Infective endocarditis caused by K. oxytoca: A Case Report

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Key Clinical Message:

Infective endocarditis caused by Klebsiella species is rare, with K. oxytoca contributing to only a tiny fraction of cases. This case highlights the role of advanced imaging and molecular techniques in the diagnosing and managing it.

KEYWORDS: Gram-negative bacteria; heart valves; infective endocarditis; *K. oxytoca* , septicemia.

Introduction:

Infective endocarditis (IE) caused by *Klebsiella* species is an infrequent occurrence, comprising less than 1.2% of native valve endocarditis cases and up to 4.1% of prosthetic valve endocarditis cases. [1,2] The majority of such cases are caused by*K. pneumoniae.* [3,4] *K. oxytoca* is an extremely rare species that can rarely cause endocarditis. Here, we present a case of *K. oxytoca* endocarditis to highlight the need for careful evaluation during the entire course of therapy and the role of advanced imaging and molecular techniques.

Case presentation:

A 77-year-old Caucasian male with no known past medical history except for benign prostatic hypertrophy presented to the emergency department with a one-week history of acute onset of progressive dyspnea and generalized swelling. Approximately three weeks prior to this emergency room visit, he had been admitted to the intensive care unit in our hospital for an episode of septic shock secondary to *K. oxytoca*bacteremia. 1 out of 2 sets of blood cultures were positive for *K. oxytoca* . He was then diagnosed and treated for two weeks for a complicated urinary tract infection. He was initially treated with intravenous ceftriaxone but later switched to oral levofloxacin given susceptibilities as reported below in **figure 1a.**

On initial evaluation in the emergency department, the patient appeared in mild distress. He was placed on a low flow nasal cannula, with oxygen saturation of 94 percent on 2 liters of supplemental oxygen. Initial vital signs were recorded as follows: Blood pressure was 116/75 mm Hg, pulse 96 beats per minute, axillary temperature 98.7 Fahrenheit and respiration 24 per minute. Physical examination was remarkable for bibasilar crackles on chest auscultation and grade 2+ pitting peripheral edema in bilateral lower extremities. He also had a grade 3 systolic murmur best heard in the 2nd left intercostal space, loudest on expiration. Otherwise, he was awake, alert and oriented to time, place and person. No Janeway lesions, no splinter hemorrhages, no Osler's nodes were noted on the rest of the clinical exam.

His laboratory studies were remarkable for leukocytosis, white blood cell count on admission was 15.6 x 10^3 cells/uL. (Reference range 4.4-11.0 x10^3 /uL). His aspartate transaminase was 78 Unit/L (reference range: 6-42 U/L) and alanine transaminase was 60 Unit/L (reference range: 0-55 U/L). C-reactive protein was 2.5 mg/dL (reference range: Less than 0.3mg/dL). Chest radiograph showed bilateral vascular congestion,

bilateral pleural effusions and enlarged cardiac silhouette as depicted in **Figure 1b** . Multiple sets of blood cultures were negative. *Bartonella* ,*Brucella* and *Coxiella* specific antibodies were negative.

Figure 1a: Blood culture report and susceptibility profile from patient's recent hospitalization, which isolated K. oxytoca

Figure 1c: The picture in upper row showing pseudoaneurysm of aortic root and the picture in the bottom revealed the pres

During hospitalization, he continued to have persistently elevated white blood cell count, with no clear explanation on workup. He was needing aggressive diuresis for his clinical picture of cardiogenic shock and associated fluid overload. Multiple sets of blood cultures remained negative after more than 72 hours of incubation. Computed tomography of abdomen and pelvis scan revealed no abscess or foci of infection.

He was started on empiric intravenous ceftriaxone given his recent history of *K. oxytoca* septicemia. Echocardiograms via both transthoracic and transesophageal approaches were pursued which showed preserved left ventricular ejection fraction however was concerning for native aortic valve endocarditis with severely thickened aortic valve and a pseudoaneurysm of aortic root.

Cardiothoracic surgery was consulted given findings of valvular endocarditis with symptoms and signs of left ventricular failure. He subsequently underwent surgical debridement of aortic annulus and left ventricular outflow tract with aortic valve replacement. Intraoperatively, chronic vegetation at the base of the right coronary cusp with fibrosis of the left ventricular outflow tract was found. Debrided valve tissue was sent for bacterial, acid-fast bacilli (AFB) test, fungal cultures and direct DNA sequencing by PCR. Gram stain and routine bacterial, mycobacterial and fungal cultures of the operative specimens were negative. However, *K. oxytoca* specific DNA was detected on the ribosomal DNA amplification by polymerase chain reaction of diseased valve/tissue.

The patient clinically improved after a few days' stay in the cardiac care unit post-surgery and was continued on intravenous ceftriaxone. He was able to be discharged home after eleven days of hospitalization and was continued on intravenous antibiotics via a peripherally inserted central catheter to complete a four-week course. On a post-hospital discharge visit to the cardiology clinic, he clinically improved and continued to participate in physical therapy sessions.

Discussion:

K. oxytoca endocarditis is an uncommon entity that can infrequently cause endocarditis and is associated with poor outcome. In comparison to more virulent organisms like *Staphylococci* , gram-negative bacteria have a lower tendency to infect native heart valves due to their reduced ability to adhere to the endocardium.[4] However, *Klebsiella* species endocarditis are responsible for high rates of complications and mortality. [2] We report a case of *K. oxytoca* endocarditis in an elderly man who presented with heart failure and severe aortic stenosis secondary to infective endocarditis.

K. oxytoca bacteremia is commonly seen in the setting of hepatobiliary tract, urinary tract, skin and soft tissue, and peritoneal infection. [2,5] The documented pathways through which K. oxytoca enters the bloodstream in cases of bacteremia are ranked in descending order as follows: the hepatobiliary tract (50%-55%), intravascular or urinary catheters (7%-16%), the urinary tract (5%-6%), skin and soft tissues (3%-5%), and the peritoneal cavity (2%-6%). Moreover, in 23% to 34% of infections, the specific entry points remain unidentified.[6,7]Although our patient didn't have any history of immunosuppression, he had other risk factors for developing gram-negative infective endocarditis including his advanced age and history of recent prolonged hospitalization for septic shock secondary to *K. oxytoca*bacteremia of urinary source.

It is well known that echocardiography is the mainstay of cardiac imaging for diagnosis of infective endocarditis.[8]Transesophageal echocardiography (TEE) is a more sensitive test compared to transthoracic echocardiogram (TTE) which is generally the first diagnostic test for patients with suspected infective endocarditis. In addition, TEE is superior to TTE for detection of cardiac complications including abscess, leaflet

perforation, and aortic pseudoaneurysm or intracardiac fistula. [9,10] As in our patient, early complications were better visualized on TEE, and the surgical intervention followed soon after. Given the high morbidity and mortality rates associated with these infections, timely surgical consultation is of paramount importance. [11]

Similarly, microbiological diagnosis of infective endocarditis is primarily based on blood culture, excised cardiac valve tissue, or infected emboli. This conventional approach has been shown to be successful in 92 to 95% of cases in which a microorganism is present.[11] In regard to our patient, a chronic vegetation at the base of the right coronary cusp with fibrosis of the left ventricular outflow tract was found on histopathological analysis confirming the diagnosis of infective endocarditis as per modified Duke's criteria. Although, the conventional microbiological analysis of cardiac valve tissue was unremarkable, *K. oxytoca* specific DNA was detected on a broad-range polymerase chain reaction technique of affected valve/tissue.

The patient in our case report had no history of intravenous drug use, nor he had any exposure to animal farms, or any active exposure to pet animals. Culture negative endocarditis due to fastidious organisms was initially considered among the differential diagnoses, however serological titer tests and automated testing of blood cultures, in addition to the use of specialized culture media (enriched broth) were unremarkable. This also highlights the fact that molecular based techniques could potentially be helpful for the identification and analysis of this life-threatening infection.

The most effective drugs against these microorganisms include third generation cephalosporins and aminoglycosides, which are generally administered together. While the ideal period for treatment is not clearly established, a recommended duration of around 6 weeks is advisable. Additional antibiotics like imipenem, aztreonam, and fluoroquinolones have also shown effectiveness against gram-negative bacteria. [12] Our patient showed clinical improvement with a four-week course of intravenous ceftriaxone.

Conclusion

This case report highlights the importance of careful evaluation and aggressive treatment of *K. oxytoca* endocarditis due to its severe complications, overall poor prognosis, and associated high mortality rate. Similarly, molecular-based techniques including universal PCR techniques can be a useful tool in certain scenarios when the standard microbiological analysis is unrevealing.

Declaration of Conflicting Interests

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CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest.

ETHICS STATEMENT

None

WRITTEN CONSENT FROM THE PATIENT

Written informed consent was obtained from the patient before the submission of the report. The patient understands that his name and initials won't be mentioned and only the images of diagnostics tests will be used.

DETAILED AUTHORS' CONTRIBUTIONS

Suju J, Sajan N collected the required case information, images, reports, and contributed to writing the original drafts of the manuscript. Sujan J contributed to conceptualize and describe the case. Suju J, Sajan N and Sujan J reviewed the literature and contributed to writing and editing the manuscript. All authors read and approved the final manuscript.

DATA AVAILABILITY STATEMENT

The data supporting the findings in this case report is available within the article and its supplementary materials.

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