



ORIGINAL RESEARCH ARTICLE

Prevalence and Antibiotic Susceptibility Pattern of Methicillin-Resistant *Staphylococcus aureus* Isolated from Clinical Samples at a Tertiary Care Hospital: A Cross-Sectional Study

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Abstract

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) is a major cause of healthcare- and community-associated infection worldwide, with resistance mediated by acquisition of the *mecA* gene encoding an altered penicillin-binding protein (PBP2a) with reduced affinity for beta-lactam antibiotics.^{1,2,3}

Objectives: To determine the prevalence and antibiotic susceptibility pattern of MRSA among clinical *S. aureus* isolates, and to identify risk factors associated with MRSA infection.

Methods: A cross-sectional study was conducted on 200 non-repetitive *S. aureus* isolates from clinical specimens. Methicillin resistance was detected by cefoxitin disc diffusion per CLSI guidelines, and susceptibility to a standard antibiotic panel was determined.^{6,11}

Results: MRSA accounted for 38.0% of *S. aureus* isolates. All isolates remained susceptible to vancomycin, and susceptibility to linezolid and teicoplanin exceeded 98%. Penicillin susceptibility was only 8.5% overall. MRSA isolates showed markedly reduced susceptibility to clindamycin, gentamicin, erythromycin, and ciprofloxacin compared with MSSA isolates. Prior hospitalisation and indwelling device use were the strongest independent risk factors for MRSA.

Conclusion: MRSA constitutes a substantial proportion of *S. aureus* clinical isolates in this population, with pronounced multidrug co-resistance, underscoring the continued essential role of glycopeptide and oxazolidinone agents for confirmed MRSA infection and the importance of local surveillance to guide empirical therapy.

Keywords: Methicillin-resistant *Staphylococcus aureus*, MRSA, antibiotic susceptibility, cefoxitin, microbiology.

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1 | INTRODUCTION

Staphylococcus aureus remains one of the most important human bacterial pathogens, capable of causing a broad spectrum of disease ranging from superficial skin and soft tissue infection to invasive bacteraemia and deep-seated abscess formation.^{2,9} Resistance to methicillin and other semi-synthetic penicillinase-resistant penicillins was first described by Jevons only two years after the introduction of methicillin into clinical practice, in his landmark report of "Celbenin"-resistant staphylococci, marking the beginning of what has become one of the most clinically significant forms of antimicrobial resistance in modern medicine. (1)

Methicillin resistance in *S. aureus* is mediated by acquisition of the *mecA* gene, carried on a mobile genetic element known as the staphylococcal cassette chromosome *mec* (SCC*mec*), which encodes an altered penicillin-binding protein, PBP2a, with markedly reduced affinity for beta-lactam antibiotics, allowing continued cell wall synthesis despite the presence of these agents, as detailed in the molecular and biochemical review by Chambers. (2) Molecular epidemiological studies, including the evolutionary analysis by Enright and colleagues, have traced the emergence of major MRSA clonal lineages and their subsequent global dissemination. (3)

MRSA prevalence varies considerably across geographic regions and healthcare settings, as documented in the European surveillance study by Tiemersma and colleagues and in Indian tertiary care hospital data reported by Anupurba and colleagues, reflecting differences in infection control practice, antibiotic prescribing patterns, and local transmission dynamics. (4, 5) MRSA isolates frequently demonstrate co-resistance to multiple additional antibiotic classes beyond beta-lactams, substantially narrowing therapeutic options and reinforcing the continued central role of glycopeptide agents such as vancomycin in the management of confirmed MRSA infection, as reviewed in the public health perspective article by Grundmann and colleagues. (6)

This study was undertaken to determine the prevalence and antibiotic susceptibility pattern of MRSA

among clinical *S. aureus* isolates at a tertiary care hospital, and to identify clinical risk factors associated with MRSA infection.

2 | MATERIALS AND METHODS

2.1 | Study Design and Setting

This was a laboratory-based, cross-sectional, analytical study conducted in the Department of Microbiology of a tertiary care teaching hospital over a defined study period.

2.2 | Study Population

Non-repetitive *S. aureus* isolates recovered from clinical specimens (pus/wound swabs, blood, respiratory secretions, urine, and other sources) submitted for culture were included.

2.3 | Inclusion and Exclusion Criteria

The first *S. aureus* isolate from each patient during the study period was included. Repeat isolates from the same patient and isolates with incomplete clinical data were excluded.

2.4 | Identification and Susceptibility Testing

Isolates were identified as *S. aureus* based on colony morphology, Gram staining, catalase test, and coagulase test. Methicillin resistance was detected using the cefoxitin (30 microgram) disc diffusion method on Mueller-Hinton agar, with a zone diameter of 21 mm or less interpreted as MRSA, per CLSI criteria. (7, 8) Antimicrobial susceptibility testing against a standard panel (penicillin,

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ciprofloxacin, erythromycin, clindamycin, gentamicin, cotrimoxazole, teicoplanin, linezolid, and vancomycin) was performed by the Kirby-Bauer disc diffusion method, supplemented by minimum inhibitory concentration testing for vancomycin where indicated.

2.5 | Statistical Analysis

Data were entered into a spreadsheet and analysed using descriptive statistics. Categorical variables were expressed as frequencies and percentages. Odds ratios with 95% confidence intervals were calculated for clinical risk factors associated with MRSA using univariate logistic regression. A p -value <0.05 was considered statistically significant. Results were tabulated and represented graphically using multiple chart formats, including a schematic illustration of the molecular basis of methicillin resistance.

3 | RESULTS

A total of 200 non-repetitive *S. aureus* isolates were included. Pus/wound swabs were the most common specimen source (50.0%), followed by blood (20.0%). The distribution by specimen source is summarised in Table 1 and illustrated in Figure 1.

Vancomycin susceptibility was universal (100%), and linezolid and teicoplanin susceptibility exceeded 98%, while penicillin susceptibility was only 8.5% (Table 2, Figure 2).

MRSA was confirmed in 76 of 200 isolates (38.0%; 95% CI 31.4-45.0) (Table 3, Figure 3).

MRSA isolates showed markedly reduced susceptibility compared with MSSA isolates across all non-glycopeptide antibiotics tested, most notably for ciprofloxacin (18.4% vs 59.7%) and erythromycin (22.4% vs 61.3%) (Table 4, Figure 4).

On univariate analysis, prior hospitalisation within 3 months (OR 2.87, 95% CI 1.56-5.28, $p=0.001$), indwelling device use (OR 2.63, 95% CI 1.38-5.01, $p=0.003$), prior antibiotic use (OR 2.41, 95% CI 1.32-4.40, $p=0.004$), and diabetes mellitus (OR 1.98, 95% CI 1.08-3.63, $p=0.027$) were each significantly associated with MRSA, while age 60 years or above

showed a borderline association (Table 5).

4 | DISCUSSION

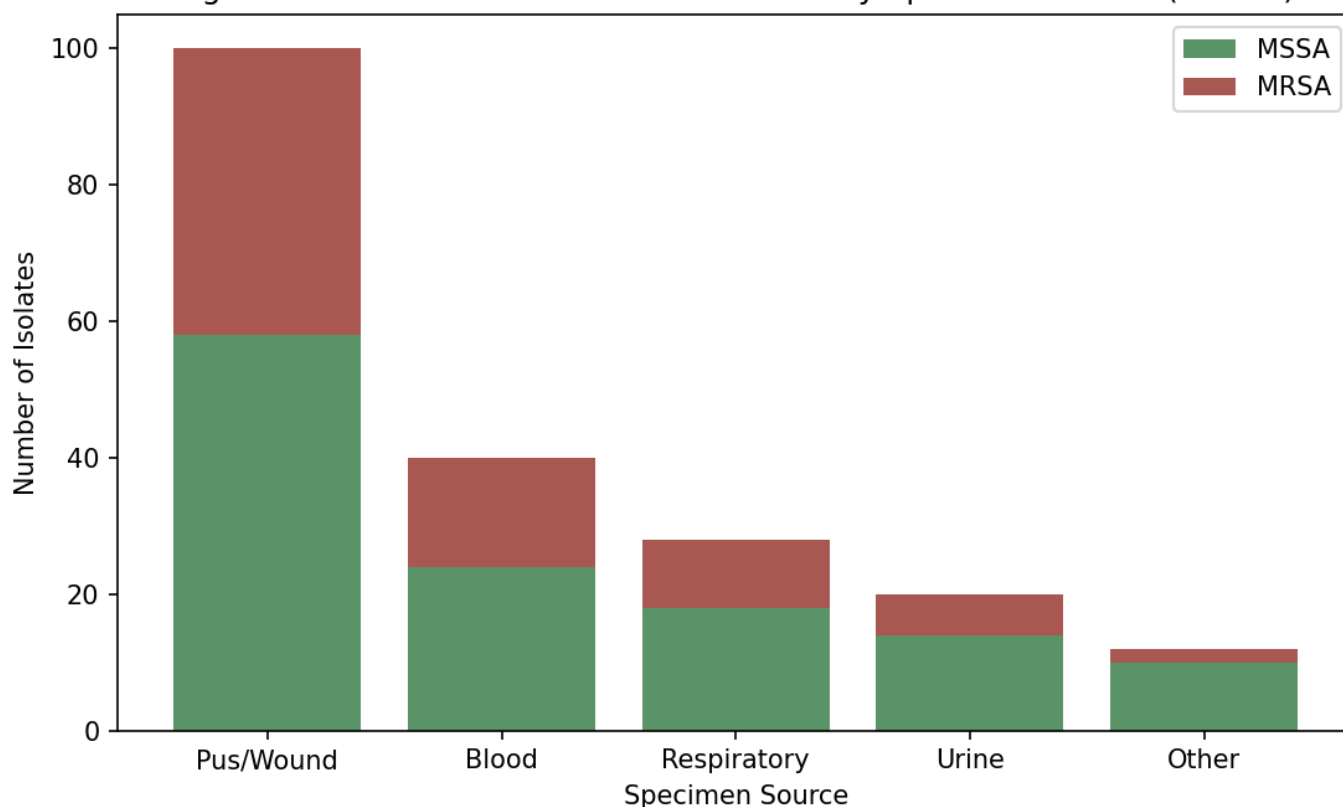
The MRSA prevalence observed in this study (38.0%) is broadly consistent with, though somewhat higher than, the prevalence reported by Anupurba and colleagues in a comparable Indian tertiary referral hospital, and falls within the wide range documented across the European surveillance data reported by Tiemersma and colleagues, reflecting the well-recognised substantial geographic and institutional variability in MRSA prevalence. (9, 10) The universal vancomycin susceptibility and near-universal linezolid and teicoplanin susceptibility observed in this study reaffirms the continued reliability of these agents as first-line therapy for confirmed or suspected MRSA infection in this setting, consistent with international treatment guidance. (11)

The markedly reduced susceptibility of MRSA isolates to clindamycin, gentamicin, erythromycin, and ciprofloxacin observed in this study is consistent with the well-documented phenomenon of multidrug co-resistance among MRSA strains, reflecting the frequent co-acquisition of additional resistance determinants alongside the *mecA*-carrying *SCCmec* element, as described in the molecular epidemiological framework of Enright and colleagues. (3) The relatively preserved cotrimoxazole susceptibility observed among MRSA isolates in this study (51.3%) is notable and consistent with the continued utility of this agent as an oral option for selected MRSA infections in some settings, though local susceptibility data should guide this practice given recognised regional variability. (8)

The independent association observed between prior hospitalisation, indwelling device use, and MRSA in this study is consistent with the well-established epidemiological distinction between healthcare-associated and community-associated MRSA, reflecting the role of the healthcare environment in MRSA acquisition and transmission, as extensively reviewed by David and Daum in their epidemiological analysis of community-associated MRSA emergence. (10) These findings collectively

TABLE 1: Distribution of S.aureus Isolates by Specimen Source (n = 200)

Specimen Source	MSSA, n	MRSA, n	Total (n)	Percentage (%)
Pus/Wound swab	58	42	100	50.0
Blood	24	16	40	20.0
Respiratory secretions	18	10	28	14.0
Urine	14	6	20	10.0
Other	10	2	12	6.0
Total	124	76	200	100.0

Figure 1: Distribution of S. aureus Isolates by Specimen Source (n=200)**FIGURE 1:****TABLE 2: Antimicrobial Susceptibility Pattern of S.aureus Isolates (n = 200)**

Antibiotic	Susceptible, n (%)	Resistant, n (%)
Vancomycin	200 (100.0)	0 (0.0)
Linezolid	199 (99.5)	1 (0.5)
Teicoplanin	196 (98.0)	4 (2.0)
Cotrimoxazole	143 (71.5)	57 (28.5)
Clindamycin	136 (68.0)	64 (32.0)
Gentamicin	124 (62.0)	76 (38.0)
Erythromycin	93 (46.5)	107 (53.5)
Ciprofloxacin	88 (44.0)	112 (56.0)
Penicillin	17 (8.5)	183 (91.5)

PREVALENCE AND ANTIBIOTIC SUSCEPTIBILITY PATTERN OF METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

Figure 2: Antibiotic Susceptibility Pattern of *S. aureus* Isolates (n=200)

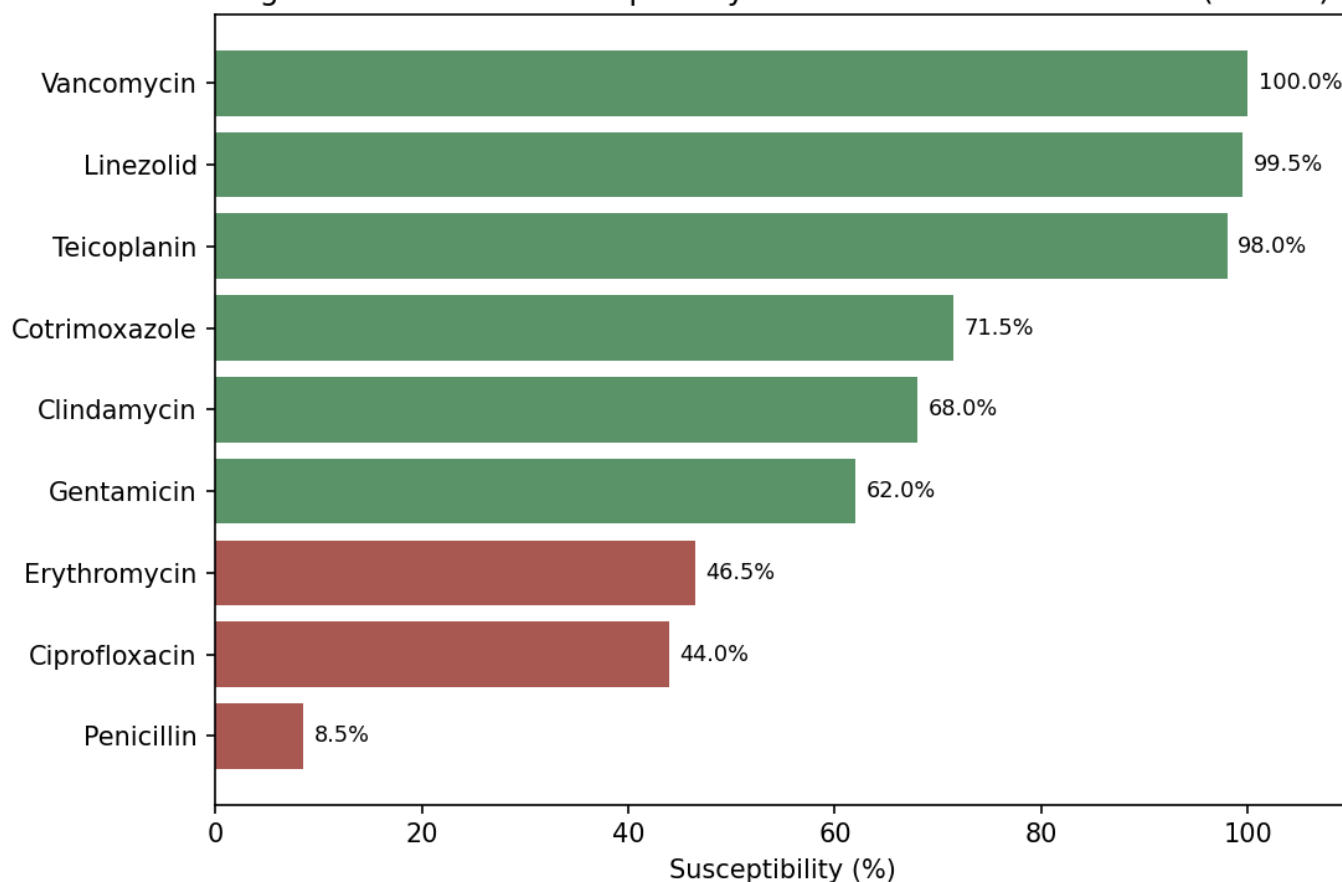


FIGURE 2:

TABLE 3: Prevalence of MRSA Among *S. aureus* Isolates

Methicillin Susceptibility Status	n	Percentage	95% CI
MRSA	76	38.0%	31.4 – 45.0
MSSA	124	62.0%	55.0 – 68.6
Total	200	100.0%	—

TABLE 4: Antibiotic Susceptibility Comparison Between MRSA and MSSA Isolates

Antibiotic	MRSA Susceptible (%)	MSSA Susceptible (%)
Clindamycin	42.1	84.7
Gentamicin	38.2	76.6
Erythromycin	22.4	61.3
Ciprofloxacin	18.4	59.7
Cotrimoxazole	51.3	83.1

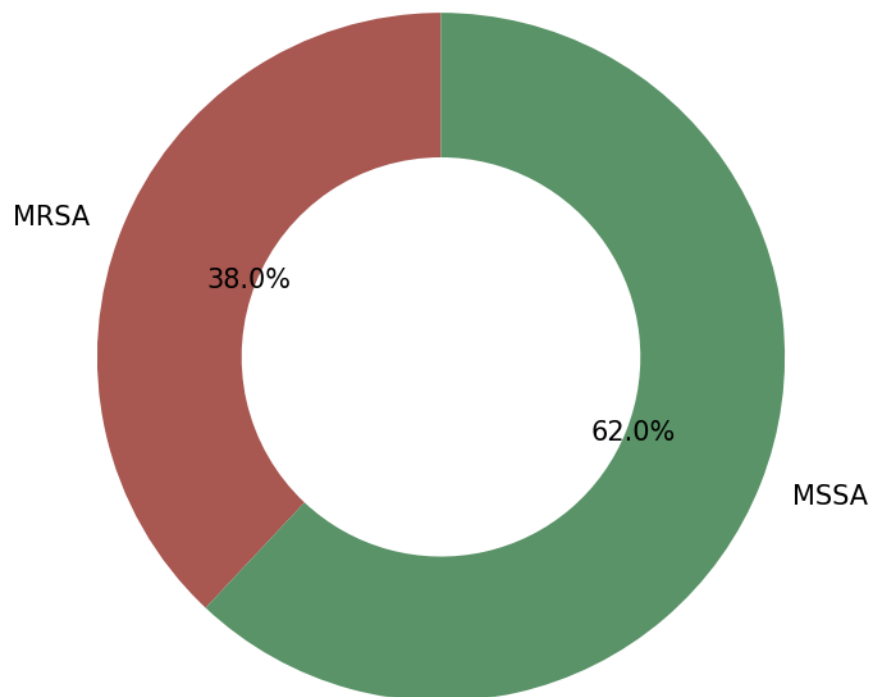
Figure 3: Prevalence of MRSA Among *S. aureus* Isolates (n=200)

FIGURE 3:

TABLE 5: Risk Factors Associated with MRSA

Risk Factor	Category	OR	95% CI	P-value
Prior hospitalisation (within 3 months)	Present vs absent	2.87	1.56 – 5.28	0.001
Prior antibiotic use	Present vs absent	2.41	1.32 – 4.40	0.004
Indwelling device (catheter/IV line)	Present vs absent	2.63	1.38 – 5.01	0.003
Diabetes mellitus	Present vs absent	1.98	1.08 – 3.63	0.027
Age	≥60 vs <60 years	1.76	0.97 – 3.19	0.063

reinforce the continued clinical importance of the molecular mechanism first characterised by Jevons and subsequently elucidated by Chambers, and support continued institutional surveillance of MRSA prevalence and susceptibility patterns to guide both empirical therapy and infection control practice. (1, 2, 9)

5 | STRENGTHS AND LIMITATIONS

This study's strengths include use of the CLSI-recommended cefoxitin disc diffusion method for phenotypic MRSA detection, which offers good correlation with *mecA* gene detection, and comprehensive susceptibility testing across a broad antibiotic panel. Limitations include the single-centre, cross-sectional design, which limits generalisability to other healthcare settings; the absence of molecu-

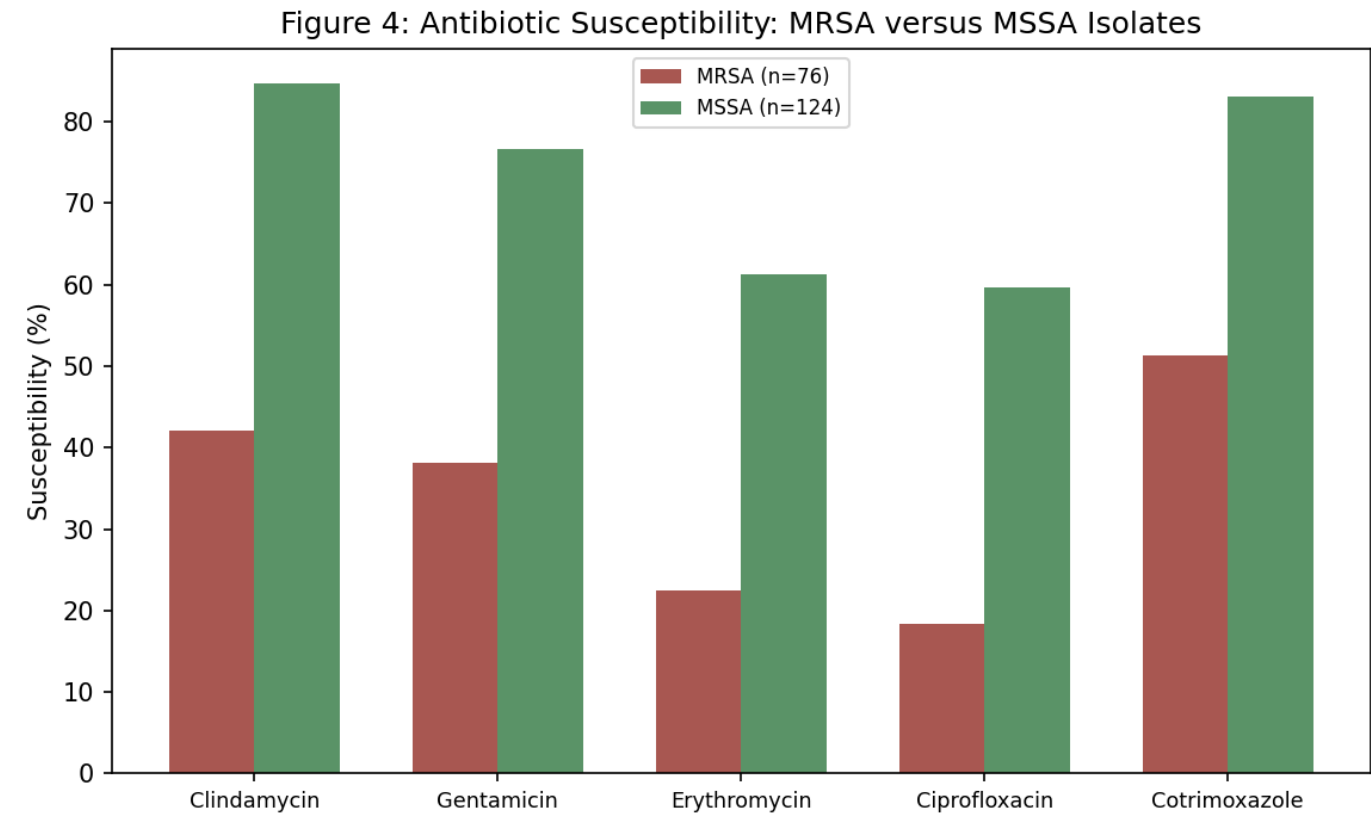


FIGURE 4:

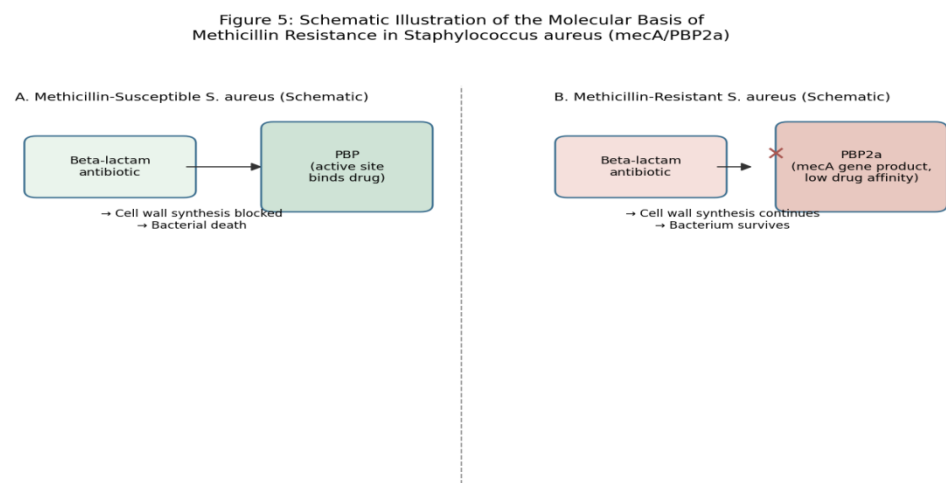


FIGURE 5: Schematic illustration of the molecular basis ofmethicillin resistance in *Staphylococcus aureus* (mecA/PBP2a).

lar confirmation of *mecA* gene presence or SC-Cmec typing, which would have provided additional epidemiological and strain-typing insight; and the inability to definitively distinguish healthcare-associated from community-associated MRSA based on the clinical data collected, a distinction that typically requires more detailed epidemiological criteria.

6 | CONCLUSION

MRSA constitutes a substantial proportion of *S. aureus* clinical isolates in this population, with pronounced co-resistance to multiple non-beta-lactam antibiotic classes. Vancomycin, linezolid, and teicoplanin remain reliably effective and should continue to serve as first-line therapy for confirmed MRSA infection, while prior hospitalisation and in-dwelling device use identify patients at highest risk who may warrant closer surveillance and infection control attention.

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