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Beyond *Staphylococcus aureus*: emerging Gram-negative isolates in pediatric impetigo in Southern Vietnam

Thi Xuan Tam Huynh,¹ Nguyen Anh Thu Tran,^{2,3} Van Ba Huynh²

¹Pham Ngoc Thach University of Medicine, Ho Chi Minh City; ²Can Tho University of Medicine and Pharmacy, Can Tho City; ³Tam Anh Hospital, Ho Chi Minh City, Vietnam

Correspondence: Thi Xuan Tam Huynh, Pham Ngoc Thach University of Medicine, No. 2, Duong Quang Trung Str., Ward 12, District 10, Ho Chi Minh City, Vietnam.

E-mail: tamhtx@pnt.edu.vn

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Abstract

Impetigo is common in tropical regions, yet local data on bacterial spectra beyond *Staphylococcus aureus* remain limited. This study aimed to characterize clinical features, bacterial isolates, and resistance-related factors among patients with impetigo in the Mekong Delta, Vietnam. We conducted a prospective cross-sectional study of 74 patients with clinically diagnosed impetigo at Can Tho City Hospital of Dermato-Venereology from May 2020 to July 2022. Demographic and clinical data were collected using a standardized form. Lesion swabs were cultured, isolates were identified by conventional microbiological methods, and antimicrobial susceptibility was assessed by disk diffusion according to Clinical and Laboratory Standards Institute procedures. Disease severity was evaluated using the Skin Infection Rating Scale (SIRS). The mean age was 4.4 ± 3.5 years; 58.1% of patients were aged 2-6 years. Non-bullous impetigo was the predominant subtype (71.6%), and most patients presented with moderate or severe disease. Cultures were positive in 43 patients (58.0%). *S. aureus* was the leading pathogen (79.1%), followed by *Escherichia coli* (11.7%), *Staphylococcus* species (4.6%), *Pseudomonas aeruginosa* (2.3%), and *Proteus* spp. (2.3%). No *Streptococcus pyogenes* was isolated. *E. coli* was detected in ecthyma and showed high resistance to antibiotic classes, including universal resistance to levofloxacin and piperacillin/tazobactam. No statistically significant association was found between *S. aureus* resistance and clinical subtype, baseline severity, prior antibiotic exposure, or disease duration. Impetigo in this setting was predominantly pediatric and *S. aureus*-associated; however, Gram-negative organisms, especially *E. coli*, may contribute to severe or ulcerative disease. Culture and susceptibility testing should be considered in complicated cases.

Introduction

Impetigo is a common superficial bacterial skin infection, particularly among children living in warm and humid climates. The disease is highly contagious and remains an important dermatologic condition in tropical countries, including Vietnam, where environmental conditions favor the growth and transmission of bacterial, viral, and fungal skin pathogens. Although impetigo is usually limited to the superficial epidermis, delayed diagnosis or inappropriate treatment may lead to local complications such as ecthyma, eczematization, and cellulitis, as well as systemic complications including visceral abscesses, acute glomerulonephritis, arthritis, sepsis, and, rarely, life-threatening infection.

The main causative organisms of impetigo are *Staphylococcus aureus* and *Streptococcus pyogenes*, either alone or in combination.¹ Other bacteria, including *Pseudomonas aeruginosa*, anaerobic bacilli such as

Proteus spp., and *Escherichia coli*, may also be isolated in some cases, contributing to heterogeneous clinical manifestations and diagnostic difficulty.² In mixed infections caused by *S. pyogenes* and *S. aureus*, the initial infection may be associated with *S. pyogenes*, followed by secondary colonization or infection with *S. aureus*; however, because *S. aureus* can produce bactericidal substances against *S. pyogenes*, culture may preferentially recover *S. aureus*.³

Treatment of impetigo generally includes local wound care and antibiotic therapy, either topical alone or combined with systemic antibiotics, depending on disease severity and extent.³ Commonly used systemic antibiotics include cephalexin, clindamycin, oxacillin, erythromycin, and amoxicillin, while topical agents such as mupirocin and fusidic acid are frequently recommended.⁴ However, the effectiveness of empirical treatment increasingly depends on the local antimicrobial susceptibility profile of the causative organisms. The emergence of antimicrobial-resistant strains, particularly penicillin-resistant *S. aureus* and methicillin-resistant *S. aureus* (MRSA), has become a major clinical concern.⁵

Antimicrobial resistance may reduce treatment efficacy, prolong disease duration, increase transmission, and contribute to treatment failure. Resistance is often driven by inappropriate antibiotic use, empirical broad-spectrum therapy, self-medication, and delayed medical care.⁶ In developing countries, *S. pyogenes* has traditionally been considered an important cause of impetigo.³ Nevertheless, recent studies have reported substantial geographic variation in bacterial etiology and resistance patterns, underscoring the need for local microbiological surveillance. Previous studies have shown high rates of resistance among *S. aureus* isolates from impetigo, particularly to penicillin, erythromycin, and other commonly used antibiotics.^{7,8}

Despite the clinical importance of impetigo, recent data on bacterial isolates and antimicrobial resistance patterns among patients with impetigo in Vietnam remain limited, particularly in the Mekong Delta region. Information on non-*S. aureus* pathogens, Gram-negative isolates, and clinical factors related to antimicrobial resistance is also scarce. Because effective treatment depends on the causative organism and its susceptibility profile, updated local evidence is essential to guide rational antibiotic selection and improve patient outcomes.

Therefore, this study was conducted to characterize bacterial isolates from patients with impetigo at Can Tho City Hospital of Dermato-Venereology during 2020-2022, describe antimicrobial resistance patterns, and explore selected clinical factors associated with antimicrobial resistance. The findings may provide useful evidence for optimizing microbiological evaluation and antibiotic use in patients with impetigo in this setting.

Materials and Methods

Study design and setting

This was a prospective descriptive cross-sectional study conducted at Can Tho City Hospital of Dermato-Venereology, Vietnam, from May 2020 to July 2022. The study was designed to determine the bacterial isolates recovered from patients with impetigo, assess their antimicrobial resistance profiles, and explore selected factors associated with antimicrobial resistance.

Study population

All patients clinically diagnosed with impetigo who attended or were treated at Can Tho City Hospital of Dermato-Venereology during the study period were screened for eligibility. Impetigo was diagnosed according to the diagnostic criteria of the Vietnamese Ministry of Health, based on the presence of flaccid bullae or vesicles that rapidly became purulent and ruptured, or thick yellow-brown or honey-colored crusted lesions.⁹ Patients were eligible if they or their legal guardians agreed to participate. For patients younger than 18 years, written informed consent was obtained from parents or guardians. Patients with severe or unstable systemic diseases were excluded.

Sample size and sampling method

The sample size was calculated using the formula for estimating a single population proportion:¹⁰

$$n = Z_{1-\alpha/2}^2 \times \frac{p(1-p)}{d^2}$$

A 95% confidence level was used, corresponding to $Z_{1-\alpha/2}=1.96$. The expected proportion of culture-positive impetigo cases was set at 92.31%, based on a previous Vietnamese study by Tran *et al.*¹¹ The allowable margin of error was 6%. The calculated minimum sample size was 74 patients. Participants were recruited by convenience sampling over time until the required sample size was reached.

Data collection and clinical assessment

Data were collected using a structured case-record form. Demographic and clinical variables included age, sex, ethnicity, residence, educational level, occupation, duration of disease before admission, previous history of topical or oral antibiotic use, contact with a potential source of infection, family history of impetigo or other skin diseases, and coexisting dermatologic conditions.

All patients underwent a standardized dermatologic examination. Recorded symptoms included pruritus, pain, burning sensation, fever, and regional lymphadenitis. Skin lesions were classified as erythema, vesicles, bullae, pustules, honey-colored crusts, erosions, or ulcers. Clinical subtypes were categorized as non-bullous impetigo, bullous impetigo, or ecthyma according to predefined clinical criteria.

Disease severity was assessed using the Skin Infection Rating Scale (SIRS), which evaluates erythema, exudation and/or purulence, erosion, pruritus and/or burning pain, and ulceration.¹² Each clinical component was scored from 0 to 3, except ulceration, which was used as a criterion for severe disease when present. Total scores were classified as follows: severe disease, 10-12 points and/or presence of ulceration; moderate disease, 6-9 points; mild disease, 2-5 points; and clinical cure, 0-1 point.

Specimen collection

Skin specimens were obtained from impetigo lesions under aseptic conditions. For each specimen, sterile gloves were used. The surrounding skin was cleaned with 70% alcohol, and pus or surface debris was gently removed using sterile gauze moistened with sterile normal saline. A sterile cotton swab pre-moistened in a sterile tube containing 5 mL of 0.15 M NaCl and 0.1% Tween 20 was then rubbed over an approximately 4 cm² area of the lesion. After collection, the swab was returned to the transport tube and sent to the microbiology laboratory for bacterial culture.

Specimens were transported in a closed container and processed within 2 hours whenever possible. If a longer transport time was required, specimens were stored and transported in a cold container according to the laboratory procedure.

Bacterial culture and identification

Specimens were processed at the microbiology laboratory of Can Tho University of Medicine and Pharmacy Hospital. Tubes containing collected specimens were mixed using a vortex mixer. A 20 µL aliquot of the specimen suspension was inoculated onto culture media and incubated at 37°C. Plates were examined after 2-4 days for bacterial growth. Suspected colonies were subcultured repeatedly to obtain pure isolates.

Bacterial identification was performed using standard microbiological methods according to the laboratory protocol. Gram staining was performed to distinguish Gram-positive and Gram-negative organisms. For Gram-positive cocci, additional phenotypic tests included catalase testing, coagulase testing, hemolysis assessment, and bacitracin susceptibility testing when appropriate. Identified organisms

were recorded as *S. aureus*, *S. pyogenes*, *E. coli*, *Staphylococcus* species, *P. aeruginosa*, *Proteus* spp., or other bacteria.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed using the agar disk diffusion method on Mueller-Hinton agar, following the standard operating procedure based on the Clinical and Laboratory Standards Institute guidelines.¹³ Mueller-Hinton agar plates were prepared with an agar depth of approximately 4 mm. Bacterial isolates were cultured overnight, and isolated colonies were suspended in 2 mL of sterile normal saline. The turbidity of each bacterial suspension was adjusted to match the 0.5 McFarland standard, corresponding to approximately 10^8 colony-forming units/mL.

The standardized suspension was spread evenly onto Mueller-Hinton agar plates. Antibiotic disks were placed on the agar surface, with adequate spacing between disks and from the edge of the plate. Plates were left at room temperature for approximately 30 minutes to allow diffusion and then incubated at 37°C for 24-48 hours. Inhibition zone diameters were measured and interpreted as susceptible, intermediate, or resistant according to the applicable standard criteria.

For Gram-positive isolates, susceptibility was tested against penicillin, oxacillin, amoxicillin/clavulanic acid, cefixime, cefuroxime, ceftriaxone, erythromycin, clindamycin, trimethoprim/sulfamethoxazole, ciprofloxacin, levofloxacin, tetracycline, vancomycin, and linezolid. For Gram-negative isolates, susceptibility was tested against levofloxacin, piperacillin/tazobactam, amoxicillin/clavulanic acid, cefuroxime, ceftriaxone, ceftazidime, cefepime, imipenem, meropenem, erythromycin, gentamicin, tobramycin, trimethoprim/sulfamethoxazole, and colistin.

Bias control

Several procedures were applied to reduce measurement and data-entry errors. All patients were recruited from a single study site, and data collection was performed by the principal investigator using a standardized form. Clinical assessments were conducted carefully and objectively, with diagnostic agreement between the investigator and treating dermatologists at the study hospital. Data were entered independently by two individuals, and the two datasets were compared to ensure consistency before analysis.

Statistical analysis

Data were managed using Microsoft Excel 2013 and analyzed using SPSS version 18.0. Categorical variables were summarized as frequencies and percentages. Quantitative variables were assessed for normality using the One-Sample Kolmogorov-Smirnov test. Normally distributed variables were presented as mean $\pm$ standard deviation, whereas non-normally distributed variables were summarized using median, minimum, and maximum values.

Associations between categorical variables were evaluated using the chi-square test or Fisher's exact test, as appropriate. Comparisons of mean quantitative variables between two time points were performed using the paired-samples *t*-test. A two-sided *p*-value of <0.05 was considered statistically significant.

Ethical considerations

The study was conducted in accordance with ethical principles for biomedical research. All participants or their guardians were informed about the study objectives, procedures, and use of study data before enrollment. Participation was voluntary, and refusal to participate did not affect clinical care. Patient confidentiality was maintained throughout the study.

Results

Patient characteristics and clinical presentation

A total of 74 patients with clinically diagnosed impetigo were included. The cohort was predominantly pediatric, with a mean age of 4.4 ± 3.5 years; ages ranged from 9 months to 14 years. Children aged 2-6 years represented the largest age group (43/74, 58.1%), followed by those older than 6 years (16/74, 21.7%) and those younger than 2 years (15/74, 20.3%). The sex distribution was nearly balanced, with 38 males (51.0%) and 36 females (49.0%). Most patients resided in rural areas (60/74, 81.0%), and the majority were of Kinh ethnicity (72/74, 97.0%).

Pruritus was the most frequent symptom reported in 71 patients (95.9%). Pain and burning sensation were less common, occurring in 29 (39.2%) and 18 patients (24.3%), respectively. Systemic manifestations were uncommon: fever was documented in 10 patients (13.5%), and regional lymphadenitis in one patient (1.4%).

The most consistent cutaneous finding was erythema, observed in all patients (74/74, 100%). Honey-colored crusts and erosions were each present in 73 patients (98.6%). Pustules and vesicles were also frequent, occurring in 59 (79.7%) and 58 patients (78.4%), respectively. Bullae were noted in 21 patients (28.4%), whereas ulcerative lesions were present in five patients (6.8%).

Non-bullous impetigo was the dominant clinical phenotype, accounting for 53 cases (71.6%). Bullous impetigo was diagnosed in 17 patients (23.0%), and ecthyma in four patients (5.4%). Cases were observed throughout the year, with the highest proportion recorded from April to June (24/74, 32.4%), followed by October to December (22/74, 29.7%), July to September (16/74, 21.6%), and January to March (12/74, 16.2%).

At presentation, most patients had moderate-to-severe disease according to the SIRS. Severe disease was recorded in 36 patients (48.6%), moderate disease in 35 patients (47.3%), and mild disease in three patients (4.1%).

Bacterial culture and pathogen distribution

Bacterial culture was positive in 43 of 74 patients (58.0%). *S. aureus* was the predominant isolate, identified in 34 of 43 culture-positive cases (79.1%). Non-*aureus* organisms accounted for the remaining isolates, including *E. coli* in five cases (11.7%), *Staphylococcus* species in two cases (4.6%), *P. aeruginosa* in one case (2.3%), and *Proteus* spp. in one case (2.3%). No *S. pyogenes* isolate was reported in the study dataset.

Pathogen distribution varied by clinical subtype. Among culture-positive patients with non-bullous impetigo, *S. aureus* was isolated in 22 of 26 cases (84.6%), whereas *E. coli* was identified in one case (3.8%) and other bacteria in three cases (11.5%). In bullous impetigo, *S. aureus* was detected in 10 of 13 cases (76.9%), *E. coli* in two cases (15.4%), and other bacteria in one case (7.7%). In ecthyma, *S. aureus* and *E. coli* were each detected in two of four cases (50.0%); other bacteria were detected in one case (25.0%). One ecthyma case yielded both *S. aureus* and *E. coli*.

Antimicrobial resistance profile of Escherichia coli

Among the five *E. coli* isolates, resistance was frequent across multiple antibiotic classes. All isolates were resistant to levofloxacin and piperacillin/tazobactam (5/5, 100%). Resistance to amoxicillin/clavulanic acid, cefuroxime, ceftriaxone, ceftazidime, cefepime, erythromycin, gentamicin, and tobramycin was observed in four of five isolates (80.0%). Imipenem resistance was detected in two isolates (60.0%). Resistance to meropenem, trimethoprim/sulfamethoxazole, and colistin was lower, occurring in one isolate each (20.0%).

Factors associated with antimicrobial resistance in Staphylococcus aureus

Exploratory analyses did not identify statistically significant associations between *S. aureus* resistance and clinical subtype, baseline disease severity, prior topical antibiotic exposure, prior oral antibiotic exposure, or duration of illness before admission (Tables 1 and 2). Across the tested antibiotics, all comparisons remained non-significant by Fisher's exact test or chi-square testing, with $p > 0.05$.

Although not statistically significant, several descriptive patterns were observed. Resistance tended to cluster among patients with non-bullous impetigo simply because this was the most common clinical subtype; however, linezolid resistance was detected only in ecthyma, and the association by clinical subtype approached but did not reach statistical significance ($p = 0.059$). Resistance to levofloxacin also showed a near-threshold difference across clinical subtypes ($p = 0.072$). With respect to baseline severity, all vancomycin-resistant and linezolid-resistant *S. aureus* isolates were found among patients with severe disease, but these differences were not statistically significant.

Prior topical antibiotic use was not associated with resistance to any tested antibiotic. In contrast, resistance was descriptively more common among patients with a history of prior oral antibiotic use for several agents, but no comparison reached statistical significance. Similarly, patients with illness duration of ≥ 7 days before admission showed numerically higher resistance rates for several antibiotics, including clindamycin, trimethoprim/sulfamethoxazole, cefixime, cefuroxime, oxacillin, ciprofloxacin, levofloxacin, vancomycin, and linezolid; however, none of these associations were statistically significant.

Discussion

In this study, impetigo predominantly affected young children, particularly those aged 2-6 years, with a mean age of 4.4 ± 3.5 years. This finding is consistent with previous reports showing that impetigo is most common in preschool-aged children.¹⁴ Children in this age group frequently attend nursery school or kindergarten, where close contact facilitates transmission. In addition, immature hygiene practices and scratching behavior may promote autoinoculation and spread of lesions.

The sex distribution was nearly equal, with a male-to-female ratio of approximately 1:1, which is consistent with prior studies reporting no major sex difference in impetigo occurrence.¹⁴ Most patients lived in rural areas. This may reflect differences in hygiene conditions, household crowding, access to early medical care, and environmental exposure. In tropical settings, heat and humidity may further impair the skin barrier and facilitate bacterial colonization.¹⁵

Pruritus was the most common symptom, reported in almost all patients. This is clinically important because scratching can rupture vesicles, pustules, and crusted lesions, allowing bacterial spread to adjacent skin and potentially worsening the infection. By contrast, systemic manifestations were uncommon, with fever and lymphadenitis observed in only a small number of patients. This finding is compatible with the superficial nature of most impetigo cases, although systemic signs may occur in more extensive or severe disease.

The most frequent lesions were erythema, honey-colored crusts, and erosions. This pattern reflects the typical evolution of impetigo lesions from erythema to vesicles or pustules, followed by rupture, erosion, crusting, and healing. Similar findings were reported by Tran *et al.*, in which erosions and honey-colored crusts were also the dominant clinical features.¹¹ These findings are also consistent with standard descriptions of non-bullous impetigo.^{3,16} Because many patients present after lesions have already ruptured, crusts and erosions are more commonly observed than intact vesicles or bullae.

Non-bullous impetigo was the most common clinical subtype, accounting for more than two-thirds of cases. This is consistent with the report by Sewon Kang, in which non-bullous impetigo represented approximately 70% of cases.³ Ecthyma was less frequent but remains clinically important because it represents a deeper form of infection and may be associated with delayed treatment, inadequate wound care, secondary infection, and scarring.

Bacterial culture was positive in 58.0% of patients, lower than the rate reported by Tran *et al.*¹¹ This difference may be partly explained by specimen transport from the dermatology hospital to the microbiology laboratory, which could have reduced bacterial recovery. In addition, the study period overlapped with the COVID-19 pandemic, which may have affected sample collection and processing.

Among culture-positive cases, *S. aureus* was the predominant pathogen, identified in 79.1% of isolates. This finding is consistent with previous studies indicating that *S. aureus* is a leading cause of impetigo.^{8,11} No *S. pyogenes* isolate was detected in our cohort. One possible explanation is that in mixed infections, *S. aureus* may suppress *S. pyogenes*, leading to preferential isolation of *S. aureus* in culture.³ Geographic variation, prior antibiotic exposure, sampling time, and laboratory methods may also influence the relative detection rates of these organisms.

The predominance of *S. aureus* was observed across clinical subtypes, particularly in non-bullous and bullous impetigo. However, in ecthyma, *S. aureus* and *E. coli* were detected with equal frequency, and one case yielded both organisms. Although *E. coli* is not traditionally considered a major cause of impetigo, it has been reported in skin and soft-tissue infections. Petkovsek *et al.* identified *E. coli* in skin

and soft-tissue infection specimens and described virulence factors that may impair epithelial repair and delay wound healing.¹⁷ Therefore, the detection of *E. coli* in ecthyma may suggest a role for Gram-negative organisms in deeper, ulcerative, chronic, or treatment-refractory lesions.

The antimicrobial resistance profile of *E. coli* isolates was notable, although interpretation is limited by the small number of isolates. Resistance was observed across multiple antibiotic classes. These findings appear higher than those reported by Petkovsek *et al.*,¹⁷ but are compatible with more recent evidence suggesting an increasing burden of multidrug-resistant *E. coli*. Ranjan *et al.* reported a high rate of multidrug resistance among *E. coli* isolates, including those from skin and soft-tissue infections.¹⁸ In addition, Marwan demonstrated that plasmids from multidrug-resistant MRSA isolates could transform *E. coli* into a multidrug-resistant strain, suggesting a possible mechanism for resistance transfer in polymicrobial infections.⁸

No statistically significant associations were found between *S. aureus* antimicrobial resistance and clinical subtype, baseline severity, previous topical antibiotic use, previous oral antibiotic use, or disease duration before admission. However, this should be interpreted cautiously because the number of *S. aureus* isolates was limited, and several subgroups were small. Descriptively, linezolid resistance was observed only in ecthyma, and all vancomycin-resistant and linezolid-resistant isolates occurred in patients with severe disease. Tran *et al.* also reported that MRSA infection was more frequent in patients with ecthyma.¹¹ These observations suggest that severe or ulcerative lesions may be associated with a higher likelihood of resistant organisms, although larger studies are needed to confirm this relationship.

Previous topical antibiotic use was not associated with resistance in this study. In contrast, patients with prior oral antibiotic exposure showed a tendency toward higher resistance rates, although the association was not statistically significant. This trend is biologically plausible because systemic antibiotic exposure may select for resistant strains. Verdú-Expósito *et al.* also reported resistant *S. aureus* isolates in patients with prior oral antibiotic use.¹⁹ Similarly, longer disease duration before admission tended to be associated with higher resistance rates, possibly reflecting delayed care, inappropriate self-medication, and prolonged bacterial colonization.

This study has several limitations. It was conducted at a single center with a modest sample size, limiting the statistical power of subgroup analyses. The number of *E. coli* isolates was small, and molecular testing was not performed to identify resistance genes, virulence factors, or clonal relationships. In addition, culture yield may have been affected by specimen transport and the COVID-19 pandemic. Despite these

limitations, the study provides useful evidence on the clinical and microbiological characteristics of impetigo in the Mekong Delta region.

Conclusions

In conclusion, impetigo in this cohort mainly affected young children and most commonly presented as non-bullous impetigo. *S. aureus* remained the predominant pathogen, but the detection of *E. coli*, particularly in ecthyma, suggests that Gram-negative bacteria may contribute to severe or ulcerative disease in selected cases. The observed resistance patterns support the importance of bacterial culture and susceptibility testing in severe, persistent, recurrent, or treatment-refractory impetigo, and highlight the need for rational antibiotic use in clinical practice.

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Table 1. The association between clinical subtype and baseline severity with antimicrobial resistance among *S. aureus* isolates.

Antibiotic	Resistant isolates by clinical subtype: non-bullous/bullous/ecthyma n (%)	p-value	Resistant isolates by baseline severity: moderate/severe n (%)	p-value
Clindamycin	21 (63.6)/10 (30.3)/2 (6.1)	1.000	14 (42.4)/19 (57.6)	0.441
Trimethoprim/ sulfamethoxazole	22 (66.7)/9 (27.2)/2 (6.1)	0.353	15 (45.5)/18 (54.5)	1.000
Cefixime	22 (66.7)/9 (27.2)/2 (6.1)	0.353	15 (45.5)/18 (54.5)	1.000
Cefuroxime	22 (66.7)/9 (27.2)/2 (6.1)	0.353	15 (45.5)/18 (54.5)	1.000
Ceftriaxone	21 (65.6)/9 (28.1)/2 (6.3)	0.588	15 (46.9)/17 (53.1)	0.492
Oxacillin	21 (67.7)/8 (25.8)/2 (6.5)	0.353	13 (41.9)/18 (58.1)	0.571
Amoxicillin/ clavulanic acid	19 (65.5)/8 (27.6)/2 (6.9)	0.734	12 (41.4)/17 (58.6)	0.634
Ciprofloxacin	14 (82.4)/2 (11.8)/1 (5.8)	0.079	10 (58.8)/7 (41.2)	0.084*
Levofloxacin	13 (81.3)/2 (12.5)/1 (6.2)	0.072	9 (56.3)/7 (43.8)	0.300
Tetracycline	11 (68.8)/4 (25.0)/1 (6.2)	0.852	7 (43.8)/9 (56.2)	0.968*
Vancomycin	2 (50.0)/1 (25.0)/1 (25.0)	0.286	0 (0.0)/4 (100.0)	0.113
Linezolid	0 (0.0)/0 (0.0)/1 (100.0)	0.059	0 (0.0)/1 (100.0)	1.000

Data are presented as resistant isolates/total isolates within each subgroup (%); p-values were calculated using Fisher's exact test unless otherwise indicated; *chi-square test.

Table 2. The association between prior antibiotic exposure and disease duration before admission with antimicrobial resistance among *S. aureus* isolates.

Antibiotic	Prior topical antibiotic use: yes/no n (%)	p-value	Prior oral antibiotic use: yes/no n (%)	p value	Disease duration before admission: <7 days/≥7 days n (%)	p value
Clindamycin	4 (12.1)/29 (87.9)	0.147	21 (63.6)/12 (36.4)	0.382	13 (39.4)/20 (60.6)	1.000
Trimethoprim/ sulfamethoxazole	5 (15.2)/28 (84.8)	1.000	20 (60.6)/13 (39.4)	1.000	13 (39.4)/20 (60.6)	1.000
Cefixime	5 (15.2)/28 (84.8)	1.000	20 (60.6)/13 (39.4)	1.000	13 (39.4)/20 (60.6)	1.000
Cefuroxime	5 (15.2)/28 (84.8)	1.000	20 (60.6)/13 (39.4)	1.000	13 (39.4)/20 (60.6)	1.000
Ceftriaxone	5 (15.6)/27 (84.4)	1.000	19 (59.4)/13 (40.6)	0.513	13 (40.6)/19 (59.4)	0.513
Oxacillin	5 (16.1)/26 (83.9)	1.000	19 (61.3)/12 (38.7)	1.000	12 (38.7)/19 (61.3)	1.000
Amoxicillin/ clavulanic acid	4 (13.8)/25 (86.2)	1.000	18 (62.1)/11 (37.9)	1.000	13 (44.8)/16 (55.2)	0.132
Ciprofloxacin	4 (23.5)/13 (76.5)	0.335	10 (58.8)/7 (41.2)	1.000	6 (35.3)/11 (64.7)	0.724*
Levofloxacin	3 (18.8)/13 (81.3)	0.648	9 (56.3)/7 (43.8)	0.533*	6 (37.5)/10 (62.5)	0.934*
Tetracycline	2 (12.5)/14 (87.5)	1.000	10 (62.5)/6 (37.5)	0.800*	8 (50.0)/8 (50.0)	0.183*
Vancomycin	1 (25.0)/3 (75.0)	0.488	2 (50.0)/2 (50.0)	0.627	1 (25.0)/3 (75.0)	1.000
Linezolid	0 (0.0)/1 (100.0)	1.000	1 (100.0)/0 (0.0)	0.647	0 (0.0)/1 (100.0)	1.000

Data are presented as resistant isolates/total isolates within each subgroup (%); p-values were calculated using Fisher's exact test unless otherwise indicated; *chi-square test.