persistent MRSA and VRE bacteremias secondary to osteomyelitis and, possibly, endocarditis after treatment failure with other antibiotics.

A 64-year-old male immigrant from Hong Kong was admitted to the hospital because of a history of acute back pain, rigors, and fever. He had chronic renal failure of unknown etiology and had been hemodialysis-dependent for 5 years. Examination revealed a temperature of 39°C and systolic and diastolic heart murmurs consistent with mitral and aortic valve regurgitation. A blood culture yielded VRE that was susceptible to amoxicillin, rifampin, and gentamicin, and MRSA that was susceptible to vancomycin, fusidic acid, and tetracycline, as determined by disc diffusion testing. The following day his right subclavian venous catheter was removed. He was treated with iv vancomycin (1 g) postdialysis and with oral rifampin (300 mg twice daily), and 48 h later his temperature had returned to normal. Culture of the venous catheter tip yielded no organisms. A bone scan was within normal limits and a trans thoracic echocardiogram revealed mitral valve regurgitation but no vegetations. The patient continued antibiotics as an outpatient and continued to dialyze via a new right femoral venous catheter.

One week later, while he was an outpatient, 8 of 8 cultures of the patient’s blood yielded VRE. The patient was readmitted to the hospital, all lines were removed, and peritoneal dialysis was commenced. The MIC of the VRE to amoxicillin, rifampin, and linezolid were 1 mg/L, 0.25 mg/L, and 0.5 mg/L, respectively; otherwise, the organism was highly resistant. Therapy was changed to iv amoxicillin 1 g q.i.d. and iv gentamicin, 160 mg postdialysis. Three additional blood cultures yielded no pathogens. A transesophageal echocardiogram demonstrated mitral and aortic valve regurgitation and a small echodense structure attached to the aortic valve, suggestive, but not typical, of endocarditis. From a cardiovascular standpoint, the patient remained stable and he was afebrile, although his back pain persisted and his C-reactive protein level (CRP, 358 mg/L) remained elevated. An MRI revealed a temperature of 39°C and systolic and diastolic heart murmurs consistent with mitral and aortic valve regurgitation. A blood culture yielded VRE that was susceptible to amoxicillin, rifampin, and gentamicin, and MRSA that was susceptible to vancomycin, fusidic acid, and tetracycline, as determined by disc diffusion testing. The following day his right subclavian venous catheter was removed. He was treated with iv amoxicillin 1 g q.i.d. and iv gentamicin, 160 mg postdialysis. Three additional blood cultures yielded no organisms, although the concentration of linezolid detected was 9.0 mg/g, as measured by HPLC (Antibiotic Reference Unit, Bristol, UK). After a total of 6 weeks treatment and 3 weeks following his operation, linezolid was discontinued. Two months after surgery, the patient was mobile and free of pain. He was afebrile, his WBC count was within normal limits, and his CRP level was 17 mg/L.

It is highly likely this patient had osteomyelitis and possibly endocarditis. Blood cultures repeatedly yielded MRSA and VRE, and no organisms were ever grown from dialysis lines. Surgical specimens failed to yield MRSA or VRE, although a high concentration of linezolid was detected in the debrided bone. We are unaware of other reports where linezolid has been successful as therapy for vertebral osteomyelitis. Despite renal failure, the drug was well tolerated and no dose adjustments were required. Our case supports the argument of those who advocate linezolid as a safe and effective antibiotic to treat serious infections caused by resistant gram-positive organisms such as MRSA and VRE.

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The Final Nail

Sir—Sobel et al. [1] provide important evidence of the mycological futility and absence of clinical benefit associated with treatment of asymptomatic funguria in nonseptic patients. Their report drives another nail (which perhaps should be the final nail?) into the coffin of antifungal therapy for asymptomatic candiduria. The only detectable outcome difference between the fluconazole and placebo groups was a somewhat
lower prevalence of candiduria among fluconazole recipients at the conclusion of therapy. Even this modest mycological difference, which nonetheless left 50% of the fluconazole group with persistent funguria, had vanished by 2 weeks after therapy. Moreover, treatment had no detectable impact on more clinically relevant end points, such as mortality, development of pyelonephritis or candidemia, and urinary symptoms.

These findings poorly support the assertion that “oral fluconazole (is/was) effective.” They also leave very much in question what was meant by “when treatment of asymptomatic or minimally symptomatic candiduria is indicated,” since, as yet, there is no clear evidence that such treatment is ever indicated. Why do clinicians persist in battling asymptomatic candiduria, in the absence of evidence of clinical benefit? Perhaps it is because funguria is not “normal.” Physicians are committed to restoring “normalcy” wherever possible, and, in the compromised, complicated patients who develop candiduria, this may represent one of the few seemingly reversible abnormalities that can be addressed. Yet the crusade to vanquish asymptomatic candiduria in hospitalized patients appears to be as futile as the pursuit to vanquish asymptomatic bacteriuria in the elderly [2].

It may be time to institute a moratorium on diagnosing and treating asymptomatic candiduria outside the context of clinical trials that are designed to establish whether there is any clinical benefit to such efforts. Further efforts to define the “how” of treating candiduria will not be helpful without an established “why” [3].

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