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Detection of Inducible and Constitutive Clindamycin Resistance in Staphylococcus aureus and Its Association with Methicillin Resistance

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ABSTRACT: **Background**: Inducible clindamycin resistance (iMLSB) in Staphylococcus aureus appears susceptible on routine testing and it is detected throgh D-test. Local data on its frequency in methicillin resistant Staphylococcus aureus (MRSA) and methicillin sensitive Staphylococcus aureus (MSSA) guide the safe use of clindamycin.

Methods: This cross-sectional study was conducted in the Department of Microbiology, Teerthanker Mahaveer Medical College and Research Centre, Moradabad, from March 2025 to February 2026. Non-duplicate S. aureus isolates from clinical specimens were isolated by standard methods. Methicillin resistance was detected by the cefoxitin disk (30 µg), and antimicrobial susceptibility by Kirby-Bauer disk diffusion according to CLSI 2025. The D-test was done on erythromycin-resistant, clindamycin-susceptible or intermediate isolates. Data were analysed in SPSS version 27 using the chi-square test ($p < 0.05$).

Results: Of 150 isolates, 91 (60.7%) were MRSA and 59 (39.3%) were MSSA. The MS phenotype was found in 68 (45.3%), constitutive MLSB in 57 (38.0%) and iMLSB in 24 (16.0%), and 81 isolates (54.0%) were clindamycin-resistant overall. Phenotypes differed significantly between MRSA and MSSA ($p = 0.0008$), with iMLSB in 19.8% versus 10.2% and cMLSB in 47.3% versus 25.4%. Phenotype also differed by specimen ($p = 0.001$): iMLSB occurred in 12 blood (20.3%) and 12 pus (15.6%) isolates. There was no significant association with age, gender, IPD/OPD or ICU location. All isolates were susceptible to vancomycin and teicoplanin, and only one was resistant to linezolid (0.7%). Resistance to fluoroquinolones exceeded 87%.

Conclusion: One in six isolates showed inducible clindamycin resistance that routine testing would have missed. Resistance was higher in MRSA and in blood isolates. The D-test should be included in routine susceptibility testing.

INTRODUCTION

Staphylococcus aureus is one of the most important bacterial pathogens of humans. It is a normal colonizer of the skin and anterior nares, but it also causes a wide range of infections, from boils, abscesses, impetigo and wound infections to bacteraemia,

endocarditis, pneumonia, osteomyelitis, septic arthritis and infections of prosthetic devices [1]. Its virulence comes from surface adhesins, immune-evasion proteins, and toxins such as the Panton-Valentine leucocidin, exfoliative toxins and toxic shock syndrome toxin [1]. In hospitals it is a leading cause of surgical site and bloodstream infections, and in the community, it causes most skin and soft tissue infections.

Antibiotic resistance has made *S. aureus* infections much harder to treat. Methicillin-resistant *S. aureus* (MRSA) carries the mecA gene, which encodes an altered penicillin-binding protein (PBP2a) with low affinity for β-lactam antibiotics. MRSA is therefore resistant to nearly all β-lactams, and these strains are often also resistant to fluoroquinolones, aminoglycosides, macrolides and other classes of antibiotics [2]. MRSA infections are linked to longer hospital stay, higher cost and higher mortality than infections with methicillin-susceptible strains (MSSA). Vancomycin, teicoplanin and linezolid remain reliable for serious MRSA infections, but they are costly, need monitoring or intravenous administration, and should be preserved for severe disease [3]. This creates a need for oral, effective and affordable options for less severe infections.

Clindamycin is one such option. It is a lincosamide that binds the 50S ribosomal subunit and inhibits protein synthesis. It is well absorbed orally, penetrates tissue and bone well, suppresses toxin production, and is inexpensive. It is widely used for MRSA skin and soft tissue infections, for step-down oral therapy, and in patients allergic to penicillins [3]. Its usefulness, however, depends on whether the isolate carries resistance that routine testing can miss.

Resistance to clindamycin is closely linked to resistance to macrolides through the macrolide- lincosamide -streptogramin B (MLSB) group of mechanisms [4]. Three phenotypes are recognised. In the constitutive MLSB (cMLSB) phenotype, the isolate is resistant to both erythromycin and clindamycin because erm genes are expressed continuously. In the MS phenotype, resistance to erythromycin is caused by an efflux pump encoded by msrA, and the isolate stays susceptible to clindamycin. In the inducible MLSB (iMLSB) phenotype, erm genes (most often ermA and ermC) encode a methyltransferase that modifies the 23S ribosomal RNA target, but expression is switched on only in the presence of an inducer such as erythromycin [4,5]. An isolate with iMLSB is resistant to erythromycin and appears susceptible to clindamycin in a standard disk test, because clindamycin alone does not induce the enzyme.

This has real clinical consequences. If an inducible-resistant isolate is reported as clindamycin-susceptible and treated with clindamycin, resistant subpopulations that constitutively express erm can be selected during therapy, and treatment can fail [6-8]. Failures have been described in skin and soft tissue infections and in invasive disease, including in children [7,8]. Detecting inducible resistance is therefore essential for safe use of clindamycin.

The D-test (disk approximation test) detects inducible resistance. An erythromycin (15 μg) disk and a clindamycin (2 μg) disk are placed 15-26 mm apart on a lawn of the isolate. Erythromycin diffuses towards clindamycin and induces the erm enzyme, so the zone around clindamycin becomes flattened on the side facing erythromycin, forming a "D" shape [9,10]. The test is simple, inexpensive, needs no special equipment, and is recommended by CLSI for erythromycin-resistant, clindamycin-susceptible staphylococci [11]. Isolates that are D-test positive should be reported as clindamycin-resistant. Despite this, the D-test is not yet part of routine susceptibility testing in many laboratories, so inducible resistance may go undetected.

The frequency of inducible clindamycin resistance varies widely between studies. Recent reports give iMLSB rates from about 15% in Qatar [12], 22.6% in western India [13] and 26.3% in Ethiopia [14] to 34% in eastern India [15]. Studies from Nepal also report a high burden of inducible resistance in tertiary care hospitals [16,17]. The differences reflect the proportion of MRSA among isolates, local macrolide and lincosamide prescribing, the patient population, and the denominator used, because some studies report percentages among all isolates and others only among erythromycin-resistant isolates [4,18]. Several studies find that inducible and constitutive resistance are more frequent in MRSA than MSSA [13,15], but findings are not consistent [12]. Distribution by specimen type, patient age, gender, and community or hospital origin has been studied much less, and resistance patterns can differ between community and hospital settings [19].

MATERIAL AND METHODS

Study design and setting

This cross-sectional study was conducted in the Department of Microbiology, Teerthanker Mahaveer medical college and research centre Moradabad, India, over a period of 1 year from March 2025 to February 2026.

Ethical approval

The study was approved by the Institutional Ethics Committee (Approval No. TMU/IEC/2024-25/PG/003).

Aim

To determine the prevalence of inducible clindamycin resistance among clinical isolates of *Staphylococcus aureus*, including MRSA, by D-test.

Objectives

1. To determine the proportion of MRSA and MSSA among clinical isolates of *S. aureus*.
2. To detect MLSB resistance phenotypes (iMLSB, cMLSB and MS phenotypes) by the D-test.
3. To compare these phenotypes between MRSA and MSSA.
4. To study their association with specimen type, age group, gender, IPD/OPD and ward/ICU.
5. To determine the antibiotic susceptibility pattern of the isolates.

Sample collection

Clinical specimens including pus, wound swabs, blood, sputum and body fluids were received from IPD and OPD patients with suspected staphylococcal infection. Only one isolate per patient was included. Duplicate isolates from the same patient and contaminants were excluded.

Isolation and identification of *Staphylococcus aureus*

Specimens were inoculated on appropriate culture media and incubated aerobically at 37°C for 18-24 hours. Isolates were identified as *S. aureus* by colony morphology, Gram stain (Gram-positive cocci in clusters), catalase test, slide and tube coagulase tests, and mannitol fermentation on mannitol salt agar with pigment production.

Detection of methicillin resistance (MRSA)

Methicillin resistance was detected by the cefoxitin disk diffusion method (30 µg) on Mueller-Hinton agar (MHA) according to CLSI 2025 guidelines. A zone diameter of ≤21 mm was interpreted as MRSA and ≥22 mm as methicillin-sensitive *S. aureus* (MSSA).

Antimicrobial susceptibility testing

Antimicrobial susceptibility was tested by the Kirby-Bauer disk diffusion method on MHA using a 0.5 McFarland inoculum suspension. The antibiotics tested included erythromycin, clindamycin, ciprofloxacin, gentamicin, cotrimoxazole, tetracycline and linezolid. Plates were incubated at 35 ± 2°C for 16-18 hours (24 hours for cefoxitin), and results were interpreted per CLSI M100 2025 breakpoints.

D-test for inducible clindamycin resistance

The D-test was performed on all isolates that were erythromycin-resistant and clindamycin-susceptible or intermediate, as per CLSI 2025 guidelines. A lawn culture of 0.5 McFarland suspension was made on MHA. An erythromycin disk (15 µg) and a clindamycin disk (2 µg) were placed 15-26 mm apart (edge to edge). Plates were incubated at 35 ± 2°C for 16-18 hours in ambient air incubator. Results were interpreted as follows: [20]

- **D-test positive (inducible MLSB, iMLSB phenotype):** flattening of the clindamycin zone adjacent to the erythromycin disk, forming a "D" shape. Isolates were reported as clindamycin-resistant.
- **D-test negative (MS phenotype):** erythromycin-resistant, clindamycin-susceptible, with a circular clindamycin zone [11,21].
- **Constitutive MLSB (cMLSB phenotype):** resistant to both erythromycin and clindamycin, with no D-zone [11,21].

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Quality control

S. aureus ATCC 25923 was used as the quality control strain for disk diffusion. ATCC 43300 (methicillin-resistant) was used as the control for the cefoxitin test. For the D-test, ATCC BAA-976 (D-test negative) and ATCC BAA-977 (D-test positive) were used as controls [11].

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 27. Results were expressed as frequencies and percentages. The association of phenotypes with specimen type, ward/intensive care unit and age group was assessed using the chi-square test, with $p < 0.05$ considered statistically significant.

RESULTS

A total of 150 non-duplicate *Staphylococcus aureus* isolates were included. Patient age ranged from 1 day to 89 years (mean ± SD, 39.7 ± 21.7 years), and the largest group was over 60 years (29, 19.3%). There were 87 males (58.0%) and 63 females (42.0%). Most isolates came from inpatients (122, 81.3%), and 43 (28.7%) came from intensive care units. Overall, 91 isolates (60.7%) were MRSA and 59 (39.3%) were MSSA. MRSA was most frequent in the 31-40year (73.1%) and over-60-year (72.4%) groups, but it did not differ significantly by age group, gender or ICU location. It was higher among OPD isolates (78.6%) than IPD isolates (56.6%), a borderline difference ($p = 0.053$) (Table 1).

Table 1. Demographic and clinical profile and MRSA proportion (N = 150)

Variable	Category	Total, n (%)	MRSA, n (%)	p-value
Age group (years)	0-10	16 (10.7)	8 (50.0)	0.234
	11-20	15 (10.0)	7 (46.7)	
	21-30	21 (14.0)	10 (47.6)	
	31-40	26 (17.3)	19 (73.1)	
	41-50	17 (11.3)	12 (70.6)	
	51-60	26 (17.3)	14 (53.8)	
	>60	29 (19.3)	21 (72.4)	
Gender	Male	87 (58.0)	51 (58.6)	0.665
	Female	63 (42.0)	40 (63.5)	
Setting	IPD	122 (81.3)	69 (56.6)	0.053
	OPD	28 (18.7)	22 (78.6)	
Location	ICU	43 (28.7)	25 (58.1)	0.828
	Non-ICU	107 (71.3)	66 (61.7)	

Distribution of specimens

Pus was the most common specimen (77, 51.3%), followed by blood (59, 39.3%) and tissue (4, 2.7%). Swabs (ear and high vaginal) and respiratory samples (sputum, endotracheal aspirate and BAL fluid) accounted for 3 isolates each (2.0%). Sterile body fluids (synovial, pleural, CSF and ascitic fluid) accounted for 4 isolates (2.7%) (Table 2).

Table 2. Distribution of isolates by specimen type

Specimen	n	%
Pus	77	51.3
Blood	59	39.3
Tissue	4	2.7
Swabs (ear, HVS, other)	3	2.0
Respiratory (sputum, ET, BAL)	3	2.0
Sterile fluids (synovial, pleural, CSF, ascitic)	4	2.7
Total	**150**	**100.0**

Phenotypic pattern of MLSB resistance

By the D-test, 68 isolates (45.3%) showed the MS phenotype, 57 (38.0%) showed constitutive MLSB (cMLSB) and 24 (16.0%) showed inducible MLSB (iMLSB). One isolate had no phenotype recorded. Overall, 81 isolates (54.0%) were clindamycin-resistant, either constitutively or inducible (Table 3).

Table 3. Distribution of MLSB phenotypes (N = 150)

Phenotype	n	%
MS phenotype (D-test negative)	68	45.3
cMLSB	57	38.0
iMLSB (D-test positive)	24	16.0
Not recorded	1	0.7
Total	**150**	**100.0**

Phenotype according to methicillin resistance

The phenotypes differed significantly between MRSA and MSSA (p = 0.0008). The MS phenotype was more common in MSSA (64.4%), whereas iMLSB (19.8% vs 10.2%) and cMLSB (47.3% vs 25.4%) were more common in MRSA. Clindamycin resistance (iMLSB + cMLSB) was seen in 61 of 91 MRSA (67.0%) and 21 of 59 MSSA (35.6%) isolates (Table 4).

Table 4. MLSB phenotypes in MRSA and MSSA

Phenotype	MRSA (n=91), n (%)	MSSA (n=59), n (%)	p-value
iMLSB	18 (19.8)	6 (10.2)	0.0008
cMLSB*	43 (47.3)	15 (25.4)	
MS phenotype	30 (33.0)	38 (64.4)	

Phenotype detection

iMLSB was most frequent in patients over 60 years (31.0%), in males (19.5%) and in OPD patients (21.4%). None of these differences was statistically significant (age p = 0.266, gender p = 0.309, setting p = 0.288, ICU location p = 0.807) (Table 5).

Table 5. MLSB phenotypes by age, gender, setting and location, n (%)

Variable	Category	MS	cMLSB	iMLSB	p-value
Age group (years)	0-10	7 (43.8)	8 (50.0)	1 (6.3)	0.266
	11-20	11 (73.3)	2 (13.3)	2 (13.3)	
	21-30	11 (52.4)	8 (38.1)	2 (9.5)	
	31-40	10 (38.5)	11 (42.3)	5 (19.2)	
	41-50	9 (52.9)	6 (35.3)	2 (11.8)	
	51-60	11 (42.3)	12 (46.2)	3 (11.5)	
	>60	9 (31.0)	11 (37.9)	9 (31.0)	
Gender	Male	36 (41.4)	34 (39.1)	17 (19.5)	0.309

	Female	32 (50.8)	24 (38.1)	7 (11.1)	
Setting	IPD	59 (48.4)	45 (36.9)	18 (14.8)	0.288
	OPD	9 (32.1)	13 (46.4)	6 (21.4)	
Location	ICU	18 (41.9)	17 (39.5)	8 (18.6)	0.807
	Non-ICU	50 (46.7)	41 (38.3)	16 (15.0)	

Phenotype detention according to specimen

iMLSB was seen in 12 blood isolates (20.3%) and 12 pus isolates (15.6%), and in none of the other specimens. cMLSB was more common in blood (54.2%), whereas the MS phenotype predominated in pus (58.4%). The distribution of phenotypes differed significantly by specimen type ($\chi^2 = 18.42$, df = 4, p = 0.001) (Table 6).

Table 6. MLSB phenotypes by specimen type, n (%)

Specimen	MS	cMLSB	iMLSB	Total	p-value
Blood	15 (25.4)	32 (54.2)	12 (20.3)	59	0.001
Pus	45 (58.4)	20 (26.0)	12 (15.6)	77	
Others*	8 (57.1)	6 (42.9)	0	14	

*Tissue, swabs, respiratory samples and sterile fluids.

Antibiotic susceptibility pattern

All isolates (100%) were susceptible to vancomycin and teicoplanin. Only one isolate (0.7%) each was resistant to linezolid and minocycline. Resistance was highest to ciprofloxacin (90.7%), levofloxacin (88.7%), ofloxacin (87.3%) and azithromycin (77.3%). Resistance to most agents was significantly higher in MRSA than in MSSA, except for cotrimoxazole (p = 0.093) and tetracycline (p = 0.052) (Table 7).

Table 7. Antibiotic resistance pattern of MRSA and MSSA, n (%)

Antibiotic	Total (N=150)	MRSA (n=91)	MSSA (n=59)	p-value
Azithromycin	116 (77.3)	82 (90.1)	34 (57.6)	<0.001
Cefoxitin	101 (67.3)	84 (92.3)	17 (28.8)	<0.001
Ciprofloxacin	136 (90.7)	88 (96.7)	48 (81.4)	0.003
Clarithromycin	77 (51.3)	59 (64.8)	18 (30.5)	<0.001
Clindamycin	74 (49.3)	56 (61.5)	18 (30.5)	<0.001
Cotrimoxazole	67 (44.7)	46 (50.5)	21 (35.6)	0.093
Doxycycline	37 (24.7)	28 (30.8)	9 (15.3)	0.034
Erythromycin	101 (67.3)	71 (78.0)	30 (50.8)	<0.001
Gentamicin	70 (46.7)	49 (53.8)	21 (35.6)	0.031
Levofloxacin	133 (88.7)	88 (96.7)	45 (76.3)	<0.001
Minocycline	1 (0.7)	1 (1.1)	0	1.000
Ofloxacin	131 (87.3)	86 (94.5)	45 (76.3)	0.002
Tetracycline	27 (18.0)	21 (23.1)	6 (10.2)	0.052
Linezolid	1 (0.7)	1 (1.1)	0	1.000

DISCUSSION

Staphylococcus aureus is a leading cause of skin, bloodstream and device-related infections, and its virulence and multidrug resistance make it a constant challenge in hospitals [1,2]. Clindamycin is often used for MRSA skin and soft tissue infections

because it is oral, inexpensive and penetrates tissue well [3]. Its use depends on detecting inducible resistance, which routine disk diffusion misses. In this study of 150 isolates, 60.7% were MRSA and 54.0% were clindamycin-resistant (16.0% inducible, 38.0% constitutive). Phenotype was significantly associated with methicillin resistance and with specimen type.

In this MRSA prevalence of 60.7% lies between recent reports of 18.9% from western India [13], 36.9% from Ethiopia [14] and 72% from Qatar [12]. This variation reflects differences in hospital type, case mix and infection-control practice. Our tertiary care setting, with 81.3% inpatients and 39.3% blood isolates, likely explains the high rate. MRSA was more frequent in outpatients (78.6%) than inpatients (56.6%), a borderline difference ($p = 0.053$), and this warrants a check for community-acquired strains. Age, gender and ICU location were not significantly associated with MRSA.

In this study iMLSB in 16.0%, cMLSB in 38.0% and the MS phenotype in 45.3% of isolates. Our iMLSB rate is close to the 15.5% reported from Qatar among all isolates [12] and lower than the 22.6% from Vadodara [13], 26.3% from Ethiopia [14] and 34% from West Bengal [15]. Our cMLSB rate (38.0%) is similar to the 40% in the West Bengal study [15] but far higher than the 4.2% in the Vadodara study [13]. Differences across studies come from local macrolide and lincosamide use, which drives erythromycin resistance [4,18], from the proportion of MRSA, and from the denominator used, since some studies report percentages only among erythromycin-resistant isolates. Nepalese studies also report a substantial burden of inducible resistance in tertiary hospitals [16,17]. We used the CLSI D-test [9-11], which supports comparison with these studies.

Phenotype differed significantly between MRSA and MSSA ($p = 0.0008$). iMLSB (19.8% vs 10.2%) and cMLSB (47.3% vs 25.4%) were commoner in MRSA, and the MS phenotype in MSSA (64.4%). This agrees with the West Bengal study, which found iMLSB in 40% of MRSA and 22% of MSSA [15], and with the Vadodara study, which reported a significant association between MRSA and iMLSB [13]. Not all studies agree. In the Qatari series, iMLSB was similar in MRSA and MSSA among erythromycin-resistant isolates (58% and 63.6%) [12]. Multidrug resistance in MRSA, heavy antibiotic exposure in hospitals and spread of resistant clones probably in this study.

iMLSB is caused by ribosomal methylation encoded by erm genes, and erythromycin induces expression that then affects clindamycin [4,5]. The isolates appear clindamycin-susceptible on routine testing and are missed unless the D-test is done [6]. The 24 iMLSB isolates in our study (16.0%) would otherwise have been reported susceptible. Clindamycin failure has been documented with inducible-resistant isolates [7,8]. Guidelines advise against clindamycin for D-test-positive MRSA in serious infections [3,23], and for minor skin infections the decision should be individualised.

Phenotype differed significantly by specimen ($\chi^2 = 18.42$, $df = 4$, $p = 0.001$). Blood isolates were mostly cMLSB (54.2%) with 20.3% iMLSB, while pus isolates were mostly MS (58.4%). Bloodstream isolates come largely from hospitalised patients with heavy antibiotic exposure, and community-type strains more often show the efflux-mediated MS phenotype. Constitutive and inducible resistance have been shown to differ between community and tertiary hospital settings [19]. This explanation is a hypothesis, because we did not analyse source of infection.

Resistance was very high to ciprofloxacin (90.7%), levofloxacin (88.7%), ofloxacin (87.3%) and azithromycin (77.3%), and higher in MRSA than in MSSA for most agents, as expected for multidrug-resistant MRSA [2]. Fluoroquinolones and macrolides should not be used empirically in our setting. Resistance to cotrimoxazole (44.7%), doxycycline (24.7%) and tetracycline (18.0%) was moderate, so these remain options for MRSA skin infections when susceptible [3]. All isolates were susceptible to vancomycin and teicoplanin, and resistance to linezolid and minocycline was found in one isolate each (0.7%). Even a single linezolid-resistant isolate matters for surveillance.

CONCLUSION

Inducible clindamycin resistance was present in 16.0% of isolates and overall clindamycin resistance exceeded 50%, higher in MRSA than MSSA and in blood than pus. Routine D-testing should be part of standard susceptibility testing to avoid inappropriate clindamycin use. Vancomycin, teicoplanin, linezolid and minocycline remained highly active. Larger multicentre studies with molecular data and clinical outcomes are needed.

Strength Limitations of this study

The present study used CLSI-standard methods with quality control strains and analysed MLSB phenotypes in relation to methicillin resistance, patient variables and specimen type. The study has some limitations. It was conducted at a single centre, and some subgroups were small, which limited the statistical power of subgroup analyses. Methicillin resistance was detected

phenotypically and was not confirmed by mecA or PBP2a testing, and clonal typing was not performed. In a few isolates, the cefoxitin disk result did not agree with the final MRSA/MSSA classification, and molecular testing would have resolved these discrepancies. Clinical outcomes were not studied, so the link between iMLSB and treatment failure could not be assessed directly.

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