Candida lusitaniae: An Uncommon Cause of Prosthetic Valve Endocarditis

*Candida lusitaniae,* first described as a human pathogen in 1979 [1], usually affects granulocytopenic patients [2]. Prosthetic valve endocarditis (PVE) has not been described previously. We describe a case of PVE due to *C. lusitaniae* following aortic valve replacement (AVR).

A 41-year-old woman with congenital aortic stenosis who had undergone a commissurotomy during childhood and two subsequent AVRs presented with refractory congestive heart failure (CHF) due to an ascending aortic aneurysm. She underwent her third AVR with a Medtronic Hall tilting disk valve (Medtronic, Inc., Minneapolis) an aortic conduit, and reimplantation of the coronary arteries on 21 February 1994. She continued to receive warfarin for anticoagulation; a permanent pacemaker was placed postoperatively.

Three weeks later she developed CHF, and blood cultures yielded *C. lusitaniae* (BACTEC NR 730 System, Becton Dickinson, Sparks, MD). MICs were obtained using a broth microdilution method [3] (table 1). Initially, the patient had no peripheral stigmata suggestive of endocarditis. Later, however, she developed a few subungual splinter hemorrhages. A transthoracic echocardiogram (TTE) and a transesophageal echocardiogram (TEE) showed marked left atrial enlargement, left ventricular dilatation, and mitral regurgitation, but no vegetations. Results of a gallium scan were normal. HIV serology was negative. No other diagnosis likely to result in candidemia was found.

A treatment regimen including amphotericin B, 0.6 mg/kg daily, and 5-fluorocytosine, 1.5 g orally every 6 hours, was instituted. Peak and trough levels for 5-fluorocytosine were 71 μg/mL and 69 μg/mL, respectively. A total of 1.4 g of amphotericin B was administered over 5 weeks before central venous access was no longer available. Fluconazole, 200 mg b.i.d., was given in addition to the 5-fluorocytosine; therapy was complicated by right-ankle hemarthrosis due to concomitant treatment with warfarin. In addition, the patient had diarrhea, which resolved when therapy with 5-fluorocytosine was discontinued. She was not willing to undergo another AVR, but she received fluconazole for 4 months.

One year after the third AVR and 7 months after completing antifungal treatment, the patient developed fever and flu-like symptoms. Blood cultures again yielded *C. lusitaniae,* and treatment with amphotericin B was resumed. A TEE did not demonstrate any vegetations. The patient was again reluctant to undergo an AVR, and she resumed treatment with amphotericin B, 1.2 mg/kg three times per week; she received 1.5 g of amphotericin B over the next 3 months. Her fungemia did not resolve, and a repeated TEE now suggested a vegetation. On 9 June 1995, she underwent her fourth AVR with a St. Jude prosthetic valve and replacement of the aortic conduit. A diffuse vegetation was found, extending inferiorly into the ventricular cavity and superiorly into the aorta; cultures yielded *C. lusitaniae.* Treatment with amphotericin B was continued and treatment with 5-fluorocytosine was resumed. The serum fungistatic titer was >1:1,024, and the fungicidal titer was 1:16. MICs and minimum fungicidal concentrations (MFCs) were obtained for the isolate recovered from her valve (table 1).

The patient was discharged 1 month postoperatively, and she continued to receive oral fluconazole. She was readmitted 6 weeks later with refractory CHF. A TEE obtained on readmission showed generalized left ventricular dysfunction but no vegetation on the prosthetic valves or native valves. Blood cultures remained negative. Right heart catheterization suggested biventricular failure due to her successive surgeries, despite adequate valvular function. The patient elected not to pursue heart transplantation and died 18 months after her third AVR; permission was not granted for a postmortem examination.

Table 1. MICs and minimum fungicidal concentrations for *C. lusitaniae* isolates over treatment time.

<table>
<thead>
<tr>
<th>Agent</th>
<th>Parameter, value (μg/mL)</th>
<th>Interpretive criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>March 1994</td>
<td>June 1994</td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>MIC, ≤0.05</td>
<td>MIC, 0.8</td>
</tr>
<tr>
<td></td>
<td>MFC, ND</td>
<td>MFC, 1.6</td>
</tr>
<tr>
<td>5-fluorocytosine</td>
<td>MIC, ≤0.2</td>
<td>MIC, 0.4</td>
</tr>
<tr>
<td></td>
<td>MFC, ND</td>
<td>MFC, 6.25</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>MIC, 0.4</td>
<td>MIC, 0.2</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>MIC, 3.1</td>
<td>MIC, ND</td>
</tr>
</tbody>
</table>

NOTE. Values presented in μg/mL. Susceptibilities of blood culture isolate and the isolate from a resected valve, performed by the same facility (Microbiology Reference Laboratory, Cypress, CA) using the same methods. MFC = minimum fungicidal concentration. ND = not done.
PVE caused by Candida species is associated with a 5-year survival rate of only 50% [4, 5]. Organisms commonly involved include Candida albicans and Candida parapsilosis. Historically, medical treatment with amphotericin B and 5-fluorocytosine has been unsuccessful. Successful treatment of C. lusitaniae PVE, like other forms of fungal PVE, probably requires replacement of the infected valve and prolonged suppressive antifungal therapy. The etiology of our patient’s C. lusitaniae PVE is presumed to be perioperative, with the infection occurring during either the third AVR or the pacemaker placement. The infection failed to resolve despite prolonged treatment with amphotericin B and 5-fluorocytosine, relapsing 7 months later. After the fourth AVR, the fungemia resolved; however, the patient was left with fatal biventricular failure. Unfortunately, AVR in the presence of left ventricular dysfunction is associated with significant morbidity and mortality. C. lusitaniae has developed resistance to amphotericin B during therapy [6, 7]. Although the MICs of amphotericin B and 5-fluorocytosine for the organism isolated from the infected valve were higher than those for the original blood isolate (table 1), fungicidal titers were readily achieved using these agents after the infected valve was replaced.

Two outbreaks of PVE due to C. parapsilosis following cardiac surgery have been described that were associated with high mortality rates [8, 9]. We found no evidence of an outbreak of C. lusitaniae infection at our hospital. At the time of our patient’s illness, the microbiology laboratory routinely identified yeast isolates from blood, urine, sputum, and normally sterile sites to the species level. During the period from 1994 to 1995, the only C. lusitaniae isolates among over 2,000 isolates identified to the species level were from our patient (P. Leist, personal communication).

Several TTEs and TEEs did not show evidence of PVE despite prolonged fungemia. The fourth AVR revealed a large vegetation extending to the left ventricular outflow tract and aorta; this was not evident on the TEE. Although transesophageal echocardiography is a diagnostic tool for the evaluation and management of PVE and is superior to transthoracic echocardiography, interpretation of this test can be compromised by acoustic shadowing from the prosthetic valve, sewing ring, or conduit [10].

Barry Wendt, Lisa Haglund, Ali Razavi, and Ranjit Rath
Department of Internal Medicine, Good Samaritan Hospital, Cincinnati, Ohio

References

Splenic Abscess Caused by Propionibacterium avidum as a Complication of Cardiac Catheterization

Cardiac catheterization is a well-known cause of infection, both locally at the site of catheter insertion and systemically due to transient bacteremia caused by catheter insertion. The majority of cardiac catheterization–associated infections are attributable to staphylococcal species; most of these infections occur as cellulitis or abscess at the catheter-insertion site. On rare occasions, metastatic foci of infections involve heart valves (endocarditis), vascular bodies (osteomyelitis), or cause bacterial seeding in other organs. We describe the first case of cardiac catheterization leading to the formation of a splenic abscess. Moreover, Propionibacterium avidum is an unusual cause of splenic abscess. To our knowledge, there is only one other case report of splenic abscess due to P. avidum.

In September 1997 a 79-year-old man presented to our emergency department with a 3-day history of high fever, rigors, lethargy, weakness and a 1-day history of shortness of breath. He was 6 weeks status post myocardial infarction, which had occurred in Colombia, and he had undergone cardiac catheterization with angioplasty at that time. Three days after this procedure, he had daily fevers (temperature, to 38.5°C), left upper-quadrant abdominal pain, and left-shoulder pain (worse on inspiration). A CT scan of the abdomen, obtained in Colombia, revealed a homogeneous, cystic splenic mass measuring 12.5 × 12.8 cm. This lesion was believed to be a splenic hematoma, possibly secondary to a splenic infarction caused by atheromatous embolization due to the cardiac catheterization.

A transesophageal echocardiogram was obtained that revealed no evidence of endocarditis. Blood cultures were negative. The patient was considered high risk for surgery because of his recent

Reprints or correspondence: Dr. Ameet Vohra, Department of Medicine, Mount Sinai Medical Center, 4300 Alton Road, Miami Beach, Florida 33140.