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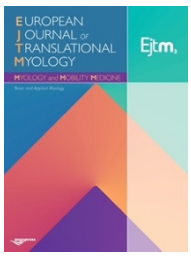

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Antimicrobial resistance and virulence gene patterns of *Staphylococcus aureus* in infectious mastitis: implications for inflammatory myopathies of the lactating breast

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Abstract

With the present work, we aimed to investigate antimicrobial resistance and virulence gene patterns of Staphylococcus aureus in lactating patients with infectious mastitis and evaluate their potential impact on inflammatory myopathies of the lactating breast. Between January 2021 and April 2024, 158 lactating patients with culture-confirmed infectious mastitis were treated at Hangzhou Linping District Maternal and Child Health Hospital. Among these, 119 isolates were identified as *S. aureus* (82 MRSA, 37 MSSA). Antimicrobial susceptibility and virulence genes were analyzed. Muscle involvement was inferred indirectly from clinical presentation, including marked local induration, tenderness extending to deeper breast tissue, and reduced breast mobility. No imaging or biopsy was performed to confirm myopathic changes directly. *S. aureus* was the predominant pathogen. Both MRSA and MSSA showed high resistance to penicillin G, erythromycin, and Clindamycin, while all isolates were susceptible to nitrofurantoin, linezolid, vancomycin, and rifampicin. MRSA exhibited higher resistance than MSSA ($p<0.05$). Frequent resistance genes included aac(6')/aph(2''), blaZ, mecA, aph(3')-III, and qacA/B. Virulence genes hla, clfA, clfB, and fnbA were common; pvl was less frequent in MRSA ($p<0.05$). MRSA infections were associated with stronger local inflammation and increased clinical markers possibly related to muscle involvement, raising the possibility of an association with myopathic changes in lactating breast tissue. *S. aureus*, particularly MRSA, is the main pathogen in lactating mastitis. Specific virulence genes may

influence the severity of local inflammation and myopathic changes, highlighting implications for inflammatory myopathies in the lactating breast.

Key words: lactation; infectious mastitis; *Staphylococcus aureus*; antimicrobial resistance; virulence factors; myopathies; muscle inflammation.

Infectious mastitis is a common condition with a high incidence among lactating women.[1] It is usually caused by poor milk drainage and improper breastfeeding techniques, leading to milk stasis and subsequent infection during lactation. Clinically, patients often present with breast erythema, swelling, pain, and fever, accompanied by palpable masses and difficulty in milk expression.[2,3] Pathogens may invade through erosions or wounds on the nipple, entering the subareolar lymphatic vessels and interlobular spaces, thereby causing breast cellulitis and seriously affecting breastfeeding.[4,5]

Staphylococcus aureus, a highly pathogenic Gram-positive bacterium, is a common causative agent of infectious mastitis in lactating women and can cause infections of the skin, soft tissues, bones, joints, and multiple organ systems. In recent years, the detection rate of Methicillin-Resistant *S. aureus* (MRSA) in breast milk and pus samples from lactating women has been increasing. MRSA is characterised by high levels of antimicrobial resistance and complex resistance mechanisms, which may contribute to increased infection-related mortality.[6]

Therefore, analysing the pathogen spectrum and antimicrobial resistance profiles in lactating patients with infectious mastitis is of great clinical significance for guiding the rational use of antibiotics. Previous studies[7,8] have shown that the pathogenicity of *S. aureus* is closely related to

the virulence genes it carries. Thus, investigating the distribution of virulence genes in MRSA and Methicillin-Susceptible *S. aureus* (MSSA) is essential. Based on this, the present study aimed to analyse the antimicrobial susceptibility and virulence gene profiles of *S. aureus* isolated from lactating patients with infectious mastitis, providing a reference for clinical diagnosis and treatment in this population, Notably, *S. aureus* infections are also implicated in muscle complications such as pyomyositis and inflammatory myopathies, suggesting that mastitis caused by virulent strains may have broader systemic muscular implications. The findings are presented as follows.

Materials and Methods

Clinical data

A total of 158 lactating patients with culture-positive infectious mastitis, admitted to Hangzhou Linping District Maternal and Child Health Hospital between January 2021 and April 2024, were included in this study. Among them, Staphylococcus aureus was isolated in 119 cases, comprising 82 strains of MRSA and 37 strains of MSSA. Bilateral involvement was observed in 37 patients, and unilateral involvement in 121. Patients ranged in age from 20 to 45 years, with a mean age of (30.67 ± 2.58) years. Among them, 66 were outpatients and 92 were hospitalized.

Inclusion criteria

All patients met the diagnostic criteria for infectious mastitis during lactation,[9] including breast pain, poor milk drainage, local masses in the mammary gland, and signs of inflammation in the affected area (increased skin temperature, redness, swelling, and tenderness). Systemic symptoms included fever, chills, generalized sweating, dizziness, and fatigue. All patients had positive pathogen cultures and no evidence of upper respiratory or other infections, fever, or pre-existing mastitis prior to lactation. Coagulation function was within the normal range.

Exclusion criteria

Patients were excluded if they had inflammatory breast cancer, non-lactational mastitis, untreated breast tuberculosis or other unresolved breast diseases, severe dysfunction of major organs (heart, liver, kidney), coexisting malignancies, recent use of immunosuppressants or antimicrobial agents, or a history of immunological disorders.

This study was approved by the Medical Ethics Committee of our hospital.

Detection of S. aureus *resistance and virulence genes*

Colonies of *S. aureus* grown on sheep blood agar were selected and transferred to centrifuge tubes containing double-distilled water. After mixing, proteinase K (0.2 mg/mL) and lysostaphin (16 U/mL) were added. The mixture was incubated at 37 °C for 1 hour. Bacterial DNA was extracted using the boiling method. Briefly, a bacterial colony was transferred into 600 μL of lysis buffer, vortexed thoroughly, and boiled for 15 minutes. The sample was then cooled and centrifuged at 12,000 rpm for 10 minutes. The supernatant was collected and adjusted to a DNA concentration of 50–100 ng/μL to serve as the PCR template.

Polymerase Chain Reaction (PCR) was employed to detect the expression of resistance and virulence genes in S. aureus. Primer sequences are shown in Table 1 and were designed and synthesized by GenScript Biotech (Nanjing, China).

The PCR reaction mixture consisted of: 2 μL of DNA template, 1 μL each of forward and reverse primers, and 12.5 μL of 2× SYBR Premix Ex Taq; double-distilled water was added to a final volume of 25 μL.

PCR conditions were as follows: initial denaturation at 94 °C for 5 minutes; 40 cycles of denaturation at 94 °C for 30 seconds, annealing at 55 °C for 30 seconds, and extension at 72 °C for 1 minute; followed by a final extension at 72 °C for 10 minutes.

Distilled water served as the negative control, and target gene DNA fragments were used as positive controls. PCR products were subjected to 2% agarose gel electrophoresis and visualized using a gel documentation system.

Pathogen detection and antimicrobial susceptibility testing

In patients with suspected infection, breast milk or pus from the lactiferous ducts was collected prior to antimicrobial treatment after cleaning the nipple and surrounding skin. For patients with breast abscesses, puncture fluid or discharge from ruptured lesions was collected as the specimen. All samples were inoculated onto sheep blood agar and incubated at 37 °C for 24–48 hours. Bacterial identification was performed using a fully automated microbial identification system (VITEK-32, bioMérieux, France). Specimen collection and strain identification were conducted according to relevant standard operating procedures.[10] Pathogens were identified based on colony morphology, Gram staining, biochemical characteristics, and other features. Duplicate isolates from the same patient were excluded.

Antimicrobial susceptibility testing was performed using the disk diffusion method (OXOID, UK), and interpretative criteria were based on relevant references.[11] MRSA was defined as isolates with an oxacillin Minimum Inhibitory Concentration (MIC) ≥ 4 mg/L. Colonising strains and contaminants were excluded. Quality control strains were obtained from the ATCC culture collection, including *S. aureus* ATCC 25923, Klebsiella pneumoniae ATCC 700603, and Escherichia coli ATCC 25922.

Statistical analysis

A p-value < 0.05 was considered statistically significant. Statistical analyses were conducted using SPSS version 26.0. Categorical variables were expressed as n (%) and compared using the chi-square test. Continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm s$), and between-group comparisons were performed using the independent samples t-test. Normality was assessed by the Shapiro-Wilk test.

Results

Detection of pathogenic bacteria in infectious mastitis during lactation

A total of 158 pathogenic strains were isolated from 158 lactating patients with infectious mastitis. The majority were Gram-positive bacteria. Staphylococcus aureus was detected in 119 cases, accounting for the highest proportion (Table 2).

***Detection of drug resistance genes in* S. aureus**

Among the 119 *S. aureus* isolates, the most frequently detected resistance genes were *aac(6')/aph(2")*, *blaZ*, *mecA*, *aph(3')-III*, and *qacA/B*, with detection rates of 100.00%, 100.00%, 100.00%, 94.96%, and 84.87%, respectively (Table 3).

Antimicrobial resistance in MRSA and MSSA

Both MRSA and MSSA strains showed high resistance to penicillin G, erythromycin, and Clindamycin, while remaining susceptible to nitrofurantoin, linezolid, vancomycin, and rifampicin. Compared with MSSA, MRSA showed significantly higher resistance rates to Penicillin G, Erythromycin, Clindamycin, and Tetracycline ($p<0.05$) (Table 4).

Detection of virulence genes in MRSA and MSSA

Virulence genes hla, clfB, clfA, and fnbA were commonly detected in both MRSA and MSSA isolates. The detection rate of pvl was significantly lower in MRSA compared with MSSA ($p<0.05$). No statistically significant differences were observed for the other virulence genes ($p>0.05$).

Table 5. Detection of virulence genes in MRSA and MSSA [n (%)]. To provide a concise overview, the main resistance and virulence patterns are summarized in Table 6.

Discussion

Infectious mastitis during lactation is often caused by milk stasis resulting from inadequate milk drainage. The condition is closely associated with pathogen invasion, as microorganisms can directly enter the lactiferous ducts and ascend to the lobules where they proliferate. Additionally, the decomposition products of milk can serve as a nutrient medium for bacterial growth, which further facilitates infection.[12,13] Clinically, infectious mastitis in lactating women is characterized by local signs of inflammation such as redness, swelling, heat, and pain, and it can significantly impair breastfeeding, maternal well-being, and infant nutrition and health.[14,15]

The main pathogen involved is Staphylococcus aureus, which often carries multiple virulence genes and exhibits strong pathogenicity. Inadequate or delayed treatment may result in recurrence and pose serious health risks to both mother and infant.[16,17] Although a wide range of antibiotics is available for the treatment of infectious mastitis, the frequent use of broad-spectrum agents has led to increasing antimicrobial resistance. Therefore, understanding the distribution and resistance profiles of causative bacteria is essential for guiding rational antibiotic use in clinical practice.

This study investigated the antimicrobial susceptibility and virulence gene characteristics of *S. aureus* in lactating patients with infectious mastitis and yielded several important findings. Among the 158 strains isolated, Gram-positive bacteria were predominant, consistent with previous studies.[181,9] Based on susceptibility testing, targeted antimicrobial therapy may be applied.

We further found that *S. aureus* was isolated in 119 cases, with MRSA and MSSA both showing high resistance to penicillin G, erythromycin, and clindamycin, while remaining susceptible to nitrofurantoin, linezolid, vancomycin, and rifampicin. MRSA demonstrated significantly higher resistance rates to penicillin G, erythromycin, clindamycin, and tetracycline compared with MSSA. These findings suggest that clinicians should avoid the use of agents with high resistance rates—such as penicillin G, erythromycin, clindamycin, and tetracycline—when treating MRSA-related mastitis. Instead, antibiotics with higher susceptibility rates, including nitrofurantoin, linezolid, vancomycin, and rifampicin, may be more appropriate. The elevated resistance could be related to increased and repeated exposure to antimicrobials, resulting in structural changes in pathogens and

altered drug susceptibility. It should also be noted that certain antibiotics may be secreted into breast milk, and breastfeeding should be suspended during treatment when necessary.

The pathogenicity of *S. aureus* is determined by its virulence factors, which play roles in colonization, invasion, and proliferation. Multiple virulence factors act in concert and are closely associated with the development of infectious mastitis during lactation.[20,21] Previous studies[22] have shown that important virulence genes of S. aureus include those encoding enterotoxins, hemolysins, exfoliative toxins, leukocidins, and toxic shock syndrome toxin. Among these, Panton–Valentine Leukocidin (PVL) is an extracellular toxin capable of disrupting cell membranes, leading to cell lysis and more severe infections.[23] The hla gene encodes alpha-hemolysin, which induces hemolysis by inserting into the hydrophobic membrane of red blood cells and forming pores.[24] The clfB gene, part of the clumping factor adhesin family, is critical for *S. aureus* colonization and infection, particularly in lactating women. ClfA, another clumping factor, is a surface-associated protein that mediates bacterial adherence to host extracellular matrix.[25] FnbA encodes fibronectin-binding protein A, which promotes adhesion to fibronectin on host cells, facilitating tissue invasion.[26]

Our results showed high detection rates of hla, clfB, clfA, and fnbA in both MRSA and MSSA isolates. Interestingly, the detection rate of pvl was significantly lower in MRSA than in MSSA. This suggests that S. aureus strains causing infectious mastitis in lactating women often carry multiple virulence genes with strong pathogenic potential. The lower detection of pvl in MRSA may be attributed to the lysogenic conversion mechanism in MSSA via bacteriophage transduction, which enhances pvl expression and enables horizontal gene transfer among different strains. Therefore, greater attention should be paid to the presence of pvl in MSSA isolates.

Resistance genes such as aac(6′)/aph(2″) and aph(3′)-III are aminoglycoside resistance determinants. blaZ is associated with penicillin resistance. The mecA gene encodes Penicillin-Binding Protein 2a (PBP2a), an alternative form of the native penicillin-binding protein in *S. aureus*. The qacA/B genes encode efflux pump proteins that confer resistance to antiseptics and disinfectants, including quaternary ammonium compounds, guanidine derivatives, and biguanides.

In this study, the most frequently detected resistance genes in *S. aureus* were aac(6′)/aph(2″), blaZ, mecA, aph(3′)-III, and qacA/B, indicating that these genes are common in S. aureus strains isolated from lactating women with infectious mastitis. Although the presence of resistance genes does not always correspond directly to phenotypic resistance, it is likely influenced by the combined effects of multiple genes.

This study has several limitations, including a relatively small sample size and its single-center design, which may introduce bias and limit the generalizability of the findings. Despite these limitations, the study provides valuable insights into the antimicrobial susceptibility and virulence gene characteristics of *S. aureus* in lactating women with infectious mastitis. Future research with a larger and more diverse patient population is warranted to validate and extend these findings.

In conclusion, most pathogens isolated from lactating women with infectious mastitis were Gram-positive bacteria, with *S. aureus* being the most prevalent. MRSA exhibited higher resistance rates to penicillin G, erythromycin, clindamycin, and tetracycline than MSSA. The most frequently detected resistance genes included aac(6′)/aph(2″), blaZ, mecA, aph(3′)-III, and qacA/B. The pvl virulence gene was less frequently detected in MRSA than in MSSA. These findings may help guide targeted clinical management strategies to reduce the risk and progression of infectious mastitis during lactation.

As presented in Table 6, there was a marked resistance to penicillin G, erythromycin, and clindamycin, however, Vancomycin and rifampicin continued to be effective. hla, clfB, and clfA were virulence genes that were common and pvl was more common in MSSA. These patterns directly impact clinical management.

Our findings raise the possibility of muscular complications in severe *S. aureus* mastitis, although this link remains speculative. Certain virulence factors such as α-hemolysin and pvl have been implicated in tissue invasion and muscle injury in previous studies, but direct evidence from our cohort is lacking. Future investigations using muscle imaging, cytokine profiling, or biopsy samples are needed to confirm whether mastitis-related inflammation can contribute to secondary myopathic

changes.

Clinical implications

Our results are important for the treatment of lactational mastitis. Both MRSA and MSSA strains showed significant resistance to penicillin G, erythromycin, clindamycin, and tetracycline, indicating empirical treatment with these antibiotics is inappropriate. This is in line with more recent evidence showing the global ineffectiveness of macrolides and tetracyclines for MRSA.[27-29] On the other hand, all isolates were fully susceptible to vancomycin, rifampicin, linezolid, and nitrofurantoin, suggesting these antibiotics remain valid treatment options. Vancomycin is crucial for severe or treatment-resistant cases, and linezolid, although reserved for less severe cases, provides oral therapy and good tissue penetration.[30,31] While nitrofurantoin is rarely employed for mastitis, its reliable antibiotic action might make it useful in select cases. Additionally, virulence gene profiling may help determine the severity of the infection. The presence of pvl or hla is associated with severe inflammation and tissue necrosis.[30,32] Therefore, the clinician's awareness that strains with these virulence factors mastitis necessitate enhanced surveillance and, in some cases, more intensive treatment adjustments is important.

This is consistent with emerging literature on pyomyositis and infectious myopathies, where S. aureus virulence determinants are key drivers of muscle injury.[33]

From a translational standpoint, incorporating antimicrobial resistance data and virulence profiling into clinical decision-making may help optimize antibiotic selection, anticipate complications, and reduce recurrence rates. Future clinical guidelines should consider integrating such microbiological insights into standard mastitis management protocols.

Potential confounding factors, such as prior antibiotic exposure or breastfeeding practices, were not controlled in this analysis. This may have influenced resistance rates and clinical outcomes.

Given that this was a single-center study with a relatively small cohort, the results may not be completely generalizable. Patterns of resistance are shaped by local antibiotic prescribing habits as

well as local infectious disease trends. For this reason, the results should be applied with caution when considering populations beyond the scope of our study region.

List of Abbreviations

MRSA – Methicillin-Resistant Staphylococcus aureus

MSSA – Methicillin-Susceptible Staphylococcus aureus

PCR – Polymerase Chain Reaction

MIC – Minimum Inhibitory Concentration

PVL – Panton–Valentine Leukocidin

hla – Alpha-Hemolysin gene

clfA – Clumping Factor A gene

clfB – Clumping Factor B gene

fnbA – Fibronectin-Binding Protein A gene

aac(6')/aph(2") – Aminoglycoside Resistance Gene

blaZ – Beta-Lactamase Gene

mecA – Methicillin Resistance Gene

aph(3')-III – Aminoglycoside Resistance Gene

qacA/B – Quaternary Ammonium Compound Resistance Genes

SPSS – Statistical Package for the Social Sciences

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Conflict of interest

The authors declare no potential conflict of interest, and all authors confirm accuracy.

Ethics approval

This study was approved by the Medical Ethics Committee of Hangzhou Linping District Maternal and Child Health Hospital.

Informed consent

All patients participating in this study signed a written informed consent form for participating in this study.

Patient consent for publication

Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

Availability of data and materials

All data generated or analyzed during this study are included in this published article.

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Table 1. Primer sequences used for resistance and virulence gene detection in *S. aureus*.

Gene	**Forward primer (5′–3′)**	**Reverse primer (5′–3′)**	**Product size (bp)**

aac(6′)/aph(2″)	GTAGAAATGACTGAACGTCCGATAA	CCAATTCCACATTCTTTCGGTCTAA	310
blaZ	ATCATTAGGTAAAATGTCTGGACATGATCCA	GCATCAAGTGTATTGGATAGCAAAAGC	433
mecA	GTGAAGATATACCAAGTGATT	ATGCGCTATAGATTGAAAGGAT	147
aph(3′)-III	CTGATTACTATCCAAGAAATTCGATTG	CTTTCCAGCCTACTTTTTTATCAGT	209
qacA/B	ATTGGCGTGGCTTCAGTGCT	CGTTTCTTCCGTAGTTGCATTTG	292
ermA	GTTCAAGAACAATCACAGAG	GGATCAGGAAAAGGACATTTTAC	533
tetM	ATGACAGAATACTTATTAAGTGCTGGC	TGTATCAACCAATAATAGTCTGAATGT	360
ermB	GGTTATCAATGTGCGGGTGG	CGGCACTTTTTTCTCTTCGG	102
ermC	GAAGATCTATCACAAGATGAAATA	ATGGATTTCACTGGTGTTATTACA	210
msrB	GCGTCAAAATAATTCCAAGG	CAATCTGTCCAATCAAGCGAG	567
tetK	ATGGAAATACAACAAACACAC	CTGCTTGAATGCGCCAAAC	258
tetL	CAATCTGTCCAATCAAGCGAG	GCGTCAAAATAATTCCAAGG	642
linA	CGCGGAAAGGAACGGGTTTT	TTAGCGTCTAGCGTTCTGCC	519
msrA	AATACACCAACGCCTCCAAG	TTGTTGCCGCTTTTGCTCTC	400
hla	AGTTTATAGCGAAGAAGG	AGTTTATAGCGAAGAAGG	447
clfB	GTTATGGTGGTGGAAGTGCTG	CGCTCTTATCTCCTGTTTCTGG	1041

clfA	ATGGGACAACGAAGTAGCA	GCTTCATCTTCAGAACCTG	1149
fnbA	TAGGAACTGAAAATGGTCAC	GAAGCAATCAGAAAACACTC	1027
hlb	GCCAAAGCCGAATCTAAG	GCGATATACATCCCATGGC	834
fnbB	TAGGAACTGAAAATGGTCAC	GAGTATGTAATTATTTCTTGG	973
pvl	ATCATTAGGTAAAATGTCTGGACATGATCCA	GCATCAAGTGTATTGGATAGCAAAAGC	433
seh	CAACTGCTGATTTAGCTCAG	GTCGAATGAGTAATCTCTAGG	359
sec	CTTGTATGTATGGAGGAATAACAA	TGCAGGCATCATATCATACCA	284

Table 2. Detection of pathogenic bacteria in infectious mastitis during lactation.

Pathogen	**Number of isolates**	**Proportion (%)**
Gram-negative bacteria	12	7.59
Escherichia coli	4	2.53
Enterobacter cloacae	4	2.53
Pseudomonas aeruginosa	3	1.90
Klebsiella pneumoniae	1	0.63
Gram-positive bacteria	146	89.87
Staphylococcus aureus – MRSA	82	51.90
Staphylococcus aureus – MSSA	37	23.42
Staphylococcus epidermidis	9	5.70

Enterococcus faecalis	8	5.06
Streptococcus agalactiae	6	3.80
Staphylococcus haemolyticus	4	2.53
Total	158	100.00

Table 3. Detection of resistance genes in *S. aureus.*

Resistance gene	Positive isolates (n)	Detection rate (%)
aac(6')/aph(2")	119	100.00
blaZ	119	100.00
mecA	119	100.00
aph(3')-III	113	94.96
qacA/B	101	84.87
ermA	60	50.42
tetM	54	45.38
ermB	30	25.21
ermC	30	25.21
msrB	26	21.85
tetK	24	20.17
tetL	12	10.08
linA	6	5.04
msrA	6	5.04

Table 4. Antimicrobial resistance in MRSA and MSSA.

Antimicrobial agent	MRSA			MSSA		
	No. tested	No. resistant	Resistance rate (%)	No. tested	No. resistant	Resistance rate (%)
Nitrofurantoin	82	2	2.44	37	0	0.00
Gentamicin	82	4	4.88	37	2	5.41
Levofloxacin	82	7	8.54	37	2	5.41
Ciprofloxacin	82	7	8.54	37	3	8.11
Penicillin G	82	75	91.46[a]	37	29	78.38
Erythromycin	82	54	65.85[a]	37	17	45.95
Linezolid	82	0	0.00	37	0	0.00
Vancomycin	82	0	0.00	37	0	0.00
Clindamycin	82	54	65.85[a]	37	17	45.95
Rifampicin	82	0	0.00	37	0	0.00
Tetracycline	82	41	50.00[a]	37	2	5.41

*p=0.034, ($p<0.05$) compared with MSSA group

Table 5. MRSA and MSSA virulence gene carrying status [*n*(%)].

Virulence gene	**MRSA**	**MSSA**	Total
	Number of isolates (n = 82) / Detection rate (%)	Number of isolates (n = 37) / Detection rate (%)	
Hla	81(98.78)	35(94.59)	116(97.48)

ClfB	80(97.56)	35(94.59)	115(96.64)
ClfA	53(64.63)	28(75.68)	81(68.07)
FnbA	56(68.29)	24(64.86)	80(67.23)
Hlb	35(42.68)	14(37.84)	49(41.18)
FnbB	32(39.02)	13(35.14)	45(37.82)
PVL	29(35.37)[a]	21(56.76)	50(42.02)
seh	11(13.41)	3(8.11)	14(11.76)
Sec	6(7.32)	3(8.11)	9(7.56)

*p=0.034, ($p<0.05$) compared with MSSA group

Table 6. Summary of major resistance and virulence profiles of *S. aureus* in infectious mastitis.

Category	**Key Findings**	**Clinical Implication**
High resistance (>60%)	Penicillin G (MRSA 91.5%, MSSA 78.4%), Erythromycin (MRSA 65.8%, MSSA 46.0%), Clindamycin (MRSA 65.8%, MSSA 46.0%)	Avoid use in empirical therapy
Low resistance (<10%)	Nitrofurantoin, Linezolid, Vancomycin, Rifampicin	Remain effective treatment options
Common virulence genes (>90%)	hla, clfB, clfA, fnbA	Associated with strong pathogenicity

Differential virulence	pvl lower in MRSA (35.4%) vs MSSA (56.8%)	May affect severity and muscle involvement