Acinetobacter endocarditis: a rare nosocomial infection of native heart valves

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ABSTRACT

Acinetobacter baumannii is an opportunistic bacterial pathogen predominantly associated with hospital-acquired infections. Here we present a case of infective endocarditis of native Mitral and Aorta valves caused by A. baumannii in a 73-year-old man. He underwent surgical excision and Pathologic specimen showed A. baumannii growth after 48 hours that was extensively drug-resistant (XDR). He was treated with colistin and tigecycline. Finally, he discharged with no important complication. To our best knowledge, it is the first case of Acinetobacter endocarditis has ever been reported in Iran. Although XDR A. baumannii is a life-threatening pathogen, proper and timely treatment can be life-saving.

Keywords: Acinetobacter baumannii; Endocarditis; Heart valve; Nosocomial infection; Multidrug resistance

INTRODUCTION

Acinetobacter baumannii is an opportunistic bacterial pathogen predominantly associated with hospital-acquired infections and potential for substantial antimicrobial resistance (1). It is widely distributed in the environment and in the hospital setting such as on environmental surfaces, the health care workers hands, mechanical ventilators and dialysis machines. Most common infections include pneumonia, bacte- remia, meningitis, skin, soft tissue and urinary tract infection (2).

Reports of Acinetobacter endocarditis are very rare and most cases have been noted to involve prosthetic valve. It occurs mainly in hospitalized patients with predisposing factors (3). Native valve endocarditis caused by Acinetobacter is acute and aggressive and is more likely to be fatal than the prosthetic valve involvement (4). Increasing resistance to antibiotics, makes this infection difficult to treat and associated with higher mortality (3).

To our best knowledge, it is the first case of Acinetobacter endocarditis has ever been reported in Iran.

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CASE PRESENTATION

A 73-year-old man admitted to Erfan hospital, Tehran, Iran, with fever, generalized weakness and dyspnea. He had history of cholecystectomy due to cholelithiasis 56 days ago. Following surgery, he gradually developed shortness of breath but he did not seek care. Continued dyspnea and addition of fever, took him to the emergency room. He had history of hypertension and no history of addiction or diabetes mellitus. At presentation, temperature was 38°C, respiratory rate 18 breaths per minute, heart rate 84 beat per minute, blood pressure 130/70 mmHg and oxygen saturation 94%. Heart auscultation indicated early systolic and diastolic murmur. Laboratory test showed a total leukocyte count of 8700 with 90% polymorphs and anemia (Hb 7.5 g/dl). His erythrocyte sedimentation rate was 19 mm/h and his serum level of C-reactive protein was 29 mg/dl. Blood urea, serum creatinin, sodium and potassium were in normal ranges. His chest CT scan demonstrated mild plural effusion in both lungs. In transthoracic echocardiography he had moderate MR and moderate TR with a mobile calcified mass on aorta valve. Transesophageal echocardiography indicated Mitral valve thickening with severe regurgitation and perforation of anterior leaflet; Aortic valve thickening with severe insufficiency and perforation of non-coronary cusp (NCC); mobile mass measuring about 6x7 mm on NCC and mild pericardial effusion posterior to right atrium (Fig. 1). Three sample of blood culture by hourly intervals was negative. After 2 weeks of treatment with vancomycin, ampicillin/sulbactam and gentamicin, there was no improvement in the patient. So, he underwent surgical excision and valves replacement that revealed vegetation on aorta and mitral valves. Pathologic specimen showed severe neutrophilic infiltration with areas of necrosis, granulation tissue, fibrous deposition and bacterial colonization (Fig. 2). Tissue sample was also placed in sterile screw-cap container and drops of sterile saline were added to keep it moist. It was transported to the microbiological laboratory in 15 minutes. The sample was cultured on MacConkey agar and chocolate blood agar and after 48 hours Gram-stained smear showed Gram-negative pleomorphic cocobacilli which grew as non lactose fermenting colonies. The organism was oxidase negative, catalase positive and non mobile. The isolate was identified as Acinetobacter baumannii complex based on glucose oxidation and Triple Sugar Iron (TSI) test.

Fig. 1. Transesophageal echocardiography shows 6 × 7 mm vegetation (white circle) on the NCC

Fig. 2. Histopathological section of endocardial tissue demonstrating neutrophilic infiltration (yellow arrow) and Acinetobacter colonization (green arrow) in hematoxylin and eosin staining (magnification 400)

Antimicrobial susceptibility testing was performed using the Kirby-Bauer disc diffusion method as per clinical and laboratory standard institute recommendation and based on that, the organism was XDR and was only sensitive to colistin. According to the antibiotic susceptibility pattern, medical treatment of the patient changed to intra-venous colistin and tigecycline. Finally, he discharged 42 days after operation and antibiotic therapy with no important complication and no vegetation on follow up echocardiography.

DISCUSSION

Acinetobacter baumannii has a high incidence among immunocompromised patients, in particular,
patients with a history of prolonged (>90 days) hospital stay (5). Other risk factors for developing Acinetobacter infections include immune suppression, older age, presence of co-morbidity, major trauma, invasive procedures, previous antibiotic use and presence of indwelling catheters or mechanical ventilation (6). In the case of endocarditis with rare organisms, several case reports have been published, like a case of IE due to Faclamia hominis in an adult patient with rheumatic mitral stenosis (7) or a rare case of Corynebacterium striatum endocarditis on a bioprosthetic aortic valve (8). There are few reports of endocarditis due to A. baumannii. In most of these reports, underlying heart disease as a risk factor has been reported and is often seen in patients with prosthetic valve or heart valve disorder, as reported by Qian Chen in 2015, Mauro Sturiale in 2016 and S Senthil Kumar in 2008 (9-11). Our patient had no history of heart disorder but he had a history of cholecystectomy and hospitalization 56 days before.

Despite advances in the treatment of infective endocarditis, it remains challenging (3). In recent years, MDR pathogens have increasingly become a serious global concern with regard to both community-acquired and nosocomial infections. A. baumannii has been designated as a “red alert” pathogen, due to its extensive antibiotic resistance spectrum; making selection of an appropriate empirical treatment difficult, so antibiotic therapy should be performed following antimicrobial susceptibility testing. Nevertheless, delay in beginning correct treatment may have adverse effects on patient health (6). In most cases of reported Acinetobacter endocarditis, the pathogen was MDR (9, 10) and treatment was performed based on antibiotic susceptibility testing, but in the case of Acinetobacter endocarditis reported by S. Senthil Kumar in 2008 in India, the isolate was found to be sensitive to a majority of the antibiotics tested (11). Differences in antibiotic susceptibility patterns may be explained by differences in time and place where the studies were performed.

Yoshinori Sato Showed tigecycline killed MDR Acinetobacter more effectively than colistin, although colistin and tigecycline are both considered effective against MDR Acinetobacter (12). These results are consistent with the results reported by Khoshrood S in southwestern Iran who reported 87% and 12.8% of Acinetobacter isolates were MDR and XDR, respectively. Colistin and tigecycline with 2.8% and 45.7% resistance rates were the most effective antibiotics (13). Fereshteh Ezadi reported 38% and 61.9% of Acinetobacter isolates were carbapenem-susceptible and carbapenem-resistant, respectively. In addition, 94.36% of isolates were susceptible to colistin (14). Our patient had negative blood culture results, so initially he was treated with vancomycin, ampicillin/sulbactam and gentamicin but after the culture of tissue specimen was positive for XDR A. baumannii, according to the result of antimicrobial susceptibility testing, treatment was changed to a combination of colistin and tigecycline.

Due to the poor prognosis of critically ill patients who acquire Acinetobacter infections, attribution to a definite mortality rate is difficult; however the mortality rates have ranged 23-68% (15). Fortunately, our patient recovered after surgical and medical treatment and discharged with no important complication.

CONCLUSION

To our best knowledge, it is the first case of Acinetobacter endocarditis has ever been reported in Iran. Although MDR or XDR Acinetobacter baumannii is a life-threatening pathogen, especially in nosocomial setting and is a great concern, proper and timely treatment can be life-saving. In addition, more effective strategies and surveillance must be used to prevent the dissemination of Acinetobacter baumannii in the hospital.

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ACINETOBACTER ENDOCARDITIS

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