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The Prevalence of Inducible Clindamycin Resistance *Staphylococcus aureus* among Various Clinical Specimens in Khartoum state Sudan

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ABSTRACT

Background : *S. aureus* is frequently associated with skin infections, pneumonia, surgery wounds, bacteraemia, osteomyelitis and endocarditis, being considered one of the most important pathogens of the human being, both at the community level and at nosocomial infections, and may become serious if caused by antimicrobial resistant strains, especially methicillin-resistant S. aureus (MRSA) strains, which are resistant to most of the antimicrobial agents, methicillin-sensitive S.aureus (MSSA) and isolates with reduced susceptibility and resistance to vancomycin, which is the last drug for the treatment of MRSA infections. So, this study aimed to detect the existence of inducible resistance of S. aureus to Clindamycin in Khartoum-Sudan among patients attended to Suba University Hospital.

Methods : The study was performed as cross-sectional one, 53 clinical isolates of S. aureus obtained from (34 females and 19 males) with different clinical condition among patients attended to Suba University Hospital in Khartoum-Sudan from April to August 2017. To detect inducible clindamycin resistant by using D test. In addition to that MRSA / MSSA all the isolates screened for methicillin resistant by using 1 µg oxacillin then examined for inducible clindamycin resistant by D test. In addition to that examine for antimicrobial susceptibility profile which include vancomycin, gentamycin, tetracycline and co-trimoxazole. The data were analyzed using Statistical Package for Social Science, version 22, P. value <0.05 was considered statistically significant

Results : out of 53 isolates, 36 S. aureus isolated resistant to Clindamycin, 26 (72.2%) were MRSA and 10 (27.8%) were MSSA by means of D test, while 17 (32.1%) of isolates were sensitive 9 (53%) MRSA and 8 (47 %) MSSA. Comparing Induced clindamycin resistance showed equally distribution among MSSA and MRSA isolates, giving no significant difference as P- value 0.167.

Conclusion : This study showed that D.test to detect inducible clundamycin resistance in staphylococcus aureus.

Keywords : Staphylococcus Aureus, Inducible Clindamycin Resistance (iCR).

I. INTRODUCTION

S. aureus is one of the most frequent causes of bacterial infections in skin, bone, tissue, and blood, with bacteremia being one of the most severe presentations (1), or as a marker of severe infections (2). Bacteremia refers to viable bacteria in the blood; it can be asymptomatic which occurs in normal daily activities such as conducting oral hygiene and after minor medical procedures. In a healthy person, these clinically benign infections are transient without further sequelae. But, when immune response mechanisms fail or become overwhelmed, bacteremia becomes a bloodstream infection that can evolve into many clinical spectrums and is differentiated as septicemia. Untreated and clinically significant bacteremia progresses to systemic inflammatory response syndrome (SIRS), sepsis, septic shock, and multiple organ dysfunction syndromes (MODS) (3-5). Bacteremia may be the best-described manifestation of S. aureus infection, it has been recognized that, the prevalence, prognosis, and outcome of S. aureus bacteremia (SAB) in industrialized regions of the world (6). SAB is often associated with severe complications, such as secondary deep infections, and a reported mortality rate of 20–25% (7). However, nearly half of cases are classified as uncomplicated infections associated with a significantly lower risk of relapse and death (8-9). Uncomplicated SAB is partly due to in-hospital infections related to invasive procedures and the use of peripheral and central venous catheters, as well as community-acquired skin and soft tissue infections (10,11). The extensive use of antibiotics worldwide is closely related to the increasing issues of antimicrobial resistance and antibiotic-associated infections (12,13).

As *S. aureus* is the one of the most common organisms causing nosocomial (infections occurred in hospitals) and community-acquired infections in every region of the world, so increasing prevalence of resistance among *Staphylococci* is an increasing problem(4) The emergence of community-associated methicillin-resistant *Staphylococcus aureus (MRSA)* infections in the late 1990s(14,15) resulted in a shift in the recommendations for antimicrobial therapy for staphylococcal infections from penicillinase-resistant β-lactam agents active against methicillin-susceptible *S. aureus* (MSSA) to other drugs included clindamycin(21-25) . So, Clindamycin is not recommended for any infection caused by an inducible resistant isolate. MRSA isolates with inducible clindamycin resistance (iCR) are resistant to erythromycin and sensitive to clindamycin on routine testing. D-test can help to determine whether clindamycin could be used as a therapeutic option (26).

Clindamycin is a food and drug agency (FDA)-approved for the treatment of septicemia, intra-abdominal infections, lower respiratory infections, gynecological infections, bone, and joint infections, and skin and skin structure infections as well as in the treatment of streptococcal pharyngitis, acne vulgaris, bacterial vaginosis, and severe pelvic inflammatory disease(27) Clindamycin acting through preventing peptide bond formation, so inhibiting protein synthesis by reversibly binding to 50S ribosomal subunits. Depending on the organism, infection site, and drug concentration, clindamycin may be a bacteriostatic or bactericidal antibiotic. Administrated orally, absorption cannot take place until clindamycin palmitate becomes hydrolyzed in the gastrointestinal (GI) tract. It then distributes across the body in tissue and other regions containing blood. Clindamycin cannot efficiently penetrate meninges very well and is therefore not an antibiotic of choice for infections of the cerebrospinal fluid (CSF). As it travels through the bloodstream, clindamycin is primarily bound to protein (27-30).

Development of drug resistance in *S. aureus* has led to the use of older antibiotics such as macrolide, lincosamide, and streptogramin B (MLSB) antibiotic

(31-33). So extensive use of these antibiotics in serious staphylococcal infections has caused the emergence of *S. aureus* resistant to MLSB antibiotics (34). Resistance occurs by different mechanisms to these microbiologically related antibiotics. Resistance due to active efflux encoded by *msr* (A) gene confers resistance to macrolides and streptogramin B (MS phenotype) but not to clindamycin. Ribosomal target modification, another mechanism of resistance, confers resistance to macrolide, type B streptogramin and also to clindamycin (MLS_B phenotype). MLS_B resistance in *staphylococci* is either constitutive ($cMLS_B$), where rRNA methylase is always produced or inducible ($iMLS_B$), where methylase is only produced in the presence of an inducer, and is encoded by erm (A) or erm (C) gene (35-36). Patients infected with $iMLS_B$ strains of *staphylococcus* if treated with clindamycin can develop constitutive resistance during therapy and subsequently result in treatment failure (37).

Strains with inducible resistance to clindamycin are difficult to detect in the routine laboratory (38), so lack of identity of inducible clindamycin resistance leads to false laboratory reports and could lead to clinical failure when clindamycin is used therapeutically and cause treatment problems (39-40). On the other hand, labeling of staphylococci as clindamycin resistant, while they are only resistance to erythromycin, could stop prescription of clindamycin, in cases that infections have occurred by truly clindamycin-susceptible staphylococcal isolates (38-41). A simple laboratory test (as titled D-zone test) can differentiate between staphylococci that have inducible *erm* genes-mediated resistance and those which have efflux pump-mediated resistance (42).

II. MATERIAL AND METHOD

This study was cross sectional study conducted during 5 months from April to August 2017, of targeting patients admitted in Soba Universal hospital. Patients were 53, females' contributors were 34(64.2%), while 19 (35.8%) males were involved. Collecting different samples, they were processed to isolate S. aureus, included pus, body fluids, and urine. S. aureus isolates were identified by standard biochemical techniques (43). Bacteria were sub cultured on Manitol Salt Agar then followed by aerobic overnight incubation at 37°c. The isolates were re- identified based on colonial morphology, Gram's stain, and other biochemical tests according to the Gram Stain. Detection of MRSA: Oxacillin susceptibility testing was performed with 1µg oxacillin disks on Mueller Hinton Agar, incubated aerobic overnight at 37°C. The interpretation of the bacterial susceptibility results was recorded as "resistant," intermediate and "sensitive." The antibiotic susceptibility was determined according with methods of Clinical and Laboratory Standards Institute (CLSI) for the purpose of the study

Antimicrobial susceptibility testing was done by Kirby-Bauer's disc diffusion method using various antimicrobial agents like Vancomycin (30 1µg), oxacillin (1 g), gentamycin (10 g), erythromycin (15 g), as per CLSI guidelines (44). For detection of methicillin resistance, 1 g of oxacillin disc was placed; plates were incubated at 35°C for 24h. Isolates with zone diameters ≤10 mm was labeled as methicillin resistant (43). For detection of inducible clindamycin resistance, a disk approximation test was performed by placing a 2 mg clindamycin disc from 19 mm away from the edge of a 15 mg erythromycin disc. Following overnight incubation at 37°C.

Inducible clindamycin resistance was confirmation by forming of a flattening shape of the clindamycin inhibition zone (D shape) around the erythromycin disk which indicated erythromycin had induced clindamycin resistance. Furthermore, the staphylococcal isolates were grouped to two different phenotypes, these phenotypes were: D positive phenotype (inducible resistant to clindamycin and Resistant for erythromycin) and D Negative (Sensitive for both erythromycin and clindamycin).

III. RESULTS AND DISCUSSION

In this study a total of 53 S. aureus isolates were collected from samples (pus, urine and body fluids) of 34 (64.2%) females and 19 (35.8%) males as in Figure 1.

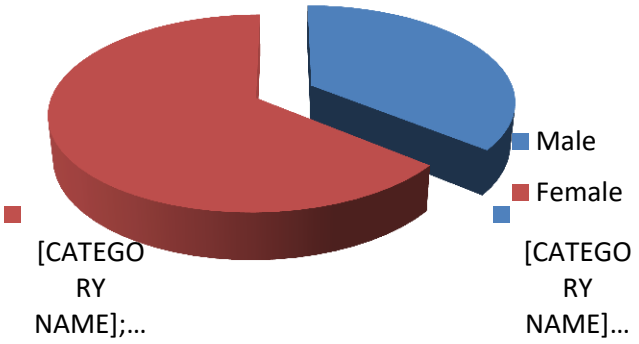

Figure 1 : shows the distribution of gender across the study participants.

The distribution of isolates among males and females did not showed statistically significant difference as p value= 0.302 (significant difference obtained when p value <0.05).

Majority of isolated S. aureus were obtained from pus (50 %) followed by urine (23%); blood and body fluids (27%). A total of 36 (67.9%) S. aureus isolates were resistant to Clindamycin. Other antibiotic was included as they were set at the disc, so S. aureus isolates sensitive to vancomycin 51(96.2%), Gentamycin38 (71.7%), and Tetracycline31 (58.5 %) and Co-Trimoxazole 35(66.0%) as in table 1.

Table 1 : Antimicrobial Susceptibility Profile 0F S. aureus Isolates

Antimicrobials	Resistant	Sensitive
	No (%)	No (%)
Vancomycin	2 (3.8%)	51(96.2%)
Gentamycin	15(28.3%)	38(71.7%)
Tetracycline	22(41.5%)	31(58.5%)
Co-trimoxazole	35(66.0%)	18(34.0%)
Erythromycin	37(69.8%)	16(30.2%)

Out of the 53 isolates there were 36(67.9%) S. aureus isolates resistant to Clindamycin and 17 (32.1%) were sensitive to clindamycin. Out of 36 S. aureus isolates resistant to Clindamycin there were 26 (72.2%) were MRSA and 10 (27.8%) were MSSA, while 17 S. aureus isolate sensitive to clindamycin there were 9(52.9) were MRSA and8(47.1) were MSSA. Comparing Induced clindamycin resistance among MSSA and MRSA isolates, there was no significant difference (P- value 0.167) as shown in table 2.

Table 2 : comparing the frequency distribution of D-test among S. aureus isolates

	D-test		P- Value
Isolates	Resistant	Sensitive	
MSSA	10 (27.8%)	8 (47.1%)	0.167
MRSA	26 (72.2%)	9 (52.9%)	
Total	36(100%)	17(100%)	

The association of induced clindamycin resistance and other antibiotics was insignificant including Vancomycin, Gentamycin, tetracycline P value > 0.05, while there was association with Co-trimoxazole and Erythromycin P value 0.009 and 0.000, the different frequency of resistant and sensitivity of S. aureus isolates to D-test in comparison to the other antimicrobial test as in table 3.

Table 3 : Antimicrobial profile among clindamycin sensitive and resistant

Antimicrobials	D-test		Total	
	Resistant	Sensitive		P. value
Vancomycin				
Resistant	2 (3.8%)	0 (0.0%)	2 (3.8%)	0.457
Sensitive	34 (64.2%)	17 (32.1%)	51 (96.2%)	
Erythromycin				
Resistant	36 (67.9%)	1 (1.9%)	37 (69.8%)	0.000
Sensitive	0 (0.0%)	16 (30.2%)	16 (30.2%)	
Co-trimoxazole				
Resistant	28 (52.8%)	7 (13.2%)	35 (66.0%)	0.009
Sensitive	8 (15.1%)	10 (18.9%)	18 (34.0%)	
Tetracycline				
Resistant	14 (26.4%)	8 (15.1%)	22 (41.5%)	0.573
Sensitive	22 (41.5%)	9 (17.0%)	31 (58.5%)	
Gentamycin				
Resistant	12 (22.6%)	3 (5.7%)	15 (28.3%)	0.237
Sensitive	**24 (45.3%)**	**14 (26.4%)**	**38 (71.7%)**	

Discussion

In this study a 53 isolates of S. aureus were collected from different samples compose of 64.2% females and 35.8% males. Majority of isolated s. aureus were obtained from pus (50 %) followed by urine (23%); blood and body fluids (27%). over all clindamycin resistant S. aureus among study 36/53(67.9). Other antibiotics susceptibly profile of s. aureus included sensitive to vancomycin51 (96.2%), Gentamycin38(71.7%), and Tetracycline31(58.5 %). Out of the 36 S. aureus isolates resistant to Clindamycin, 26/36 (72.2%) were MRSA and 10/36 (27.8%) were MSSA, while 17/53 (32.1%) of isolates were sensitive to Clindamycin, 9/17 (53%) MRSA and 8/17 (47 %) MSSA. Induced clindamycin resistance showed equally distribution among MSSA and MRSA isolates, giving no significant difference as P- value 0.167. The association of induced clindamycin resistance and other antibiotics was insignificant including Vancomycin, Gentamycin, tetracycline P value0.457and 0.237and 0.573 respectively. , while there was association with Co-trimoxazole and Erythromycin P value 0.009 and 0.000, the different frequency of resistant and sensitivity of S. aureus isolates to D-test in comparison to the other antimicrobial test.

An agreement with study was conducted in Microbiology laboratory of Nepal Medical College

and Teaching Hospital, Kathmandu, Nepal to find the incidence of different phenotypes of MLSB resistance among S. aureus from clinical samples and their association with methicillin resistance. Of the 270 clinical isolates of S. aureus68/270(25.1%) were MRSA. clindamycin resistance was seen in113/270(41.8%) isolates. Resistance to clindamycin were higher in MRSA as compared to MSSA (clindamycin-resistance: 79.4% Vs 41.8%). (45)

Dis agreement obtained by study was aimed to detect methicillin resistance and iCR among *S. aureus* isolates, effectiveness of some commonly used antibiotics and correlation between methicillin resistance and iCR. The study included 46 *S. aureus* isolates subjected to Kirby-Bauer's disk diffusion method for antibiotic susceptibility testing (AST) to estimate MRSA and resistance to some commonly used antibiotics. D-test was employed to detect iCR. Eleven of the 46 (23.9%) isolates tested were MRSA. Overall, 19 (41.3%) isolates showed of iCR. Vancomycin and linezolid were found to be 100 per cent effective. A positive Karl-Pearson's coefficient of correlation (0.89) between methicillin resistance and iCR was obtained (46).

IV.CONCLUSION

This study conclude that inducible clindamycin resistance in S.aureus and D.test can be used as simple and reliable method to detect clindamycin resistance in routine clinical laboratories

also comparing induced clindamycin resistance has equally distribution among MRSA and MSSA isolates giving no difference

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