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(RESEARCH ARTICLE)

Antimicrobial resistance profile and molecular detection of MecA gene in methicillin resistant *Staphylococcus aureus* from patients in selected general hospitals in Abuja municipal, Nigeria

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Abstract

Staphylococcus aureus (*S. aureus*) is globally recognized as an important pathogen associated with both hospital and community acquired infections. Studies on antibiotic resistance profile of *S. aureus* and carriage of mecA gene in methicillin resistant isolates from patients attending selected general hospitals in Abuja Municipal, Nigeria was carried out. Three hundred and sixty (360) clinical samples (200 urine, 50 high vaginal swabs, 60 ear swab and 40 wound swabs) were collected from Asokoro General Hospital (AGH), Garki Hospital Abuja (GHA) and Wuse General Hospital (WGH); and *S. aureus* was isolated and identified using standard microbiological methods. Antibiotic susceptibility testing of the isolates was carried out using disc diffusion method. Molecular detection of mecA gene in methicillin resistant isolates was carried out using the polymerase chain reaction method. The total occurrence of *S. aureus*was 15.3% (55/360); and the occurrence in relation to the selected hospitals was high in GHA (22.7%) and low in AGH (10.3%). The occurrence of *S. aureus* was highest in wound swabs in all the hospitals in the order: GHA (47.1%) > AGH (40%) > WGH (35.7%). The isolates from all the hospitals were highly (≥ 50.0%) resistant to all the antibiotics tested; but moderately (≤ 40.0%) to gentamicin and levofloxacin. The occurrence of multi-drug resistant (MDR) isolates in the selected hospitals was high in GHA (27%) but low in AGH (12%). Of the 32 oxacillin resistant isolates, mecA gene was detected in 30 (93.8%). The *S. aureus* isolates were less resistant to gentamicin and levofloxacin and most of the oxacillin resistant isolates harbored mecA gene.

Keywords: *Staphylococcus aureus*; Methicillin resistance; mecA

1. Introduction

Staphylococcus aureus (*S. aureus*) is a gram positive coagulase positive coccus in the family of staphylococeae [1] and a usual resident of human skin and mucous membrane [2]. This organism have been widely reported as a causative agent of infections such as bacteremia, endocardiatis, urinary tract infection (UTIs) and soft tissue infections [3-4]; both in hospital and community settings.

The first methicilin resistant *S. aureus* (MRSA) was detected in the early 1960 [2-3]. Methicillin resistant *Staphylococcus aureus* is resistant to a wide group of antibiotics known as the β-lactams including penicillin and cephalosporins [5]. The MRSA is also known as oxacillin resistant *S. aureus* (ORSA) because methicillin and

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oxacillinare members of the same generation of penicillin [2]. *Staphylococcus aureus* isolates resistant to methicillin is caused by acquisition of *mec*Agene that produces an alternative penicillin-binding-protein (PBP2a) which has lower affinity for β-lactam antibiotics [2-6].

Methicillin resistant *S. aureus* constitute a crucial global health challenge to hospitals all over the world due to its emergence and spread of the isolates with decreased susceptibility to numerous classes of antibiotics [7] that are difficult to contain and be treated [8]. Infections caused by MRSA are a serious problem both in the community and hospital practice, affecting people of all ages and gender [9-10]. The existence of multidrug resistance MRSA strains in patients with cases has reduced the available options of managing the pathogen which in turn requires innovative means of counteracting the pathogen and the infection [11].

Few studies on molecular detection of *mec*A gene in *S. aureus* isolates are have been reported elsewhere in Nigeria [2-5-13] but no such from the area under study. It is thus necessary to investigate the carriage of *mec*A genes *S. aureus* isolates in the area under study. This study investigated the presence of *mec*A genes in MRSA isolates from patients in selected general hospitals in Abuja Municipal, Nigeria.

2. Material and methods

2.1. Study location

The study was carried out in three selected general hospitals in the Abuja Municipal namely: Asokoro General Hospital (AGH), Garki Hospital Abuja (GHA) and Wuse General Hospital (WGH). These general hospitals were chosen because they represent the oldest and busiest of all the general hospitals in the Abuja Municipal.

2.2. Ethical approval

The ethical approval for the study was obtained from Research and Ethics Committee of the Federal Capital Territory, Abuja, Nigeria. This approval was obtained after due consideration of a proposal for the study by the requisite authority.

2.3. Sample collection

A total number of 360 clinical samples namely 173 urine (80 AGH, 44 GHA, 49 WGH), 86 high vaginal swabs (19 AGH, 27 GHA, 40 WGH), 60 ear swabs (8 AGH, 22 GHA, 30 WGH) and 41 wound swabs (10 AGH, 17 GHA, 14 WGH) were collected from specimens submitted to the collection centres of the laboratories in the selected hospitals; and then transported to the Microbiology laboratory in Nasarawa State University, Keffi, for analysis.

2.4. Isolation of *Staphylococcus aureus*

*Staphylococcus aureus*was isolated from clinical samples by modification of the method earlier described [13]. Briefly, a loopful of sample was streaked on mannitol salt agar (MSA: Oxoid Ltd, Basingstoke, UK) and the plate was incubated at 37ºC for 24 h. Golden yellow colonies that grew on MSA were considered presumptive *S. aureus*.

2.5. Identification of *Staphylococcus aureus*

The presumptive *S. aureus* was identified by gram staining and biochemical tests (catalase, coagulase and oxidase test) as earlier described [13].

2.6. Confirmation of *Staphylococcus aureus* using KBOO HiStaph™ kit

The suspect *S. aureus* isolates which was Gram positive cocci, clusters, catalase-positive and coagulase-positive were confirmed using KBOO HiStaph kits following manufacturer's instructions. 2 pure colonies of 24-h nutrient agar (Oxoid Ltd., Basingstoke, UK) culture of suspected *S. aureus* were transferred into 5ml of sterile normal saline and adjusted to the turbidity equivalent to McFarland 0.5. The kit was aseptically opened by peeling off the sealing foil and 50 µl of the standardized suspension was inoculated into each well and the well was sealed off using the sealing foil and incubated at 37°C for 24 h. After incubation, 3 drops of Baritt reagent was added to well No.1; followed by 1 drop of Baritt reagent B. also 2 drops of NaOH (Sigma-Aldrich Laborchemikalien, GmbH) was added to well No.2 and the results was interpreted as per standard given in the identification index.

2.7. Antibiotic susceptibility testing

The antibiotic susceptibility testing of the isolates was carried out as earlier described by Clinical and Laboratory Standards Institute (CLSI, 2014). Briefly, three (3) pure colonies of *S. aureus* was inoculated in to 5 ml sterile 0.85% (w/v) NaCl (normal saline) and the turbidity of the *S. aureus* suspension adjusted to the turbidity equivalent to 0.5 McFarland's standard. The McFarland's standard was prepared as follows; 0.5 ml of 1.172% (w/v) $BaCl_2.2H_2O$ was added into 99.5 ml of 1% (w/v) H_2SO_4. An oxacillin disc (1 µg) was used to detect methicillin resistance.

A swab stick was soaked in standardized bacteria suspension and streaked on Mueller-Hilton agar (Oxoid Ltd, Basingstoke, UK) plates and the antibiotic discs (Oxoid Ltd, Basingstoke, UK) aseptically placed at the centre of the plates and allowed to stand for one hour pre-diffusion time. The plate was then incubated at 37°C for 24 h in an incubator ((Model 12-140E, Quincy Lab Inc., USA). The diameter zone of inhibition in millimetre was measured and the result of the susceptibility was interpreted in accordance with the susceptibility break point earlier described by the CLSI [14].

2.8. Determination of multiple antibiotic resistance (MAR) index

The MAR index of the antibiotic resistant isolates was determined using the formula as described [15]:

$$MAR\ Index = \frac{\text{Numberof antibiotics to which isolate was resistant}}{\text{Total number of antibiotics tested}}$$

2.9. Classification of antibiotic resistance

Antibiotic resistance in the isolates were classified into: multidrug resistance (MDR: non-susceptible to ≥1 agent in ≥3 antimicrobial categories); extensive drug resistance (XDR: non-susceptible to ≥1 agent in all but ≤2 antimicrobial categories); pan drug resistance (PDR: non-susceptible to all antimicrobial listed); and non-multi drug resistance (NMDR) [16].

2.10. Molecular detection of *mec*A gene in methicillin resistant *Staphylococcus aureus*

Staphylococcus aureus isolates resistant to oxacillin were considered methicillin resistant. These isolates were screened for *mec*A gene as described herewith.

2.10.1. DNA extraction

TheDNA of methicillin resistant *S. aureus* isolates were extracted using boiling method as described [17]. Briefly following purification, 1 pure colony of a MRSA isolate was inoculated into 2 ml of Luria-Bertani (LB: Oxoid Ltd., Basingstoke, UK) broth and incubated at 37°C for 8 h and 2 ml of LB broth culture was transferred into Eppendorf tube and microcentrifuged at 3200 rpm for 2 min at room temperature and the supernatant was discarded leaving the cells and the cells were washed twice with washing buffer. 0.5 ml of sterile phosphate buffer was added to the pellet and vortexed for 5 sec after which it was heated at 90°C for 10 min and rapid cooling was done by transferring the tubes into freezer for 10 min and thereafter it was centrifuged at 3200 rpm for 1 min to separate the DNA and the cell debris and 300µl of the supernatant containing the DNA was transferred into 2 ml Eppendorf tube and stored at -10°C until used.

2.10.2. Amplification of target mecA gene

The DNA amplification of target *mecA* gene in methicillin resistant *S. aureus* isolates was carried out by PCR method as earlier described [18]. Briefly, the PCR reaction was carried out in 25 µl reaction volumes which were made up of 5 µl of master mix (Qiagen), 2.4 µl of primers, 0.5 µl of $MgCl_2$, 1.5 µl of DNA template and 15.6 µl of nuclease free water. The primers used, their sequences and amplicon sizes are as described in Table 1. The reaction tubes were placed in the hole of the thermal cycler (Model TC-312, Techne, England) and the door of the thermal cycler was closed and the *mecA* gene was amplified under the following condition; Initial denaturation at 95°C for 3 min, followed by 33 cycles of amplification of 94°C for 1 min, 53°C for 30 sec, initial extension at 72°C for 1 min with a final extension at 72°C for 6 min.

Table 1 Primer used, sequence and amplicon size

Target gene	Sequence	Amplicon size (bp)	References
*mec*A	(F)5´AAAATCGATGGTAAAGGTTGGC (R)´AGTTCTGCAGTACCGGATTTGC	533	[13]

2.10.3. Agarose gel electrophoresis

Eight microliter of the amplified target *mec*A gene was separated using 1.5% agarose in agarose gel electrophoresis to determine the base pair of the *mec*A gene and 1500 bp DNA ladder was used as a standard.

2.11. Statistical analysis

The data obtained in this study was analysed using chi-square by use of Smith Statistical Package (SSP) version (2.80) and the significance was determine at 95% confidence interval.

3. Results and discussion

3.1. Isolation and identification of *Staphylococcus aureus*

The cultural, morphological and biochemical characteristics of *S. aureus* isolated from patients in selected general hospitals in Abuja Municipal, Nigeria is as shown in Table 2.

Table 2 Cultural, morphological and biochemical characteristics of *Staphylococcus aureus* from patients in selected general hospitals in Abuja Municipal, Nigeria

Cultural characteristics		Golden yellow colonies on MSA
Morphological Characteristics	Gram stain	+
	Morphology	Cocci in cluster
Biochemical Characteristics	Cat	+
	Coa	+
	Vp	+
	Akp	+
	ONPG	-
	Ur	+
	Arg	+
	Man	+
	Su	+
	Lac	+
	Ar	-
	Rf	-
	Tr	+
	Mal	+
Inference		*S. aureus*

MSA = Mannitol Salt agar, + = Positive, - = Negative, Cat = Catalase, Coa = Coagulase, Vp= Voges-Proskauer, Akp= Alkaline Phosphate, ONPG = Ortho-Nitrophenyl-β-galactoside, Ur= Urease, Arg = Arginine, Man = Mannitol, Su = Sucrose, Lac = Lactose, Ar = Arabinose, Rf = Raffinose, Tr = Trehalose, Mal = Malt

3.2. Occurrence of *Staphylococcus aureus* in patients

The occurrence of *S. aureus* in the patients in the selected general hospitals is shown in Figure 1. The overall occurrence was 15.3% (55/360); and the occurrences in relation to the selected hospitals were of the order: GHA (22.7%) > WGH (13.5%) > AGH (10.3%).

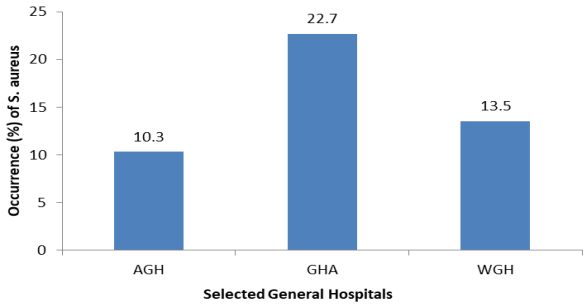

Figure 1 Overall occurrence of *Staphylococcus aureus* in patients from selected general hospitals in Abuja Municipal, Nigeria. (AGH = Asokoro General Hospital; GHA= Garki Hospital Abuja; WGH = Wuse General Hospital)

The occurrences in relation to the clinical samples are as shown in Figure 2. The order of occurrence in urine was: GHA (20.5%) > WGH (12.2%) > AGH (7.5%); in High Vaginal Swab: GHA (18.5%) > WGH (12.5%) > AGH (5.3%); in Ear Swab: GHA (13.6%) > AGH (12.5%) > WGH (6.7%); in wound swab: GHA (47.1%) > AGH (40%) > WGH (35.7%).

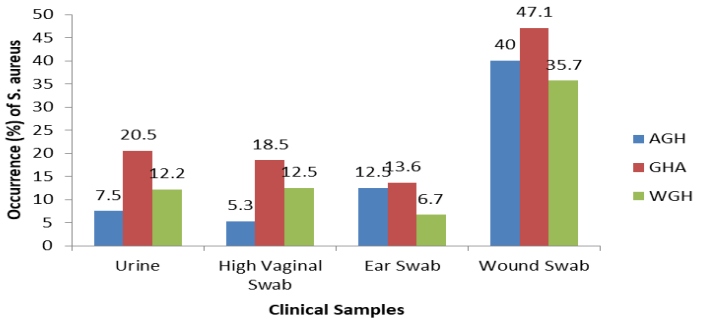

Figure 2 Occurrence of *Staphylococcus aureus* in relation to the clinical samples from patients in selected general hospitals in Abuja Municipal, Nigeria. (AGH = Asokoro General Hospital; GHA= Garki Hospital Abuja; WGH = Wuse General Hospital)

3.3. Antibiotic resistance profile of the *Staphylococcus aureus* isolates

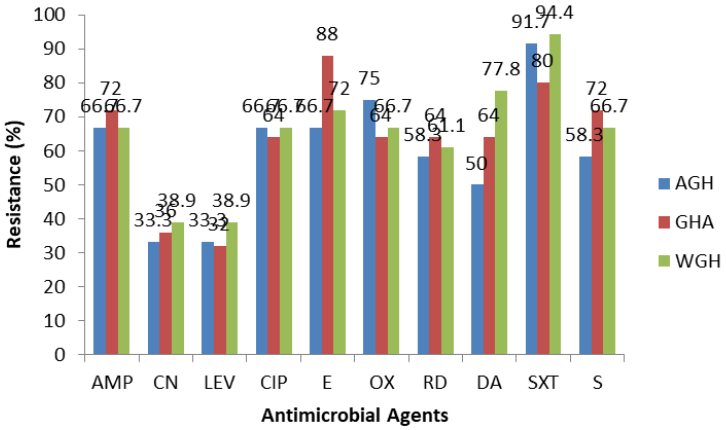

Figure 3 Antibiotic Resistance of *Staphylococcus aureus* from patients in selected general hospitals in Abuja Municipal Nigeria. (AGH = Asokoro General Hospital; GHA= Garki Hospital Abuja; WGH = Wuse General Hospital; AMP = Ampicillin; CN = Gentamicin; LEV = Levofloxacin; CIP = Ciprofloxacin; E = Erythromycin; OX = Oxacillin; RD = Rifampicin; DA = Clindamycin; SXT = Sulphamethoxazole/Trimethoprim; S = Streptomycin)

Antibiotics resistance profile of the *S. aureus* isolates is as shown in Figure 3. The isolates from AGH showed highest resistance to Sulphamethoxazole/Trimethoprim (91.7%) and least resistance to Gentamicin and Levofloxacin (33.3%); those from GHA showed highest resistance to Erythromycin (88.0%) and the least resistance to Gentamicin (33.3%) and Levofloxacin (32.0%); isolates from WGH showed highest resistance to Sulphamethoxazole/Trimethoprim (94.4%) and least resistance to Gentamicin and Levofloxacin (38.9%).

3.4. Antibiotic resistance phenotypes of the *Staphylococcus aureus* isolates

Antibiotic resistance phenotypes of the antibiotic resistant *S. aureus* isolates are as shown in Table 3. The most common phenotypes in the isolates from all the selected hospitals were AMP, CN,CIP,E,OX,RD,DA,SXT,S and AMP-LEV-CIP-E-OX-RD-DA-SXT-S with an occurrence of 1.8% each.

Table 3 Antibiotic resistance phenotypes of *Staphylococcus aureus* from patients in selected general hospitals in Abuja Municipal, Nigeria

Antibiotic Resistance Phenotypes	No. (%) Occurrence of Phenotype		
	AGH	GHA	WGH
S,SXT	1(1.8)	0(0.0)	0(0.0)
SXT,OX	0(0.0)	0(0.0)	1(1.8)
E,SXT,S	0(0.0)	1(1.8)	0(0.0)
AMP,DA,S	0(0.0)	0(0.0)	1(1.8)
RD,SXT,OX,CIP	1(1.8)	0(0.0)	0(0.0)
AMP,S,SXT,LEV	0(0.0)	0(0.0)	1(1.8)
AMP,SXT,OX,CN	0(0.0)	0(0.0)	1(1.8)
S,SXT,CIP,E	0(0.0)	1(1.8)	0(0.0)
AMP,CN,LEV,E	0(0.0)	1(1.8)	0(0.0)
CIP,E,RD,SXT	0(0.0)	1(1.8)	0(0.0)
AMP,E,RD,DA,SXT	0(0.0)	1(1.8)	0(0.0)
AMP,LEV,CIP,E,SXT	1(1.8)	0 (0.0)	0(0.0)
AMP,E,DA,SXT,S	0(0.0)	1(1.8)	0(0.0)
E,OX,RD,SXT,S	1(1.8)	0(0.0)	0(0.0)
AMP,RD,S,SXT,OX	0(0.0)	1(1.8)	0(0.0)
AMP,SXT,OX,E,DA	1(1.8)	0(0.0)	0(0.0)
S,SXT,CIP,E,DA	0(0.0)	0(0.0)	1(1.8)
AMP,OX,CN,CIP,LEV	1(1.8)	0(0.0)	0(0.0)
AMP,S,SXT,CIP,E	0(0.0)	1(1.8)	0(0.0)
AMP,RD,SXT,OX,CIP	0(0.0)	0(0.0)	1(1.8)
RD,S,SXT,OX,CIP,DA	0(0.0)	1(1.8)	0(0.0)
AMP,S,SXT,CIP,E,DA	0(0.0)	1(1.8)	0(0.0)
CN,LEV,CIP,E,DA,SXT	0(0.0)	0(0.0)	1(1.8)
AMP,CN,CIP,E,SXT,S	1(1.8)	0(0.0)	0(0.0)
CN,CIP,E,OX,RD,S	0(0.0)	1(1.8)	0(0.0)
AMP,CN,LEV,E,RD,SXT	0(0.0)	1(1.8)	0(0.0)
CIP,E,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	1(1.8)
AMP,CIP,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	0(0.0)
CN,E,OX,RD,DA,SXT,S	1(1.8)	0(0.0)	0(0.0)
CN,CIP,E,RD,DA,SXT,S	0(0.0)	0(0.0)	1(1.8)
AMP,OX,CN,CIP,E,DA,LEV	0(0.0)	1(1.8)	0(0.0)
RD,S,SXT,OX,CIP,E,DA	0(0.0)	1(1.8)	1(1.8)
AMP,CIP,E,OX,DA,SXT,S	0(0.0)	0(0.0)	1(1.8)
AMP,CN,CIP,E,OX,DA,S	0(0.0)	1(1.8)	0(0.0)
AMP,RD,SXT,OX,CIP,DA,LEV	1(1.8)	0(0.0)	0(0.0)
AMP,RD,SXT,OX,CN,CIP,E	0(0.0)	1(1.8)	0(0.0)
AMP,RD,SXT,OX,E,DA,LEV	0(0.0)	0(0.0)	1(1.8)
AMP,E,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	0(0.0)
AMP,CN,LEV,CIP,E,RD,DA,S	0(0.0)	0(0.0)	1(1.8)
AMP,CIP,E,OX,RD,DA,SXT,S	1(1.8)	0(0.0)	1(1.8)
AMP,LEV,E,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	1(1.8)
AMP,CN,LEV,E,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	0(0.0)
AMP,CN,CIP,E,OX,RD,DA,SXT,S	1(1.8)	1(1.8)	1(1.8)
AMP,CN,LEV,CIP,E,OX,RD,DA,SXT	0(0.0)	1(1.8)	1(1.8)
AMP,LEV,CIP,E,OX,RD,DA,SXT,S	1(1.8)	1(1.8)	1(1.8)
AMP,CN,LEV,CIP,E,OX,RD,DA,SXT,S	0(0.0)	1(1.8)	0(0.0)

AMP = Ampicillin; CN = Gentamicin; LEV = Levofloxacin; CIP = Ciprofloxacin; E = Erythromycin; OX = Oxacillin; RD = Rifampicin; DA = Clindamycin; SXT = Sulphamethoxazole/Trimethoprim; S = Streptomycin

3.5. Multiple antibiotics resistance (MAR) indices of the isolates

All the *S. aureus* isolates were MAR isolates. The MAR indices of the isolates are as shown in Table 4. All the isolates had MAR index of ≥ 0.2; the most common were: 0.5 in AGH (33.3%), 0.7 in GHA (28.0%) and 0.7 in WGH (27.8%).

Table 4 Multiple Antibiotics Resistance Index (MAR) of *Staphylococcus aureus isolates* from patients in selected general hospitals in Abuja Municipal, Nigeria

No. of antibiotics isolate is resistant to (a)	No. of antibiotics tested (a)	MAR Index (a/b)	No. (%) MAR isolates		
			AGH (n=12)	GHA (n=25)	WGH (n=18)
10	10	1.0	0(0.0)	1(4.0)	0(0.0)
9	10	0.9	2(16.7)	4(16.0)	3 (16.7)
8	10	0.8	1(8.3)	1(4.0)	3(16.7)
7	10	0.7	2(16.7)	7(28.0)	5(27.8)
6	10	0.6	1(8.3)	4(16.0)	1(5.6)
5	10	0.5	4(33.3)	4(16.0)	2(11.1)
4	10	0.4	1(8.3)	2(8.0)	3(16.7)
3	10	0.3	0(0.0)	1(4.0)	1(5.6)
2	10	0.2	1(8.3)	0(0.0)	1(5.6)

AGH = Asokoro General Hospital; GHA= Garki Hospital Abuja; WGH = Wuse General Hospital

3.6. Classification of antibiotic resistance in the isolates

The classification of antibiotic resistance in the *S. aureus* isolates into multidrug resistance (MDR), Extensive drug resistance (XDR), Pan-drug resistance (PDR) and non-multi drug resistance (NMDR) categories is as shown in Figure 4. The order of occurrence of MDR isolates in the selected hospitals was: GHA(27%) > WGH (14%) > AGH(12%); XDR was found only in AGH at 1%; and PDR only in WGH at 1%.

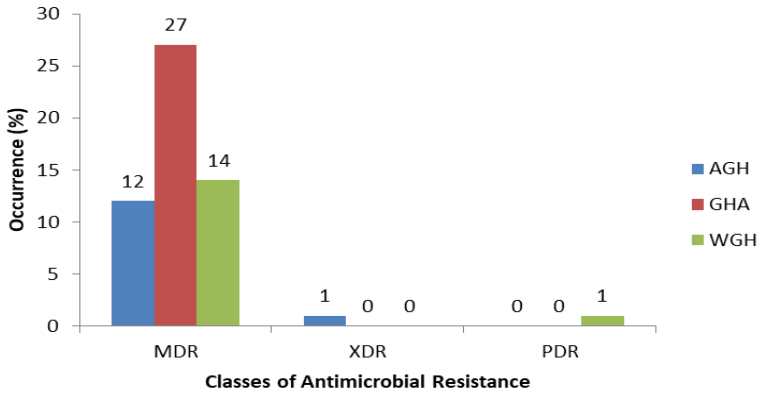

Figure 4 Classification of Antibiotic Resistance in *Staphylococcus aureus* from patients in selected general hospitals in Abuja Municipal, Nigeria. (AGH = Asokoro General Hospital; GHA= Garki Hospital Abuja; WGH = Wuse General Hospital; MDR = Multi Drug Resistance; XDR = Extensive Drug Resistance; PDR = Pan Drug Resistance)

3.7. Occurrence of *mec*A gene in the methicillin resistant *Staphylococcus aureus* isolates

The occurrence of *mec*A gene in the methicillin resistant *S. aureus* isolates is as shown in Figure 5. The order of occurrence of *mec*A gene in the screened isolates was: GHA (100%) > WGH (90%) > AGH (80%). The DNA bands of *mec*A genes in methicillin resistant *S. aureus* are as shown in Figure 7 and 8.

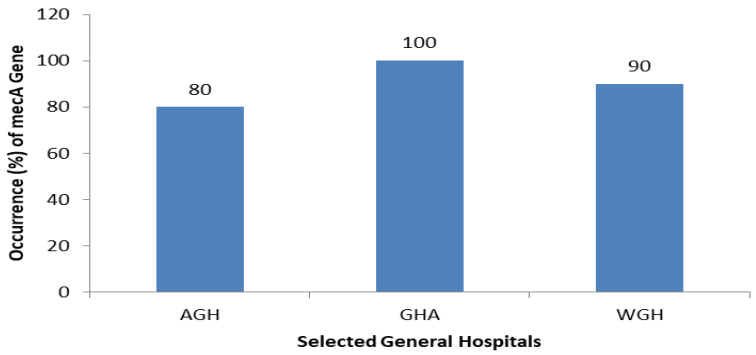

Figure 5 Occurrence of *mec*A gene in methicillin resistant *Staphylococcus aureus* in clinical samples from patients in selected general hospitals in Abuja Municipal, Nigeria. *(*AGH=Asokoro General Hospital, GHA=Garki General Hospital, WGH = Wuse General Hospital).

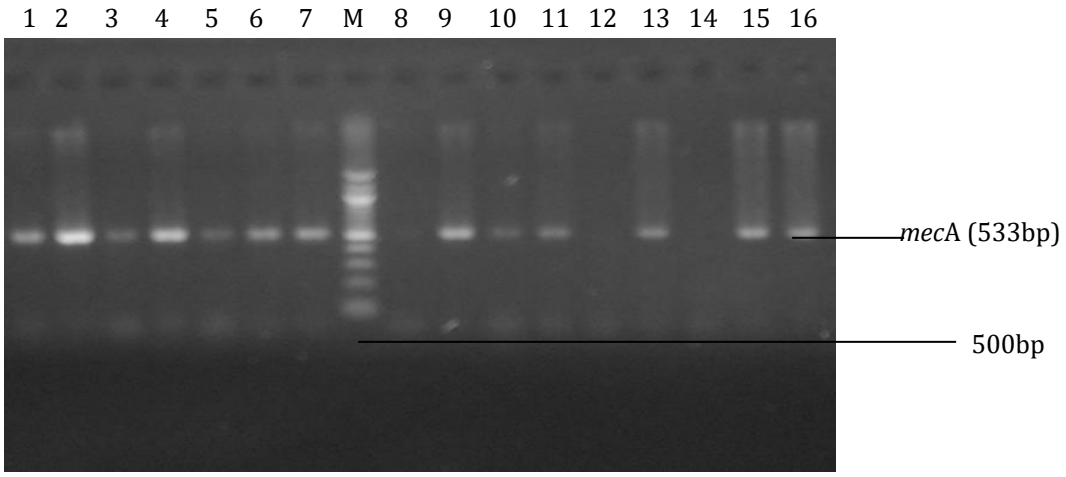

Figure 7 Agarose gel electrophoresis of the amplified *mecA* genes from the *Staphylococcus aureus*isolates. Lanes 1, 2, 3 to 7, 9, 10, 11, 13, 15, and 16 represent the *mec*A band, Lane M represents the 1500bp molecular ladder, while other lanes show no bands.

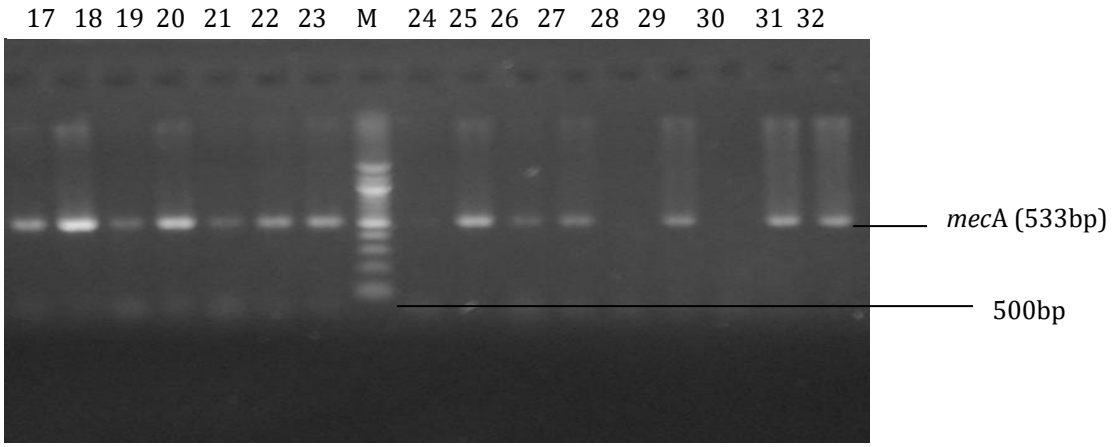

Figure 8 Agarose gel electrophoresis of the amplified *mec*A genes from the *Staphylococcus aureus*isolates. Lanes 17, 18, 19 to 23, 25, 26, 27, 29, 31, and 32 represent the *mec*A band, Lane M represents the 1500bp molecular ladder, while other lanes show no bands.

3.8. Relationship between carriage of *mec*A gene and antibiotic resistance in the isolates

The relationship between *mec*A positive MRSA isolates and antibiotics resistance is as shown in Figure 6. The *mec*A positive MRSA isolates in AGH were highly resistant to erythromycin, rifampicin, clindamycin, sulphamethoxazole/trimethoprim and streptomycin with percentage resistance of 100% but showed low resistance to gentamicin with percentage resistance of 16.7%. In GHA, the *mec*A positive MRSA were highly resistant to rifampicin and sulphamethoxazole/trimethoprim with percentage resistance of (100%) but less resistant to levofloxacin with percentage resistance of 23.1%. In WGA, the *mec*A positive MRSA isolates were more resistant to rifampicin and streptomycin with percentage resistance of 100% but less resistant to gentamicin with a percentage resistance of 16.7%.

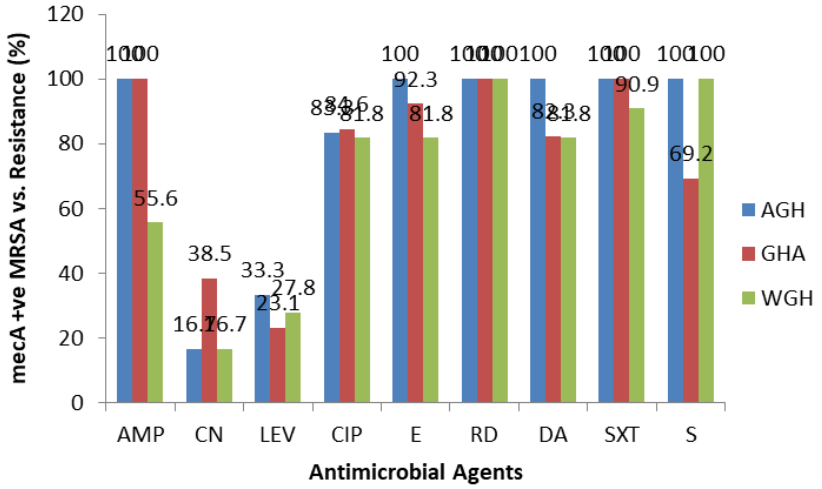

Figure 6 Relationship between *mec*A positive MRSA and antibiotics resistance

The overall occurrence of *S. aureus* in wound swab, HVS, ear swab and urine observed in this study was expected and this is in agreement with the study earlier reported [19]. It is also similar with studies [20-21-22]. The percentage occurrence of *S. aureus* in clinical samples such as wound swab, HVS, ear swab and urine observed in this study was higher than the study earlier reported [22]. Our finding shows the percentage occurrence of *S. aureus*in selected hospitals was higher in wound swab and this finding is consistent with the study earlier reported [23] where *S. aureus* was mostly observed in wound swabs (32.6%), Urine(23.8%) and 29.4%[22-23] and 13.8% [24] and in partial agreement with Obiazi and Garba[25-26] at 17% but in contrast with report elsewhere [27] who reported a high percentage occurrence of *S. aureus* in urine than other clinical samples and also [26] who reported a higher rate in vaginal swab at 39%. The high incidence of *S. aureus* observed among the clinical specimens shows the versatility of this organism amongst other bacteria which makes it the most endemic pathogen in clinical settings. The occurrence of the organism in the clinical samples namely, Urine, Wound swab, ear swab and eye swab observed in this study suggest the organism may likely be responsible for the infection such as UTI, wound infection, deep tissue infections, including osteomyelitis, arthritis, endocarditis, and cerebral pulmonary, renal and breast abscesses[28].

The *S. aureus* isolates in the selected hospitals were less resistant to levofloxacin and gentamicin in this study and this observation is in agreement with the study earlier reported [20-29] but in contrast with [30-31-32]. The low resistance of *S. aureus* to these antibiotics may be due to the fact that such antibiotics may not have been commonly prescribed for treatment of *S. aureus* infection in the location of study and also the parenteral use of gentamicin reduces its abuse. The isolates from selected hospitals as observed in this study were more resistant to ampicillin (A), sulphamethoxazole/trimethoprim (SXT), erythromycin, ciprofloxacin(CIP), rifampicin (RD), and clindamycin (DA), streptomycin (S) was not surprising and this is in agreement with the study earlier described [33] though these antibiotics used are commonly prescribed for treatment of *S. aureus* infections and the resistance observed may be due to abuse and inappropriate use of antibiotics as prescribed by physician. Also the high resistance is understandable since all MRSA strains have been variously reported to be resistant to all β-lactam an antibiotic of which ampicillin is one [34]. There is high level of antibiotic abuse in this environment arising from self-medication which is often associated with inadequate dosage and failure to comply to treatment and availability of antibiotics to consumers across the counter with or without prescription. The percentage resistance of *S. aureus* isolates in the selected hospitals to ciprofloxacin, sulphamethoxazole/trimethoprim observed in this study was higher than in study reported [35] which reported at a rate of 7.3% and 29.8% respectively but similar to study described [20] The low

susceptibility of *S. aureus* isolates to erythromycin and ciprofloxacin was higher than 56.0% and 44.0% resistance to erythromycin and ciprofloxacin as earlier reported [36]. Our findings are also very similar to those of a study done in Taiwan in which resistance rates of 94.9% and 71.8% to erythromycin, sulphamethoxazole/trimethoprim respectively were observed but lower resistance rate for gentamicin at 36.4% as opposed to the study done at 78.2% [37]. The high resistance to sulphamethoxazole/trimethoprim found in this study supports the findings of Kapatamoyo [38] who found sulphamethoxazole/trimethoprim resistance rates (among Staphylococci isolates) at 86% and recommended that it should not be used for treatment of acute bacterial infections [38]. The result of our finding on low susceptibility of *S. aureus* isolates in selected hospitals to clindamycin contradict with 24.4% resistance to clindamycin as earlier described [3]. However, it was similar in the study of 74.5% reported by Fayomi in Ekiti State, 86.5% in Taiwan [37] Ciprofloxacin is another potential antibiotic in the treatment of MRSA infections. Ciprofloxacin and other quinolone antibiotics have been proposed as possible alternatives to parenteral vancomycin therapy on the basis of several in vitro and in vivo animal model data [39] but in this study the resistance rate to ciprofloxacin was high (65.5%) which is consistent with those of several other studies [26-39] but differs from those of Kapatamoyo and others [38], who found very low ciprofloxacin resistances rates.

The percentage resistance of *S. aureus* in selected hospitals to Oxacillin antibiotics observed in this study was higher than a previous study in Maiduguri at a rate of 12.5% [40], Ibadan 30.4% [41]. The high resistance of *S. aureus* to oxacillin observed in this study is in agreement with previous study reported elsewhere in a similar study [20-27-42]. The low susceptibility of *S. aureus* isolates to oxacillin observed in this study is similar with. 78.3%, 89%, 61.5% and 80% resistance as earlier reported [12-43-44]. Thus oxacillin resistant *S. aureus* are commonly isolated from clinical samples.

In this study a total number of 46 resistance patterns were observed which is closely related with a study from South Africa which detected 61 resistance patterns and multi-drug resistance of (81.5%) [45]. The most common occurring phenotypes show that these antibiotics may have been abused or commonly used in the treatment of *S. aureus* infections in the selected hospitals.

Most of the *S. aureus* isolates in the selected hospitals were multidrug resistant (MDR). The MDR among MRSA strains was higher than the methicillin sensitive strains. These results are in agreement with global findings of MRSA strains being multi-drug resistant [46-47-48]. This might be as a result of the fact that that a large number of the bacteria isolates have been pre exposed to several of the antibiotics. The resistance may also be due to a combination of microbial characteristics such as selective pressure on antimicrobial usage. Also the transmission of drug resistant organisms due to technological and societal changes might also contribute to the high resistance. Other factors may include an increase in irrational consumption of antibiotics and transmission of resistant isolates between people [49].

This study showed that all the MRSA isolates were significantly less sensitive to antibiotics as compared with MSSA isolates. Although methicillin resistant *S. aureus* are not necessarily more virulent than methicillin-susceptible *S. aureus*, treatment options are often severely limited by multidrug resistance [1]. Methicillin resistant *S. aureus* infections are more resistant to some treatments than methicillin-sensitive *Staphylococcus aureus* (MSSA). This study also showed that not all of oxacillin resistant *S. aureus* isolates were mecA gene positive and is similar to studies previously reported [50-51] and this shows that resistance to oxacillin may not necessarily be due to modification of penicillin binding protein which is similar to a report by Mojtaba[52]. This discrepancy between phenotypic and genotypic resistance in the isolates has been reported [53-54] which could be due to other mechanism of resistance to methicillin such as the presence or over expression of β-lactamase enzymes and chromosomal mutations like the acquisition of modified PBPs [54-55].

The existence of these borderline (low-level resistant) strains emphasise the need to screen *mecA*negative strains for other resistance mechanisms although this was not evaluated in this study. The percentage occurrence of *mecA* gene in oxacillin resistant *S. aureus* observed in this study 96.4% is not surprising though higher than 22.2%, 38.0% and 38.0% earlier reported [56-57]. The occurrence of *mecA* in oxacillin resistant *S. aureus* suggest that the gene may be responsible resistance to oxacillin antibiotics and the isolates may also be referred to as methicillin resistant *Staphylococcus aureus* (MRSA) since methicillin and oxacillin are in the class of β-lactam resistant penicillin antibiotics. The high resistance of *mecA* positive MRSA isolates to antibiotics namely sulphamethoxazole/trimethoprim, erythromycin, streptomycin, ciprofloxacin, clindamycin and rifampicin observed in this study was expected and this finding is in agreement with the study earlier reported [58] that MRSA isolates are as well resistant to other class of antibiotics and the high level of resistance of MRSA to other class of antibiotics in this study suggest that this isolate may likely cause staphylococcal infection that may be difficult to be treated. Drug

resistant *S. aureus*, especially the methicillin-resistant strains in health care and community settings is an increasingly reported event and this makes the treatment of infections caused by this organism very difficult [20-58].

4. Conclusion

The *Staphylococcus aureus* isolates were less resistant to gentamicin and levofloxacin and most of the oxacillin resistant isolates harbored *mec*A gene.The occurrence of MRSA strains in patients has reduced the available options of managing the pathogen which in turn requires innovative means of counteracting the pathogen and the infection.

Compliance with ethical standards

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Disclosure of conflict of interest

Authors have declared that no conflict of interests exists.

Statement of ethical approval

Ethical approval was obtained from the Research and Ethics Committee of the Federal Capital Territory, Abuja, Nigeria for the study.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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