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CASE REPORT

Gram-negative bacteria causing infective endocarditis: Rare cardiac complication after liver transplantation

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Abstract

Bacterial endocarditis is a rare complication amongst solid organ transplant recipients and is often linked to bacteremia. Majority of these recipients do not have underlying valvular heart disease or congenital valvular abnormalities. *Staphylococoocusaureus* and *Enterococcus* species are the most commonly isolated organisms. There are very few reports of gram-negative bacteria causing endocarditis in liver transplant recipients. We report a 51-year-old male, a liver transplant recipient, who developed bacterial endocarditis of the mitral valve due to extended spectrum of betalactamase producing strain of *Escherichia coli* and was managed successfully with antibiotics.

Key words: Bacteria; Infective endocarditis; Liver transplantation

Core tip: A pre-transplant cardiac assessment should include a careful evaluation for underlying valvular pathology. Bacterial endocarditis can however still occur in liver transplant recipients with normal cardiac valves. Gram negative bacteria though rare can be a causative agent for infective endocarditis. High index of suspicion for bacterial endocarditis is essential when investigating transplant recipients for fever of uncertain origin.

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INTRODUCTION

Infective endocarditis is a rare complication affecting solid-organ transplant recipients. Common isolates include *Staphylococoocusaureus* (*S. aureus*), *Enterococci* and *Aspergillus*. Gram negative bacilli causing bacterial endocarditis of mitral valve has hitherto not been reported. We report bacterial endocarditis of the mitral valve due to extended spectrum of betalactamase (ESBL) producing strain of *Escherichia coli* (*E. coli*) following a deceased donor liver transplantation.

CASE REPORT

A 51-year-old male, a hypertensive, underwent a deceased donor liver transplant (LT) for ethanol induced end-stage

liver disease. Pre-transplant cardiac work up included an echocardiogram (ECHO), adenosine stress SPECT study and coronary angiogram. ECHO showed a mild dilation of the left atrium, normal left ventricular systolic function with an ejection fraction of 65%. There was annular calcification of the mitral valve with extension into the posterior leaflet and a mild regurgitation. The aortic valve was sclerotic. Pulmonary artery systolic pressure was 40 mmHg with mild tricuspid regurgitation. Adenosine stress SPECT study revealed a small area of inducible ischemia in the left anterior descending artery but the coronary angiogram showed no significant disease in the epicardiac arteries. Right heart study showed normal pulmonary artery pressures.

Three weeks prior to LT, the patient had fever. Urine and blood cultures grew Escherichia coli and the patient was treated with appropriate antibiotics for 2 wk. Subsequent cultures of both blood and urine prior to LT were sterile. Donor and recipient cultures on the day of transplant were also sterile. Post-operative period was uneventful and he was discharged with good graft function after 3 wk. One month later, the patient attended the liver clinic with high-grade fever and chills. On examination, he was haemodynamically stable, conscious, oriented but febrile (temperature 39 degree Celsius). Cardiovascular system examination revealed a grade 3/6 pansystolic murmur at the mitral area. Other organ system examination was normal.

Investigations revealed hemoglobin of 7.2 gm/dL, total white cell count of 14400 cell/mm^3 with predominant neutrophils (86%), elevated ESR (92 mm/h) and C reactive protein of 289 mg/L. Serum creatinine was 1.8 mg/dL. Urine examination showed proteinuria and plenty of red blood cells. Blood and urine cultures were positive for ESBL producing strain of *E. coli*, which was sensitive to Meropenem, Tigecycline and Amikacin. ECHO showed multiple echogenic mobile masses on the mitral valve leaflet especially the posterior leaflet with moderate to severe grade of mitral regurgitation.

A diagnosis of infective endocarditis of the mitral valve due to ESBL *E. coli* was made. Patient was treated with meropenem and tigecycline for 6 wk though cultures were sterile a week after initiation of antibiotics. Follow-up ECHO at 8 wk showed mild mitral regurgitation with vegetations on the valve. Repeat ECHO after 48 wk showed mild mitral regurgitation without vegetations on mitral valve.

DISCUSSION

Bacterial infections are common in the post LT period and occur in 33%-68% of LT recipient[1]. In a recent study from our centre, *Klebsiella* and *E. coli* were the two common organisms responsible for post LT infection[2]. Common sources were the respiratory tract, urinary tract and blood stream infection[2].

Infective endocarditis after LT is rare. The prevalence of bacterial endocarditis among liver transplant recipients has been reported to be around 1.7%. Unlike in general population, bacterial endocarditis in transplant recipients can occur even in normal cardiac valves. The common causative agents for valvular endocarditis and mural endocarditis are *S.aureus* and *Aspergillus* respectively[3]. Other uncommon bacterial organisms causing infective endocarditits are *Enteroccocusfecalis*, ESBL producing *Klebsiellaterrigena* and *Propionibacterium acnes*[4,5]. *E.coli* causing infective endocarditis has been reported in pulmonary valve[6]. To our knowledge, this is the first report of ESBL producing *E. coli* induced mitral valve endocarditis.

Bacteria producing ESBL enzymes are resistant to most betalactam antibiotics such as penicillin and cephalosporins. ESBL is found exclusively in Gram negative organisms such as *Klebsiellapneumoniae*, *Klebsiellaoxytoca*, *E. coli* species and *Acinetobacterburkholderia*. Risk factors for infection with ESBL producing organisms include prolonged hospital or intensive care unit stay, prolonged mechanical ventilation, central venous or arterial catheters, bladder catheter, emergency abdominal surgery and prolonged exposure to antibiotics.

In conclusion, a high index of suspicion for endocarditis is essential, especially in the setting of new auscultation findings at cardiac valve areas. Multiple blood cultures are necessary to make the correct diagnosis. Initiating empirical broad-spectrum antibiotics at the earliest prior to getting culture sensitivity is important in LT recipients.

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