

Inducible Clindamycin Resistance and Antibigram of Staphylococcus: A Hospital-Based Study**Subhashree Mohapatra¹, Naveen Kumar Medi², Nikunja Kumar Das³, Sudipti Sahu⁴, Satyaram Satapathy⁵, Nirupama Chayani⁶**¹Assistant Professor Department of Microbiology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha²Senior Consultant, Department of Nephrology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha³Professor, Department of Microbiology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha⁴Assistant Professor, Department of Microbiology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha⁵Assistant Professor, Department of Microbiology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha⁶Professor, Department of Microbiology, DRIEMS Institute of Health Sciences and Hospital, Tangi, Cuttack, Odisha

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Abstract

Introduction: Infections of the skin and soft tissues are now frequently caused by Staphylococcus. Methicillin-resistant Staphylococcus aureus (MRSA) and inducible clindamycin resistance (iMLSB) have emerged as significant challenges in the treatment of Staphylococcal infections, and medication resistance has grown. The purpose of this study was to identify MRSA and iMLSB and to determine the pattern of antibiotic susceptibility among the isolates.

Materials and Methods: 150 isolates of Staphylococcus were studied for detecting the antibiotic resistance pattern and also to detect MRSA using cefoxitin disc and oxacillin E test. iMLSB resistance among MRSA strains was detected using D test.

Results: Out of 150 isolates of Staphylococcus, 110 (73%) isolates were of Staphylococcus aureus and 40 (26%) isolates were of Coagulase-negative Staphylococci. Staphylococcus was most sensitive to vancomycin and linezolid, followed by clindamycin. Penicillin was the least sensitive antibiotic. 32 (21.3%) strains of Staphylococcus aureus were MRSA. Among them, 18 (56.2%) were erythromycin resistant, and 08 (44%) of erythromycin-resistant strains were found to be inducible clindamycin resistant.

Conclusion: Testing of all the isolates of Staphylococcus for antibiotic resistance and identifying the MRSA isolates along with iMLSB resistance is important in determining the antibiotic sensitivity, which will prevent treatment failure.

Keywords: Antibigram, Methicillin-Resistant Staphylococcus Aureus, Inducible Clindamycin Resistance.

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Introduction

Skin and soft tissue infections are now often caused by both Staphylococcus aureus and Coagulase-negative Staphylococcus (CoNS). Staphylococcus aureus infections are more common in people with diabetes, cancer, sepsis, tissue necrotizing pneumonia, eczema, vascular disorders, and lung diseases [1]. Previously thought to be pollutants, CoNS, which are typically found on the skin as commensal bacteria, are now recognized as important contributors to human illness. The two most frequently isolated CoNS from clinical

samples are Staphylococcus epidermidis and Staphylococcus saprophyticus [2]. Biofilms, PIA (polysaccharide intracellular adhesion), Bap (biofilm-associated protein), and toxins are the main factors behind the pathogenicity of Staphylococcus epidermidis [3]. The first antibiotic to treat Staphylococcus aureus infections was penicillin, which was initially made available in the early 1940s. However, plasmid outbreaks that propagated the β -lactamase gene throughout the whole Staphylococcus aureus species caused a

significant decrease in the potency of penicillin within ten years. Methicillin-resistant *Staphylococcus aureus* (MRSA) strains were discovered in clinical samples within a few years of the introduction of penicillinase-resistant β -lactam antibiotics (methicillin). This is because the *mecA* gene, which encodes a distinct penicillin-binding protein (PBP2A) with low affinity for β -lactam antibiotics, was acquired.

Epidemic clones of MRSA developed multidrug resistance by the 1980s and proliferated globally, becoming a major cause of hospital-acquired illnesses [4].

Due to the extensive usage of the macrolide-lincosamide-streptogramin B (MLSB) family of antibiotics following the emergence of MRSA, some strains of *Staphylococcus aureus* developed resistance to MLSB antibiotics. This was often caused by *erm* genes altering the target site [5]. The purpose of this study was to identify MRSA and inducible MLSB resistance in *Staphylococcus aureus*, as well as to ascertain the antibiotic sensitivity pattern of *Staphylococci*.

Materials and Methods

This study was conducted at the Department of Microbiology at a tertiary care hospital, Odisha and included 150 *Staphylococcus* isolates collected from various clinical samples such as aspirates, body fluids, urine, vaginal swabs, pus, and sputum. The isolates were identified as *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Staphylococcus saprophyticus* based on Gram stain, colony morphology, and biochemical tests.

Antibiotic susceptibility was tested using the standardized Kirby-Bauer disc diffusion method, following guidelines from the Clinical and Laboratory Standards Institute. The antibiotics tested included Amikacin (30 μ g/ml), gentamicin (10 μ g/ml), cotrimoxazole (1.25/23.75 μ g/ml), clindamycin (2 μ g/ml), linezolid (30 μ g/ml), ofloxacin (5 μ g/ml), erythromycin (15 μ g/ml), penicillin (10 units), and vancomycin (30 μ g/ml).

A cefoxitin (30 μ g) disc was used to determine methicillin resistance in all *Staphylococcus aureus* strains. Resistance was defined as an inhibitory zone of less than 21 mm, while susceptibility was indicated by a zone of 22 mm or more [6].

A Mueller-Hinton agar plate supplemented with 2% NaCl was used to measure the E-test oxacillin

strip's Minimum Inhibitory Concentration (MIC) of methicillin-resistant *Staphylococci*. Oxacillin E-test MICs of < 2 μ g/ml and $\geq$ 4 μ g/ml, respectively, were used to characterize methicillin susceptibility and resistance [7,8]. The double disc diffusion test (D-test) was used to identify MRSA strains exhibiting inducible MLSB resistance. A 0.5 McFarland suspension of *Staphylococcus aureus* and an erythromycin (15 μ g) disk were added to Mueller-Hinton agar, with the disks positioned 15 mm (edge to edge) apart from the clindamycin (2 μ g) disk. The plates were examined for D-shaped flattening of the area around the clindamycin disk after an overnight incubation at 37 °C. MRSA strains that tested positive in the D-test were considered to have inducible MLSB resistance. Strains resistant to both erythromycin and clindamycin were classified as constitutive MLSB resistant, while those resistant to erythromycin but susceptible to clindamycin were categorized as MS phenotype.

Results

Of the 150 *Staphylococcus* isolates, 40 (26%) were of CoNS and 110 (73%) were of *Staphylococcus aureus*. *Staphylococcus epidermidis* (55%) and *Staphylococcus saprophyticus* (45%), respectively, were the most prevalent CoNS. Vancomycin (100%) and Linezolid (100%) were the most effective antibiotics against *Staphylococcus aureus*, followed by clindamycin (81.1%), amikacin (68.3%), gentamicin (65.8%), erythromycin (58.9%), cotrimoxazole (51.2%), ofloxacin (35.8%), and penicillin (5.9%). Vancomycin (100%) and Linezolid (100%) were the most effective antibiotics, followed by clindamycin. The CoNS isolates showed no penicillin sensitivity. (Table 1) By using the cefoxitin disc diffusion test and the oxacillin E test, 32 (21.3%) strains of *Staphylococcus aureus* were shown to be methicillin-resistant. A MIC of 4 μ g/ml for oxacillin was often noted among the MRSA strains in the investigation. (Table 2) Vancomycin was the most effective treatment for MRSA strains, followed by clindamycin and linezolid. (Graph 1, Table 3) 18 (56.2%) of the 32 MRSA isolates exhibited erythromycin resistance. Five (27.0%) MRSA strains were constitutive MLSB, eight (44%) were inducible MLSB, and five (27.0%) were of the MS phenotype. (Table 4)

Table 1: Antibiotic sensitivity pattern of Staphylococcus

S.NO	Organisms	P (%)	AK (%)	G (%)	Co (%)	Cd (%)	Lz (%)	Of (%)	E (%)	Va (%)
1.	Staphylococcus aureus Total no.- 110	7 (6.3)	80 (72.7)	77 (70.0)	60 (54.5)	93 (84.5)	110 (100)	42 (38.1)	69 (62.7)	110 (100)
2.	Staphylococcus epidermidis Total no.- 22	0 (0)	14 (63.6)	13 (59.0)	14 (63.6)	14 (63.6)	22 (100)	10 (45.4)	10 (45.4)	22 (100)
3.	Staphylococcus saprophyticus Total no.- 18	0 (0)	10 (55.5)	09 (50.0)	08 (44.4)	18 (100)	18 (100)	08 (44.4)	09 (50.0)	18 (100)
Total = 150		7 (4.6)	104 (69.3)	99 (66.6)	82 (54.6)	125 (83.3)	140 (93.3)	60 (40)	88 (58.6)	150 (100)

Table 2: MIC of oxacillin for methicillin-resistant Staphylococcus aureus isolates

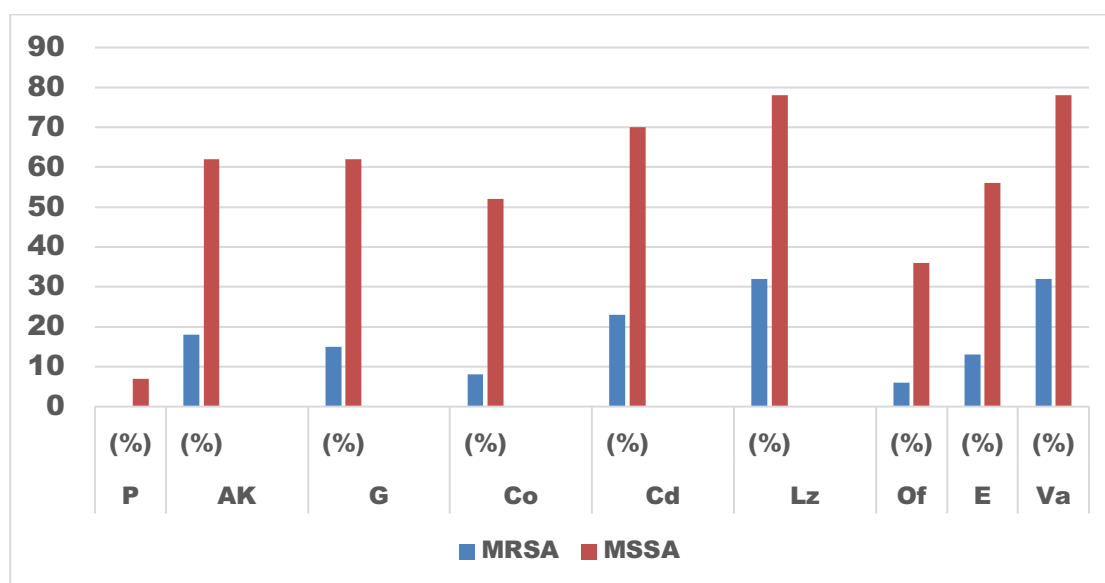
MIC value in µg/ml	No of isolates (%)	MIC value in µg/ml	No of isolates (%)
4		9	(28.1%)
6		8	(25.0%)
8		5	(17.2%)
192		2	(6.8%)
>256		8	(25.0%)
Total		32	

Table 3: Antibiotic sensitivity pattern of Staphylococcus aureus

S.No	MRSA/ MSSA	P (%)	AK (%)	G (%)	Co (%)	Cd (%)	Lz (%)	Of (%)	E (%)	Va (%)
1	MRSA Total no. 32	0 (0)	18 (56.2)	15 (46.8)	8 (25.0)	23 (71.8)	32 (100)	6 (18.7)	18 (56.2)	32 (100)
2.	MSSA Total no. 78	7 (8.9)	62 (79.4)	62 (79.4)	52 (66.6)	70 (89.7)	78 (100)	36 (46.1)	56 (71.7)	78 (100)
Total	110	7	80	77	60	93	110	42	69	110

Table 4: Distribution of inducible clindamycin resistance among MRSA isolates

S.NO	Susceptibility pattern (Phenotype)	MRSA isolates (%)
1.	Erythromycin – R, Clindamycin –R (constitutive MLSB)	05 (27%)
2.	Erythromycin – R, Clindamycin –S D test positive (inducible MLSB)	08 (44%)
3.	Erythromycin – R, Clindamycin –S D test negative (MS phenotype)	05 (27%)
Total		18

**Graph 1: Antibiotic sensitivity pattern of Staphylococcus aureus**

P-Penicillin, Ak-Amikacin, G-Gentamicin, Co-Cotrimoxazole, Cd-Clindamycin, Lz-Linezolid, Of-Ofloxacin, E-Erythromycin, Va-Vancomycin

Discussion

The Staphylococcal isolates were most susceptible to vancomycin (100%) and linezolid (100%), followed by clindamycin (83.3%). Only 4.6% of *Staphylococcus aureus* isolates were susceptible to penicillin, while 54.6% were susceptible to cotrimoxazole, and 58.6% to erythromycin. Research by Krithikaa et al. found that the antibiotics to which *Staphylococcus aureus* was most sensitive were linezolid (91.1%), vancomycin (80%), clindamycin (68.5%), erythromycin (51.5%), cotrimoxazole (46%), and penicillin (20%), in decreasing order. [9]. According to Sajjanar V et al., CoNS is completely susceptible to linezolid and vancomycin. The antibiotics erythromycin (33.3%) and clindamycin (50%) were the least effective against CoNS. More than 50% resistance to clindamycin and erythromycin has been observed in other studies [10,11]. MRSA was found in 32 (29.0%) of the *Staphylococcus aureus* isolates. An increased incidence of MRSA has been reported in certain studies. Giacometti et al. reported that 54.4% of isolates included MRSA [12]. In 2005, Jain A examined 97 *Staphylococcus aureus* isolates and discovered that 75.26 percent of them were resistant to methicillin [13].

The percentage of MRSA isolates that were resistant to clindamycin, erythromycin, and amikacin was 13.8%, 55.2%, and 38%, respectively. In a review done by Gebremariam et al, among the different studies included, there were more than 70% MRSA strains showing resistance to penicillin and erythromycin, but resistance to clindamycin and amikacin was less than 50% [14].

Five (27%) of the 18 (40.6%) erythromycin-resistant MRSAs had constitutive MLSB, eight (44%) had inducible clindamycin resistance, and the remaining five (27%) had an MS phenotype. In various other studies, Erythromycin resistance ranged from 50% to 59%, whereas inducible clindamycin resistance ranged from 33% to 42% [9,15,16]. A decreased rate of inducible clindamycin resistance has been reported in certain studies [17,18].

Conclusion

The rise in resistance to widely used antibiotics has made treating *Staphylococcus aureus* infections more difficult in recent years. Even the CoNS, which was previously considered a contaminant, has developed antibiotic resistance. Additionally, the development and ongoing increase of MRSA strains have raised serious concerns about how to manage infections.

The cefoxitin disc test can be routinely used to detect MRSA strains by the phenotypic method. Clindamycin's relative affordability, high

bioavailability, and superior tissue penetration have made it a key antibiotic for treating Staphylococcal infections. It is crucial to identify the *Staphylococcus aureus* strains that have evolved resistance to clindamycin yet appear susceptible in vitro.

Over 25% of the MRSA isolates in our study that seemed to be clindamycin-sensitive were found to be resistant by the D test. If clindamycin had been used to treat such infections, the treatment would have failed. Therefore, treatment of Staphylococcal infections would greatly benefit from accurate antibiotic sensitivity testing and the identification of MRSA and inducible clindamycin resistance by the D test.

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