Novel Nosocomial Infections by *Stenotrophomonas maltophilia*: First Reported Case from Lucknow, North India

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Received 27 December 2002/Returned for modification 24 March 2003/Accepted 2 June 2003

We describe a case of empyema with infected ascites caused by *Stenotrophomonas maltophilia*, which has rarely been reported as pathogenic. The source was determined to be a disinfectant solution. The isolate was sensitive to a newer carbapenem-meropenem, and the patient was treated successfully. This case represents a novel dual presentation of a nosocomial infection by the isolate in question.

CASE REPORT

A 72-year-old female was admitted to our hospital in June 2002 with progressive abdominal distention and breathlessness. She was diagnosed previously with carcinoma of the left ovary (stage IV) but was not cooperative regarding her treatment and follow-up. On admission, there was bilateral pleural effusion, ascites, peded edema, and hepatomegaly. Her total leukocyte count was \(7.5 \times 10^9/liter\), and her platelet count was \(155 \times 10^9/liter\). Her hemogram, coagulation profile, and blood biochemistry were within the normal limits. Her carcinoembryonic antigen-125 level was 334 King Armstrong U/liter (normal value, less than 35 U/liter). A chest radiograph revealed bilateral pleural effusion. An abdominal ultrasonographic examination confirmed the presence of ascites and disclosed parenchymal liver metastasis, but because of massive ascites, pelvic structures could not be properly visualized. Transvaginal sonography (Logic 500 instrument [no. RT 3200; GE WIPRO] with a 7.5-MHz transducer) showed an irregular (approximately 10 by 12 cm) mass that was partially cystic and partially solid with few echo-dense areas.

The patient was afebrile on admission. Pleural and ascitic fluids were found to be transudative in nature, and cultures were sterile. There were malignant cells on cytologic examination. From the third day on, she developed a fever with chills and intense abdominal pain. The ascitic and pleural fluids were again tapped and found to be exudative in nature, with 5 to 6 polymorphonuclear leukocytes per oil immersion field, and 1 to 2 polymorphonuclear leukocytes per oil immersion field were found to be sterile. The sensitivity of *S. maltophilia* was tested against amikacin (30 \(\mu\)g), tobramycin (10 \(\mu\)g), piperacillin (100 \(\mu\)g), carbenicillin (100 \(\mu\)g), ceftriaxone (30 \(\mu\)g), ceftazi dime (30 \(\mu\)g), ciprofloxacin (5 \(\mu\)g), levofloxacin (5 \(\mu\)g), gatifloxacin (5 \(\mu\)g), cefoperazone-sulbactam (75 and 10 \(\mu\)g, respectively), pipercillin-tazobactam (100 and 10 \(\mu\)g, respectively), ticarcillin-clavulanic acid (75 and 10 \(\mu\)g, respectively), cefpirome (30 \(\mu\)g), and meropenem (10 \(\mu\)g), and the organism was sensitive in vitro to meropenem and ciprofloxacin only by the modified Stokes method (1). Skin disinfectants commonly used in hospital wards, like betadine (10% povidone iodine), chlorhexidine gluconate (0.5% alcoholic solution), ethanol (70%), and savlon (0.3% chlorhexidine gluconate and 3% cetrimide; 60 to 90 ml in 1.0 to 1.5 liters of water), and other materials, like needles, cotton packs, etc., used during pleural and ascitic fluid tapping and supposed to be sterile were also subjected to culture and, if required, sensitivity testing. Povidone iodine showed growth of the same isolate with the same sensitivity pattern. The values of the API 20 NE and Rapid NF Plus systems in identifying the isolate from the patient and that from povidone iodine were exactly the same.

The patient was put on meropenem (1 g given intravenously every 8 h) for 2 weeks. A repeat culture done 3 days later grew *S. maltophilia* with the same sensitivity pattern. However, the patient was afebrile from the fourth day on. On the seventh day, ascitic and pleural fluid cultures were sterile. The patient was ultimately treated with neoadjuvant chemotherapy, followed by interval debulking.

Discussion. *S. maltophilia* has emerged as an important cause of morbidity and mortality in hospitalized patients, particularly in intensive care units. This organism was originally classified as *Pseudomonas maltophilia* but was transferred to the genus *Xanthomonas* in 1993 (8) and subsequently became the sole member to the genus *Stenotrophomonas* (13). The patient was afebrile at the time of admission and became infected during her hospital stay. Her pleural and ascitic fluid samples were sterile at first, which shows that the infection was acquired after hospitalization during an invasive procedure like tapping, which was probably not done with ade-
quate aseptic precautions. On surveillance, growth of S. maltophilia was found in the ascitic and pleural fluids, as well as in the povidone iodine solution used. The antibiogram and the values of the API 20 NE and Rapid NF Plus systems for the isolate from the disinfectant (povidone iodine) were identical to those of the isolate from the pleural and ascitic fluids. Immediately, the whole batch of povidone iodine solution was discarded and hospital authorities were informed.

Transmission of nosocomial infections associated with contaminated disinfectant solutions, as in the present study, has been reported by Vartivarian et al. (12) and Wishart et al. (15); isolation from sustained-release ganciclovir implants (3) or hospital water (7) has also been reported. The organism has also been reported to cause urinary tract infection, mucocutaneous and soft tissue infections, bacteremia, pneumonia, endocarditis, mastoiditis, osteochondritis, and meningitis (2, 4, 6, 10, 14). However, no other case of peritonitis and empyema together in the same patient at the same time due to this organism has been reported.

Therapy for infections with these pathogens is challenging because of their resistance to most antimicrobial agents and the variable antimicrobial susceptibility of different strains (9). However, initial empirical use of trimethoprim-sulfamethoxazole and ticarcillin-clavulanic acid in combination is recommended (11). The organism infecting our patient was found to be multidrug resistant, even to the expanded-spectrum cephalosporin cefpirome, newer quinolones like levofloxacin and gatifloxacin, and the latest β-lactam–β-lactamase inhibitor combinations. She was treated successfully with meropenem, a carbapenem recently introduced into our country that is not yet used routinely in our hospitals.

Therefore, in any routine laboratory, whenever any atypical Pseudomonas strain is grown, a detailed history of the case should be taken, the organism should be identified to the species level, and the source of the infection must be determined. Newer drugs like meropenem, which was found to be very effective in our case, should always be tested in vitro against such isolates. Finally, strict vigilance is required to check routinely the various skin disinfectants used in the hospital.

REFERENCES