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Detection of the *mecA* gene and methicillin-resistant *Staphylococcus aureus* isolated from inanimate surfaces and healthcare workers in tertiary hospitals in Abia state, Nigeria

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Abstract

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) has become a public health issue. The purpose of this study was to detect MRSA among *S. aureus* isolates and to detect the presence of the *mecA* gene among selected MRSA isolates.

Methods: A total of 206 *S. aureus* isolates identified after the necessary biochemical tests were tested for methicillin resistance using the cefoxitin disk diffusion method. The Kirby-Bauer disk diffusion method was used to determine the antibiotic susceptibility pattern of the isolates. Some MRSA isolates were tested for the presence of the *mecA* gene.

Results: A total of 122 *S. aureus* isolates were recovered from inanimate surfaces, with footwear showing the highest contamination rate (39.34%, n = 48), while 84 *S. aureus* isolates were obtained from the palms of the hands and nostrils of healthcare workers, with the nostrils harboring the highest proportion (83.33%, n = 70). Antibiotic susceptibility testing was carried out on the *S. aureus* isolates. The *S. aureus* isolates were more resistant to ampicillin, erythromycin, cotrimoxazole, and gentamicin. A total of 206 *S. aureus* isolates were tested for MRSA using cefoxitin disk diffusion, and 43.69% (n = 90) were methicillin-resistant. A total of 20 *S. aureus* isolates suspected to be MRSA were tested for the *mecA* gene, and only 4 isolates (20% of the total isolates tested by polymerase chain reaction) were positive for the *mecA* gene.

Conclusion: The presence of MRSA in the two tertiary hospitals poses a risk to healthcare and community environments by contaminating healthcare workers, patients, visitors, and surrounding surfaces.

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Introduction

Infections acquired in hospitals or other healthcare facilities are a significant public health issue (1). In developed nations, the prevalence of healthcare-associated infections (HAIs) is already high, affecting between 5% and 15% of patients admitted to standard wards and up to 50% of critically ill patients in intensive care units (ICUs) (2,3).

It is obvious that monitoring the hospital environment is crucial for preventing nosocomial infections. Although hand-to-hand contact is likely the greatest risk, surface contamination may also serve as a potential source of infection. Multidrug-resistant (MDR) microorganisms may contaminate high-contact communal items, such as telephones and medical charts, as well as the surfaces of frequently used medical equipment, necessitating laborious, expensive, and complicated operations to improve patient safety (4,5). The problem of environmental contamination presents an even greater challenge in the intensive care unit (ICU), where critically ill patients have multiple risk factors for nosocomial infections (6).

This issue becomes especially significant when multiresistant bacteria, such as non-fermenting rods, methicillin-resistant Staphylococcus, and vancomycin-resistant enterococci, arise in hospital settings (7-9).

MRSA, methicillin-resistant *S. aureus*, is among the most prevalent bacterial strains causing hard-to-control infections in hospital settings (10).

Coagulase-positive staphylococci (Such as *S. aureus*) and coagulase-negative staphylococci (*S. epidermidis* and *S. saprophyticus*) are germs that readily contaminate the hospital environment. People with venous catheters, individuals with diabetes, and healthcare workers are more susceptible to *S. aureus*. However, methicillin-resistant *S. aureus* (MRSA) poses the greatest threat because it causes nosocomial infections with exceptionally high rates of morbidity and mortality (11). *S. aureus* was one of the frequently isolated pathogens between 2006 and 2007 linked to nosocomial infections reported to the National Healthcare Safety Network. Of hospital supplies and equipment contaminated with *S. aureus*, 56% involved MRSA. Colonized or infected individuals are the most significant source of MRSA in hospitals because they can readily contaminate nearby electronic and medical equipment. MRSA can persist on dry surfaces for several months (12,13). Although the contaminated hands of healthcare workers are the main route by which MRSA is spread to patients, evidence indicates that patient infections can also result from exposure to MRSA-contaminated surfaces (14,15).

According to evidence from the literature review, *S. aureus* can survive on dry surfaces for anywhere from one week to three years (16). Although there is evidence that *S. aureus* may survive in both home and clinical environments, the dissemination of these organisms, especially MRSA, across the population has received less attention (17). According to certain studies, *S. aureus* may survive on a wide range of inanimate surfaces, including polyethylene for 90 days, sterile packaging for 266 days, screw-cap bottles for 318 days, and polypropylene for more than 1097 days (18). However, these results appear to be based on optimization of the experimental setup. These studies employed an inoculum of 107-109 CFU and staphylococcal strains that were highly resistant to desiccation. Although these findings were clear and consistent in the laboratory, they may not truly represent *S. aureus* survival in the general population. Because of their limited survival on dry surfaces for longer than 24 hours, dangerous and pathogenic *S. aureus* strains, such as MRSA, which cause hospital-associated infections, are rarely isolated from environmental fomites (19).

The epidemiology of MRSA, particularly on inanimate surfaces, has remained understudied in Abia State, Nigeria, and this may have contributed to its increase in hospital-acquired infections. The main aim of this study was to detect MRSA among *S. aureus* isolates and to detect the presence of the *mecA* gene among selected MRSA isolates.

This study was necessary because the majority of isolates recovered from inanimate surfaces, nostrils, and palms of the hands of healthcare workers in tertiary hospitals in Abia State were *S. aureus*.

Methods

This cross-sectional study was carried out in selected hospital wards in Federal Medical Centre, Umuahia, and Abia State University Teaching Hospital, Aba. Ethical approval was obtained from the healthcare facilities before sampling and analysis. Informed consent was obtained from the participating healthcare workers with utmost confidentiality.

The present study focused on 206 *S. aureus* isolates collected from inanimate surfaces, palms of the hands, and nostrils of healthcare workers after laboratory analysis. One hundred and twenty-two *S. aureus* isolates were collected from inanimate surfaces, while 84 *S. aureus* isolates were collected from the palms of the hands and nostrils of healthcare workers. A total of 14 *S. aureus* isolates were collected from the palms of the hands, while 70 *S. aureus* isolates were collected from nostrils. Sterile swab sticks dampened with sterile water were used to swab the surfaces of mattresses, tables, footwear, clinical coats, bed sheets, and pillows. These inanimate surfaces were selected based on their frequent use and direct contact with patients, healthcare workers, and visitors. Sterile swab sticks dampened with sterile water were used to swab the palms of the hands of healthcare workers, and sterile swab sticks were used to swab the nostrils of healthcare workers. To ensure that each surface area was well covered, the swab sticks were rotated.

Gram-stained isolates were identified using standard biochemical tests after the swab sticks were inoculated onto appropriate media (Blood agar and mannitol salt agar) and incubated for 24 to 48 hours at 37°C. Pigment production, acid production, coagulase, and catalase tests were the biochemical tests used to confirm *S. aureus* isolates from mannitol salt agar (20).

The disk diffusion method was used to test antibiotic susceptibility, and the results were interpreted in accordance with Clinical Laboratory Standards Institute guidelines (21). Sterile cotton wool swabs were dipped into a suspension of the organism's overnight growth prepared to the density of a 0.5 McFarland opacity standard to inoculate Mueller-Hinton culture plates. Any excess liquid from the swab was then expressed using the spread plate procedure before inoculation.

The antibiotic disks (Biomark, India) used had the following concentrations: tetracycline, 30 µg; ampicillin, 10 µg; meropenem, 10 µg; gentamicin, 10 µg; erythromycin, 5 µg; ciprofloxacin, 5 µg; cotrimoxazole, 25 µg; cefuroxime, 10 µg; augmentin, 30 µg; cefalexin, 10 µg; vancomycin, 30 µg; ceftazidime, 10 µg; chloramphenicol, 10 µg; ceftriaxone, 30 µg; cefotaxime, 30 µg; and amikacin, 30 µg.

To ensure that the growth was confluent or near confluent, the control and test plates were examined after overnight incubation. The diameter of each zone of inhibition was measured in mm using a ruler on the underside of the plate. Growth began at the endpoint of inhibition (22).

The Clinical and Laboratory Standards Institute criteria (23) were followed when conducting the test. Each isolate was suspended to a turbidity of 0.5 McFarland standard before being plated onto a Mueller-Hinton agar plate (Hardy Diagnostics, USA). On each plate, a 30 µg cefoxitin disk (Oxoid) was placed. Zone sizes were assessed after a 24-hour incubation period at 35°C. Resistance was defined as an inhibitory zone size of less than or equal to 19 mm. ATCC 33591 (MRSA) and ATCC 29213 (MSSA) were used as control strains.

To provide a detailed explanation, each isolate was cultured in 1 mL of sterile water in different 1.5 mL microcentrifuge tubes. For one minute, the samples were centrifuged at 10,000 rpm, and the supernatants were discarded.

After adding 200 µL of lysis buffer to each tube containing the pelleted samples, a vortex mixer was used to mix it thoroughly for a few seconds. The samples were incubated in a heating block machine (Biobase) at 55°C for 10 minutes. The samples were allowed to cool after incubation and were then centrifuged at 10,000 rpm for 30 seconds.

After this step, 200 µL of absolute ethanol was added to each sample, and the supernatants were carefully transferred into well-labeled spin columns fitted into collection tubes without dislodging the pellets.

The spin columns were centrifuged at 10,000 rpm for 1 minute, and the flow collected in the collection tubes was discarded.

Using 500 µL of wash buffer 1 and 2, respectively, the DNA trapped on the silica membrane in the spin columns was washed.

At each washing stage, the spin columns were centrifuged at 10,000 rpm for 30 seconds to allow the wash buffer to pass into the collection tubes.

To remove any traces of ethanol, the spin columns were spin-dried at 14,000 rpm. DNA samples were eluted into well-labeled, nuclease-free 1.5 mL microcentrifuge tubes with 50 µL of elution buffer. After elution, the DNA samples were stored at -20°C until further analysis (24).

The *mecA* gene was amplified by polymerase chain reaction (PCR) using specific primer pairs (*mecA*-F: 5'-AAAATCGATGGTAAAGGTTGGC-3'; *mecA*-R: 5'-AGTTCTGCAGTA CCGGATTTGC-3') (24).

For each isolate's DNA samples, a total reaction volume of 25 µL was prepared using 5X HOT FIREPol Blend Master Mix with 7.5 mM MgCl2 (Solis Biodyne). This was diluted to 1X concentration using 1X Blend Master Mix buffer (Solis Biodyne), 1.5 mM MgCl2, 200 µM of each deoxynucleoside triphosphate (dNTP) (Solis Biodyne), 25 pMol of each forward and reverse primer (BIOMERS, Germany), 2 units of Hot FIREPol DNA polymerase (Solis Biodyne), proofreading enzyme, 5 µL of extracted DNA, and sterile distilled water.

After five minutes of initial denaturation at 95°C, 30 amplification cycles of 30 seconds at 95°C, 30 seconds at 58°C, and 45 seconds at 72°C were carried out using a PTC 200 gradient thermal cycler Eppendorf. A final extension phase of 10 minutes at 72°C followed. Electrophoresis was performed at 80 V for 60 minutes after the resultant amplicons were separated on a 1.5% agarose gel. After electrophoresis, ethidium bromide staining and a UV transilluminator were used to view the DNA bands. A 100 bp DNA ladder was used as the DNA molecular weight marker (24).

Statistical analysis

SPSS statistical software version 20.0 was used for data analysis. Categorical variables, such as the frequency of the bacterial isolates, were summarized using proportions expressed as percentages.

Results

The prevalence of *S. aureus* on inanimate surfaces is provided in Table 1. Of the 122 *S. aureus* isolates collected from inanimate surfaces, footwear harbored the highest proportion (39.34%, n = 48), followed by mattresses (38.52%, n = 47), while clinical coats had the lowest proportion (0.82%, n = 1). At a 95% confidence level, the calculated t-test value (4.366) was greater than the critical t-test value (2.015), indicating that the result was statistically significant and unlikely to be due to random chance.

Table 1. Prevalence of *Staphylococcus aureus* on inanimate surfaces

Items	No. Examined	No. of Bacterial Isolates *Staphylococcus Aureus*	Percentage
Mattress	256	47	38.52
Bedsheet	103	11	9.02
Clinical coat	68	1	0.82
Footwear	108	48	39.34
Pillow	69	7	5.74
Table	96	8	6.56
Total	700	122	-
Percentage	-	45.69	-
P < 0.05 = Significant			

Table 2 shows the prevalence of *S. aureus* among healthcare workers. Eighty-four *S. aureus* isolates were recovered from healthcare workers, with the nostrils having the highest proportion (83.33%, n = 70). At a 95% confidence level, the calculated t-test value (4.320) was less than the critical t-test value (6.314), indicating that the result was not statistically significant.

When the prevalence of *S. aureus* on inanimate surfaces and among healthcare workers was compared, healthcare workers had a higher percentage (68.85%) than inanimate surfaces (45.69%).

The antibiotic susceptibility profile of *S. aureus* isolates is provided in Table 3. The isolates were more susceptible to meropenem, augmentin, tetracycline, ceftazidime, cefuroxime, and cephalexin. Of the 206 *S. aureus* isolates tested for MRSA using cefoxitin disk diffusion, 43.69% (n = 90) were methicillin-resistant (Table 4).

Table 2. Prevalence of *Staphylococcus aureus* among healthcare workers

Site of Collection	No. Examined	No. of Bacterial Isolates *Staphylococcus Aureus*	Percentage
Palm	125	14	14.67
Nostril	175	70	83.33
Total	300	84	-
Percentage	-	68.85	-
P > 0.05 = Not Significant			

Table 3. Antibiotic susceptibility profile of *Staphylococcus aureus* isolates

Antibiotics	Isolates (%)
Ampicillin	70 (33.98)
Meropenem	160 (77.67)
Erythromycin	60 (29.13)
Tetracycline	140 (67.96)
Cotrimoxazole	60 (29.13)
Cefuroxime	150 (72.81)
Gentamicin	70 (33.98)
Ciprofloxacin	75 (36.41)
Augmentin	140 (67.96)
Vancomycin	100 (48.54)
Ceftazidime	120 (58.25)
Cephalexin	130 (63.11)

Table 4. MRSA detection by cefoxitin disk diffusion method

Isolates	Total No	MRSA	Percentage
Staphylococcus Aureus	206	90	43.69

Key: MRSA = Methicillin-Resistant *Staphylococcus Aureus*

The agarose gel electrophoresis of the *mecA* gene (532 bp) is shown in Figure 1. Most of the isolates tested were negative for the *mecA* gene (Negative: 16; Positive: 4).

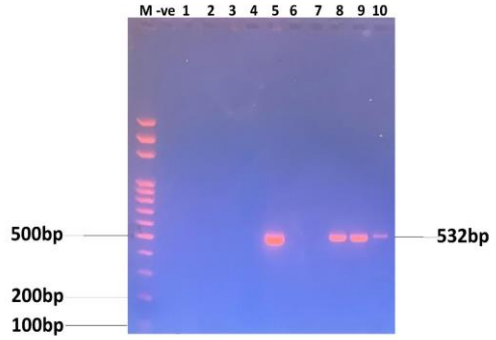

Figure 1. Agarose gel electrophoresis amplification of the *mecA* gene (532 bp) of *S. aureus* isolates. M, Marker (100 bp); lanes 1, 2, 3, 4, 6, and 7, negative; lanes 5, 8, 9, and 10, positive

Discussion

S. aureus (45.69%) was the prevalent bacterium isolated from inanimate surfaces/fomites in this study. These results are comparable to those of the Brazilian study, which found that *S. aureus* was the primary organism isolated from fomites 53.3 percent of the time (25). The findings of Munveshyaka et al. (26) on inanimate surfaces and equipment are also consistent with these findings. *S. aureus* can form biofilms on inanimate surfaces/fomites, and these biofilms can be more resistant to disinfectants and cleaning efforts, making them a more persistent source of infection.

S. aureus was observed more frequently in the nostrils than on the palms of the hands in this study, which corresponds to the findings of Pant and Sharma (27) and Junu et al. (28). This result is consistent with the notion that *S. aureus* most frequently colonizes the nostrils. Compared with nasal carriers, there are fewer reports of hand carriers. Compared with Mukhiya et al. (29) and Pant and Sharma (27), the prevalence of palm-of-hand carriers was lower, which may suggest that the healthcare workers who took part in this study practiced better hand hygiene. In this investigation, every hand carrier was also a nasal carrier, and the isolates from both locations shared similar antibiograms, indicating that they were phenotypically identical. The notion that hands are the primary vector for spreading *S. aureus* from surfaces to the nostrils and from the nostrils to surfaces is further supported by the possibility that these carriers tend to pick their noses.

The bacterial isolates were resistant to frequently used antibiotics. Erythromycin (29.13%), cotrimoxazole (29.13%), gentamicin (33.98%), ampicillin (33.98%), and ciprofloxacin (36.41%) had low susceptibility rates against *S. aureus*. *S. aureus* was more resistant to these drugs. This could be due to their availability, cost, and misuse. This is in line with the findings of Sanusi et al. (30). Adam et al. (31), on the other hand, found that the highest rate of *S. aureus* resistance was to trimethoprim-sulfamethoxazole. This could be due to the development of resistance mechanisms such as reduced antibiotic accumulation within the bacteria and bypassing the antibiotic's inhibitory effects.

Of the 390 bacterial isolates obtained from the two tertiary hospitals in Abia State, 206 (52.82%) were positive for *S. aureus*. Mukhiya et al. (32) and Firesbhat et al. (33) reported the presence of *S. aureus* on hospital surfaces in Nepal and Ethiopia, respectively, despite the scarcity of data regarding the prevalence of MRSA in Nigeria. These results contradict those of this investigation. The findings of this study are in line with those of Anyadoh et al. (34) and Sanusi et al. (30), who found that more than 50% of hospital surfaces were contaminated with *S. aureus*.

The rise of MRSA, which is resistant to cephalosporins, a class of antibiotics commonly used to treat Staphylococcus infections, as well as monobactams and all beta-lactam drugs, has worsened the emergence of *S. aureus* infections (35). Identifying MRSA accurately requires prompt and early diagnosis because MRSA infections lead to treatment issues and spread (36).

In this study, 90 (43.69%) *S. aureus* isolates were found to be resistant by the cefoxitin disk diffusion test for MRSA. This is comparable to the study by Khairullah et al. (37), which revealed that 47.62% of the isolates tested positive for MRSA. In comparison, Adam et al. (31) and Oguzkaya-Artan et al. (38) reported 2.1% of MRSA positivity, while Sanusi et al. (30) reported 5.4%. According to this study, a number of variables, such as improper hand hygiene and failure to clean surfaces, might contribute to the development of MRSA contamination on fomites/inanimate surfaces and healthcare personnel. MRSA contamination raises the possibility of the spread of hard-to-treat Staphylococci, posing a major public health danger.

According to Miragaia (39), *mecA* genotyping by PCR remains the primary recommendation despite its inability to be carried out on a regular basis, and phenotypic detection of MRSA via disk diffusion has not yet yielded fully reliable results. Nevertheless, because of its speed and affordability, disk diffusion identification of MRSA is still often utilized (40).

Oxacillin and cefoxitin diffusion disks exhibit the same 100% sensitivity and 74.07% and 92.59% specificities, respectively (41). However, because there is still a high percentage of false positives with the oxacillin disk diffusion approach, a number of earlier investigations found that cefoxitin disk diffusion had a higher sensitivity level than oxacillin for identifying MRSA (42). According to Vyas et al. (41), beta-lactamase hyperproduction may contribute to false positives by causing oxacillin resistance to manifest phenotypically without a genotypic resistance mechanism.

A total of 20 suspected methicillin-resistant *S. aureus* isolates were tested for the *mecA* gene, and only 4 isolates (20% of all isolates tested by PCR) were *mecA* gene-positive. This might be due to false positives in the phenotypic analysis. Suspected MRSA isolates were found to have the *mecA* gene, as shown by the PCR results. This is similar to Khirullah et al. (37), where 30% of the MRSA isolates tested were *mecA* gene-

positive. However, this contrasts with Sanusi et al. (30), who reported that 83.33% of MRSA isolates were positive for the *mecA* gene. These findings are in line with the study by Ramandinianto et al. (43). Because it can increase the expression of PBP2a, which the *mecA* gene encodes, cefoxitin is an effective inducer of *mecA* gene expression (44). This also agrees with Reichmann and Pinho (45).

Conclusion

This study was conducted to detect the *mecA* gene and MRSA from *S. aureus* isolated from inanimate surfaces, palms of the hands, and nostrils of healthcare workers in selected hospital wards in the two tertiary hospitals in Abia State. This study found an MRSA prevalence of 43.69% and *mecA* gene positivity of only 20%.

The hospital wards evaluated in this study showed the presence of bacterial pathogens on inanimate surfaces and in the nostrils of healthcare workers, with *S. aureus* being the prevalent bacterial isolate in this study. Nasal carriage was higher than hand carriage in this study.

The presence of MRSA in the two tertiary hospitals poses a risk to healthcare and community environments by contaminating healthcare workers, patients, visitors, and surrounding surfaces. This could ultimately lead to community exposure to resistant bacteria. Therefore, prevention and control measures are required to stop the spread of *S. aureus* infections on fomites/inanimate surfaces and among healthcare personnel at tertiary hospitals in Abia State.

However, the unavailability of molecular laboratories in close proximity to run the *mecA* gene analysis immediately limited this study.

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Ethical Statement

The ethics committees of the Federal Medical Centre, Umuahia (Code: FMC/QEH/G.596/Vol.10/746), and Abia State University Teaching Hospital (Code: ABSUTH/MAC/117/VOL1/60) approved the study.

Conflicts of Interest

The authors have no competing interests.

Author Contributions

EUO: Conceptualization; Methodology; Data curation; Formal analysis; Funding acquisition; Investigation; Resources; Visualization; Writing-Original draft; Writing-Review and Editing. IO: Supervision; Methodology. EON: Methodology; Investigation; Interpretation.

Data Availability Statement

All data are available upon request from the corresponding author.

Use of Artificial Intelligence

The authors declare that no artificial intelligence tools were used in this work.

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