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Neonatal Outbreak of Methicillin-Resistant *Staphylococcus aureus* Clone Geraldine: A Bundle of Measures to Halt Transmission

Methicillin-resistant *Staphylococcus aureus* (MRSA) outbreaks are frequent in neonatal intensive care units (ICUs).[1] Toxic-shock-syndrome toxin 1 (TSST-1)–producing MRSA Geraldine clone represented 6.3% of invasive MRSA isolates in France in 2006 and 2007,[2] and has been implicated in one outbreak among newborns.[3] We describe here a neonatal MRSA Geraldine clone outbreak.

On March 31, 2014, a TSST-1–positive MRSA was isolated in bronchial aspirates from 2 ICU neonates (case patients 3 and 4) (Figure 1). Our subsequent investigation identified 2 prior cases of TSST-1 MRSA carriage, the index case 1 by umbilical swab in November 2013 and case 2 by bronchial aspirate in December 2013. A case was defined as a positive culture for an MRSA strain expressing TSST-1 and/or specific antibiotic susceptibility in a patient hospitalized in the neonatal ICU or general neonatal ward. In total, we identified 8 cases (7 cases of carriage and 1 skin infection) over a 9-month period (Figure 1). All case patients were premature (26–30 weeks gestation; mean birth weight, 975.2 g) and were hospitalized in the neonatal ICU. Among them, the mean interval of MRSA carriage detection was 25.1 hospitalization days, and mean length of ICU stay was 33.1 days. During the outbreak, case surveillance consisted of weekly nasal *S. aureus* carriage screening of the neonates of both wards; this procedure remained in place for 5 months after the last case was discovered. All MRSA isolates expressed resistance to penicillin G, methicillin, kanamycin, tobramycin, and fucidic acid according to guidelines of the French Antibiogram Committee. All of the isolates were typed by the National Reference Center for staphylococci (*S. aureus* Genotyping Identibac, Alere, Waltham, MA) and were identified as the Geraldine clone, which is characterized by the following criteria: (1) sequence type ST5, *agr*2, (2) positivity for TSST-1, enterotoxins SEC, SED, SEJ, SEL, and SER as well as the *egc* locus, and (3) negativity for Panton-Valentine leukocidin.[4] All case isolates underwent molecular analysis except strains from cases 1 and 2, as these strains had not been stored.

Immediately after the alert, we implemented contact precautions (ie, glove and gown usage) for HCP in contact with infected and colonized neonates. We also held information meetings for healthcare personnel (HCP) and audited HCP practices. The audit revealed a lack of consistency in standard precaution application and hygiene practices. The control measures implemented consisted of team support for multidrug-resistant bacteria management, standard precautions, and hand-hygiene reinforcement. We focused on the use of hydroalcoholic solutions, lack of hand jewelry verifications, and daily changes of work outfits. We assessed the effectiveness of these measures using indicators such as bedsore prevalence, cleaning activities records, environmental samples, and compliance with hand hygiene procedures, which was assessed by hydroalcoholic solutions consumption according to French guidelines.[5] Compliance to the minimum hydroalcoholic consumption, calculated according to clinical activity, increased from 57.4% 6 months before the outbreak to 84.4% during the outbreak to 102.9% 6 months after the outbreak.

We sought environmental links between cases. In total, 60 environmental swabs and 20 surface samples from patient rooms, drug preparation area, transfrontanellar ultrasound apparati, and x-ray devices were tested between May 3 and June 25, 2014. No medical devices or environmental sources were found to be involved in transmission.

Despite the control measures, transmission continued. Some carrier neonates were hospitalized in neighboring rooms (Figure 1), suggesting possible cross transmission via HCP hands, especially because HCP compliance to the measures was not consistent at the beginning of the outbreak. In addition, *S. aureus* may have been spread by airborne transmission by HCP.[6] The long interval between the first 2 and subsequent 6 cases also pointed to HCP carriage. HCP are often involved in horizontal MRSA transmission to neonates,[6,8] and HCP decolonization is a proven outbreak control measure.[3,7,8] We opted for universal decolonization of all HCP, both permanent and rotating staff (including students, radiology technicians, radiologist physiotherapist, psychologist, milk-bank technicians, cleaning staff, social workers, laboratory couriers, and secretaries), regardless of their screening results, in order to cover the risk of false negatives due to intermittent carriage. We sampled both the noses and throats of the HCWs to improve sensitivity.[9] Decolonization consisted of a 5-day course based on twice-daily mupirocin nasal ointment and daily showering with chlorhexidine soap,[10] which were dispensed to each HCP during screening interviews to promote adherence. HCP voluntarily participated in decolonization; no

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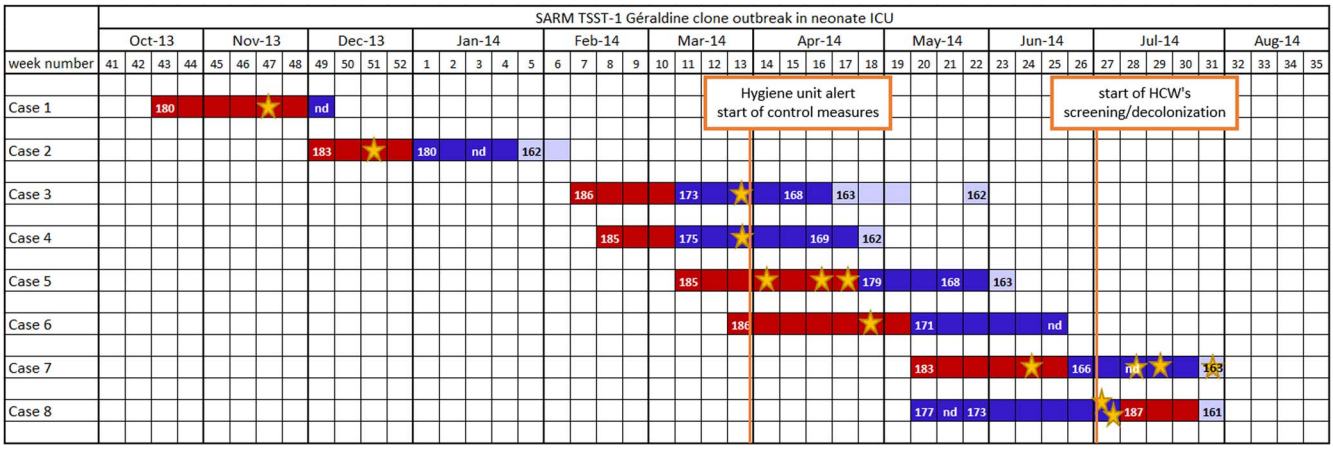

FIGURE 1. Synoptic table of the cases and wards. Each square represents a week: red for the neonatal ICU, dark blue for the general neonatal ward, and light blue for the mother–neonate unit to which neonates are transferred when their condition allows, usually close to hospital discharge. The numbers in the squares represent the room number, and stars represent isolations of the outbreak clone.

one refused. No adverse event related to decolonization was recorded. Our strategy was aggressive because the same bundle of standard control measures plus non-exhaustive HCP decolonization failed to control an MRSA outbreak in another neonatal ICU.[11] The first round of screening involved all 155 HCP working with the neonates during July and August 2014. Overall, 61 HCP were *S. aureus* carriers (39.3%); among them, 2 (1.3%) carried the outbreak strain. Both were nurses, 1 nurse worked in neonatal ICU and the other one in general neonatal ward. They had been in contact with the case patients since the outbreak started. We were not able to determine whether the transmission originated with an HCP or a patient. These results confirmed our working hypothesis and justified the decolonization strategy. We checked screening efficacy with a second round of screening 1 month later on 30 HCP including the 2 identified carriers, both of whom tested negative. In the subsequent screening round, global *S. aureus* carriage decreased from 53.3% to 20%. The overall cost of the outbreak was US$18,821 (17,600 euros), which included consumption of protections for contact precautions for HCP and neonates, follow-up screening, and decolonization treatments.

In conclusion, we have described an outbreak of the MRSA Geraldine clone in a neonatal department, which was finally controlled by screening and decolonizing all HCP. The screening results suggested circulation of the outbreak clone between HCP and patients, which explains the persistence of new cases despite classical control measures. Our findings support universal HCP decolonization during neonatal outbreaks of MRSA TSST-1.

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Absence of Correlation Between Vancomycin Consumption and Minimum Inhibitory Concentration of Methicillin-Resistant *Staphylococcus aureus* Isolates

Methicillin-resistant *Staphyloccocus aureus* (MRSA) is an important human pathogen; it is among the most common causes of healthcare-associated infection.[1] Despite the use of appropriate antimicrobial therapy, MRSA invasive infections carry a high mortality rate.[2,3] Vancomycin is a mainstay of therapy in MRSA infections, and although it has been used since 1950, resistance is uncommon.[1] Vancomycin minimal inhibitory concentration (MIC) "creep" is a phenomenon in which the vancomycin MIC in *S. aureus* isolates progressively reaches higher values (MIC ≥1.5 mg/L) within the susceptibility range.[4]

Monitoring antimicrobial usage remains a cornerstone of antimicrobial stewardship programs. There is limited evidence of a correlation between MRSA active antimicrobial agent consumption and the emergence of resistance.[5] In this study, we aimed to assess the existence of vancomycin MIC creep among MRSA isolates obtained from blood cultures and to determine whether a correlation exists between vancomycin consumption and variations in the MIC over time.

This study was performed at the Hospital Nossa Senhora da Conceição, a tertiary-care public hospital located in Porto Alegre, Brazil. The study period extended from June 2012 to February 2016, and data for the period were obtained from computerized databases. Isolates from the same episode of bacteremia were counted only once. The isolates were identified using a Vitek-2 system (bioMérieux, Marcy-I'Etoile, France). The vancomycin MIC was determined either by broth microdilution (BMD), according to Clinical & Laboratory Standards Institute (CLSI) recommendations, or by Etest strips (bioMérieux). Vancomycin utilization was expressed in defined daily doses (DDD) per 100 patient days, processed according to the Anatomical Therapeutic Chemical classification system, in which the DDD of vancomycin is 2 g.

The χ^2 test was used to compare proportions. Correlation between variables was tested using the Pearson correlation coefficient, and variation of MIC over time was calculated using analysis of variance (ANOVA). Two-tailed *t* tests were utilized, and a value of $P \leq .05$ was considered significant. Statistical analyses were performed using the JMP 9 program (SAS Institute, Cary, NC).

A total of 186 isolates were included in the analysis. During the study period, the laboratory applied 2 different methodologies: for data from June 2012 to November 2013, BMD was used, and for data from December 2013 to February 2016, an Etest was performed. The MIC ranged from 0.25 to 2.0 mg/L in the first period (for which BMD was used), and the MIC varied from 0.5 to 2.0 mg/L in the second period (for which an Etest was used).

Vancomycin MIC geometrical mean increased significantly in the study period from 0.766 to 1.966 mg/L ($P < .0001$) when the 2 different methodologies were combined. Analyzing the 2 periods separately, no significant variation was observed for the BMD period. However, for the second period (ie, the Etest period), a significant increase in MIC geometrical mean from 0.791 to 1.966 mg/L was observed ($P = .0003$) (Figure 1). There was an increase in the modal MIC from 0.5 to 2.0 mg/L over the whole period ($P < .0001$). Analyzing modal MIC only in the Etest period, there was an increase from 1.0 to 2.0 mg/L ($P = .003$). The proportion of isolates with MIC > 1.0 mg/L ranged from 6.5% to 100% ($P = .001$) during the Etest period.

Both vancomycin utilization and its relation to the total number of hospitalized patients remained stable, varying between 4,488 and 6,449 DDD per 100 patient days ($P = .223$). No correlation was observed between vancomycin utilization and the MIC geometric mean, nor with modal MIC, even when the 2 different methodology periods were analyzed separately. A separate correlation coefficient between MIC geometrical mean and vancomycin utilization for the Etest period alone was poor ($r = 0.524$; $P = .148$).

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