

Antimicrobial Susceptibility and Methicillin Resistance among Coagulase-Negative Staphylococci Isolated from Neonatal Sepsis: Phenotypic–Genotypic Correlation and Implications for Antimicrobial Stewardship

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ABSTRACT

Background: Coagulase-negative staphylococci (CoNS) are important causes of late-onset neonatal sepsis and are increasingly associated with methicillin resistance and multidrug resistance. Because neonates have limited physiological reserve and antimicrobial choices are constrained by toxicity, local susceptibility surveillance is essential. This paper evaluates the antimicrobial susceptibility profile and methicillin-resistance findings in tertiary-care hospital study.

Methods: The study analysed 100 CoNS isolates recovered from blood cultures of clinically suspected neonatal sepsis cases. Species identification and antimicrobial susceptibility testing were performed using the Vitek 2 Compact system. Susceptibility was reported for penicillin G, oxacillin, gentamicin, clindamycin, erythromycin, vancomycin, teicoplanin, linezolid, ciprofloxacin, cotrimoxazole, daptomycin, and tetracycline. Methicillin resistance was assessed phenotypically by oxacillin MIC screening and ceftioxin disk diffusion. Phenotypically resistant isolates were tested for the *mecA* gene by conventional polymerase chain reaction, using a 310-bp amplicon as the positive target.

Results: All 100 isolates were susceptible to vancomycin, linezolid, and daptomycin; 94% were susceptible to teicoplanin, 80% to tetracycline, and 69% to cotrimoxazole. Susceptibility was lower for gentamicin at 53%, penicillin G and oxacillin at 52% each, ciprofloxacin at 33%, clindamycin at 29%, and erythromycin at 20%. The study reported high resistance to erythromycin and clindamycin and moderate resistance to ciprofloxacin, penicillin, oxacillin, and cotrimoxazole. Phenotypic testing identified 48% of isolates as methicillin resistant by both oxacillin MIC screening and ceftioxin disk diffusion. Among these 48 isolates, 42 (87.5% of phenotypically resistant isolates; 42% of all CoNS) carried *mecA*. Six phenotypically resistant isolates were *mecA* negative. *S. epidermidis* accounted for most phenotypically resistant isolates and most *mecA*-positive isolates.

Conclusions: The local CoNS antibiogram demonstrates preserved activity of vancomycin, linezolid, and daptomycin but substantial resistance to several commonly used oral and parenteral agents. The 48% phenotypic methicillin-resistance rate and 42% *mecA*-mediated resistance rate support routine resistance surveillance. Ceftioxin disk diffusion and oxacillin MIC screening were concordant in the source study, while molecular testing identified a phenotypic–genotypic discordance that merits further investigation. Results should support, not replace, individualized neonatal treatment, rapid de-escalation, and infection-prevention measures.

Keywords: coagulase-negative staphylococci; neonatal sepsis; antimicrobial susceptibility; methicillin resistance; *mecA*; ceftioxin; oxacillin; antimicrobial stewardship.

INTRODUCTION

Antimicrobial therapy is central to the survival of neonates with suspected sepsis, but the same urgency that saves lives can generate avoidable antimicrobial exposure. Neonatal sepsis often presents with nonspecific signs such as temperature instability, poor feeding, apnoea, lethargy, respiratory distress, or circulatory compromise. Blood culture remains the reference diagnostic test, yet its sensitivity is influenced by blood volume, prior antibiotic administration,

and the low density or intermittent nature of bacteraemia 1. Clinicians therefore frequently begin empirical broad-spectrum therapy before culture results are available, creating a narrow window in which microbiology must guide refinement of treatment.

This challenge is particularly important for CoNS. They are commensal organisms of skin and mucous membranes but also opportunistic pathogens in premature infants, especially those with central venous catheters, endotracheal tubes, parenteral nutrition, and prolonged hospitalization. Their capacity to form biofilms on foreign material promotes persistence and reduces antimicrobial penetration 11 12. CoNS are also reservoirs of antimicrobial-resistance determinants, including the *mecA* gene, which encodes the low-affinity penicillin-binding protein PBP2a. PBP2a allows cell-wall synthesis to continue despite exposure to most beta-lactam antibiotics 2 13.

Methicillin resistance has consequences beyond the failure of penicillin or oxacillin. In clinical practice, a methicillin-resistant staphylococcus is generally considered resistant to most beta-lactams, including many cephalosporins and carbapenems, even when an individual in vitro result may appear favorable. This sharply narrows the role of first-line beta-lactam therapy and often leads to consideration of glycopeptides or other reserve agents. Vancomycin remains a major treatment option for serious MR-CoNS infection, but its nephrotoxicity, need for therapeutic monitoring, and ecological importance make indiscriminate use undesirable 3.

Reliable detection is therefore essential. Cefoxitin disk diffusion is widely used as a surrogate marker for *mecA*-mediated resistance because it induces expression of the resistance phenotype more consistently than oxacillin in many staphylococci 14 17. Oxacillin MIC methods provide a quantitative alternative, while PCR detection of *mecA* identifies the principal genetic determinant directly. However, phenotype and genotype are not always identical. A resistant phenotype without detectable *mecA* may reflect alternative mechanisms, technical factors, heteroresistance, other target genes such as *mecC*, or limitations in the assay. Conversely, a gene may be present but expressed weakly or not at the level expected from a routine susceptibility test 4 15.

Local antibiograms are indispensable because resistance varies by hospital, unit, species, specimen type, and patient population 16 19 20. A national or international estimate cannot substitute for NICU-specific data. The supplied study document provides a detailed susceptibility profile for 100 CoNS isolates recovered from neonatal sepsis cases and includes phenotypic and genotypic methicillin-resistance results. It reports preserved susceptibility to vancomycin, linezolid, daptomycin, and teicoplanin, alongside substantial resistance to erythromycin, clindamycin, ciprofloxacin, penicillin, and oxacillin.

The objectives of this paper are to describe the reported CoNS antibiogram; quantify the phenotypic and *mecA*-associated methicillin-resistance burden; examine the species distribution of resistant isolates; assess the concordance between oxacillin MIC, cefoxitin disk diffusion, and PCR; and translate the findings into practical recommendations for antimicrobial stewardship and NICU infection prevention.

MATERIALS AND METHODS

Study design and isolates

The analysis is based on the prospective cross-sectional institutional study. The study was conducted in the Department of Microbiology at Index Medical College Hospital & Research Centre, Indore, Madhya Pradesh. Blood samples were collected from clinically suspected neonatal sepsis cases before initiation of antimicrobial therapy where possible. One hundred CoNS isolates were included in the final analytical cohort after exclusion of samples yielding other organisms and isolates regarded as contaminants according to the clinical and laboratory criteria described in the study.

Identification and antimicrobial susceptibility testing

Blood culture bottles were incubated in the BacT/ALERT system, and positive bottles were sub cultured on sheep blood agar. Preliminary identification used Gram staining, catalase testing, and slide and tube coagulase tests. Final identification and antimicrobial susceptibility testing were performed with the Vitek 2 Compact 60 system. The reported antimicrobial panel included penicillin G, oxacillin, gentamicin, clindamycin, erythromycin, vancomycin, teicoplanin, linezolid, ciprofloxacin, cotrimoxazole, daptomycin, and tetracycline.

Phenotypic detection of methicillin resistance

A cefoxitin disk containing 30 µg was applied to a standardized 0.5 McFarland suspension lawned on Mueller–Hinton agar. Plates were incubated at 37°C for 24 hours. According to the breakpoint, an inhibition zone of 24 mm or less was interpreted as methicillin resistant and 25 mm or more as susceptible. Oxacillin MIC screening was performed using the Vitek 2 system. Both methods were used as phenotypic indicators of methicillin resistance.

Detection of *mecA* by PCR

All isolates classified as methicillin resistant by phenotypic testing were subjected to PCR. DNA was extracted by a dry-heat method from fresh subcultures. The reported primers were: forward 5'-GTAGAAATGACTGAACGTCCGATAA-3' and reverse 5'-CCAATTCCACATTGTTTCGGTCTAA-3'. The

amplification protocol included initial denaturation, 35 cycles of denaturation, annealing, and extension, followed by a final extension. PCR products were electrophoresed on 2% agarose gel, and a distinct 310-bp band was interpreted as positive for *mecA*.

RESULTS

Overall susceptibility profile

The most favourable results were observed for vancomycin, linezolid, and daptomycin, with 100% susceptibility among the 100 CoNS isolates. Teicoplanin susceptibility was 94%. Tetracycline and cotrimoxazole showed susceptibility of 80% and 69%, respectively. Gentamicin susceptibility was reported as 53%, whereas penicillin G and oxacillin each showed 52% susceptibility. The least favourable results were observed for ciprofloxacin at 33%, clindamycin at 29%, and erythromycin at 20%.

Antimicrobial agent	Susceptible isolates or percentage reported	Resistance reported
Vancomycin	100/100 (100%)	0%
Linezolid	100/100 (100%)	0%
Daptomycin	100/100 (100%)	0%
Teicoplanin	94/100 (94%)	3%
Tetracycline	80/100 (80%)	20%
Cotrimoxazole	69/100 (69%)	31%
Gentamicin	53/100 (53%)	30%
Penicillin G	52/100 (52%)	48%
Oxacillin	52/100 (52%)	48%
Ciprofloxacin	33/100 (33%)	59%
Clindamycin	29/100 (29%)	71%
Erythromycin	20/100 (20%)	79%

The study classified erythromycin and clindamycin resistance as high. Resistance to ciprofloxacin, penicillin G, oxacillin, and cotrimoxazole was classified as moderate. Tetracycline and gentamicin were described as having low resistance in the source report, although the gentamicin table does not provide a complete distribution across susceptible, intermediate, and resistant categories.

Staphylococcus epidermidis susceptibility

S. epidermidis accounted for 56 of the 100 CoNS isolates. Its susceptibility pattern broadly mirrored the overall CoNS profile. All 56 isolates were susceptible to vancomycin, linezolid, and daptomycin. Susceptibility to teicoplanin was 91.07%, tetracycline 80.35%, cotrimoxazole 73.21%, gentamicin 60.71%, penicillin G 44.64%, oxacillin 44.64%, ciprofloxacin 37.5%, and clindamycin 37.5%. Erythromycin had the lowest susceptibility at 21.42%.

Antimicrobial	<i>S. epidermidis</i> susceptible	<i>S. epidermidis</i> resistant
Vancomycin	56/56 (100%)	0%
Linezolid	56/56 (100%)	0%
Daptomycin	56/56 (100%)	0%
Teicoplanin	51/56 (91.07%)	3.57%
Tetracycline	45/56 (80.35%)	19.64%
Cotrimoxazole	41/56 (73.21%)	26.78%
Gentamicin	34/56 (60.71%)	16.07%

Antimicrobial	<i>S. epidermidis</i> susceptible	<i>S. epidermidis</i> resistant
Penicillin G	25/56 (44.64%)	55.35%
Oxacillin	25/56 (44.64%)	55.35%
Ciprofloxacin	21/56 (37.5%)	51.78%
Clindamycin	21/56 (37.5%)	62.5%
Erythromycin	12/56 (21.42%)	76.78%

Phenotypic methicillin resistance

Oxacillin MIC screening identified 48% of the 100 CoNS isolates as resistant and 52% as susceptible. Cefoxitin disk diffusion produced the same distribution: 52 resistant and 48 susceptible according to the source report. The document states that the two phenotypic methods were concordant. The 48 phenotypically resistant isolates included 31 *S. epidermidis* isolates, 11 *S. haemolyticus*, five *S. hominis*, and one *S. lugdunensis* 21 22.

Species	Phenotypically methicillin-resistant isolates	Percentage of 48 resistant isolates
<i>S. epidermidis</i>	31	64.58%
<i>S. haemolyticus</i>	11	22.91%
<i>S. hominis</i>	5	10.41%
<i>S. lugdunensis</i>	1	2.08%
Total	48	100%

mecA detection and phenotype–genotype comparison

Conventional PCR detected *mecA* in 42 of the 48 phenotypically resistant isolates, equivalent to 87.5% of phenotypically resistant isolates and 42% of the full 100-isolate cohort. Six isolates were phenotypically resistant but *mecA* negative. Among the 42 *mecA*-positive isolates, 25 were *S. epidermidis* (59.52%), 11 were *S. haemolyticus* (26.19%), five were *S. hominis* (11.9%), and one was *S. warneri* (approximately 2.38%).

The distribution of *mecA*-positive isolates differs slightly from the distribution of phenotypically resistant isolates because one *S. lugdunensis* isolate was phenotypically resistant but not included among the *mecA*-positive results, while one *S. warneri* isolate was *mecA* positive. These observations reinforce that species-level characterization and molecular confirmation can add information beyond a single resistance phenotype.

DISCUSSION

The principal finding is a substantial methicillin-resistance burden among CoNS recovered from neonatal sepsis cases. Phenotypic resistance was present in 48% of isolates, while 42% carried *mecA*. Even the lower of these two estimates indicates that nearly one in two isolates would not be suitable for routine beta-lactam therapy if the isolate represented true bloodstream infection. The result is clinically important because CoNS are common in LOS, where empirical treatment may need to account for both Gram-negative organisms and resistant Gram-positive organisms.

The susceptibility profile also demonstrates that resistance is not confined to methicillin. Erythromycin and clindamycin showed the highest reported resistance, at 79% and 71%, respectively. Ciprofloxacin resistance was reported at 59%, while penicillin G and oxacillin resistance were each 48%. These findings are consistent with the broader observation that CoNS readily accumulate resistance determinants under hospital antimicrobial pressure. A 2020 study of clinical CoNS isolates found high *mecA* carriage and substantial resistance in intensive-care and foreign-body-related specimens, while retaining activity of vancomycin, linezolid, and teicoplanin 5. The exact percentages cannot be transferred between hospitals, but the pattern supports local susceptibility testing rather than reliance on historical assumptions.

The 100% susceptibility to vancomycin, linezolid, and daptomycin is reassuring but should not be interpreted as a reason to use these agents indiscriminately 23. Vancomycin is valuable for serious MR-CoNS infection, yet it has nephrotoxic potential and exerts selection pressure. Linezolid is generally reserved for selected situations because of its pharmacological and toxicity considerations. Daptomycin is not routinely used for every neonatal bloodstream

infection and may be limited by age-specific evidence, pharmacokinetics, and clinical context. The central stewardship principle is therefore to use the narrowest effective agent for the shortest evidence-supported duration after the organism, clinical significance, source, and susceptibility profile are known [6](#).

The concordance between oxacillin MIC screening and cefoxitin disk diffusion is operationally useful. Laboratories in resource-limited settings may not have immediate molecular capacity, whereas cefoxitin testing is relatively accessible and can be incorporated into routine susceptibility workflows. The result is consistent with evidence that cefoxitin disk diffusion and oxacillin-based methods can perform well as phenotypic approaches when standardized to current laboratory guidance [5](#) [7](#). Nevertheless, breakpoints, inoculum, incubation conditions, species, and quality-control procedures must be current and validated. [8](#).

The *S. epidermidis* data are particularly relevant because this species accounted for 56% of all isolates and 64.58% of the phenotypically resistant group. Its susceptibility to penicillin G and oxacillin was only 44.64%, while erythromycin susceptibility was 21.42% and clindamycin susceptibility 37.5%. These results suggest that the dominant species also carries much of the local resistance burden. The finding is not surprising: *S. epidermidis* is abundant on human skin, survives in healthcare environments, attaches to foreign material, and can acquire mobile resistance elements. It also means that interventions directed at device-associated transmission may have both epidemiological and antimicrobial-resistance benefits.

The high resistance to macrolides and clindamycin limits their use as reliable empirical alternatives. Clindamycin may be useful for selected susceptible isolates, but inducible resistance must be considered where relevant and tested according to current laboratory standards. Erythromycin resistance can be a marker of broader macrolide resistance and frequently coexists with resistance to other classes. Ciprofloxacin susceptibility at 33% is also too low to support empirical use for neonatal CoNS sepsis. Penicillin G susceptibility at 52% means that half of isolates may remain susceptible, but the result cannot be used before species identification and susceptibility testing, particularly when methicillin resistance is present.

Teicoplanin susceptibility at 94% provides a potential alternative within the glycopeptide class, although neonatal dosing, local availability, and clinical indications must be considered. Tetracycline and cotrimoxazole showed relatively higher susceptibility, but these agents are not automatically interchangeable with first-line neonatal bloodstream-infection therapy. Age-specific safety, pharmacokinetics, tissue penetration, bactericidal activity, and the severity of infection all matter. An antibiogram describes in vitro activity; it does not independently determine the correct regimen for an individual infant.

The study reinforces the need to separate empirical therapy from definitive therapy. Empirical regimens should reflect local patterns and the expected source of infection, while definitive therapy should be narrowed once cultures and susceptibilities are available. If CoNS is judged a contaminant, antibiotics should not be continued solely because a skin commensal was isolated. If the isolate represents true infection, the clinical team should evaluate the device, repeat cultures when appropriate, assess for metastatic infection, and use an agent active against the isolate. Early discontinuation in culture-negative infants with low clinical probability can reduce unnecessary exposure, but the decision must be individualized and guided by serial examination and laboratory data [6](#) [9](#).

The resistance findings also have infection-control implications [18](#). CoNS can colonize neonates, healthcare workers, and the NICU environment. The *mecA* gene is carried on staphylococcal cassette chromosome *mec* elements that can move between strains and species. Thus, MR-CoNS are not merely a treatment problem; they may act as reservoirs of resistance genes within the hospital ecosystem [10](#). Hand hygiene, appropriate barrier precautions, environmental cleaning, line-care bundles, cohorting during outbreaks, and careful device handling are foundational. Routine decolonization should not be introduced without local epidemiological justification because it may create additional selective pressure and has uncertain benefit for all neonatal populations.

A neonatal antimicrobial stewardship program should use the local data in a structured way. First, the laboratory should publish a NICU-specific antibiogram at regular intervals, separating blood isolates from colonizing or non-sterile-site isolates and avoiding duplicate isolates from the same episode. Second, the laboratory should ensure current quality control for cefoxitin, oxacillin, and molecular assays. Third, clinicians and microbiologists should establish criteria for interpreting CoNS-positive cultures, including the number of positive cultures, time to positivity, clinical features, central-line status, and repeat-culture results. Fourth, pharmacy and infection-control teams should audit vancomycin starts, duration, therapeutic monitoring, renal toxicity, and de-escalation. Finally, outcome surveillance should include mortality, duration of bacteremia, line removal, readmission, and neurodevelopmental follow-up where feasible.

CONCLUSION

The tertiary-care study demonstrates a substantial antimicrobial-resistance burden among CoNS isolated from neonatal sepsis cases. Vancomycin, linezolid, and daptomycin retained complete in vitro activity, and teicoplanin remained

highly active, whereas erythromycin, clindamycin, ciprofloxacin, penicillin, and oxacillin showed important resistance. Phenotypic methicillin resistance occurred in 48% of isolates and *mecA* was detected in 42%. Concordant cefoxitin and oxacillin screening supports their routine use when properly standardized, but the six phenotype-positive/*mecA*-negative isolates show why discordant results should be investigated rather than ignored. Local antibiograms, judicious reserve-antibiotic use, rapid de-escalation, and rigorous device-associated infection prevention are necessary to protect effective treatment options for neonates.

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