ORIGINAL ARTICLE

# Antimicrobial susceptibility pattern and plasmid-mediated antibacterial resistance in *Staphylococcus aureus* and coagulase-negative staphylococci

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**Abstract**

**Background**: *Staphylococcal infections* constitute problems to health care institutions. *Its* resistance to antibiotic has been associated with resistant plasmids (R-plasmid) that have the ability to mediate the production of drug inactivated enzymes such as β-lactamase.

**Method**: Forty five *Staphylococcus aureus* (*S. aureus)* and 15 Coagulase-negative Staphylococci (CoNS) were isolated from clinical samples and isolates subjected to antibiotic susceptibility testing, plasmid curing and plasmid DNA isolation.

**Result**: The highest percentages isolates were recovered from urine samples and the least from high vagina swab and wound swab for *S. aureus* and CoNS respectively. The antibiogram showed that majority of *S. aureus (*95.6%) was resistant to cefuroxime and ceftazidime, while CoNS (93.3%) were more resistance to cefuroxime and gentamycin. (p=0.17). 24.4% *S. aureus* and 20% CoNS were resistant to more than 5 antibiotics with multiple antibiotic resistances (MAR) index of 0.33 to 0.89 for *S. aureus* and 0.56 to 0.78 for CoNS. Most of the *S. aureus* and CoNS were cured of their resistant markers showing that their R-markers may have been borne on plasmid. However, resistance to ciprofloxacin and ceftriaxone for CoNS may have been borne on chromosome as all the resistant markers were not cured. The percentage of *S. aureus* and CoNS cured of their R-plasmid were similar (p=0.25). Plasmids ranging from 2.03kbp to 23.13kbp were harboured by both *S. aureus* and CoNS.

**Conclusion**: There is widespread antimicrobial resistance patterns and diverse plasmid profile of *S. aureus* and CoNS in this study. This data will be a useful baseline for further epidemiological investigations.

**Keywords**: Plasmid, *Staphylococcus*, susceptibility, antimicrobials, resistance, curing

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## Introduction

Staphylococci are Gram-positive, facultative anaerobes, non-motile asporogenous spherical bacteria that characteristically divide in more than one plane to form irregular cluster with diameter ranging from 0.5 – 1.5um [1-3]. Staphylococci are widely distributed in various niches such as clinical environments and food plants. Species are classified as coagulase positive (e.g. *Staphylococcus aureus)* and coagulase-negative staphylococci (e.g. *Staphylococcus epidermidis* and *Staphylococcus saprophyticus*.)

*Staphylococcus aureus (S. aureus)* is an opportunistic pathogen affecting both immunocompetent and immunocompromised individuals frequently resulting in high morbidity and complications which constitute problems to health care institutions [4]. It is associated with a variety of clinical infections including septicemia, pneumonia, wound sepsis, septic arthritis, post-surgical toxic shock syndrome as well as scalded skin syndrome in humans [5, 6]. *S. aureus* is the commonest caused of infections in the hospitals and is most liable to infect newborn babies, old and malnourished persons, patients with diabetes and other chronic diseases [7]. *S. aureus* has a record of developing resistance quickly and successfully to antibiotics and has overcome many of the therapeutic agents that have been developed in the past 50years [8].

Coagulase-negative Staphylococci (CoNS) previously dismissed as contaminants are now emerging as important potential pathogens and are one of the main causal agents of bacteraemia in patients with indwelling medical devices [9]. In the last two decades, CoNS have also emerged as significant pathogens, especially in immunocompromised patients, premature newborns, prosthetic-valve endocarditis, surgical wound, central nervous system shunt infections, intravascular catheter-related infections, peritoneal dialysis-related infections, urinary tract infections, arthritis, and infections of prosthetic joints [10]. The cost related to infections caused by them in the hospital settings are enormous and represents a major health care burden [11].

Multiple resistances by microorganisms to antibiotic may be an indication of the presence of resistance factors such as R-plasmids which contain one or more resistance genes, each encoding a single antibiotic resistance

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phenotype [12]. It has been shown that R-plasmid mediated antibiotic resistance can spread in a population subjected to heavy antibiotic therapy [13]. Furthermore, the use of antibiotics perpetuated antibiotic resistant plasmids in communities such as Nigeria where there is unrestricted use of antimicrobial.

The staphylococci appear to become drug resistant more readily than most other bacteria. The appearance of drug resistant strains isolated from pathologic processes have followed the introduction of various antibiotics into general use and the proportion of resistant strains found have continuously increased.

At present, *Staphylococcal* resistance to antibiotic has been associated with resistant plasmids (R-plasmid) that have the ability to mediate the production of drug inactivated enzymes such as β-lactamase [13] and other related functions [14, 15]. Plasmids allow the movement of genetic material, including antimicrobial resistance genes between bacterial species and genera. Plasmid profiles determination is the earliest DNA-based method used as serotype-specific reference patterns for detecting certain strain with possible variation in plasmid content which is very important in epidemiological studies [16, 17]. This study was therefore carried out to determine the antibiotic susceptibility pattern and plasmid-mediated resistance of *Staphylococcus aureus* and Coagulase negative Staphylococci from clinical samples.

**Materials and Methods**

*Isolation of bacterial species*

Forty five and fifteen strains of *S. aureus* and CoNS respectively isolated by standard procedures [18] from 100 clinical specimens sent to Medical Microbiology Laboratory of University of Benin Teaching Hospital (UBTH) were evaluated in this study.

*Preparation of Inoculum*

The overnight cultures of the test organisms were inoculated onto peptone water and vortex thoroughly. The turbidity of the bacterial suspensions were then adjusted and compared with 0.5 McFarland standard.

*Antibiotic Susceptibility Testing*

The antibiotic susceptibility pattern of the bacterial species isolated was performed on Muller-Hinton agar (MHA) plates by disk diffusion method as described by the Clinical and Laboratory Standards Institute (CLSI) with slight modification. Each test organism suspension showing the turbidity equivalent to that of a 0.5 McFarland standard was transferred onto dried agar plates in duplicate and was spread evenly with a sterile cotton swab dipped into suspension, rotated several times, gently pressed onto the inside wall of the tube above the fluid level to remove excess inoculum from the swab. The swab is then streaked over the entire surface of the Agar plate three times with plate rotated approximately 60° each time to ensure even distribution of the inoculum. A final sweep of the swab is made around the Agar rim [19]. The appropriate antibiotic disks containing the following antibiotics: ciprofloxacin (30µg), ceftriaxone (30µg), ofloxacin (25µg), amoxicillin-clavulanic acid (25µg), cefuroxime (30µg), gentamycin (10µg), ceftazidime (30µg), cloxacillin (10µg) and cefixime (30µg) were aseptically placed on the agar surface using sterile forceps and the plates were incubated at 37°C for 24hrs. The degree of susceptibility of the test isolates to each antibiotic was interpreted as either sensitive (S) or resistant (R) based on CLSI guidelines.

*Determination of Multiple Antibiotic Resistance (MAR) Index*

Multiple antibiotic resistance (MAR) index was determined using the formula MAR=$x/y$, where $x$ is the number of antibiotics to which test isolate displayed resistance and $y$ is the total number of antibiotics to which the test organism has been evaluated for sensitivity [18].

*Plasmid curing*

In other to determine the location (Plasmid-borne or chromosomal) of the drug resistance marker(s), curing experiments were performed using 10% sodium dodecyl sulphate (SDS). For this purpose, 0.2ml of overnight culture was added to 5ml nutrient broth containing 10% SDS and incubated at 37°C. After 24hrs, the broth cultures were agitated to homogenize the content and were sub-cultured unto Mueller-Hinton agar (MHA) plates. The plates were incubated at 37°C for 24hrs after which colonies were screened for antibiotic resistance by the disk diffusion method. Cured markers were determined by comparison between the pre- and post-curing antibiogram of isolates [20].

*Plasmid DNA isolation*

The plasmid DNA isolation was carried out in Obong-Lahor laboratory, Benin City, Nigeria. Selected strains of *S. aureus* and CoNS that were cured of their resistance markers were subjected to plasmid DNA isolation according to the protocol of Birboim and Doly [21]. The isolated DNA extracts were subjected to Agarose gel electrophoresis on 0.8% (w/v) agarose gel in a 0.5x concentration of Tris-borate EDTA (TBE) buffer, stained with ethidium bromide. After electrophoresis, plasmid DNA bands were viewed by fluorescence of bound ethidium bromide under a short wave UV light transilluminator and the DNA bands were matched with those for lambda DNA Hind III digest molecular weight markers. The approximate molecular weight of each plasmid was consequently obtained by extrapolation on graphical plots of molecular weight of marker against the distance travelled by the respective band [18, 22].

*Statistical analysis:*
Mann-Whitney statistics as used to test for significant differences in the data obtained. All statistical analyses were carried out using the SPSS 17.0 window based program. Significance difference was defined as p? 0.05.

**Results**
The frequency of occurrence of *S. aureus* and CoNS from the clinical samples showed that the highest percentage of these isolates were collected from urine samples and the least percentage of isolates were recovered from high vaginal swabs and wound swab for *S. aureus* and CoNS respectively (Table 1).

Table 1: Frequency of occurrence of *Staphylococcus aureus* and Coagulase negative Staphylococci from clinical samples

| Clinical samples | Staphylococcus aureus, n (%) | Coagulase negative Staphylococci, n (%) |
|---|---|---|
| Urine | 21(46.7) | 4(36.4) |
| Wound swab | 6(13.3) | 1(9.1) |
| Urethral swab | 4(8.9) | - |
| Blood | 4(8.9) | 2(18.2) |
| Endocervical swab | 3(6.7) | 3(27.3) |
| Ear swab | 4(8.9) | 3(27.3) |
| High vagina swab | 1(2.2) | - |
| Pus | 2(4.4) | 2(18.2) |
| Total | 45(45) | 15(15) |

Table 2: Antibiotic susceptibility profile and multiple antibiotic resistances index of *Staphylococcus aureus* and Coagulase negative Staphylococci

| | *Staphylococcus aureus* | | | | Coagulase negative Staphylococci (CoNS) | | | |
|---|---|---|---|---|---|---|---|---|
| Antibiotics | Sensitive n(%) | Resistance n(%) | MAR Index[a] | | Sensitive n(%) | Resistance n(%) | MAR Index[b] | |
| | | | MAR | n(%) | | | MAR | n(%) |
| Ceftriaxone | 33(73.3) | 12(26.7) | 0.33 | 1(2.22) | 10(66.7) | 5(33.3) | 0.33 | 0 |
| Ofloxacin | 26(57.8) | 19(42.2) | 0.44 | 12(26.7) | 3(20) | 12(80) | 0.44 | 0 |
| Ciprofloxacin | 19(42.2) | 26(57.8) | 0.56 | 11(24.4) | 10(66.7) | 5(33.3) | 0.56 | 3(20) |
| Gentamycin | 24(31.1) | 31(68.9) | 0.67 | 5(11) | 1(6.7) | 14(93.3) | 0.67 | 9(60) |
| Cloxacillin | 16(35.6) | 29(64.4) | 0.78 | 14(31.1) | 3(20) | 12(80) | 0.78 | 3(20) |
| Cefuroxime | 2(4.4) | 43(95.6) | 0.89 | 2(4.4) | 1(6.7) | 14(93.3) | 0.89 | 0 |
| Ceftazidime | 2(4.4) | 43(95.6) | Total | 45(100) | 3(20) | 12(80) | Total | 15(100) |
| Cefixime | 30(66.7) | 15(33.3) | | | 8(53.3) | 7(46.7) | | |
| Amoxicillin-clavulanic acid | 13(28.9) | 32(71.1) | | | 5(33.3) | 10(66.7) | | |

MAR= multiple antibiotic resistance; p = 0.19 for a vs b

The antimicrobial susceptibility of *Staphylococcus aureus* and CoNS as shown in Table 2 revealed that *S. aureus* was highly resistant to cefuroxime and ceftazidime at the same rate (95.6%), followed by amoxicillin-clavulanic acid (71. 1%), while CoNS were resistant to cefuroxime and gentamycin at the same rate (93.3%), closely followed by ofloxacin, ceftazidime, and cloxacillin at the same rate (80%). The result further showed that there is no significant difference between the resistance and sensitivity of *S. aureus* to all the antibiotics (p=0.38). However, the resistance traits exhibited by CoNS to all the antibiotics was significantly higher than its sensitivity (p=0.01). Moreover, resistance to all the antibiotics by both *S. aureus* and CoNS showed no significant difference (p=0.17). In this study, the most effective antimicrobial agent against both *S. aureus* and CoNS was ceftriaxone as only 26.7% of *S. aureus* and 36.4% of CoNS were resistant to it. The multiple antibiotic resistance (MAR) indexes of *S. aureus* and CoNS are also shown in Table 2. The antibiotic resistant *S. aureus* have MAR index of 0.33 to 0.89, while the antibiotic resistant CoNS have MAR index of 0.56 to 0.78. The findings also revealed that 24.4% of *S. aureus* and 20% of CoNS were resistant to at least five antibiotics. There is no significance difference between the MAR indexes of *S. aureus* and CoNS (p=0.19)

Table 3 shows the antimicrobial resistant pattern of *S. aureus* and CoNS. Twenty (20) and 8 resistance pattern was shown by *S. aureus* and CoNS respectively. The most common resistance pattern for *S. aureus* was Ciprofloxacin (*cip), cefuroxime (cxm),* ceftazidime *(caz),cloxacillin (clo)* closely followed by amoxicillin-clavulanic acid (*amc*), *cefuroxime (cxm),gentamycin (gm),ceftazidime (caz),cloxacillin (clo)*. In the same vein, the most common resistance pattern for CoNS was ofloxacin (*ofx),* amoxicillin-clavulanic acid *(amc), cefuroxime (cxm), gentamycin (gm), ceftazidime (caz), cloxacillin (clo)* and ceftriaxone (*ctx), ofloxacin (ofx), cefuroxime (cxm), gentamycin (gm), cloxacillin (clo), ocefix (cfx)*.

The effects of plasmid curing using 10% SDS on the drug resistance determinants of *S. aureus* and CoNS are depicted in Table 4. Resultantly, some of the resistance markers were stably lost (excluding ciprofloxacin and ceftriaxone for CoNS. In the findings, 77% and 71% *S. aureus* and CoNS respectively were cured of their cefuroxime resistance markers. The result also revealed that the number of *S. aureus* (P=0.03) and CoNS (P=0.02) resistant to the various antibiotics after curing were significantly lower than those before curing. Also, the percentage of *S. aureus* cured of their R-plasmid were not significantly different from those of CoNS (p=0.25).

Table 3: Resistance pattern of *Staphylococcus aureus and Coagulase negative Staphylococci*

| | *Staphylococcus aureus* | | | Coagulase negative Staphylococci (CoNS) | | |
|---|---|---|---|---|---|---|
| S/N | Resistance pattern | No. of antibiotics | No. of isolates | Resistance pattern | No. of antibiotics | No. of isolates |
| 1. | cxm,caz,clo | 3 | 1 | cip,ofx,caz,clo,cfx | 5 | 3 |
| 2. | amc,cxm,caz,cfx | 4 | 1 | ctx,ofx,amc,cxm,gm,caz | 6 | 2 |
| 3. | cip,cxm,caz,clo | 4 | 6 | ofx,amc,cxm,gm,caz,clo | 6 | 2 |
| 4. | amc,cxm,gm,caz | 4 | 2 | cip,amc,cxm,gm,caz,clo | 6 | 1 |
| 5. | cip,ctx,gm,clo | 4 | 2 | amc,cxm,gm,caz,clo,cfx | 6 | 2 |
| 6. | cxm,gm,caz,cfx | 4 | 1 | ctx,ofx,cxm,gm,clo,cfx | 6 | 2 |
| 7. | cip,cxm,gm,caz,clo | 5 | 3 | cip,ofx,amc,cxm,gm,caz,clo | 7 | 1 |
| 8. | amc,cxm,gm,caz,clo | 5 | 5 | cip,ofx,cxm,gm,caz,clo,cfx | 7 | 2 |
| 9. | ofx,amc,cxm,caz,clo | 5 | 2 | | | |
| 10. | amc,cxm,gm,caz,cfx | 5 | 1 | | | |
| 11. | cip,ofx,amc,cxm,gm,caz | 6 | 1 | | | |
| 12. | cip,amc,cxm,caz,clo,cfx | 6 | 2 | | | |
| 13. | ctx,amc,cxm,gm,caz,clo | 6 | 2 | | | |
| 14. | cip,ofx,amc,cxm,gm,caz,clo | 7 | 2 | | | |
| 15. | ofx,,amc,cxm,gm,caz,clo,cfx | 7 | 2 | | | |
| 16. | cip,ctx,ofx,amc,cxm,gm,caz | 7 | 4 | | | |
| 17. | cip,ofx,amc,cxm,caz,clo,cfx | 7 | 2 | | | |
| 18. | ctx,ofx,amc,cxm,gm,caz,cfx | 7 | 2 | | | |
| 19. | cip,ofx,amc,cxm,gm,caz,cfx | 7 | 2 | | | |
| 20. | cip,ctx,ofx,amc,cxm,gm,caz,cfx | 8 | 2 | | | |

Key: cip=ciprofloxacin, ctx=ceftriaxone, ofx=ofloxacin, amc=amoxicillin-clavulanic acid, cxm=cefuroxime, gm=gentamycin, caz=ceftazidime, clo=cloxacillin, cfx=cefixime.

The results of the plasmid profile of selected strains as shown in Table 5 revealed that both *S. aureus* and CoNS that were cured of their R-markers harboured plasmid bands of different molecular size. The plasmid bands ranges from 2.03 – 23.13kb with 23.13kb plasmid being the most abundant.

Table 4: Plasmid curing analysis of resistant *Staphylococcus aureus* and Coagulase negative Staphylococci

| | *Staphylococcus aureus* | | | *Coagulase negative Staphylococci (CoNS)* | | |
|---|---|---|---|---|---|---|
| Antibiotics | No. resistant (pre-curing)[b] | No. resistant (post-curing)[c] | No.(%) cured[a] | No. resistant (pre-curing)[d] | No. resistant (post-curing)[e] | No.(%) cured[a] |
| Ceftriaxone | 12 | 6 | 6(50) | 5 | 5 | 0 |
| Ofloxacin | 19 | 14 | 5(26) | 12 | 8 | 4(33) |
| Ciprofloxacin | 26 | 23 | 3(12) | 5 | 5 | 0 |
| Gentamycin | 31 | 23 | 8(26) | 14 | 8 | 6(43) |
| Cloxacillin | 29 | 21 | 8(28) | 12 | 5 | 7(58) |
| Cefuroxime | 43 | 10 | 33(77) | 14 | 4 | 10(71) |
| Ceftazidime | 43 | 21 | 22(51) | 12 | 5 | 7(58) |
| Cefixime | 15 | 12 | 3(20) | 7 | 6 | 1(14) |
| Amoxicillin-clavulanic acid | 32 | 23 | 9(28) | 10 | 6 | 4(40) |

a (not significant different, P= 0.25), b and c (significantly different, P=0.03), d and e (significantly different, P=0.02)

Table 5: Plasmid profile analysis of *Staphylococcus aureus* and Coagulase negative Staphylococci

| Code no. of isolate | No. of plasmid | Plasmid size (kbp) |
|---|---|---|
| Sa 5 | 1 | 23.13 |
| Sa 7 | 1 | 23.13 |
| Sa 9 | 1 | 2.03 |
| Sa 14 | 1 | 9.42 |
| Sa 15 | 1 | 6.56 |
| Sa 16 | 1 | 23.13 |
| Sa 20 | 1 | 2.03 |
| Sa 21 | 1 | 23.13 |
| Sa 27 | 1 | 23.13 |
| Sa 40 | 1 | 23.13 |
| CoNS 10 | 1 | 2.03 |
| CoNS 12 | 1 | 23.13 |
| CoNS 13 | 1 | 9.42 |
| CoNS 3 | 1 | 23.13 |
| CoNS 6 | 1 | 23.13 |

Key: Sa= *S. aureus*, CoNS= Coagulase negative staphylococci

## Discussion

Multiple drug resistant *S. aureus* and CoNS are recognized as a major cause of nosocomial infections

with resultant significant morbidity and mortality rates [23]. Coagulase negative Staphylococci have become one of the predominant pathogens in hospitalized patients and the number of infections caused by these organisms is on the increased [24, 25].

The result of the antimicrobial susceptibility pattern showed that *S. aureus* was highly susceptible to ceftriaxone, cefixime and ofloxacin at different rate, while CoNS were also susceptible to ceftriaxone and ciprofloxacin at the same rate. This finding is closely similar to earlier studies. [18] Another study revealed that *S. aureus* were highly susceptible to gentamycin, ofloxacin and ciprofloxacin [26]. Their result is almost similar to the findings of this study with respect to ofloxacin, but is contrary to this study with respect to gentamycin and ciprofloxacin.

*S. aureus* exhibits remarkable versatility in their behaviour towards antibiotics and its capacity to produce human diseases had not diminished even with introduction of antibiotics [27]. *S. aureus* has been reported to exhibit resistance to beta-lactam antibiotics. In this study however, *S. aureus* is sensitive to ceftriaxone (73.3%) and cefixime (66.7%), but highly resistant to cefuroxime and ceftazidime (95.6%) (both beta-lactam antibiotics). Bacterial resistance to beta-lactam antibiotics is primarily due to the production of β-lactamase which splits the ring of the antibiotics rendering them inactive [28]. *S. aureus* resistant to beta-lactam antibiotics have been frequently associated with devastating nosocomial infections [29, 30]. Other resistant profile of *S. aureus* in this study includes amoxicillin-clavulanic acid (71.1%), gentamycin (68.9%), cloxacillin (64.4) and ciprofloxacin (57.8%). Various studies have reported *S. aureus* resistance to antibiotics such as cloxacillin and gentamycin [31], amoxicillin-clavulanic acid [32], gentamycin and amoxicillin-clavulanic acid [13], ciprofloxacin and gentamycin [33] and cloxacillin [34].

Coagulase negative Staphylococci have been reported to be resistant to a wide spectrum of antibiotics [17]. The findings that CoNS were resistant to cefuroxime and gentamycin (93.3%), ofloxacin, ceftazidime and cloxacillin (80%), amoxicillin-clavulanic acid (66.7%) is almost similar to previous study [35]. Another study also showed that CoNS were resistant to cloxacillin, gentamycin and ofloxacin [36]. The multiple antibiotic resistance (MAR) index, when applied to a single isolate, is defined as a/b, where "a" represents the number of antibiotics to which the isolate was resistant and "b" represents the number of antibiotics to which the isolate was exposed. MAR index higher than 0.2 identifies organisms that originate from high-risk sources of contamination, where antibiotics are often used. MAR indices less than, or equal to 0.2, identify strains from environments where antibiotics are seldom or never used [37]. In this study, *S. aureus* has MAR index of 0.33 to 0.89, while CoNS has MAR index of 0.56 to 0.78. The MAR indices of these organisms are higher than 0.2 *which indicate* that a very large proportion of the bacterial isolates have been exposed to several antibiotics. Inappropriate practices like misuse and abuse of antibiotics by unskilled practitioners can lead to emergence of resistance in bacteria. Degraded antibiotics, self-medication, fake drugs, inadequate hospital control measures can as well promote the development of resistance in clinical isolates [38, 39]. In developing countries like Nigeria, self medication is a common practice and this might probably be a major cause of antibiotic resistance in clinical isolates since patients only think of going to the hospitals when they are unable to treat themselves [39].

Plasmid replication is inhibited by various agents especially sodium dodecyl sulphate that intercalates between the bases of DNA, without inhibiting the chromosomal DNA replication. In order to confirm the location (chromosomal or plasmid) of the antibiotic resistant markers, plasmid curing analysis were conducted using 10% sodium dodecyl sulphate (SDS). Although curing provides only the preliminary evidence that genetic traits are of extrachromosomal nature, but loss of growth on antibiotic containing plates shows that the multidrug resistant genes may be plasmid-borne. In this study, some of the resistant markers were stably lost in some isolates. Shahid and Malik [40] showed that after curing, loss of antibiotic resistance was concomitant with the lost of plasmid content. Thus, resistance by these isolates may have been borne on plasmid. All ciprofloxacin and ceftriaxone resistance markers on CoNS were not lost, thereby showing the chromosomal location of these two markers.

Resistances to high levels of antibiotics have been associated in most instances to the presence of plasmids [41- 43]. In this study, the most commonly encountered plasmids were of size 23.13kb. This is similar to earlier reports [18, 22, 41 and 44]. The emergence of resistant plasmids in this study could be due to overzealous desire to treat every infection, diagnosed or not and to the over the counter availability of antibiotics [43, 45, 46]. Plasmid profile analysis has been shown to be a good epidemiological tool in investigating epidemics or outbreaks of bacterial resistance [47].

**Conclusion**

In conclusion, this study highlighted widespread antimicrobial resistance patterns and diverse plasmid profile of *S. aureus* and CoNS from a tertiary hospital in Nigeria. It is hoped that this information will be a useful baseline for further epidemiological investigations. There is also the need for consistent on-going

antimicrobial resistance surveillance for important and commonly isolated clinically significant pathogens of staphylococcal species to form the basis for developing and implementing measures that can reduce the burden of antimicrobial resistance and prevent a probable impending public health burden.

**Competing Interest**

Authors have declared that there is no competing interest.

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