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Original Research Article

Antibiotic resistance trends and species distribution of coagulase negative *Staphylococci* in a tertiary hospital setting

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ABSTRACT

Background: Coagulase-negative *Staphylococci* (CoNS) have emerged as significant opportunistic pathogens, especially in nosocomial settings. Their increasing resistance to commonly used antibiotics has heightened the need for species-level identification and susceptibility profiling. Aim of this study was to speciate clinical isolates of CoNS, determine their antibiogram and study biofilm formation.
Methods: In this cross-sectional study, 84 clinically significant CoNS isolates were identified by Standard biochemical tests and VITEK 2 system. Antibiotic susceptibility was determined by Kirby-Bauer disc diffusion method following CLSI (Clinical and laboratory standards institute) guidelines. Biofilm formation was assessed by tissue culture plate (TCP) method.
Results: A total 84 clinically significant CoNS isolates were identified from blood, pus, urine, sterile body fluids and catheter tips. Out of 84 isolates, *S. epidermidis* (46.43%) was the most common species, followed by *S. haemolyticus* (22.61%) and *S. saprophyticus* (10.71%). Majority of CoNS were isolated from blood culture (57.15%) followed by pus (21.42%) and urine (14.29%). High resistance was observed against penicillin (92.9%), followed by gentamicin (63.1%), and cotrimoxazole (61.9%). The 54.8% isolates were MRCoNS (Methicillin resistant coagulase negative *Staphylococci*), with 21.42% showing inducible clindamycin resistance (ICR) among the 84 CoNS. All MRCoNS were sensitive to vancomycin and linezolid. Biofilm production was noted in 61.90% of isolates.
Conclusions: Species-level identification and antibiotic resistance profiling of CoNS is crucial in clinical settings to avoid treatment failures. The high prevalence of MRCoNS and biofilm producers necessitates strict infection control and antibiotic stewardship.

Keywords: Coagulase-negative *Staphylococci*, VITEK 2, Antibiotic susceptibility, MRCoNS, Inducible clindamycin resistance, Biofilm

INTRODUCTION

Coagulase-negative *Staphylococci* (CoNS) are commensal inhabitants of skin and mucous membranes of mammals. They may be found in mouth, blood, genitourinary tract and upper respiratory tract of the host.[1]

However, in the recent years, an increased recognition of CoNS from common contaminants to agents of hospital and community acquired infections is noted.[2]

CoNS bacteraemia occurs due to long term use of indwelling central venous catheters, administration of parenteral nutrition and previous antibiotics, patient illness (oncology, burn and high-risk nursery) and other predisposing factors including intensive care unit stay, adherence to infection control practices and handwashing practices of medical staff.[3]

Recent studies have established the role of CoNS as causative agent of infections of blood stream, urinary tract,

surgical sites, deep seated prosthetic implants and infections in seriously ill and immunocompromised patients (ICU patients, premature new born and cancer and transplant patients).[2,4] Several species of CoNS are involved in pathogenesis of various infections like native and prosthetic valve endocarditis, osteomyelitis, peritonitis in patients undergoing ambulatory peritoneal dialysis, infections of cerebrospinal fluid shunts and community acquired and nosocomial wound infections.[5] These infections are difficult to treat because of multidrug resistant behaviour of these organisms.[6]

In addition to this, CoNS possess a loosely bound exopolysaccharide layer called as biofilm which contributes to increased association with infections and reduced antibiotic susceptibility.[3] Therefore, rapid and reliable identification of CoNS at species level becomes the need to predict the potential pathogenicity or antibiotic susceptibility of all the clinical isolates.[7]

METHODS

This cross-sectional study was conducted from June 2022 to July 2024 in the Department of Microbiology, Government Medical College, Nagpur. A total of 84 CoNS isolates with clinical significance were identified in the present study.

Inclusion and exclusion criteria

All the CoNS isolates from various clinical specimens like blood, pus, urine, body fluids (ascitic fluid, pleural fluid, cerebrospinal fluid (CSF) and peritoneal fluid) and catheter tips from patients of all age groups and both the sexes were included in the study while clinical samples yielding polymicrobial growth were excluded.

Identification

All the clinical specimens were processed by standard microbiological techniques. Initial identification was done by colony morphology and Gram staining (Gram-positive cocci in clusters). Genus *Staphylococcus* was identified based on positive catalase test, furazolidone (100 µg) susceptibility and resistance to bacitracin (0.04U). Further, slide and tube coagulase test were performed to identify CoNS.[8]

Speciation

To differentiate between various species of CoNS, we performed standard biochemical tests (ornithine decarboxylase, nitrate reduction, phosphatase, urease production, carbohydrate fermentation of xylose, trehalose, glucose, lactose, sucrose, maltose, mannose, mannitol) and novobiocin (5 µg) susceptibility.[8] All the isolates were also confirmed by VITEK® 2 Compact system Gram positive cards. This system utilizes fluorescence-based biochemical reactions and proprietary algorithms to deliver species-level identifications within 6-8 hours.[9]

Antimicrobial susceptibility testing

Antimicrobial susceptibility was determined by the Kirby-Bauer disc diffusion method on Mueller-Hinton agar, in accordance with the CLSI 2022 standards. Antibiotic discs tested include penicillin (10 units), cefoxitin (30 µg), erythromycin (15 µg), clindamycin (2 µg), cotrimoxazole (1.25/23.75 µg), doxycycline (30 µg), tetracycline (30 µg), linezolid (30 µg), chloramphenicol (30 µg), levofloxacin (5 µg), gentamycin (10 µg), nitrofurantoin (300 µg) and norfloxacin (10 µg). Zone diameters were measured after 18±2 hours of incubation at 35±2°C and interpreted as per the CLSI breakpoints.[10]

Methicillin resistance detection

Cefoxitin resistance (zone≤21 mm for *S. lugdunensis* and zone≤24 mm for *S. epidermidis* and other species of CoNS) was used as a surrogate marker for mecA-mediated methicillin resistance.[10] Isolates resistant to cefoxitin were classified as MRCoNS and retained for further analysis for determination of vancomycin MIC (Minimum inhibitory concentration) using E-strip.[11]

ICR (D-zone test)

To detect erm-mediated ICR, erythromycin (15 µg) and clindamycin (2 µg) discs were placed 15-20 mm apart on agar plates inoculated with test isolates. A blunted (D-shaped) zone adjacent to the clindamycin disc indicates inducible resistance.[10]

Biofilm detection by microtitre plate method

Biofilm formation was assessed by the method of Christensen et al.[12] A single colony from blood agar was inoculated into 2 ml TSB (Trypticase soy broth) with 1% glucose and was incubated overnight at 36±1°C. Then, 200 µl of culture was added to the microtitre plate wells and incubated overnight. The wells were emptied, washed once with PBS (Phosphate buffer saline) of pH 7.2, and fixed with 2% sodium acetate for 10 minutes. After discarding, 0.1% crystal violet was added and kept for 30 minutes. The wells were washed and air-dried for one hour and their OD (optical density) was measured at 620 nm. Biofilm production of CoNS was classified as non-producer (OD≤0.120), moderate (OD of 0.120-0.240) or strong (OD>0.240).[13]

All the data in the study was analyzed using descriptive statistics; species distribution, resistance rates, and biofilm categories were expressed as percentages.

RESULTS

A total of 84 clinically significant CoNS were isolated from various clinical specimens, of which the maximum

number of isolates were obtained from blood cultures (57.15%), followed by pus (21.42%) and urine (14.29%). Other specimens were ascitic fluid (1.19%), pleural fluid (1.19%), CSF (1.19%), peritoneal fluid (1.19%), ear discharge (1.19%) and DLC (Double lumen catheter) tip culture (1.19%). *S. epidermidis* (46.43%) was the most common species causing CoNS infection followed by *S. hemolyticus* (22.61%) and *S. saprophyticus* (10.71%). *S. lugdunensis*, *S. hominis hominis* and *S. xylosus* each represented 4.77% of the total CoNS isolates, while 2.39% were identified as *S. warneri* and *S. cohnii*, and *S. capitis* was identified in 1.19%.

Most commonly isolated species from blood was *S. epidermidis* (43.76%), followed by *S. hemolyticus* (22.91%), *S. hominis hominis* (8.34%), *S. xylosus* (8.34%), *S. lugdunensis* (6.26%) and others. Abscess and wound infections were also mainly due to *S. epidermidis* (55.56%), followed by *S. hemolyticus* (38.89%) and *S. lugdunensis* (5.55%). CoNS isolates identified from urine were mostly *S. saprophyticus* (75%) and *S. epidermidis* (25%). One isolate of *S. epidermidis* was also isolated each from other specimens like ascitic fluid, pleural fluid, CSF, ear discharge and DLC tip. However, one isolate of *S. hemolyticus* was isolated from the peritoneal fluid (Table 1).

Out of 84 CoNS isolates, 52 strains were positive for biofilm formation and 32 were categorized as non-biofilm producers by microtitre plate method. Out of the 52 positives, 38.10% of CoNS were strong biofilm producers and the 23.80% were moderate biofilm producers (Figure 1).

Biofilm formation was maximum by *S. epidermidis* (53.8%) followed by *S. hemolyticus* (21.1%) and *S. saprophyticus* (11.5%) by microtitre plate method. Biofilm formation was also shown by other species of CoNS like *S. lugdunensis*, *S. hominis hominis*, *S. xylosus*, *S. warneri* and *S. cohni cohni* (Table 2).

Out of 84 isolates, 92.9% were resistant to penicillin, followed by 63.1% to gentamycin, 61.9% to cotrimoxazole, 57.2% to tetracycline and 52.4% to levofloxacin. Methicillin resistance was observed in 54.8% isolates, referred as MRCoNS. Resistance to erythromycin was 51.2% and clindamycin was 45.3% while ICR was shown by 18 (21.42%) CoNS isolates.

All the isolates were sensitive to linezolid. Among the 12 urinary isolates tested, norfloxacin resistance was found in 50% and the resistance to nitrofurantoin was 25% (Figure 2).

MIC for vancomycin was tested only for MRCoNS. It was observed that all the 46 MRCoNS were sensitive to vancomycin with 28 (60.8%) isolates showing MIC 2 µg/ml and 18 (39.1%) isolates showing MIC 4 µg/ml.

Table 1: Species wise distribution of CoNS in various clinical specimens.

Species	Blood, n=48 (57.15%)	Pus, n=18 (21.42%)	Urine, n=12 (14.29%)	Others, n=6 (7.14%)	Total, n=84 (100%)
S. epidermidis	21 (43.76)	10 (55.56)	3 (25)	5 (83.3)*	39 (46.4)
S. haemolyticus	11 (22.91)	7 (38.89)	-	1(16.7) **	19 (22.6)
S. saprophyticus	-	-	9 (75)	-	9 (10.7)
S. lugdunensis	3 (6.26)	1 (5.55)	-	-	4 (4.7)
S. hominis hominis	4 (8.34)	-	-	-	4 (4.7)
S. xylosus	4 (8.34)	-		-	4 (4.7)
S. warneri	2 (4.16)	-	-	-	2 (2.3)
S. cohni cohni	2 (4.16)	-	-	-	2 (2.3)
S. capitis	1 (2.08)	-	-	-	1 (1.2)

*Five other specimen include ascitic fluid, pleural fluid, CSF, ear discharge and DLC tip one each. **Peritoneal fluid (1). These fluids make 1.19% each of the total CoNS isolates.

Table 2: Biofilm formation by various species of CoNS.

CoNS species, n=84	Biofilm producer isolates, n=52 (%)
***S. epidermidis*, (n=39)**	28 (53.8)
***S. haemolyticus*, (n=19)**	11 (21.1)
***S. saprophyticus*, (n=9)**	06 (11.5)
***S. lugdunensis*, (n=4)**	02 (3.8)
***S. hominis hominis*, (n=4)**	01 (2)
***S. xylosus*, (n=4)**	02 (3.8)
***S. warneri*, (n=2)**	01 (2)
***S. cohni cohni*, (n=2)**	01 (2)
***S. capitis*, (n=1)**	00 (0)

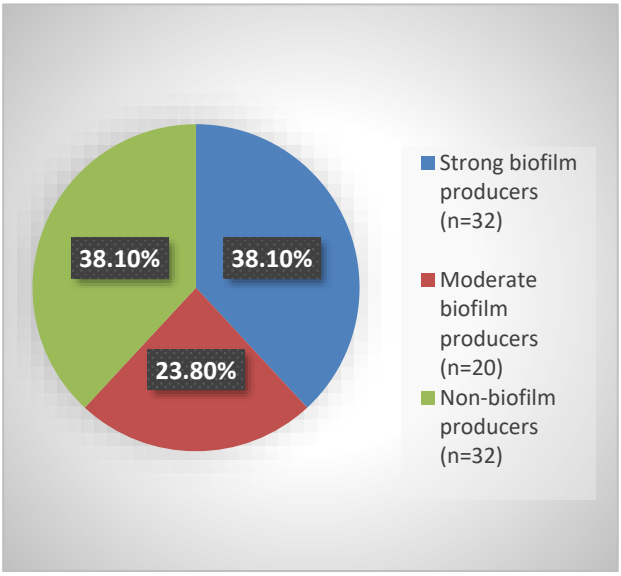

Figure 1: Strength of biofilm formation in CoNS isolates, (n=84).

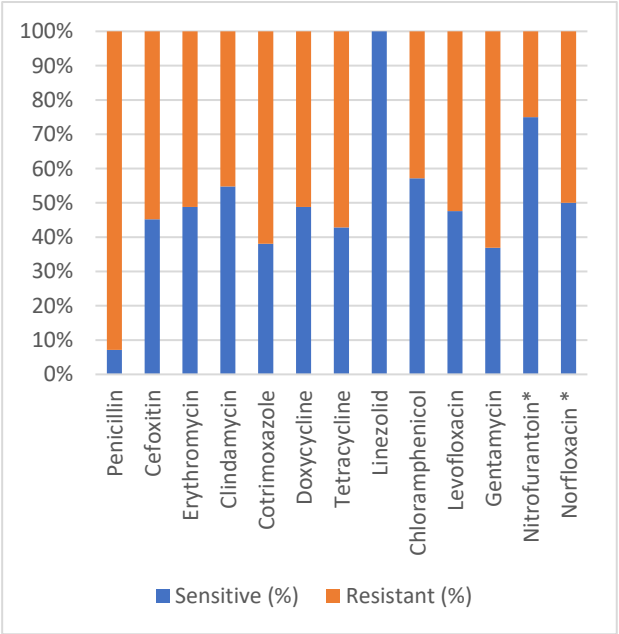

Figure 2: Antibiogram of clinical isolates of CoNS.
*Nitrofurantoin and Norfloxacin were tested only for urinary isolates of CoNS i. e. 12 isolates.

DISCUSSION

In clinical laboratories, identification of *Staphylococci* is often limited to screening for *Staphylococcus aureus*, while isolates that are not *S. aureus* are typically reported collectively as CoNS. However, with the growing recognition of the pathogenic potential of CoNS, understanding the epidemiology and virulence of individual species has become increasingly important. As a result, the need for rapid and precise species-level identification of CoNS has gained prominence in recent years.[7,14]

In the present study, 57.15% of the CoNS were isolated from blood cultures, 21.42% from pus, 14.29% from urine, 1.19% each from ascitic fluid, pleural fluid, CSF, peritoneal fluid, ear discharge and DLC tip culture. Similar results were published by Usha et al who reported that out of 102 CoNS, blood (54) was the most common source for CoNS isolates, followed by pus (32), and urine (12).[6] Also, Chavan et al in her study reported that 54.9% of the CoNS were isolated from blood cultures, 26.5% from pus and 15.8% from urine.[15] However contrasting results were published by Singh et al who reported 88 (65.2%) CoNS isolates from urine, 37 (27.5%) from blood, 3 (2.2%) from pus, followed by 2 (1.5%) from catheter tip, 2 (1.5%) from wound swab, 1 (0.7%) each from aural swab, sputum and ascitic fluid.[7] Also, Roopa et al reported that out of 112 CoNS, 87 (77.6%) were isolated from wounds, 7 (6.2%) from sputum, 6 (5.3%) each from blood and urine, 2 (1.7%) each from body fluids, throat swabs and ear swabs.[1] The differences observed in the distribution of CoNS among clinical specimens in different studies may have resulted due to variable number of clinical specimens involved in the studies.

In this study, most common species of CoNS isolated from all the sources were *S. epidermidis* (46.43%), followed by *S. haemolyticus* (22.61%), *S. saprophyticus* (10.71%), *S. lugdunensis* (4.77%), *S. hominis hominis* (4.77%), *S. xylosus* (4.77%), *S. warneri* (2.39%), *S. cohni cohni* (2.39%) and *S. capitis* (1.19%). This distribution pattern correlates well with the results published by Bora et al where *S. epidermidis* was 46%, *S. haemolyticus* was 28.2% and *S. lugdunensis* was 30.8%.[16] Also, in studies by Roopa and Kavitha *S. epidermidis* constituted the predominant species; 50.8% and 46.84% respectively.[1,17] However, contrasting findings were observed in a study by Raina et al which shows predominance of *S. haemolyticus* (25%) and *S. warneri* (20%), followed by *S. epidermidis* (11.67%), *S. simulans, S.capitis. S. cohnii*, *S. lugdunensis*, *S. schleferi* and *S. lentus*.[18] The identification of species increases knowledge of the pathogenicity of the various species of CoNS, providing useful epidemiologic information and contributing to isolate being clinically significant.

This study demonstrated that out of 48 CoNS isolated from blood, 43.76% isolates were *S. epidermidis*, 22.91% were *S. haemolyticus*, 8.34% each were *S. hominis hominis* and *S. xylosus*, 6.26% were *S. lugdunensis*, 4.16% each were *S. cohni cohni* and *S. warneri*; and 2.08% were *S. capitis*; this data correlates well with the findings by Shreshta et al, in which *S. epidermidis* was the most common species of CoNS isolated from blood cultres.[19] In a study by Dilip et al out of 55 CoNS isolated from blood, 28 were *S. epidermidis* and 18 were *S. haemolyticus* followed by other species of CoNS while out of 32 pus swabs, *S. epidermidis* and *S. hemolyticus* were 14 and 10 respectively followed by other CoNS species.[20] Also, majority of the CoNS isolated from urine samples were *S. saprophyticus* (75%), followed by *S. epidermidis* (25%). In a study by Dilip et al all the 5 CoNS isolated from urine

were *S. saprophyticus*.[20] Also, one isolate of *S. epidermidis* was isolated from ear discharge in our study which also correlates with the study by Dilip et al.[20] In a study by Raina et al 3 CoNS were isolated from CSF which were pathogenic, and we have isolated one clinically significant CoNS from CSF.[18]

Bacteria that are directly or indirectly attached to the polymer surface produce biofilm which is an extracellular polysaccharide material that is amorphous and mucoid. This is believed to be the primary mechanism underlying both auto-aggregation and bacterial adhesion to plastic surfaces. The colonizing and virulence factor, i.e. biofilm formation was evaluated in this study. Fifty-two (61.9%) out of 84 CoNS has produced biofilm in this study. Comparable results of biofilm formation have been previously published by, Halim et al and Mathur et al which was found to be 47% and 53.9% respectively.[13,21] In contrast, biofilm producing species of CoNS were more in studies by Shrestha et al, Oliveria et al and Meriem et al which were 71.8 %, 73% and 83% respectively.[19,22,23] In our study, strong biofilm producer CoNS were 38.1% and 23.8% CoNS were moderate biofilm producers and in a study by Halim et al 14.4% CoNS were strong biofilm producers while 39.4% were moderate producers.[13]

In this study, among the biofilm producer species of CoNS, *S. epidermidis* (53.8%), *S. haemolyticus* (21.1%) and *S. saprophyticus* (11.5%) were the first, second and third highest species followed by other species of CoNS like *S. lugdunensis*, *S. hominis hominis*, *S. xylosus*, *S. warneri* and *S. cohni cohni* whereas no biofilm production was reported by *S. capitis*. Similarly, in a study by Manandhar et al biofilm production was determined quantitatively by TCP method that demonstrated strong and moderate biofilm producers in 35 (16.4%) and 55 (25.7%) CoNS isolates respectively.[24] However, in a study by Raina et al biofilm production by TCP method was highest in *S. hemolyticus* (57.7%), followed by *S. epidermidis* (21.2%) and *S. hominis* (19.2%).[25]

Antibiotic susceptibility testing has shown variability in which out of 84 CoNS isolates, 92.9% were resistant to penicillin, followed by gentamycin (63.1%), cotrimoxazole (61.9%), tetracycline (57.2%) and levofloxacin (52.4%). Resistance to erythromycin was 51.2% and to clindamycin it was 45.3% while no resistance to linezolid was observed. MRCoNS (Methicillin-resistant CoNS) were 54.8% and MSCoNS (Methicillin-susceptible CoNS) were 45.2% and all the 46 MRCoNS were sensitive to vancomycin. Among the 12 urinary CoNS isolates tested for antibiotic susceptibility, norfloxacin resistance was found in 50% and nitrofurantoin in 25% isolates.

In a study by Dilip out of 110 CoNS isolates, penicillin resistance was shown by 95.5% of isolates, followed by erythromycin (80.9%), clindamycin (52.7%), cotrimoxazole (36.3%) and gentamycin (5.26%) while methicillin resistance was shown by 17.3% isolates.[20] Similarly, in a study by Manandhar et al penicillin resistance was observed in 91.6% of CoNS isolates.[24] And in a study by Singh et al MRCoNS were 58.5% and MSCoNS were 41.5% whereas no resistance to linezolid and vancomycin was detected which correlates with the findings of our study.[7] Golia et al in their study reported the antibiotic susceptibility testing of CoNS isolates which showed maximum resistance to penicillin (95.5%), followed by erythromycin (71.6%), cefoxitin (66.4%), gentamicin (24.6%), linezolid 23 (17.2%) and levofloxacin 9 (6.7%).[26] In a study by Singh et al out of 59 CoNS, 57.6% were MRCoNS with an overall high prevalence of resistance to the non-β-lactam antimicrobials was observed including clindamycin (69.4%) erythromycin (62.7%), ciprofloxacin (45.7%) and cotrimoxazole (40.7%); also, all the isolates were susceptible to vancomycin and linezolid.[2] In various studies done by Kashid et al, Raina et al and Manandhar et al the MRCoNS were 35.7%, 46.6% and 66.8% respectively while in a study conducted by Al-Janabi et al MRCoNS were 96.8%.[18,24,27,28] In a study by Nasaj MRCoNS were 55% and MSCoNS were 45% out of 91 CoNS isolates and all CoNS species (both MRCoNS and MSCoNS) were sensitive to vancomycin.[29] However, in a study by Geetanjali et al vancomycin sensitive CoNS were only 69%.[30]

Our study reported ICR in 18 (21.42%) CoNS isolates from which 11 (13.09%) were MRCoNS and 7 (8.33%) were MSCoNS. In a study by Thapa et al, out of 51 CoNS isolates, 39.21% of isolates reported ICR from which 43% were MRCoNS and in a study by Yilmaz et al 25.7% MRCoNS were inducible clindamycin resistant.[31,32] However, in a study by Kalbhor et al 11 MRCoNS and 31 MSCoNS were identified of which inducible clindamycin resistant isolates were 5 and 8 respectively.[33]

Limitations

This is a single centre study. Molecular methods of gene detection for antibiotic resistance were not performed in this study.

CONCLUSION

In conclusion, CoNS, long regarded as mere commensals or contaminants in clinical specimens, are now increasingly recognized as significant opportunistic and nosocomial pathogens. Different CoNS species are linked to specific infection types, biofilm (slime) formation capabilities, and distinctive antibiotic resistance patterns. Therefore, precise species-level identification, assessment of biofilm production, and antimicrobial susceptibility testing are essential to better understand their pathogenic potential and epidemiological significance. It is crucial not to overlook CoNS as trivial isolates, especially when recovered from valuable clinical samples, as accurate interpretation can greatly benefit patient management and outcomes.

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