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S aureus With Reduced Susceptibility to Vancomycin

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Since 1996, vancomycin-intermediate *Staphylococcus aureus* (VISA; vancomycin minimum inhibitory concentration [MIC]=8-16 µg/mL) has been identified in Europe, Asia, and the United States. The emergence of reduced vancomycin susceptibility in *S aureus* increases the possibility that some strains will become fully resistant and that available antimicrobial agents will become ineffective for treating infections caused by such strains. The CDC recently reported the fourth case of confirmed VISA from a patient in the United States.

The case was a 63-year-old woman with methicillin-resistant *Staphylococcus aureus* bacteremia (MIC<1 µg/mL) who was transferred from a long-term–care facility to an Illinois hospital (hospital A) in April 1999. The patient had a history of frequent hospitalizations for complications of hemodialysis-dependent, end-stage renal disease and intravascular access, including two failed arteriovenous grafts, multiple central venous catheter-associated infections, and intermittent receipt of vancomycin therapy through June 1998. Thirteen days after hospital admission and 25 days after initiating vancomycin therapy (median vancomycin serum concentration=12.7 µg/mL; range, 12.1 µg/mL-20.9 µg/mL), a culture from her blood grew *S aureus* with an MIC of 4 µg/mL; the blood culture was tested using the Vitek system (bioMerieux, Hazelwood, MO). Three subsequent blood specimens drawn within the next 3 days grew *S aureus* with MICs of 8 µg/mL on confirmatory testing. The isolates, identical by pulsed-field gel electrophoresis, were resistant to penicillin, oxacillin, clindamycin, erythromycin, ciprofloxacin, and rifampin but susceptible to trimethoprim-sulfamethoxazole, and tetracycline, gentamicin and had intermediate susceptibility to chloramphenicol. No VISA strains were recovered from other body sites. An echocardiogram demonstrated a mitral valve vegetation, but the patient declined surgical intervention. Despite treatment with intravenous vancomycin, rifampin, and tobramycin, the patient died 10 days after the first VISA blood specimen was drawn; the cause of death was endocarditis.

The VISA isolate was interpreted as "susceptible" at 4 µg/mL by the Vitek system. Susceptibility results were confirmed by the CDC. The CDC's Interim Guidelines for Prevention and Control of Staphylococcal Infection Associated with Reduced Susceptibility to Vancomycin were implemented in hospital A. None of 10 family members or 171 healthcare workers screened by nares culture was colonized with VISA. No other VISA isolates were identified in other hospitalized patients.

The acronyms VISA and GISA (glycopeptide-intermediate *S aureus)* have been used in the United States to describe *S aureus* isolates with reduced susceptibility to vancomycin. The National Committee for Clinical Laboratory Standards published interpretive criteria defining both. The term GISA is a technically more accurate description of VISA strains, because all isolates have shown intermediate level MICs to the glycopeptide drugs, vancomycin, and teicoplanin. However, clinicians may not recognize the term *glycopeptide,* and the acronym *VISA* is used more frequently.

The CDC seeks laboratory reports of confirmed cases of VISA infection for an ongoing nationwide epidemiological study. Information on confirmatory testing, investigation therapy, and infection control guidelines can be obtained from the CDC's Hospital Infections Program, National Center for Infectious Diseases, telephone (404) 639-6413; http://www.cdc.gov/ncidod/hip/vanco/vanco.htm; or by e-mailing SEARCH @cdc.gov. The recovery of *S aureus* with reduced susceptibility to vancomycin (eg, MIC>4 µg/mL) should be reported promptly to local and state health departments and to the CDC, infection-control precautions should be implemented, and an epidemiological investigation should be conducted.

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