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, only the *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), vertebral 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.

Clinical Infectious Diseases 1998;26:770–1
© 1998 by The University of Chicago. All rights reserved.
1058-4838/98/2603–0045$03.00
myocardial infarction; therefore, he was treated conservatively with the expectation that the hematoma would resolve spontaneously. His medical history was significant for diet-controlled diabetes and hypertension.

On physical examination, the patient appeared well nourished and comfortable at rest, although he was lethargic. He was febrile (temperature, to 38.9°C), and coarse crackles were heard at both lung bases, with decreased air entry at the left lung base. There was mild left-upper-quadrant tenderness of the abdomen; however, the spleen was not palpable, and there were no peritoneal signs. There was no evidence of peripheral embolism suggestive of endocarditis.

Laboratory studies revealed a WBC count of 13.1 × 10^9/L with a left shift. Four sets of blood cultures were negative, and a CT scan of the abdomen showed a large, hypodense cystic splenic mass (figure 1), unchanging from that observed on the CT scan obtained in Colombia. Because of the clinical deterioration of the patient, the high suspicion of a splenic abscess, and the danger of splenic rupture, a laparotomy was performed. On laparotomy, an enlarged spleen with a large abscess was observed and was decompressed and resected. Cultures of the abscess were performed. A portion of the diaphragm that was necrotic and fixed to the upper dome of the spleen was resected, and the diaphragm was repaired. The 240-g resected spleen contained a 15-cm abscess filled with fibrinopurulent material. The abscess wall was partially fibrotic and contained granulation tissue. Histological examination revealed many cholesterol emboli within splenic arterioles adjacent to the abscess and in random sections of the spleen. The splenic abscess yielded *P. avidum* in pure culture.

Splenic abscesses are not common. However, they may occur in patients with sickle hemoglobinopathies, trauma, bactereemia, splenic infarctions, hematomas, or histories of intravenous drug use. The most common cause of splenic abscess is metastasis from an existing source of infection, e.g., endocarditis (usually due to staphylococcal species), disseminated tuberculosis, and salmonellosal bacteremia. The etiology of splenic abscess in our patient is unclear, but possible explanations include undetected endocarditis or a predisposing splenic lesion such as a hematoma, an infarction, or a cyst. Although histological examination did not show evidence of any of these conditions, it is unlikely that bacteremia due to a low-virulence pathogen would result in abscess formation within a healthy spleen [1]. To our knowledge, this is the first case of splenic abscess as a complication of cardiac catheterization. Further, the fact that *P. avidum* was the source of the infection suggests that there was direct contamination during the cardiac catheterization. In addition, because of the fibrosis surrounding the splenic abscess, it is possible to estimate an abscess age of ~2–8 weeks, a finding consistent with cardiac catheterization as the cause, given that the procedure was undertaken 6 weeks earlier.

*P. avidum* is a pleomorphic, gram-positive, non-spore-forming anaerobic bacillus that comprises part of the normal skin flora in moist areas such as the groin, axillae, and perianal region. Like other *Propionibacterium* species, *P. avidum* is of low pathogenicity and rarely causes infection in the absence of predisposing conditions such as surgery, instrumentation, immunodeficiency, the presence of foreign bodies, malignancy, or trauma. In this case the predisposing condition was cardiac catheterization.

*P. avidum* is susceptible to most common antibiotics, except metronidazole. Dunne et al. [2] reported the only other case in the English-language literature of *P. avidum* causing splenic abscess. The abscess occurred in a 61-year-old man following coronary artery bypass grafting. His symptomology was similar to that of our patient, and the organism was eventually cultured from the abscess fluid after splenectomy was performed. Dunne et al. reported that the portal of entry for the organism was postulated to be the lower-extremity venous-graft donor site, and preexisting areas of splenic fibrosis found on pathological examination, possibly related to a previous episode of malaria, may have predisposed the spleen to bacterial seeding and abscess formation.

Although associated rarely with infection, *Propionibacterium* species are known to cause localized cutaneous infections, conjunctivitis, and dental infections. In the susceptible host, the organisms can also cause severe, life-threatening infections such as endocarditis, meningitis, septic arthritis, osteomyelitis, abscesses, and peritonitis [3]. *Propionibacterium* species should be suspected in infections that occur after instrumentation and in cases with other predisposing conditions. Gram staining showing the presence of pleomorphic, gram-positive rods should raise the suspicion of infection due to *Propionibacterium* species, and the presence of such organisms should not be dismissed as contamination for susceptible hosts.

Ameet Vohra, Enma Saiz, Joseph Chan, Jose Castro, Raphael Amaro, and Jamie Barkin

Department of Medicine, Mount Sinai Medical Center, Miami Beach, Florida

References