Pseudo-, Xantho-, and now

Stenotrophomonas maltophilia:

New kid on the block

Stenotrophomonas maltophilia, with its checkered taxonomic past, has emerged as one of the most frequently isolated nonfermenting Gram-negative bacilli in the clinical microbiology laboratory and has become a leading cause of serious life-threatening nosocomial infection over the past 10 years (1-7). The organism was first described as a new species of Pseudomonas in 1960 when it was isolated from human and animal sources, water and milk (8,9) and was initially named Pseudomonas maltophilia. It was reclassified from the Pseudomonas genus to the genus Xanthomonas in 1983 based on studies finding significant rRNA homology differences between the two genera as well as guanine plus cytosine content, differences in cellular enzymes, fatty acids, ubiquinone and growth characteristics (10,11). The taxonomic status of this organism was challenged again in the early 1990s on the basis of multiple significant phenotypic differences, and the creation of a new genus, Stenotrophomonas, was proposed in 1993 (12). The new genus, Stenotrophomonas, carries only one species, S maltophilia, whereas the genus Xanthomonas continues to house several species. The name of the new genus, Stenotrophomonas, is derived from the Greek words, stenus (narrow), trophus (one who feeds) and monas (a unit) and quite aptly describes the organism which requires few substrates and can thrive in water sources.

S maltophilia is commonly isolated from water and soil samples, animal sources, plant material and foods, and has been isolated from hospital equipment, water faucets, water traps, sinks, suction catheters and respirometers. The organism is usually commensal but can be a part of the transient flora of hospitalized patients (2,3). Although initially considered a colonizer when recovered from clinical specimens, S maltophilia has risen to compete with Pseudomonas aerugi-
Xanthomonas maltophilia as the leading cause of nonfermenting Gram-negative bacillary infections. Characteristically, infection occurring with this organism is serious and most often affects the immunocompromised host. The infections with which it has been associated include bacteremia, endocarditis, meningitis, lower respiratory tract infections and soft tissue infections (1-7). The most common underlying disease in the host is malignancy, particularly hematological (3-7). It has been suggested (13) that the altered biochemical environment associated with malignant lesions might select for the growth of a minimally virulent X. maltophilia but the pathogenicity of this organism in patients without malignancy has been demonstrated. Mortality has been reported to be as high as 38% in patients with bacteremia due to X. maltophilia (2).

Risk factors for X. maltophilia colonization or infection include prior broad spectrum antimicrobial use, neutropenia, central venous catheterization, unresolved underlying illness and a prolonged hospital stay (2,3,13). Since the organism is not considered a part of the normal gastrointestinal flora colonization is generally thought to occur from exogenous sources within the hospital environment, often water or medical equipment sources. The epidemiology of colonization and cross-transmission of this organism within the hospital environment will be delineated with the use of newer molecular subtyping techniques (13-15).

X. maltophilia presents a therapeutic challenge because it is an inherently antibiotic-resistant organism, exhibiting resistance to aminoglycosides, extended spectrum beta-lactams and quinolones (1,13,16,17). Broad spectrum resistance to aminoglycosides is believed to be due to decreased permeability, whereas the elaboration of two chromosomally mediated inducible beta-lactamases combined with altered outer membrane permeability confers resistance to beta-lactam agents. One of these beta-lactamases is a zinc dependent enzyme that breaks down carbapenems and confers resistance to aminoglycosides (10). Mutations in the outer membrane proteins are believed to confer resistance to quinolones (17). There are also difficulties with standard susceptibility test methodologies, and disc diffusion test methods are not considered to be reliable (18).

Trimethoprim-sulfamethoxazole, ticarcillin-clavulanic acid, doxycycline and clinafloxacin appear to be the most predictably active agents (19,20) on the basis of agar dilution and time-kill curves.

The emergence of X. maltophilia as a significant pathogen in immunocompromised patient populations comes as no surprise. With increasing numbers of such patients and increasing use of broad spectrum antimicrobials the numbers of infections will likely continue to increase. The prevention and control of infections due to this organism will require continued surveillance, monitoring of resistance trends, application of appropriate infection control practices (including the environment) and prudent hospital antimicrobial restriction policies.

REFERENCES


