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RESEARCH ARTICLE OPEN ACCESS

Inducible Clindamycin Resistance and its Molecular Characterisation among Isolates of *Staphylococcus aureus* from A Tertiary Care Centre in South Kerala

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Abstract

A rise in methicillin-resistant *S. aureus* (MRSA) infections in hospitals and the population has led to an increase in Macrolide–Lincosamide–Streptogramin type B (MLSB) antibiotic resistance. This has led to increased treatment costs, length of hospital stay and morbidity, as therapy has grown increasingly challenging. As a result, it is imperative that *S. aureus* isolates and their susceptibility patterns especially to clindamycin and erythromycin, be accurately identified and reported. Of the 341 (100%) *S. aureus* strains that were recovered from various clinical samples, such as blood, pus, urine, and sputum, 267 (78.6%) were MSSA (methicillin-sensitive *S. aureus*) and 74 (21.4%) were MRSA. iMLSB (inducible MLSB) resistance detected by D-test was present in 42 (12.3%) isolates, out of which 23 (15.6%) were MSSA and 19 (33.9%) were MRSA. Real-time PCR was done for gene detection on all iMLSB positive isolates. The majority of the isolates - 22 (52.4%) showed both *erm*A and *erm*C genes; out of which 13 (56.5%) were MSSA, whereas 9 (47.4%) were MRSA. To correctly identify the true phenotypes sensitive to clindamycin and those showing iMLSB resistance, a reasonably priced, D-test (double disc diffusion test) can be included in routine antibiotic susceptibility testing in clinical settings for all erythromycin-resistant staphylococcal isolates. By following this technique, clinicians treating patients with infections brought on by inducible clindamycin-resistant strains will be guided to forego clindamycin from their treatment regimens, helping to prevent therapeutic failures.

Keywords: D-test, *Staphylococcus aureus*, Inducible Clindamycin Resistance, *erm* Genes, iMLSB, *erm*A, *erm*C, PCR

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INTRODUCTION

Since the emergence of MRSA, there have been few therapeutic options for treating staphylococcal infections. Among the antibiotics in the macrolide-lincosamide-streptogramin B (MLSB) family is one such substitute, clindamycin which is most commonly used, because of its superior pharmacokinetic properties.[1] It can be used as a substitute antibiotic for those allergic to penicillin and as a follow-up treatment following intravenous therapy because of its potent oral absorption,[2] and to treat outpatients. It also accumulates in abscesses and does not require dosage modifications even in cases of renal impairment.[3]

The acquirement of erythromycin resistance methylase (*erm*) genes, encoding enzymes that methylate the 23S rRNA, is the most common cause of resistance to MLSB antibiotics.[4,5] Three methylase genes are present in staphylococci: *erm*A, *erm*B, and *erm*C. *erm*A gene expression is induced by erythromycin, whereas *erm*B and *erm*C are either constitutive or inducible.[6] Antibiotic-resistant *S. aureus* strains with *erm* genes exhibit cross-resistance to MLSB antibiotics. The *msr*A gene codes for the efflux mechanism, which is the other mechanism that causes the MS phenotype.

A decline in the drug's efficacy could be caused by inducible clindamycin resistance.

Figure 1. Blood agar with β-hemolytic colonies of *S. aureus*

The modern necessity for directing treatment is the *in vitro* identification of macrolide resistance and accurate susceptibility test interpretation.[7] In this study, the D-test was performed to asses the resistance patterns of erythromycin and clindamycin. The *erm* gene that induced inducible clindamycin resistance was found using polymerase chain reaction (PCR). Understanding a pathogen's precise antibiotic susceptibility pattern is essential for making informed therapeutic decisions. Therefore, while using clindamycin to treat staphylococcal infections, figuring out the fundamental processes that result in MLSB antibiotic resistance is crucial.

Aims and objectives

To identify using the D-test, the proportion of *S. aureus* isolates with inducible clindamycin resistance and identify the resistant genes among them by molecular methods.

MATERIALS AND METHODS

A descriptive study was conducted in the Microbiology department of a tertiary care centre in South Kerala. Over the course of a year, 341 non-duplicate isolates of *S. aureus* were obtained from a variety of clinical samples obtained from IPD and OPD patients. Excluded from the study were clinical isolates from individuals receiving clindamycin treatment already. The organism was identified using colony characteristics, golden yellow β-hemolytic colonies on blood agar (Figure 1), Gram staining, catalase test, coagulase

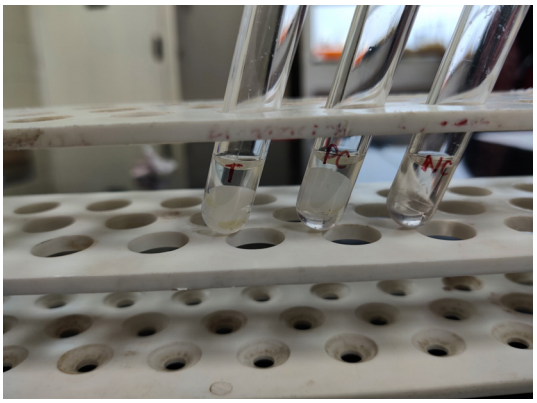

Figure 2. Tube coagulase test

test (Figure 2), standard biochemical reactions, and the VITEK 2 automated system. The Kirby Bauer disc diffusion method was utilized to perform and evaluate the antibiotic susceptibility testing, adhering to the CLSI guidelines 2020. Isolates of MRSA were identified using the cefoxitin disc diffusion method on Mueller-Hinton agar.

Disc diffusion test (D-zone test) for the detection of inducible clindamycin resistance

In addition to routine antibiotic susceptibility testing, on a Mueller-Hinton Agar plate, bacterial suspension of *S. aureus* with a turbidity of 0.5 McFarland was inoculated. Erythromycin (15 µg) disc was kept 15-26 mm apart from clindamycin (2 µg) disk and incubated for 16-18 hours at 35 °C+/-2 °C (ambient air), along with quality control strain *S. aureus* ATCC 25923. Flattening of the zone of inhibition near the erythromycin disc (D-zone), indicated the presence of inducible clindamycin resistance (iMLSB) (Figure 3). Clindamycin resistance was also indicated by hazy growth within the zone of inhibition around the antibiotic, even in the absence of a distinct D-zone.[8]

Interpretation of Erythromycin resistant phenotypes

Isolates classified as MS phenotypes were those that were clindamycin sensitive (≥21 mm) and erythromycin resistant (≤13 mm). Both erythromycin (≤13 mm) and clindamycin (≤14 mm) resistant isolates were interpreted as constitutive MLSB (cMLSB) phenotypes. Inducible MLSB (iMLSB) phenotypes were identified in erythromycin-resistant and clindamycin-sensitive isolates (exhibiting a D-shaped inhibitory zone surrounding clindamycin that flattened towards erythromycin disc).[8]

Real-time PCR

Next, using the HELINI Antibiotic Resistance gene Real-time PCR kit, the *erm*A/*erm*C genes of *S. aureus* isolates showing iMLSB resistance were analysed. All procedures were performed in accordance with universal safety precautions in a biosafety cabinet (level 2). Using the HELINI Purefast® Bacterial DNA mini spin prep kit, genomic DNA was recovered from *S. aureus* isolates. Until it was utilized for PCR, the isolated DNA was kept at -20 °C. Helini Biomolecules designed the primer sequences that were used for the amplification of the *erm*A and *erm*C genes for identification (Table 1). The *erm*A/C primer and probe mix consisted of forward and reverse primers and the TaqMan probe which was

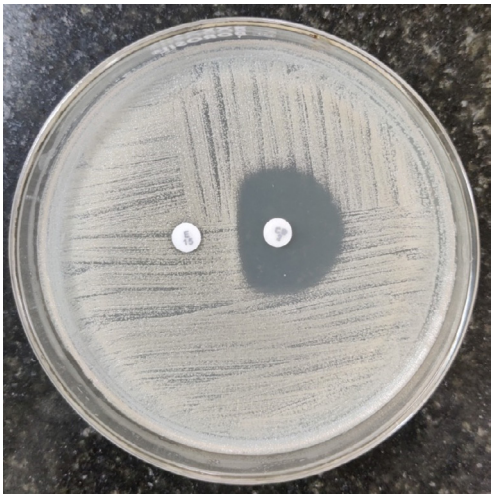

Figure 3. D zone (Inducible Clindamycin resistance)

Table 1. *erm*A and *erm*C primers and probes

	Primer	Probe
*erm*A	5'-TCAGGAAAAGGACATTTTACC-3'	5'-GAGCTTTGGGTTTACTATTAATGG-3'
*erm*C	5'-CTTGTTGATCACGATAATTTCC-3'	5'-CATAAGTACGGATATAATACGCA-3'

Table 2. *erm* gene detection mix for samples

Components	*erm*A	*erm*C
Probe PCR Master mix	10 µl	10 µl
*erm*A primer probe mix	2.5 µl	-
IC primer probe mix	2.5 µl	-
*erm*C primer probe mix	-	2.5 µl
PCR grade water	-	2.5 µl
Purified DNA	10 µl	10 µl
Final reaction volume	25 µl	25 µl

Table 3. Qualitative interpretation of results

Test sample	Negative control	Internal control	Positive control	Interpretation
Positive	Negative	Positive	Positive	Positive (*erm*A/C specific DNA detected)
Negative	Negative	Positive	Positive	Negative (No *erm*A/C specific DNA detected). Undetectable amounts of *erm*A/C specific DNA in a sample
Negative	Negative	Negative	Negative	Repeat (Experiment fail)
Positive	Positive	Positive	Positive	Repeat (Experiment fail)

fluorescently labelled with fluorescein amidite (FAM) (Table 2).

After addition of all the reagents, the PCR tubes were centrifuged to remove any air bubbles that could interfere with fluorescence detection, following which they were placed in the thermocycler (QuantStudio™ 5 Real-Time PCR System) and the PCR run was started. The procedure comprised an initial activation of the Taq enzyme at 95°C for 15 minutes, followed by 40 cycles of denaturation, annealing, and extension (20 seconds at 95 °C, 20 seconds at 60 °C, and 20 seconds at 72 °C, respectively). The qualitative interpretation of results were done as per manufacturer's instructions (Table 3 and Figures 4, 5 and 6).

RESULTS

In a total of 341 (100%) *S. aureus* strains isolated from various clinical samples, 267 (78.6%) were MSSA and 74 (21.4%) were MRSA (Figure 7).

Of the 341 total *S. aureus* isolates, 203 (59.5%) were erythromycin-resistant. The D-test

Table 4. iMLSB, cMLSB and MS phenotypes in isolates of *S. aureus* resistant to erythromycin

Pattern of resistance	Frequency	Percentage
Inducible clindamycin Resistance (D-test positive)	42	12.3
Constitutive clindamycin resistance	73	21.4
MS Phenotype	88	25.8

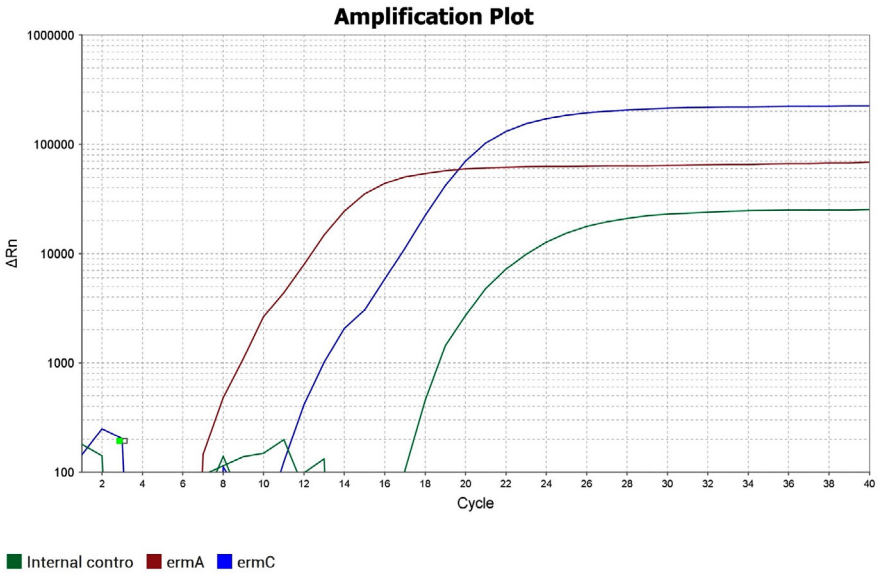

Figure 4. Amplification plots *erm*A and *erm*C

was performed for erythromycin-resistant and clindamycin-sensitive isolates. A total of 42 (12.3%) isolates tested positive for iMLSB resistance by D-test (Table 4). Out of the 42 inducible clindamycin resistant strains (D-test positive), 23 (15.6%) were MSSA, and 19 (33.9%) were MRSA. P-value was calculated by chi-square test with df -1, the p-value was 0.004 and therefore considered statistically significant (Table 5).

Real-time PCR was used to detect the *erm* gene in all staphylococcal D-test positive iMLSB phenotypes, and majority of the isolates [22 (52.4%)], had both *erm*A and *erm*C genes (Table 6).

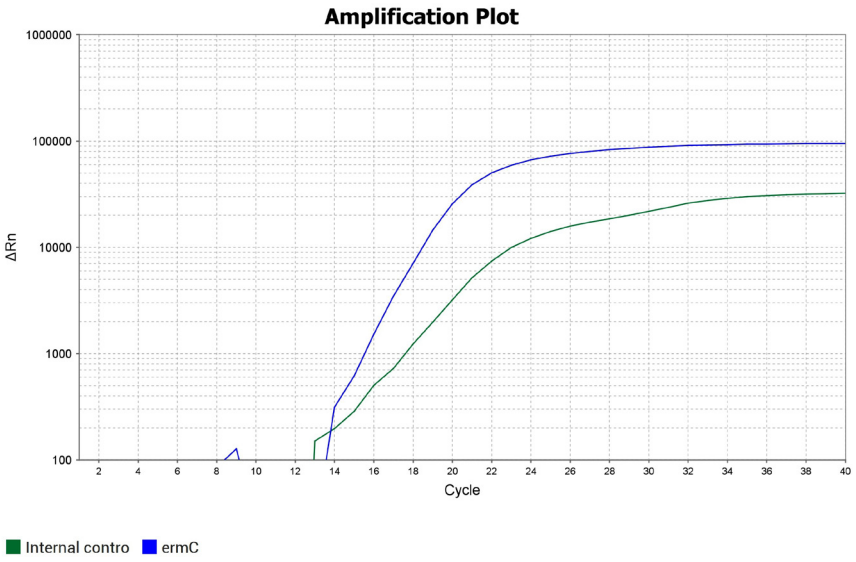

Figure 5. Amplification plot *erm*C

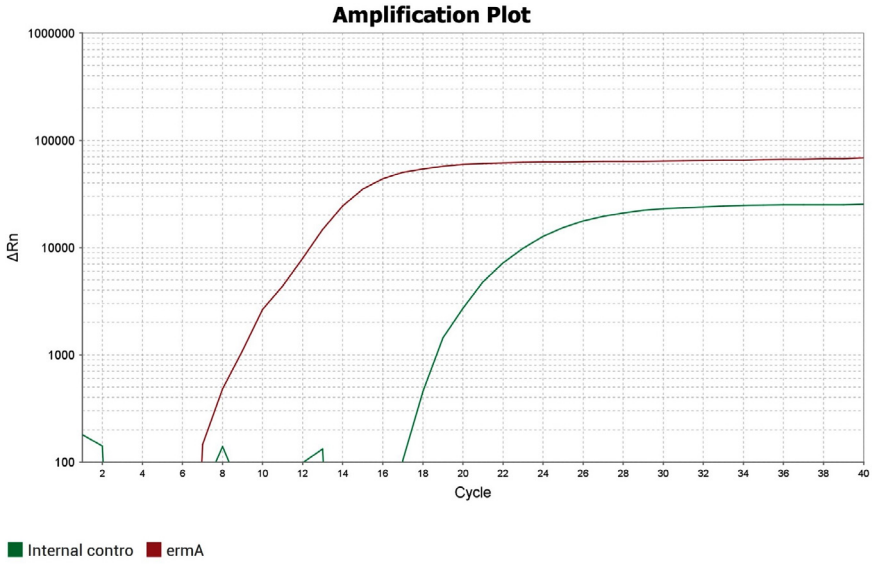

Figure 6. Amplification plot *erm*A

DISCUSSION

Despite the availability of multiple potent anti-staphylococcal drugs, *S. aureus*, has proved its adaptability by being a major cause of illness and death. In hospitals as well as communities, this pluripotent pathogen induces disease by toxin- and non-toxin-mediated routes, ranging from catastrophic systemic infections to comparatively mild infections of the skin and soft tissues.[9] MRSA prevalence has become progressively global,[7] due to which therapy has become problematic due to their multidrug-resistant nature. As a result, prevention of infection by staphylococci is more vital than ever.[10] Thus, specialized surveillance systems are required to track and stop the spread of MRSA infections within the community.[11]

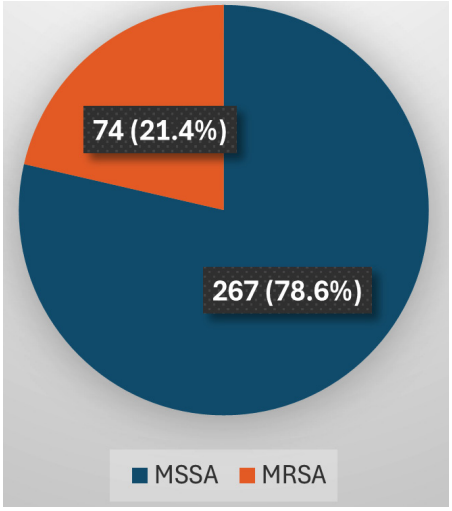

Figure 7. Distribution of *S. aureus* based on methicillin sensitivity

Table 5. Distribution of iMLSB among MRSA and MSSA resistant to erythromycin

Inducible Clindamycin Resistance (D-test positive)	Methicillin sensitivity		Total	Chi-square value	p-value
	MSSA	MRSA			
Present	23 (15.6%)	19 (33.9%)	42 (20.7%)	8.260	0.004
Absent	124 (84.4%)	37 (65.1%)	161 (79.3%)		
Total	147 (100%)	56 (100%)	203 (100%)		

Table 6. Distribution of *erm* genes among MSSA and MRSA strains showing iMLSB phenotype

PCR gene	Methicillin sensitivity		Total	Chi-square value	p-value
	MSSA	MRSA			
*erm*A gene	3 (13.0%)	2 (10.5%)	5 (11.9%)	3.268	0.352
*erm*C gene	5 (21.7%)	8 (42.1%)	13 (31.0%)		
Both *erm*A and *erm*C gene	13 (56.5%)	9 (47.4%)	22 (52.4%)		
Others	2 (8.7%)	0 (0.0%)	2 (4.8%)		
Total	23 (100%)	19 (100%)	42 (100%)		

Table 7. *S. aureus* MLSB phenotypes compared with those from other studies

Phenotypes	Fiebelkorn K.R. et al.[2] Texas, 2003 (n = 114)	Gadepalli R. et al.[14] India, 2006 (n = 200)	Yilmaz G. et al.[1] Turkey, 2007 (n = 883)	Gupta V. et al.[16] Chandigarh, India, 2009 (n = 200)	N. Pal et al.[17] Jaipur, India, 2010 (n = 851)	Mokta et al.[18] Shimla, India 2015, (n = 350)	Present study (n = 341)
iMLSB	34 (29.8%)	42 (21%)	175 (19.81%)	36 (18%)	101 (11.8%)	48 (13.71%)	42 (12.3%)
cMLSB	39 (34.2%)	53 (26.5%)	224 (25.36%)	38 (19%)	202 (23.7%)	60 (17.14%)	73 (21.4%)
MS phenotype	41 (35.9%)	24 (12%)	39 (4.41%)	64 (32%)	127 (14.9%)	29 (8.28%)	88 (25.8%)

Table 8. Comparing the MLSB phenotypes of MRSA and MSSA with those from other studies

Author's name	MSSA			MRSA		
	iMLSB	cMLSB	MS Phenotype	iMLSB	cMLSB	MS Phenotype
Yilmaz et al.[15] Turkey, 2007 (n = 883)	62 (14.8%)	19 (4.5%)	15 (3.6%)	113 (24.4%)	205 (44.2%)	24 (5.2%)
Gupta et al.[16] Chandigarh, India, 2009 (n = 200)	26 (17.3%)	15 (10%)	56 (37.3%)	10 (20%)	23 (46%)	8 (16%)
Deotale et al.[1] Wardha, India, 2010 (n = 247)	2 (1.6%)	0 (0%)	5 (4.0%)	34 (27.6%)	9 (7.3%)	30 (24.3%)
Debdas et al.[19] Assam,India, 2011 (n = 379)	5 (2%)	8 (3%)	45 (16%)	18 (18%)	23 (23%)	47 (48%)
Mittal et al.[20] Lucknow, India, 2012 (n = 260)	13 (8.4%)	7 (4.5%)	25 (16.1%)	47 (44.8%)	9 (8.6%)	14 (13.3%)
Sasirekha et al.[21] Bangalore, India, 2013 (n = 153)	13 (8.49%)	12 (7.84%)	20 (13.07%)	1 (0.65%)	8 (5.22%)	9 (5.88%)
Lall et al.[22] Delhi, India, 2014 (n = 305)	10 (6%)	8 (4.8%)	52 (31.5%)	52 (37.1%)	23 (16.6%)	32 (22.8%)
Mokta et al.[18] Shimla,India, 2015 (n = 350)	25 (9.32%)	36 (13.43%)	18 (6.71%)	23 (28.04%)	24 (29.26%)	11 (13.41%)
Chatterjee et al.[23] India, 2018 (n = 197)	16 (12%)	7 (6%)	0	18 (22.5%)	36 (45%)	2 (2.5%)
Present study (n = 341)	23 (15.6%)	55 (37.4%)	69 (46.9%)	19 (33.9%)	18 (32.1%)	19 (33.9%)

Clindamycin has strong *in vitro* and *in vivo* activity, which makes it an excellent alternative for treating MRSA infections,[12] however, because of an inducible mechanism of resistance, it becomes resistant in the presence of erythromycin due to cross-resistance among members of the MLSB families. Inducible resistance to clindamycin hampers its effectiveness. To demonstrate such inducible resistance (iMLSB phenotype), the D-test or double disc diffusion agar inhibitory assay is employed.[13]

In the present study, a total of 42 (12.3%) isolates were tested positive for iMLSB resistance by D-test. Our observation regarding iMLSB was in concordance with the report by Pal et al. and Mokta et al. Some investigators reported a higher incidence of iMLSB resistance, compared to our study (Table 7). The incidence of iMLSB was higher among MRSA [19(33.9%)] in our study, which was in concordance with all the other studies. Sasirekha et al. reported a lower incidence of iMLSB among MRSA strains (Table 8).

In this study, real-time PCR was done on 42 iMLSB positive isolates and majority-22 isolates (52.4%) had both *erm*A and *erm*C genes, followed by 13 (31%) isolates that showed only the *erm*C gene and 5 (11.9%) isolates that showed only the *erm*A gene. Among 22 isolates that showed both *erm*A and *erm*C genes, 13 (56.5%) were MSSA, and 9 (47.4%) were MRSA. Out of the 13 isolates that showed only the *erm*C gene, 5 (21.7%) were MSSA, and 8 (42.1%) were MRSA. Out of the 5 isolates that showed only the *erm*A gene, 3 (13%) were MSSA, and 2 (10.5%) were MRSA. In 2 MSSA isolates, neither the *erm*A nor the *erm*C genes could be detected, and these isolates could posses the *erm*B or *msr*A gene.

In a study done by Fiebelkorn et al., in Texas, genetic analysis of 19 samples of *S. aureus* with iMLSB revealed the *erm*A gene in 18 samples, while the remaining one contained the *erm*C gene.[3] Another study by Matthew et al., in Australia reported that among 28 iMLSB isolates genotyped, 8 had *erm*A and the rest had *erm*C.[6] Both these studies showed that the predominant genes were *erm*A and *erm*C and this was in concordance with our study.

Because of the evolution of antimicrobial resistance, precise drug susceptibility data is the need of the hour for appropriate therapeutic decisions. The results of this study indicate that all clinical laboratories should routinely subject staphylococcal isolates displaying both clindamycin-susceptible and erythromycin-resistant patterns to the D-test, which measures inducible clindamycin resistance.

In our study, we concluded that the D-test can be employed as an alternate method to PCR in laboratories with resource-limited settings. This test may aid in avoiding therapeutic failure by providing confident laboratory reports leading to the omission of clindamycin in patients infected with inducible clindamycin-resistant staphylococci. The D-test is essential for accurate discrimination between iMLSB resistance and true susceptibility. As long as inducible MLSB resistance is regularly and consistently identified, clindamycin can be utilized to treat individuals infected with true clindamycin-sensitive bacteria in a safe and effective manner.[17]

Periodic surveys are essential and must be performed to track changes in the prevalence of iMLSB strains and the evolution of strains with diverse patterns of susceptibility. This will help us to restrict the spread of these strains and formulate an efficient policy for antimicrobial usage control.

CONCLUSION

In a total of 341 (100%) *S. aureus* strains isolated from various clinical samples like pus, urine, blood, and sputum, majority [267 (78.6%)] were MSSA. iMLSB resistance was detected by disc diffusion induction test or "D-test" in 42 (12.3%) isolates, out of which 23 (15.6%) were MSSA and 19 (33.9%) were MRSA. Inducible resistance was found to be significantly higher among MRSA when compared to MSSA.

Real-time PCR was done for gene detection in all iMLSB positive isolates and the majority of the isolates - 22 (52.4%) showed both *erm*A and *erm*C genes, out of which 13 (56.5%) were MSSA and 9 (47.4%) were MRSA.

In this study, we describe a simple and inexpensive double-disc diffusion test (D-test) that can be incorporated into routine antibiotic susceptibility testing in clinical laboratories for all erythromycin-resistant staphylococcal isolates. This test enables accurate differentiation between inducible MLSB resistance and true clindamycin susceptibility, thereby guiding clinicians in avoiding clindamycin use in cases with inducible resistance and preventing potential therapeutic failures. While the D-test remains a practical and cost-effective tool for routine use, real-time PCR offers significant advantages for the detection of *erm* genes, including higher sensitivity, specificity, and the ability to provide rapid, precise molecular identification of resistance mechanisms. The integration of both phenotypic and genotypic methods can enhance diagnostic accuracy and support more informed antimicrobial stewardship.

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None.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTION

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

FUNDING

None.

DATA AVAILABILITY

All datasets generated or analyzed during this study are included in the manuscript.

ETHICS STATEMENT

This study was approved by the Institutional Ethics Committee, Sree Gokulam Medical College & Research Foundation, Kerala, India, vide reference number SGMC-IEC-No.: 34/463/09/2019.

INFORMED CONSENT

Written informed consent was obtained from the participants before enrolling in the study.

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