Section: Microbiology

Original Research Article

INDUCIBLE CLINDAMYCIN RESISTANCE IN STAPHYLOCOCCUS AUREUS ISOLATES FROM CLINICAL SAMPLES AT A TERTIARY CARE FACILITY IN NORTH INDIA: A PHENOTYPIC ANALYSIS

Amrita Gupta[1], Anjalee Chhetri[2], Brij Nandan Singh[3], Saumya Singh[4]

[1]Assistant Professor, Department of Microbiology, United Institute of Medical Sciences, Prayagraj, UttarPradesh, India
[2]Lab Head, Agilus Diagnostics, Fortis Escorts Hospital, Neelam Bata Chowk, Faridabad, Haryana.
[3]Associate Professor, Department of Microbiology, Autonomous State Medical College, Sonbhadra, Uttar Pradesh, India.
[4]Professor, Department of Microbiology, United Institute of Medical Sciences, Prayagraj, Uttar Pradesh, India.

Received *: 05/06/2026*
Received in revised form *: 20/07/2026*
Accepted *: 04/08/2026*

Corresponding Author:
Dr. Amrita Gupta,
Assistant Professor, Department of Microbiology, United Institute of Medical Sciences, Prayagraj, UttarPradesh, India.
Email: guptaamrita1512@gmail.com

DOI: 10.70034/ijmedph.2026.3.384

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 2411-2417

ABSTRACT

Background: Staphylococcus aureus is one of the most important pathogens responsible for community-acquired as well as hospital-acquired infections. Increasing antimicrobial resistance, particularly methicillin-resistant Staphylococcus aureus (MRSA), has considerably limited therapeutic options. Clindamycin is frequently used for the treatment of staphylococcal infections because of its excellent pharmacokinetic properties and ability to suppress toxin production. However, inducible resistance mediated by the macrolide-lincosamide-streptogramin B (MLSB) resistance mechanism may lead to therapeutic failure if not detected during routine susceptibility testing. The Clinical and Laboratory Standards Institute (CLSI) recommends the double-disc diffusion test (D-test) for phenotypic detection of inducible clindamycin resistance. The objective is to determine the prevalence of inducible clindamycin resistance among clinical isolates of Staphylococcus aureus and to compare the distribution of resistance phenotypes between MRSA and methicillin-sensitive Staphylococcus aureus(MSSA) isolates.

Materials and Methods: A laboratory-based cross-sectional study was conducted in the Department of Microbiology at a tertiary care centre in North India. A total of 356 non-duplicate clinical isolates of Staphylococcus aureus recovered from various clinical specimens were included. Identification was performed using conventional microbiological methods. Antimicrobial susceptibility testing was carried out by the Kirby–Bauer disc diffusion method according to CLSI guidelines. Methicillin resistance was detected using the cefoxitin (30 µg) disc, while inducible clindamycin resistance was identified by the D-test using erythromycin and clindamycin discs.

Results: The phenotypic assay showed that 65 (18.3%) were inducible MLSB (iMLSB), 44 (12.4%) were constitutive MLSB (cMLSB), 66 (18.5%) had an MS phenotype, and 181 (50.8%) were completely susceptible. Inducible clindamycin resistance was proportionally higher among MRSA isolates than MSSA isolates.

Conclusion: Significant proportions of S. aureus strains were found to have inducible resistance to clindamycin. D-test needs to become part of antimicrobial susceptibility testing to avoid false susceptibility results and reduce therapy failures; thereby improving and strengthening antimicrobial stewardship.

Keywords: Staphylococcus aureus; MRSA; Clindamycin; D-test; Inducible Clindamycin Resistance; MLSB Phenotype; Antimicrobial Resistance.

INTRODUCTION

Staphylococcus aureus is still one of the most important pathogenic bacteria in contemporary medicine. This microorganism causes a variety of diseases, such as skin and soft tissue infections, surgical infections, bloodstream infections, pneumonia, osteomyelitis, septic arthritis, infective endocarditis, and device-associated infections. The rise of antibiotic resistant pathogens has greatly contributed to morbidity, mortality, lengthened hospital stay, and increased healthcare costs on a global scale.[1]

MRSA isolates are resistant to β-lactam drugs and often to a variety of non-β-lactam antimicrobial drugs, reducing the number of treatment possibilities available. While glycopeptides, including vancomycin, still constitute the primary treatment for MRSA infections, concerns regarding nephrotoxicity, reduced susceptibility, cost, and the requirement for parenteral administration have prompted the use of effective alternative agents whenever appropriate.[2]

Clindamycin is commonly used in treating staphylococcal infections owing to its high oral bioavailability, good tissue penetration, intracellular distribution, and toxin suppression properties. Due to its pharmacological properties as well as the availability of clindamycin in oral and parenteral forms, clindamycin plays a crucial role in the treatment of infections involving the skin and soft tissues, osteoarticular infections, as well as infections of toxins mediated by S. aureus, especially in people who are allergic to β-lactam antibiotics.[3]

Resistance to macrolide-lincosamide-streptogramin B (MLSB) antibiotics is primarily mediated by methylation of the 23S ribosomal RNA target site encoded by erm genes. Expression of these genes may occur constitutively, resulting in resistance to both erythromycin and clindamycin (constitutive MLSB phenotype), or inducibly, wherein isolates appear susceptible to clindamycin during routine laboratory testing but express resistance following exposure to macrolides such as erythromycin (inducible MLSB phenotype). In contrast, resistance mediated by the msrA gene results in the MS phenotype, characterized by erythromycin resistance with preserved susceptibility to clindamycin due to an active efflux mechanism.[4]

The inducible MLSB phenotype is clinically important because isolates harbouring inducible resistance may be incorrectly reported as clindamycin susceptible during routine susceptibility testing. Administration of clindamycin in such cases may induce expression of the erm gene during therapy, leading to constitutive resistance and subsequent clinical treatment failure. To minimize this risk, the Clinical and Laboratory Standards Institute (CLSI) recommends routine performance of the double-disc diffusion (D-test) for all erythromycin-resistant and clindamycin-susceptible Staphylococcus aureus isolates.[5,6]

The D-test is a simple, inexpensive, and reliable phenotypic method that differentiates inducible MLSB resistance, constitutive MLSB resistance, and the MS phenotype. Because it can be incorporated into routine antimicrobial susceptibility testing without additional laboratory infrastructure, it plays an important role in improving susceptibility reporting and supporting antimicrobial stewardship programmes.[6,7]

Several studies from India and other countries have demonstrated considerable geographical variation in the prevalence of inducible clindamycin resistance, with consistently higher rates among MRSA isolates than MSSA isolates. Such variation highlights the importance of institution-specific surveillance for guiding empirical therapy, monitoring antimicrobial resistance trends, and developing local antibiograms.[7-9]

Given the limited contemporary data from tertiary care centres in North India, the present study was undertaken to determine the prevalence of inducible clindamycin resistance among clinical isolates of Staphylococcus aureus, compare resistance phenotypes between MRSA and MSSA isolates, and evaluate the role of routine D-testing in improving antimicrobial susceptibility reporting and guiding appropriate antimicrobial therapy.

Aim & Objectives: To determine the prevalence of inducible clindamycin resistance among clinical isolates of Staphylococcus aureus obtained from patients attending a tertiary care centre in North India.

Primary Objective: To determine the prevalence of inducible clindamycin resistance among Staphylococcus aureus isolates using the D-test.

Secondary Objectives

1. To determine the prevalence of MRSA and MSSA among clinical isolates.
2. To evaluate the antimicrobial susceptibility profile of Staphylococcus aureus.
3. To determine the distribution of susceptible, MS phenotype, inducible MLSB (iMLSB), and constitutive MLSB (cMLSB) phenotypes.
4. To compare clindamycin resistance phenotypes between MRSA and MSSA isolates.
5. To assess the utility of routine D-testing in improving antimicrobial susceptibility reporting.

MATERIALS AND METHODS

Study Design: A laboratory-based, cross-sectional observational study was conducted in the Department of Microbiology of a tertiary care teaching hospital in North India.

Study Duration: The study included consecutive, non-duplicate Staphylococcus aureus isolates recovered during the defined study period (June 2024 to December 2025) from clinically significant specimens recived to the microbiology laboratory.

Study Population: A total of 356 non-duplicate clinical isolates of Staphylococcus aureus obtained

from various clinical specimens were included in the analysis.

Inclusion Criteria

The study included all non-duplicate clinical isolates of Staphylococcus aureus recovered from clinically significant specimens received in the Department of Microbiology during the study period. Specimens included pus, wound swabs, blood, respiratory samples, urine, body fluids, and other clinically relevant samples submitted from both inpatient and outpatient departments.

Exclusion Criteria

- Duplicate isolates obtained from the same patient.
- Colonization isolates without clinical evidence of infection.
- Isolates with incomplete microbiological records.
- Contaminated specimens.

Isolation and Identification of Staphylococcus aureus: Clinical specimens were processed according to standard microbiological procedures. Isolates suspected to be Staphylococcus aureus were identified on the basis of colony morphology, Gram staining, catalase test, slide coagulase test, tube coagulase test, and other conventional biochemical tests. The identification procedures followed standard laboratory protocols similar to those described in previous Indian studies on inducible clindamycin resistance.[10,11,7]

Antimicrobial Susceptibility Testing: Antimicrobial susceptibility testing (AST) was performed by the Kirby–Bauer disc diffusion method on Mueller–Hinton agar according to the Clinical and Laboratory Standards Institute (CLSI) recommendations.[5,12] The antibiotics included in the study were Penicillin, Cefoxitin, Erythromycin, Clindamycin, Ciprofloxacin, Gentamicin, Cotrimoxazole, Tetracycline, Vancomycin, Linezolid

Methicillin resistance was detected using the Cefoxitin (30 µg) disc. Isolates with inhibition zones interpreted as resistant according to CLSI criteria were classified as MRSA, whereas susceptible isolates were classified as MSSA.[5]

Detection of Inducible Clindamycin Resistance: All erythromycin-resistant and clindamycin-susceptible isolates were subjected to the D-test according to CLSI recommendations.[5,6]

Briefly,

- A standardized 0.5 McFarland suspension was inoculated on Mueller–Hinton agar.
- Erythromycin (15 µg) and Clindamycin (2 µg) discs were placed 15–20 mm apart (edge to edge).
- Plates were incubated aerobically at 35 ± 2°C for 16–18 hours.

The D-test was interpreted as follows:

Sensitive phenotype

- Sensitive to erythromycin
- Sensitive to clindamycin

MS phenotype

- Resistant to erythromycin
- Sensitive to clindamycin
- Circular inhibition zone around clindamycin (D-test negative)

Inducible MLSB (iMLSB)

- Resistant to erythromycin
- Sensitive to clindamycin
- Flattening of inhibition zone adjacent to erythromycin producing the characteristic "D-shaped" appearance (D-test positive)

Constitutive MLSB (cMLSB)

- Resistant to both erythromycin and clindamycin

These interpretive criteria were based on CLSI recommendations and the original description of the D-test by Fiebelkorn et al.[5,6]

Interpretation of phenotypes of E and CD

Phenotype	Erythromycin (E)	Clindamycin(CD)	D test Result	Character of Phenotype
E and CD Susceptible (Sensitive Phenotype)	S	S zone size ≥ 21 mm	Not applicable	
MS Phenotype	R zone size ≤13 mm	S zone size ≥ 21 mm	NEGATIVE	Circular zone of Inhibition around CD
Inducible Phenotype (iMLSB)	R zone size ≤13 mm	S zone size ≥ 21 mm	POSITIVE	D Shaped zone of inhibition around CD with flattening towards E disc
Constitutive Phenotype (cMLSB)	R zone size ≤ 13 mm	R zone size ≤14 mm	NEGATIVE	

Quality Control: Quality control for antimicrobial susceptibility testing was maintained using standard reference strains recommended by CLSI. Internal quality assurance procedures were followed throughout the study.[5]

Data Collection: The following variables were recorded for each isolate: age, sex, clinical specimen, hospital location, MRSA/MSSA status, antibiotic susceptibility profile, D-test result, and clindamycin resistance phenotype.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using Python and SPSS. Descriptive statistics included frequencies and percentages. Categorical variables were compared using the Chi-square test. A p-value <0.05 was considered statistically significant. Tables for frequencies, cross-tabulation, antibiogram, pie charts, stacked bar charts, and Chi square statistics were produced using the Python script for data visualization and analysis.

RESULTS

A total number of 356 non-repetitive clinical isolates of Staphylococcus aureus were used for the study. Among these, 233 (65.4%) isolates were obtained from male patients and 123 (34.6%) isolates were obtained from female patients. The male-to-female ratio was 1.9:1, indicating a predominance of infections among male patients.

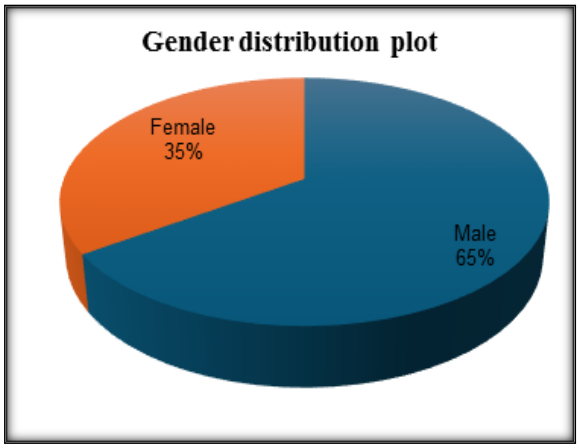

Figure 1: Gender distribution of study participants

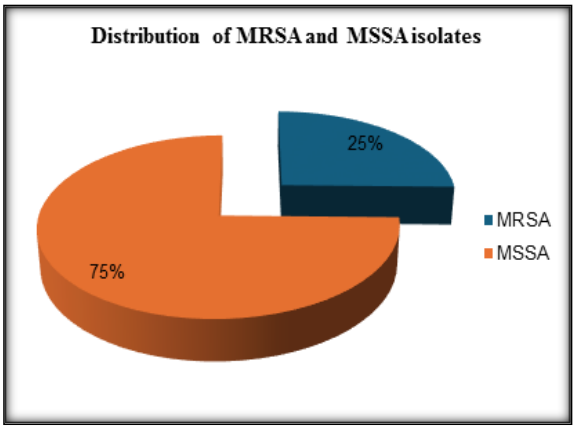

Figure 2: Distribution of MRSA and MSSA isolates (pie chart).

Table 1: Gender Distribution of Patients

Gender	Number	Percentage
Male	233	65.4
Female	123	34.6
Total	356	100

Table 2: Distribution of MRSA and MSSA

	Number	Percentage
MRSA	90	25.3
MSSA	266	74.7
Total	356	100

Among the 356 isolates, 90 (25.3%) were identified as MRSA and 266 (74.7%) were MSSA, indicating that approximately one-quarter of all clinical isolates exhibited methicillin resistance.

Table 3: Antibiotic Resistance Profile of Staphylococcus aureus

Antibiotic	Resistant n (%)
Penicillin	100
Cotrimoxazole	70.8
Ciprofloxacin	64.0
Gentamicin	62.0
Tetracycline	59.0
Erythromycin	58.7
Clindamycin	30.6
Cefoxitin	25.3
Vancomycin	0
Linezolid	0

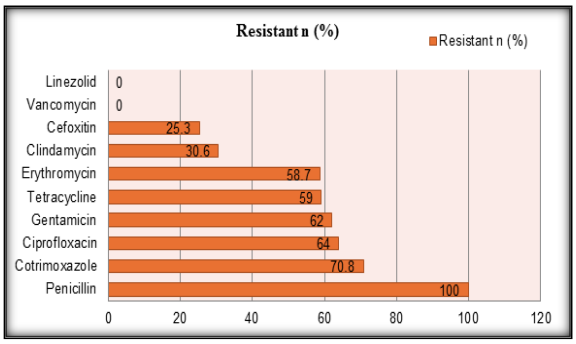

Figure 3: Antibiogram showing resistance pattern of S. aureus isolates.

Susceptibility testing to different antibiotics revealed varied levels of resistance. Resistance to penicillin was 100%, suggesting that β-lactam resistance is prevalent among Staphylococcus aureus isolates. High resistance was also seen to cotrimoxazole (70.8%), ciprofloxacin (64.0%), gentamicin (62.0%), tetracycline (59.0%), and erythromycin (58.7%). Resistance to clindamycin was observed in 30.6% of isolates, and methicillin resistance, determined by cefoxitin testing, was 25.3%. Notably, all isolates were susceptible to vancomycin and linezolid, making these agents the most effective antibiotics against the study isolates.

Table 4: Phenotypic Distribution of Clindamycin Resistance

Phenotype	Number	Percentage
Sensitive	181	50.8
MS phenotype	66	18.5
iMLSB	65	18.3
cMLSB	44	12.4
Total	356	100

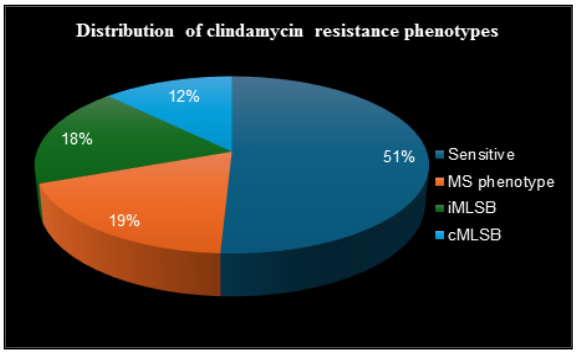

Figure 5: Pie chart showing distribution of clindamycin resistance phenotypes.

Resistance phenotypes from D-testing yielded four resistance categories. Of the 356 isolates, 181 (50.8%) were found to be completely sensitive, 66 (18.5%) exhibited the MS phenotype, 65 (18.3%) were of the iMLSB phenotype, and 44 (12.4%) were of the cMLSB phenotype. Resistance by inducibility was thus higher than resistance by constitutivity, underscoring the need for the D-test.

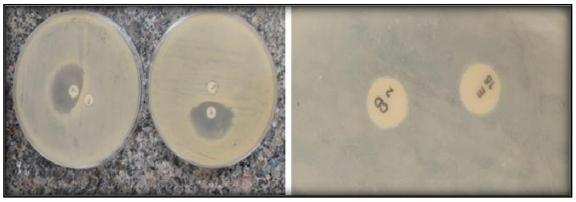

Image 1a. iMLSB **Image 1b. cMLSB**

Table 5: Distribution of Clindamycin Resistance Phenotypes in MRSA and MSSA

Phenotype	MRSA (n=90)	MSSA (n=266)
Sensitive	24	157
MS phenotype	26	40
iMLSB	23	42
cMLSB	17	27

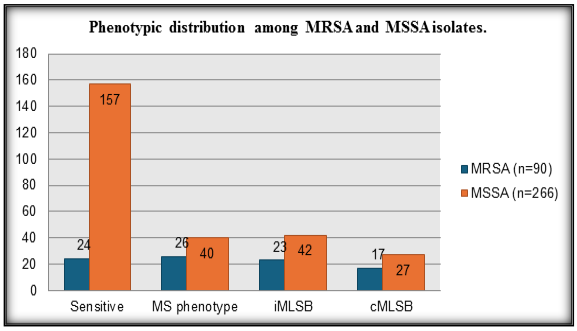

Figure 7: Bar chart demonstrating phenotypic distribution among MRSA and MSSA isolates.

Resistance phenotypes for MRSA and MSSA strains were compared, and the results showed that inducible and constitutive resistance to clindamycin was more common among MRSA isolates. Among MRSA isolates, the sensitive phenotype accounted for 24 (26.7%), the MS phenotype for 26 (28.9%), iMLSB for 23 (25.6%), and cMLSB for 17 (18.9%). Among MSSA isolates, the sensitive phenotype accounted for 157 (59.0%), the MS phenotype for 40 (15.0%), iMLSB for 42 (15.8%), and cMLSB for 27 (10.2%). These findings indicate that inducible clindamycin resistance was proportionally higher among MRSA isolates.

The chi-square test was conducted to assess the relationship between methicillin resistance and clindamycin resistance phenotypes. The contingency table had four groups (sensitive, MS phenotype, iMLSB, and cMLSB) with stratification based on whether isolates were MRSA or MSSA. This produced a chi-square value with its associated p-value using the Python program. Overall, the distribution demonstrated a higher proportion of inducible and constitutive clindamycin resistance among MRSA isolates compared with MSSA isolates, supporting the need for routine D-testing in methicillin-resistant strains.

DISCUSSION

The increasing prevalence of antimicrobial resistance among Staphylococcus aureus has become a major therapeutic challenge worldwide. Clindamycin continues to be an attractive treatment option because of its excellent oral bioavailability, superior tissue penetration, ability to inhibit bacterial toxin production, and effectiveness against both methicillin-sensitive Staphylococcus aureus (MSSA) and selected methicillin-resistant Staphylococcus aureus (MRSA) infections. However, inducible resistance mediated by erm genes may not be detected by routine antimicrobial susceptibility testing, potentially resulting in inappropriate clindamycin therapy and subsequent treatment failure. Consequently, routine detection of inducible clindamycin resistance by the D-test has become an important component of antimicrobial stewardship programs.[3,6,7,13]

The present study evaluated 356 non-duplicate clinical isolates of Staphylococcus aureus recovered from patients attending a tertiary care centre in North India. This sample size is larger than those reported by Sida et al. (297 isolates) and Kalbhor et al. (318 isolates), providing a robust dataset for evaluating local resistance patterns.

Distribution of MRSA: In the current study, 25.3% of isolates were found to be MRSA, whereas 74.7% of isolates were MSSA. The prevalence of MRSA seen in the study is similar to that reported by Kalbhor et al., where the prevalence of MRSA was 20.44%, and Sida et al., who noted 30.3% MRSA isolates. The

moderate prevalence of MRSA seen in the study could be because of proper infection prevention and control strategies, rational use of antimicrobials, and infection control measures in hospitals. However, one of every four isolates of S. aureus being methicillin resistant is clinically significant because infections due to MRSA cause increased morbidity, lengthened hospital stays, increased treatment costs, and limited treatment options.[1,14,15,16]

Antibiotic Resistance Pattern: The antibiogram derived from the above study showed 100% resistance to penicillin, which is consistent with the well-known prevalence of β-lactamase production in S. aureus. Several Indian studies by Kalbhor et al. and Sida et al. have similarly shown penicillin resistance close to 100%. Other antibiotics showing high resistance levels include cotrimoxazole (70.8%), ciprofloxacin (64.0%), gentamicin (62.0%), tetracycline (59.0%), and erythromycin (58.7%). This indicates increased prevalence of multidrug resistance among S. aureus strains and highlights the need for local antibiograms.[5,14,15]

However, importantly, all isolates in the current study were still sensitive to vancomycin and linezolid. This is in agreement with previous reports by Kalbhor et al. and Sida et al. who also did not observe any resistance to the above antimicrobials. These results indicate that vancomycin and linezolid may still be used as viable treatment options for MRSA infection.[14,15]

Prevalence of Inducible Clindamycin Resistance: The main goal of the current study was to establish the frequency of the inducible resistance to clindamycin. In the 356 strains isolated in the study, 65 (18.3%) exhibited inducible MLSB resistance. This frequency can be considered as average compared to previous works. For example, Sida et al. reported 13.46% of the inducible resistance, while Kalbhor et al. observed 20.13%. Likewise, Majhi et al. reported a frequency of approximately 22% of the inducible resistance in S. aureus.[14,15,17]

The variation observed among different studies may be attributed to differences in geographical location, antibiotic prescribing practices, infection control measures, patient population, prevalence of MRSA, and local antimicrobial stewardship programs. Despite these regional differences, all published studies consistently demonstrate that inducible clindamycin resistance represents a clinically significant proportion of S. aureus isolates and should not be overlooked during routine susceptibility testing.[6,7,17]

Constitutive MLSB Resistance: Constitutive MLSB resistance was observed in 12.4% of isolates in the present study. This finding closely resembles the results reported by Kalbhor et al. (13.52%) and is comparable with those described in earlier Indian studies.[14,17]

Unlike inducible resistance, constitutive resistance is readily identified during routine susceptibility testing because isolates exhibit resistance to both erythromycin and clindamycin. However, the presence of constitutive resistance further narrows available therapeutic options and reflects widespread dissemination of erm-mediated resistance mechanisms.[3,18]

MS Phenotype: The MS phenotype accounted for 18.5% of isolates. This phenotype results from the msrA-mediated active efflux mechanism and generally retains susceptibility to clindamycin because ribosomal target modification is absent. Recognition of the MS phenotype is clinically important because clindamycin remains an appropriate therapeutic option in these isolates, unlike those exhibiting inducible resistance.[3,18]

Association between MRSA and Inducible Resistance: One of the most important observations of the present study was the higher prevalence of inducible clindamycin resistance among MRSA isolates compared with MSSA isolates.

Among MRSA isolates, approximately one-fourth demonstrated inducible resistance, whereas the proportion was lower among MSSA isolates. Similar findings have consistently been reported by Majhi et al., Sida et al., and Kalbhor et al., all of whom observed significantly higher frequencies of inducible resistance among MRSA isolates than MSSA isolates.[14,15,17]

This association is clinically relevant because MRSA isolates already possess limited treatment options. Failure to detect inducible resistance may result in inappropriate use of clindamycin and eventual therapeutic failure.[3,6]

Clinical Implications: The findings of the present study have several practical implications for routine clinical microbiology laboratories.

Antimicrobial susceptibility testing is not sufficiently sensitive to confirm the presence of inducible clindamycin resistance. As a result, some isolates will incorrectly be reported as clindamycin susceptible. Performing the D-test along with the routine antimicrobial susceptibility testing requires little additional effort. The test is cost-effective, technically easy to perform, and can be done at the same time as routine Kirby-Bauer testing. D-test routine performance will increase the sensitivity of laboratory reporting and help physicians with antimicrobial selection. Institution-specific surveillance results from studies like the current one provide the basis for antimicrobial stewardship programs.[3,6,7,13]

Strengths of the Study: The current research has several strengths. It has a relatively high sample size of 356 isolates, larger than that of other similar Indian studies, and includes both MRSA and MSSA isolates. The study employed a CLSI-approved D-test technique together with antimicrobial susceptibility pattern analysis, and it generated an institutional antibiogram for empirical treatment. Statistical analysis and visualization were performed using Python software, and the study provides current data on the local epidemiology of the North India region.

Limitations: Despite its strengths, the present study has certain limitations. It was conducted at a single

tertiary care centre; therefore, the findings may not be generalizable to other regions. Molecular characterization of resistance genes (such as ermA, ermB, ermC, and msrA) was not performed, clinical treatment outcomes of patients receiving clindamycin were not evaluated, and minimum inhibitory concentrations (MICs) for clindamycin were not determined.

Future multicentre studies incorporating molecular characterization and clinical outcome assessment would provide a more comprehensive understanding of inducible clindamycin resistance.

CONCLUSION

The current study reports a high prevalence of inducible resistance to clindamycin in clinical isolates of Staphylococcus aureus obtained from a tertiary care centre in North India. About 20% of the isolates showed inducible MLSB pattern, being more frequent among MRSA isolates. The isolates showed complete resistance to penicillin while remaining susceptible to vancomycin and linezolid. The results emphasize the inability of routine susceptibility test to detect inducible resistance and highlight the need for carrying out the D-test in erythromycin-resistant and clindamycin-susceptible isolates. Routine implementation of the D-test will improve the accuracy of antimicrobial susceptibility reporting, reduce inappropriate clindamycin use, minimize therapeutic failure, and strengthen institutional antimicrobial stewardship programs.

Acknowledgements: The authors acknowledge the technical staff of the Department of Microbiology, Agilus Diagnostics & Fortis Escorts Hospital Faridabad, Haryana for their assistance in specimen processing, culture, antimicrobial susceptibility testing, and maintenance of laboratory records. The authors also thank the hospital administration for providing the necessary laboratory infrastructure and support to conduct this study.

REFERENCES

1. Tong SYC, Davis JS, Eichenberger E, Holland TL, Fowler VG Jr. Staphylococcus aureus infections: epidemiology, pathophysiology, clinical manifestations, and management. Clin Microbiol Rev. 2015;28(3):603-61. doi:10.1128/CMR.00134-14.
2. Stevens DL, Bisno AL, Chambers HF, Dellinger EP, Goldstein EJC, Gorbach SL, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. Clin Infect Dis. 2014;59(2):e10-52. doi:10.1093/cid/ciu296.
3. Leclercq R. Mechanisms of resistance to macrolides and lincosamides: nature of the resistance elements and their clinical implications. Clin Infect Dis. 2002;34(4):482-92. doi:10.1086/324626.
4. Roberts MC, Sutcliffe J, Courvalin P, Jensen LB, Rood J, Seppala H. Nomenclature for macrolide-lincosamide–streptogramin B resistance determinants. Antimicrob Agents Chemother. 1999;43:2823–30. doi: 10.1128/aac.43.12.2823. [DOI] [PMC free article] [PubMed] [Google Scholar]
5. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing. 35th ed. CLSI supplement M100. Wayne (PA): Clinical and Laboratory Standards Institute; 2025.
6. Fiebelkorn KR, Crawford SA, McElmeel ML, Jorgensen JH. Practical disk diffusion method for detection of inducible clindamycin resistance in Staphylococcus aureus and coagulase-negative staphylococci. J Clin Microbiol. 2003;41(10):4740-4. doi:10.1128/JCM.41.10.4740-4744.2003.
7. Prabhu K, Rao S, Rao V. Inducible clindamycin resistance in Staphylococcus aureus isolated from clinical samples. J Lab Physicians. 2011;3(1):25-7. doi:10.4103/0974-2727.78558.
8. Thapa D, Shrestha B, Singh A, et al. Staphylococcus aureus with inducible clindamycin resistance among patients attending a tertiary care hospital, Kathmandu, Nepal. Ann Clin Microbiol Antimicrob. 2021;20:82. doi:10.1186/s12941-021-00486-5.
9. Khursheed M, Sulaiman M, Alhassan AM, et al. Clindamycin-resistant Staphylococcus aureus: investigation into phenotypic and genotypic profiles. PLoS One. 2025;20(8):e0329467. doi:10.1371/journal.pone.0329467.
10. Cheesbrough M. District laboratory practice in tropical countries. Part 2. 2nd ed. Cambridge: Cambridge University Press; 2006.
11. Forbes BA, Sahm DF, Weissfeld AS. Bailey & Scott's diagnostic microbiology. 14th ed. St Louis (MO): Elsevier; 2017.
12. Bauer AW, Kirby WMM, Sherris JC, Turck M. Antibiotic susceptibility testing by a standardized single disk method. Am J Clin Pathol. 1966;45(4):493-6. doi:10.1093/ajcp/45.4_ts.493.
13. Banik A, Khyriem AB, Gurung J, et al. Inducible and constitutive clindamycin resistance in Staphylococcus aureus and coagulase-negative staphylococci in a northeastern Indian tertiary care hospital. J Infect Dev Ctries. 2015;9(7):725-31.
14. Kalbhor P, Wanjare V, Shrikhande S. Detection of inducible and constitutive clindamycin resistance among clinical isolates of Staphylococcus aureus in a tertiary care hospital. Indian J Appl Res. 2018;8(9):70-72
15. Sida H, Chauhan B, Pethani J, Patel L, Shah P. Inducible clindamycin resistance among clinical isolates of Staphylococcus aureus. Natl J Med Res. 2015;5(4):268-71.
16. Chambers HF, DeLeo FR. Waves of resistance: Staphylococcus aureus in the antibiotic era. Nat Rev Microbiol. 2009;7(9):629-41. doi:10.1038/nrmicro2200.
17. Majhi S, Dash M, Mohapatra D, Mohapatra A, Chayani N. Detection of inducible and constitutive clindamycin resistance among Staphylococcus aureus isolates in a tertiary care hospital, Eastern India. Avicenna J Med. 2016;6(3):75-80. doi:10.4103/2231-0770.184066.
18. Steward CD, Raney PM, Morrell AK, et al. Testing for induction of clindamycin resistance in erythromycin-resistant isolates of Staphylococcus aureus. J Clin Microbiol. 2005;43(4):1716-21. doi:10.1128/JCM.43.4.17161721.2005.