

that its prevalence in immunocompromised Thai children (20%) was higher than that previously reported in a healthy Thai population (17.5%) (4) and in those with recurrent abdominal pain (11.3%) (5). The prevalence in children <5 years of age was high compared with that reported from Perez-Perez et al. (17.4% vs. 5%) (4). Although unintentional eradication of *H. pylori* after multiple courses of antimicrobial drugs in such patients could explain the low prevalence in some studies, commonly prescribed antimicrobial drugs without antisecretory agents may be unable to cure the infection.

The major limitations of this preliminary study were the use of different diagnostic methods in the various studies and the lack of healthy controls. Thus, a well-designed case-control study is needed. However, the prevalence of infection with *H. pylori* in the immunocompromised children was high, and these patients appear to be more susceptible to this infection in early life.

**Prakaimuk Nutpho\***  
and **Nuthapong Ukarapol\***

\*Chiang Mai University, Chiang Mai, Thailand

#### References

1. Sabbi T, de Angelis P, Colistro F, Dall'Oglio L, di Abriola GF, Castro M. Efficacy of noninvasive tests in the diagnosis of *Helicobacter pylori* infection in pediatric patients. *Arch Pediatr Adolesc Med*. 2005;159:238-41.
2. Cacciarelli AG, Marano BJ, Gualtieri NM, Zuretti AR, Torres RA, Strapoli AA, et al. Lower *Helicobacter pylori* infection and peptic ulcer disease prevalence in patients with AIDS and suppressed CD4 counts. *Am J Gastroenterol*. 1996;91:1783-4.
3. Matsukawa Y, Itoh T, Nishinarita S, Ohshima T, Horie T, Aizawa S, et al. Low seroprevalence of *Helicobacter pylori* in patients with leukemia [letter]. *Am J Hematol*. 1999;60:253.
4. Perez-Perez GI, Taylor DN, Bodhidatta L, Taylor DN, Bodhidatta L, Wongsrichanalai J, et al. Seroprevalence of *Helicobacter pylori* infections in Thailand. *J Infect Dis*. 1990;161:1237-41.
5. Ukarapol N, Lertprasertsuk N, Wongsawadi L. Recurrent abdominal pain in children: the utility of upper endoscopy and histopathology. *Singapore Med J*. 2004;45:121-4.

Address for correspondence: Nuthapong Ukarapol, Department of Pediatrics, Faculty of Medicine, Chiang Mai University, Chiang Mai, Thailand; fax: 66-53-946-461; email: nukarapo@chiangmai.ac.th

## Community Case of Methicillin-resistant *Staphylococcus aureus* Infection

**To the Editor:** Community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) is an emerging infectious disease worldwide and is increasingly reported in Asia (1). We describe a community case of invasive MRSA infection, which appeared as bacteremia and pneumonia; CA-MRSA was initially suspected, and eventually the patient was treated successfully with ampicillin/sulbactam.

A 52-year-old man with chronic eczema was admitted to the Prince of Wales Hospital, Hong Kong, with fever and chills. Before admission, he had been treated for infected eczematous lesions for several weeks with oral ampicillin, cloxacillin, and cefazolin. He had no history of hospitalization in the past 10 years, and none of his family members were health-care workers. Examination showed an oral temperature of 40°C, blood pressure 95/55 mm Hg, and no audible murmur. Cellulitis in the left leg complicated his eczematous skin lesions. Chest radiograph showed right-middle-zone pneumonia. Neutrophilia

(leukocytes  $15.5 \times 10^9/L$ , neutrophils 86%), thrombocytopenia (platelets  $55 \times 10^9/L$ ), prolonged activated partial thromboplastin time (43.6 s), and elevated bilirubin level (31  $\mu\text{mol/L}$ ) were observed. Two initial blood cultures grew gram-positive cocci in clusters, identified as *S. aureus* by positive results for catalase and slide/tube coagulase and a negative result for ornithine decarboxylase. Intravenous cloxacillin (2 g every 6 h) was given on days 2-5. Antimicrobial drug susceptibility testing was performed by the disk-diffusion method (1  $\mu\text{g}$  oxacillin/disk, Mueller-Hinton agar, 2% NaCl), followed by MIC determination with the agar dilution method in accordance with NCCLS (former National Committee for Clinical Laboratory Standards, now Clinical and Laboratory Standards Institute) recommendations (2). One blood isolate was identified as methicillin-resistant *S. aureus* (MRSA), with an oxacillin MIC 4  $\mu\text{g/mL}$ . The other isolate was identified as methicillin-sensitive *S. aureus* (MSSA), with an oxacillin MIC of 0.5  $\mu\text{g/mL}$ . In view of a possible CA-MRSA infection (which could have been  $\beta$ -lactam-resistant), cloxacillin was substituted with intravenous vancomycin plus rifampin on day 5.

However, the patient's condition progressively deteriorated from day 2 to day 10 with persistent fever, chills, hypotension, and hemoptysis. A repeated chest radiograph showed small lung cavities with fluid, and a thoracic computed tomographic scan confirmed multiple lung abscesses. Results of an initial transthoracic echocardiograph were normal, but a subsequent transesophageal echocardiograph demonstrated tricuspid valve vegetation.

The MRSA isolate was susceptible to gentamicin, cotrimoxazole, erythromycin, ciprofloxacin, clindamycin, fusidic acid, tetracycline, chloramphenicol, vancomycin, and rifampin; a different pattern of multidrug-resistant

Table. Comparison between methicillin-sensitive and methicillin-resistant strains of *Staphylococcus aureus*

	Strain and major resistance mechanism*			
	MSSA/penicillinase production	BORSA/novel methicillinase ± penicillinase hyperproduction	CA-MRSA/PBP alteration	HA-MRSA/PBP alteration
PBP2a detection (e.g., latex-agglutination method)	–	–	+	+
<i>mecA</i> gene detection (e.g., PCR method)	–	–	+ (SCC <i>mec</i> IVa)	+
PVL gene detection (PCR method)	Infrequent (<5%)	Data limited	Frequent (>66%–100%)	Infrequent (<5%)
Coresistance to non-β-lactam antimicrobial drugs	±	±	+	+++
Usual antimicrobial drugs to which MSSA is susceptible	PRP (e.g., cloxacillin), β-lactam/β-lactamase-inhibitor combinations (e.g., ampicillin/sulbactam); linezolid, vancomycin, erythromycin, clindamycin, trimethoprim-sulfamethoxazole, fluoroquinolones, rifampin, gentamicin, fusidic acid, tetracyclines	PRP (e.g., cloxacillin), β-lactam/β-lactamase-inhibitor combinations (e.g., ampicillin/sulbactam), other drugs to which MSSA is potentially susceptible	Vancomycin, linezolid, rifampin, gentamicin, trimethoprim-sulfamethoxazole, fusidic acid, tetracyclines, fluoroquinolone, clindamycin†	Vancomycin, linezolid; ± fusidic acid, rifampin, gentamicin, trimethoprim-sulfamethoxazole, fluoroquinolones‡

\*MSSA, methicillin-susceptible *Staphylococcus aureus*; BORSA, borderline oxacillin-resistant *S. aureus*; MRSA, methicillin-resistant *S. aureus*; CA-MRSA, community-associated MRSA; HA-MRSA, hospital-associated MRSA; PBP, penicillin-binding protein; PCR, polymerase chain reaction; PVL, Panton-Valentine leukocidin; PRP, penicillinase-resistant penicillins; +, positive; –, negative. ±, occasionally present; +++, usually present.  
†Concern over inducible clindamycin resistance; also, macrolide resistance is common.  
‡Fluoroquinolone resistance increasing.

MRSA isolates from that usually found in our facility (2,3). The isolate was also susceptible to ampicillin/sulbactam, with an equivalent breakpoint MIC <8/4 µg/mL by disk testing (2). Latex detection for PBP2a (Slidex MRSA-Detection, bioMérieux, Marcy l'Etoile, France) and polymerase chain reaction (PCR) detection for *mecA* were both negative, predicting nonresistance to oxacillin (2–5). A nitrocefin-disk test was positive for β-lactamases, and a 4-fold reduction in MIC was demonstrated in the presence of sulbactam (6). Panton-Valentine leukocidin (PVL) gene locus was not detected (1). Community-acquired BORSA (borderline oxacillin-resistant *S. aureus*), infective endocarditis, and lung abscesses were diagnosed. Intravenous ampicillin/sulbactam (3 g every 6 h) was given on day 10 with rifampin; vancomycin treatment was stopped. Defervescence occurred 3 days later, subsequent blood cultures became sterile, and radiographic changes gradually resolved. Ampicillin/sulbactam was given for 6 weeks without complication.

As this case suggests, BORSA can sometimes be confused with CA-MRSA because of similar clinical signs and symptoms and overlapping oxacillin MICs (2–8 µg/mL and 4–64 µg/mL, respectively) (1,4,6). Both pathogens can appear as community-acquired infections and may be related to previous antimicrobial drug usage (6,7). Although CA-MRSA has been associated with soft tissue infections and necrotizing pneumonia (7,8), MSSA or BORSA strains can also cause these diseases. Thus, in view of potentially different treatment options, when MRSA isolates (e.g., oxacillin MICs ≥4–8 µg/mL) are associated with community-acquired, serious infections (e.g., blood isolates) and are not multidrug resistant, one can consider *mecA* (or PBP2a) testing to delineate the resistance mechanism (Table). If *mecA* is present, further testing for PVL gene locus with or without staphylococcal chromosomal cassette *mec* (SCC*mec*)

type IV can be performed to diagnose CA-MRSA; if *mecA* is not detected, further testing for BORSA may be indicated, and β-lactam therapy should be evaluated individually. If these pathogens are not differentiated and all are treated as CA-MRSA, a non-β-lactam antimicrobial drug, such as vancomycin, will be used (1,4,7,8). However, for serious and deep-seated *S. aureus* infections (e.g., bacteremia, endocarditis), vancomycin is inferior to β-lactam antimicrobial drugs, even when in vitro testing indicates susceptibility. Treatment failures have been encountered (4). Linezolid is a good alternative but limited by availability and cost, and clindamycin therapy can be associated with inducible resistance. For BORSA-associated infections, β-lactam antimicrobial drugs, including high-dose penicillinase-resistant penicillins (PRPs) (e.g., cloxacillin) or β-lactam/β-lactamase-inhibitor combinations (e.g., ampicillin/sulbactam) are regarded as treatments of choice (4,6,9).

type IV can be performed to diagnose CA-MRSA; if *mecA* is not detected, further testing for BORSA may be indicated, and β-lactam therapy should be evaluated individually. If these pathogens are not differentiated and all are treated as CA-MRSA, a non-β-lactam antimicrobial drug, such as vancomycin, will be used (1,4,7,8). However, for serious and deep-seated *S. aureus* infections (e.g., bacteremia, endocarditis), vancomycin is inferior to β-lactam antimicrobial drugs, even when in vitro testing indicates susceptibility. Treatment failures have been encountered (4). Linezolid is a good alternative but limited by availability and cost, and clindamycin therapy can be associated with inducible resistance. For BORSA-associated infections, β-lactam antimicrobial drugs, including high-dose penicillinase-resistant penicillins (PRPs) (e.g., cloxacillin) or β-lactam/β-lactamase-inhibitor combinations (e.g., ampicillin/sulbactam) are regarded as treatments of choice (4,6,9).

BORSA initially described non-heteroresistant strains of *S. aureus* with oxacillin MIC  $\leq 2$  mg/L, which produce ample  $\beta$ -lactamases and are rendered fully susceptible to PRP by  $\beta$ -lactamase-inhibitors (4,6). Subsequent BORSA strains described have had higher oxacillin MICs (4–8 mg/L) (4). The proportion of BORSA among clinical isolates of *S. aureus* varies (1.4%–12.5%) but is usually  $\geq 5\%$  (4,10). A BORSA infection outbreak among dermatology patients with severe skin diseases has also been reported (10). Postulated resistance mechanisms include overproduction of conventional penicillinases, production of an inducible, plasmid-mediated, membrane-bound methicillinase, and in some cases, point mutations of penicillin-binding-proteins (4). The clinical importance of BORSA is unknown since early clinical/animal data suggest treatment efficacy of PRP (against strains with MIC  $\leq 2$  mg/L) (4,6,9). Whether BORSA with higher oxacillin MICs (4–8 mg/L) will respond equally well to PRP is less clear. Further studies into the treatment of BORSA, including pharmacokinetic considerations, are needed (4). However, high-dose  $\beta$ -lactam/ $\beta$ -lactamase inhibitor combinations (e.g., ampicillin/sulbactam), as shown in animal models, are at least as effective as PRP (9). In conclusion, our report suggests that *mecA* (or PBP2a) detection may help manage serious, community-acquired, non-multidrug-resistant MRSA infections because of the potential confusion between BORSA and CA-MRSA.

Lee Nelson,\* Clive S. Cockram,\*  
Grace Lui,\* Rebecca Lam,\*  
Edman Lam,\* Raymond Lai,\*  
and Margaret Ip\*

\*Prince of Wales Hospital, Chinese University of Hong Kong, Hong Kong SAR, People's Republic of China

## References

- Vandenesch F, Naimi T, Enright MC, Lina G, Nimmo GR, Heffernan H, et al. Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. *Emerg Infect Dis*. 2003;9:978–84.
- National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial susceptibility testing. Fifteenth informational supplement. NCCLS document M100-S15. Wayne (PA): The Committee; 2005.
- Ip M, Lyon DJ, Chio F, Enright MC, Cheng AF. Characterization of isolates of methicillin-resistant *Staphylococcus aureus* from Hong Kong by phage typing, pulsed-field gel electrophoresis, and fluorescent amplified-fragment length polymorphism analysis. *J Clin Microbiol*. 2003;41:4980–5.
- Chambers HF. Methicillin resistance in staphylococci: molecular and biochemical basis and clinical implications. *Clin Microbiol Rev*. 1997;10:781–91.
- Brown DF. Detection of methicillin/oxacillin resistance in staphylococci. *J Antimicrob Chemother*. 2001;48(Suppl 1):65–70.
- Varaldo PE. The “borderline methicillin-susceptible” *Staphylococcus aureus*. *J Antimicrob Chemother*. 1993;31:1–4.
- Naimi TS, LeDell KH, Como-Sabetti K, Borchardt SM, Boxrud DJ, Etienne J, et al. Comparison of community- and health care-associated methicillin-resistant *Staphylococcus aureus* infection. *JAMA*. 2003;290:2976–84.
- Francis JS, Doherty MC, Lopatin U, Johnson CP, Sinha G, Ross T, et al. Severe community-onset pneumonia in healthy adults caused by methicillin-resistant *Staphylococcus aureus* carrying the Panton-Valentine leukocidin gene. *Clin Infect Dis*. 2005;40:100–7.
- Hirano L, Bayer AS. Beta-lactam-beta-lactamase-inhibitor combinations are active in experimental endocarditis caused by beta-lactamase-producing oxacillin-resistant staphylococci. *Antimicrob Agents Chemother*. 1991;35:685–90.
- Balslev U, Bremmelgaard A, Svejgaard E, Havstrem J, Westh H. An outbreak of borderline oxacillin-resistant *Staphylococcus aureus* (BORSA) in a dermatological unit. *Microb Drug Resist*. 2005;11:78–81.

Address for correspondence: Margaret Ip, Department of Microbiology, Prince of Wales Hospital, Chinese University of Hong Kong, Ngan Shing St, Hong Kong SAR, People's Republic of China; fax: 852-2647-3227; email: margaretip@cuhk.edu.hk

## *Rickettsia massiliae* Human Isolation

**To the Editor:** The number of new rickettsial species that cause diseases in humans is rapidly increasing (1). Moreover, many of the species first described in ticks have been recently shown to be pathogenic. Of the 10 species or subspecies found to be pathogens after 1984, a total of 7 were first isolated from ticks (2). We report the first isolation of *Rickettsia massiliae* from a patient. The bacterium was isolated in Sicily in 1985 and identified in 2005.

A 45-year-old man was hospitalized in Palermo, Italy, on June 6, 1985, for fever and a rash. He had been febrile since May 25 and did not respond to antimicrobial drug treatment using cefamezin, a first-generation cephalosporin. On examination, he had a necrotic eschar on his right ankle, a maculopapular rash on his palms and soles (online Appendix Figure 1, available at <http://www.cdc.gov/ncidod/EID/vol12no01/05-0850-G1.htm>), and slight hepatomegaly. Leukocyte count was normal; he received tetracyclines for 13 days and fully recovered. He seroconverted (from 0 to 1:80 between day 11 and day 24) by indirect immunofluorescence to *Rickettsia conorii* (*R. conorii* spot, bioMérieux, Marcy l'Étoile, France).

Four milliliters of heparinized blood sampled before treatment were inoculated in a 25-cm<sup>2</sup> flask containing Vero cells and incubated at 33°C in a CO<sub>2</sub> incubator (1). Direct immunofluorescence test on a sample of the patient's serum was positive 7 days later. The strain was stored for 20 years and tested in 2005 at the Unité des Rickettsies for identification, and *R. massiliae* was identified. DNA was extracted from the cell culture supernatant and used as template in 2 previously described polymerase