A case of Ochrobactrum anthropi-induced septic shock and infective endocarditis

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ABSTRACT

Ochrobactrum anthropi is a gram-negative rod of low virulence. Infections due to this organism are uncommon; however in immunocompromised hosts it can cause severe infections. Among the many infections it can cause, infective endocarditis is very rare. Even rarer is infective endocarditis of the native valves, as Ochrobactrum antropi affects damaged or prosthetic valves almost exclusively. This case describes native valve endocarditis due to Ochrobactrum anthropi.

KEYWORDS: infective endocarditis, sepsis, Ochrobactrum anthropi

CASE REPORT

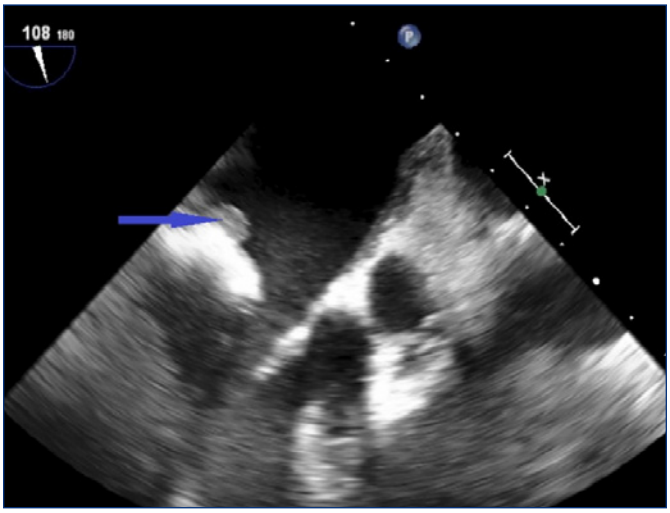
A 58-year-old woman with a past history of atrial fibrillation not on anticoagulation, end- stage renal disease with a failed kidney transplant, now on dialysis, coronary artery disease, moderate aortic stenosis, and pacemaker for 3rd degree heart block came in with chest pain for 2 days. She described the pain as substernal, sharp, non-radiating and 8/10 in intensity. She denied any difficulty breathing, fevers, chills or palpitations. She was a smoker in the past, and denied drug use.

Her vitals were: Temperature 98.1 degree Fahrenheit, heart rate 90/min, blood pressure 128/61 mmHg, respiratory rate of 16/min and oxygen saturation was 100% on room air. On examination she had a regular heart rate and a 2/6 ejection systolic murmur was heard in the aortic area, consistent with her history of aortic stenosis. No other murmurs were heard, and her lungs were clear. The rest of the examination was remarkable for a dialysis catheter in internal jugular vein and a scar at the site of the kidney transplant. Laboratory work-up showed hemoglobin of 8.1mg/dl which was her baseline. Her white count was not elevated, creatinine was 6.7mg/dl, and electrolytes were within normal range. The 1st troponin was negative. Electrocardiogram (EKG) showed ventricle-paced rhythm, Sgarbossa score was 0. The chest X-ray showed cardiomegaly which was stable compared to previous X-ray. Her pain responded to nitroglycerine and she was admitted to rule out acute coronary syndrome and to have an ischemia work-up after consultation with her cardiologist. Her home dose of aspirin, atorvastatin, metoprolol, and clopidogrel were continued.

She was monitored on telemetry without any significant events. Her chest pain remained under control. The 2nd troponin was negative, however 3rd was very mildly elevated at 0.034 ng/ml. A stress test was scheduled for the next day after an inpatient cardiology consult. However, the next day around midnight she became confused and hypotensive, with a blood pressure of 60/40 mm Hg. The heart rate at that time was 120/min, and she remained afebrile. She was initially given normal saline and then albumin, but there was minimal improvement of the blood pressure. She was also noted to have a fever of 103 Fahrenheit on a repeat set of vital signs. Examination at that time did not reveal anything new: there was no obvious skin infection, her dialysis catheter site was clean, and no new murmurs were heard. Work-up at that time included complete blood count, troponin and blood culture drawn from periphery and her dialysis catheter. EKG was unchanged from previous. She was treated empirically for hospital-acquired infection with vancomycin and piperacillin/tazobactum. Due to persistently low blood pressure she was started on phenylephrine drip. Her blood pressure responded to phenylephrine, and her mental status also improved very quickly. Within 24 hours she was weaned off the phenylephrine drip. Her blood pressure remained stable, and she did not have a fever again. Her troponin continued to trend up and peaked at 0.5ng/ml. In the absence of chest pain and EKG changes, this was considered demand ischemia due to septic shock. Her white cell count was never elevated throughout this process.

The blood culture from her dialysis catheter and periphery grew Ochrobactrum anthropi sensitive to Meropenam and resistant to Beta Lactams. Subsequently, Meropenam was started and previous antibiotics were discontinued. The dialysis catheter was also replaced, the tip of original catheter was found to have a thick biofilm. The tip was also sent for culture, and it also grew the same organism. Repeat blood cultures remained negative. Considering the fact that she had a pacemaker, a transthoracic echocardiogram was performed, which did not show any vegetation. A transesophageal echocardiogram was then done, which showed 1 cm vegetation on Mitral Valve (**Figure 1**). She subsequently had her pacemaker leads removed, given the presence of vegetation, and semi-permanent epicardial pacemaker leads were placed. She was treated with antibiotics for 6 weeks without any further complications.

Figure 1. Transesophageal echocardiogram showing vegetation, marked with blue arrow. The vegetation is about 1cm x 0.7cm in size, and adherent to mitral valve annulus.



DISCUSSION

Ochrobactrum anthropi is an aerobic gram negative rod of low virulence which is widely distributed in aquatic environment. In many ways it bears resemblance to Pseudomonas. Due to its low virulence, Ochrobactrum anthropi rarely affects immunocompetent hosts, but can cause a variety of infections in immunocompromised hosts. It is thought to be associated with central lines, catheters and other foreign objects. [1,2] It has binding abilities similar to those of Staphylococcus epidermidis and Staphylococcus aureus,[3] which would explain its association with central lines and other foreign bodies. In our case the internal jugular dialysis catheter was the most likely portal of entry. Transplant-related infections due to contaminated pharmaceuticals have also been reported.[4]

Even though Ochrobactrum anthropi has been reported to be a cause of various infections more frequently over the last 20 years, it still remains a very rare cause of endocarditis. In almost all the reported cases of endocarditis, the affected individuals had a prosthetic valve or rheumatic valve disease.[4-7] This case though, describes undamaged native valve endocarditis. To our knowledge, only 1 case of undamaged native valve endocarditis has been reported previously.[8]

Ochrobactrum anthropi is resistant to most beta lactams, and usually needs treatment with carbapeman, ciprofloxacin, trimethoprim/sulfamethoxazole or gentamycin. [8,9] The resistant to beta lactams is due to production of AmpC β-lactamase by the organism.[10] This β-lactamase is chromosomal, inducible, and is resistant to inhibition by clavulanic acid.[11]

Even though it is considered to have low virulence, Ochrobactrum anthropi can cause severe infections in immunocompromised hosts. Therefore it is important to consider this bacterium in immunocompromised hosts who deteriorate suddenly. Similarly once the organism is identified, antibiotics should be switched to carbapeman, ciprofloxacin, trimethoprim/sulfamethoxazole, or gentamycin to cover it appropriately, even before sensitivities are available. It is uncertain whether every patient with positive blood culture for Ochrobactrum anthropi should be screened for endocarditis, but patients who have any hardware in their heart (prosthetic valves, pacemakers, defibrillators), which was true in our case, should get a transthoracic echocardiogram, and even a transesophageal echocardiogram to rule out infective endocarditis. As described in our case above, transthoracic echocardiogram can miss vegetation, which can result in a shorter duration and insufficient treatment of the endocarditis.

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Disclosures

None

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