis has been intravenous drug abuse, recent studies have revealed that healthcare contact and the presence of prosthetic cardiac devices are primary risk factors for IE secondary to non-HACEK Gram-negative bacteria. We present a case of Enterobacter endocarditis in a patient with no prior history of valvular heart disease, implanted endovascular device, or intravenous drug abuse. The patient was treated successfully with carbapenem monotherapy. We have reviewed 43 cases of Enterobacter endocarditis reported in the literature to date. Clinical summary and management of IE secondary to Enterobacter based on all the published cases is outlined.

Keywords Enterobacter · Endocarditis

Introduction

Gram-negative microorganisms are rarely implicated in causing infective endocarditis (IE).

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A recent prospective cohort study, the International Collaboration on IE Prospective Cohort Study (ICE-PCS), analyzed 2,761 cases of definite IE from 61 hospitals in 28 countries and reported that only 1.8% of patients had endocarditis due to non-HACEK bacteria [1]. Most of these cases were attributed to *Escherichia coli* and Pseudomonas [1]. Enterobacter was found to cause IE in only 2 patients in this study, thus revealing the extreme rarity of this pathogen in causing endocarditis [1]. Important risk factors for non-HACEK Gram-negative IE were the presence of an implanted endovascular device and healthcare contact [1].

We present a case of Enterobacter endocarditis in a 56-year-old man with no prior history of heart disease, endovascular prostheses, or intravenous drug abuse. He received medical therapy that resulted in a favorable outcome. We have reviewed the clinical information, treatment options, and outcomes of 43 prior cases of Enterobacter endocarditis reported in the literature to date.

Case report

A 56-year-old African-American man with past medical history of urinary bladder carcinoma presented to another institution with intermittent fevers of 1-week duration. He had undergone cystectomy with neobladder reconstruction 4 years previously. On the day prior to admission, his urine had appeared dark and foul-smelling. He also complained of exertional shortness of breath, weakness, and fatigue, but denied chest pain, abdominal pain, nausea, vomiting, diarrhea, rash, or myalgia. There were no sick contacts or recent travel.

The patient's medical history included recurrent urinary tract infections, hypertension, diabetes mellitus, peripheral vascular disease, and depression. He did not have any prior

cardiac disease and denied any drug allergies. His medications included risperidone, aspirin, amlodipine, insulin glargine, and escitalopram. He was a retired steel mill worker. He smokes one pack of cigarettes per day, but did not drink alcohol or use recreational drugs.

His temperature was 38.9°C, heart rate 125 beats per min, blood pressure 76/40 mmHg, and a respiratory rate of 40 breaths/min, saturating 96% on a 100% face mask. Lung examination revealed diminished breath sounds and rales bilaterally. He was tachycardic; no murmurs or rubs were heard. Abdominal examination revealed soft, but distended, abdomen with bowel sounds appreciated in all quadrants. There was an abdominal scar from the prior cystectomy.

The leukocyte count was 26,300/mm^3 with neutrophils 65%, and bands 31%. Hemoglobin was 9.2 g/dl, creatinine was 2.5 mg/dl, and bicarbonate was 9 mmol/l. Urine analysis revealed leukocyte esterase, 197 white blood cells per high-power field, 13 red blood cells per high-power field, and many bacteria. Chest X-ray showed bilateral pulmonary nodular airspace opacities. Blood and urine cultures were collected. Vancomycin, ciprofloxacin, and cefepime were administered.

Owing to his critical hemodynamic parameters, he was transferred to the intensive critical care unit at our institution for further management. Vasopressor agents were started. The patient was also found to have decompensating respiratory status owing to increased work of breathing and was intubated orally. Blood, urine, and respiratory cultures were sent. The antibiotics started at the other institution were continued.

On hospital day 2, blood and urine cultures remained negative, but respiratory cultures grew *Enterobacter cloacae*. The patient's condition began to improve and he was weaned off all pressor agents on day 4 of hospitalization. Results from the outside institution revealed growth of *Enterobacter cloacae* in all four bottles of 2 sets of blood cultures. Urine culture also grew *Enterobacter cloacae*. All the isolates of Enterobacter were susceptible to trimethoprim/sulfamethoxazole, gentamicin, tobramycin, aztreonam, ceftriaxone, ceftazidime, ciprofloxacin, ertapenem, imipenem, and meropenem. Antibiotics were deescalated to cefepime.

A computed tomography (CT) scan of the chest, done on day 4 of hospitalization to better characterize the pulmonary nodular opacities seen on chest X-ray, demonstrated disseminated, poorly marginated, cavitating and solid non-calcified pulmonary lesions. Bronchoscopy was performed that revealed thick mucous secretions, but no endobronchial lesions were seen. Bronchoalveolar lavage (BAL) specimens were sent to pathology and microbiology. To further elucidate the composition of the pulmonary nodules, a CT-guided biopsy of a right upper lobe nodule was performed. While cytology from BAL was negative for any malignancy, Gram stain of the aspirate from the biopsy showed Gram-negative rods, and cultures grew *Enterobacter cloacae*. A transesophageal echocardiogram was done that showed a layer of echodense material in the left atrium along the anterior and anterolateral mitral annulus. A definitive diagnosis of infective endocarditis was made. Cefepime was changed to meropenem and the patient received a total of 6 weeks of antimicrobial therapy. He was discharged on hospital day 19. A CT scan of the chest was repeated 3 weeks into treatment and revealed resolving pulmonary nodules.

Discussion

Enterobacter endocarditis, although rare, is an important entity that clinicians should bear in mind for a few reasons. Firstly, the increase in the number of invasive procedures such as the implantation of endovascular devices has resulted in a rise in non-HACEK Gram-negative endocarditis [1, 2]. ICE-PCS reported that implanted endovascular devices and healthcare contact were primary risk factors for non-HACEK Gram-negative endocarditis [1]. Intravenous drug abuse, once thought to be the main risk factor for this infection, is actually rare compared to the above-mentioned risk factors [1]. While our patient neither had an endovascular device nor any prior valvular pathology, the altered urinary tract anatomy predisposed him to urinary tract infection that resulted in bacteremia and endocarditis. Neobladder surgery has been shown to contribute to recurrent urinary tract infections [3]. Secondly, the in-hospital mortality rate of patients with non-HACEK Gram-negative endocarditis can be significant [1]. Finally, with rising Gram-negative resistance patterns, treatment options may be limited and expert opinion may be warranted to treat such serious infections [4].

We conducted a literature search on all previously reported cases of endocarditis caused by Enterobacter. Forty-three cases, including the present case, have been published in the medical literature so far. Table 1 outlines demographic parameters, risk factors such as underlying heart disease and co-morbid conditions, treatment modalities, and outcomes for all the cases listed individually. Table 2 summarizes key clinical information from all the cases.

With regards to treatment, surgery was performed in about 40% of the 43 cases. Surgical intervention has generally been considered as the mainstay of treatment for non-HACEK endocarditis because of the high mortality with medical management alone [5]. More recent observations from the ICE-PCS have shown that cardiac surgery does not lead to improved outcomes for non-HACEK

Table 1 Epidemiological and clinical information of 43 reported cases of Enterobacter endocarditis

Reference no.	Age (years), sex	Valve involved	Relevant past medical history	Surgery	Treatment	Outcome	Enterobacter species
[6]	28, F	MV	IV drug abuser	N	Streptomycin, sulfadiazine	S	Aerogenes
[7]	65, M	AV	Questionable rheumatic heart disease, prostatic hypertrophy, transurethral resection of prostrate, prostatic abscess	N	Penicillin, streptomycin	D	Aerogenes
[8]	22, F	MV	Ureteral calculus, cystoscopy with meatotomy	N	Penicillin, streptomycin, sulfadiazine, sulfathiazole, polymyxin B	D	Aerogenes
[9]	NR, NR	TV	Questionable valve trauma secondary to pulmonary hypertension, pulmonary arteriosclerosis	N	NR	D	Aerogenes
[10]	NR, NR	MV	Prosthetic valve	Y	Methicillin, chloramphenicol, erythromycin	D	Aerogenes
[11]	54, M	AV, MV	Prosthetic valve, UTI	N	Chloramphenicol, kanamycin, cephalothin	S	Cloacae
[12]	44, M	AV	Aortic stenosis and insufficiency	Y	Kanamycin, streptomycin, chloramphenicol	D	Cloacae
[13]	48, M	AV, MV	IV drug abuse	N	Kanamycin, gentamicin, chloramphenicol	D	Aerogenes
[14]	57, F	NR	None	N	Ampicillin, cloxacillin, kanamycin	D	Cloacae
[15]	3.5, M	NR	Rheumatic heart disease	N	NR	D	Aerogenes
[15]	8, F	NR	VSD	N	NR	S	Aerogenes
[16]	23, M	AV	Prosthetic valve	Y	Hydracillin	S	Aerogenes
[17]	32, F	AV, MV	Rheumatic heart disease, mitral annuloplasty	Y	Colistin, chloramphenicol	D	Cloacae
[18]	13, F	AV	Congenital aortic stenosis, underwent valvotomy prior to onset of symptoms	N	Carbenicillin, kanamycin	S	Cloacae
[19]	30, NR	AV, MV	IV drug abuse, rheumatic heart disease	N	Gentamicin, penicillin	S	Cloacae
[20]	52, M	TV	IV drug abuse	N	Chloramphenicol, streptomycin	S	Aerogenes
[20]	24, M	TV	IV drug abuse, pulmonary embolus	N	Nafcillin	S	Aerogenes
[21]	28, M	TV	IV drug abuse	Y	Cefanone, gentamicin	S	Cloacae
[22]	15, M	TV	Penetrating foreign body through tricuspid valve	Y	Gentamicin, carbenicillin	S	Cloacae
[23]	NR	MV	Prosthetic valve	Y	Gentamicin, carbenicillin	S	Cloacae
[24]	42, F	TV	None	N	Gentamicin, ampicillin	D	Cloacae
[25]	41, M	MV	Prosthetic valve	N	Tobramycin, gentamicin	D	Cloacae
[26]	20, F	NR	Patch closure of IVS	NR	Tobramycin, carbenicillin	S	Cloacae
[27]	34, M	AV	Congenital aortic stenosis	N	Gentamicin, dicloxacillin	D	Cloacae
[28]	60, F	PE	Pacemaker	Y	Ceftriaxone, gentamicin	S	Cloacae
[29]	49, F	MV	None	Y	Ceftizoxime, amikacin	S	Cloacae
[30]	76, NR	MV	Transvenous pacer, mitral regurgitation	Y	Imipenem	S	Aerogenes

Table 1 continued

Reference no.	Age (years), sex	Valve involved	Relevant past medical history	Surgery	Treatment	Outcome	Enterobacter species
[31]	25, M	TV	h/o *Staphylococcus aureus* tricuspid endocarditis	N	Ciprofloxacin, ceftriaxone	S	Agglomerans
[32]	50, M	AV, MV	Tooth extraction	N	Penicillin, gentamicin, amikacin	S	Agglomerans
[33]	57, F	MV	Prosthetic valve, rheumatoid arthritis, non-Hodgkin's lymphoma	N	Ceftazidime, gentamicin	S	Cloacae
[34]	72, M	NR	Prosthetic valve, wound infection	N	NR	D	Cloacae
[35]	54, M	Atrial septum	Orthotopic heart transplant	Y	Tobramycin, imipenem, ciprofloxacin	S	Cloacae
[36]	4, M	Shunt	Cyanotic heart disease s/p insertion of right Blalock-Taussig shunt	N	Netilmicin, ciprofloxacin	S	Cloacae
[37]	28, M	MV	Rheumatic heart disease	N	Ciprofloxacin, netilmicin	S	Cloacae
[38]	16 Days, NR	RA, LA	None	N	Gentamicin cefotaxime	D	Cloacae
[39]	76, M	MV	Rheumatic mitral stenosis requiring commissurotomy, prosthetic valve	Y	Ciprofloxacin	S	Cloacae
[40]	22, M	IVS	Complete correction of tetralogy of Fallot 16 years prior	Y	Ceftriaxone, gentamicin	S	Agglomerans
[41]	39, M	MV	Prosthetic mitral valve, rheumatic heart disease	Y	Cefotaxime, gentamicin, imipenem, vancomycin	D	Aerogenes
[42]	25, M	AV, MV	Mitral valve prolapse, mitral regurgitation, pulmonary hypertension	Y	Cefotaxime, aminoglycoside, vancomycin	S	Cloacae
[43]	7, M	RA	Central venous catheter one year prior	N	Meropenem, gentamicin	S	Cloacae
[44]	56, M	MV	Rheumatoid arthritis, mucosal abscess of lower lip	Y	Gentamicin, ciprofloxacin	S	Cloacae
[45]	40, NR	MV	Prosthetic valve	Y	Imipenem and ciprofloxacin	D	Cloacae
Present case	56, M	MV	Neobladder reconstruction with intermittent catheterization	N	Meropenem	S	Cloacae

NR not reported, *MV* mitral valve, *TV* tricuspid valve, *AV* aortic valve, *RA* right atrium, *LA* left atrium, *IVS* interventricular septum, *PE* pacemaker electrode, *S* survived, *D* died, *Y* yes, *N* no, *UTI* urinary tract infection, *VSD* ventricular septal defect, *h/o* history of, *s/p* status post

Gram-negative bacillus endocarditis [1]. On reviewing the overall mortality rates based on the cases of endocarditis secondary to Enterobacter, the mortality rate was 30% in those who underwent surgery compared to 44% in those who did not. Five of the 17 patients that underwent surgery for Enterobacter endocarditis unfortunately died. One of them had concomitant Staphylococcal and fungal endocarditis that was found postmortem [10]. One of the patients died due to perforation of a mycotic aneurysm [12]. Another patient died abruptly about 2 weeks postoperatively secondary to acute onset of cyanosis and hypotension [17]. Causes of death in 2 patients were acute renal failure and acute respiratory failure [41, 45]. No definite conclusion, however, can be drawn from this data, given that it reflects the observation of a small number of cases over 60 years with potential confounding factors such as selection bias for surgery, use of different antimicrobials, co-morbid conditions, and possible differences in medical care over this wide time span.

With regards to antimicrobial therapy, the usual recommendation is that combination therapy should be routinely utilized in treating non-HACEK Gram-negative endocarditis [5]. This is probably based on older studies, while the results of a more recent study conducted by Morpeth et al. [1] did not reveal a statistically significant benefit among patients who received combination therapy over those who were administered monotherapy. Our review results revealed that the majority of patients with Enterobacter endocarditis were treated with dual antibiotics (Table 1). The most common combination was a

Table 2 Clinical summary of all 43 cases of Enterobacter endocarditis

Clinical findings	Proportion (%)
Age: mean 37 years; range 16 days to 76 years	
Pediatric (<18 years old)	6/43 (13.95)
Adult	34/43 (79)
Not reported	3/43 (7)
Gender	
Male	25/43 (58.18)
Female	11/43 (25.58)
Not reported	7/43 (16.28)
Prior valvular/heart disease	
Prosthetic valve	11/43 (25.58)
Rheumatic heart disease	5/43 (11.63)
Valve trauma	2/43 (4.65)
Congenital heart disease (aortic stenosis, VSD, corrected tetralogy of Fallot, cyanotic heart disease	5/43 (11.63)
Aortic stenosis and insufficiency	1/43 (2.33)
Mitral valve prolapse with mitral regurgitation and pulmonary hypertension	1/43 (2.33)
Microbiology	
E. cloacae	26/43 (60.46)
E. aerogenes	12/43 (27.90)
E. agglomerans	3/43 (6.98)
Location of vegetations	
Mitral valve	20/43 (46.51)
Tricuspid valve	7/43 (16.28)
Aortic and mitral valve	6/43 (13.95)
Atrial walls	2/43 (4.65)
Interventricular septum	1/43 (2.33)
Pacemaker electrode	1/43 (2.33)
Unknown location	5/43 (11.63)
Surgery intervention	
Surgery performed	18/43 (41.86)
No surgery indicated	25/43 (58.14)
Prognosis after treatment completion	
Survived	27/43 (62.79)
Died	16/43 (37.21)

betalactam with an aminoglycoside. Studies in animals have shown that the addition of an aminoglycoside to betalactams produces a more rapid and extensive reduction in vegetation titers in *Enterobacter aerogenes* endocarditis compared to betalactam monotherapy [46]. The rationale behind employing dual therapy is synergism to amplify the rapidity and extent in kill-time and to prevent the emergence of resistance [4]. We chose monotherapy with a carbapenem for a few reasons. Firstly, based on recent data that did not support the routine use of combination therapy, we did not feel compelled to use more than one antibiotic. Secondly, we had data for susceptibilities to the isolate of Enterobacter and did not double-cover for possible resistance to one of the antibiotics. We switched from cefepime to meropenem to utilize the antimicrobial agent most stable against AmpC betalactamase that Enterobacter is known to produce [4]. This was done more as a caution because we did not have the information on production of this enzyme in our isolate. Finally, monotherapy with carbapenem has yielded favorable results in another case of Enterobacter endocarditis [30].

To summarize, Enterobacter is a rare cause of endocarditis, but clinicians should consider this as a possibility in the right clinical setting. Surgery should not be the default management option for all cases of Gram-negative endocarditis because medical management per se is a viable modality. One should be cognizant of the high rates of mortality with this condition.

Conflict of interest None.

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