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Investigating the Prevalence of the mecA Gene and Antibiotic Resistance of methicillin-resistant *Staphylococcus aureus* in Biological Samples from Hospitalized Patients

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A B S T R A C T

Staphylococcus aureus is a Gram-positive bacterium that can cause various diseases and infections. Penicillin and methicillin are examples of β-lactam antibiotics, the first line of defense against *Staphylococcus aureus* infections. Methicillin-Resistant *Staphylococcus aureus* (MRSA) is still one of the leading causes of hospital-acquired infections associated with morbidity, mortality, and cost. MRSA can be hospital-acquired (HA-MRSA) or community-associated (CA-MRSA) infections. The main objective of this study is to screen MRSA among HA-MRSA to determine the prevalence of antibiotic susceptibility patterns of MRSA among patients. Furthermore, we identify the *mecA* gene, which produces a penicillin-binding protein (PBP2a) with a low affinity for β-lactam antibiotics. This study was done on the patients of Kathmandu Model Hospital, Nepal, and the samples were processed at the Microbiology laboratory of Kathmandu Model Hospital. Data analyses were done from Microsoft Excel and GraphPad Prism. DNA extraction was done from the classical CTAB method with minor modifications, and *mecA* gene-specific primers were used to detect the gene in the samples. Out of 4383 samples, 848 (21.00%) samples have growth, and 190(22.4%) were Staphylococcus aureus. Among *Staphylococcus aureus* 52 (27.36%) were Methicillin resistant *Staphylococcus aures*. Antibiotic susceptibility tests were done to characterize MRSA isolates. Most of the isolates were resistant to Amikacin (69.25%), followed by Ampicillin (53.8%), Chloramphenicol (78.84%), Cotrimoxazole (53.8%), Gentamycin (67.3%), Ofloxacin (15.39%), Erythromycin (71.15%) Vancomycin and Teicoplanin (3.84%). In our study, 50 (96.15%) out of 52 MRSA strains showed the presence of the *mecA* gene, while 3.85% showed the absence of the *mecA* gene.The frequency of MRSA infections in HA-MRSA was comparatively high, with a greater abundance of the *mecA* gene that confers resistance. Regular surveillance of HA-MRSA and genetic profiling of the mecA gene are essential for reducing MRSA infection.

Introduction

According to the 2014 WHO Global Health Report on antimicrobial resistance in all WHO regions, Methicillin-Resistant *Staphylococcus aureus* (MRSA) infections was above 20 % and increased risk of death and associated

health care costs (Organization WHO,2014 and Lee BY et al. 2013). MRSA is a worldwide problem not localized to any geographic area (Carroll, 2008). Hospital-acquired MRSA is the most common cause of hospital-acquired infections (Archer, 1998 and Deresinski, 2005). The most often reported invasive MRSA-related illnesses are septic shock (56%), pneumonia (32%), endocarditis (19%), bacteremia (10%), and cellulitis (6%) (Iwamoto et al. 2013). In general, the frequency of infections caused by MRSA has increased in the last decade (Moran, 2006). Antimicrobial resistance (AMR), which is currently responsible for roughly 5 million deaths annually, has been made worse by the inability to implement infection prevention practices completely and the sharp rise in antibiotic use (Remschmidt *et al.,* 2017). Several genetic factors contribute to the multi-drug resistance in HA-MRSA. HA-MRSA has the *SCCmec* gene cassettes that include a *mecA* gene. The *mecA* gene encodes for an alternative penicillin-binding protein 2a (PBP-2a) with low affinity to β-lactams (Lim and Strynadka, 2002).

When β-lactams block native PBPs, the *mecA* gene is necessary for cell wall production. Even within the same species, isolates carrying the same *mecA* gene frequently displayed varying levels of resistance, suggesting that strain-specific factors may be necessary for manifesting methicillin resistance (Wielders et al 2002).

The main purpose of this research is to detect the presence of the *mecA* gene among the collected samples. Since the horizontal gene transfer of the *mec* gene family has been found to increase the prevalence of MRSA in the community, this study aims to provide general information on how predominant this gene is. This study also seeks to evaluate the efficacy of the disk diffusion test in detecting methicillin-resistant *Staphylococcus aureus.*

Materials and Methods

Sampling

This study takes clinical samples from different biological specimens such as pus, wound, swab, blood, urine, sputum, tissue, nasal swab, and ET. The samples were collected with a sterile swab in Kathmandu Model Hospital. A total of 4285 samples were collected and cultured on nutrient agar. For molecular analysis, MRSA isolates were transported to the Central Department of Biotechnology by taking all the Biosafety and Biosecurity measures according to the guidelines published by the National Public Health Laboratory.

Isolation and Identification of *Staphylococcus aureus*

Clinical specimens were cultured in Nutrient Agar and then sub-cultured in 5% sheep blood agar and mannitol salt agar under sterile conditions. The plates were incubated at 37°C under aerobic conditions. Bacterial isolates with golden yellow color on MSA were further analyzed via Gram's staining, catalase test, slide, and tube coagulase test. Isolates exhibiting the characteristics and properties of *Staphylococcus aureus* were further subcultured in blood agar for confirmation.

Antibiotic susceptibility testing

The modified Kirby-Bauer disk diffusion method was used to perform antibiotic susceptibility testing on Mueller Hinton Agar, following the guidelines set by the Clinical Laboratory and Standards Institute (CLSI, 2020). The commercial antibiotics discs and concentrations used were Amikacin(25µg), Ampicillin(10µg), Penicillin G (30µg), Gentamicin(30µg), Cotrimoxazole (25µg), cefoxitin (30µg), Erythromycin (30µg), Ofloxacin, Chloramphenicol, Teicoplanin, Linezolid, and vancomycin (30µg). A bacterial suspension equivalent to 0.5 McFarland turbidity standard was prepared for inoculation. The plates were incubated at 37°C for 24 hours in Mueller-Hinton agar (MHA) supplemented with 2% NaCl. An inhibition zone diameter of each antimicrobial was then measured and interpreted as resistant (R), intermediate (I), and sensitive (S) according to CLSI guidelines. MRSA-positive strains were confirmed by those *S. aureus* strains that were resistant to cefoxitin and had a zone size < 21mm.

Extraction of DNA

DNA was extracted by the CTAB- the boiling method, in which the pellet of loopful bacteria was resuspended in a TE buffer, followed by adding lysozyme to the mixture. Then, proteinase K and 30 µL of SDS were added after brief incubation until the suspension became clear. Next, preheated CTAB/NaCl (65°C) was added to the suspension. Then an equal volume of phenol/chloroform/isoamyl alcohol (25:24:1) was added to collect the upper aqueous phase of DNA. To the collected supernatant, isopropanol was added, and DNA was precipitated by adding ethanol to the mixture.

Amplification of the *mecA* gene from *Staphylococcus aureus*

For the amplification of the *mecA* gene from *Staphylococcus aureus,* crude lysates were utilized as a DNA template. The primers for this study were used by Vatansever et al., 2016 [14]: Forward Primer (*mecA*PF1) 5'- ACTGCTATCCACCCTCAAAC- 3' and reverse primer (*mecA* PR1) 5'- CTGGTGAAGTTGTAATCTG G-3'. DNA amplification was done in a 10µl of the reaction mixture with 5 µl of master mix, 1 µl of each forward and reverse primer, 1 µl of DNA, and 2 µl of nuclease-free water. The PCR conditions are initial denaturation at 95ºC for 120 seconds, denaturation at 95ºC for 30 seconds, annealing at 56.2ºC for 30 seconds, extension at 72ºC for 20 seconds, and 29 amplification cycles at 72ºC for 5 minutes. After PCR, the amplicon was analyzed by running the samples in 1.5% agarose gel stained with ethidium bromide. The resulting amplicon length of 163 bp was confirmed as a positive sample for the *mecA* gene.

Quality control

The inoculation and culture were carried out using aseptic techniques to ensure contamination-free conditions. The media were checked for the growth of pure cultures of microorganisms. The *S. aureus* ATCC 25923 species was used as a control organism for identification (using Gram staining and culture) and antibiotic susceptibility testing. The Mueller-Hinton agar (MHA) thickness was kept at 4 mm, and the pH was maintained between 7.2 and 7.4. A control smear was stained whenever a new batch of stains was prepared to ensure proper staining reactions. All procedures were conducted under strict aseptic conditions. Equipment such as microscopes, incubators, centrifuges, refrigerators, water baths, autoclaves, anaerobic jars, and hot air ovens was checked regularly to ensure their proper functioning and the reliability of the results. The results were recorded neatly and clearly.

Statistical Analysis

The data was entered into a standard format computer database, checked for errors, and verified. Data maintained in the computer sheets were organized and analyzed using SPSS software (Version 21.6) and GraphPad Prism (Version 9.5.1). Data are presented in appropriate tables, figures, charts, and graphs by calculating percentages, rates, etc. Appropriate statistics were applied wherever applicable.

Results and Discussion

Distribution of Clinical Samples and Bacterial Growth

Samples were collected from different biological specimens, including pus, wound swab, blood, urine, sputum, tissue, nasal swab, and ear and throat (ET) samples at Kathmandu Model Hospital, Kathmandu, Nepal. Among the 4,285 clinical samples processed, 848 (21%) showed bacterial growth, while 3,437 (79%) showed no growth. A higher proportion of bacterial growth was observed among male patients compared to females (Fig. 1).

Gender-wise Distribution of MRSA Isolates

Among the 52 MRSA-positive isolates, 29 (56%) were obtained from male patients and 23 (44%) from female patients, indicating a higher prevalence of MRSA among males (Fig. 2).

Age-wise Distribution of MRSA and Multidrug Resistance

The study population ranged from 2 to 80 years of age. Analysis of MRSA distribution across age groups revealed higher multidrug resistance among patients aged 31–36 years, 37–42 years, and above 56 years. In contrast, isolates from patients aged 49–56 years showed greater susceptibility to the antibiotics tested (Fig. 3). Overall, an increasing trend in antibiotic resistance was observed with advancing age.

Antibiotic Susceptibility Pattern of MRSA Isolates

Antibiotic susceptibility testing revealed that all MRSA isolates showed 100% resistance to penicillin and cefoxitin. High levels of resistance were also observed for chloramphenicol (78.84%), erythromycin (71.15%), amikacin (69.25%), gentamicin (67.3%), ampicillin (53.8%), and cotrimoxazole (53.8%). In contrast, high susceptibility was observed for vancomycin (96.15%), ofloxacin (84.61%), teicoplanin (100%), and linezolid (100%). Only erythromycin showed a small proportion of intermediate resistance (Table 1).

Gender-based Antibiotic Susceptibility Patterns

Female MRSA isolates showed higher resistance to cefoxitin, while remaining highly sensitive to vancomycin, teicoplanin, linezolid, and doxycycline (Fig. 4). Similarly, MRSA isolates from male patients demonstrated resistance to cefoxitin and penicillin, with high susceptibility to linezolid and doxycycline (Fig. 5).

Distribution of MRSA among Clinical Specimens

MRSA isolates were most frequently recovered from pus samples (n = 37), followed by blood (n = 3), sputum (n = 3), urine (n = 2), ET samples (n = 2), nasal swab (n = 1), wound swab (n = 1), and tissue samples (n = 1). The highest prevalence of MRSA was observed in pus samples, indicating their major contribution to MRSA-associated infections in this study (Fig. 6).

Molecular Detection of the *mecA* Gene

PCR analysis was performed to detect the presence of the *mecA* gene among hospital-acquired MRSA isolates. Out of the 52 MRSA isolates, 50 (96%) were positive for the *mecA* gene, confirming the genetic basis of methicillin resistance in most isolates (Fig. 7).

Concordance Between Phenotypic and Genotypic Resistance

A strong concordance was observed between phenotypic methicillin resistance and molecular detection of the *mecA* gene, validating the reliability of conventional antibiotic susceptibility testing supported by PCR-based confirmation.

Table.1 Antibiotic susceptibility pattern of MRSA isolates

S.No.	Antibiotics	Sensitivity		
		Sensitive	Intermediate	Resistance
1.	Amikacin	16(30.7%)	0	36(69.25%)
2.	Ampicillin	24(46.15%)	0	28(53.8%)
3.	Cefoxitin	0	0	52(100%)
4.	Chloramphenicol	11(21.15%)	0	41(78.84%)
5.	Cotrimoxazole	24(46.15%)	0	28(53.8%)
6.	Ofloxacin	44(84.61%)	0	8(15.39%)
7.	Penicillin	0	0	52(100%)
8.	Gentamycin	17(32.69%)	0	35(67.3%)
9.	Erythromycin	14(26.9%)	1(1.92%)	37(71.15%)
10.	Vancomycin	50(96.15%)	0	2(3.84%)
11.	Teicoplanin	52(100%)	0	0
12.	Linezolid	52(100%)	0	0

Figure.1 Distribution pattern of bacteria. Among the total samples of 4,285, 2341(54.63%) were male, and 1944 (45.36%) were female (a). Similarly, among 190 samples, 52 (27.37%) were Methicillin Resistant *Staphylococcus aureus* (MRSA), and the other 138 (72.63%) were MRSA negative (b).

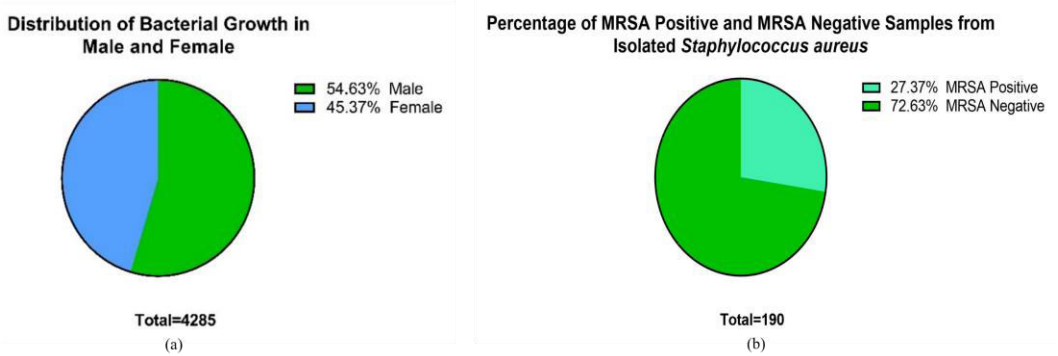


Figure.2 Frequency of positive MRSA samples between males and females. Among the 52 MRSA-positive, 56% were male, and 44% were female.

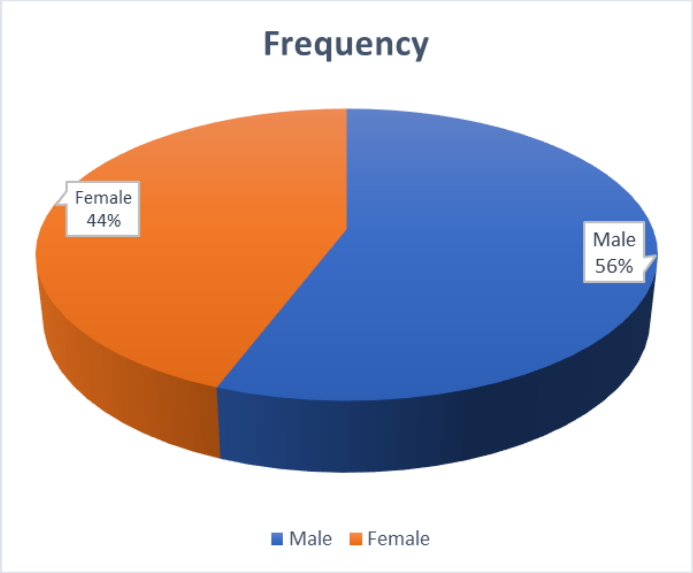


Figure.3 Age-wise distribution of antibiotic resistance and susceptibility among MRSA isolates.

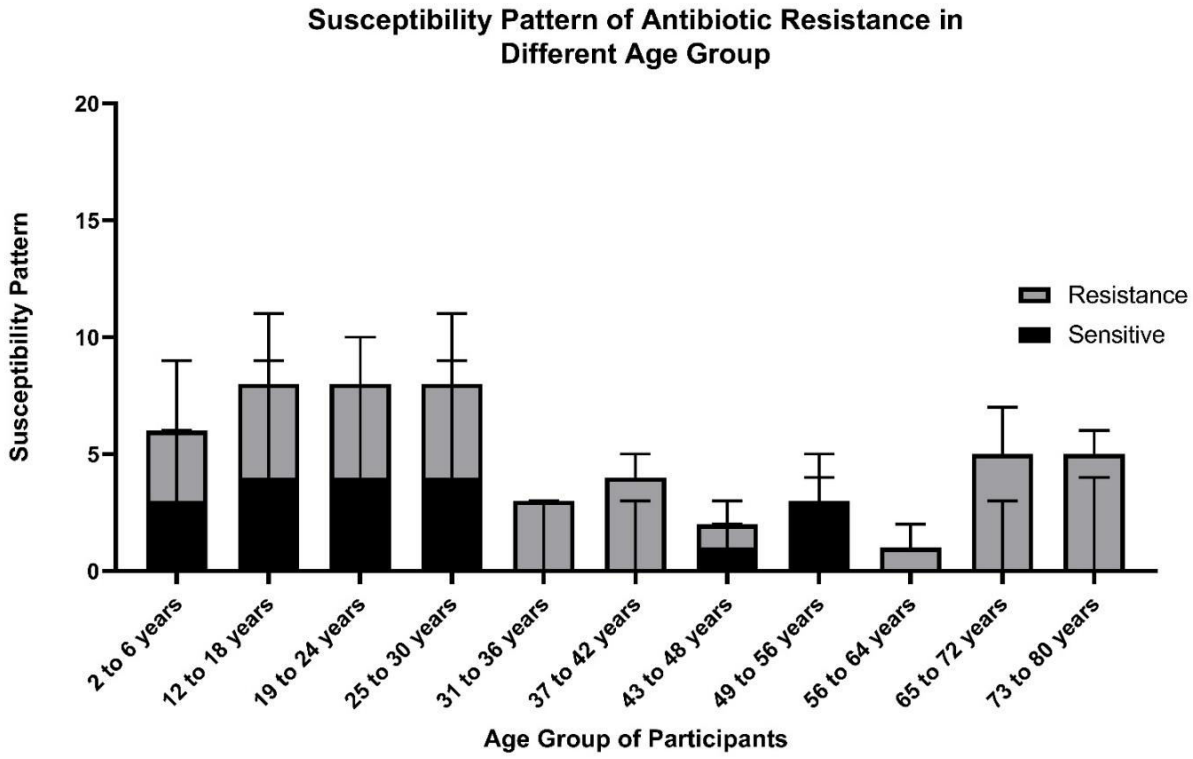


Figure.4 Antibiotic susceptibility pattern of MRSA isolates from female clinical specimens

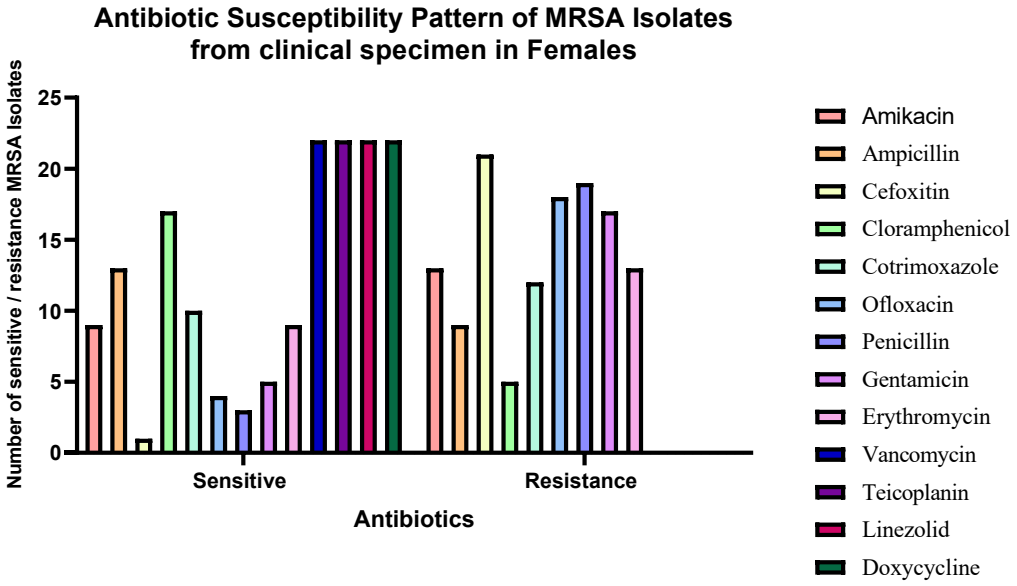


Figure.5 Antibiotic susceptibility pattern of MRSA isolates from male clinical specimens.

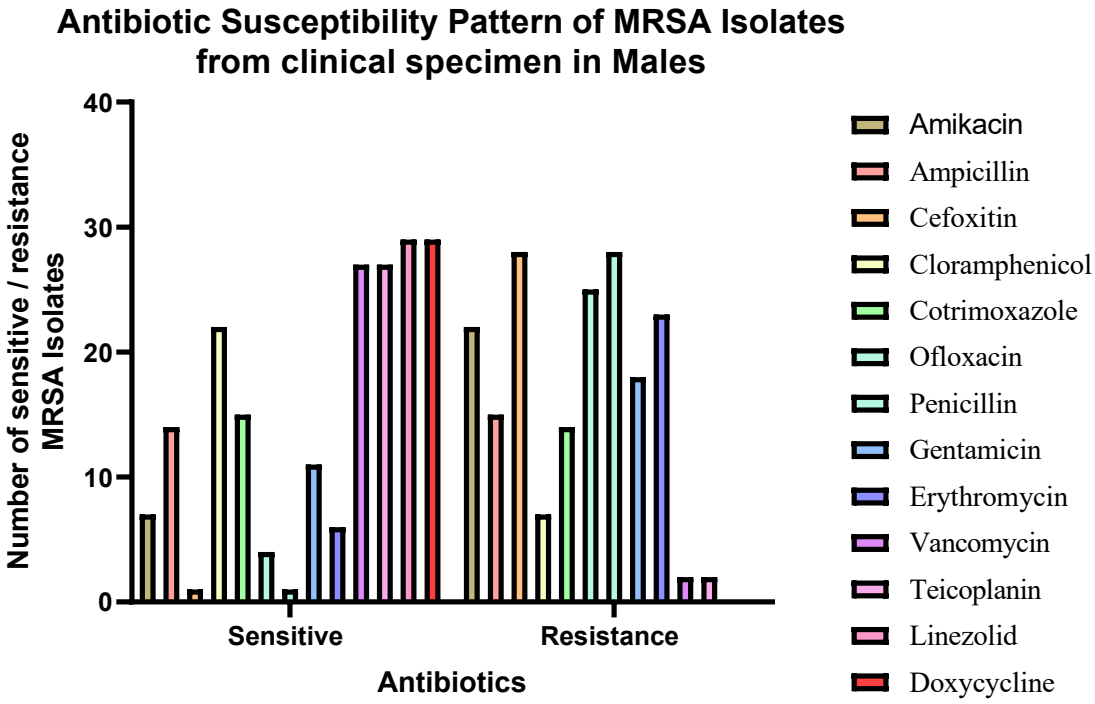


Figure.6 Distribution of MRSA isolates among different clinical specimens

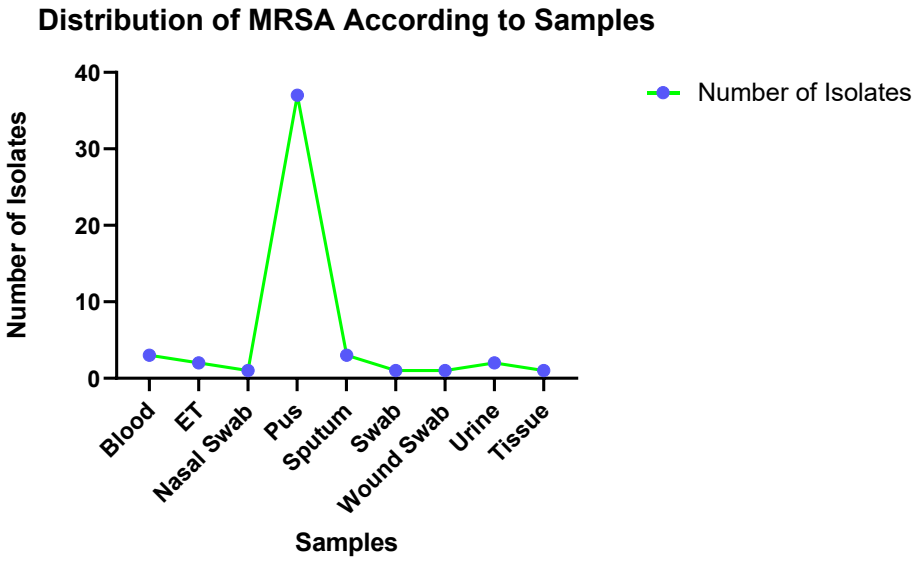
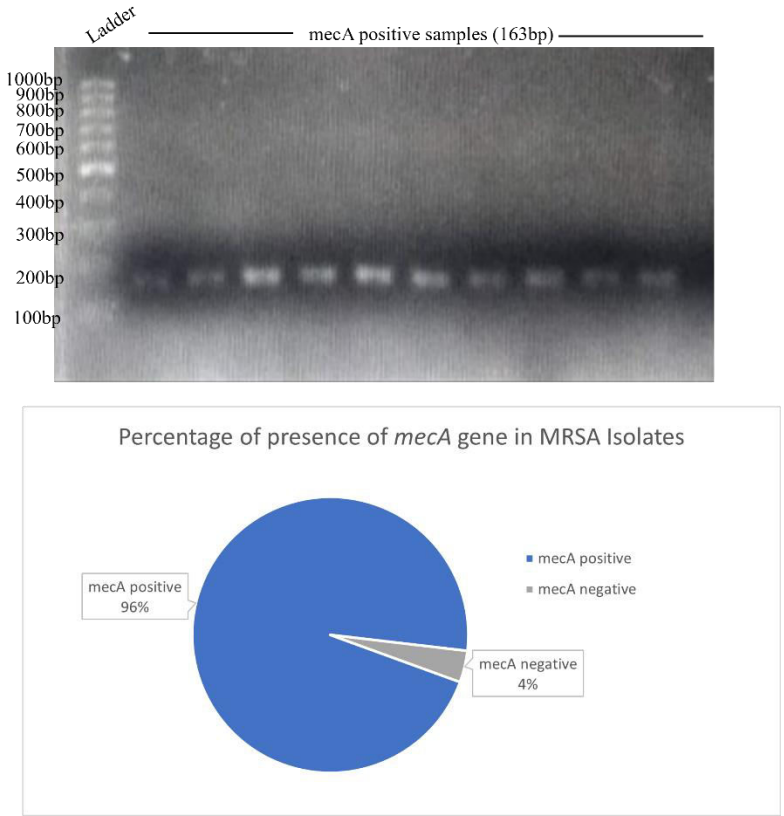


Figure.7 Molecular detection of the mecA gene among MRSA isolates



Methicillin-resistant *Staphylococcus aureus* (MRSA) is a significant pathogen that has emerged in the last four decades, producing both nosocomial and community-acquired infections. Rapid and reliable diagnosis of methicillin resistance in *S. aureus* is critical for administering appropriate antimicrobial treatment and managing MRSA nosocomial dissemination (Vatansever *et al.,* 2016).

The main aim of this study is to highlight the trend of the emergence of MRSA and detect the frequency of *mecA* in the isolated MRSA strains. Similarly, from this study, we could elucidate the resistance of MRSA to the empirical drugs used in hospitals and medical centers in Nepal.

One of the most significant discoveries in this investigation is the prevalence of MRSA strains (27.37%) among isolated *Staphylococcus aureus* from different biological specimens. MRSA strains were identified via the conventional method, the Kirby-Bauer disk method (Al-Ruaily and Khalil, 2011). The incidence of MRSA in our study was higher in males than in females (56% vs 44%). This finding accords with the meta-analysis conducted by Ghia et al. 2020 where the male population across the studies accounted for 60.4% of cases as opposed to 39.6% in females. Furthermore, 7-year long-term research by Kupfer et al. revealed that male gender was a significant risk factor for acquiring MRSA. A higher abundance of MRSA in males has been attributed to behavioral practices that increase the rate of MRSA infections.

In addition to gender, we sought to analyze the distribution of MRSA between age groups. Our study found that the age group from 56 to 80 had all resistant MRSA strains, following the trend in other literature (Shukla *et al.,* 2009). Since age has been regarded as an independent factor for MRSA incidence by the Centers for Disease Control and Prevention (CDC) or the Robert Koch Institute, some things might be more consistent with the results (Diller *et al.,* 2008). However, older people have more risk factors that must be accounted for.

Compared to the previous studies by different authors, such as Pai V et al. 2020, we found a higher population of MRSA strains in the Pus sample (71%). In other investigations, pus was shown to have a more significant proportion of Methicillin-resistant *Staphylococcus aureus* than blood, urine, wound swab, and sputum (Shahi *et al.,* 2018).

Similarly, we analyzed the antibiogram of isolated MRSA from different clinical samples. All MRSA samples were found to be resistant to penicillin and cefoxitin. The resistance of MRSA to β-lactam antibiotics such as penicillin arises due to the mutation of penicillin-binding protein (Khanal *et al.,* 2018). Chloramphenicol is effective against a large population of MRSA isolates worldwide (Lee *et al.,* 2018). But, in our study, we found 78.84% of strains of MRSA resistant to it. High resistance to chloramphenicol may be related to local antibiotic prescription habits. Resistance to erythromycin and gentamycin has rapidly developed. Due to the emerging resistance of MRSA to these classes, it has not been advised for the treatment of MRSA (Garau *et al.,* 2009). In this comparative analysis, vancomycin, teicoplanin, and linezolid were the most effective antibiotics. The synergistic actions of linezolid/vancomycin and linezolid/teicoplanin are effective against MRSA isolates (Neupane *et al.,* 2025). The present study results revealed and accorded with the superiority of linezolid and teicoplanin in treating MRSA, as evident in many studies worldwide (Jacqueline *et al.,* 2003; Sabir *et al.,* 2014; Chen *et al.,* 2018). Thus, this antibiogram of MRSA isolates shows that Teicoplanin and Linezolid, which belong to the glycopeptide and oxazolidinone classes, respectively, are the most effective antibiotics.

Almost 96% of the clinical samples in our study showed the *mecA* gene. The mecA gene's high prevalence in MRSA indicates the potential resistance to the beta-lactam group. *mecA* gene is a gold standard for identifying MRSA (Fu *et al.,* 2013). Our results of the high *mecA* gene correlate with the study conducted by Haydeh E et al., 2019 and McTavish, SM et al., 2019. In our study, only 4% didn't have *mecA* gene. Some studies have reported a low occurrence of the *mecA* gene (McTavish *et al.,* 2019; Bhatt *et al.,* 2016). The increased prevalence of the mecA gene in our study might be attributed to the fact that the samples were obtained from a regular diagnostic lab where there is a mixture of patients from the intensive care unit, extended stay of patients in the hospital, frequent use of invasive medical procedures, and haphazard use of multiple antibiotics (Pournajaf *et al.,* 2014).

Various intrinsic factors play a role in the development of resistance, suppressing the expression of the *mecA* gene. In a recent investigation, five major SCC *mec* types, *mecA,* and the PBP2 gene product were missing; the isolates remained phenotypically resistant, indicating

the possibility of β-lactamase hyperproduction (Becker *et al.,* 2014). Similarly, another study revealed that specific changes in amino acids on protein binding cascades (PBPs 1,2 and 3), which play a crucial role in developing MRSA resistance, might alter the expression of *mecA* in resistant strains (Dhungel *et al.,* 2021).

This study has a few limitations, including a shorter study duration, a smaller sample size, and a single study site. Future research might expand on the findings by doing a longitudinal study at several tertiary care hospitals to enhance the results. Due to limited resources, a minimum inhibitory concentration (MIC) was not possible, which might have provided some further insights into this study. Nonetheless, this study emphasizes the phenotypic and molecular methods for detecting MRSA, providing a valuable reference for future studies on MRSA in Nepal and developing countries. Comprehensive information on the antibiogram of MRSA strains can be beneficial for tertiary centers where nosocomial infections are significantly high.

Data availability

The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

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Author contributions

Sita Neupane: Conceived the original idea and designed the model and wrote the manuscript. Conceptualization, Methodology, Investigation, Resources, Writing - Original Draft Preparation, Supervision. Aastha Acharya: Designed the model and the computational framework and analysed the data. Methodology, Software, Validation, Formal Analysis, Investigation, Resources, Data Curation, Writing - Original Draft Preparation, Writing - Review & Editing, Visualization. Samiran Subedi: Designed the model and the computational framework and analysed the data Software, Validation, Formal Analysis, Investigation, Writing - Review & Editing

Declarations

Ethical Approval Not applicable.

Consent to Participate Not applicable.

Consent to Publish Not applicable.

Conflict of Interest The authors declare no competing interests.

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