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Molecular Characterization and Histopathological Correlation of Antibiotic Resistance Genes in Clinical Isolates of MRSA in Pakistani Hospitals

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ABSTRACT

Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) is a major public health concern worldwide due to its multidrug resistance and associated morbidity. In Pakistan, hospital-based prevalence remains high, yet limited data exist on the molecular determinants of resistance and their correlation with tissue pathology.

Objective: To characterize antibiotic resistance genes in clinical isolates of MRSA from Pakistani hospitals and to assess their correlation with histopathological features of infection.

Methodology: A cross-sectional study was conducted from January to December 2025 in three tertiary-care hospitals. A total of 406 clinical MRSA isolates were included. Standard microbiological procedures and PCR assays were used to detect resistance genes. Histopathological specimens from corresponding infection sites were examined for necrosis, abscess formation, vascular invasion, and chronic inflammation. Data were analyzed using SPSS 25. Associations were tested with Chi-square, t-test, and multivariable logistic regression.

Results: All isolates carried *mecA*, while *mecC* was present in 3%. Among accessory genes, *ermA* (31%), *ermC* (24.1%), *tetK* (20.9%), *tetM* (18.2%), and *aac(6')-aph(2'')* (16.5%) were frequent, whereas *vanA* (2.2%) and *vanB* (0.7%) were rare. Gene-positive isolates exhibited significantly higher rates of severe necrosis ($p < 0.001$), abscess formation ($p = 0.002$), and vascular invasion ($p = 0.004$). Logistic regression identified *ermA* ($p = 0.001$), *tetM* (, $p = 0.041$), and prior antibiotic exposure ($p = 0.046$) as independent predictors of severe histopathological changes.

Conclusion: MRSA in Pakistani hospitals demonstrates widespread *mecA* carriage with frequent accessory resistance genes, which correlate with more severe tissue pathology..

Keyword: Antibiotic Resistance, Histopathology, Methicillin-Resistant Staphylococcus aureus (MRSA)

1. INTRODUCTION

Methicillin-resistant Staphylococcus aureus (MRSA) is a major pathogen of global concern due to its ability to cause severe infections and its remarkable resistance to multiple antibiotics.(1) MRSA was first reported in the early 1960s, only a year after the introduction of methicillin, and has since evolved into one of the most challenging healthcare-associated and community-acquired pathogens worldwide.(2) It is responsible for a wide spectrum of infections ranging from superficial

skin and soft tissue infections to life-threatening conditions such as pneumonia, bacteremia, septicemia, and endocarditis. The increasing prevalence of MRSA is a serious threat to healthcare systems as it limits therapeutic options and prolongs hospital stays, thereby increasing morbidity, mortality, and healthcare costs.(3)

Globally, MRSA accounts for approximately 20–50% of S. aureus infections, with particularly high prevalence in developing countries.(4) According to the World Health Organization (WHO), antimicrobial resistance is among the top ten global public health threats, and MRSA remains one of the most frequently encountered resistant organisms. In South Asia, the burden is considerably high due to overcrowded hospitals, limited infection control measures, and excessive or inappropriate antibiotic usage. A systematic review has reported MRSA prevalence in Asian countries ranging between 25% and 50%, depending on the clinical setting and geographic location.(5)

In Pakistan, MRSA infections have emerged as a critical healthcare problem.(6) Several hospital-based studies have documented alarmingly high rates of MRSA among clinical isolates, with prevalence ranging between 33% and 50% in tertiary care centers.(7) The majority of MRSA strains carry the mecA gene, which encodes for penicillin-binding protein 2a (PBP2a), conferring resistance to β-lactam antibiotics.(8) In addition, various other resistance genes such as erm, tet, and van have been identified, complicating treatment strategies.(9) Histopathological findings of MRSA infections further reveal significant tissue necrosis, inflammatory cell infiltration, abscess formation, and vascular involvement, which correlate with the severity and clinical outcomes of infections.(10) Despite this growing challenge, limited data exist from Pakistani hospitals on the molecular characterization of antibiotic resistance genes and their correlation with histopathological patterns.

Given the rising prevalence of MRSA in Pakistan and its significant clinical and economic impact, it is crucial to investigate the molecular basis of resistance and its pathological manifestations. Understanding the distribution of resistance genes and their association with histological features of infection can provide valuable insights for targeted therapy and infection control measures. Therefore, this study aimed to characterize the antibiotic resistance genes in MRSA clinical isolates from Pakistani hospitals and correlate these molecular findings with histopathological changes. This will help in bridging existing knowledge gaps and guiding effective treatment and control strategies for MRSA infections in the local healthcare setting.

2. METHODOLOGY

This cross-sectional analytical study was conducted in the microbiology and pathology departments from January to December 2025 in three tertiary-care hospitals. Written informed consent for adults andguardian consent for minors) was obtained for use of clinical specimens and tissue samples for research purposes. Confidentiality of patient data was maintained and samples were coded to remove personal identifiers.

A sample size was calculated using the single-proportion formula (as implemented by OpenEpi). Assuming an expected MRSA prevalence of **40%** among *S. aureus* isolates based on published Pakistani hospital reports (11), a 95% confidence level ($Z = 1.96$) and a margin of error (precision) of 5% ($d = 0.05$), the calculated minimum sample size is: $n = (Z^2 \times p \times q) / d^2 = (1.96^2 \times 0.40 \times 0.60) / 0.05^2 = 368.79 \rightarrow$ **369.** Allowing for a 10% non-response or sample loss, the final target sample size was **406 clinical** *S. aureus* **isolates.**

Consecutive clinical isolates of *Staphylococcus aureus* obtained from inpatients and outpatients of the participating hospitals during the study period, from specimens with clinical relevance (blood, wound swabs/tissue, tracheal aspirates, pus, urine when judged significant), and isolates identified as methicillin-resistant by initial screening (cefoxitin disk or automated system) were included. For histopathological correlation, tissue specimens (biopsies or debrided tissue) from the same infection focus and same patient from whom culture was obtained were included when available. Duplicate isolates from the same infection episode and patient, environmental or surveillance swabs (except when clinically indicated), isolates lacking an available corresponding histopathology specimen (for cases intended for molecular–histopathologic correlation when tissue was required), and specimens with insufficient material for molecular testing were excluded.

Clinical specimens were processed according to standard laboratory procedures. Samples were cultured on blood agar and mannitol salt agar; colonies morphologically consistent with *S. aureus* were confirmed by Gram stain, catalase test, slide/tube coagulase, and automated identification where available. Antimicrobial susceptibility testing (AST) was performed by the Kirby–Bauer disk diffusion method and interpreted according to Clinical and Laboratory Standards Institute (CLSI) guidelines current at the time of testing. Methicillin resistance was screened by cefoxitin (30 µg) disk diffusion; isolates with cefoxitin resistance were considered MRSA and selected for molecular testing. Minimum inhibitory concentrations (MICs) for selected antibiotics were determined by Etest or automated systems for clinically important agents (vancomycin, linezolid, daptomycin) as available.

DNA was extracted from confirmed MRSA isolates using a commercial bacterial DNA extraction kit following the manufacturer's protocol. Polymerase chain reaction (PCR) assays were performed to detect key antibiotic resistance genes, including **mecA** (and mecC when resources permit), macrolide-lincosamide-streptogramin resistance genes (**ermA, ermB, ermC**), tetracycline resistance genes (**tetK, tetM**), aminoglycoside-modifying enzyme genes (**aac(6')-aph(2")),** and vancomycin resistance operon genes (**vanA, vanB**) if phenotypic resistance suggested their presence. PCR primers, cycling conditions, and positive/negative controls were used as described in validated protocols; a subset of PCR products were

Sanger-sequenced for confirmation and submitted to GenBank or kept for internal sequence comparison. Quality control included known reference strains for mecA-positive and mecA-negative controls.

Available surgical or biopsy tissue from infection sites were fixed in 10% neutral buffered formalin, processed, embedded in paraffin, and stained with hematoxylin & eosin (H&E). Additional special stains (e.g., Gram stain, Masson's trichrome) were used as indicated. A board-certified pathologist blinded to the molecular results examined slides and graded features including necrosis. neutrophilic infiltrate, abscess formation, vasculitis/vascular invasion, fibrin deposition, and evidence of chronic inflammation or granulation tissue. A semi-quantitative scoring system (0–3) was applied for each feature to enable statistical correlation with molecular profiles. When tissue Gram stain or immunohistochemistry was available, bacterial localization was recorded.

A standardized proforma was used to record patient demographics (age, sex), clinical data (inpatient vs outpatient, ward, underlying comorbidities, prior antibiotic exposure within 3 months), specimen type and date, AST results, molecular test results (genes detected), and histopathology scores. Laboratory results were cross-checked against hospital records to ensure accuracy. All collected data were entered into a secure electronic database (password-protected Excel or REDCap) and coded for analysis. Periodic data audits were performed to ensure completeness and to reconcile any discrepancies between culture, molecular, and histopathology records.

Data were exported to SPSS 25 for analysis. Descriptive statistics are presented as frequencies and percentages for categorical variables and mean ± standard deviation or median (IQR) for continuous variables depending on distribution. The prevalence of specific resistance genes among MRSA isolates was calculated with 95% confidence intervals. Associations between presence of resistance genes and categorical clinical/pathological variables (e.g., severe necrosis, abscess formation, prior antibiotic use) were assessed using Chi-square or Fisher's exact test as appropriate. Continuous histopathology scores were compared between gene-positive and gene-negative groups using t-test or Mann–Whitney U test based on normality. Multivariable logistic regression analysis was performed to identify independent predictors of severe histopathological features (e.g., severe necrosis or vascular invasion), adjusting for confounders such as age, comorbidity, specimen source, and prior antibiotic exposure. Odds ratios (ORs) with 95% CIs were reported. A two-tailed p-value <0.05 was considered statistically significant.

3. RESULTS

A total of 406 MRSA isolates were analyzed. The mean age of patients was 42.6 ± 18.7 years, with males slightly more represented than females. The majority of cases were from inpatients (68.7%), and wound swabs/tissue specimens accounted for the largest proportion of isolates. Prior antibiotic use within the last three months was common (62.1%), and diabetes mellitus emerged as the most frequent comorbidity (26.6%) (Table 1).

Molecular analysis confirmed the presence of the **mecA gene in all isolates,** consistent with methicillin resistance. A small subset (3%) also carried mecC. Among non-β-lactam resistance determinants**, ermA (31%) and ermC (24.1%) were the most frequent,** followed by tetracycline resistance genes tetK (20.9%) and tetM (18.2%). Aminoglycoside resistance was mediated by aac(6')-aph(2'') in 16.5% of isolates, while vancomycin resistance genes were rare (vanA 2.2%, vanB 0.7%) (Table 2).

When correlated with histopathology, the presence of resistance genes showed significant associations with tissue damage. **Severe necrosis was observed in nearly half of gene-positive cases compared with only 22.5% of gene-negative cases ($p < 0.001$).** Abscess formation ($p = 0.002$) and vascular invasion ($p = 0.004$) were also significantly more frequent in gene-positive isolates. Chronic inflammation, however, did not differ significantly between groups ($p = 0.482$) (Table 3).

Quantitative histopathology scoring further supported these findings. Gene-positive isolates demonstrated significantly higher mean necrosis scores (2.3 ± 0.7 vs. 1.6 ± 0.8, $p < 0.001$), abscess scores ($p = 0.003$), and vascular invasion scores ($p < 0.001$), indicating a stronger pathological impact of resistant strains (Table 4).

Multivariable logistic regression identified **ermA (OR 2.42, p = 0.001) and tetM (OR 1.87, p = 0.041)** as independent predictors of severe histopathological changes, even after adjusting for age, comorbidity, and prior antibiotic exposure. Prior antibiotic use itself was also a significant risk factor (OR 1.69, $p = 0.046$). Diabetes mellitus showed a trend towards significance but did not reach statistical cutoff ($p = 0.072$) (Table 5).

Table 1. Demographic and Clinical Characteristics of Patients with MRSA Isolates (n = 406)

Variable	Mean ± SD/ n(%)
Age (years), mean ± SD	42.6 ± 18.7
Gender	

Male	238 (58.6%)
Female	168 (41.4%)
Inpatient vs Outpatient	
Inpatient	279 (68.7%)
Outpatient	127 (31.3%)
Common Specimen Types	
Wound swabs/tissue:	175 (43.1%)
Blood	123 (30.3%)
Respiratory samples	73 (18.0%)
Urine	35 (8.6%)
Prior Antibiotic Use (last 3 months)	
Yes	252 (62.1%)
No	154 (37.9%)
Major Comorbidities	
Diabetes:	108 (26.6%)
CKD	42 (10.3%)
Malignancy	36 (8.9%)
None	220 (54.2%)

Table 2. Distribution of Antibiotic Resistance Genes among MRSA Isolates (n = 406)

Resistance Gene	Positive, n (%)
mecA	406 (100%)
mecC	12 (3.0%)
ermA	126 (31.0%)
ermC	98 (24.1%)
tetK	85 (20.9%)
tetM	74 (18.2%)
aac(6')-aph(2'')	67 (16.5%)
vanA	9 (2.2%)
vanB	3 (0.7%)

Table 3. Correlation of Resistance Genes with Histopathological Findings

Histopathological Feature	Gene-Positive Cases (%)	Gene-Negative Cases (%)	p-value
Severe Necrosis	62 (49.2%)	54 (22.5%)	<0.001
Abscess Formation	71 (56.3%)	87 (36.3%)	0.002

Vascular Invasion	29 (23.0%)	25 (10.4%)	0.004
Chronic Inflammation	88 (69.8%)	158 (65.9%)	0.482

Table 4. Comparison of Histopathology Severity Scores between Gene-Positive and Gene-Negative Groups

Histopathology Score (0–3)	Gene Positive (Mean ± SD)	Gene Negative (Mean ± SD)	Test Statistic	p-value
Necrosis Score	2.3 ± 0.7	1.6 ± 0.8	t = 6.28	<0.001
Abscess Score	2.1 ± 0.9	1.7 ± 0.8	U = 9483	0.003
Vascular Invasion Score	1.4 ± 0.6	0.9 ± 0.5	t = 5.11	<0.001

Table 5. Multivariable Logistic Regression Predicting Severe Histopathological Features (Severe Necrosis or Vascular Invasion as Outcome)

Predictor Variable	Adjusted OR	95% CI	p-value
Presence of ermA gene	2.42	1.41 – 4.14	0.001
Presence of tetM gene	1.87	1.02 – 3.42	0.041
Prior antibiotic use (Yes vs No)	1.69	1.01 – 2.83	0.046
Age > 50 years	1.15	0.67 – 1.97	0.612
Diabetes mellitus	1.73	0.95 – 3.13	0.072

4. DISCUSSION

In this cross-sectional study of 406 MRSA isolates we found universal detection of **mecA,** low-level presence of **mecC** (3%), and frequent carriage of non–β-lactam resistance determinants (ermA 31%, ermC 24.1%, tetK 20.9%, tetM 18.2%). These molecular patterns broadly align with several Pakistani reports that document high mecA prevalence and variable rates of additional resistance genes, although absolute proportions differ between studies. For example, a 2021 genotypic characterization of clinical *S. aureus* from Pakistan reported widespread mecA carriage with multiple SCCmec types and frequent multidrug resistance, consistent with our finding of a dominant mecA background underpinning MRSA in hospital settings.(12)

Relative to the literature on mecC, our low mecC frequency (3%) is also consistent with recent Pakistani surveillance that found mecC to be uncommon but detectable in a minority of isolates. Idrees et al. (2023) reported both mecA and sporadic mecC detection in southern Punjab, emphasizing that mecC remains rare but should not be ignored in molecular surveillance programs. The presence of mecC in a small subset of isolates in our data supports routine inclusion of mecC-targeted assays in comprehensive molecular workflows.(13)

Our observed frequencies of macrolide resistance genes (ermA/ermC) and tetracycline genes (tetK/tetM) are comparable to multiple Pakistani and regional studies that document frequent co-carriage of non–β-lactam resistance determinants in MRSA, reflecting extensive antibiotic pressure in clinical practice. Khan et al. (2021) described multidrug-resistant MRSA clones in clinical settings with coexisting aminoglycoside, macrolide, and tetracycline resistance determinants—a pattern that mirrors our detection of aac(6′)-aph(2″) (16.5%) alongside erm and tet genes. Such co-resistance can complicate empiric therapy and explains why we saw rare but present van genes only in a very small fraction of isolates.(12)

Comparing our pathology correlations with prior work, the strong association between resistance-gene carriage and **increased tissue damage** (higher necrosis, abscess formation and vascular invasion; necrosis $p < 0.001$, abscess $p = 0.002$, vascular invasion $p = 0.004$) extends the clinical concern beyond treatment failure to more severe host pathology. Direct histopathological correlation studies of MRSA genotypes are scarce, but other investigations have linked particular virulence genotypes/clones with more aggressive clinical presentations. For example, studies that combined molecular typing with clinical outcomes reported that multidrug-resistant clones frequently produce more severe soft-tissue disease and are tied to higher rates of invasive infection—this finding complements our histologic observations.(14, 15)

Our multivariable model identified **ermA (adjusted OR 2.42, p = 0.001)** and **tetM (adjusted OR 1.87, p = 0.041)** as independent predictors of severe histopathological features, while prior antibiotic exposure was an additional independent risk factor (OR 1.69, p = 0.046). These results suggest that resistance determinants—or the genetic backgrounds that carry them—may be markers for more pathogenic clones or that prior antibiotic pressure selects for strains that both resist therapy and cause worse tissue injury. Similar inferences have been drawn in genomic outbreak and molecular epidemiology studies elsewhere: investigations using whole-genome methods have shown that certain resistance-associated lineages are also enriched for virulence determinants and for nosocomial transmission potential, producing both clinical severity and treatment challenges.(14, 16)

When placed in a regional and global context, some differences are apparent. Community and livestock-associated MRSA surveys from Pakistan and neighboring regions have reported variable mecA prevalence and different distributions of SCCmec types and accessory resistance genes, likely reflecting local antibiotic usage patterns, infection-control practices, and sampling frames (clinical vs. food/animal vs. environmental). For example, a 2020 Frontiers report from Rawalpindi–Islamabad showed MRSA presence in nonclinical reservoirs (meat and slaughterhouse samples), underscoring environmental reservoirs that could seed clinical strains and influence the local resistance gene pool. Our study's focus on clinical isolates from tertiary hospitals naturally yields higher multidrug-resistance rates than community/environmental surveys.(15)

There are also close parallels with international studies that document low but important prevalence of vancomycin resistance genes and occasional mecC-positive strains; these global findings underscore the need for vigilance even when such genes are rare. Studies from other low- and middle-income settings (e.g., Nigeria, Iran) report similar challenges of co-resistance and the emergence of VRSA/van gene carriage in small subsets, again matching our detection of vanA/vanB as rare events (vanA 2.2%, vanB 0.7%). Such cross-country comparisons suggest that while mecA remains the dominant mechanism for methicillin resistance, the accessory resistome shows local heterogeneity.(17)

Limitations of our study should temper interpretation. First, although our sample size was large and powered for prevalence estimates, the cross-sectional design prevents causal inferences about whether gene carriage *causes* more severe histopathology or whether both reflect underlying clone virulence and host factors. Second, we used targeted PCR for canonical resistance genes rather than whole-genome sequencing (WGS); WGS would better resolve clonal relationships, mobile elements (SCCmec subtypes, plasmids, prophages), and linkage between resistance and virulence genes, an approach used in recent outbreak/genomic studies that has clarified transmission dynamics and pathogenicity. Finally, our isolates came from tertiary hospitals and therefore may over-represent more severe or treatment-exposed cases compared with community samples; this selection bias likely contributes to higher multidrug resistance rates relative to nonclinical surveys.

5. CONCLUSION

Our findings of universal mecA carriage, frequent co-resistance genes (erm, tet, aac), and a clear association between resistance gene carriage and more severe histopathological injury (notably necrosis and vascular invasion) are concordant with the regional molecular-epidemiologic literature and with international genomic reports of aggressive multidrug-resistant MRSA clones. The results reinforce the need for routine molecular surveillance (including mecC and van gene screening), integration of molecular typing or WGS into infection-control programs, and stewardship interventions to reduce antibiotic pressure that selects for highly resistant, pathogenic strains. Future work should combine WGS, virulence profiling, and prospective clinical outcome measures to disentangle whether resistance genes are causal mediators of tissue damage or markers of virulent lineages.

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