ORIGINAL ARTICLE

Molecular Characterization of Methicillin-Resistant *Staphylococcus aureus* Spread by Neonates Transferred From Primary Obstetrics Clinics to a Tertiary Care Hospital in Korea

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OBJECTIVE. To investigate the characteristics and origins of methicillin-resistant *Staphylococcus aureus* (MRSA) strains isolated from neonatal patients admitted to a tertiary care hospital from local and primary care obstetrics clinics.

DESIGN. Molecular typing study.

SETTING. A 1,278-bed tertiary care hospital (Samsung Medical Center) and 2 primary obstetrics clinics in Seoul, Korea.

PATIENTS. The genotypic characteristics of 12 MRSA samples isolated from 11 neonatal patients transferred from 2 primary care obstetrics clinics to a tertiary care hospital were investigated by means of multilocus sequence typing, *spa* (staphylococcal protein A) typing, and SCC*mec* typing. Ten MRSA strains isolated from workers and environments in the associated obstetrics clinics were also investigated.

RESULTS. Although the antibiograms of isolates from 2 obstetrics clinics differed, no strain showed multidrug resistance to antimicrobials. Multilocus sequence typing analysis showed that all 22 MRSA isolates analyzed in this study had sequence type 1 (with the allelic profile 1-1-1-1-1-1-1), sequence type 493 (62-1-1-1-1-1-1), or a novel sequence type (25-1-1-1-1-1-1) and that all belonged to a single clonal complex (clonal complex 1). Moreover, they all contained SCC*mec* type IVA and the identical *spa* type (UJEBKBP). These genotypic characteristics are similar to those of typical community-associated MRSA strains rather than the hospital-acquired MRSA strains common in Korea.

CONCLUSION. The findings of this study suggest that community-acquired MRSA strains can spread in primary care clinics and be imported into tertiary care settings.

Infect Control Hosp Epidemiol 2006; 27:593-597

Methicillin-resistant *Staphylococcus aureus* (MRSA) is usually viewed as a major nosocomial pathogen that is associated with hospitals and healthcare facilities. However, recently, community-associated MRSA (CA-MRSA) infections have been increasingly reported. Traditionally, CA-MRSA isolates have been differentiated from healthcare-associated MRSA (HA-MRSA) isolates on the basis of the time of onset of infection after admission to a hospital and the presence of risk factors such as recent hospitalization, admission from another hospital, nursing home residence, injection drug abuse, previous antimicrobial treatment, and underlying illness (eg, cardiovascular and pulmonary disease, diabetes, malignancy, or chronic skin disease).[1-9]

CA-MRSA strains are also genotypically and phenotypically distinct from HA-MRSA strains. Unlike multidrug-resistant HA-MRSA strains, CA-MRSA strains are often susceptible to most antimicrobials, except β-lactam agents.[3,10] The presence of SCC*mec* type IV is also a characteristic of CA-MRSA strains.[3] In addition, CA-MRSA strains have diverse sequence types, as determined by multilocus sequence typing (MLST), that are distinct from those of HA-MRSA strains.[8,11] Pulsed-field gel electrophoresis has also confirmed the distinctiveness of CA-MRSA strains.[12]

In 2004, we isolated several MRSA strains from neonatal patients transferred from primary obstetrics clinics to a tertiary care hospital. In the present study, we characterized these MRSA strains from neonates with several molecular typing methods: MLST, SCC*mec* typing, *spa* (staphylococcal protein A) typing, and in vitro antimicrobial susceptibility testing. To elucidate the origin of the MRSA strains isolated from the neonatal patients, we also isolated and investigated MRSA strains obtained from healthcare workers and the primary obstetrics clinic environments from which neonatal patients were transferred.

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Received July 22, 2005; accepted October 12, 2005; electronically published May 18, 2006.

METHODS

Study Sites and Patients

Neonatal patients were transferred from 2 primary care obstetrics clinics in Seoul, Korea, to our 1,278-bed tertiary care hospital (Samsung Medical Center, Seoul, Korea), where MRSA samples were isolated.

Bacterial Isolates

Screening swab samples of the anterior nostrils and skin of all the neonatal patients transferred from other hospitals or clinics were obtained and cultured for MRSA at admission as a routine procedure to prevent influx of MRSA to our unit. We investigated 11 neonatal patients transferred from 2 obstetrics clinics during March to June 2004 and 12 MRSA strains isolated from these patients (6 isolates from each clinic). The 2 obstetrics clinics (A and B) are representative of other clinics in Seoul, Korea, in terms of size. During the study period, 12 and 11 neonatal patients were transferred from clinics A and B, respectively.

The MRSA strains were isolated from 9 (75%) of 12 neonatal patients from clinic A and from 7 (64%) of 11 neonatal patients from clinic B. Of the 9 patients from clinic A with MRSA isolated, 2 (22%) were born vaginally; 4 (57%) of the 7 patients from clinic B were born vaginally. The others were born by cesarean section. The patients were transferred to the neonatal intensive care unit in our hospital immediately after birth. Two MRSA strains were isolated from the nostrils and skin of a neonatal patient transferred from obstetrics clinic A. We screened the hands and nostrils of all the healthcare workers (36 from clinic A and 25 from clinic B) and the frequently contaminated environments of 2 clinics for MRSA carriage or colonization by obtaining swab specimens with wet cotton-tipped swabs. This screening was performed one day after the isolation from the neonates of the MRSA strains included in this study. A total of 10 MRSA isolates were collected from the hands and nostrils of workers and from the obstetrics clinic environment. Of these, 1 isolate was from a nasal swab specimen obtained from a nurse working in clinic A, and 9 isolates were from a physician, nurses, and the environment of clinic B (Table). In both clinic A and clinic B, workers whose MRSA clones were shared with an infant had cared for that infant. Cultures of isolates were done with blood agar plates, and identification of MRSA was performed with the Vitek 2 system (bioMérieux Vitek).

Molecular Typing

Chromosomal DNA for MLST, for multiplex polymerase chain reaction (PCR) SCC*mec* typing, and for *spa* typing was extracted by a simple boiling-lysis method. MLST was performed as described elsewhere.[13,14] The allelic profiles of strains were defined by alleles at 7 MLST loci, in the following order: *arcC, aroE, glpF, gmk, pta, tpi,* and *yqiL*. Each unique allelic profile was designated as an sequence type, which was determined by comparison with the database at the MLST Web site (http://saureus.mlst.net). A sequence type not found in the database was submitted to the MLST Web site. The SCC*mec* types were determined by multiplex PCR as described elsewhere.[15] Each isolate also underwent *spa* typing, as described elsewhere.[16,17] The *spa* gene was amplified using the primers SpaF (5′-GAC GAT CCT TCA GTG AGC AAA G-3′) and SpaR (5′-GCA GCA ATT TTG TCA GCA GTA G-3′). Amplified *spa* gene fragments were purified and sequenced. Each repeat sequence differing from others by least 1 point mutation is designated with alphabetical letters,[16] as described on the *spa* typing Web site (http://www.ridom.de/spaserver).[18] MRSA strain sequences published previously were used as controls.[14] The presence of the *pvl* gene was determined by PCR, as described elsewhere.[19]

Antimicrobial Resistance Testing

In vitro susceptibility testing was performed using the broth microdilution method according to guidelines issued by the Clinical Laboratory Standards Institute.[20] Twelve antimicrobial agents were tested: oxacillin, penicillin, ciprofloxacin, clindamycin, erythromycin, gentamicin, nitrofurantoin, rifampin, tetracycline, trimethoprim-sulfamethoxazole, vancomycin, and teicoplanin. Interpretive criteria for susceptibility were those indicated by the Clinical Laboratory Standards Institute.[20] *S. aureus* ATCC 49619 was used as a control strain in each set of tests.

RESULTS

The Table summarizes the sources, the molecular typing results, and the antimicrobial susceptibilities of the 22 MRSA isolates analyzed in this study. Eleven neonatal patients between 1 and 13 days of age were transferred from primary obstetrics clinics to our hospital. The MRSA isolates were cultured 1 or 2 days after transfer. The patients received diagnoses of hyperbilirubinemia, wet lung syndrome, hemophilia, neonatal sepsis, feeding intolerance, hypoxemia, or gastroenteritis. However, none of the patients had clinical illness secondary to *S. aureus* infection.

Of 6 MRSA strains from neonates from obstetrics clinic A, 5 strains had sequence type (ST) 1 (with the allelic profile 1-1-1-1-1-1-1) and 1 strain (A356N) had a novel sequence type (with the allelic profile 25-1-1-1-1-1-1), which is a single-locus variant of ST1. Although 2 MRSA strains (A356N and A357N) were isolated from the hand and nostrils of one patient, they had different sequence types. Only 1 MRSA strain (A-01) was isolated from a nurse at obstetrics clinic A, and it had the same sequence type (a single-locus variant of ST1) as A356N. Seven MRSA strains associated with obstetrics clinic A produced the same antibiogram: they were resistant to only gentamicin and tetracycline, in addition to oxacillin and penicillin.

Of the 6 MRSA strains from neonates from obstetrics clinic B, 4 had ST1 and the other 2 showed ST493 (with the allelic

TABLE. Characteristics of the Methicillin-Resistant *Staphylococcus aureus* Strains Analyzed in This Study

Clinic, strain[a]	Source specimen (personnel)	Infected subject's underlying disease or condition	Sequence type (allelic profile)	SCC*mec* type	*spa* type	Antimicrobial resistance pattern[b]
Clinic A						
A216N	Unknown	Feeding intolerance	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A356N	Hand	Feeding hypoxemia	New (25-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A357N	Nares	Feeding hypoxemia	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A372N	Unknown	Hypoxemia	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A413N	Hand	Feeding intolerance	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A555N	Nares	Acute gastroenteritis	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
A-01	Nares (nurse)	…	New (25-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Tet
Clinic B						
B006N	Nares	Hyperbilirubinemia	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Ery, Tet
B140N	Unknown	Wet lung syndrome	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B273N	Hand	Hemophilia	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B295N	Unknown	Neonatal seizure	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B464N	Hand	Neonatal sepsis	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B570N	Hand	Neonatal sepsis	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-01	Nares (doctor)	…	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-02	Nares (nurse)	…	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-03	Nares (nurse)	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery, TMP-SMX
B-04	Hand (nurse)	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-05	Nares (nurse)	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery, TMP-SMX
B-06	Hand (nurse)	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-07	Table	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-08	Sink	…	ST1 (1-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery
B-09	Scale	…	ST493 (62-1-1-1-1-1-1)	IVA	UJEBKBPE	Gen, Ery

NOTE. Ery, erythromycin; Gen, gentamicin; *spa,* staphylococcal protein A; Tet, tetracycline; TMP-SMX, trimethoprim-sulfamethoxazole.

[a] The letter N denotes an MRSA strain isolated from 1 or more neonates.

[b] Strains were resistant to oxacillin and penicillin but susceptible to all other antimicrobials except those listed.

profile 62-1-1-1-1-1-1), which is also a single-locus variant of ST1. These isolates were resistant to only gentamicin and erythromycin, except for 1 isolate (B006N), which was resistant to erythromycin and tetracycline. Of the 9 MRSA strains isolated from a physician, nurses, and the environment of obstetrics clinic B, 3 had ST1 and 6 had ST493. Although 7 strains were resistant to gentamicin and erythromycin, as were MRSA strains from neonates, the remaining 2 strains (B-03 and B-05) were resistant to trimethoprim-sulfamethoxazole, gentamicin, and erythromycin (in addition to oxacillin and penicillin). All 22 MRSA strains had SCC*mec* type IVA and *spa* type 286 (UJEBKBPE) and were negative for the *pvl* gene.

DISCUSSION

In spring 2004, we isolated an increased number of MRSA strains from neonatal patients who were transferred to our hospital from local and primary care obstetrics clinics. In the neonatal intensive care unit, MRSA strains were isolated from 22 (22%) of 102 neonatal patients who were transferred from other hospitals in 2003 and from 51 (36%) of 143 patients in 2004, which was a significant increase ($P = .02$; Fisher exact test). Although most MRSA strains from neonatal patients did not seem to be linked with severe infection, their characteristics and origin are important to infection control in a hospital. Moreover, carriage of MRSA often precedes infection with the same strain.[21] Therefore, an understanding of MRSA transmission is highly relevant to control of infection.

In the present study, the molecular characteristics of 12 MRSA strains isolated from 11 neonatal patients at 2 obstetrics clinics were investigated. The 2 obstetrics clinics were selected as representatives, because MRSA strains were continuously isolated from transferred neonatal patients. To elucidate strain origins, we also isolated and investigated MRSA strains from workers and the environments of the relevant primary obstetrics clinics. Our results show that MRSA strains isolated from neonates may have originated in the obstetrics units where the neonates were born. Before this study was done, the obstetrics clinics were cleaned using house detergent and were occasionally disinfected using 75% alcohol. The infants shared bathtubs in those clinics. The healthcare workers with MRSA carriage in those clinics were treated with topical application of mupirocin ointment. Originally, only soap and water were used for hand hygiene, but an alcohol-based hand rub was introduced after this study. The clinic environment is now regularly disinfected using alkyldiaminoethylglycine (Tego; Th. Goldschmidt) solution.

ST1, which was the main MLST type identified in strains

from neonatal patients transferred from both clinics, is one of the CA-MRSA strain genotypes most frequently found, along with ST8, particularly in North America.[8,11] Although the other 2 sequence types detected, ST493 and a novel sequence type, are single-locus variants of ST1, and thus belong to clonal complex 1, they could not be found in the MLST databases. We previously investigated the molecular characteristics of MRSA strains isolated at teaching hospitals in Asia and at 8 tertiary care hospitals in Korea[14,22] and found no isolates belonging to clonal complex 1 among 74 MRSA strains from 12 Asian countries and only 3 isolates of ST1 and ST514 (with the allelic profile 1-31-1-1-1-1-1) from 1 Korean hospital. Thus, clonal complex 1 is considered a rare clone in Korean hospitals.

Although 9 MRSA strains isolated from neonates at 2 obstetrics clinics showed the same sequence type (ST1), SCC*mec* type (type IVA), and *spa* type (UJEBKBPE), antibiograms were different for the MRSA strains from the 2 clinics. All isolates from clinic A were resistant to gentamicin and tetracycline, but most isolates from clinic B were resistant to gentamicin and erythromycin. Such a difference in the antibiogram indicates that the MRSA strains from the 2 clinics have a different origin, and this may be relevant in clinical settings. In addition, all isolates from neonatal patients included in this study were susceptible to ciprofloxacin and trimethoprim-sulfamethoxazole, which was significantly different from the antibiogram of HA-MRSA strains (average, 38% and 44% of strains resistant, respectively) in our hospital. In addition, the novel sequence type was found only in clinic A, whereas ST493 was exclusively found in clinic B. These findings indicate that the MRSA isolates of neonatal patients from the 2 clinics have different origins.

Controversy exists regarding whether the increased prevalence of CA-MRSA strains truly represents the emergence of a new pathogen or whether it is related to an increased recognition of HA-MRSA in patients with undocumented healthcare contacts. Moreover, it has been reported that many CA-MRSA strains are actually acquired in hospital-associated sites.[23] These MRSA strains from individuals with risk factors show molecular characteristics similar to HA-MRSA strains.[3] However, some CA-MRSA strains have distinct genotypic and antibiogram differences from HA-MRSA, which may be an indicator of true community acquisition.[19] Moreover, these so-called "true CA-MRSA" strains with distinct genotypes are being increasingly reported. Our findings document that strains biologically similar to previously reported CA-MRSA strains are being imported into a neonatal hospital setting from obstetrics clinics. If we applied a strict epidemiologic definition for CA-MRSA, which may exclude patients transferred from other settings, these isolates would be inappropriately classified as HA-MRSA. In actuality, most epidemiologic studies that use such a definition consider newborns to be from the community unless hospitalized for more than 1 week. Molecular methods seem to be more accurate for distinguishing HA-MRSA from CA-MRSA.[11] Actually, the spread of HA-MRSA into the community and the migration of CA-MRSA strains into hospitals have also been observed on several occasions.[24-26] The transmission of CA-MRSA strains into hospitals may be an inevitable result of the presence of a widespread community reservoir,[27,28] and this introduction of CA-MRSA strains into neonatal units presents challenges to hospital infection control systems and treatment modalities. In addition, emergence and/or transmission of CA-MRSA may affect treatment options for outpatients. For example, clindamycin and trimethoprim-sulfamethoxazole would be suitable for many neonatal patients without any concerns, because MRSA strains from transferred neonates were susceptible to such antimicrobials, unlike HA-MRSA strains.

In summary, we investigated the molecular characteristics of MRSA isolates from neonates transferred from primary care obstetrics clinics. Although our study is limited by the fact that maternal vaginal cultures could not be obtained, the isolates showed the same genotypic and phenotypic characteristics as MRSA isolates obtained from healthcare workers and obstetrics clinic environments and were similar to known CA-MRSA. Our findings suggest that true CA-MRSA strains are already present in the community in Korea and that obstetrics clinics with low antibiotic pressure are reservoirs of CA-MRSA strains, even though true CA-MRSA infection has not been reported yet.

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ACKNOWLEDGMENTS

We thank the physicians and nurses at the obstetrics clinics for their assistance with the bacterial isolation.

This study was supported by the Asian-Pacific Research Foundation for Infectious Diseases (ARFID).

REFERENCES

1. Campbell KM, Vaughn AF, Russell KL, et al. Risk factors for community-associated methicillin-resistant *Staphylococcus aureus* infections in an outbreak of disease among military trainees in San Diego, California, in 2002. *J Clin Microbiol* 2004; 42:4050-4053.
2. Chambers HF. The changing epidemiology of *Staphylococcus aureus? Emerg Infect Dis* 2001; 7:178-182.
3. Charlebois ED, Perdreau-Remington F, Kreiswirth B. Origins of community strains of methicillin-resistant *Staphylococcus aures. Clin Infect Dis* 2004; 39:47-54.
4. Cookson BD. Methicillin-resistant *Staphylococcus aureus* in the community: new battlefronts, or are the battles lost? *Infect Control Hosp Epidemiol* 2000; 21:398-403.
5. Dufour P, Gillet Y, Bes M. Community-acquired methicillin-resistant *Staphylococcus aureus* infections in France: emergence of a single clone that produces Panton-Valentine leukocidin. *Clin Infect Dis* 2002; 35:819-824.
6. Eady EA, Cove JH. Staphylococcal resistance revisited: community-acquired methicillin resistant *Staphylococcus aureus:* an emerging problem for the management of skin and soft tissue infections. *Curr Opin Infect Dis* 2003; 16:103-124.
7. Saïd-Salim B, Mathema B, Kreiswirth BN. Community-acquired meth-

icillin-resistant *Staphylococcal aureus*: an emerging pathogen. *Infect Control Hosp Epidemiol* 2003; 24:451-455.
8. Vandenesch F, Naimi T, Enright MC, et al. Community-acquired methicillin-resistant *Staphylococcus aures* carrying Panton-Valentine leukocidin genes: worldwide emergence. *Emerg Infect Dis* 2003; 9:978-984.
9. Crawford SE, Daum RS. Epidemic community-associated methicillin-resistant *Staphylococcus aureus. Pediatr Infect Dis J* 2005; 24:459-450.
10. Hussain FM, Boyle-Vavra S, Bethel CD, Daum RS. Current trends in community-acquired methicillin-resistant *Staphylococcus aureus* at a tertiary care pediatric facility. *Pediatr Infect Dis J* 2000; 19:1163-1166.
11. Okuma K, Iwakawa K, Turnidge JD, et al. Dissemination of new methicillin-resistant *Staphylococcus aureus* clones in the community. *J Clin Microbiol* 2002; 40:4289-4294.
12. McDougal LK, Steward CD, Killgore GE, et al. Pulsed-field gel electrophoresis typing of oxacillin-resistant *Staphylococcus aureus* isolates from the United States: establishing a national database. *J Clin Microbiol* 2003; 41:5113-5120.
13. Enright MC, Day NPJ, Davies CE, Peacock SJ, Spratt BG. Multilocus sequence typing for characterization of methicillin-resistant and methicillin-susceptible clones of *Staphylococcus aureus. J Clin Microbiol* 2000; 38:1008-1015.
14. Ko KS, Lee JY, Suh JY, Oh WS, Peck KR, Lee NY, Song, JH. Distribution of major genotypes of methicillin-resistant *Staphylococcus aureus* clones in Asian countries. *J Clin Microbiol* 2005; 43:421-426.
15. Oliveira DC, de Lencastre H. Multiplex PCR strategy for rapid identification of structural types and variants of the *mec* element in methicillin-resistant *Staphylococcus aureus. Antimicrob Agents Chemother* 2002; 46: 2155-2561.
16. Koreen L, Ramaswamy SV, Graviss EA, Naidich S, Musser JM, Kreiswirth BN. *spa* Typing method for discriminating among *Staphylococcus aureus* isolates: implications for use of a single marker to detect genetic micro- and macrovariation. *J Clin Microbiol* 2004; 42:792-799.
17. Shopsin B, Gomez Z, Montgomery SO, et al. Evaluation of protein A gene polymorphic region DNA sequencing for typing of *Staphylococcus aureus* strains. *J Clin Microbiol* 1999; 37:3556-3563.
18. Harmsen D, Claus H, Witte W, Rothgänger J, Claus H, Turnwald D, Vogel U. Typing of methicillin-resistant *Staphylococcus aureus* in a university hospital setting by using novel software for *spa* repeat determination and database management. *J Clin Microbiol* 2003; 41:5442-5448.
19. Fey PD, Said-Salim B, Pupp ME, et al. Comparative molecular analysis of community- or hospital-acquired methicillin-resistant *Staphylococcus aureus. Antimicrob Agents Chemother* 2003; 47:196-203.
20. Clinical and Laboratory Standards Institute (CLSI). *Performance Standards for Antimicrobial Susceptibility Testing: Fifteenth Informational Supplement.* Wayne, PA: CLSI; 2005. CLSI/NCCLS document M100-S15.
21. von Eiff C, Becker K, Machka K, Stammer H, Peters G. Nasal carriage as a source of *Staphylococcus aureus* bacteremia. *N Engl J Med* 2001; 344: 11-16.
22. Ko KS, Kim YS, Song JH, et al. Genotypic diversity of methicillin-resistant *Staphylococcus aureus* isolates in Korean hospitals. *Antimicrob Agents Chemother* 2005; 49:3583-3585.
23. Tambyah PA, Habib AG, Ng TM, Goh H, Kumarasinghe G. Community-acquired methicillin-resistant *Staphylococcus aureus* infection in Singapore is usually "health-associated." *Infect Control Hosp Epidemiol* 2003; 24:436-438.
24. Eckhardt C, Halvosa JS, Ray SM, Blumberg HM. Transmission of methicillin-resistant *Staphylococcal aureus* in the neonatal intensive care unit from a patient with community-acquired disease. *Infect Control Hosp Epidemiol* 2003; 24:460-461.
25. Healy CM, Hulten KG, Palazzi DL, Campbell JR, Baker CJ. Emergence of new strains of methicillin-resistant *Staphylococcus aureus* in a neonatal intensive care unit. *Clin Infect Dis* 2004; 39:1460-1466.
26. Saiman L, O'Keefe M, Graham PL III, et al. Hospital transmission of community-acquired methicillin-resistant *Staphylococcus aureus* among postpartum women. *Clin Infect Dis* 2003; 37:1313-1319.
27. Bratu S, Eramo A, Koper R, et al. Community-associated methicillin-resistant *Staphylococcus aureus* in hospital nursery and maternity units. *Emerg Infect Dis* 2005; 11:808-813.
28. Carleton HA, Diep BA, Charlebois ED, Sensabaugh GF, Perdreau-Remington F. Community-adapted methicillin-resistant *Staphylococcus aureus* (MRSA): population dynamics of an expanding community reservoir of MRSA. *J Infect Dis* 2004; 180:1730-1738.