Epidemiology of MDROs

Management of Multidrug-Resistant Organisms in Healthcare Settings (2006)

KEY POINTS

Epidemiology of MDROs from the Management of Multidrug-Resistant Organisms in Healthcare Settings (2006) guideline.

ON THIS PAGE

Trends

Prevalence of MDROs varies temporally, geographically, and by healthcare setting (70, 71). For example, VRE emerged in the eastern United States in the early 1990s, but did not appear in the western United States until several years later, and MDRSP varies in prevalence by state (72). The type and level of care also influence the prevalence of MDROs. ICUs, especially those at tertiary care facilities, may have a higher prevalence of MDRO infections than do non-ICU settings (73, 74). Antimicrobial resistance rates are also strongly correlated with hospital size, tertiary-level care, and facility type (e.g., LTCF) (75, 76). The frequency of clinical infection caused by these pathogens is low in LTCFs (77, 78). Nonetheless, MDRO infections in LTCFs can cause serious disease and mortality, and colonized or infected LTCF residents may serve as reservoirs and vehicles for MDRO introduction into acute care facilities (78-88). Another example of population differences in prevalence of target MDROs is in the pediatric population. Point prevalence surveys conducted by the Pediatric Prevention Network (PPN) in eight U.S. PICUs and 7 U.S. NICUs in 2000 found ≤4% of patients were colonized with MRSA or VRE compared with 10-24% were colonized with ceftazidime- or aminoglycoside-resistant gram-negative bacilli; < 3% were colonized with ESBL-producing gram negative bacilli. Despite some evidence that MDRO burden is greatest in adult hospital patients, MDRO require similar control efforts in pediatric populations as well (89).

During the last several decades, the prevalence of MDROs in U.S. hospitals and medical centers has increased steadily (90, 91). MRSA was first isolated in the United States in 1968. By the early 1990s, MRSA accounted for 20%-25% of *Staphylococcus aureus* isolates from hospitalized patients (92). In 1999, MRSA accounted for >50% of *S. aureus* isolates from patients in ICUs in the National Nosocomial Infection Surveillance (NNIS) system; in 2003, 59.5% of *S. aureus* isolates in NNIS ICUs were MRSA (93). A similar rise in prevalence has occurred with VRE (94). From 1990 to 1997, the prevalence of VRE in enterococcal isolates from hospitalized patients increased from <1% to approximately 15% (95). VRE accounted for almost 25% of enterococcus isolates in NNIS ICUs in 1999 (94), and 28.5% in 2003 (93).

GNB resistant to ESBLs, fluoroquinolones, carbapenems, and aminoglycosides also have increased in prevalence. For example, in 1997, the SENTRY Antimicrobial Surveillance Program found that among *K. pneumoniae* strains isolated in the United States, resistance rates to ceftazidime and other third-generation cephalosporins were 6.6%, 9.7%, 5.4%, and 3.6% for bloodstream, pneumonia, wound, and urinary tract infections, respectively (95). In 2003, 20.6% of all *K. pneumoniae* isolates from NNIS ICUs were resistant to these drugs (93). Similarly, between 1999 and 2003, *Pseudomonas aeruginosa* resistance to fluoroquinolone antibiotics increased from 23% to 29.5% in NNIS ICUs (74). Also, a 3-month survey of 15 Brooklyn hospitals in 1999 found that 53% of *A. baumannii* strains exhibited resistance to carbapenems and 24% of *P. aeruginosa* strains were resistant to imipenem (10). During 1994-2000, a national review of ICU patients in 43 states found that the overall susceptibility to ciprofloxacin decreased from 86% to 76% and was temporally associated with increased use of fluoroquinolones in the United States (96).

Lastly, an analysis of temporal trends of antimicrobial resistance in non-ICU patients in 23 U.S. hospitals during 1996-1997 and 1998-1999 (97) found significant increases in the prevalence of resistant isolates including MRSA, ciprofloxacin-resistant *P. aeruginosa*, and ciprofloxacin- or

ofloxacin-resistant *E. coli.* Several factors may have contributed to these increases including: selective pressure exerted by exposure to antimicrobial agents, particularly fluoroquinolones, outside of the ICU and/or in the community (7, 96, 98); increasing rates of community-associated MRSA colonization and infection (99, 100); inadequate adherence to infection control practices; or a combination of these factors.

Important concepts in transmission

Once MDROs are introduced into a healthcare setting, transmission and persistence of the resistant strain is determined by the availability of vulnerable patients, selective pressure exerted by antimicrobial use, increased potential for transmission from larger numbers of colonized or infected patients ("colonization pressure") (101, 102); and the impact of implementation and adherence to prevention efforts. Patients vulnerable to colonization and infection include those with severe disease, especially those with compromised host defenses from underlying medical conditions; recent surgery; or indwelling medical devices (e.g., urinary catheters or endotracheal tubes (103, 104)). Hospitalized patients, especially ICU patients, tend to have more risk factors than non-hospitalized patients do, and have the highest infection rates. For example, the risk that an ICU patient will acquire VRE increases significantly once the proportion of ICU patients colonized with VRE exceeds 50% (101) or the number days of exposure to a VRE-patient exceeds 15 days (105). A similar effect of colonization pressure has been demonstrated for MRSA in a medical ICU (102). Increasing numbers of infections with MDROs also have been reported in non-ICU areas of hospitals (97).

There is ample epidemiologic evidence to suggest that MDROs are carried from one person to another via the hands of HCP (106-109). Hands are easily contaminated during the process of care-giving or from contact with environmental surfaces in close proximity to the patient (110-113). The latter is especially important when patients have diarrhea and the reservoir of the MDRO is the gastrointestinal tract (114-117). Without adherence to published recommendations for hand hygiene and glove use (111) HCP are more likely to transmit MDROs to patients. Thus, strategies to increase and monitor adherence are important components of MDRO control programs (106, 118).

Opportunities for transmission of MDROs beyond the acute care hospital results from patients receiving care at multiple healthcare facilities and moving between acute-care, ambulatory and/or chronic care, and LTC environments. System-wide surveillance at LDS Hospital in Salt Lake City, Utah, monitored patients identified as being infected or colonized with MRSA or VRE, and found that those patients subsequently received inpatient or outpatient care at as many as 62 different healthcare facilities in that system during a 5-year span (119).

Role of colonized HCP in MDRO transmission

Rarely, HCP may introduce an MDRO into a patient care unit (120-123). Occasionally, HCP can become persistently colonized with an MDRO, but these HCP have a limited role in transmission, unless other factors are present. Additional factors that can facilitate transmission, include chronic sinusitis (120), upper respiratory infection (123), and dermatitis (124).

Implications of community-associated MRSA (CA-MRSA)

The emergence of new epidemic strains of MRSA in the community, among patients without established MRSA risk factors, may present new challenges to MRSA control in healthcare settings (125-128).

Historically, genetic analyses of MRSA isolated from patients in hospitals worldwide revealed that a relatively small number of MRSA strains have unique qualities that facilitate their transmission from patient to patient within healthcare facilities over wide geographic areas, explaining the dramatic increases in HAIs caused by MRSA in the 1980s and early 1990s (129). To date, most MRSA strains isolated from patients with CA-MRSA infections have been microbiologically distinct from those endemic in healthcare settings, suggesting that some of these strains may have arisin *de novo* in the community via acquisition of methicillin resistance genes by established methicillin-susceptible *S. aureus (*MSSA) strains (130-132). Two pulsed-field types, termed USA300 and USA400 according to a typing scheme established at CDC, have accounted for the majority of CA-MRSA infections characterized in the United States, whereas pulsed-field types USA100 and USA200 are the predominant genotypes endemic in healthcare settings (133).

USA300 and USA400 genotypes almost always carry type IV of the staphylococcal chromosomal cassette (SCC) *mec*, the mobile genetic element that carries the *mec*A methicillin-resistance gene (133, 134). This genetic cassette is smaller than types I through III, the types typically found in healthcare associated MRSA strains, and is hypothesized to be more easily transferable between *S. aureus* strains.

CA-MRSA infection presents most commonly as relatively minor skin and soft tissue infections, but severe invasive disease, including necrotizing pneumonia, necrotizing fasciitis, severe osteomyelitis, and a sepsis syndrome with increased mortality have also been described in children and adults (134-136).

Transmission within hospitals of MRSA strains first described in the community (e.g. USA300 and USA400) are being reported with increasing frequency (137-140). Changing resistance patterns of MRSA in ICUs in the NNIS system from 1992 to 2003 provide additional evidence that the new epidemic MRSA strains are becoming established healthcare-associated as well as community pathogens (90). Infections with these strains have most commonly presented as skin disease in community settings. However, intrinsic virulence characteristics of the organisms can result in clinical manifestations similar to or potentially more severe than traditional healthcare-associated MRSA infections among hospitalized patients. The prevalence of MRSA colonization and infection in the surrounding community may therefore affect the selection of strategies for MRSA control in healthcare settings.

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