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First Isolate of Vancomycin-Resistant *Staphylococcus aureus*—Japan

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The first known isolate of *Staphylococcus aureus* resistant to vancomycin (VRSA) has been reported from Japan. The CDC received the strain from Dr. Keiichi Hiramatsu of Jutendo University in Tokyo, Japan, and confirmed the isolation of a strain of *S aureus* with a vancomycin minimum inhibitory concentration (MIC) of 8 µg/mL (broth microdilution). The disk diffusion zone size was 18-19 mm (considered intermediate resistance by the National Committee for Clinical Laboratory Standards). It is a methicillin-resistant *Staphylococcus aureus* (MRSA) strain that is susceptible to synergid and arbekacin (a drug used in Japan) but few other drugs. It was isolated from a surgical-site infection (undrained abscess) of a 4-month-old boy who had surgery to correct pulmonary atresia and been treated with vancomycin for 29 days without improvement. The child developed purulent discharge after surgery, MRSA was isolated, and the child was given vancomycin plus arbekacin, with healing of the wound. Ten days later, fever developed, and a subcutaneous abscess was found;

ampicillin-sulbactam was added to his regimen. He improved, but relapsed and developed an abscess at the surgical site 12 days post-therapy. Additional therapy with arbekacin and ampicillin-sulbactam, along with drainage of the abscess, cleared the infection. The baby was discharged and apparently has been well since.

Similar MRSA strains (by pulsed-field gel electrophoresis), although with vancomycin MICs of only 2-4 µg/mL, have been seen in several hospitals in Japan. In Japan, screening on brain-heart infusion agar with 4 µg/mL of vancomycin, held for 48 hours at 37°C, reveals growth of strains that have post-induction MICs of 4-8 µg/mL. In other words, strains with initial vancomycin MICs of 2-4 jump to 4-8 after exposure to vancomycin. The strain received by the CDC from Japan had a stable MIC of 8.

Dr. Hiramatsu describes this strain as "homoresistant" and the others as "heteroresistant" to vancomycin, much like oxacillin. The strain described above, called Mu-50, is negative for *vanA*, *vanB*, *vanC1*, *vanC2*, and *vanC3*. The mechanism of resistance is unknown. Dr. Fred Tenover from the CDC made the following points about this VRE isolate:

"First, such strains have not yet been recognized in the United States, and, second, the organism was obtained from an undrained abscess; if this strain were present in the blood, normal doses of vancomycin should have been effective in eliminating it."

Rather than VRSA, the CDC is calling this VISA because of its intermediate resistance to vancomycin. Infectious disease experts have commented that this may not have been a treatment failure, but rather a failure to drain an abscess, because the infection resolved after drainage. This case is scheduled for publication in the July 1997 issue of *Antimicrobial Chemotherapy* and in the *MMWR* in August. The CDC currently is working on a draft guideline for hospitals on the control of VRSA; however, this draft has not yet been approved by the CDC's Hospital Infection Control Practices Guidelines Committee.

FROM: Tenover F. Letter to microbiologists published on ASM's *Clinical Micronet* (clinmicronet@asmusa.org), May 27, 1997; and the article "Staph germ resists potent drug." *Chicago Tribune* May 27, 1997; section A.