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PREVALENCE OF ERYTHROMYCIN AND INDUCIBLE CLINDAMYCIN RESISTANCE IN STAPHYLOCOCCI ISOLATED FROM CLINICAL SAMPLES IN WESTERN NEPAL: IMPLICATIONS FOR ANTIMICROBIAL STEWARDSHIP

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ABSTRACT

Background

Staphylococci are major opportunistic pathogens with increasing antimicrobial resistance. Inducible clindamycin resistance (iMLSB) is often undetected by routine susceptibility testing, leading to treatment failure. In Nepal, data on iMLSB are limited and D-testing is not routinely performed.

Objectives

To determine the prevalence of erythromycin resistance and inducible clindamycin resistance among clinical staphylococcal isolates at a tertiary care hospital in western Nepal, and to evaluate methicillin resistance and multidrug resistance patterns.

Methods

A cross-sectional study was conducted from May 2024 to April 2025. A total of 374 non-duplicate staphylococcal isolates were identified by standard microbiological methods. Antimicrobial susceptibility testing was performed by Kirby-Bauer disc diffusion as per CLSI 2024 guidelines. Methicillin resistance was detected using cefoxitin, and inducible clindamycin resistance was detected by the D-test.

Results

Staphylococcus aureus accounted for 293 (78.3%) isolates and coagulase-negative staphylococci (CoNS) for 81 (21.7%). Methicillin resistance was present in 57.7% of *S. aureus* (MRSA) and 63.0% of CoNS (MRCoNS). Multidrug resistance was observed in 43.7% of *S. aureus* and 38.3% of CoNS. Erythromycin resistance was 56.3% in *S. aureus* and 60.5% in CoNS. Among all isolates, the D-test revealed iMLSB in 35.6%, cMLSB in 43.6%, and MS phenotype in 20.9%. All isolates were susceptible to linezolid and vancomycin.

Conclusions

There is a high prevalence of methicillin resistance, multidrug resistance, and inducible clindamycin resistance among staphylococcal isolates. Routine D-zone testing is essential to detect iMLSB and avoid clindamycin treatment failure.

Keywords: *Coagulase-negative staphylococci, D-test, Inducible clindamycin resistance, Staphylococcus aureus, MLSB phenotype*

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INTRODUCTION

Staphylococci, notably *S. aureus* and CoNS, are both common skin and mucous membrane commensals and clinically important opportunistic pathogens. They cause infections ranging from superficial skin and soft tissue infections to life-threatening conditions such as bacteraemia, infective endocarditis, and septic shock [1]. Their importance is increased by transmissibility, diverse virulence factors, and strong ability to acquire antimicrobial resistance, making them a major and persistent global public health concern across healthcare and community settings [2]. Both healthcare-associated and community-acquired infections are common, with asymptomatic colonization contributing significantly to transmission dynamics in hospitals and the community [3].

Methicillin-resistant staphylococci (MRS), carrying *mecA* or *mecC* genes and detected using cefoxitin testing, are now endemic in healthcare settings worldwide. CLSI interpretive criteria recommend cefoxitin disc diffusion as a reliable and standardized method for detecting methicillin resistance in staphylococci in routine laboratories [5]. These strains are often multidrug resistant (MDR), limiting available therapeutic options and complicating clinical management and infection control practices [3,4].

The burden of methicillin-resistant *Staphylococcus aureus* (MRSA) remains high in low- and middle-income countries, including Nepal. A Nepal study reported significant MRSA prevalence and concerning resistance patterns, indicating ongoing circulation in both hospital wards and community settings [6]. This has increased reliance on macrolides and lincosamides as important alternative agents for empirical and targeted therapy [7].

Erythromycin and clindamycin inhibit bacterial protein synthesis by binding to the 50S ribosomal subunit and are widely used when β-lactams cannot be used or are contraindicated [7]. However, resistance to these agents is increasing globally and has become a clinically significant therapeutic challenge requiring continuous monitoring.

Macrolide-lincosamide resistance is mainly mediated by *erm* genes causing methylation of the 23S rRNA component of the 50S ribosomal subunit, producing MLSB cross-resistance [8]. This may be constitutive (cMLSB) or inducible (iMLSB), where clindamycin resistance appears only in the presence of erythromycin induction [9]. Failure to detect inducible resistance may lead to inappropriate therapy and clinical treatment failure [10].

Another mechanism involves *msrA* efflux pumps, causing resistance to macrolides and streptogramin B while preserving clindamycin susceptibility (MS phenotype) [11]. Differentiating these phenotypes is essential for appropriate antimicrobial selection and optimizing patient outcomes in clinical practice.

MLSB resistance varies geographically and is influenced by antibiotic use, prescribing behavior, and infection control practices [12]. Studies show rising inducible clindamycin resistance, especially in settings with high macrolide consumption and inadequate stewardship [13,14]. In Nepal, data are limited and routine D-test performance is inconsistent in many diagnostic laboratories, increasing the risk of missed iMLSB detection and therapeutic failure [15]. This study aimed to determine erythromycin resistance and inducible clindamycin resistance among clinical staphylococcal isolates in a tertiary hospital in western Nepal. Methicillin resistance and multidrug resistance patterns were also assessed to provide a comprehensive resistance profile supporting evidence based empirical therapy, rational antibiotic use, and strengthening antimicrobial stewardship programs in the region in tertiary care hospital settings of western Nepal region.

METHODS

This cross-sectional descriptive study was conducted in the Microbiology Laboratory of Nepalgunj Medical College, Kohalpur, Banke, Nepal, a tertiary care teaching hospital affiliated with Kathmandu University. The study was carried out over a 12-month period from May 2024 to April 2025.

Ethical clearance was obtained from the Institutional Review Committee of Nepalgunj Medical College (Ref: 46/080–081). Written informed consent was obtained from all adult participants, while assent along with parental or guardian consent was obtained for minors prior to sample collection.

Sample Size and Sampling Technique

A total of 374 non-duplicate staphylococcal isolates were included in the study. The sample size was estimated based on an anticipated prevalence of inducible clindamycin resistance of approximately 30% from preliminary data at the study site, with a 95% confidence interval and 5% margin of error. A census sampling technique was employed, wherein all eligible clinical samples received during the study period were included.

Clinical specimens including pus, wound swabs, high vaginal swabs, blood, urine, sputum, and other body fluids from patients of all age groups and both sexes attending outpatient and inpatient departments

were included if they yielded significant growth of staphylococci. Only the first isolate from each patient was considered. Samples showing mixed growth, insignificant growth, improper labeling, or leakage were excluded. Patients who had received antibiotics within the 48 hours of sample collection were also excluded.

Isolation and Identification of Isolates

All specimens were processed according to standard microbiological procedures [16]. Urine samples were inoculated onto cysteine lactose electrolyte deficient (CLED) agar and incubated aerobically at 37 °C for 24 hours. Pus, swabs, sputum, and body fluids were cultured on MacConkey agar (without crystal violet) and 5% sheep blood agar and incubated at 37 °C for 24–48 hours. Blood samples were inoculated into brain heart infusion (BHI) broth at a 1:10 ratio, incubated at 37 °C, and subcultured onto MacConkey and blood agar at 24 hours, 48 hours, and on day 7.
Isolates were identified as staphylococci based on colony morphology, Gram staining (Gram-positive cocci in clusters), catalase positivity, and coagulase testing (both slide and tube methods). *Staphylococcus aureus* was confirmed by positive catalase and coagulase tests, mannitol fermentation on mannitol salt agar, and pigment production on nutrient agar. Coagulase-negative staphylococci (CoNS) were identified by negative coagulase tests, lack of mannitol fermentation, and further speciated using conventional biochemical tests including urease, phosphatase, pyrrolidonyl arylamidase, novobiocin susceptibility, and carbohydrate fermentation tests according to the methods described by Kloos and Schleifer [17,18].

Antimicrobial Susceptibility Testing

Antimicrobial susceptibility testing was performed using the Kirby–Bauer disc diffusion method on Mueller-Hinton agar (MHA) in accordance with Clinical and Laboratory Standards Institute (CLSI) guidelines [5]. A standardized bacterial suspension equivalent to 0.5 McFarland was inoculated uniformly onto MHA plates. The following antibiotic discs (HiMedia Laboratories, India) were applied: gentamicin (10 µg), ciprofloxacin (5 µg), clindamycin (2 µg), Co-trimoxazole (1.25/23.75 µg) chloramphenicol (30 µg), linezolid (30 µg), cefoxitin (30 µg), erythromycin (15 µg), vancomycin (30 µg), and tetracycline (30 µg). Novobiocin (30 µg) was used for identification of *S. saprophyticus*. Plates were incubated at 37 °C for 18–24 hours, and inhibition zone diameters were measured and interpreted according to CLSI 2024 breakpoints [5]. Multidrug resistance (MDR) was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial classes [19].

Detection of Methicillin Resistance

Methicillin resistance was determined phenotypically using the cefoxitin disc (30 µg) diffusion method on Mueller-Hinton agar (MHA) following overnight incubation at 37 °C for 24 hours. Zone diameters were interpreted according to Clinical and Laboratory Standards Institute (CLSI) M100 guidelines. Isolates exhibiting a zone of inhibition ≤24 mm were classified as methicillin-resistant (MRSA or MRCoNS), while those with a zone diameter ≥25 mm were considered methicillin-susceptible [19].

Detection of Inducible Clindamycin Resistance (D-Test)

Inducible clindamycin resistance was detected using the disc approximation (D-test) method as described by Fiebelkorn et al. [9]. A 0.5 McFarland suspension of each isolate was inoculated onto MHA plates. Erythromycin (15 µg) and clindamycin (2 µg) discs were placed 15–20 mm apart (edge-to-edge). After incubation at 37 °C for 18–24 hours, the zones of inhibition were examined. Flattening of the clindamycin inhibition zone adjacent to the erythromycin disc, producing a characteristic “D” shape, was interpreted as inducible MLSB (iMLSB) resistance. Isolates resistant to both erythromycin and clindamycin without zone flattening were classified as constitutive MLSB (cMLSB). Isolates resistant to erythromycin but susceptible to clindamycin with a negative D-test were categorized as having the MS phenotype (efflux-mediated resistance) [19].

Data Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics, including frequencies and percentages, were calculated for demographic variables, specimen distribution, antimicrobial resistance patterns, and MLSB phenotypes. Associations between categorical variables were assessed using the chi-square test where appropriate. A p-value of < 0.05 was considered statistically significant.

RESULTS

A total of 374 non-duplicate clinical isolates of staphylococci were included in this study. Of these, 293 (78.3%) were identified as *Staphylococcus aureus*

and 81 (21.7%) as coagulase-negative staphylococci (CoNS).

Among *S. aureus* isolates, 174 (59.4%) were obtained from male patients and 119 (40.6%) from female patients. For CoNS isolates, 38 (46.9%) were from males and 43 (53.1%) from females.

The distribution of staphylococcal isolates across various clinical specimens is summarized in Table 1. Pus and wound swabs were the most common source of *S. aureus* isolates (56.3%), whereas urine samples were the predominant source of CoNS isolates (48.1%).

Table 1: Sample wise distribution of *S. aureus* and CoNS isolates (n=374)

Clinical Specimen	*S. aureus* (n=293) n (%)	CoNS (n=81) n (%)
Pus/Wound Swab	165 (56.3)	17 (21.0)
High Vaginal Swab	54 (18.4)	7 (8.6)
Blood	7 (2.4)	5 (6.2)
Urine	43 (14.7)	39 (48.1)
Sputum	21 (7.2)	12 (14.8)
Body Fluids	3 (1.0)	1 (1.2)

The antibiotic susceptibility patterns of staphylococcal isolates are presented in Table 2. All isolates were susceptible to linezolid and vancomycin (100%). High rates of resistance were observed for cefoxitin and erythromycin. And notably CoNS isolates exhibited higher resistance than *S. aureus* to gentamicin, ciprofloxacin, and tetracycline.

Table 2: Antibiotic susceptibility of *S. aureus* and CoNS isolates (n=374)

Antibiotic (Disc)	*S. aureus* Resistant n (%)	CoNS Resistant n (%)	Total Resistant n (%)
Gentamicin (10 µg)	26 (8.9)	23 (28.4)	49 (13.1)
Ciprofloxacin (5 µg)	89 (30.4)	41 (50.6)	130 (34.8)
Clindamycin (2 µg)	35 (11.94) *	15 (18.5)	50 (13.4)*
Co-trimoxazole (1.25/23.75 µg)	150 (51.2)	28 (34.6)	178 (47.6)
Chloramphenicol (30 µg)	45 (15.4)	12 (14.8)	57 (15.2)
Linezolid (30 µg)	0 (0.0)	0 (0.0)	0 (0.0)
Cefoxitin (30 µg)	169 (57.7)	51 (63.0)	220 (58.8)
Erythromycin (15 µg)	165 (56.3)	49 (60.5)	214 (57.2)
Vancomycin (30 µg)	0 (0.0)	0 (0.0)	0 (0.0)
Tetracycline (30 µg)	13 (4.4)	24 (29.6)	37 (9.9)

*Percentages based on the number of isolates tested for clindamycin.

Among *S. aureus* isolates, 169 (57.7%) were methicillin-resistant (*MRSA*) as determined by cefoxitin disc diffusion. Of the 81 CoNS isolates, 51 (63.0%) were methicillin-resistant (MRCoNS).

Multi-drug resistance (MDR), defined as resistance to at least one agent in three or more antimicrobial classes, was observed in 128 (43.7%) of *S. aureus* isolates and 31 (38.3%) of CoNS isolates.

The D-test was performed to determine the prevalence of MLSB resistance phenotypes. Table 3 summarizes the distribution among MRSA, MSSA, MRCoNS, and MSCoNS.

Table 3: Distribution of MLSB phenotypes among staphylococcal isolates (n=374)

Phenotype	MRSA (n=169) n (%)	MSSA (n=124) n (%)	MR-CoNS (n=51) n (%)	MS-CoNS (n=30) n (%)	Total (n=374) n (%)
iMLSB	67 (39.6)	39 (31.5)	19 (37.3)	8 (26.7)	133 (35.6)
cMLSB	87 (51.5)	32 (25.8)	29 (56.9)	15 (50.0)	163 (43.6)
MS	15 (8.9)	53 (42.7)	3 (5.9)	7 (23.3)	78 (20.9)

Overall, the constitutive MLSB (cMLSB) phenotype was most prevalent (43.6%), followed by inducible MLSB (iMLSB, 35.6%) and the MS phenotype (20.9%). Among MRSA isolates, cMLSB predominated (51.5%), whereas among MSSA, the MS phenotype was most frequent (42.7%). The cMLSB phenotype indicates constitutive resistance to both erythromycin and clindamycin, meaning clindamycin is clinically ineffective against these isolates. Among the 81 CoNS isolates, *S. epidermidis* was most common (45.7%), followed by *S. saprophyticus* (24.7%), *S. haemolyticus* (12.3%), *S. hominis* (8.6%), *S. lugdunensis* (4.9%), and *S. Cohnii* (3.7%).

DISCUSSION

The present study evaluated the prevalence of erythromycin resistance and inducible clindamycin resistance among clinical isolates of staphylococci in a tertiary care hospital in western Nepal, alongside methicillin resistance and overall antimicrobial susceptibility patterns. These findings provide important insights for guiding empirical therapy and strengthening antimicrobial stewardship, particularly in resource limited settings where routine D-testing is not consistently performed [15]. The results highlight a concerning burden of antimicrobial resistance among both *Staphylococcus aureus* and coagulase-negative staphylococci (CoNS), reinforcing the need for continuous surveillance and evidence based clinical decision making.

In this study, *Staphylococcus aureus* accounted for the majority of isolates (78.3%), with CoNS comprising 21.7%, a distribution consistent with previous reports from Nepal and similar healthcare

settings [21]. This predominance reflects the well established pathogenic role of *S. aureus* in a wide range of clinical infections. The high proportion of isolates obtained from pus and wound swabs (56.3%) further supports its strong association with skin and soft tissue infections, which remain among the most common presentations in both community and hospital settings [21]. In contrast, CoNS were most frequently isolated from urine samples, emphasizing their clinical significance in urinary tract infections, particularly those caused by *S. saprophyticus* and other uropathogenic species [22]. These findings underscore the dual role of staphylococci as both commensals and opportunistic pathogens, capable of causing diverse infections depending on host and environmental factors.

The prevalence of methicillin-resistant *S. aureus* (MRSA) observed in this study (57.7%) is comparable to recent reports from Nepal, where MRSA rates have been consistently high [6,13]. This high prevalence is of considerable clinical concern, as MRSA infections are associated with increased morbidity, prolonged hospital stays, and higher healthcare costs. Similarly, the high proportion of methicillin resistant CoNS (63.0%) highlights their emerging importance as reservoirs of antimicrobial resistance genes within healthcare settings [23]. CoNS, often dismissed as contaminants, are increasingly recognized as significant pathogens, particularly in immunocompromised patients and those with indwelling medical devices. Their ability to harbor and transfer resistance determinants, including *mec* genes, contributes to the broader dissemination of resistance within hospital environments.

Multidrug resistance (MDR) was observed in a substantial proportion of both *S. aureus* (43.7%) and CoNS (38.3%) isolates, indicating a high burden of resistance across multiple antibiotic classes. This finding aligns with global trends of increasing MDR among staphylococci and reflects the cumulative impact of inappropriate antibiotic use, lack of stringent infection control measures, and limited antimicrobial stewardship in many settings [2]. The presence of MDR strains significantly restricts therapeutic options and necessitates the use of last-line agents such as vancomycin and linezolid, which, although effective, must be used judiciously to prevent the emergence of further resistance.

Erythromycin resistance was notably high among both *S. aureus* (56.3%) and CoNS (60.5%), indicating widespread resistance to macrolides in the study setting. This high prevalence may be attributed to the frequent and often empirical use of macrolides in clinical practice, which exerts selective pressure favoring resistant strains. While clindamycin resistance detected by routine disc diffusion appeared relatively lower, the D-test revealed a significant proportion of isolates harboring inducible resistance mechanisms. This discrepancy highlights a critical limitation of routine susceptibility testing and underscores the importance of additional phenotypic methods for accurate detection of resistance [9,20].

The prevalence of inducible clindamycin resistance (iMLSB phenotype) in this study (35.6%) is consistent with findings from other studies conducted in Nepal and South Asia [13,24]. This level of inducible resistance is clinically significant, as isolates that appear susceptible to clindamycin in vitro may develop resistance during therapy, leading to treatment failure. Notably, a higher proportion of MRSA isolates exhibited the iMLSB phenotype, suggesting a strong association between methicillin resistance and inducible macrolide-lincosamide-streptogramin B resistance. This association may be explained by the co-localization of resistance genes on mobile genetic elements, facilitating their simultaneous transfer and expression.

The constitutive MLSB (cMLSB) phenotype was the most prevalent resistance pattern observed in this study (43.6%), indicating that a large proportion of isolates express resistance continuously to all MLSB antibiotics. This finding further limits the utility of clindamycin in empirical therapy, particularly in settings with high cMLSB prevalence. In contrast, the MS phenotype, mediated by efflux mechanisms, was more common among methicillin-susceptible *S. aureus* (MSSA), consistent with previous reports [11]. From a clinical standpoint, isolates with the MS phenotype remain susceptible to clindamycin, emphasizing the importance of accurate phenotypic differentiation to avoid unnecessary exclusion of effective antibiotics.

The clinical implications of these findings are substantial. Clindamycin is widely used in the treatment of staphylococcal infections due to its excellent tissue penetration, oral bioavailability, and ability to inhibit toxin production [7]. However, the presence of inducible resistance poses a significant risk of therapeutic failure if not detected prior to treatment initiation. Therefore, reliance solely on routine susceptibility testing without performing the D-test may lead to inappropriate antibiotic selection and suboptimal patient outcomes [10]. Incorporation of the D-test into routine laboratory protocols is thus essential, particularly for erythromycin resistant isolates.

The resistance patterns observed in this study are broadly consistent with regional data from Nepal and neighboring countries, where similar trends in MRSA prevalence and MLSB resistance have been reported [14]. However, in contrast to high income countries, where stricter antimicrobial stewardship and infection control measures are implemented, lower rates of inducible resistance are typically observed [12]. This disparity highlights the critical role of healthcare infrastructure, antibiotic regulation, and surveillance systems in controlling the spread of antimicrobial resistance.

From a public health perspective, the high burden of resistance observed in this study underscores the urgent need for comprehensive antimicrobial stewardship programs. Such programs should focus on rational antibiotic prescribing, regular surveillance of resistance patterns, and continuous education of healthcare professionals. In addition, strengthening laboratory capacity for accurate and timely detection of resistance mechanisms is essential for guiding appropriate therapy and improving patient outcomes.

Despite its strengths, including a relatively large sample size and comprehensive phenotypic characterization, this study has certain limitations. Being a single-center study, the findings may not be fully generalizable to other regions. Furthermore, molecular characterization of resistance genes was not performed, which could have provided deeper insights into the genetic basis and transmission dynamics of resistance. Nevertheless, the study provides valuable baseline data for the region and highlights key areas for future research.

Overall, the findings of this study emphasize the growing challenge of antimicrobial resistance among staphylococci in Nepal. The high prevalence of methicillin resistance, multidrug resistance, and inducible clindamycin resistance calls for urgent and coordinated efforts to optimize antibiotic use, enhance diagnostic capabilities, and implement effective infection control strategies.

CONCLUSIONS

This study demonstrates a high prevalence of methicillin resistance, multidrug resistance, and inducible clindamycin resistance among staphylococcal isolates in western Nepal. The iMLSB phenotype was present in more than one third of isolates, particularly among MRSA, highlighting the potential for clindamycin treatment failures if the D-test is not routinely performed. These findings highlight the importance of strengthening antimicrobial susceptibility testing capabilities, implementing routine D-test for erythromycin resistant staphylococci, and promoting antimicrobial stewardship to preserve the efficacy of available treatment options.

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