ORIGINAL ARTICLE

Molecular Characterization of Methicillin-Resistant *Staphylococcus aureus* Isolates from Two Tertiary Hospitals in Thailand

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Background: Methicillin-resistant *Staphylococcus aureus* (MRSA) remains a major cause of healthcare-associated infections with substantial morbidity and mortality. Optimizing treatment and infection-control strategies is crucial to minimizing its spread in healthcare settings.

Objective: To characterize MRSA isolates from two tertiary hospitals in Thailand, including Thammasat University Hospital (TU) and Songklanagarind Hospital (PSU) at the molecular level, and to identify clinical and microbiological factors associated with treatment failure.

Materials and Methods: Whole genome sequencing (WGS) was performed using the Illumina MiSeq. SCC*mec*Finder, PubMLST, and *spa*Typer were used to determine SCC*mec*, MLST, and *spa* types, respectively. Phylogenetic trees were constructed with RaxML (54 samples). Vancomycin MICs were determined by the broth microdilution (BMD) method, and susceptibility data for other antibiotics (by disk diffusion method) were obtained from hospital medical records. Clinical and microbiological data were analyzed using STATA, version 17. The isolates and medical records were provided from both hospitals with official permission.

Results: The predominant genotypes were SCC*mec* type IV, ST239, and t037, with CC8 being the most common clonal complex in both hospitals. Most isolates were vancomycin-susceptible *S. aureus* (VSSA). However, a few heteroresistant vancomycin-intermediate *S. aureus* (hVISA) strains were detected. Isolates were highly susceptible to fusidic acid and linezolid but resistant to β-lactams, macrolides, and clindamycin. Pneumonia and acute kidney injury were independently associated with treatment failure.

Conclusion: The findings revealed multiple genetic lineages and identified clinical and microbiological characteristics of MRSA-infected patients associated with treatment outcomes. These results highlight the need for prompt and appropriate therapy, continuous susceptibility surveillance, and rigorous infection-control measures to prevent the spread of MRSA. In addition, the phylogenetic tree provided valuable insights into the evolutionary relationships among the isolates, facilitating the identification of clonal lineages and transmission patterns, which are essential for infection control and epidemiological surveillance.

Keywords: Methicillin-resistant *Staphylococcus aureus* (MRSA); Whole genome sequencing (WGS); Healthcare-associated infection (HAI); Vancomycin; Molecular typing; Clinical outcome

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Staphylococcus aureus is a common gram-positive pathogen that colonizes human skin and mucosa and can cause a wide range of infections, from mild skin and soft tissue infections to life-threatening diseases such as pneumonia, bacteremia, and endocarditis[1-3]. The emergence of methicillin-resistant *S. aureus* (MRSA) has become a major global health concern due to its ability to acquire multiple antibiotic resistance mechanisms, leading to limited treatment options and increased morbidity and mortality[4,5]. MRSA infections are associated with prolonged hospital stays, higher medical costs, and greater healthcare resource utilization compared with methicillin-susceptible *S. aureus* (MSSA) infections[5]. In Thailand, MRSA remains prevalent in hospital settings, with multiple clones circulating among healthcare facilities[6,7]. However, clinical predictors associated with treatment failure in MRSA-infected patients have not been comprehensively

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characterized.

At the molecular level, MRSA lineages are typically classified using staphylococcal cassette chromosome *mec* (SCC*mec*) typing, multilocus sequence typing (MLST), and *spa* typing, which provide essential insights into genetic diversity, antimicrobial resistance, and transmission pathways[8,9]. Integrating clinical and genomic data may help identify predictors of poor outcomes and guide infection-control strategies more effectively[10].

The objective of this research is to characterize MRSA strains from two tertiary hospitals at the molecular level and to analyze clinical and microbiological data, including patient demographics, underlying conditions, infection site, and specific bacterial characteristics (such as antibiotic resistance), associated with treatment outcomes. These findings are essential for optimizing treatment and preventing the further spread of MRSA in healthcare settings.

Materials and Methods

Bacterial isolates

Eighty-one MRSA isolates were obtained from clinical specimens collected at two tertiary hospitals, including Thammasat University Hospital (TU) with 33 specimens (collected between 2012 and 2021) and Songklanagarind Hospital, Prince of Songkla University (PSU) with 48 specimens (collected between 2019 and 2021).

Clinical data collection and statistical analysis

Clinical and microbiological data, including demographic characteristics, comorbidities, antimicrobial susceptibility profiles, and clinical outcomes, were retrieved from the Microbiology laboratories at TU and PSU. All statistical analyses were performed using Stata Statistical Software, version 17 (StataCorp LLC, College Station, TX, USA). Variables with p-values less than 0.10 in univariable analysis were entered into multivariable logistic regression models to identify independent predictors of clinical failure among MRSA-infected patients. Results were presented as adjusted odds ratios (aORs) with 95% confidence intervals (CIs), and p-values less than 0.05 were considered statistically significant. Records with missing data from two patients were excluded from the analysis.

Antimicrobial susceptibility test

Minimum inhibitory concentrations (MICs) of vancomycin were determined using the broth microdilution method in accordance with Clinical and Laboratory Standards Institute (CLSI) guidelines[11]. Antimicrobial susceptibility data for other antibiotics determined by the disk diffusion method were obtained from the medical records of the two hospitals. The disk diffusion test was performed according to CLSI guidelines[12]. The antibiotics tested included vancomycin, sulfamethoxazole/trimethoprim, fusidic acid, rifampicin, trimethoprim, cefpirome, clindamycin, linezolid, mupirocin, teicoplanin, azithromycin, erythromycin, penicillin, oxacillin, and tetracycline. The MRSA strains showing vancomycin MIC values of 1.5 to 2 µg/mL were considered to exhibit MIC creep.

Population analysis profile-area under the curve (PAP-AUC)

The presence of heteroresistant vancomycin-intermediate *S. aureus* (hVISA) was confirmed using the modified PAP-AUC method[13]. MRSA isolates were serially diluted and plated on brain heart infusion (BHI) agar containing vancomycin concentrations ranging from 0 to 8 µg/mL. After incubation at 35°C for 48 hours, colony counts (log_{10} CFU/mL) were plotted against vancomycin concentrations to calculate the area under the curve (AUC). Isolates with an AUC ratio of 0.9 or more relative to the hVISA reference strain Mu3 were defined as hVISA.

Molecular typing

Genomic DNA was extracted using the GF-1 Bacterial DNA Extraction Kit (Vivantis, Malaysia) according to the manufacturer's instructions. The presence of the *mecA* gene was confirmed by polymerase chain reaction (PCR) using specific primers. Sequencing libraries were prepared using the Nextera XT DNA Library Preparation Kit, and sequencing was performed on an Illumina MiSeq platform to generate 250 bp paired-end reads. Raw reads were quality-checked with FastQC and trimmed using Trimmomatic. High-quality reads were then de novo assembled using SPAdes, and genome annotation was conducted with Prokka. SCC*mec* typing was performed using SCC*mec*Finder, while *spa* typing was determined by sequencing the polymorphic X region of the *spa* gene and assigning *spa* types with *spa*Typer. MLST was determined based on the *S. aureus* PubMLST database (https://pubmlst.org/organisms/staphylococcus-aureus). Phylogenetic relationships were inferred from core-genome single-nucleotide polymorphism (SNP) analysis using RAxML, and clustering and

Table 1. Comparison of clinical and microbiological characteristics between patients with different clinical outcomes of MRSA infection

Characteristics	Total (n=79)	Treatment		p-value
		Failure	Success	
Clinical characteristics				
Sex; n (%)				0.427
• Male	47 (59.5)	15 (31.9)	32 (68.1)	
• Female	32 (40.5)	13 (40.6)	19 (59.4)	
Age (year); mean±SD	52.40±25.96	51.58±29.74	52.85±23.95	0.836
Length of stay (day); median (IQR)	23 (10, 37)	21 (3, 34)	23 (11, 37)	0.569[m]
Prior MRSA infection in previous 3 months; n (%)	15 (19.0)	7 (25.0)	8 (15.7)	0.313
Prior vancomycin exposure; n (%)	13 (16.5)	4 (14.3)	9 (17.7)	0.763
Hospitalization within 1 month of MRSA infection; n (%)	22 (27.9)	8 (28.6)	14 (27.5)	0.915
Hospitalization >3 months of MRSA infection; n (%)	5 (6.3)	4 (14.3)	1 (2.0)	0.051[f]
ICU admission; n (%)		4 (14.3)	7 (13.7)	1.000[f]
Concurrent conditions; n (%)				
Chronic kidney disease	17 (21.5)	6 (21.4)	11 (21.6)	0.988
Acute kidney injury	12 (15.2)	9 (32.1)	3 (5.9)	0.003[f]*
Chronic hemodialysis	4 (5.1)	2 (7.1)	2 (3.9)	0.612[f]
Cerebrovascular disease	14 (17.7)	5 (17.9)	9 (17.7)	0.981
Cardiovascular disease	19 (24.1)	3 (10.7)	16 (31.4)	0.054[f]
Congestive heart failure	5 (6.3)	2 (7.1)	3 (5.9)	1.000[f]
Cirrhosis liver cirrhosis	4 (5.1)	2 (7.1)	2 (3.9)	0.612[f]
Diabetes mellitus	22 (27.9)	5 (17.9)	17 (33.3)	0.142
Cancer	23 (29.1)	12 (42.9)	11 (21.6)	0.046*
Recent surgery	24 (30.4)	6 (21.4)	18 (35.3)	0.2
Transplant	1 (1.3)	1 (3.6)	0 (0.0)	0.354[f]
Immunosuppressant	6 (7.6)	3 (10.7)	3 (5.9)	0.660[f]
Implanted device	16 (20.3)	6 (21.4)	10 (19.6)	0.847
Bedridden	11 (13.9)	4 (14.3)	7 (13.7)	1.000[f]
Drug; n (%)				
Adequate glycopeptide use within 48 hours	12 (15.2)	1 (3.6)	11 (21.6)	0.047[f]*
Adjustment of vancomycin trough level	11 (13.9)	5 (17.9)	6 (11.8)	0.45
Infectious source; n (%)				
Pneumonia	26 (32.9)	15 (53.6)	11 (21.6)	0.004*
Wound/skin or soft tissue	28 (35.4)	13 (46.4)	15 (29.4)	0.130
Bone/joint	6 (7.6)	0 (0.0)	6 (11.8)	0.084[f]
Surgical site	7 (8.9)	0 (0.0)	7 (13.7)	0.047[f]*
Central venous	5 (6.3)	0 (0.0)	5 (9.8)	0.155[f]
Others	37 (46.8)	10 (35.7)	27 (52.9)	0.142
Microbiological characteristics				
Co-microbe resistant strains infection; n (%)	47 (59.5)	19 (67.9)	28 (54.9)	0.261
Days to negative culture (days); median (IQR) (n=37)	11 (6, 18)	15 (8, 23)	8 (5, 17)	0.094[m]

MRSA=Methicillin-resistant *Staphylococcus aureus*; ICU=intensive care unit; SD=standard deviation; IQR=interquartile range

Statistical analyses were performed using Fisher's exact test (f), chi-square test, independent t-test, and Mann-Whitney U test (m), * Statistically significant at the 0.05 level (α=0.05)

visualization were generated with iTOL.

Ethical approval

The present study was approved by the Human Research Ethics Committee of Thammasat University (Medicine), approval number 219/2563.

Results

Analysis of clinical and microbiological data revealed that clinical treatment failure was significantly associated with acute kidney injury (p=0.003), cancer (p=0.046), and pneumonia (p=0.004), as shown in Table 1.

Table 2. Multivariable analysis of factors associated with clinical failure in patients with MRSA infections

Factors	Total (n=79)		Male (n=47)		Female (n=32)	
	Adjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Pneumonia	3.37 (1.13 to 10.01)	0.029*	7.16 (1.50 to 34.24)	0.014*	1.36 (0.21 to 8.38)	0.742
Acute kidney injury	7.80 (1.70 to 35.72)	0.008*	9.81 (1.48 to 65.18)	0.018*	8.35 (0.62 to 111.66)	0.109
Cancer	2.33 (0.74 to 7.28)	0.146	0.65 (0.11 to 3.70)	0.625	11.61 (1.64 to 82.01)	0.014*
p-value#	0.825		0.835		0.998	
Area under the curve value	0.746		0.787		0.779	

OR=odds ratio; CI=confidence interval

Statistical analyses were performed using multiple binary logistic regression, # p-value indicated by Hosmer-Lemeshow goodness-of fit, * Statistically significant at the 0.05 level (α=0.05)

Multivariable logistic regression was performed to identify independent factors associated with clinical failure. The analysis demonstrated that pneumonia (adjusted OR 3.37, 95% CI 1.13 to 10.01, p=0.029) and acute kidney injury (adjusted OR 7.80, 95% CI 1.70 to 35.72, p=0.008) remained independent predictors of clinical failure. Interestingly, in sex-stratified analyses, cancer was an independent predictor of treatment failure among females (adjusted OR 11.61, 95% CI 1.64 to 82.01, p=0.014), whereas pneumonia was an independent predictor among males (adjusted OR 7.16, 95% CI 1.50 to 34.24, p=0.014), as presented in Table 2. The discriminative ability of the model was evaluated using the AUC. The AUC values were 0.7460 (95% CI 0.6131 to 0.8789) for the total population, 0.7875 (95% CI 0.6119 to 0.9631) for males, and 0.7794 (95% CI 0.5839 to 0.9750) for females. These values indicate acceptable discriminative performance in predicting clinical failure among MRSA patients (p<0.001).

The majority of isolates from both hospitals were classified as vancomycin-susceptible *S. aureus* (VSSA), indicating that VSSA was the predominant phenotype across both sites.

MRSA isolated from TU and PSU were tested for antimicrobial susceptibility. All isolates remained fully susceptible to vancomycin, while fusidic acid maintained consistently moderate to high activity. Linezolid, tested only in TU, demonstrated complete susceptibility, and mupirocin remained active. In contrast, widespread resistance to β-lactam antibiotics (penicillin, oxacillin, and cefpirome) and macrolides (erythromycin and azithromycin) was evident in both centers. Notably, PSU isolates displayed particularly high resistance to clindamycin and erythromycin. Resistance to sulfamethoxazole or trimethoprim, rifampicin, and tetracycline was also observed (Table 3).

Vancomycin MIC testing showed that most isolates from both TU (87.9%) and PSU (85.4%) had MIC values within 0.25 to 1 μg/mL, whereas a smaller proportion exhibited elevated MICs of 1.5 to 2 μg/mL (TU: 12.1% and PSU: 14.6%). Slightly higher frequencies of elevated MICs were observed among isolates from PSU compared with TU. In addition, the strains from TU and PSU were found hVISA 3.0% and 2.4%, respectively (Figure 1).

Table 3. Antimicrobial susceptibility test of MRSA strains isolated from TU and PSU

	Resistant n (%)	No of tested strains n
TU strains (n=33)		
Antibiotic		
• Vancomycin	0 (0.0)	16
• Sulfamethoxazole trimethoprim	7 (21.9)	32
• Fusidic acid	4 (13.3)	30
• Rifampicin	5 (31.3)	16
• Trimethoprim	10 (62.5)	16
• Cefpirome	16 (100)	16
• Clindamycin	7 (21.2)	33
• Linezolid	0 (0.0)	33
• Mupirocin	1 (6.3)	16
• Teicoplanin	0 (0.0)	16
• Azithromycin	7 (41.2)	17
• Erythromycin	7 (41.2)	17
• Penicillin	14 (82.4)	17
• Oxacillin	10 (58.8)	17
• Tetracycline	2 (12.5)	16
PSU strains (n=48)		
Antibiotic		
• Clindamycin	33 (100)	33
• Erythromycin	45 (93.8)	48
• Fusidic acid	1 (2.2)	46
• Fosfomycin	0 (0.0)	27
• Vancomycin	0 (0.0)	42
• Oxacillin	47 (100)	47

TU=Thammasat University Hospital; PSU=Songklanagarind Hospital

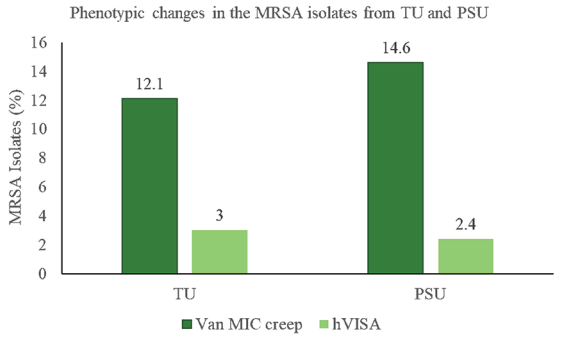

Figure 1. Phenotypic changes in the MRSA isolates from TU and PSU.

Table 4. Prevalence of PVL genes among SCC*mec* type IV MRSA isolates

MRSA strains	Total SCC*mec* type IV n	Complete PVL (lukS/F) n (%)	Incomplete PVL (lukF only) n (%)
TU	4	3 (75.0)	1 (25.0)
PSU	8	4 (50.0)	4 (50.0)

MRSA=Methicillin-resistant *Staphylococcus aureus*; TU=Thammasat University Hospital; PSU=Songklanagarind Hospital
Complete PVL: TU13, TU14, TU15, PSU39, PSU41, PSU44, PSU45
Incomplete PVL: TU7, PSU1, PSU2, PSU25, PSU26

Analysis of the correlation between genomic data and clinical characteristics revealed significant findings. Notably, within the SCC*mec* type IV group, 50% of the isolates were identified as ST8 and carried the complete pvl gene (lukS/F-PV). This specific cluster was strongly associated with severe clinical pathologies, including pneumonia observed in the TU strains, including TU13, TU14, and TU15, and necrotic skin infections, such as the dry gangrene detected in the PSU45, as shown in Table 4.

However, pathogenicity was not exclusively dependent on the presence of the pvl gene. In cases where pvl was incomplete (lukF-PV) but clinical symptoms were severe (TU7), the presence of superantigens was noted, suggesting these may serve as alternative key factors in triggering inflammation. The predominance of SCC*mec* type IV, combined with a diverse array of virulence factors, underscores the circulation of highly pathogenic strains within the hospital setting. This necessitates rigorous and continuous surveillance.

SCC*mec* typing of MRSA isolates from TU (29 isolates) and PSU (35 isolates) revealed distinct distributions between the two tertiary hospitals. Among TU isolates, type IV was most frequent (37.9%), followed by type II (10.3%) and type I (3.4%). In contrast, PSU isolates were type IV (22.9%), followed by type I (14.3%) and type II (5.7%). These findings indicate that SCC*mec* type IV was the leading genotype in both hospitals, although the relative proportions differed between the two settings (Figure 2B).

MLST demonstrated diverse clonal lineages among the 64 MRSA isolates. The most frequent sequence types in the TU strains were ST239 (27.6%), followed by ST22 (24.1%), ST8 (10.3%), and ST764 (10.3%). Similarly, ST239 was predominant at 25.7% among PSU strains, followed by ST1232 (11.4%), ST8 (8.6%), and ST188 (5.7%). These results indicated that ST239 remains the major MRSA lineage circulating in both hospitals, whereas other sequence types varied in prevalence between sites (Figure 2A).

The *spa* typing revealed high genetic heterogeneity among MRSA isolates from both hospitals. The predominant *spa* types in TU were t037 (27.6%) and t032 (24.1%), followed by t008 (10.3%) and t045 (10.3%). Similarly, in PSU isolates, t037 was most frequent (25.7%), followed by t001 (17.1%) and t571 (8.6%). Several additional *spa* types were sporadically detected in both hospitals. These findings highlighted the genetic diversity and complex clonal structure of MRSA circulating in clinical settings (Figure 2C).

Phylogenetic analysis of MRSA isolates from TU and PSU revealed six major clonal complexes as CC1, CC97, CC8, CC5, CC45, and CC22. These clusters demonstrated the genetic diversity and evolutionary relationships of MRSA circulating between the two hospitals. However, the phylogenetic tree did not clearly distinguish isolates with vancomycin MIC creep from those without creep, suggesting that this phenotype was not associated with a specific lineage (Figure 3).

Discussion

According to the results, MRSA infections in two tertiary hospitals in Thailand are influenced by both clinical factors and genetic diversity. Pneumonia and acute kidney injury were independent predictors of clinical failure, consistent with previous reports indicating that pulmonary involvement and renal dysfunction contribute to worse outcomes in MRSA infections[14,15]. In this cohort, cancer also emerged as a predictor of poor outcomes among female patients, reflecting increased susceptibility due to immunocompromised status, comorbidities, or sex-related physiological and healthcare-access differences. The observed sex-specific differences

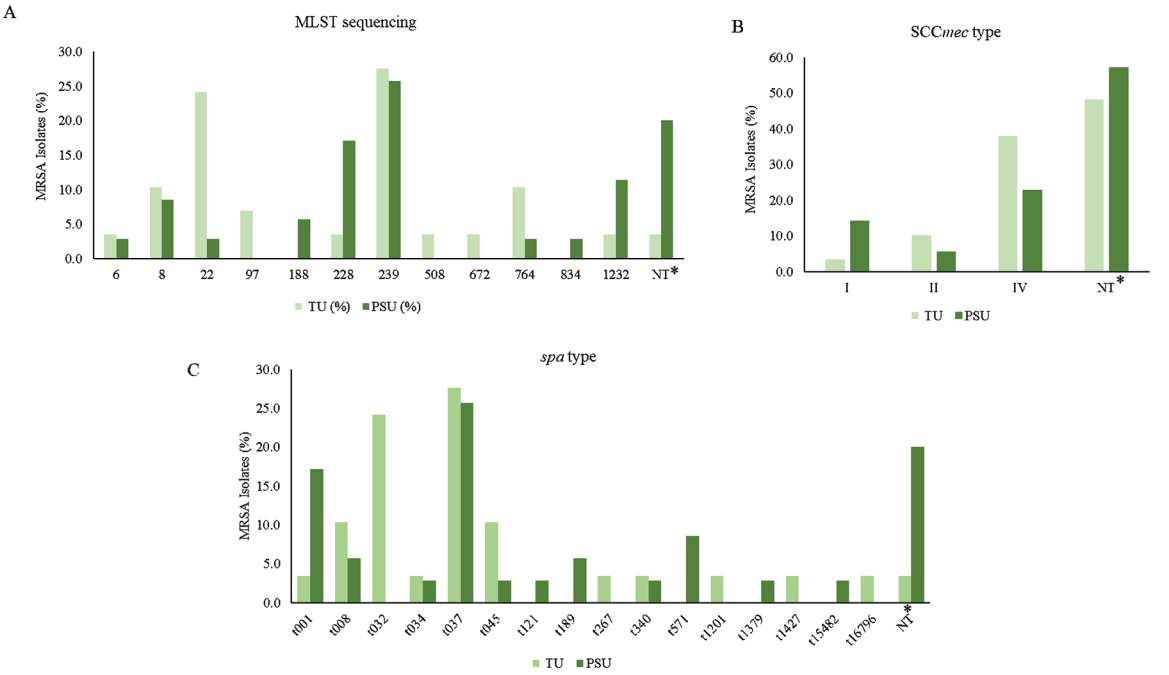

Figure 2. Distribution of molecular types of MRSA isolates from TU and PSU.

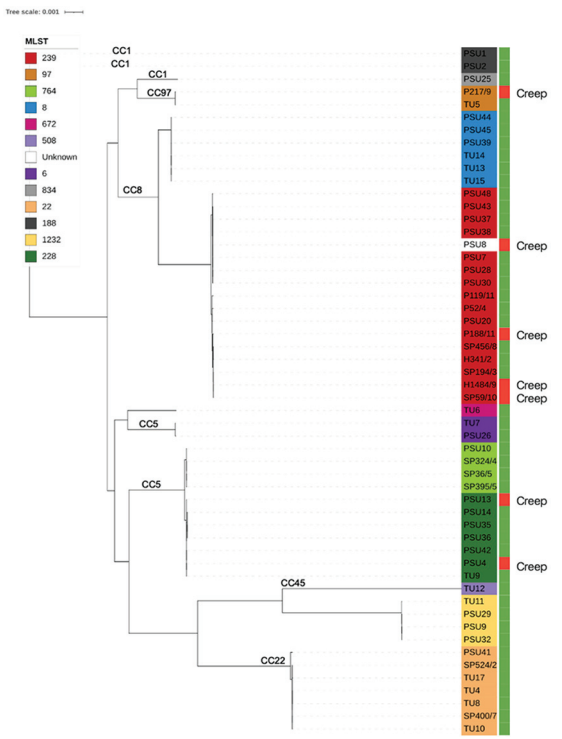

Figure 3. Phylogenetic tree of MRSA isolates generated and annotated using iTOL. The clonal complex (CC), sequence type (ST), and hospital origin (Thammasat University Hospital, TU, and Songklanagarind Hospital, PSU) are indicated.

in predictors of treatment failure highlight the need for tailored clinical management. Female patients with underlying malignancy may require intensified monitoring and early optimization of antimicrobial therapy due to immunocompromised status. In contrast, male patients with pneumonia may benefit from alternative agents with better pulmonary penetration, such as linezolid, to reduce the risk of treatment failure.

Pneumonia and surgical interventions were associated with microbiological failure, aligning with previous studies linking respiratory infections and invasive procedures to reduce bacterial clearance[16-18]. Distinct antimicrobial susceptibility profiles between the two hospitals, along with temporal shifts observed at TU, indicate that MRSA resistance is dynamic and locally variable. These findings are consistent with regional reports describing variations in clonal composition and antimicrobial resistance across Southeast Asia[19].

In this study, 41.2% of the TU isolates exhibited an oxacillin-susceptible phenotype despite being genetically confirmed as *mecA*-positive MRSA. This phenomenon, known as oxacillin-susceptible *mecA*-positive *S. aureus* (OS-MRSA), typically results from heterogeneous expression or low-level transcription of the *mecA* gene[20,21]. Clinically, these isolates are of high concern because they may be misidentified as

MSSA, leading to inappropriate beta-lactam therapy and potential treatment failure[22] (Table 3).

Molecular typing revealed SCC*mec* type IV and sequence type ST239 as the predominant clones, consistent with previous Thai and regional studies identifying ST239 as a major hospital-associated and multidrug-resistant MRSA lineage[23-25].

The prevalence of ST8-SCC*mec* IV harboring PVL in cases of severe pneumonia and dry gangrene aligns with the well-documented hypervirulence of the USA300 lineage[26,27]. However, the clinical severity observed in PVL-negative cases underscores the critical role of superantigens as alternative inflammatory drivers[28]. The circulation of these diverse virulent strains within hospital settings necessitates enhanced molecular surveillance[29].

These results underscore the need for continuous molecular surveillance and effective antimicrobial stewardship programs to monitor emerging MRSA lineages and to guide evidence-based empirical therapy. The findings highlight the importance of hospital-specific resistance monitoring and targeted infection-control measures to reduce treatment failure and prevent MRSA dissemination within Thai healthcare settings.

Limitation

The data are limited due to the small number of isolates from only two centers and the retrospective nature of data collection, which may not fully represent the nationwide MRSA epidemiology. Moreover, whole genome sequencing (WGS) was performed on a subset of isolates; therefore, some minor genotypes may have been underrepresented.

Conclusion

The findings highlight the importance of early recognition of pneumonia and comorbid conditions, along with prompt and appropriate treatment, to improve clinical outcomes. Vancomycin, teicoplanin, and linezolid remain key therapeutic options, emphasizing the need for continuous antimicrobial surveillance and stewardship to guide appropriate antibiotic use and control the MRSA spread.

MRSA infections in Thai tertiary hospitals are driven by both established and emerging clones, exhibit high-level resistance to multiple antibiotic classes, and are associated with defined clinical risk factors for treatment failure. Continuous monitoring of clinical risk factors, antibiotic susceptibility patterns, and molecular epidemiology is crucial to inform effective therapy and infection-control strategies.

Future nationwide surveillance integrating genomic and clinical data across multiple Thai hospitals will be essential to monitor the evolution of MRSA lineages, guide evidence-based treatment, and strengthen infection control policies.

What is already known about this topic?

MRSA isolates from patients at TU were previously studied at the molecular level between 2012 and 2015. However, WGS data were not determined. The later MRSA isolates exhibited distinct antibiograms, and isolates from PSU during the same period had never been characterized at the molecular level.

What does this study add?

This study provides a comprehensive genomic characterization of MRSA isolates using WGS to obtain detailed insights into their genetic diversity. In addition, it identifies risk factors associated with treatment failure, offering valuable evidence for surveillance, targeted infection-control measures, and the prevention of MRSA dissemination in Thai healthcare settings.

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Authors' contributions

LN performed experiments and clinical data analysis. PPh, WP, and WR carried out bioinformatics analyses. PP interpreted the results. SK designed and directed the research project. LN, PP, and SK wrote the main manuscript with input from all authors. All authors reviewed and approved the final manuscript.

Data availability statement

The remaining sequencing data for the other 54 isolates that support the findings of this study

are available from the corresponding author upon reasonable request.

Ethics approval and consent to participate

This study was approved by the Human Research Ethics Committee of Thammasat University (Faculty of Medicine) (Approval No. 219/2563). Informed consent was waived by the Institutional Review Board.

Clinical trial registration

This study is not a clinical trial; therefore, clinical trial registration is not applicable.

Use of artificial intelligence

No artificial intelligence (AI) tools were used to generate or analyze data in this study.

Conflicts of interest

The authors declare no conflict of interest.

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