

Short Communication

Nosocomial Methicilin-Resistant *Staphylococcus aureus* Endocarditis with Splenic Abscess in a Pregnant Woman

Zulal Ozkurt*, Bilgehan Erkut¹, Ayten Kadanali, Azman Ates¹ and Ibrahim Yekeler²

Department of Clinical Bacteriology and Infectious Diseases and

¹Department of Cardiovascular Surgery, Ataturk University School of Medicine, Erzurum and

²Department of Cardiovascular and Thoracic Surgery, Siyami Ersek Hospital, Istanbul, Turkey

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SUMMARY: A 36-year-old, 7-week-gravida patient with catheter-related nosocomial infective endocarditis due to methicillin-resistant *Staphylococcus aureus* (MRSA) is presented in this paper. The patient was admitted to our hospital because of carbon monoxide intoxication. After 14 days, MRSA catheter-related bacteremia developed. The central venous catheter was immediately removed, and teicoplanin therapy was started. Because of persistent fever, leukocytosis, and high C-reactive protein values, endocarditis was suspected. A transesophageal echocardiogram revealed 19-mm vegetation on her mitral valve, confirming the diagnosis of endocarditis. Gentamicin and rifampicin were added to the therapy regimen, and the dose of teicoplanin was increased to 12 mg/kg/day. After 8 days, a splenic abscess was detected by ultrasonography. Vegetation excision, mitral valve replacement by open-heart surgery and splenectomy were performed in the same operation. Antibiotherapy was continued for 6 weeks after surgery, and the patient's condition improved. The development of endocarditis could be prevented by proper clinical practices.

Nosocomial infective endocarditis (NIE) is defined as acute IE occurring within 48 to 72 h or more of admission to a hospital and/or endocarditis directly resulting from a hospital-based procedure performed during a previous hospital stay within 8 weeks of admission (1). NIE is a complication of nosocomial bacteremia and is associated with a high mortality rate. It develops secondary to new therapeutic modalities such as intravenous catheters, hyperalimentation lines, pacemakers, dialysis shunts, etc. (2). Colonized intravascular catheters are the most common identified source of nosocomial endocarditis, accounting for one- to two-thirds of all reported cases (3).

A 36-year-old, 7-week-gravida woman was admitted to our hospital in a comatose condition because of carbon monoxide (CO) intoxication. She was immediately treated with high-flow oxygen and supportive medical therapy, recovering her consciousness completely nearly 10 days after CO exposure. After 14 days of hospitalization, the clinical findings of the patient improved, but her fever remained at 38-39°C. On physical examination, hyperemia and purulence were detected around the central venous catheter. The laboratory results revealed the following: hemoglobin (Hb), 8.7 mg/dL; white blood cells (WBCs), $19,900 \times 10^3/\text{mm}^3$ (with 87% polymorphonuclear leukocyte [PNL]); erythrocyte sedimentation rate (ESR), 80 mm/h; C-reactive protein (CRP), 17 mg/L; lactate dehydrogenase (LDH), 1,669 IU/L; and urine red blood cells (RBCs), $17/\text{mm}^3$. All other results were normal. Blood and catheter cultures were performed, and methicillin-resistant *Staphylococcus aureus* (MRSA) was isolated from both the blood and central venous catheter cultures. The catheter was removed and teicoplanin 6 mg/kg/day (once per day) was initiated. Physical examination showed



Fig. 1. Vegetation in echocardiography.

a grade 3 systolic murmur at the apex. Because of persistent fever, leukocytosis and high CRP values, the patient was suspected to have endocarditis. A transesophageal echocardiogram (TEE) revealed 19-mm vegetation on her mitral valve (Fig. 1), and the diagnosis of endocarditis was established following Duke's criteria. Gentamicin (1 mg/kg per 8-h IV) and rifampicin (600 mg/day, per os) were added to the therapy regimen, and the dose of teicoplanin was increased to 12 mg/kg/day. Subsequent blood cultures were performed three times under therapy to evaluate MRSA. After 8 days of therapy, the patient complained of pain on her left side, and a splenic abscess was detected by ultrasonography. A medical abortion was performed due to exposure to long-term therapy with high doses of drugs and planned surgery. A computerized tomography evaluation confirmed the existence of the splenic abscess (Fig. 2), and MRSA was isolated from abscess material. Vegetation excision, mitral valve replacement by open-heart surgery and splenectomy were performed in the same operation. Gentamicin was continued for 14 days at the

*Corresponding author: Mailing address: Saglik Sok, Gez Konutlari, No:4, Dadaskent, Erzurum, Turkey. Tel: +90 442 316 63 33 ext. 2025, Fax: +90 442 316 63 40, E-mail: zozkurt@atauni.edu.tr

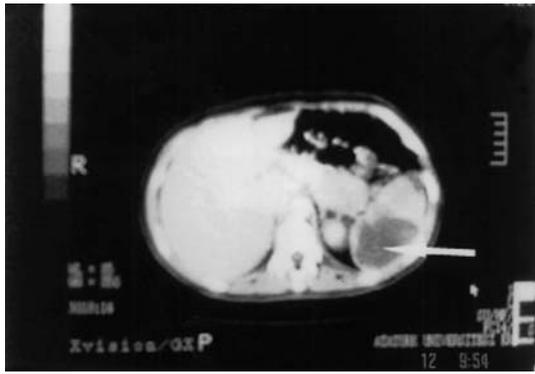


Fig. 2. The splenic abscess in computerized tomography.

same dose, and teicoplanin (6 mg/kg/day, once per day) and rifampicin (the same dose) were continued for 6 weeks after surgery. The patient's condition improved completely.

NIE is a rare but serious complication of nosocomial bacteremia and its case fatality rate is greater than 50% (4). The source of nosocomial endocarditis is usually detectable, and intravascular devices are the source of bacteriemia in 50.8-85.7% of all nosocomial endocarditis cases (5,6). In the present study, the source of NIE was a central venous catheter.

Nosocomial endocarditis is most often reported to be due to predominant *S. aureus* and coagulase-negative staphylococci (77.4%) and, less often, to streptococci (1). In nearly half of all cases, NIE is caused by MRSA strains (2). The risk of endocarditis is reported to be 31.7% in patients with *S. aureus* bacteremia (7). Irrespective of the epidemiological origin of *S. aureus* bacteremia, mortality remains high particularly among nosocomial endocarditis cases (8). Therefore, patients with *S. aureus* bacteremia should be aggressively evaluated for endocarditis. TEE has been reported to have higher sensitivity than transthoracic echocardiogram (TTE) in detecting cardiac lesions (81.4% versus 33.9%) (5). TTE does not reliably exclude vegetation, and TEE is therefore recommended to detect NIE in patients with *S. aureus* bacteremia (9). Both TEE and Duke's criteria should assist in the early and accurate identification of *S. aureus* NIE leading to the early application of appropriate antibiotic therapy (1,5). In the light of these considerations, we used TEE to diagnose NIE in our patient. A predisposing heart disease or prosthesis valve is not a mandatory condition of NIE (6). Llinares et al. (6) report 21 cases of nosocomial endocarditis in patients with no prosthesis valve. Our case also had no history of any heart disease or prosthesis valve.

Llinares et al. (6) found involvement of the left cavities in 76.2% of their cases, and involvement of the right cavities was also detected in cases in which an intravascular catheter was known to be the source of NIE. The course is frequently fulminant when it involves the mitral or aortic valve, with widespread metastatic infection to distant organs, i.e., the central nervous system, the heart, spleen, lungs, kidneys and joints, and results in death in approximately 40% of patients; the mortality rate often exceeds 50% in patients over 50 years of age (2,8). In the present case, a splenic abscess was detected as a metastatic infection.

The recommended therapy for MRSA endocarditis is vancomycin for 4-6 weeks, and teicoplanin, which shows similar or better activity than vancomycin, may be a useful alternative (2). Teicoplanin penetration in cardiac tissue is

good with mean tissue levels of 70.6 and 139.8 mg/l being achieved by single doses of 6 and 12 mg/kg, respectively (10). Glycopeptides are not effective as other more rapidly bactericidal agents when used as monotherapy against MRSA infection. Combination therapy has proven superior in animal experiments; human studies to date are few and inconclusive (10), however, combination therapy resulted in a more rapid rate of eradication of the bacteremia. Many authorities still use combination therapy for short periods of time for left-sided *S. aureus* IE, especially in fulminant cases (2), however, better success rates have been achieved with combination therapy in staphylococcal endocarditis, particularly *S. aureus* (78% versus 42%) (10). Although we used teicoplanin, clinical improvement was not achieved in the early period of the disease. Thus, rifampicin and gentamicin were added for their synergic effect, and the teicoplanin dose was increased to the recommended doses (12 mg/kg/day) for endocarditis.

Another feature of the present case was pregnancy. The incidence of infective endocarditis during pregnancy has been reported to be 0.006%. During pregnancy, infective endocarditis shows a high mortality for both mother and fetus, with a maternal mortality rate of nearly 33%, and a fetal mortality rate which can reach 29% (11). Jorge et al. (12) reported mortality rates of 47% (maternal), and 63% (fetal) in 11 cases. Campuzano et al. (13) reviewed the literature from 1965 to 2002, and reported maternal mortality rates of 22.1% and fetal mortality rates of 14.7% in 68 cases, finding, furthermore, that valve-specific maternal mortality was 42.1, 21.7 and 9.5% for the aortic, mitral and tricuspid valves, respectively.

Education of health personnel, proper hand hygiene, aseptic technique during insertion and catheter site care are essential for the prevention of catheter-related infections (14). NIE occurs in a definable sub-population of hospitalized patients, but may be prevented by the application of currently recommended preventive and therapeutic practices, and improved care of intravascular devices (1).

Finally, nosocomial endocarditis is, to some extent, an emerging nosocomial infection but is a preventable disease. Preventive measures must be strictly implemented in hospital practice.

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