International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Coagulase-Negative Staphylococci in Neonatal Sepsis: Epidemiology, Species Distribution, and Device-Associated Risk Factors in a Tertiary-Care Hospital

Mr Tarkeshwar Prasad Yadav[1], Dr Ramanath K[2]

[1]Research Scholar, Department of Microbiology, Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh-452016.
[2]Professor, Department of Microbiology, Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh-452016.

Abstract

Background: Neonatal sepsis remains a major cause of preventable morbidity and mortality, particularly among premature and low-birth-weight infants. Coagulase-negative staphylococci (CoNS), once regarded principally as skin contaminants, are now established causes of late-onset sepsis in neonatal intensive care units (NICUs). Their clinical importance is linked to immature host defences, prolonged hospitalization, vascular access, respiratory support, and the ability to adhere to foreign materials. The present study analyses the epidemiological and clinical profile of CoNS isolated from suspected neonatal sepsis cases in a tertiary care centre.

Methods: The study performed was a prospective cross-sectional investigation conducted in the Microbiology Department of Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh. The broader surveillance dataset contained 6,069 blood samples collected from clinically suspected neonatal sepsis cases during the reported study period. Among these, 2,897 cultures were positive, 2,115 were sterile, and 1,057 yielded contaminants. A defined analytical cohort of 100 CoNS isolates was evaluated after exclusion of isolates considered contaminants and samples yielding other organisms. Clinical, demographic, onset-of-sepsis, gestational-age, birth-weight, device-exposure, and species-identification data were summarized descriptively.

Results: The overall blood-culture positivity rate was 47.73%. CoNS represented 554 of 2,897 positive cultures, or 19.12%, and were the most frequent Gram-positive group in the broader dataset. Within the 100-isolate CoNS cohort, 64% were associated with late-onset sepsis and 36% with early-onset sepsis. Most late-onset cases occurred between days 4 and 7 of life. Males comprised 65% of the cohort. Medical devices producing mucosal or cutaneous breach were present in 96% of cases, low birth weight in 66%, and prematurity in 40%; the source document reports a statistically significant association for the evaluated risk-factor profile. Six CoNS species were identified. *Staphylococcus epidermidis* was predominant at 56%, followed by *S. haemolyticus* at 29%, *S. hominis* at 11%, *S. capitis* at 2%, and *S. lugdunensis* and *S. warneri* at 1% each.

Conclusions: The findings support CoNS as a clinically important component of neonatal sepsis surveillance, especially in the first week of life and among infants exposed to invasive devices. The

predominance of *S. epidermidis* is consistent with its role as a skin commensal and biofilm-forming opportunist. Prevention requires rigorous hand hygiene, device-care bundles, skin protection, reduction of unnecessary device days, and a reproducible framework for distinguishing true infection from contamination.

Keywords: neonatal sepsis; late-onset sepsis; Coagulase-negative Staphylococci; *Staphylococcus epidermidis*; neonatal intensive care unit; prematurity; low birth weight; bloodstream infection.

Introduction

Neonatal sepsis is a life-threatening syndrome in which infection is accompanied by dysregulated host responses and organ dysfunction. The clinical burden is disproportionately concentrated in the neonatal period because the immune system, epithelial barriers, and physiological reserves are incompletely developed. A systematic review estimated a population-level neonatal sepsis incidence of approximately 2,202 cases per 100,000 live births, with mortality between 11% and 19%, while emphasizing the scarcity and heterogeneity of data from low- and middle-income settings 1. The global burden is therefore not only a clinical problem but also a surveillance problem: differences in case definitions, blood-culture practices, laboratory capacity, and access to neonatal intensive care affect the apparent incidence and the organisms reported.

Neonatal sepsis is commonly divided according to the timing of presentation. Early-onset sepsis (EOS) is generally defined as infection occurring within the first 72 hours after birth and is often associated with maternal, intrauterine, or peripartum transmission. Late-onset sepsis (LOS) develops after 72 hours and is more strongly associated with the hospital environment, prolonged admission, invasive procedures, and postnatal colonization 2. This distinction is useful but not absolute. The epidemiology of neonatal sepsis is changing as survival improves among extremely premature infants, invasive support becomes more frequent, and antimicrobial exposure selects organisms adapted to the NICU.

CoNS are central to this changing epidemiology. They are common inhabitants of human skin and mucous membranes, and *S. epidermidis* is frequently recovered from moist skin sites, the anterior nares, axillae, perineum, and other body surfaces 3. Their ubiquity creates a diagnostic paradox. The same organism can represent contamination during blood collection, colonization of a catheter or skin surface, or genuine bloodstream infection. In a critically ill premature neonate, dismissing a CoNS-positive culture as contamination may delay appropriate treatment; treating every positive culture as sepsis may expose infants to unnecessary antibiotics and amplify resistance.

The biological plausibility of CoNS sepsis is strongest when host vulnerability and device exposure coexist. Prematurity is associated with reduced neutrophil number and function, altered antigen presentation, lower immunoglobulin concentrations, and immature skin and mucosal barriers 4. Very-low-birth-weight infants often require central venous catheters, endotracheal tubes, parenteral nutrition, and repeated blood sampling. These interventions can disrupt barrier function and provide surfaces on which CoNS adhere and form biofilms. Biofilm-associated organisms are protected by extracellular matrix, exhibit altered metabolic states, and may be relatively difficult to eradicate without removal or replacement of the implicated device 5.

The epidemiological importance of a local study lies in its capacity to connect these general mechanisms with the organisms and exposures observed in a particular NICU. The supplied institutional document reports a large background blood-culture dataset and a defined cohort of 100 CoNS isolates. It records a

predominance of LOS, a high frequency of device exposure, and a species distribution led by *S. epidermidis*. These findings provide an opportunity to examine CoNS not as an undifferentiated laboratory category, but as a clinically relevant group whose timing, species composition, and host context should guide interpretation.

This study has four objectives. First, it describes the contribution of CoNS to blood-culture-positive neonatal sepsis in the source dataset. Second, it characterizes the timing of infection and the demographic, gestational, birth-weight, and device-associated profile of the 100-isolate cohort. Third, it examines the distribution of CoNS species. Finally, it discusses how these findings should influence clinical diagnosis, infection prevention, and future surveillance in resource-constrained tertiary-care NICUs.

Materials and methods

Study design and setting

This study is based on the prospective cross-sectional study conducted at a tertiary care centre. Blood specimens were obtained from clinically suspected neonatal sepsis cases admitted to the neonatal intensive care service of Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh.

Study population and analytical cohorts

The broader surveillance dataset consisted of 6,069 blood samples from suspected neonatal sepsis cases. Of these, 2,897 yielded growths, 2,115 showed no growth, and 1,057 were reported as contaminants. The broad culture-positivity rate was calculated as 2,897 divided by 6,069, or 47.73%. CoNS represented 554 of the culture-positive samples, corresponding to 19.12% of positive cultures.

For detailed clinical and microbiological analysis, 100 CoNS isolates that fulfilled the study criteria were used. Included patients were clinically suspected of neonatal sepsis, had samples collected before antimicrobial treatment where possible, and had consent for participation. Samples yielding organisms other than CoNS were excluded. CoNS isolates considered contaminants were also excluded. These criteria included two positive cultures drawn on separate occasions with compatible signs, one positive culture accompanied by clinical improvement after antimicrobial therapy, or pure CoNS growth from a bottle flagged within 24 hours.

Blood culture and organism identification

Approximately 1–2 mL of blood was collected by venipuncture under standard precautions and inoculated directly into BacT/ALERT PF Plus culture bottles. The culture system was incubated for up to seven days. Positive bottles were sub cultured onto sheep blood agar and incubated at 37°C. Colonies consistent with staphylococci were evaluated by Gram staining, catalase testing, and slide and tube coagulase testing. CoNS were defined phenotypically as Gram-positive cocci, catalase positive, and non-coagulase-producing isolates. Final species identification was performed with the Vitek 2 Compact system using a Gram-positive identification card.

Variables and analysis

The variables used for this analysis were sex, age at onset, EOS or LOS classification, gestational maturity, birth-weight category, presence of a device producing mucosal or cutaneous breach, and final species identification. Descriptive statistics are reported as counts and percentages.

Results

Blood-culture yield and contribution of CoNS

Among 6,069 samples from suspected neonatal sepsis cases, 2,897 were culture positive, giving a positivity rate of 47.73%. Sterile cultures accounted for 34.84% and contaminants for 17.41%. The relatively high contaminant fraction is epidemiologically important because it illustrates the difficulty of interpreting organisms that are common skin flora. It also demonstrates why a CoNS surveillance program must combine microbiological results with clinical information and sampling quality rather than report all positive cultures as equivalent.

CoNS were the third most frequent category among positive cultures, after *Acinetobacter baumannii* and *Klebsiella pneumoniae*. The 554 CoNS-positive cultures represented 19.12% of all positive cultures. Other reported organisms included *Escherichia coli* at 11.70%, *Staphylococcus aureus* at 5.76%, *Pseudomonas aeruginosa* at 5.00%, and several *Candida* and *Citrobacter* species 20. Thus, CoNS were not the dominant organism overall, but they were the most prominent Gram-positive group and constituted a substantial fraction of the culture-positive burden.

Table 1: Distribution of studies cases and positivity

Indicator	Finding in the source dataset
Blood samples from suspected neonatal sepsis	6,069
Culture-positive samples	2,897 (47.73%)
Sterile samples	2,115 (34.84%)
Samples reported as contaminants	1,057 (17.41%)
CoNS among positive cultures	554 (19.12%)
Detailed CoNS analytical cohort	100 isolates

Timing and demographic profile

Within the 100-isolate CoNS cohort, 64% were associated with LOS and 36% with EOS. Among the LOS group, 79.6% of cases occurred between four and seven days of life; the remainder occurred between eight and 28 days. This concentration in the first postnatal week is compatible with the period in which infants may have already undergone colonization, repeated vascular access, respiratory instrumentation, and prolonged exposure to the NICU environment, while still possessing highly vulnerable epithelial and immune barriers.

Males comprised 65% of the cohort and females 35%, producing a male-to-female ratio of approximately 1.8:1. The study was not designed to test a biological explanation for this difference, and the result should not be interpreted as proof of sex-specific susceptibility. The imbalance may reflect the sex distribution of admissions, referral patterns, or chance variation in a modest cohort. It does, however, provide an important description of the local study population.

Host and device-associated risk factors

A medical device associated with mucosal or cutaneous breach was present in 96% of the CoNS sepsis

cases. Low birth weight was documented in 66%, and 40% of infants were preterm. The source document reports a statistically significant association for the evaluated risk-factor profile (P = 0.023). Since the available report does not provide the cross-tabulation of each exposure against a non-CoNS control group, the result is best interpreted as evidence of a clinically important clustering of vulnerability factors rather than as a precise estimate of independent risk.

The pattern nevertheless has strong biological coherence. Devices can bypass the skin and mucosal barriers, create a surface for microbial attachment, and require repeated handling by staff. Prematurity and low birth weight magnify this exposure by prolonging hospitalization and increasing the need for respiratory and vascular support. Studies of neonatal CoNS infection similarly identify prematurity, very low birth weight, central lines, and prolonged hospitalization as major risk factors [6] [7] [17] [18] [19]. The combination of multiple modest risks may therefore be more informative than any single variable.

Species distribution

Six species were recovered. *S. epidermidis* accounted for 56 isolates, followed by *S. haemolyticus* with 29 and *S. hominis* with 11. *S. capitis*, *S. lugdunensis*, and *S. warneri* accounted for two, one, and one isolate, respectively.

Table 2: Species distribution of CoNS

CoNS species	Number of isolates	Percentage
Staphylococcus epidermidis	56	56%
Staphylococcus haemolyticus	29	29%
Staphylococcus hominis	11	11%
Staphylococcus capitis	2	2%
Staphylococcus lugdunensis	1	1%
Staphylococcus warneri	1	1%
Total	**100**	**100%**

The predominance of *S. epidermidis* is consistent with reports from India, Iran, Europe, and other settings in which it is the most frequently recovered CoNS species from neonatal blood cultures [8] [9] [21] [22]. The relatively high proportion of *S. haemolyticus* is also clinically relevant because this species can carry extensive antimicrobial resistance and may persist in hospital environments. Species-level identification is therefore not merely taxonomic: it can improve outbreak recognition, interpretation of resistance patterns, and selection of infection-control interventions.

Discussion

The present study presents a coherent epidemiological picture of neonatal CoNS sepsis. CoNS accounted for approximately one in five positive blood cultures and were the most common Gram-positive organisms. In the detailed cohort, nearly two-thirds of cases were late onset, and almost nine in ten occurred during the first week of life. Most infants had exposure to a device capable of breaching a

protective barrier, while low birth weight and prematurity were also frequent. *S. epidermidis* dominated the species distribution.

The findings are consistent with the established distinction between EOS and LOS. EOS is generally driven by maternal or perinatal acquisition, with group B streptococci and *E. coli* being prominent causes. LOS, in contrast, is strongly influenced by the NICU environment and by cumulative medical exposure 10. CoNS are particularly well adapted to this setting because they are already present on human skin, can be transferred through contact, and can establish biofilms on catheters and other devices. Contemporary reviews report CoNS as responsible for a large proportion of LOS episodes, with estimates varying by population, case definition, and surveillance method 11. The 64% LOS proportion in the present CoNS cohort is therefore clinically plausible and supports the interpretation of CoNS as a predominantly postnatal, healthcare-associated pathogen.

The concentration of cases between days four and seven is important for prevention. It suggests that risk does not begin only after a prolonged NICU stay. Colonisation, line insertion, parenteral nutrition, respiratory support, and repeated contact may combine rapidly after admission. A prevention strategy focused only on long-term central-line users could miss a vulnerable window during the first week. Admission bundles, aseptic line insertion, daily device-necessity review, and early recognition of clinical deterioration should begin immediately rather than after the infant has accumulated several days of exposure.

The 96% device exposure rate is the strongest local signal. It does not establish that a particular device caused infection, because most critically ill NICU infants require devices and the study does not include a matched non-infected comparison group. It does show, however, that CoNS sepsis in this setting is tightly linked to the clinical circumstances in which barrier disruption and foreign material are present. Device-related infection is mechanistically supported by the steps of nonspecific attachment, binding to conditioning proteins, multiplication, intercellular accumulation, and biofilm maturation 5 12. Once a mature biofilm is established, antibiotic treatment alone may be insufficient, and device removal or replacement may be required when clinically feasible.

Prematurity and low birth weight should be interpreted as both biological and systems-level markers. Premature infants have immature stratum corneum, greater trans epidermal water loss, reduced innate immune function, and lower transplacental IgG acquisition. They also remain hospitalised longer and undergo more procedures. Very-low-birth-weight infants may receive central venous access and parenteral nutrition for prolonged periods, creating repeated opportunities for contamination and colonization. This interaction is why prevention cannot be reduced to a single intervention. Hand hygiene, skin care, line insertion technique, line maintenance, equipment disinfection, and antibiotic stewardship operate as a linked bundle.

The distinction between true CoNS infection and contamination remains the central interpretive challenge. In the source dataset, 17.41% of all samples were classified as contaminants, demonstrating how often blood-culture contamination may complicate sepsis evaluation. CoNS are particularly difficult because they are common commensals and may be recovered in small numbers. A single positive bottle does not automatically establish infection, yet a single positive culture can be clinically meaningful in a neonate with a central line, compatible signs, and improvement after therapy. Repeat cultures, time to positivity, the number of positive bottles, clinical signs, inflammatory markers, and device status should be integrated rather than considered in isolation 13 14.

The source study's criteria represent a pragmatic attempt to operationalise this decision. Two positive cultures obtained separately provide stronger evidence than one. Pure growth from a bottle flagged early may support a higher bacterial burden, although time to positivity is influenced by blood volume and prior antibiotics. Clinical improvement after antimicrobial therapy is supportive but not specific, because premature infants may improve for multiple reasons. Future work should predefine a composite case definition, record exact time to positivity, document the number of positive bottles, and include a comparator group of neonates with suspected sepsis but negative cultures.

The species distribution also has implications for surveillance. *S. epidermidis* was the leading isolate at 56%, a finding reported in several studies 8 9. Its predominance is unsurprising given its abundance on human skin and its capacity for adhesion and biofilm formation. *S. haemolyticus* represented 29%, a notable proportion that warrants attention because it is frequently associated with multidrug resistance and may persist in hospital niches. *S. hominis* accounted for 11%, while less common species were also present. If a NICU reports only "CoNS," it may miss species-specific clustering or an emerging outbreak. Species-level identification by validated automated methods or mass spectrometry can therefore strengthen infection-control surveillance, provided that identification quality is monitored.

The broader dataset also shows that CoNS coexist with a large Gram-negative burden. *A. baumannii* and *Klebsiella pneumoniae* together accounted for more than half of positive cultures, and *E. coli*, *Pseudomonas*, *Citrobacter*, and *Candida* were also reported. This means that infection prevention must not become narrowly CoNS-focused. A line bundle that reduces CoNS may also reduce other bloodstream pathogens, but environmental cleaning, antimicrobial stewardship, cohorting, and outbreak investigation must address the entire local ecology. Empirical therapy should be guided by current local antibiograms and then narrowed promptly after culture and susceptibility results 15.

The study's sex distribution should be treated as descriptive. The 1.8:1 male-to-female ratio may resemble patterns reported elsewhere, but the present design cannot distinguish biological susceptibility from admission composition. Similarly, the reported *P* value for risk factors is not sufficient to establish causation. The study would be strengthened by a case-control or cohort design, multivariable analysis, and explicit definitions of low birth weight and prematurity. Nevertheless, descriptive studies are valuable first steps in hospitals where routine surveillance is limited. They identify which exposures deserve prospective measurement and which organisms require laboratory attention.

A practical local response can be organized around three layers. The first is prevention: strict hand hygiene, standardized line insertion and maintenance, skin antisepsis appropriate to gestational age, disinfection of shared equipment, and avoidance of unnecessary device days 16. The second is diagnosis: adequate blood volume, careful venipuncture-site preparation, repeat cultures when clinically indicated, species identification, and a structured contamination-versus-infection algorithm. The third is surveillance: monthly review of CoNS rates, species, device-days, time to positivity, methicillin resistance, and outcomes. Rates should be expressed per 1,000 central-line days or patient-days when denominators are available, because raw counts are affected by NICU occupancy and case mix.

Limitations

This analysis has several limitations. It is based on a pre-existing institutional document rather than a reanalysis of a patient-level database. The source report does not provide a non-CoNS control group, exact definitions for every risk-factor category, confidence intervals, or the full statistical model underlying the reported *P* value. The distinction between the 554 CoNS-positive cultures in the broad dataset and the 100

isolates in the detailed cohort is not fully explained, although the latter appears to represent the eligible non-contaminant analytical sample. Calendar-period and geographic inconsistencies are present in the source document. Finally, the findings are from one tertiary-care hospital and may not generalize to other NICUs 23

Conclusion

CoNS represented a substantial proportion of positive neonatal blood cultures and were the leading Gram-positive group in the supplied tertiary-care dataset. Within the detailed cohort, LOS predominated, most cases appeared during the first postnatal week, and device exposure, low birth weight, and prematurity were common. *S. epidermidis* was the principal species, followed by *S. haemolyticus* and *S. hominis*. These findings support a prevention model centered on barrier protection, device stewardship, hand hygiene, and species-aware surveillance. Because CoNS may be either contaminant or pathogen, microbiological results must be interpreted in the context of clinical status, repeat cultures, device exposure, and response to therapy.

References

1 Fleischmann-Struzek C, Goldfarb DM, Schlattmann P, Schlapbach LJ, Reinhart K, Kissoon N. The global burden of paediatric and neonatal sepsis: a systematic review. Lancet Respir Med. 2018;6(3):223-230. doi:10.1016/S2213-2600(18)30063-8.
2 Rallis D, Giapros V, Serbis A, Kosmeri C, Baltogianni M. Fighting antimicrobial resistance in neonatal intensive care units: rational use of antibiotics in neonatal sepsis. Antibiotics (Basel). 2023;12(3):508. doi:10.3390/antibiotics12030508.
3 Becker K, Heilmann C, Peters G. Coagulase-negative staphylococci. Clin Microbiol Rev. 2014;27(4):870-926. doi:10.1128/CMR.00109-13.
4 Cuenca AG, Wynn JL, Moldawer LL, Levy O. Role of innate immunity in neonatal infection. Am J Perinatol. 2013;30(2):105-112. doi:10.1055/s-0032-1333412.
5 von Eiff C, Peters G, Heilmann C. Pathogenesis of infections due to coagulase-negative staphylococci. Lancet Infect Dis. 2002;2(11):677-685. doi:10.1016/S1473-3099(02)00438-3.
6 Marchant EA, Boyce GK, Sadarangani M, Lavoie PM. Neonatal sepsis due to coagulase-negative staphylococci. Clin Dev Immunol. 2013;2013:586076. doi:10.1155/2013/586076.
7 Berlak N, Shany E, Ben-Shimol S, Chertok IA, Goldinger G, Greenberg D, et al. Late onset sepsis: comparison between coagulase-negative staphylococci and other bacteria in the neonatal intensive care unit. Infect Dis (Lond). 2018;50(10):764-770. doi:10.1080/23744235.2018.1484965.
8 Bhatt P, Patel A, Patel A, Patel S, Ranjan S, Shah S. Prevalence and molecular characterisation of methicillin resistance among coagulase-negative staphylococci at a tertiary care centre. Indian J Med Microbiol. 2016;34(3):301-305.
9 Malakian A, Dehghan S, Abbasi Montazeri E, Aramesh MR, Dehdashtian M, Aletayeb SMH. 16S rRNA-based molecular identification of coagulase-negative Staphylococcus species in neonates with sepsis and their antibiotic resistance patterns in Ahvaz, Iran. BMC Microbiol. 2025;25(1):114. doi:10.1186/s12866-025-03778-8.
10 Simonsen KA, Anderson-Berry AL, Delair SF, Davies HD. Early-onset neonatal sepsis. Clin Microbiol Rev. 2014;27(1):21-47. doi:10.1128/CMR.00031-13.

11 Russell NJ, Barday M, Okomo U, Dramowski A, Sharland M, Bekker A. Early-versus late-onset sepsis in neonates: time to shift the paradigm? Clin Microbiol Infect. 2024;30(1):38-43. doi:10.1016/j.cmi.2023.08.012.
12 Mack D, Fischer W, Krokotsch A, Leopold K, Hartmann R, Egge H, et al. The intercellular adhesin involved in biofilm accumulation of Staphylococcus epidermidis is a linear beta-1,6-linked glucosaminoglycan. J Bacteriol. 1996;178(1):175-183. doi:10.1128/jb.178.1.175-183.1996.
13 Huebner J, Goldmann DA. Coagulase-negative staphylococci: role as pathogens. Annu Rev Med. 1999;50:223-236. doi:10.1146/annurev.med.50.1.223.
14 Krajcinovic SS, Dordevic M, Ruzic Zecevic D, Vukadin M, Milosevic B. Risk factors for neonatal sepsis and method for reduction of blood culture contamination. Malawi Med J. 2015 Mar;27(1):20-4. (6):e2522. doi: 10.4314/mmj.v27i1.6..
15 Iroh Tam PY, Bendel CM. Diagnostics for neonatal sepsis: current approaches and future directions. Pediatr Res. 2017;82(4):574-583. doi:10.1038/pr.2017.134.
16 Adams-Chapman I, Stoll BJ. Prevention of nosocomial infections in the neonatal intensive care unit. Curr Opin Pediatr. 2002;14(2):157-164. doi:10.1097/00008480-200204000-00007.
17 Vergnano S, Menson E, Kennea N, Embleton N, Russell AB, Watts T, et al. Neonatal infections in England: the NeonIN surveillance network. Arch Dis Child Fetal Neonatal Ed. 2011;96(1):F9-F14. doi:10.1136/adc.2009.178798.
18 Goldstein ND, Eppes SC, Ingraham BC, Paul DA. Characteristics of late-onset sepsis in the NICU: does occupancy impact risk of infection? J Perinatol. 2016;36(9):753-757. doi:10.1038/jp.2016.84.
19 Afonso EDP, Blot S. Effect of gestational age on the epidemiology of late-onset sepsis in neonatal intensive care units: a review. Expert Rev Anti Infect Ther. 2017;15(10):917-924. doi:10.1080/14787210.2017.1379394.
20 Becker K, Heilmann C, Peters G. Coagulase-negative staphylococci: pathogenicity and resistance. Clin Microbiol Rev. 2014;27(4):870-926. doi:10.1128/CMR.00109-13.
21 Krediet TG, Jones ME, Janssen K, Gerards LJ, Fleer A. Prevalence of molecular types and mecA gene carriage of coagulase-negative staphylococci in a neonatal intensive care unit: relation to nosocomial septicemia. J Clin Microbiol. 2001;39(9):3376-3378. doi:10.1128/JCM.39.9.3376-3378.2001.
22 Nash C, Chu A, Bhatti M, Alexander K, Schreiber M, Hageman JR. Coagulase-negative staphylococci in the neonatal intensive care unit: are we any smarter? NeoReviews. 2013;14(6):e284-e293. doi:10.1542/neo.14-6-e284.
23 Marchant EA, Boyce GK, Sadarangani M, Lavoie PM. Neonatal sepsis due to coagulase-negative staphylococci. Clin Dev Immunol. 2013;2013:586076. doi:10.1155/2013/586076.