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Methicillin-resistant *Staphylococcus aureus* in Nurseries

Denise Bratcher, DO*

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Dr Bratcher did not disclose any financial relationships relevant to this article.

Objectives

After completing this article, readers should be able to:

1. Describe the clinical manifestations of methicillin-resistant *Staphylococcus aureus* (MRSA) infections in neonates.
2. Delineate the role of community-acquired strains of MRSA in the development of sepsis in neonatal intensive care units (NICUs).
3. Describe the route of transmission of MRSA.
4. List the measures necessary to control MRSA in NICUs.

Introduction

Staphylococcus aureus has been recognized as a common nosocomial pathogen in nurseries since devastating outbreaks caused serious infections in the 1950s. Adults are commonly colonized, typically involving the nose and skin, with reported rates of carriage ranging from 30% to 50%. Skin colonization with *S aureus* can occur within 24 to 48 hours of birth from contact with the skin of adults or the environment. *S aureus* colonization of newborns varies from 20% to 90% over the first week after birth, with the most common sites including the anterior nares, umbilicus, and skin (groin and axillae). The probability of staphylococcal colonization increases with prolonged duration of hospitalization. *S aureus* is the second most common pathogen causing late-onset sepsis in very low-birthweight infants in neonatal intensive care units (NICUs).

Since its emergence in the 1960s in the United States, methicillin-resistant *S aureus* (MRSA) has become a common health care-associated pathogen. According to 2001 data from the National Nosocomial Surveillance System, it comprises 55% of *S aureus* strains causing nosocomial infection. Traditional risk factors for MRSA colonization or infection have included contact with the health care system, such as recent hospitalization, surgery, close contact with hospitalized individuals, or prior antimicrobial treatment. Numerous outbreaks of MRSA in NICUs have been described, and MRSA has become a major, sometimes endemic, problem in many NICUs. As complex, prolonged care for infants at the extremes of gestational age and birthweight has become increasingly common, so too has the impact of MRSA in this setting.

Although MRSA traditionally remained almost exclusive to hospitals, long-term care facilities, or other similar institutional settings, since the late 1990s, studies have revealed a significant prevalence of community-acquired MRSA (CA-MRSA) colonization or infections among individuals who have no known risk factors for MRSA acquisition. The prevalence of CA-MRSA acquisition differs among geographic locales, but the increase in CA-MRSA infections has the potential to create reservoirs among family members of hospitalized patients, leading to an increase in the prevalence and risk of nosocomial infections due to MRSA. More recently, these genetically distinct strains have emerged as a significant cause of sepsis in neonates in NICUs and have caused disseminated infection with substantial morbidity and mortality.

Abbreviations

BLR:	beta-lactamase-resistant
CA-MRSA:	community-acquired methicillin-resistant *Staphylococcus aureus*
CLSI:	Clinical Laboratory Standards Institute
MRSA:	methicillin-resistant *Staphylococcus aureus*
NICU:	neonatal intensive care unit
NTED:	neonatal toxic shock-like exanthematous disease
PFGE:	pulsed-field gel electrophoresis
PVL:	Panton-Valentine leukocidin
TSST-I:	toxic shock syndrome toxin type I

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Microbiology

Staphylococcal organisms are nonmotile aerobic or facultatively anaerobic cocci that grow readily in simple microbiologic media. Most strains of *S aureus* produce hemolysis on blood agar within 24 to 36 hours of plating, and many produce a deep yellow or golden pigment. They are typically recognized as gram-positive clusters that are catalase- and coagulase-positive.

S aureus elaborates a wide variety of extracellular toxins responsible for the virulence of the organisms. These include alpha-, beta-, and delta-hemolysins; coagulases; leukocidin; hyaluronidase; staphylokinase; bacteriocins; the epidermolytic toxins; toxic shock syndrome toxin type I (TSST-I); and the enterotoxins. The latter three have specific roles in staphylococcal infection or disease. Epidermolytic toxins A and B cause the various skin manifestations of staphylococcal scalded skin syndrome by cleaving and separating cell layers within the epidermis. TSST-I has been associated with toxic shock syndrome and is responsible for inducing the major physiologic changes of that presentation, often acting as a superantigen. Enterotoxins are heat-stable exotoxins involved in staphylococcal food poisoning and possibly other syndromes. The Panton-Valentine leukocidin (PVL) attacks leukocytes exclusively and kills human polymorphonuclear leukocytes and macrophages. Genes encoding for PVL often are identified among CA-MRSA strains. In general, MRSA does not appear to be more virulent than its methicillin-susceptible counterpart.

Soon after the introduction of semisynthetic beta-lactamase-resistant (BLR) penicillins in the early 1960s, *S aureus* acquired the *mec* regulon, which coded for resistance to methicillin, oxacillin, nafcillin, cloxacillin, dicloxacillin, and the related cephalosporins, creating a pattern of resistance now known as MRSA. The penicillin-binding protein called PBP2a or PBP2′ mediates resistance to the BLR semisynthetic penicillins by a novel cell wall, which has decreased affinity for BLR beta-lactam antimicrobial agents. MRSA tends to accumulate unrelated resistance determinants, leading to the typical multidrug resistance pattern noted among health care-associated strains, which usually are predictably susceptible only to vancomycin. Gentamicin and rifampin, as adjuncts to therapy, also may reflect susceptibility.

CA-MRSA strains generally retain susceptibility to a variety of drugs, including clindamycin, gentamicin, trimethoprim-sulfamethoxazole, and possibly fluoroquinolones. Many CA-MRSA strains also exhibit inducible resistance to macrolides, lincosamides, and streptogramin B (MLS_Bi phenotype). Initial susceptibility reports note resistance to erythromycin but susceptibility to clindamycin. Although isolates appear clindamycin-susceptible, resistance may develop among such strains during therapy. A disk diffusion induction test (D-test) is used to determine clindamycin susceptibility. A distorted "D-shaped" zone of inhibition is observed around the clindamycin disk if an erythromycin disk is placed nearby, indicating inducible resistance. D-testing is now standard in determining staphylococcal susceptibilities. Genetically, CA-MRSA strains also differ in the size of methicillin-resistance staphylococcal cassette chromosome *mec* (*SCCmec*), a unique mobile genetic element integrated into the *S aureus* chromosome that they carry, potentially enhancing their mobility and transferability between strains.

Methicillin resistance in staphylococcal isolates can be detected by the methods recommended by guidelines of the Clinical Laboratory Standards Institute (CLSI), formerly the National Committee for Clinical Laboratory Standards. Interpretative standards for susceptibility testing also are defined by CLSI. *S aureus* isolates are deemed methicillin-resistant if the minimal inhibitory concentration by dilution methods is 4 mcg/mL or greater and susceptible if the concentration is 2 mcg/mL or less. MRSA now is reported commonly based on oxacillin susceptibility; oxacillin resistance implies resistance to all penicillins, including antistaphylococcal penicillins, all cephalosporins, all beta-lactamase inhibitor combinations, all carbapenems, and loracarbef. Newer standards suggest that cefoxitin disk diffusion testing may improve the reliability of routine detection of MRSA.

Pulsed-field gel electrophoresis (PFGE) remains the gold standard for MRSA typing, based on its discriminatory power, reliability, and published criteria for interpretation of the profiles. Molecular typing techniques are invaluable tools for the assessment of potential MRSA outbreaks and should be encouraged in such scenarios. Use of gene probes for the *mecA* gene can identify MRSA isolates. There are currently eight unique pulsed-field type lineages endemic to the United States in the national database.

Clinical Manifestations

The clinical manifestations of MRSA infections among neonates vary widely from simple pustular skin lesions to more invasive disease expression (Table 1). Bloodstream infections due to MRSA are common in NICUs, accounting for up to 90% of all *S aureus* bloodstream infections in one NICU. *S aureus* bacteremia in the neonate is historically associated with septic shock and can be rapidly fatal. Catheter-related bacteremia is most

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Table 1. Clinical Syndromes Reported in Association With Methicillin-resistant *Staphylococcus aureus* Infections in Neonates

Dermatologic
• Cellulitis
• Impetigo (± bullae)
• Pustulosis
• Scalded skin syndrome
• Soft-tissue infection/abscess
• Exanthematous disease (NTED)
Head/Neck
• Facial cellulitis
• Lymphadenitis
• Conjunctivitis
Cardiac
• Sepsis
• Endocarditis
• Infected thrombus
• Septic thrombophlebitis
Genitourinary
• Urinary tract infection
Respiratory
• Pneumonia
• Bacterial tracheitis
• Lung abscess
• Poststernotomy mediastinitis
• Septic pulmonary emboli
Bone/joint
• Osteomyelitis
• Septic arthritis
• Iliopsoas abscess
Central Nervous System (CNS)
• Meningitis
• CNS shunt-related infection
Gastrointestinal
• Peritonitis
• Necrotizing enterocolitis

common; endocarditis and septic thrombophlebitis can result. Meningitis is rare but reported in association with MRSA bacteremia or neurologic surgery, especially with implantable devices.

Dermatologic manifestations are among the most common presentations of MRSA infections because the skin often becomes colonized initially and serves as a portal for deeper and more invasive infections. Skin infections reported in the literature include staphylococcal scalded skin syndrome, pustulosis, impetigo (including the bullous form), and soft-tissue infection and abscess, such as newborn mastitis and breast abscess.

Facial cellulitis, sometimes accompanied by underlying infantile osteomyelitis, is reported. Conjunctivitis is also noted, although growth of MRSA from conjunctival drainage without associated symptoms often simply reflects MRSA colonization.

MRSA pneumonia, septic pulmonary emboli, lung abscess, and bacterial tracheitis complicated by subglottic stenosis are described in the literature. Poststernotomy mediastinitis due to MRSA and other surgical site infections also are reported. Reports of urinary tract infections, peritonitis, and necrotizing enterocolitis, believed to be due to MRSA on the basis of isolation from stool specimens, also are noted. Bone and joint MRSA infections, such as osteomyelitis and septic arthritis, are described commonly, including reports of iliopsoas abscesses.

In a recent analysis of 90 episodes of health care-associated MRSA bacteremia in an NICU in Taiwan (Chuang et al, 2004), the most common clinical diagnoses were catheter-related infections (54%), skin and soft-tissue infections (21%), bacteremia without a focus (20%), and pneumonia (17%). Conjunctivitis (7%), bone and joint infections (3%), meningitis (2%), and peritonitis (1%) accounted for the remainder of cases, with more than one site of infection noted among 23 infants. Prolonged MRSA bacteremia was common; 10 infants had blood cultures positive for MRSA on more than 3 days. Metastatic complications developed among 18% of infants who had MRSA bacteremia, and 9% developed recurrent infection, with a median time interval to recurrence of 29.5 days. Overall mortality in this analysis was 18%, with 1.2% attributable to MRSA. Infants in this study were predominantly preterm (76%) (median gestational age of 31 weeks), had received prior antibiotic therapy (96%), had a central venous catheter (79%), were receiving hyperalimentation (62%), and weighed less than 1,500 g at birth (53%).

Another recent retrospective cohort study from Texas Children's Hospital in Houston (Healy et al, 2004) provided the first documentation that MRSA with genetic characteristics attributable to community-acquired strains can be the principal cause of MRSA infections in neonates who have never left the NICU. Of 17 infants who had *S aureus* bacteremia, eight had MRSA infec-

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tions. Isolates from six of these eight infants carried the *SCCmec* genes characteristic of CA-MRSA, one isolate was typical of a health care-associated strain, and the other was untypable. Seven infants (88%) had severe sepsis, which presented at a median age of 26 days. Other manifestations included endocarditis, septic emboli, necrotizing pneumonia, pleural empyema, pneumatoceles, lung abscess, cellulitis, skin abscess, osteomyelitis, and orbital cellulitis and abscess related to a dacryocele-associated infection. Overall mortality was 38%, although all three infants who died had MRSA isolates characteristic of community-acquired strains, raising mortality in this group to 50%. Most infants in this study were also preterm (75%) (mean gestational age of 29.4 weeks), had central venous catheters (88%), were receiving hyperalimentation (63%), and weighed less than 1,500 g at birth (88%).

Since 1992, NICUs in Japan have witnessed a pandemic spread of a single clone of MRSA that has led to the development of a new neonatal disease known as neonatal toxic shock-like exanthematous disease (NTED). NTED is characterized by a systemic exanthem, fever, thrombocytopenia, and low-positive C-reactive protein values. Symptoms regress spontaneously without antibiotic therapy in term neonates, but most preterm neonates develop more severe symptoms that require antimicrobial therapy. Most MRSA isolates from NTED patients were found to produce TSST-1 and staphylococcal enterotoxin C. MRSA is highly prevalent among NICU patients in Japan and is predominantly multidrug-resistant. The spread of MRSA in NICUs in Japan has been attributed to overcrowding, high rates of extremely low-birthweight babies, understaffing, lack of infection control measures, and overuse of antibiotics.

Diagnosis

Isolation of MRSA from culture of an otherwise sterile body fluid is typically diagnostic of infection. Care must be taken to avoid overdiagnosis of MRSA infection among heavily colonized infants based on culture results from nares, conjunctivae, umbilicus, or tracheal aspirates, which simply may reflect colonization. *S aureus* rarely is a contaminant when isolated from a blood culture. Repeated isolation from blood cultures suggests true infection, and patients should be evaluated for possible underlying suppurative foci or metastatic spread, including endocarditis. Aspiration and drainage of involved sites of infection, purulent fluid collections, or abscesses not only frequently are therapeutic, but also assist in recovery of an organism that guides selection of appropriate antimicrobial coverage.

Treatment

Intravenous vancomycin has become the mainstay of therapy for MRSA infections because it is the only reliably effective agent. Antimicrobial dosing guidelines for neonates are available in the 2003 American Academy of Pediatrics *Red Book* and in other resources. Vancomycin serum concentrations can vary significantly, depending on clinical circumstances and other medications. Vancomycin serum concentrations and renal function should be monitored, with dosing adjusted accordingly to maximize therapeutic effect and minimize toxicity.

Although the duration of therapy for serious infections depends on the site and severity of infection, prolonged antimicrobial regimens typically are indicated in the treatment of *S aureus* infections due to their propensity to persist and recur. Gentamicin or rifampin often is added to vancomycin, depending on the severity and site of infection. Rapid resistance emerges if rifampin is administered as a single agent, however.

In the following discussion, durations of therapy are those that commonly are recommended, although few data support them. Minimal duration of therapy for uncomplicated staphylococcal bacteremia is 10 days, although many experts suggest 2 to 3 weeks. The duration of therapy for catheter-associated bacteremia is somewhat controversial. Catheter removal is always optimal and should be considered as a standard course of treatment when peripheral or temporary central venous catheters are involved. Parenteral vancomycin therapy for 10 to 14 days is prescribed. This sometimes creates a dilemma because maintaining peripheral intravenous infusions for many days is not always easy, but peripherally inserted central catheter lines may contribute to persistence of the infection or the introduction of a new pathogen.

If a patient requires a surgically implanted catheter (ie, Hickman or Broviac) for ongoing care and central access options are limited, in situ treatment of the bloodstream infection can be attempted. Immediate removal of an infected catheter is indicated if a patient develops hypotension at any time during therapy for a catheter-related infection. Adjunctive therapy with rifampin in combination with vancomycin may be indicated in such situations. If the bacteremia clears immediately without evidence of associated thrombus or thrombophlebitis, 10 to 14 days of parenteral therapy is appropriate. If bacteremia persists for more than 3 days, immediate catheter removal is indicated, with continued evaluation for evidence of metastatic foci of infection.

Endocarditis due to MRSA requires 4 to 6 weeks of closely monitored therapy; gentamicin typically is added

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for the first 1 to 2 weeks as adjunctive therapy. Similar regimens are prescribed for septic thrombophlebitis or MRSA bacteremia complicated by the presence of a thrombus. Staphylococcal pneumonia, particularly if complicated by empyema or lung abscess, requires 3 to 4 weeks of therapy. Osteomyelitis caused by MRSA requires 4 to 6 weeks of parenteral vancomycin therapy, and septic arthritis commonly is treated for 3 weeks, with rifampin adjunctive therapy often used in such scenarios.

Among the options for antimicrobial therapy in neonates who have CA-MRSA are clindamycin, trimethoprim-sulfamethoxazole, and linezolid. However, consideration must be given to full susceptibility results, including D-test; the need for bactericidal versus bacteriostatic activity in specific clinical circumstances; and data regarding pharmacokinetics, safety, and efficacy of these agents in neonatal patients. Consultation with a pediatric infectious diseases specialist is indicated when treating complicated MRSA infections.

Surgical drainage of purulent fluid collections or abscesses and removal of foreign bodies and implanted devices, such as central venous catheters or central nervous system shunts, when involved, is critical to successful treatment of invasive MRSA infections. Antimicrobial therapy alone frequently fails to eradicate *S aureus* completely from implantable devices.

Vancomycin resistance issues have presented an emerging dilemma as MRSA prevalence increases. Strains of *S aureus* with intermediate- and high-level resistance to vancomycin have been reported, and the primary risk factor for the emergence of these strains is increased use of vancomycin. In light of these concerns, decisions to incorporate vancomycin into empiric antimicrobial regimens for late-onset neonatal sepsis should be made on the basis of local epidemiology, with particular attention to the prevalence of CA-MRSA in the community.

Transmission

MRSA commonly is transmitted to neonates from the hands of colonized health care workers early in their NICU stays. Colonized health care workers may serve as a reservoir for transmission, and patient-to-patient transmission among colonized infants via transient hand colonization of health care workers is well documented. Evidence of maternal-to-infant transmission via colonized nares and genital secretions, placental transmission, mastitis, and contaminated human milk also is described. The spread of MRSA in a NICU also has been attributed previously to understaffing, overcrowding, and mixing of patients.

As the prevalence of CA-MRSA has increased in different regions, the potential to increase the prevalence and risk of nosocomial infection due to CA-MRSA also exists. Saiman and associates (2003) reported a hospital outbreak of CA-MRSA among postpartum women who developed skin and soft-tissue infections (mastitis, wound infection, cellulitis, and pustulosis). Surveillance cultures of hospital employees, the hospital environment, and newborns were negative. Similarly, the study by Healy and colleagues (2004) documented introduction of community strains of MRSA into an NICU. The authors speculated that horizontal transmission may have occurred from colonized visitors or health care workers, but evidence of a single outbreak did not exist.

In a separate report by Eckhardt and associates (2003), a newborn who had sepsis at the time of delivery acquired MRSA from his mother, who presented with chorioamnionitis. Routine surveillance cultures revealed MRSA colonization in a different neonate in the same NICU 7 days later. The isolates were susceptible only to vancomycin, gentamicin, and rifampin, consistent with a health care-associated MRSA strain but different from strains recovered within the institution. PFGE pattern documented one clonal PFGE subtype, which suggested community acquisition of MRSA. Further history revealed the mother's role as caretaker for an individual who had a chronic wound infection and was believed to be the likely source of her colonization.

As the prevalence of CA-MRSA increases, reservoirs of infection may be created among family members of hospitalized infants and health care workers. These cases illustrate the challenges of preventing nosocomial transmission of MRSA when it is acquired in a community.

Control

MRSA outbreaks in NICUs can be prolonged and difficult to eradicate. Aggressive infection control measures frequently are necessary to avoid or terminate such outbreaks (Table 2). Patients who have known MRSA infection or colonization should be managed routinely with contact precautions for the duration of their hospitalization, based on persistent carriage. Cohorting of MRSA infected/colonized infants and staff designated to care for them is recommended during outbreaks. In outbreaks, surveillance cultures may be useful to define the involved population of infants. Emphasis on appropriate hand hygiene between all patient encounters and reinforcement of basic infection control principles is beneficial. Designating an infection control practitioner to ensure appropriate attention to hand hygiene, aseptic technique, cohorting, and correct isolation procedures has been successful in eradication of endemic MRSA

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Table 2. Control Measures for Methicillin-resistant *Staphylococcus aureus* (MRSA) Outbreaks in Neonatal Intensive Care Units (NICUs)

- Routine contact precautions for known MRSA infected/colonized infants
- Cohorting of MRSA infected/colonized infants and designated staff
- Surveillance cultures
  - —Infants transferred from referring nurseries
  - —NICU population during outbreaks to define involvement
- Emphasis on appropriate hand hygiene
- Reinforcement of basic infection control principles
- Designated NICU infection control practitioner
- Maintenance of appropriate staff-to-patient ratios
- Avoidance of overcrowded conditions
- Appropriate umbilical care
- Topical mupirocin for decolonization
- Antiseptic baths
- Consideration of staff nasal cultures, if outbreak persists
  - —Topical mupirocin for decolonization

from one NICU. Assuring appropriate staff-to-patient ratios and avoiding overcrowded conditions have been associated with limiting nosocomial infection spread. Routine culturing of staff is controversial and likely not cost-effective; targeted cultures of specific personnel involved in the care of infants who have MRSA infection or colonization occasionally may be indicated in outbreak circumstances only if MRSA infections persist. Mupirocin has been used successfully to eradicate nasal carriage of MRSA in neonatal patients and staff in outbreak settings. Application of triple dye, iodophor ointment, or hexachlorophene powder to the umbilical stump has been shown to reduce MRSA colonization. Hexachlorophene baths were very effective at decreasing colonization and limiting spread of *S aureus* outbreaks in the past. Hexachlorophene use is limited, however, due to neurotoxicity concerns, particularly among preterm infants. Chlorhexidine gluconate for bathing has been used in older children and adults as a component of decolonization regimens. Its use in neonates merits further study.

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NeoReviews Quiz

5. *Staphylococcus aureus* produces a variety of extracellular toxins responsible for the virulence of the microorganism. Of the following, the toxin with the *most* specific role in the causation of staphylococcal disease is:

   A. Bacteriocin.
   B. Coagulase.
   C. Enterotoxin.
   D. Hyaluronidase.
   E. Leukocidin.

6. Community-acquired methicillin-resistant *Staphylococcus aureus* strains exhibit inducible resistance to several antimicrobial agents. Of the following, the microbiologic test that can *most* accurately determine inducible resistance of clindamycin is:

   A. Cefoxitin disk diffusion.
   B. Disk diffusion induction test.
   C. Erythromycin resistance.
   D. Minimal inhibitory concentration.
   E. Pulsed field gel electrophoresis

7. The clinical manifestations of MRSA infection vary widely among newborns. Of the following, the *most* common presentation of MRSA infection in the neonate is:

   A. Conjunctivitis.
   B. Enterocolitis.
   C. Meningitis.
   D. Osteomyelitis.
   E. Skin infection.

8. Since its emergence in the 1960s in the United States, MRSA has become a common health care-associated pathogen. Of the following, the *most* accurate statement regarding this pathogen is that:

   A. MRSA comprises less than 50% of staphylococcal strains that cause nosocomial infection.
   B. MRSA is more virulent than its methicillin-susceptible counterpart.
   C. MRSA is often a contaminant when isolated from blood culture.
   D. Pulsed-field gel electrophoresis is the gold standard for MRSA typing.
   E. Routine culturing of personnel in the nursery is warranted as a means for eradication of MRSA.

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