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Original Research Article

Inducible Clindamycin Resistance Among *Staphylococcal* isolates at A Tertiary Care Centre, Gujarat

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Abstract:

Introduction: The increasing incidence of a variety of infections due to *Staphylococcus aureus* and other species of genus *Staphylococcus*, especially, its expanding role of community-associated methicillin -resistant *S. aureus* (MRSA)—has led to emphasis on the need for safe and effective agents to treat both systemic and localized staphylococcal infections. Clindamycin has been used successfully to treat variety of infections due to MRSA in adults and children. There is a mechanism of macrolide resistance in *Staphylococcus* spp. which also affects the lincosamide and type B streptogramin characterizing the so-called Macrolide-Lincosamide- type B Streptogramin ($MLS_{B)}$ resistance, whose expression can be constitutive ($cMLS_B$) or inducible ($iMLS_B$) and is encoded mainly by ermA and ermC genes. The $cMLS_B$ resistance is easily detected by susceptibility testing used in the laboratory routine, but $iMLS_B$ resistance is not. Simple laboratory testing (the erythromycin-clindamycin "D-zone" test) can separate strains that have the genetic potential (i.e., the presence of erm genes) to become resistant during therapy from strains that are fully susceptible to clindamycin.

Aim: The study was planned to detect prevalence of $iMLS_{B,}$ cMLSB and MS phenotype resistance of clinical isolates of *Staphylococcus aureus* and Coagulase Negative *Staphylococci* (CONS) at tertiary care hospital, Gujarat

Material and Methods: This cross-sectional study was conducted on 105 isolates of *Staphylococci* from 1stMay 2021to 31st December 2021 at Microbiology section of Central Diagnostic Laboratory using Vitek 2 compact and manual disk diffusion method on Muller Hinton Agar. D test was performed on all the isolates of *Staphylococci.*

Results: Out of 105 tested isolates, 19.05% were D test positive (iMLSB), 9.52% were constitutive phenotypes (cMLSB), 26.66% were D test negative (MS phenotype).The prevalence rate of both (iMLSB) and (cMLSB) was higher in Methicillin resistant isolates compared to Methicillin sensitive isolates.

Conclusion: This study revealed recent magnitude of inducible clindamycin phenotype which could be easily missed while performing Kirby–Bauer disk diffusion method.So, it is recommended for clinical microbiology laboratory to routinely perform D-test in all clinically isolated *Staphylococci to* prevent treatment failure.

Keywords: methicillin resistant Staphylococcus aureus, methicillin-sensitive Staphylococcus aureus Inducible clindamycin resistance, Constitutive clindamycin resistance, D test, Macrolide-Lincosamide- type B Streptogramin ($MLS_{B)}$ resistance.

Introduction

One of the most prevalent no socomial and community-acquired infections, Staphylococcus aureus, has recently emerged as an issue that is only becoming worse due to its growing drug resistance. [1] Erythromycin (a macrolide) and clindamycin (a lincosamide) are widely used in treatment of *Staphylococcus aureus* infections. MRSA-related pneumonia, soft-tissue infections, and musculoskeletal infections in both adults and children have been successfully treated with clindamycin. [2] Clindamycin represents an attractive option for

several reasons. Firstly, good oral absorption of clindamycin makes it suitable for outpatient therapy or as follow-up after intravenous therapy. Secondly, it has high tissue penetrations (except for the central nervous system) and accumulation in abscesses and no need for renal dosing adjustments. Thirdly, clindamycin can be used as an alternative antibiotic in patients allergic to penicillin. Fourthly, community-acquired methicillin-resistant *S.*

aureus, which has rapidly emerged in recent years as a cause of skin and soft-tissue infections, has shown susceptibility to clindamycin. Finally, it has been shown that clindamycin inhibits the production of toxins and virulence factors in gram-positive organism through inhibition of protein synthesis. [3] Widespread use of macrolide-lincosamide streptogramin (MLS) antibiotic, has led to an increase in a number of *Staphylococci* acquiring cross-resistance to MLS antibiotic. This cross resistance to MLS antibiotic (MLS resistance) in *Staphylococci*, is generally attributable to one of two mechanisms.

First is an active efflux, due to energy dependent pump, which expels antimicrobial agents from the bacterial cell.

Efflux mechanism is encoded by msr (A) gene and confers resistance to macrolides and type B streptogramin, but clindamycin remains active (MS phenotype). Second mechanism is modification of drug- binding site on the bacterial ribosome, mediated by ribosomal methylases, which leads to the reduced binding of MLS antibiotics. Ribosomal methylases are encoded by erm genes [erm(A) or erm(C) in *Staphylococci*] and results in resistance to macrolides, lincosamide and type B streptogramin (MLSB resistance). Phenotypic expression of this resistance can be constitutive (cMLSB resistance phenotype) and inducible (iMLSB resistance phenotype).

In vitro, *staphylococcal* isolates with cMLSB phenotypes are resistant to all MLSB antibiotics, whereas those with iMLSB phenotypes demonstrate resistance to macrolides, while appearing susceptible to lincosamide and type B streptogramin.[4,12] There are reports of clinical failures of clindamycin in treating patients with iMLSB resistance phenotype, attributed to selection for a mutation in the macrolide responsive promoter region upstream of the erm gene and emergence of cMLSB resistance isolates, leading some investigators to recommend that clindamycin therapy be avoided for *S. aureus* isolates that display the iMLSB resistance phenotype.

On the other hand, labelling all erythromycin resistant *S. aureus* as clindamycin resistant may prevent the use of clindamycin in cases where it would be effective therapy. [3] Because they appear in vitro to be erythromycin resistant and clindamycin sensitive when not placed next to each other, it was highly challenging to identify the inducible clindamycin resistance in the normal laboratory.

Clindamycin in vivo therapy in such circumstances may result in clinical therapeutic failure. In the event of a different mode of resistance mediated by the msrA genes, such as antibiotic efflux, staphylococcal isolates show erythromycin resistance and clindamycin sensitivity both in vivo and in vitro, and the strain normally does not develop clindamycin resistance throughout treatment. Determining inducible clindamycin resistance phenotypes in vitro was crucial to preventing clinical treatment failure in the resistance instance caused by the erm gene. [1]

For detection of iMLS-resistant strains, Clinical and Laboratory Standards Institute (CLSI) has developed a phenotypic method called the double disk diffusion test (D-test). [5, 6] There was a significant regional variance in the prevalence of inducible clindamycin resistance.

There aren't many studies on the prevalence of S. aureus with inducible clindamycin resistance in Gujarat. [1]

Studies carried out in two Brazilian states with clinical isolates *Staphylococcus spp*. reported the cMLSB phenotype as the most frequent. Coutinho et al. have also evaluated the occurrence of the erm genes among the isolates analyzed.[7]

Prevalence of Inducible clindamycin resistance varies from 3.5% to 45% in *Staphylococcal spp*. in various studies.[8] However, the frequency of cMLSB and iMLSB resistance varies among different hospitals and there are other resistance mechanisms that confer resistance to only one or two classes of the MLSB complex.[7]

Increasing frequency of methicillin resistant *Staphylococcus aureus* (MRSA) infections and Because of its superior pharmacokinetic qualities, Clindamycin is the preferred medication for treating such infections as a result of evolving patterns in antimicrobial resistance..[9]

Objectives

Primary objective: Determine the prevalence of inducible clindamycin resistance using phenotypic (D-test) as well as Vitek 2 compact automated methods in clinical isolate of *Staphylococcus aureus* and *Coagulase Negative Staphylococci (CONS)* at Shree Krishna Hospital, Karamsad.

Secondary objectives:

- To determine association between methicillin (oxacillin) resistance and inducible clindamycin resistance.
- To compare the manual method (D test) with automated system (Vitek 2 Compact) to determine methicillin (oxacillin) resistance and inducible clindamycin resistance.

Material and Method

This descriptive cross-sectional study was conducted at Microbiology section of Central Diagnostic Laboratory, Shree Krishna Hospital,

Karamsad from July 2021-January 2022 after duly approved by Institutional Ethics Committee (IEC).

Clinical specimens of all the indoor and outdoor patients of all age groups received for culture and antimicrobial susceptibility testing (AST) at Shree Krishna Hospital, Karamsad Microbiology laboratory were processed according to laboratory standard operative procedure for bacterial isolate isolation and identification.

Samples from which *Staphylococcus aureus* and *Coagulase-Negative Staphylococci (CONS)* have been isolated were included in the study. The same isolate that was obtained in duplicate from the single patient from the same site of infection, were considered as a single isolate. No specific exclusion criteria were there. All samples underwent inoculation on Nutrient, Sheep Blood, and MacConkey agar before being incubated aerobically for 24 hours at 37 °C.

The initial method for identifying Staphylococcal species was colony morphology on 5 percent sheep blood agar. Using Gram stain, catalase test, and coagulase test by conventional microbiological methods. Cream to golden yellow colonies with or without haemolysis were selected for further identification and antimicrobial susceptibility Testing

(AST) [3].

Identification and Antimicrobial susceptibility Testing (AST) of all the isolates was performed as per the standard protocols using Vitek 2 Compact automated system (Biomerieux, France) and manual methods as per the standard protocols in CLSI.

All *Staphylococcus* species isolates were further classified as methicillin sensitive or methicillin resistant based on their Oxacillin susceptibility and Inducible clindamycin resistance positive or negative (IDC) as determined by the Vitek 2 Compact automated system.

1. **Detection of Methicillin resistance (Oxacillin Resistance) in *Staphylococcus species* by manual method** [4,5]:

All *Staphylococcal* species were tested for methicillin resistance by using Cefoxitin disc (30µg) by disc diffusion method (table 1). All *Staphylococcal* species were tested for D test as per CLSI guidelines to detect inducible clindamycin resistance (table 2). [11,12]

Table 1: Manual method of detecting Methicillin (Oxacillin) Resistance in *Staphylococcus* species. [4,5]

Test	Detecting mecA-Mediated Resistance using cefoxitin
Test method	Disk diffusion
Medium	Muller Hinton agar
Antimicrobial concentration	30µg Cefoxitin disk
Incubation condition	33 to 35 °C; ambient air
Incubation Length	16-18 hours
QC recommendation routine	S. aureus ATCC 25923-mecA negative (zone 23-29mm)

Interpretation:

- Isolates with cefoxitin zone size ≥22 mm = negative for mecA-mediated resistance (Oxacillin sensitive)
- Isolates with cefoxitin zone size ≤21 mm =positive for mecA – mediated resistance
- (Oxacillin resistant)

Table 2: Detection of Inducible Clindamycin Resistance (ICR) in *Staphylococcus species*(4,5)

Test Method	Disk Diffusion (D-Zone test)
Organism group (applies only to organisms resistant to erythromycin and susceptible or intermediate to clindamycin)	All *Staphylococcus spp.*
Medium	Muller Hinton Agar or blood agar plate used with MIC tests
Antimicrobial concentration	5µg erythromycin and 2µg clindamycin disk spaced 15-26 mm apart
Incubation conditions	35C± 2°C; ambient air
Incubation Length	16-18 hours
QC recommendation routine	*S. aureus* ATCC 25923 for routine QC of erythromycin and clindamycin disks

Interpretation:

- Flattening of the D-zone (inducible clindamycin resistance): the zone of inhibition next to the erythromycin disc. (Image 1)
- Even if there is no D-zone visible, hazy development within the zone of inhibition.

around clindamycin indicates clindamycin resistance.

- Isolates resistant to both erythromycin and clindamycin was considered as Constitutive resistance phenotypes (cMLS$_B$).
- Isolates resistant to erythromycin and susceptible to clindamycin (Dtestnegative) was considered as MS phenotype.
- For quality control, S. aureus ATCC 25923, S. aureus ATCC 43300, S. aureus ATCC BAA-976, and S. aureus ATCC BAA-977 were employed.

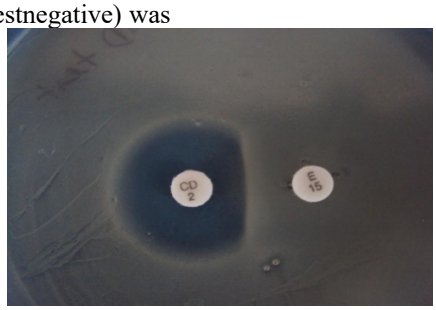
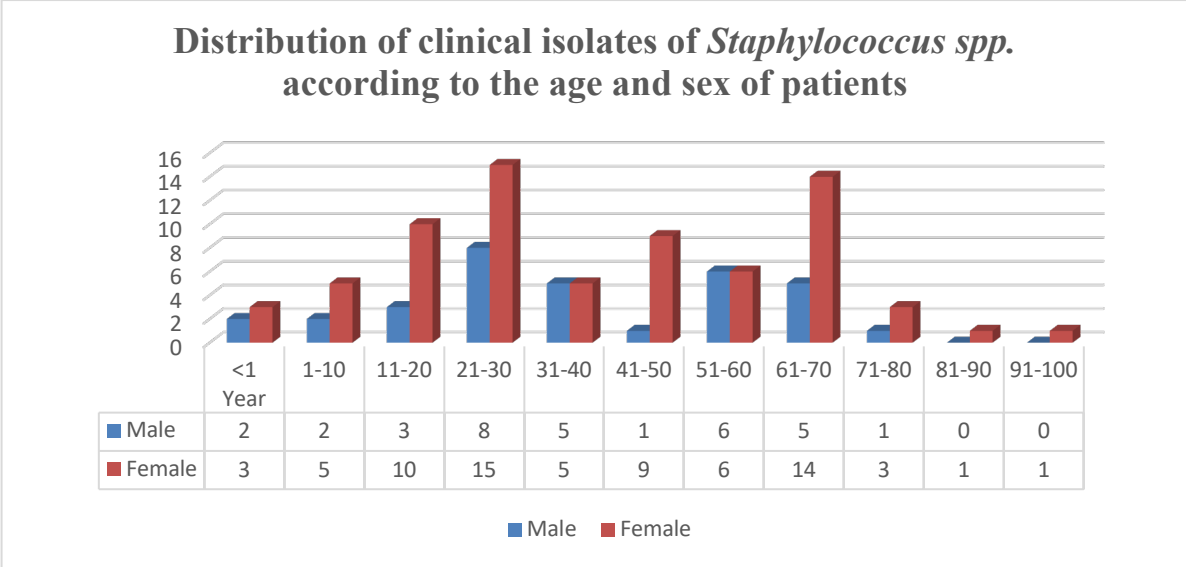


Figure 1: D-zone test

Results:

During the study period of July 2021-January 2022, a total of 105 isolates of *Staphylococcal* species identified at Microbiology laboratory were included in the study. Both *Staphylococcus aureus* and *Coagulase-Negative Staphylococci (CONS)* isolated from various clinical sample included in study as shown in table 4. Age & Sex wise distribution of 105 isolates is shown in Figure 2.

Figure 2: Distribution of clinical isolates of *Staphylococcus spp.* according to the age and sex of patients. (N=105)



Maximum 23 (22%) *Staphylococcus spp.* were isolated from 21-30 years of age group followed by 19(18%) from 61 to 70 years and 13(12%) from 11-20 years. Isolation of Staphylococcus species was more in female than male in almost each age group.

Table 3: Distribution of clinical isolates of *Staphylococcus spp.* according to the Specimen (N=105)

Specimen	*S. aureus* (%)	*CONs* (%)	Total (%)
Pleural fluid	2(1.90%)	0(0)	2(1.90%)
Tissue	3(2.85%)	0(0)	3(2.85%)
Endotracheal secretion	5(4.76%)	0(0)	5(4.76%)
Sputum	1(0.95%)	0(0)	1(0.95%)
Tracheostomy Secretion	1(0.95%)	0(0)	1(0.95%)
Pus	41(39%)	0(0)	41(39%)
Nasal swab	1(0.95%)	0(0)	1(0.95%)
Swab	6(5.71%)	1(0.95%)	7(6.66%)
Blood	18(17.14%)	19(18.09%)	37(35%)

Urine non catheterized	6(5.71%)	0(0)	6(5.71%)
Pericardial fluid	1(0.95%)	0(0)	1(0.95%)
Total	85(80.95%)	20(19.04%)	105(100)

As shown in table 4, a total of 85 (81%) *S. aureus* & 20 (19%) *CONs* were isolated. *Staphylococcus* species was predominantly isolated from Pus 41 (39%) followed by nasal swab 19 (18.09%) Blood sample 37 (35%), Swab (6.66%), Urine non catheterized 6 (5.71%) and Pleural fluid (2%).

Table 4: Distribution of the MS, iMLS$_B$ and cMLS$_B$ resistance phenotypes among *S. aureus*and *CONS* isolates. (n= 105)

	***S. aureus* (%)**	***CONS* (%)**	**Total (%)**	**p value**
MS	21(20%)	7(6.66%)	28 (26.66%)	0.423
iMLS$_B$	17(16.19%)	3(2.85%)	20 (19.05%)	0.758
cMLS$_B$	4 (3.80%)	6(5.71%)	10 (9.52%)	0.003
Total	42 (40%)	16(15.23%)	58 (55.24%)	

As shown in table 5, in 58(55.24%) isolates of *Staphylococcus* species, different resistance phenotypes were identified and no phenotypic resistancewas observed *in* 47 (44.76%) isolates of *Staphylococcus* species. A total of 28 (26.66%) isolates were identified as MS phenotype &10 (9.52%) isolate were identified as cMLSB phenotype and 20 (19.05%) isolates showed iMLSB phenotype. In *Staphylococcus aureus*, iMLSB was seen in 17 (16.19%) isolates, cMLSB seen in 4 (3.80%) isolates and the MS phenotype was seen in 21 (20%) isolates. In *CoNS* isolates., iMLSB was seen in 3(2.85%) isolates, cMLSB seen in6 (5.71%) and the MS phenotype was seen in 7(6.66%) isolates. Distribution of MS phenotype and iMLSB phenotype among *S. aureus*& CONs was statistically not significant but statistically significant difference observed in cMLSB phenotype among *S. Aureus* & CONs. Coagulase negative staphylococcus showing more constitutive resistance phenotype compared to *S. aureus.*Also, resistance phenotypes distribution is shown among methicillin resistant and methicillin sensitive *Staphylococcal* isolates in table 6

Table 5: Distribution of the MS, iMLS$_B$ and cMLS$_B$ resistance phenotypes among Methicillin Resistant (MRS) & Methicillin susceptible (MSS) *Staphylococcal* isolates (n=105)

	MRS			**MSS**		
	***S. aureus* (%)**	***CONS* (%)**	**Total (%)**	***S. aureus* (%)**	***CONS* (%)**	**Total (%)**
MS	10(9.52%)	6(5.71%)	16(15.23%)	11 (10.47%)	1(0.95%)	12 (11.42%)
iMLS$_B$	17(16.19%)	2(1.90%)	19 (18.09%)	0 (0)	1(0.95%)	1(0.95%)
cMLS$_B$	4 (3.80%)	6(5.71%)	10 (9.52%)	0 (0)	0 (0)	0
Total	31(29.52%)	14(13.33%)	45(42.85%)	11 (10.47%)	2(1.90%)	12 (11.42%)

A total of 45 (42.85%) of Methicillin resistant *Staphylococci*& 12 (11.42%) Methicillin sensitive *Staphylococci* were identified in present study. Comparison done for MRS and Inducible clindamycin resistance detection between manual and VITEK method as shown in table 7. Out of 45 MRS, 19 iMLSB and out of 12 MSS only 1 iMLSB phenotype isolated that shows tendency of MRS isolate develop inducible resistance to Clindamycin. Similar finding were observed in study conducted by Amit Banik et al in his study where 15.38% MRS showing iMLSB phenotype and only 5.31% MSS showing inducible resistance to Clindamycin. [24]

Table 6: Comparison between Vitek & Manual Methods for Detection of MRS & ICR.

Resistance phenotype	**Vitek 2 Compact**	**Manual method on MHA**
Methicillin (oxacillin) Resistance	56	51
Inducible Clindamycin Resistance (ICR) iMLSB	20	20

A total 56 MRSA were positive as per Vitek 2 compact & 51 MRSA were positive by manual disc diffusion method on MHA. A total of five (5) MRS were positive in Vitek 2 compact and were negative in Manual method on MHA even after repeat manual testing. For ICR, Vitek 2 & manual methods gave similar results in isolates.

Discussion

Staphylococcus aureus infections are becoming more common, and in particular, the growing importance of community-associated, methicillin-resistant S. aureus (MRSA) has highlighted the need for safe, efficient treatments for both systemic and localised staphylococcal infections. Clindamycin has been used well to treat MRSA-related pneumonia, soft-tissue infections, and

musculoskeletal infections in both adults and children. There was a mechanism of macrolide resistance in *Staphylococcus* species which also affects the effectiveness of lincosamide and type B streptogramin characterizing the so-called Macrolide Lin

cosamides- type B Streptogramins (MLSB) resistance, whose expression can be constitutive (cMLSB) or inducible (iMLSB) and was encoded mainly by ermA and ermC genes. The cMLSB resistance was easily detected by susceptibility testing used in the laboratory routine, but iMLSB resistance was not detected. Simple laboratory tests can distinguish between bacteria that are completely susceptible to clindamycin and strains that have the genetic potential—i.e., the presence of erm genes—to develop resistance during therapy. One such test is the erythromycin clindamycin "D-zone" test. The present study was planned to characterize the phenotypic and molecular profiles of iMLSB resistance of clinical isolates of *Staphylococcus aureus* and *Coagulase Negative Staphylococci (CONS)* at Shree Krishna Hospital, Karamsad.We can say from this setup, if the prevalence of iMLSB is less/negative than it gives idea about the rate and reason than the rate of therapeutic success will be high for *Staphylococcal* infection.

In our present study, prevalence of iMLSB was19.05 % among 105 isolates of *Staphylococcus*species, which was further divided in Methicillin resistant (18.09%) and Methicillin sensitive phenotype (0.95%). As per shown in table 8, similar prevalence of iMLSB phenotype among MRS is found in study conducted by Amit et al (15.38%)and among MSS, iMLSB prevalence rate is 5.31% [20] Similar findings are seen in study conducted by Koppad et alwhere low prevalence of iMLSB was found among MSS (2.94%) compared to MRS (14.71%) [15].So, most of the studies reported the same finding of higher rate of inducible clindamycin resistance in MRS compared to MSS as shown in table 8. This finding suggests that empirical clindamycin treatment in MSS infections is effective but not due to infections with MRS especially where rate of iMLSB is high. D test must be done prior to prescribing clindamycin empirically in MRS infections. Higher prevalence rate of iMLSB phenotype among MRS as well as MSS was found in previous studies compared to present study as shown in table 8.

Constitutive clindamyc in resistance in present study was 9.52%. We found cMLSB in only methicillin resistant group not in methicillin sensitive phenotype. Compared to other studies prevalence rate of cMLSB was always higher in MRS compared MSS phenotype as shown in table 8. In our study,cMLSB phenotype is reported more in coagulase negative *Staphylococci*(5.71%)compared to *Staphylococcus aureus* (3.80%) and it is statistically significant. This may be due to antimicrobial profile of microbial population this area. Study

conducted by Koppad et al shows similar finding as our study where cMLSB prevalence rate was 8.8% in MRS phenotype [15]. Studies conducted by Lyall et al [14], Kumar et al [16], Seifi N et al [18], Subasini et al [21], Koppad et al [15] have also shown lower prevalence rate of cMLSB among MSS phenotype where we don't find any cMLSB isolate in MSS phenotype. Most studies conducted have shown higher prevalence of inducible clindamycin resistance compared to constitutive clindamycin resistance. This important finding signifies the need to incorporate this simple and reliable D test in to our routine practice to find out this hidden resistance phenotype.

Prevalence rate of *Staphylococcal* isolates having efflux pump mediated resistance mechanism to Macrolides - MS phenotype in our study is 26.66%. Further MS phenotype is more prevalent in MRS (15.23%) compared to MSS (11.42%). Study conducted by Seifi N et al [18] shows similar finding of MS phenotype in MRS (15.91%) and MSS (9.76%) isolates. This finding is useful as it clearly states that Clindamycin can be effectively used as treatment option in such group of patients after doing D test. While in some other studies conducted globally demonstrate more prevalence in MSS compared to MRS as shown in table 8.

The MRSA prevalence in various studies like Lyall,et al, reported 91 % MRSA , Kumar R et al. reported 70 %, Bidani et al. reported 33.3%, Timisina, Shrestha sing &Timalsina reported 26.6%, Adhikari et al. reported 25.1% in their study which is higher when compared to our result (42.85%). [1,13,14,16,17,19]. Prevalence rate of MRSA was observed in different countries of South Asia like Karachi 43% by Perwaiz S, Barakzi Q, Farooqi BJ et al, in, Nepal 38% and 40% by Tiwari HK, Das AK, Sapkota D et al, in and by Sanjana RK, Shah R, Chaudhary N et al, respectively which are lower when compared to the results. [14]Tremendous increase in the methicillin resistant isolates in the hospital was observed over the period of time. This difference might be because they used oxacillin disc diffusion method for detection of MRSA on the other hand, we used cefoxitin disc diffusion for the detection of MRSA. Out of 105 isolates, both Vitek 2 compact and D-test, manual method on Muller Hinton agar detected same number of isolates (20) of inducible clindamycin resistance. So, both the methods are reliable for detection of iMLSB.

In developing country like India D test can be implemented as a routine screening test for erythromycin resistant isolate to avoid treatment

failure. On the other hand, Vitek 2 compact identified 56 MRS phenotype compared to manual Kirby Bauer disc diffusion method identified 51 isolates of MRS. As previously mentioned, this difference might be because they used oxacillin disc diffusionmethod for detection of MRSA on the other hand, we used cefoxitin disc diffusion for the detection of MRSA. The Reason of this variation found in these resistance phenotypes rate in various studies is different bacteriological and their antimicrobial susceptibility profile, geographical population variation, difference in their genetic establishment, difference in antimicrobial prescription practice, variation in infection prevention practice. With emergence of various multidrug resistance among bacteriological profile of one geographic area over the period of time, the prevalence rate of these resistance phenotypes will also change, so periodic surveillance should be done to identify the current pattern of resistance phenotype to decide appropriate empiric therapy.

Table 8: Distribution of iMLSB, cMLSB and MS phenotypes in *Staphylococcus* species from various studies and comparison to present study

Author's name	MRS (%)			MSS (%)		
	iMLSB	cMLSB	MS phenotype	iMLSB	cMLSB	MS phenotype
Adhikari et al (1)	27.9	54.4	10.3	5.9	20.8	14.8
Lyall et al (14)	33.2	22.1	44.6	34.6	7.5	46
Koppad et al (15)	14.71	8.8	17.65	2.94	8.82	47.06
Kumar et al (16)	27	21	6	10	7	3
Seifi N et al (18)	20.45	52.30	15.91	4.88	7.32	9.76
Amit et al (20)	15.38	30	8.47	5.31	1.77	15.05
Subasini et al (21)	24.8	23.3	22.5	17.5	8.7	15
Kiran et al (22)	28.04	29.26	13.41	9.32	13.43	6.71
Present study	18.09	9.52	15.23	0.95	0	11.42

Conclusion

In this era of continues emergence of antimicrobial resistance due to uncontrolled use and prescription of antibiotics, treatment of *Staphylococcal* infections becomes tricky. As we found rising prevalence of inducible clindamycin resistance among MRSA isolates, Clindamycin cannot be started as empiric treatment without prior testing. Vitek 2 compact is definitely preferable method for detection of both methicillin resistance and inducibleclindamycin resistance than manual method. But in developing countries like India, D test can be considered good and preferable method to identify this hidden resistance phenotype to avoid treatment failure. Microbiologists must perform D test as chip, reliable and accurate method in routine practice especially in erythromycin resistant isolate. Clinicians must be aware of this fact before judiciously prescribing antibiotics. This will make clindamycin available as excellent alternative for staphylococcal infection for future generations. In our locality, as far as our knowledge no such study conducted for inducible clindamycin resistance recently. By simply doing D test routinely, clindamycin treatment failure can be prevented.

Limitation

Due to lack of availability of advanced molecular diagnostic facility, we were not able to perform molecular basis of resistance phenotypes.

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