In reviewing her history and clinical record, we realized she had undergone flexible sigmoidoscopy in December 1983 and did not receive antibiotic prophylaxis. Over the ensuing six months, the patient lost 5 kg (11 lb) of weight, had recurrent bouts of nausea and vomiting and had repetitive episodes of dizziness. Due to the patient's complex set of medical problems and multiple medications, consideration of the diagnosis of prosthetic valve endocarditis was not entertained until June 1984, at which time an astute resident drew blood specimens for culture. Two days later a heavy growth of enterococcus was identified in the blood and the patient underwent a six week course of intravenous antibiotic therapy. No other source for the enterococcemia could be elucidated.

While the 1977 American Heart Association (AHA) guidelines for prevention of bacterial endocarditis clearly recommend antibiotic prophylaxis for patients with prosthetic valves undergoing sigmoidoscopy,[2] a large percentage of endoscopists do not follow these guidelines.[3] Even in retrospect, the gastroenterologist who carried out this patient's sigmoidoscopy felt that prophylaxis was not indicated.

The case described above is at least the third now reported in the literature of enterococcal endocarditis following sigmoidoscopy. Perhaps all endoscopists and primary care physicians would do well to review the AHA guidelines for prophylaxis, particularly as screening flexible sigmoidoscopy is becoming more widespread in primary care offices.

JANE L. MURRAY, MD
Assistant Director
Family Practice Residency Program
Santa Monica Hospital Medical Center
1225-15th Street
Santa Monica, CA 90404

REFERENCES

1. Rodriguez W, Levine JS: Enterococcal endocarditis following flexible sigmoidoscopy. West J Med 1984 Jun; 140:951-953
2. Kaplan EL, Anthony BF, Bisno A, et al: AHA Committee Report—Prevention of bacterial endocarditis. Circulation 1977 Jul; 56:139A-143A
3. Meyer GW: Prophylaxis of infective endocarditis during gastrointestinal procedures: Report of a survey. Gastrointest Endosc 1979 Jan; 25:1-2

Methicillin-Resistant *Staphylococcus aureus*

TO THE EDITOR: We wish to report three cases of community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) infection in intravenous drug abusers. MRSA is a well-recognized nosocomial pathogen[1,2] and has been identified as a community-acquired pathogen among drug addicts in Detroit.[3] To our knowledge MRSA is not widely recognized as a cause of community-acquired disease in this area.

Reports of Cases

CASE 1. The patient, a 45-year-old drug abuser—a "skin popper"—was admitted with multiple cutaneous abscesses, from which grew MRSA (sensitive to chloramphenicol, gentamicin, trimethoprim-sulfamethoxazole and vancomycin). Although blood cultures were negative, a urine culture showed more than 10^5 colonies per ml of MRSA with identical sensitivities.

CASE 2. A 32-year-old woman was admitted with MRSA bacteremia (sensitive to chloramphenicol, gentamicin, trimethoprim-sulfamethoxazole, clindamycin and vancomycin). She was a heroin addict who said she had not shared needles. Three months previously she had been admitted to hospital for endocarditis due to a methicillin-sensitive *Staphylococcus aureus*.

CASE 3. A 33-year-old woman, a drug abuser, was admitted with chronic osteomyelitis of the symphysis pubis. Blood culture and biopsy of the site revealed MRSA with sensitivities identical to those in case 1. The patient denied drug injection in the groin region.

All three patients had a history of multiple admissions to hospital and frequent antibiotic administration before the admission during which MRSA was recovered.

Traditional empiric therapy for intravenous drug abusers with presumptive staphylococcal infections has included coverage with a penicillinase-resistant antibiotic, typically nafcillin or a first-generation cephalosporin. Our experience suggests that in a seriously ill intravenous drug abuser in whom *Staphylococcus aureus* is suspected, especially when there is a history of recent hospital admission or antibiotic use, empiric therapy should include vancomycin pending culture and sensitivity results.

ALAN LIFSON, MD
Fellow, Infectious Diseases
BARBARA PAUL, MD
Mount Zion Hospital
PO Box 7921
San Francisco, CA 94120

REFERENCES

1. Haley RW, Hightower AW, Khabbaz RF, et al: The emergence of methicillin-resistant Staphylococcus aureus infections in United States hospitals. Ann Intern Med 1982 Sep; 97:297-308
2. Locksley RM, Cohen ML, Quinn TC, et al: Multiple antibiotic-resistant Staphylococcus aureus: Introduction, transmission, and evolution of nosocomial infection. Ann Intern Med 1982 Sep; 97:317-324
3. Levine DP, Cushing RD, Jui J, et al: Community-acquired methicillin-resistant Staphylococcus aureus endocarditis in the Detroit Medical Center. Ann Intern Med 1982 Sep; 97:330-338

Reye's Syndrome and Salicylates

TO THE EDITOR: With regard to the role of salicylates in Reye's syndrome,[1] I would suggest an additional comment to Dr Trauner's summary recommendation "to try other means of reducing fever and discomfort in children with viral illnesses and, in particular, flulike illnesses and varicella . . . ." We should also be counseling parents as to the role of fever in the immunological response to infection and to decondition them from using antipyretics routinely when fever and discomfort are modest. My guess is that the association between Reye's syndrome and salicylates would not exist were it not for the large population of children who receive aspirin (and sometimes alternately acetaminophen) on a regular basis for any temperature elevation.

ELLIOT M. SACKS, MD
Family Practice
Valley Medical Group of Lompoc, Inc
Lompoc, CA 93436

REFERENCE

1. Trauner DA: Reye's syndrome (Medical Progress). West J Med 1984 Aug; 141:206-209