



Research Article

Detection of *mecA* and *mecC* genes among Methicillin-Resistant *Staphylococcus aureus* from Wounds of Patients Attending Selected Hospitals in Kaduna, Nigeria

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ABSTRACT

Phenotypically identified Methicillin-Resistant *Staphylococcus aureus* (MRSA) that lacks *mecA* or *mecC* raises questions about the molecular basis of the observed resistance and the reliability of molecular detection. Present study detects *mecA* and *mecC* gene among MSRA from wound of patients attending selected hospitals in Kaduna State, Nigeria. A cross-sectional laboratory based study was conducted between May and December 2025. Two hundred wound swab samples comprising surgical, ulcer, scald and burn wounds were collected from consenting patients attending the selected hospitals. *S. aureus* was isolated and identified based on standard morphological and biochemical procedure. Phenotypic detection of MRSA was performed by disk diffusion method using cefoxitin (30 µg). Ten (10) MRSA isolates were randomly selected for detection of *mecA* and *mecC* genes. A total of 78 *S. aureus* were recovered from 200 wound swabs with ulcer wounds having the highest occurrence of *S. aureus* (52.27%), followed by surgical wounds (42.35%), scald wounds (28.57%) and burn wounds (25.58%). There is significant statistical association ($p = 0.04216$) between the occurrence of *S. aureus* and type of wound. Of the 78 *S. aureus* isolates, 61 (78.21%) were phenotypically identified as MRSA. PCR results showed that 7 of 10 MRSA isolates screened carried *mecA* gene and none of the 10 MRSA screened carried *mecC*. The fact that 3 phenotypically identified MRSA lacks *mecA* and no isolates had *mecC* shows that methicillin resistance cannot be always explained by these two genes. A combination of phenotypic and molecular methods is recommended for accurate detection of MRSA.

Keywords: *mecA*; *mecC*; MRSA; PCR; *S. aureus*

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INTRODUCTION

Staphylococcus aureus is an important bacterial pathogen associated with wound infection. It may employ several adaptive strategies, including antimicrobial resistance in order to persist in wound infection. Prior to 1961, *S. aureus* isolates were known to be susceptible to methicillin and related antibiotics, today resistance to these drugs has been a remarkable success achieved by *S. aureus* (Hiramatsu *et al.*, 2014). Formerly, methicillin resistance in *S. aureus* has been attributed to *mecA* gene alone, which encodes modified penicillin

binding protein (PBP2a) and its carried on mobile genetic element known as staphylococcal cassette chromosome *mec* (*SCCmec*) (Muller & Andam, 2025). In year 2011, a novel methicillin resistance gene (*mecC*) was detected from MRSA of animal origin and later found in human (Patersen *et al.*, 2013; Deplano *et al.*, 2014). Another methicillin resistance gene (*mecB*) which is plasmid borne, found to be originated from *Micrococcus spp* and later documented in *S. aureus* (Becker *et al.*, 2018). Other molecular mechanisms such as hyper production of beta lactamase, point mutations and deletions within

SCCmec, amino acid substitution in normal penicillin binding protein 2 (PBP2) may also account for methicillin resistance mechanism in *S. aureus* (Umar *et al.*, 2023; Li *et al.*, 2025). Although, antibiotic susceptibility testing which serve as phenotypic detection of MRSA is routinely performed in clinical laboratories, there is still a problem of molecular basis of phenotypically identified MRSA from wounds of patients in Kaduna State, Nigeria. In particular, local evidence on the expression of methicillin resistance by *S. aureus* and presence of *mecA* and *mecC* remains limited. The identification of phenotypically identified MRSA that lacks *mecA* and *mecC* also raises an important problem as to whether such isolates uses alternative resistance mechanism. Therefore, the present study screened for the presence of *mecA* and *mecC* genes among MRSA from wound of patients attending selected hospitals in Kaduna State, Nigeria.

MATERIALS AND METHODS

Study Area

The study area is Kaduna State, Nigeria. The selected hospitals include a referral hospital and 2 general hospitals namely; Ahmadu Bello University Teaching hospital, Shika, Nigeria, Hajiya Gambo Sawaba General Hospital and Yusuf Dantsoho Memorial Hospital, Kaduna, Nigeria. The hospitals were chosen based on their relevance to the research objective, availability of relevant patient population and the approval given to collect wound swab for the study.

Ethical Approval and Patients' Consent

Ethical approval was granted to this study by the Health Research Ethics Committees (HRECs) of Kaduna State Ministry of Health (MOH/ADM/744/VOL.1/1110031) and Ahmadu Bello University Teaching Hospital, Shika-Zaria, Nigeria (NHREC/ABUTH-HREC/29/08/23). Informed consent was obtained from all individual participant recruited into the study.

Collection of Wound Swabs

A total of 200 wound swab specimens involving surgical wound, ulcer wound, scald and burn wound were collected from consenting patients attending the selected hospitals from May to December 2025.

Isolation and Identification of *Staphylococcus aureus*

All collected wound swabs were inoculated onto the surface of prepared Mannitol salt agar plates, using streak plate method. The streaked plates were then incubated at 37°C for 48 hours. To obtain pure isolates, distinct well separated yellow colonies were sub-cultured onto prepared nutrient agar (NA) plates incubated at 37°C for 24hrs. Stock isolates were made

in nutrient agar slants and kept at 4°C in a refrigerator. *S. aureus* isolates were identified based on their morphological and biochemical characteristics using Gram staining, Catalase test, Coagulase test, DNase test, Novobiocin sensitivity test, Haemolysis on blood agar and sugar fermentation tests (Cheesbrough, 2004; Shrestha *et al.*, 2017 and Alabi *et al.*, 2022).

Phenotypic Detection of Methicillin Resistant *Staphylococcus aureus*

Phenotypic screening of methicillin resistant *Staphylococcus aureus* was carried out by agar disc diffusion technique following CLSI (2024) guidelines. Briefly, Mueller-Hinton Agar was prepared and sterilized in an autoclave set at 121°C for 15 minutes. A standard inoculum was prepared from well isolated 24 hour colonies by suspending colonies in 4 mL physiological saline and adjusting the turbidity to a 0.5 McFarland standard. Using sterile swab stick, inoculum of the test organism was obtained and the excess fluid were removed, and then swabbed evenly onto the surface of a well prepared and dried Mueller Hinton Agar. Cefoxitin disk (30 µg) was placed onto the prepared Mueller-Hinton Agar and incubated at 35°C for about 18 hours. Isolates exhibiting a zone of inhibition ≤ 21 mm surrounding the cefoxitin disk were classified as MRSA isolates (CLSI, 2024).

Detection of *mecA* gene and *mecC* gene by Polymerase Chain Reaction Technique.

Ten (10) isolates identified as MRSA by disc diffusion using cefoxitin (30 µg) disks were randomly chosen for PCR detection of *mecA* and *mecC*. The procedure involved is outlined below:

DNA extraction

Crude genomic DNA for PCR was extracted from the isolates using the heat lysis method. Colonies from overnight culture of the isolates were transferred into Eppendorf tubes containing 1 mL of nuclease-free water and boiled at 100°C for 5 minutes in a heating block and subsequently frozen at -4°C for 7 mins. The preparation was centrifuged at 12,000 rpm for 5 minutes (Espinosa *et al.*, 2013). The supernatant were pipetted out into a sterile Eppendorf tubes and used as the DNA template for PCR.

PCR amplification

Standard PCR assay was performed using the primers set (Table 1). A volume of 1 µL of prepared DNA (0.5 µg) was added to a final volume of 25 µL PCR mixture containing 10 µL of 2X Master Mix (Ampliqon, Denmark), including 1X PCR buffer, 1.5 mmol/L MgCl₂, 0.15 mmol/L dNTP, and 1.25 IU *Taq* DNA polymerase, (Ampliqon Co., Denmark), 0.7 µL of 0.8 µmol/L each primer and 12.6 µL of sterile nuclease

free water. The thermal cycling protocol for PCR comprises 95°C for 3 min, followed by 33 cycles of 94°C for 1 min, 53°C for 30 s and 72°C for 1 min, then a final extension at 72°C for 6 min.

Detection of PCR product

The amplified products were visualized by electrophoresis in 2% agarose gel stained with ethidium bromide as described by Abdalla *et al.* (2014).

Table 1: Primer sequence used for the detection of *mecA* and *mecC* genes

Primer	Sequence (5' - 3')	Amplicon size	Reference
<i>mecA</i> (F)	TCCAGATTACAACCTCACCAGG	162 bp	Stegger <i>et al.</i> (2012)
<i>mecA</i> (R)	CCACTTCATATCTTGTAAACG		Cikman <i>et al.</i> (2019)
<i>mecC</i> (F)	GAAAAAAGGCTTAGAACGCCTC	138 bp	Stegger <i>et al.</i> (2012)
<i>mecC</i> (R)	GAAGATCTTTCCGTTTTCAGC		Cikman <i>et al.</i> (2019)

Table 2: PCR Amplification Conditions

Step	Stage of PCR	Temperature	Time
1	Initial denaturation	95°C	3 minutes
2	Denaturation	94°C	1 minute
3	Annealing	53°C	30 seconds
4	Extension	72°C	1 minute
5	Final extension	72°C	6 minutes

RESULT

Distribution of *Staphylococcus aureus* isolates based on type of wound

A total of 78 *S. aureus* isolates were identified with ulcer wounds having the highest percentage with 52.27% (n = 23/44), followed by Surgical wounds 42.35% (n = 36/85). Scald wound had 28.57% (n = 8/28) and burn wounds had the least percentage, 25.58% (n = 11/43). The relationship is statistically significant (p = 0.04216) (Table 3).

Occurrence of Methicillin Resistant *Staphylococcus aureus* by disc diffusion

Out of the 78 *Staphylococcus aureus* tested using cefoxitin (30 µg), 61 were identified as Methicillin Resistant *Staphylococcus aureus* with proportion of 78.21% among the *S. aureus* isolates (Figure 1).

Occurrence of *mecA* and *mecC* genes among Methicillin Resistant *Staphylococcus aureus*

PCR results of ten (10) randomly selected MRSA isolates were presented in Table 4. The Gel electrophoregram revealed the presence of *mecA* gene at 162 base pairs in lanes 1, 2, 5, 6, 7, 8 and 9, while lanes 3, 4 and 10 showed no amplification of the *mecA* gene (Plate II). There was no amplification of *mecC* gene in any of the isolate screened (Plates III).

Table 3: Distribution of *Staphylococcus aureus* isolates based on type of wound

Type of Wound	No. of Sample	No. of Isolates (%)	χ^2	Df	p-Value
Ulcer	44	23 (52.27)	8.1944	3	0.04216
Surgical	85	36 (42.35)			
Burn	43	11 (25.58)			
Scald	28	8 (28.57)			
Total	200	78 (39)			



Plate 1: Muller-Hinton agar plate showing zone of inhibition of cefoxitin (30 µg) disk against *S. aureus*

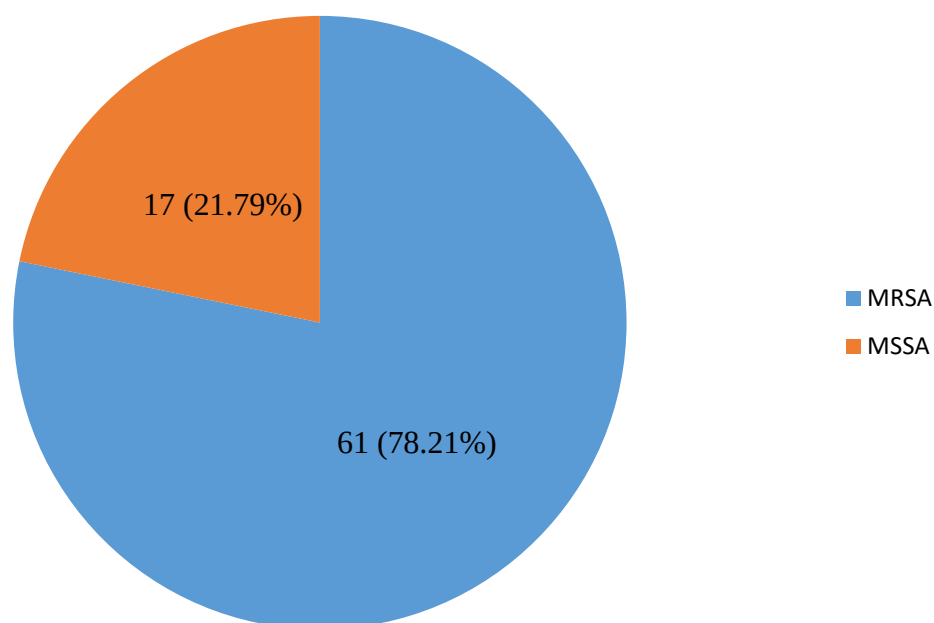


Fig. 1: Proportion of Methicillin Resistant *Staphylococcus aureus* among *S. aureus*

Table 4: PCR results for the selected MRSA isolates screened

Isolate code	Phenotype	<i>MecA</i>	<i>MecC</i>
Y14	Resistant	+ve	-ve
H1	Resistant	+ve	-ve
A68	Resistant	-ve	-ve
H2	Resistant	-ve	-ve
A1	Resistant	+ve	-ve
Y2	Resistant	+ve	-ve
Y5	Resistant	+ve	-ve
A37	Resistant	+ve	-ve
A16	Resistant	+ve	-ve
H11	Resistant	-ve	-ve

Keys: A- Ahmadu Bello University, Teaching Hospital, Shika, Nigeria. Y- Yusuf Dantsoho Memorial Hospital, Tudun Wada Kaduna, H- Hajiya Gambo Sawaba General Hospital, Zaria Kaduna, +ve – positive, -ve – negative

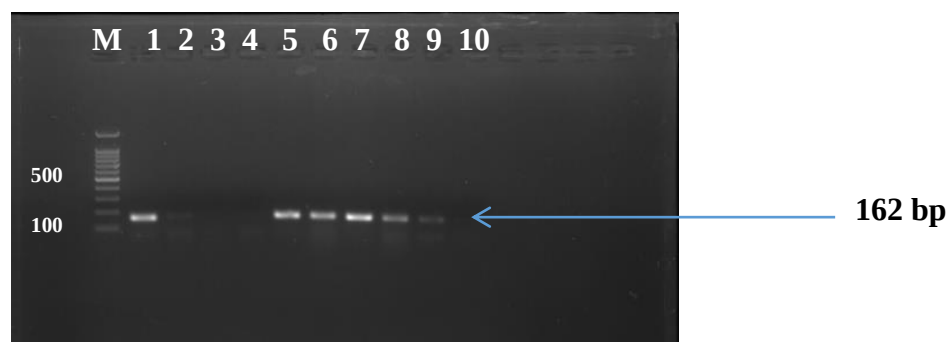


Plate II: Agarose Gel Photo of *mecA* gene (162bp) detected in Methicillin-Resistant *Staphylococcus aureus*

Keys: Lane M: Molecular Marker; Lanes: 1, 2, 5, 6, 7, 8 and 9 (positive for *mecA*); Lanes: 3, 4 and 10 (Negative for *mecA*)



Plate III: Gel Electrophoregram of *mecC* gene (138bp) not detected in Methicillin-Resistant *Staphylococcus aureus*

Keys: Lane M: Molecular Marker; Lanes: 1-10: (Negative for *mecC*)

DISCUSSION

Methicillin-Resistant *Staphylococcus aureus* (MRSA) remains one of the leading pathogen involved in wound infection (Chen *et al.*, 2026). In our study, the percentage occurrence of *S. aureus* was highest among ulcer wound (52.27%; 23/44), followed by surgical and scald wounds with 42.35% (36/85) and 28.57% (8/28) respectively. The least was 25.58% (11/43) from burn wound. The occurrence of *S. aureus* observed in ulcer wound is comparable with findings reported from other studies (Abdulaziz *et al.* 2022; Anweke, 2026). Studies on burn wounds have shown great variation with the reports on occurrence of *S. aureus* in the range of 1.37% to 72% (Bature *et al.*, 2021; Abdulaziz *et al.*, 2022; Sampson *et al.*, 2022; Anweke, 2026). Similarly, studies on surgical wound have reported varying rates of *S. aureus* occurrence in the range of 5.48% to 33.3% (Abdulaziz *et al.*, 2022; Sampson *et al.*, 2022). Regardless of the types of wound, infection by *S. aureus* may be influenced by frequent exposure to contaminated environment, poor wound management, as well as patients' characteristics such as immunosuppression (Hussain *et al.*, 2005; Bature *et al.*, 2021). The chi square test

showed statistical association ($p = 0.04216$) between types of wound and occurrence of *S. aureus* with ulcer and surgical wounds more associated with it occurrence.

In the present study, occurrence of MRSA was 78.21% (61/78), while MSSA account for 21.79% (17/78). This proportion was higher compared to reports from other studies. A recent study has reported 23.7% and 76.3% of MRSA and MSSA occurrence respectively (Baba *et al.*, 2026). Another study also reported 27.5% and 72.5% in respect of MRSA and MSSA occurrence (Omosigho *et al.*, 2026). In Rwanda, similar study reported the occurrence of MRSA and MSSA in tertiary hospital with 48% and 52% respectively (Usengimana *et al.*, 2026). Similarly, a study in India recorded 42% and 58% of MRSA and MSSA occurrence respectively (Manem & Kumar, 2026). In contrast, the proportion observed in the present study was lower than the 90.35% MRSA reported from Pakistan (Ilyas *et al.*, 2026). Thus, the studies collectively demonstrate substantial geographical and clinical variation rather than a consistent regional pattern. Several factors may account for differences observed among the studies

including antibiotic misuse, patients' characteristics, living conditions, study duration or differences in sample size.

MecA is widely used as a major molecular marker for methicillin resistance in *S. aureus*, while *mecC* occurs less frequently in many clinical isolates (Stegger *et al.*, 2012; Idrees *et al.*, 2023). In the present study, *mecA* was detected in seven of the ten MRSA isolates, whereas *mecC* was not detected in any of the isolates. The absence of *mecC* is consistent with reports from Turkey, Kenya and Iraq in which *mecC* was not detected among MRSA isolates (Cikman *et al.*, 2019; Hama *et al.*, 2024; Ogeto *et al.*, 2026). However, other studies have reported *mecC* in a proportion of MRSA isolates, indicating that the distribution of this resistance gene varies geographically and by study population (Idrees *et al.*, 2023; Iliyasu *et al.*, 2026). The detection of *mecA* in seven of ten selected isolates also agrees with reports showing that *mecA* is the predominant molecular determinant identified in many MRSA isolates, while a few phenotypically identified MRSA isolates may lack the gene (Stegger *et al.*, 2012; Cikman *et al.*, 2019; Idrees *et al.*, 2023). Similarly, studies from Nigeria have reported MRSA isolates without detectable *mecA* (Oluduro *et al.*, 2023; Sulaiman *et al.*, 2025; Seriki *et al.*, 2026). The present finding therefore supports the need to distinguish between phenotypically identified MRSA from detection of individual resistance genes.

Across these studies, absence of *mecA* and or *mecC* in MRSA could easily be traced to mutation in the *staphylococcal cassette chromosome mec* (*SCCmec*) which carries the *mec* gene complex (Scharn *et al.*, 2022) or the presence of alternative resistance mechanism such as hyper production of beta-lactamases and amino acid substitution in normal penicillin binding protein 2 (PBP2) which might led to proliferation of borderline oxacillin-resistant *Staphylococcus aureus* (BORSA) and Modified *S. aureus* (MODSA). Presence of other methicillin resistance genes such as *mecB* could also be another factor (Ogeto *et al.*, 2026).

Major limitation of the present study is that detection of *mecA* and *mecC* was performed only on 10 out of 61 MRSA isolates due to paucity of fund. Consequently, the findings may not fully represent the overall occurrence of *mecA* and *mecC* among the MRSA recovered in the study. Future studies involving larger number of isolates and additional methicillin resistance determinants such as *mecB* and other alternative mechanisms of resistance are recommended.

CONCLUSION

In conclusion, the preliminary molecular detection of *mecA* and *mecC* among MRSA recovered from wounds of patients attending selected hospitals in Kaduna state highlight *mecA* as the predominant gene mediating methicillin resistance in *S. aureus* whereas *mecC* remains uncommon in the study population. The observation that some MRSA lacks both *mecA* and *mecC* suggest possible involvement of alternative resistance mechanism and it underscores the needs for broader molecular characterization of MRSA in Kaduna State.

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Authors' Contributions

The study was conducted in collaboration among all the authors and approved the manuscript.

Competing Interest

The authors have no conflict of interest

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