DOI: 10.7860/NJLM/2025/77170.2911

Original Article

Microbiology Section

Prevalence of *Staphylococcus aureus* Isolated from Clinical Samples that Exhibits Inducible Clindamycin Resistance: A Cross-sectional Study

AKANSHA GOYAL[1], AKANSHA ARYA[2], KAJAL RAJPUT[3], SAPNA CHAUHAN[4]

ABSTRACT

Introduction: The resistance to Macrolide-Lincosamide-Streptogramin B (MLS_B) in *Staphylococcus aureus* is often caused by *erm* genes, which can either be constitutive MLS_B ($cMLS_B$) or inducible MLS_B (iMLSB). Clindamycin (CLI), a lincosamide, and erythromycin (ERY), a macrolide, both act by binding to the 50S ribosomal subunits of bacterial cells, inhibiting protein synthesis. Methylation of the ribosomal target site in iMLSB-resistant isolates displays resistance to ERY but allows susceptibility to CLI. $iMLS_B$ resistance emerges when a strong inducer of the methylase enzyme, such as ERY, is present. Identifying $iMLS_B$ resistance is crucial for effectively managing *S. aureus*.

Aim: To detect the prevalence of inducible CLI resistance in *S. aureus* isolates from various clinical samples and to determine the association between methicillin resistance and inducible CLI resistance in *S. aureus* isolates.

Materials and Methods: The study was a cross-sectional study conducted at Muzaffarnagar Medical College and Hospital, Muzaffarnagar, Uttar Pradesh, India over a period of six months, from January 2024 to June 2024. A total of 100 isolates of *S. aureus* obtained from various clinical samples were included. Simple random sampling was employed to ensure an unbiased selection of isolates. Only culture-confirmed *S. aureus* isolates were considered, while duplicate isolates and samples showing polymicrobial growth were excluded from the study. The sample population comprised individuals of varying ages and genders who sought treatment at the hospital during the study period due to symptoms indicating potential infections. The *S. aureus* isolates were initially identified using standard biochemical techniques and were then tested for susceptibility using the modified Kirby-Bauer disc diffusion method on Mueller-Hinton agar plates, following Clinical and Laboratory Standards Institute (CLSI) guidelines. The CLSI guidelines were also followed to test for inducible resistance to CLI using the 'D test' method. Data were entered and analysed using the Statistical Package for the Social Sciences (SPSS) software, version 24.0, with statistical significance considered at a p-value <0.05.

Results: Routine disc diffusion testing was employed to examine antibiotic susceptibility in 100 *S. aureus* strains, revealing that 44 (44%) displayed resistance to ERY. Out of the 100 strains, 22 (22%) showed $iMLS_B$ resistance, 27 (27%) were $cMLS_B$-positive, and 15 (15%) had the Macrolide-Streptogramin B (MS) phenotype. In this study, it was discovered that 18 (82%) of the *S. aureus* isolates that were positive for the D test were Methicillin-Resistant *S. aureus* (MRSA), while 4 (18%) were sensitive to cefoxitin (MSSA).

Conclusion: This study revealed a substantial prevalence of inducible CLI resistance among *S. aureus* isolates, with a pronounced association in MRSA strains. The strong correlation between methicillin and $iMLS_B$ resistance underscores the critical need for routine D-test screening to guide precise antimicrobial therapy, mitigate treatment failures and support effective antimicrobial stewardship practices.

Keywords: D-test, Methylation, Methicillin resistant *Staphylococcus aureus*

INTRODUCTION

Staphylococcus aureus is a Gram-positive coccus from the Micrococcaceae family. Its cells can be found individually or grouped in pairs, tetrads, and grape-like structures. *Staphylococcus aureus* commonly resides in the human body without causing symptoms and is a prevalent opportunistic pathogen [1]. However, in specific circumstances, this bacterium can cause a variety of more serious infections that impact soft-tissue and the outer layers of the skin. Certain strains can form enterotoxins, which lead to cases of food poisoning. The frequency of invasive infections, such as pneumonia, bloodstream infections and infections linked to catheters or artificial devices, has been increasing due to the presence of Community-Acquired (CA) and Hospital-Acquired (HA) MRSA [2-4]. *S. aureus* Bacteremia (SAB) often leads to metastatic conditions, such as septic arthritis, osteomyelitis and Infective Endocarditis (IE) [5].

The prevalence of MRSA infections is increasing, leading to a higher demand for antibiotics like macrolides, lincosamides, and streptogramin B (MLS_B) to address *S. aureus* infections [6]. MLS_B resistance in *S. aureus* is usually caused by *erm* genes, which can be either $cMLS_B$ or $iMLS_B$. Both CLI and ERY, belonging to different drug classes-lincosamides and macrolides, respectively-inhibit protein synthesis in bacterial cells by binding to their 50S ribosomal subunits. Methylation of the ribosomal target site of staphylococci may lead to resistance against both of these antibiotics [7]. Through the constitutive resistance mechanism controlled by *msrA* genes, *S. aureus* strains exhibit resistance to ERY and susceptibility to CLI in both in-vivo and in-vitro settings. While being treated, the strains that are always resistant do not develop resistance to CLI. The isolates resistant to $iMLS_B$ display resistance to ERY but are sensitive to CLI. In the presence of ERY, a potent methylase enzyme inducer, $iMLS_B$ resistance emerges. Contrary to $cMLS_B$ resistance, standard susceptibility testing does not detect $iMLS_B$ resistance. The D-zone test can detect inducible CLI resistance, which is shown by a D-shaped inhibition zone around CLI when exposed to ERY in-vitro. Identifying $iMLS_B$ resistance is crucial for managing *S. aureus* effectively.

Alternatively, CLI usage may result in treatment ineffectiveness due to the emergence of inherent resistance [6,8]. However, the underreporting of inducible CLI resistance in *S. aureus* is primarily due to the lack of research on its identification. In hospital settings, traditional susceptibility tests for *S. aureus* paradoxically do not include detecting inducible CLI resistance. Local resistance data are crucial for effectively managing infections, maximising antibiotic use, and guiding empirical treatment.

The current study aimed to utilise the D-test to establish the prevalence of *S. aureus* strains exhibiting inducible CLI resistance $iMLS_B$ in our specific location. Present study also sought to establish the relationship between inducible CLI resistance and MRSA. The increasing worldwide problem of antibiotic resistance indicates that we may soon see the end of successful antibiotic treatment. Resistance impacts all aspects of medicine and makes it more challenging to carry out effective empirical therapy. Paradoxically, typical susceptibility tests for *S. aureus* in hospital settings still do not include the detection of methicillin and inducible CLI resistance.

Effective infection management, empirical treatment guidance and antibiotic usage optimisation all depend on local geographical data. The aim and purpose of the study were to investigate the prevalence of methicillin and inducible CLI resistance in *S. aureus* at a tertiary care hospital and to determine the association between them. The results of this surveillance study will contribute recent data to the existing literature and assist clinicians in the area in choosing the right medications, thereby enhancing the clinical management of infections.

MATERIALS AND METHODS

This cross-sectional study was conducted at Muzaffarnagar Medical College and Hospital, Muzaffarnagar, Uttar Pradesh, India from January 2024 to June 2024. Clinical samples were collected in real-time during the study period, ensuring that newly acquired data were available for analysis. The procedures followed in this study were in accordance with the ethical standards set by the committee on human experimentation (both institutional and national). The study was approved by the Institutional Ethics Committee of Muzaffarnagar Medical College, UP, India (MMC/IEC/2024/166). Informed consent was obtained from patients prior to sample collection.

Sample size calculation:

The sample size was determined using the Cochran's formula [9].

$n=Z^2\times(p\times q)/e^2$

$=1.96^2\times(0.086\times0.914)/0.06^2$

$=83.87$

Where,

n=sample size,

Z=1.96 for 95% Confidence Interval (CI),

p=D-test positive prevalence was taken from the previous study, 08.64% [10]

q=1-p,

e=margin of error, 6%

The sample size calculation yielded 83.87; however, 100 samples were included to enhance statistical power, improve reliability, and account for potential data loss, ensuring more robust and optimisation findings.

One hundred *S. aureus* isolates were obtained from various clinical samples, including pus, wound swabs, aspirates, blood and sterile fluids, and were subsequently examined.

Inclusion criteria: All inpatients and outpatients from all age and sex groups who were asked by doctors to undergo standard microbiological culture and antibiotic susceptibility tests were included in the study.

Exclusion criteria: Individuals who had taken antibiotics in the previous four weeks and patients who refused to give consent were excluded from the study. Duplicate samples from the same patient were also omitted from the study.

Study Procedure

The *S. aureus* isolates were initially identified using standard biochemical techniques (catalase test, slide and tube coagulase test). They were then tested for susceptibility using the modified Kirby-Bauer disc diffusion method on Mueller-Hinton agar plates, following CLSI guidelines [11]. The antibiotics tested included trimethoprim-sulfamethoxazole (SXT) (25 µg), ERY (15 µg), ciprofloxacin (5 µg), CLI (2 µg), linezolid (30 µg), tetracycline (30 µg), and cefoxitin (30 µg). A zone of inhibition of 19 mm or smaller around a cefoxitin disc signifies the presence of MRSA.

The CLSI guidelines were followed to test for inducible resistance to CLI using the 'D test' method [11]. An agar plate with Mueller-Hinton agar was prepared using 0.5 McFarland standard bacterial suspensions. ERY (15 µg) and CLI (2 µg) discs were placed 15 mm apart on the plate. Inducible CLI resistance was indicated by the flattening of the D-shaped zone around the CLI disc between the two discs following overnight incubation at 37°C. After the testing process, three separate characteristics were identified and examined. This interpretation applied only to strains of *S. aureus* that showed resistance to ERY. All strains that were susceptible to ERY were eliminated from consideration.

MS phenotype: This characteristic was observed in a staphylococcal strain that displayed resistance to ERY (zone size ≤13 mm) but susceptibility to CLI (zone size ≥21 mm), resulting in a circular zone of inhibition around CLI [12].

Inducible MLS_B ($iMLS_B$) phenotype: This phenotype was found in a staphylococcal strain that was resistant to ERY (zone size ≤13 mm) but susceptible to CLI (zone size ≥21 mm), exhibiting a D-shaped area of inhibition around CLI that tapered toward the ERY disc [12].

Constitutive MLS_B phenotype: Staphylococcal isolates showing resistance to CLI (zone size ≤14 mm) and ERY (zone size ≤13 mm), with a circular zone of inhibition around CLI, were categorised as having this characteristic [12].

Following the established Standard Operating Procedure (SOP) ensured data quality from data collection to laboratory identification. The culture media were prepared in accordance with the manufacturer's guidelines and sterility was assessed by incubating a representative portion (5% of the total prepared media) at 37°C overnight, followed by evaluation for bacterial growth [11,13]. Media batches exhibiting growth were discarded and replaced with freshly prepared sterile media.

A standard disc diffusion procedure was used to perform Quality Control (QC) on the ERY and CLI discs with *S. aureus* ATCC 25923. Additional QC measures were implemented using in-house selected *S. aureus* strains, representing both positive and negative D-test results, to ensure the reliability of the testing process. All HiMedia discs were utilised for both the media and the antibiotics.

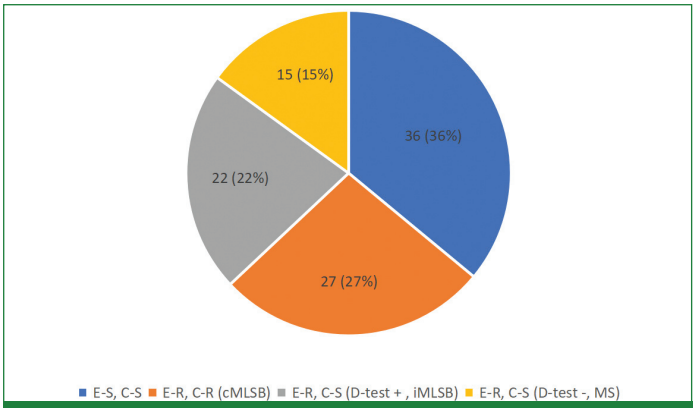
STATISTICAL ANALYSIS

Data were entered and analysed using SPSS software version 24.0, Chi-square test was used as a significance test with statistical significance established when the p-value was below 0.05.

RESULTS

Standard disc diffusion testing was employed to evaluate the antibiotic susceptibility of 100 *S. aureus* strains, revealing that 44 (44%) of them exhibited resistance to erythromycin (ERY). A total of 27 (27%) of the *S. aureus* isolates were resistant to both ERY and CLI, indicating a constitutive MLS_B phenotype; 36 (36%) were sensitive to both drugs, 22 (22%) showed an inducible $iMLS_B$ phenotype, and 15 (15%) were negative for the D-test, indicating an MS phenotype. These findings have been illustrated in [Table/Fig-1].

The association between methicillin resistance and CLI resistance is demonstrated in [Table/Fig-2]. A total of 18 (82%) of the *S. aureus* isolates that tested positive for the D-test were identified as MRSA, while 4 (18%) were sensitive to cefoxitin (MSSA) in this research.

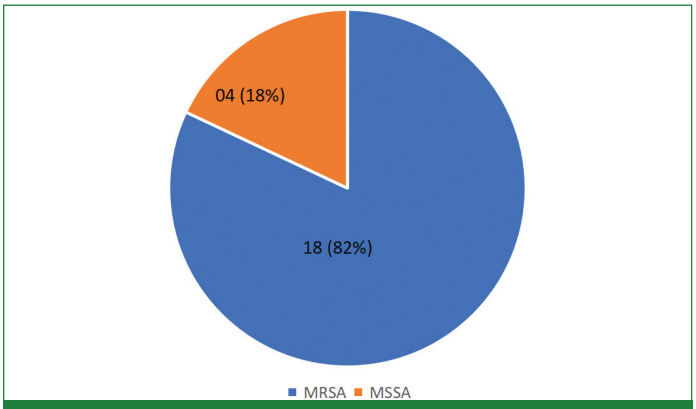


[Table/Fig-1]: Susceptibility of *S. aureus* isolates to Erythromycin (ERY) and Clindamycin (CLI).
E: Erythromycin; C: Clindamycin; S: Sensitive; R: Resistant; cMLSB: constitutive MLSB phenotype; iMLSB: inducible MLSB phenotype; MS-MS phenotype

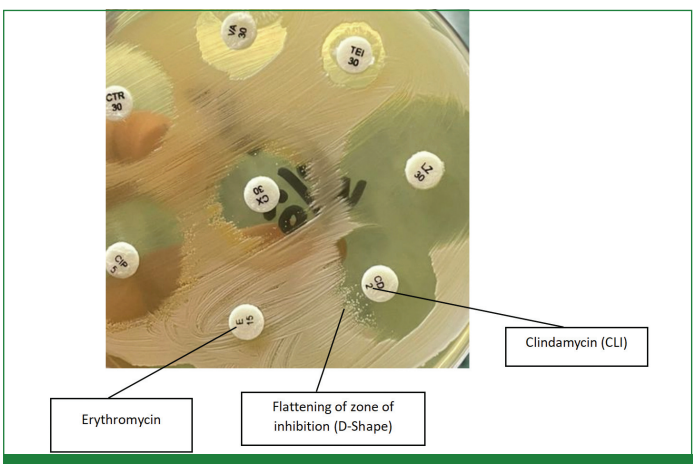
Clindamycin resistance	Methicillin resistance		Total (n)	p-value
	MRSA n (%)	MSSA n (%)		
E-S, C-S	29 (38.7)	07 (28)	36 (36)	0.355 (p>0.05, Not significant)
E-R, C-R (cMLSB)	17 (22.7)	10 (40)	27 (27)	
E-R, C-S (D-test +, iMLSB)	18 (24)	04 (16)	22 (22)	
E-R, C-S (D-test -, MS)	11 (14.7)	04 (16)	15 (15)	
Total	75 (100)	25 (100)	100 (100)	

[Table/Fig-2]: Association between resistance to methicillin and resistance to CLI.
E: Erythromycin; C: Clindamycin; S: Sensitive; R: Resistant; cMLSB: constitutive MLSB phenotype; iMLSB: inducible MLSB phenotype; MS-MS phenotype; MSSA: Methicillin sensitive *S. aureus*; MRSA: Methicillin resistant *S. aureus*

[Table/Fig-3] shows that MRSA isolates exhibited a greater proportion of constitutive and inducible resistance compared to MSSA isolates. [Table/Fig-4] illustrates the D-test showing inducible CLI resistance, with the flattening of the zone of inhibition around the CLI disc clearly observed.



[Table/Fig-3]: Association between methicillin resistance and D-test.



[Table/Fig-4]: D-test showing inducible Clindamycin (CLI) resistance.

DISCUSSION

Clindamycin (CLI) has recently emerged as an effective treatment for certain Staphylococcal infections, especially those affecting the skin and soft tissues, and as a substitute for penicillin in individuals who are allergic to it [14]. If *S. aureus* is reported to be susceptible to CLI without first undergoing testing for inducible resistance, CLI treatment may be administered inappropriately. Inducible resistance to CLI is indicated by a positive D-test.

In this current research, 44 out of 100 *S. aureus* isolates were found to be resistant to ERY, accounting for 44 (44%) of the total isolates. Every strain resistant to ERY underwent testing with the D-test. This research showed that D-test positivity was found in 22 (22%) of the *S. aureus* isolates. Numerous studies from India have documented a high prevalence of $cMLS_B$ and $iMLS_B$ phenotypes of *S. aureus*. Sefi N et al., reported 56 (26.54%) $cMLS_B$ isolates and 24 (11.37%) $iMLS_B$ isolates [15]. Another study from eastern India by Dash M et al., documented 37 (17.7%) $cMLS_B$ and 46 (22%) $iMLS_B$ isolates [16]. A study by Prabhu K et al., reported relatively lower prevalence of $cMLS_B$ and $iMLS_B$ phenotypes (18 (9.47%), 20 (10.52%)) [14]. A comparison of CLI resistance prevalence from various studies is presented in [Table/Fig-5] [10,14-18].

Author's name	E-S, C-S n (%)	$cMLS_B$ Phenotype n (%)	$iMLS_B$ Phenotype n (%)	MS Phenotype n (%)
Prabhu K et al., (2011) [14]	136 (71.57)	18 (9.47)	20 (10.52)	16 (8.42)
Sefi N et al., (2012) [15]	100 (47.39)	56 (26.54)	24 (11.37)	25 (11.85)
Mansouri S and Sadeghi J (2014) [10]	83 (51.23)	46 (28.39)	14 (8.64)	15 (9.26)
Dash M et al., (2016) [16]	85 (40.6)	37 (17.7)	46 (22)	41 (19.6)
Shetty J and Afroz Z (2017) [17]	45 (34.6)	43 (33.3)	22 (16.9)	20 (15.4)
Thapa D et al., (2021) [18]	10 (26)	07 (18)	14 (37)	07 (18)
Present study, 2025	36 (36)	27 (27)	22 (22)	15 (15)

[Table/Fig-5]: Comparative study of susceptibility of *S. aureus* isolates to Erythromycin (ERY) and Clindamycin (CL) by various authors [10,14-18].

Most of the *S. aureus* isolates that tested positive in the D-test ($iMLS_B$ phenotype), specifically 18 (82%), were found to be MRSA. This result was similar to studies by Mansouri S and Sadeghi J, Prabhu K et al., Sefi N et al., Dash M et al., as well as Shetty J and Afroz Z [10,14-17]. In contrast, the study by Thapa D et al., reported that 8 (57.2%) of D-test positive association were MSSA and 6 (42.8%) were MRSA [18]. The association of iMLSB isolates and methicillin resistance is shown in [Table/Fig-6] [10,14-18].

Author's name	Total $iMLS_B$	MRSA n (%)	MSSA n (%)
Prabhu K et al., (2011) [14]	20	12 (60)	08 (40)
Sefi N et al., (2012) [15]	24	18 (75)	06 (25)
Mansouri S and Sadeghi J (2014) [10]	14	11 (78.6)	03 (21.4)
Dash M et al., (2016) [16]	46	32 (69.6)	14 (30.4)
Shetty J and Afroz Z (2017) [17]	22	13 (59)	09 (41)
Thapa D et al., (2021) [18]	14	06 (42.8)	08 (57.2)
Present study, 2025	22	18 (82)	04 (18)

[Table/Fig-6]: Comparative study of $iMLS_B$ isolates showing methicillin resistance.

A comparative analysis of the correlation between resistance to methicillin and CLI by different authors is presented in [Table/Fig-7] [10,14-18].

Author's name	MRSA						MSSA					
	$iMLS_B$ Phenotype n (%)	$cMLS_B$ phenotype n (%)	MS phenotype n (%)	E-S, C-S phenotype n (%)	E-S, C-R	Total	$iMLS_B$ Phenotype n (%)	$cMLS_B$ Phenotype n (%)	MS Phenotype n (%)	E-S, C-S phenotype n (%)	E-S, C-R	Total
Prabhu K et al., (2011) [14]	12 (20)	10 (16.66)	08 (13.33)	30 (50)	-	60	08 (6.15)	08 (6.15)	08 (6.15)	106 (81.64)	-	130
Sefi N et al., (2012) [15]	18 (20.45)	46 (52.3)	14 (15.91)	09 (10.22)	01 (1.13)	88	06 (4.88)	09 (7.32)	12 (9.76)	91 (73.98)	05 (4.06%)	123
Mansouri S and Sadeghi J (2014) [10]	11 (11.95)	44 (47.8)	10 (10.8)	26 (28.2)	01 (1.08)	92	03 (4.28)	02 (2.8)	05 (7.1)	57 (81.4)	02 (2.85)	70
Dash M et al., (2016) [16]	32 (24.8)	30 (23.2)	29 (22.4)	38 (29.4)	-	129	14 (17.5)	07 (8.7)	12 (15)	47 (58.7)	-	80
Shetty J and Afroz Z (2017) [17]	13 (27.1)	25 (52.1)	08 (16.6)	02 (4.2)	-	48	09 (10.9)	18 (21.9)	12 (14.6)	43 (52.4)	-	82
Thapa D et al., (2021) [18]	06 (40)	03 (20)	02 (13.3)	04 (26.7)	-	15	08 (34.9)	04 (17.4)	05 (21.7)	06 (26)	-	23
Present study, 2025	18 (24)	17 (22.7)	11 (14.7)	29 (38.7)	-	75	04 (16)	10 (40)	04 (16)	07 (28)	-	25

[Table/Fig-7]: Comparative study of association between resistance to methicillin and resistance to Clindamycin (CLI) by various authors [10,14-18].

The increased occurrence of the $iMLS_B$ phenotype in MRSA infections compared to MSSA infections indicates that CLI treatment for MSSA infections is effective in many cases, whereas it may result in treatment failure for MRSA infections. In summary, present study discovered a significant occurrence of the inducible CLI resistance phenotype in this area. Authors recommend that a D-test should be conducted whenever CLI is planned for use in *S. aureus* infections to ensure effective treatment of patients. Proper and effective utilisation of CLI can reduce the need for alternative medications such as vancomycin and help avoid the emergence of resistance to these drugs.

Limitation(s)

Emphasising the main constraints of this research, such as the lack of real-time PCR assessment of gene expression and the genetic similarity among ERY-resistant strains, is crucial.

CONCLUSION(S)

The high levels of MRSA and $iMLS_B$ phenotypes in *S. aureus* underscore the importance of including the D-test and methicillin resistance test in regular susceptibility testing for *S. aureus* control. The D-test is a viable additional option for identifying inducible CLI resistance in clinical laboratory practices. Furthermore, identifying the presence of the *erm* gene in staphylococcal strains that show positive results in the D-test could help establish a standardised procedure for the test.

Authors' contribution: All authors listed have made a substantial, direct, and intellectual contribution to the work, and approved it for publication.

Data availability: All data generated or analysed during this study are included in the manuscript.

REFERENCES

[1] Stapleton PD, Taylor PW. Methicillin resistance in *Staphylococcus aureus*: Mechanisms and modulation. Science Progress. 2002;85(1):57-72.

[2] Ferry T, Perpoint T, Vandenesch F, Etienne J. Virulence determinants in *Staphylococcus aureus* and their involvement in clinical syndromes. Current Infectious Disease Reports. 2005;7(6):420-28.

[3] Kika B, Abazaj E, Petri O, Koraqi A. Prevalence of *Staphylococcus aureus* and methicillin-resistant *S.aureus* in adult hospitalized patients in "Mother Theresa" hospital center in Albania. Open Access Macedonian J Med Sci [Internet]. 2020;8(A):297-302.

[4] Frank AL, Marcinak JF, Mangat PD, Tjhio JT, Kelkar S, Schreckenberger PC, et al. Clindamycin treatment of methicillin-resistant *Staphylococcus aureus* infections in children. The Pediatr Infect Dis J. 2002;21(6):530-34.

[5] Van Hal SJ, Jensen SO, Vaska VL, Espedido BA, Paterson DL, Gosbell IB. Predictors of mortality in *Staphylococcus aureus* bacteremia. Clinical Microbiology Reviews. 2012;25(2):362-86.

[6] Levin TP, Suh B, Axelrod P, Truant AL, Fekete T. Potential clindamycin resistance in clindamycin-susceptible, erythromycin-resistant *Staphylococcus aureus*: Report of a clinical failure. Antimicrob Agents Chemother [Internet]. 2005 Feb 23 [cited 2019 Jul 21];49(3):1222-24. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC549252/.

[7] Steward CD, Raney PM, Morrell AK, Williams PP, McDougal LK, Jevitt L, et al. Testing for induction of clindamycin resistance in erythromycin-resistant isolates of *Staphylococcus aureus*. J Clin Micribiol. 2005;43(4):1716-21.

[8] Drinkovic D. Clindamycin treatment of *Staphylococcus aureus* expressing inducible clindamycin resistance. J Antimicrob Chemother. 2001;48(2):315-16.

[9] Cochran WG. Sampling Techniques. 3rd ed. New York: John Wiley & Sons; 1977.

[10] Mansouri S, Sadeghi J. Inducible clindamycin resistance in methicillin-resistant and-susceptible *Staphylococcus aureus* isolated from south east of Iran. Jundishapur J Microbiol. 2014;7(12):e11868.

[11] M100 Performance Standards for Antimicrobial Susceptibility Testing A CLSI supplement for global application [Internet]. Available from: https://clsi.org/media/tc4b1paf/m10033_samplepages-1.pdf,

[12] Lal UP, Ambhore N, Raut S, Mantri R, Malak N, Sharma P. Identification of inducible clindamycin resistance in *staphylococcus aureus* using automated Vitek-2 compact system and D test. J Med Sci Health [Internet]. 2024 Aug 15 [cited 2025 Feb 21];10(2):136-41.

[13] Mama M, Aklilu A, Misgna K, Tadesse M, Alemayehu E. Methicillin- and inducible clindamycin-resistant *Staphylococcus aureus* among patients with wound infection attending Arba Minch hospital, South Ethiopia. Int J Micribiol [Internet]. 2019 Apr 1 [cited 2019 Jul 21];2019:2965490.

[14] Prabhu K, Rao S, Rao V. Inducible clindamycin resistance in *Staphylococcus aureus* isolated from clinical samples. Journal of Laboratory Physicians [Internet]. 2011 [cited 2021 Mar 15];3(1):25-27.

[15] Seifi N, Kahani N, Askari E, Mahdipour S, Naderi NM. Inducible clindamycin resistance in *Staphylococcus aureus* isolates recovered from Mashhad, Iran. Iranian J Microbiol [Internet]. 2012;4(2):82-86.

[16] Dash M, Majhi S, 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.

[17] Shetty J, Afroz Z. Prevalence of constitutive and inducible clindamycin resistance among clinical isolates of *Staphylococcus aureus* in a tertiary care institute in North India. Int J Res Med Res. 2017;5(7):3120.

[18] Thapa D, Pyakurel S, Thapa S, Lamsal S, Chaudhari M, Adhikari N, et al. *Staphylococcus aureus* with inducible clindamycin resistance and methicillin resistance in a tertiary hospital in Nepal. Trop Med Health. 2021;49(1):99.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Microbiology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India.
2. Postgraduate Student, Department of Microbiology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India.
3. Postgraduate Student, Department of Microbiology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India.
4. Professor and Head, Department of Microbiology, Muzaffarnagar Medical College, Muzaffarnagar, Uttar Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Sapna Chauhan,
Professor and Head, Department of Microbiology, Muzaffarnagar Medical College,
Opp. Begrajpur Industrial Area, Muzaffarnagar-251001, Uttar Pradesh, India.
E-mail: connect.akansha@gmail.com; drsapnachauhan@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Dec 06, 2024
- Manual Googling: Mar 22, 2025
- iThenticate Software: Mar 24, 2025 (15%)

ETYMOLOGY: Author Origin

EMENDATIONS: 7

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Dec 06, 2024**
Date of Peer Review: **Feb 17, 2025**
Date of Acceptance: **Mar 26, 2025**
Date of Publishing: **Apr 01, 2025**