

Research Article

In Vitro Evaluation of Antimicrobial Drug Activity and Resistance-Associated Genetic Markers in Staphylococcus Aureus: A Comparative Study of Antibiotic Susceptibility and vanA/vanB Genes in Swat, Pakistan

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Received: 22.08.26, Revised: 25.09.26, Accepted: 28.10.26

ABSTRACT

Background: Methicillin-resistant Staphylococcus aureus (MRSA) is a major cause of hospital infection and vancomycin is a first-line treatment. Vancomycin resistance, usually driven by vanA and vanB genes of enterococcal origin, is rare but rising, and local data from Swat, Pakistan, are scarce.

Methods: In this cross-sectional study carried out at a tertiary care hospital in Swat from February to July 2026, a total of 145 non-duplicated laboratory confirmed samples of Staph aureus were selected for further antimicrobial susceptibility testing by disk diffusion according to CLSI standards. MRSA was defined by cefoxitin resistance. Vancomycin-resistant isolates were screened by PCR for vanA and vanB. Proportions are reported with Wilson 95% confidence intervals, and vancomycin resistance in MRSA and non-MRSA isolates was compared with Fisher's exact test.

Results: Most isolates came from pus (42.76%) and blood (28.97%). Forty-seven isolates (32.41%; 95% CI 25.3 to 40.4) were MRSA. Resistance exceeded 79% for ampicillin, aztreonam, ertapenem, tobramycin, tetracycline, and moxifloxacin, whereas susceptibility remained high for linezolid (98.62%), tigecycline (95.17%), and vancomycin (92.41%). Eight isolates (5.52%; 95% CI 2.8 to 10.5) were vancomycin-resistant, and all eight were MRSA, giving a vancomycin resistance rate of 17.02% (8/47) in MRSA, against none in the 98 non-MRSA isolates ($p < 0.001$). All eight carried vanA, and five (62.5%) also carried vanB.

Conclusion: One in three S. aureus isolates in this Swat hospital was MRSA, and resistance to conventional drugs was widespread. Vancomycin resistance was confined to MRSA and was linked to vanA, with frequent vanB co-detection that needs sequencing to confirm. MIC-based vancomycin testing, molecular surveillance, and stricter vancomycin stewardship are advisable.

Keywords: Staphylococcus Aureus, MRSA, Vancomycin Resistance, VANA, VANB, Swat, Pakistan.

INTRODUCTION

Staphylococcus aureus mostly found on the skin, and in the nostril of healthy people where it is a normal commensal, yet it is also one of the most versatile human pathogens, causing everything from skin and soft-tissue infections to bacteraemia, endocarditis, bone and joint infections, and device-related disease (1). Its ability to collect resistance determinants has

made it a central player in the wider antimicrobial resistance (AMR) crisis. One global analysis estimated that bacterial AMR was associated with 4.95 million deaths in 2019, of which 1.27 million were directly attributable to resistance (2). The World Health Organization has since kept antibiotic-resistant S. aureus on its 2024 list of high-priority bacterial pathogens (3).

Methicillin resistance is the best-known of these problems. It arises when the *mecA* or *mecC* gene produces a penicillin-binding protein with low affinity for β -lactams, leaving methicillin-resistant *S. aureus* (MRSA) untouched by most drugs in this class (4). For several decades, vancomycin has therefore been a first-line choice for serious MRSA infections (4). That reliance now carries a cost. Isolates with intermediate susceptibility (vancomycin MIC 4 to 8 $\mu\text{g/mL}$) generally develop through accumulated mutations that thicken the cell wall, whereas full resistance (MIC of 16 $\mu\text{g/mL}$ or higher) is mediated by the *vanA* gene cluster, which *S. aureus* acquires from vancomycin-resistant enterococci (4).

The first vancomycin-resistant *S. aureus* (VRSA) isolate, recovered in Michigan in 2002, had an MIC of 1024 $\mu\text{g/mL}$ and carried Tn1546 (*vanA*), which most likely moved into an MRSA strain from a co-isolated *Enterococcus faecalis* (5). A review of the first seven US cases found that every isolate was *vanA*-positive and that every patient had earlier MRSA and enterococcal infection or colonisation (6). By 2019, 52 VRSA isolates had been reported worldwide, nine of them from Pakistan (4). Reports of *vanB* in *S. aureus* are less consistent: a pooled analysis found that about 69% of VRSA isolates carried *vanA*, and noted that many studies failed to detect *vanB* (7). Pooled VRSA prevalence was 1.5% overall and had risen to 2.4% in studies after 2010 (7).

The regional picture is worrying but patchy. A systematic review of Pakistani studies published between 2009 and 2020 reported MRSA in 49% of *S. aureus* isolates, and found that only about one-third of studies used molecular methods (8). In Peshawar, 36.1% of 280 isolates were MRSA, and while disc diffusion indicated full vancomycin susceptibility, MIC testing revealed seven vancomycin-intermediate isolates (9). In a study from Lahore, 14% of 100 *S. aureus* isolates were found to be vancomycin-resistant, and amplified *vanA* was found in nine of 30 isolates (10). The neighboring countries have similar stories: all 12 VRSA isolates from Iraq were *vanA* positive and 66.6% were *vanB* positive (11), and a recent studies in Kathmandu, Nepal showed that 33.3% of the 290 MRSA were *vanA* positive and 11.1% were *vanB* positive (12).

In spite of these reports, genotype-linked data on vancomycin resistance from Swat, Khyber Pakhtunkhwa, are still limited, and local clinicians have limited evidence to guide their

empirical decisions. We therefore examined 145 *S. aureus* isolates collected at a tertiary care hospital in Swat between February and July 2026. We aimed to describe their antimicrobial susceptibility profile, to estimate the prevalence of MRSA by cefoxitin screening, to test whether vancomycin resistance is concentrated in the methicillin-resistant subset, and to detect *vanA* and *vanB* genes in the vancomycin-resistant isolates.

MATERIALS AND METHODS

Study design and setting

This cross-sectional study was carried out at a tertiary care hospital in Swat, Khyber Pakhtunkhwa, Pakistan, from February to July 2026. The hospital serves a large catchment of urban and rural patients, and its diagnostic laboratory receives specimens from both inpatient and outpatient departments.

Isolates and identification

All non-duplicate *Staph. aureus* isolates that the hospital laboratory identified and confirmed during the study period were included, giving a total of 145 isolates. Specimens were pus, blood, throat swabs, tissue, and body fluids. Identification followed the laboratory's routine procedure e.g. Gram stain, catalase, coagulase and mannitol fermentation. Confirmed isolates were stored in 30% glycerol in nutrient broth at -80°C , and subcultured before testing.

Antimicrobial susceptibility testing

Susceptibility was tested by the Kirby-Bauer disk diffusion method on Mueller-Hinton agar, using a 0.5 McFarland inoculum and procedures set out in the CLSI disk diffusion standard (13). Ten agents were tested: ampicillin, aztreonam, ertapenem, tobramycin, tetracycline, moxifloxacin, cefoxitin, vancomycin, tigecycline, and linezolid. Zone diameters were interpreted as resistant, intermediate, or susceptible using the 36th edition of the CLSI performance standards (14). *S. aureus* (ATCC 25923) served as the quality-control strain.

Definition of MRSA

Methicillin resistance was defined phenotypically by the cefoxitin disk (30 μg), which is widely used as a surrogate marker in *S. aureus* and has been applied in earlier studies from Pakistan (9, 10). Isolates resistant to cefoxitin were classified as MRSA, and all other isolates as non-MRSA.

Vancomycin susceptibility and gene detection

Vancomycin susceptibility categories followed the CLSI MIC breakpoints: susceptible (2 µg/mL or lower), intermediate (4 to 8 µg/mL), and resistant (16 µg/mL or higher) (4). Because disk diffusion cannot reliably detect reduced vancomycin susceptibility (7), MICs were determined by broth microdilution according to the CLSI dilution standard (15).

DNA was extracted from the vancomycin-resistant isolates using Qiagen genomic DNA Extraction kit. The vanA and vanB genes were amplified by conventional PCR with primers described previously (16, 17). list of primer sequences and amplicon sizes are mentioned

(Table 1). PCR amplification of the vanA and vanB genes was carried out under the following thermal cycling conditions. After an initial denaturation at 95 °C for 5 minutes, the reaction was run for 35 cycles of denaturation at 95 °C for 30 seconds, annealing for 30 seconds, and extension at 72 °C for 1 minute. The annealing temperature was 58 °C for vanA and 56 °C for vanB. A final extension at 72 °C for 5 minutes followed, and the products were held at 12 °C until further analysis. The PCR product was run on 1.5% agarose gel electrophoresis along with a 100bp ladder for band size measurements. The gel was finally visualized by gel documentation system, UV transillumination.

Table 1. List of primer sequences and their amplicon sizes

Primer and sequences	Temperature	Amplicons size
vanA-F GGGAAAACGACAATTCGCGC vanA-R GTACAATGCGCCGTTA	58°C	732bp
vanB -F ATGGGAAGCCGATAGTC vanB-R GATTTTCGTTCTCGACC	56C°	635bp

Statistical analysis

Categorical data are presented as frequencies and percentages. Ninety-five percent confidence intervals for proportions were calculated by the Wilson score method, which performs well for small samples and for proportions near 0 or 1. Vancomycin resistance in MRSA and non-MRSA isolates was compared with Fisher's exact test because one cell of the 2 × 2 table was zero. A two-sided p-value below 0.05 was considered statistically significant. Analyses were run in SPSS.

RESULTS

Characteristics of the isolates

A total of 145 *S. aureus* isolates were analyzed over the six-month study period. Pus was the commonest source (n = 62; 42.76%), followed by blood (n = 42; 28.97%) and throat swabs (n = 24; 16.55%). Tissue (n = 10; 6.90%) and body fluids (n = 7; 4.83%) made up the remainder (Table 1). Pus and blood together supplied more than 70% of the collection.

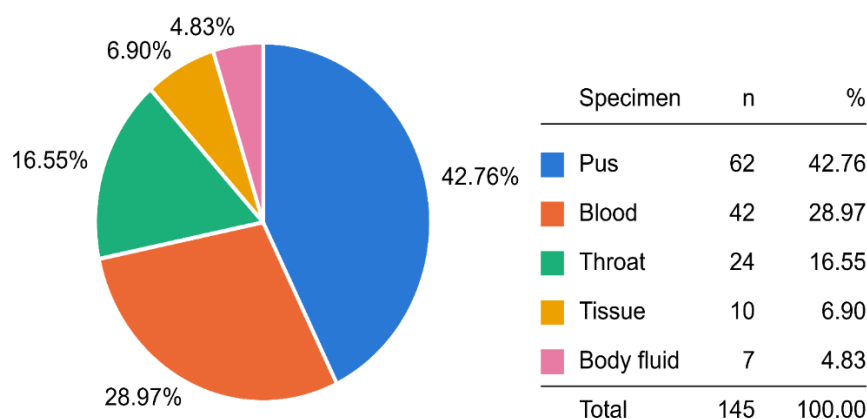


Figure 1. Distribution of Staph. aureus specimens (n=145)

Burden of methicillin resistance

By cefoxitin screening, 47 of the 145 isolates were resistant (MRSA), a prevalence of 32.41% (95% CI 25.3 to 40.4). The other 98 isolates

(67.59%; 95% CI 59.6 to 74.7) were susceptible and were classed as non-MRSA for comparative analysis.

Antimicrobial susceptibility pattern

Resistance to conventional agents was widespread (Table 2, Figure 1). Ampicillin showed the highest resistance (88.97%; 95% CI 82.8 to 93.1), followed closely by aztreonam (87.59%), ertapenem (84.83%), tobramycin (84.14%), tetracycline (80.69%) and moxifloxacin (79.31%). For all six drugs the lower confidence limit stayed above 72%, so these rates are unlikely to be a sampling

artefact. Cefoxitin resistance, at 32.41%, was considerably lower.

The picture was very different for the last-line agents. Linezolid kept its activity against 143 isolates (98.62%), tigecycline against 138 (95.17%) and vancomycin against 134 (92.41%). Resistance to these three drugs was 1.38%, 3.45% and 5.52% respectively, so ampicillin resistance was roughly 16 times higher than vancomycin resistance.

Table 2. Antimicrobial susceptibility profile of *S. aureus* isolates (N = 145)

Antibiotic	Resistant, n (%) [95% CI]	Intermediate, n (%)	Susceptible, n (%)
Ampicillin	129 (88.97) [82.8–93.1]	3 (2.07)	13 (8.97)
Aztreonam	127 (87.59) [81.2–92.0]	2 (1.38)	16 (11.03)
Ertapenem	123 (84.83) [78.1–89.8]	1 (0.69)	21 (14.48)
Tobramycin	122 (84.14) [77.3–89.2]	4 (2.76)	19 (13.10)
Tetracycline	117 (80.69) [73.5–86.3]	10 (6.90)	18 (12.41)
Moxifloxacin	115 (79.31) [72.0–85.1]	5 (3.45)	25 (17.24)
Cefoxitin	47 (32.41) [25.3–40.4]	–	98 (67.59)
Vancomycin	8 (5.52) [2.8–10.5]	3 (2.07)	134 (92.41)
Tigecycline	5 (3.45) [1.5–7.8]	2 (1.38)	138 (95.17)
Linezolid	2 (1.38) [0.4–4.9]	0 (0.00)	143 (98.62)

CI, confidence interval (Wilson method).

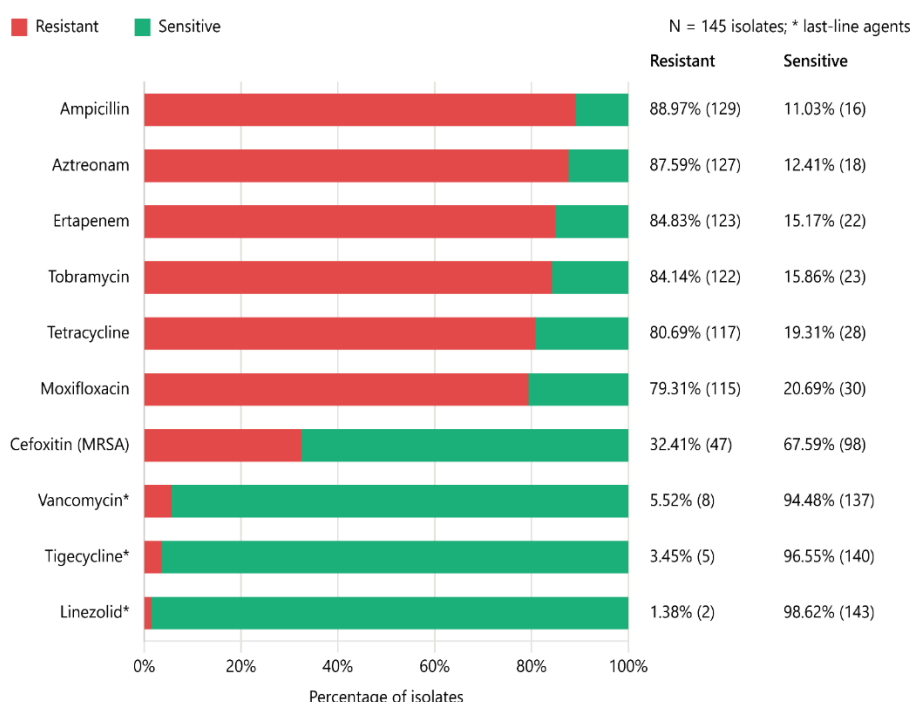


Figure 2. Percentage of *S. aureus* isolates resistant to each antibiotic (N = 145). Red bars show resistant isolates while the green indicate sensitivity in %

Vancomycin resistance and its link to MRSA status

Eight isolates (5.52%; 95% CI 2.8 to 10.5) were vancomycin-resistant and three (2.07%)

were intermediate, so 11 isolates (7.59%; 95% CI 4.3 to 13.1) showed reduced susceptibility overall. All eight resistant isolates were also MRSA. Vancomycin resistance therefore

affected 17.02% of MRSA isolates (8/47; 95% CI 8.9 to 30.1), whereas none of the 98 non-MRSA isolates was resistant (0/98; upper 95% limit 3.8%). This difference was statistically

significant (Fisher's exact test, $p < 0.001$; Table 3). In this collection, vancomycin resistance was confined to the methicillin-resistant subset.

Table 3. Vancomycin resistance according to methicillin-resistance status

Group	Vancomycin-resistant, n (%)	Not resistant, n (%)	Total	p value
MRSA	8 (17.02)	39 (82.98)	47	< 0.001*
Non-MRSA	0 (0.00)	98 (100.00)	98	
Total	8 (5.52)	137 (94.48)	145	

*Fisher's exact test (exact $p \approx 8 \times 10^{-5}$). "Not resistant" includes intermediate and susceptible isolates.

Genotypic detection of vanA and vanB

All eight vancomycin-resistant isolates were tested by PCR. The vanA gene was present in every isolate (8/8; 100%; 95% CI 67.6 to 100), while vanB was found in five (62.5%; 95% CI 30.6 to 86.3). Five isolates therefore carried both genes and three carried vanA alone; no

isolate had vanB without vanA (Table 4, Figure 3). Relative to all 145 isolates, vanA was present in 5.52% and vanB in 3.45%. Because only eight isolates were tested, the confidence intervals are wide, and these gene frequencies are descriptive rather than population estimates.

Table 4. vanA and vanB distribution among vancomycin-resistant *S. aureus* (n = 8)

Genotype	N	% (95% CI)
vanA positive	8	100 (67.6–100)
vanB positive	5	62.5 (30.6–86.3)
vanA + vanB	5	62.5 (30.6–86.3)
vanA only	3	37.5 (13.7–69.4)
vanB only	0	0 (0–32.4)

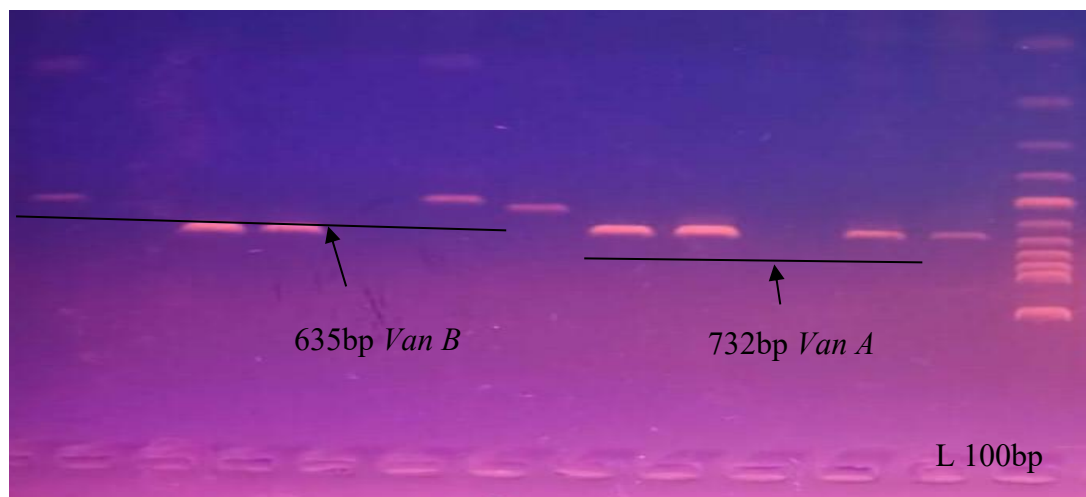


Figure 3: PCR based amplification and identification vancomycin resistant genes vanA (732bp), and VanB gene (635bp) on the right side

DISCUSSION

The aim of this study was to describe the resistance profile of *S. aureus* in a tertiary hospital in Swat and to determine if vancomycin resistance and the genes responsible for it are clustered in methicillin-resistant isolates. There are three findings that stand out. First, one-third of the isolates was MRSA, and resistance to most of the conventional drug classes was

over 79%. Second, linezolid, tigecycline and vancomycin maintained good activity overall, but 5.52% of isolates were vancomycin-resistant, all of which were MRSA. Third, all eight vancomycin-resistant isolates were vanA positive, with five of them being vanB positive. The MRSA prevalence rate of 32.41% falls within the range of published rates from other nearby settings. It is lower than the 49%

reported in a systematic review of Pakistani studies (8), and the 76% in a hospital in Lahore (10), but similar to the 36.1% recorded in Peshawar (9), and the 37% pooled estimate for India (18). Such differences are not unusual and may be due to case mix, specimen type and ward distribution as well as true geographic variation; for instance, the Indian meta-analysis showed 49% in hospital studies and 27% in community studies (18).

The susceptibility profile is a familiar one. The resistance rates of ampicillin, aztreonam, ertapenem, tobramycin, tetracycline and moxifloxacin were between 79% and 89%, which were within 2-8 percentage points of the rates reported for the same panel in Lahore (10). Similarly, Peshawar MRSA isolates were also 85.1% resistant to moxifloxacin (9). These are very high rates across unrelated drug classes, suggesting strong selective pressure, and authors who have reviewed Pakistani data have attributed this to irrational prescribing and self-medication (8). In this context, the reserve agents did a good job. Linezolid (98.62%) and tigecycline (95.17%) stayed active, which matches high tigecycline susceptibility in Lahore (10) and full linezolid susceptibility in Peshawar (9). This is reassuring, because VRSA isolates usually remain susceptible to agents such as linezolid and tigecycline (4). It should not breed complacency, however, since high-level linezolid resistance alongside vancomycin resistance has already been reported from Pakistan (19), and two of our isolates were linezolid-resistant.

Vancomycin resistance in our collection (5.52%) was well above the global pooled VRSA prevalence of 1.5%, and above the 2.4% pooled for studies after 2010 (7). It is closer to the 14% reported in Lahore (10), although a recent Indian series found 6.2% among MRSA (12), compared with 17.02% in ours. Vancomycin categories are only meaningful when they rest on MIC testing, because disk diffusion cannot reliably separate susceptible, intermediate and resistant isolates (7, 20).

The fact that no non-MRSA isolate was resistant (0/98) fits what is known about how VRSA arises. Full resistance reaches *S. aureus* through the *vanA* cluster, transferred from vancomycin-resistant enterococci (4). In the first US cases, every patient had earlier MRSA and enterococcal infection or colonisation, and all isolates were *vanA*-positive (6). MRSA is simply the strain most often exposed to vancomycin and to the enterococcal reservoir.

Our data cannot show which patients carried enterococci, but they support screening MRSA, not all *S. aureus*, when vancomycin resistance is suspected.

Detection of *vanA* in all eight isolates agrees with the literature, in which *vanA* is the cluster found in confirmed VRSA (4) and about 69% of VRSA carry it (7). The *vanB* result is harder to place. Many studies fail to detect *vanB* in VRSA (7), yet reports from Baghdad (66.6% of 12 VRSA) (11), and Shiraz (37% of isolates) (21) resemble our 62.5%, while a recent Indian series found only 11.1% (12). Several explanations are possible. True co-carriage could occur if an isolate acquired elements from more than one enterococcal donor, which is plausible in patients co-colonised with MRSA and VRE (4). Equally, cross-reactive primers, partial operons or contamination can produce false positives. The enterococcal *vanA* system has been characterized in detail in *S. aureus* (22), and some VRSA lose their resistance after subculture. Because *vanB* alone has not been a typical cause of VRSA (4), we regard the five *vanA* + *vanB* isolates as a signal to confirm, not as proof of dual carriage. Sequencing the amplicons, as done in the Lahore study (10), would settle the question.

Pus was the commonest specimen (42.76%). Wounds are the commonest source of VRSA, and they favour co-colonization with VRE and MRSA, with polymicrobial biofilms offering a setting for plasmid transfer (4, 22, 23). Most VRSA patients had received vancomycin in the preceding months (4). Reassuringly, no onward transmission has been documented to date (4), and prompt laboratory detection with standard infection-control measures prevented spread in the early US cases (6). We would therefore suggest MIC confirmation for MRSA from blood and deep infections, routine reporting of vancomycin-resistant isolates to infection-control staff, and tighter vancomycin stewardship.

Limitations

Several limitations should be kept in mind. The study was confined to one hospital over six months, so the findings may not represent other parts of Khyber Pakhtunkhwa. Only eight isolates were vancomycin-resistant, which makes the confidence intervals around the gene frequencies wide. MRSA was defined by cefoxitin, but not confirmed by *mecA*, and we did not sequence the *van* amplicons or type the strains, so the *vanB* results are not confirmed.

No clinical data were collected, so no analysis of risk factors (such as previous use of vancomycin or enterococcal colonisation) was possible. There are no CLSI breakpoints for *S. aureus* with aztreonam or ertapenem, and resistance rates should be interpreted as screening signals only. Lastly, there was no comparison of resistance between MRSA and non-MRSA isolates for each drug.

Future Recommendation

- Conduct multicenter, prospective surveillance in KP with larger sample size and longer study duration.
- Confirm methicillin resistance by *mecA* or *mecC* PCR and vancomycin by broth microdilution for all MRSA from blood and deep-seated infections.
- Sequence *vanA* and *vanB* amplicons, and perform whole genome sequencing and plasmid typing to determine if there is true *vanA*–*vanB* co-carriage and how the genes are disseminated.
- Screen for vancomycin-resistant enterococci in patients with vancomycin-resistant *S. aureus* and gather clinical information to determine risk factors and outcomes. In future analyses, report MRSA against non-MRSA comparisons, multidrug resistance and the multiple antibiotic resistance index.
- Implement vancomycin stewardship, contact precautions and early notification of resistant isolates to infection control teams.

CONCLUSION

One-third of *S. aureus* isolates were MRSA, resistance to conventional agents was prevalent, and linezolid and tigecycline were the most effective choices at this Swat hospital. Vancomycin resistance was only found in MRSA (5.52%) and all vancomycin resistant isolates had *vanA*, five of which had *vanB*. The results suggest the need for MIC testing for vancomycin, molecular confirmation by sequencing, enterococcal screening, and enhanced vancomycin stewardship, with the need for larger multicenter studies.

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