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Emerging Community-Acquired Methicillin-Resistant Staphylococcus Aureus Pneumonia

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Abstract

Background: Methicillin-resistant Staphylococcus aureus (MRSA) has been an important nosocomial pathogen worldwide for more than four decades. Community-acquired MRSA infections, generally occurring in previously healthy persons without recognizable risk factors for health care setting-related MRSA, are emerging as serious clinical and public health concerns. The most frequent of these community-based infections include skin and soft tissue infections and necrotizing pneumonias. A majority of causative community-acquired MRSA (CA-MRSA) isolates are associated with genes that encode the virulence factor, Panton-Valentine leukocidin (PVL) toxin.

Aims & Objectives: To describe six cases of CA-MRSA pneumonia recently admitted to our community hospital in Florida, and discuss the epidemiology, clinical features, and management of these expanding infections.

Methods/Study Design: The medical records of six patients with radiographically-confirmed pneumonia and positive sputum cultures for MRSA at the time of hospitalization at the Lawnwood Regional Medical Center and Heart Institute, Fort Pierce, Florida, from December 2006 through January 2007, were retrospectively reviewed. All patients were seen by one of the authors (DO), an infectious diseases consultant. Lawnwood Regional Medical Center is a 341-bed, acute care institution and regional referral center for four counties of Treasure Coast, FL. The hospital institution review board gave permission for this study.

Results/Findings: Six patients (5 men, 1 woman) with CA-MRSA pneumonia were identified. The mean patient age was 57 years (range, 32-79 years). Three patients had no history of previous hospital admission, while two patients had been last hospitalized two years prior to the study admission. Three elderly patients had known co-morbidities predisposing to pneumonia including carcinoma of the lung (2 patients), and cirrhosis, diabetes mellitus, chronic renal failure, COPD, and cardiomyopathy (1 patient each). Sputum samples were collected at the time of admission and all grew MRSA. Two isolates were resistant only to oxacillin, while four were also resistant to levofloxacin (3 isolates), erythromycin (2 isolates), ciprofloxacin (1 isolate), and/or clindamycin (1 isolate). One patient had concurrent Pseudomonas bacteremia, and another had Pseudomonas isolated from

sputum culture in addition to MRSA. All patients had abnormal chest radiographs; three had focal unilateral pneumonia, two had bilateral pneumonia, and one had a lung abscess. The latter patient also had evidence of metastatic infection with sternoclavicular osteomyelitis. Three patients required ventilatory support; two of these subjects died and one was discharged to hospice care. None of the six patients had any epidemiologic connection to one another.

Conclusion: CA-MRSA is becoming an increasingly important cause of community-acquired pneumonia in many parts of the world, and in these regions empiric antibiotic treatment guidelines should be reconsidered.

Keywords: Pneumonia, community-acquired, Staphylococcus aureus, MRSA

Introduction

Methicillin-resistant *Staphylococcus aureus* (MRSA) has been an important nosocomial pathogen worldwide for more than four decades. Community-acquired MRSA infections, generally occurring in previously healthy persons without recognizable risk factors for health care setting-related MRSA, are emerging as serious clinical and public health concerns (Bradley, 2005; Drews et al, 2006; Johnston, 1994; Karas et al, 2006; Maltezou & Giamarellou, 2006). The most frequent of these community-based infections include skin and soft tissue infections and necrotizing pneumonias (John & Schreiber, 2006; Rotas et al, 2007). A majority of causative community-acquired MRSA isolates are associated with genes that encode the virulence factor, Panton-Valentine leukocidin (PVL) toxin (Francis et al, 2005; Naas et al, 2005; Wannet et al, 2005). We describe six cases of community-acquired MRSA (CA-MRSA) pneumonia recently admitted to our community hospital in Florida, and discuss the epidemiology, clinical features, and management of these expanding infections.

Patients and Methods

The medical records of six patients with radiologically-confirmed pneumonia and positive sputum cultures for MRSA at the time of hospitalization at the Lawnwood Regional Medical Center and Heart Institute, Fort Pierce, Florida, from December 2006 through January 2007, were retrospectively reviewed. All patients were seen by one of the authors (DO), an infectious diseases consultant. Lawnwood Regional Medical Center is a 341-bed, acute care institution and regional referral center for four counties of Treasure Coast, FL. The hospital institution review board gave permission for this study.

Results

Six patients (5 men, 1 woman) with CA-MRSA pneumonia were identified. The mean patient age was 57 years (range, 32-79 years). Three patients had no history of previous hospital admission, while two patients had been last hospitalized two years prior to the study admission. Three elderly patients had known co-morbidities predisposing to pneumonia including carcinoma of the lung (2 patients), and cirrhosis, diabetes mellitus, chronic renal failure, COPD, and cardiomyopathy (1 patient each). Sputum samples were collected at the time of admisssion and all grew MRSA. Two isolates were resistant only to oxacillin, while four were also resistant to levofloxacin (3 isolates), erythromycin (2 isolates), ciprofloxacin (1 isolate), and/or clindamycin (1 isolate). One patient had concurrent Pseudomonas

bacteremia, and another had Pseudomonas isolated from sputum culture in addition to MRSA. All patients had abnormal chest radiographs; three had focal unilateral pneumonia, two had bilateral pneumonia, and one had a lung abscess. The latter patient also had evidence of metastatic infection with sternoclavicular osteomyelitis. Three patients required ventilatory support; two of these subjects died and one was discharged to hospice care.

Discussion

The incidence of CA-MRSA is increasing in around the world (e.g., in North America, Europe, Australia, Singapore) due to the epidemic spread of several epidemic clonal subtypes of resistant *Staphylococcus aureus* (Dufour et al, 2002; Holmes et al, 2005; Linde et al, 2005; Naas et al, 2005; Wannet et al, 2005; Wijaya et al, 2006). In parts of the United States, CA-MRSA infections currently exceed those caused by their methicillin-susceptible counterparts. Geographically, there are two types of CA-MRSA: one (sequence type ST30) that is worldwide (pandemic) and the other (sequence types, e.g., ST1, ST8 or ST80) that is continent-specific (Otsuka et al, 2006). Current evidence suggests that CA-MRSA strains have arisen from virulent methicillin-susceptible strains, most likely by horizontal transfer of methicillin-resistance genes from coagulase-negative staphylococci to *S. aureus*, and these clones have spread extensively around the globe via person-to-person transmission (Gosbell, 2005; Rice, 2006).

Healthcare facility-acquired MRSA (HA-MRSA) and community-acquired strains of MRSA can be distinguished by molecular fingerprinting and antibiotic susceptibility profiles. CA-MRSA strains are epidemiologically and clonally unrelated to hospital-acquired strains (Palavecino, 2004). Genetic analysis using techniques such as pulse field gel electrophoresis (PFGE) and multi-locus sequence typing (MLST), and antibiograms demonstrate that CA-MRSA isolates are distinct strains emerging de novo from CA-methicillin-susceptible isolates rather than from HA-MRSA isolates eminating from hospital settings (Mongkolrattanothai et al, 2003). They arise from a broader genetic background, and possess differing virulence genes. Community-acquired MRSA isolates possess novel methicillin resistance genetic cassettes (especially the staphylococcal cassette chromosome mec type IV), and are associated with a phage that encodes for the virulence factor, Panton-Valentine leukocidin (PVL) toxin, which is postulated to be at least partly responsible for the increased virulence of CA-MRSA compared to HA-MRSA (Boyle-Vavra S & Daum, 2007; Monecke et al, 2006; Soderquist et al, 2006). PVL toxin creates lytic pores in the cell membrane of neutrophils and induces the release of neutrophil chemotactic factors that promote inflammation and tissue destruction (Kollef & Micek, 2006).

In a nasal culture survey in Taiwan, Huang and coworkers assessed the prevalence of MRSA carriage among contacts of a case of severe CA-MRSA invasive disease (Huang et al, 2004). They identified carriage of MRSA in a substantial proportion (12.7%) of school children without apparent risk factors for MRSA.

Community-acquired MRSA strains differ from nosocomial strains in clinically relevant ways, such as in their propensity to cause a distinct spectrum of frequently bacteremic infections, especially complicated skin and soft tissue and pulmonary infections including cutaneous abscesses, necrotizing fasciitis, severe necrotizing pneumonia, empyema, and septic pulmonary emboli and other metastatic infection (John & Schreiber, 2006; Gerogianni et al, 2006; Miles et al, 2005; Kowalski et al, 2005; Gorak et al, 1999). CA-MRSA infections occur predominantly in previously healthy children and young adults, with outbreaks and severe infections being reported more frequently in children than in adults (John & Schreiber,

2006). CA-MRSA infections are also seen in epidemiologically disparate populations such as iv drug users, homeless persons, prison inmates, football players, wrestlers, native peoples, and others without established risk factors for MRSA (Gilbert et al, 2006).

Pulmonary involvement is commonly observed in patients with invasive community-acquired *S. aureus* infections (Chua & Lee, 2006), and pneumonia has been reported more often in children with CA-MRSA than in those with community-acquired methicillin-susceptible *S. aureus* (CA-MSSA) (Marcinak & Frank, 2003). The presence of genes encoding PVL is highly associated with pulmonary involvement by *S. aureus*. In 2005, Frances et al reported the first North American adults with severe community-onset MRSA pneumonia caused by strains carrying the PVL genes (Francis et al, 2005). In a study of 113 children with community-acquired *S. aureus* infections, Gonzalez et al found that 47 of 70 (67%) patients with CA-MRSA infection had abnormal pulmonary imaging findings compared with 12 of 43 (28%) patients with CA-MSSA infection ($p < 0.001$) (Gonzalez et al, 2005). Pneumonia and/or empyema, and septic emboli were the most common findings, and metastatic pulmonary disease occurred more frequently among patients with osteomyelitis. Severe necrotizing pneumonia was present in 3 children co-infected with influenza and parainfluenza virus. The presence of genes encoding PVL was investigated in 67 MRSA and 36 MSSA isolates. Abnormal chest x-ray findings were observed for 51 of 80 (64%) patients with PVL-positive isolates, compared with 2 of 23 (9%) subjects with PVL-negative isolates ($p < 0.001$). PVL remained independently associated with abnormal chest imaging findings in patients with secondary pneumonia in a multivariate analysis ($p = 0 .03$). In contrast, however, Mishaan and coworkers noted that CA-MSSA isolates were more likely to be associated with invasive infections than were CA-MRSA isolates ($p < 0.01$) (Mishaan et al, 2005).

Like MSSA, CA-MRSA can be an important and virulent cause of post-viral pneumonia. Hageman et al reported 17 cases of *S aureus* community-acquired pneumonia from 9 states during the 2003-04 influenza season, of which 15 (88%) were methicillin-resistant (Hageman et al, 2006). The median patient age was 21 years; 5 (29%) had underlying diseases, and only 4 (24%) had risk factors for MRSA. All but one patient, who died on arrival, were hospitalized. Death occurred in 5 (4 with MRSA). PVL toxin genes were detected in all isolates; 11 (85%) had only genes for PVL. All isolates had community-associated PFGE patterns, and all MRSA isolates had the staphylococcal cassette chromosome mec type Iva. Due to the presence of PVL and other potent virulence factory, CA-MRSA infections are often life-threatening and may be more serious than HA-MRSA with higher rates of morbidity and mortality.

Unlike typical multi-resistant HA-MRSA, for which vancomycin is the drug of choice, CA-MRSA are usually pan-susceptible to non-beta-lactam antimicrobials, although they are usually not susceptible to macrolides (Rice, 2006). The best antibiotic treatment for PVL-positive CAMRSA is unknown. Inexpensive oral agents for treatment of localized, CA-MRSA infection include clindamycin, trimethoprim-sulfamethoxazole, and newer tetracyclines. Clindamycin has been used successfully to treat soft-tissue and musculoskeletal infections and pneumonia due to MRSA in adults and children. However, concern over the possibility of emergence of inducible clindamycin resistance during therapy is a potential limitation (Francis et al, 2005; Frank et al, 2002; Lewis & Jorgensen, 2005; Marcinak & Frank, 2003).

Simple laboratory testing (e.g., the erythromycin-clindamycin "D-zone" test) can separate strains that have the genetic potential (i.e., via the presence of erm genes) to become resistant

during therapy from strains that are fully susceptible to clindamycin. Martinez-Aguilar et al compared clindamycin treatment of invasive infections caused by CA-MRSA (*n* = 46) and CA-MSSA *(n* = 53) in children, and found that clindamycin was effective in treating invasive infections caused by susceptible CA-MRSA isolates (Martinez-Aguilar et al, 2003).

Clindamycin appears to be more reliably effective in CA-MRSA skin infection, compared to patients receiving the drug for pneumonia. Trimethoprim-sulfamethoxazole may be effective in treating CA-MRSA cutaneous infections, but there is insufficient data to recommend its use for pulmonary or other serious invasive infections due to CA-MRSA (Marcinak & Frank, 2003). The appropriate role of newer antibiotics such as quinupristine-dalfoprinstine, linezolid, daptomycin, tigecycline in the management of CA-MRSA is not clear (Peppard & Weigelt, 2006).

Delay in starting appropriate antibiotic therapy for severe infections caused by MRSA can be life-threatening. Presently, there is a need for reconsideration of empiric antistaphylococcal antibiotic selection for seriously ill patients with suspected community-associated *S. aureus* infections. In areas newly-identified to be endemic for MRSA, vancomycin or teicoplanin may need to be incorporated into existing empiric treatment guidelines for community-acquired pneumonia (including post-influenza infection) that already include a 3rd-generation cephalosporin and either a macrolide or a respiratory fluoroquinolonne (Levison & Fung, 2006).

Conclusion

In summary, molecular biology has contributed to a better epidemiologic and clinical understanding of MRSA infections. Clinicians should be aware of the emergence of community-acquired MRSA as an important cause of serious infections, including pneumonia, arising in the community setting. Appropriate antibiotic therapy should be initiated as soon as infection when this pathogen is suspected.

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Table 1: Clinical and laboratory features of six patients with community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) pneumonia.

Patient No.	Age (yrs)	Sex	Underlying disease(s)	Radiology	Microbiology	Resistant to	Treatment	Outcome
1	45	M	-	Lung abscess; strernoclavicular osteomyelitis	MRSA (sputum)	Oxacillin Erythromycin Levofloxacin	Linezolid	Recovered
2	44	M	-	Bilateral pneumonia	MRSA (sputum) *Pseudomonas aeruginosa* (sputum)	Oxacillin Erythromycin Ciprofloxacin	Ciprofloxacin plus clindamycin	Recovered
3	32	M	-	Bilateral pneumonia	MRSA (sputum) *Pseudomonas aeruginosa*	Oxacillin	Vancomycin (rash), then tigecycline plus aztreonoam	Died
4	79	M	Lung cancer, cirrhosis	Right-sided pneumonia	MRSA (sputum)	Oxacillin	Vancomycin	Improved, then hospice care
5	68	F	Diabetes, heart failure, chronic renal failure	Pneumonia	MRSA (sputum)	Oxacillin Levofloxacin	Linezolid	Died
6	78	M	COPD, lung cancer	Right-sided pneumonia	MRSA (sputum)	Oxacillin Levofloxacin Clindamycin	Vancomycin	Recovered