Tropical Journal of Pharmaceutical Research August 2026; 25 (8): 1143-1151
ISSN: 1596-5996 (print); 1596-9827 (electronic)

Available online at http://www.tjpr.org
https://dx.doi.org/10.4314/tjpr.v25i8.11

Original Research Article

Incidence of vancomycin-resistant *Staphylococcus aureus* from clinical isolates in a tertiary health facility in Northern Nigeria

Olatunji Kayode Aremu[1]*, Josiah Ademola Onaolapo[2], Yakubu Kokori Ibrahim[2], Igwe J Chibueze[3]

[1]Department of Pharmaceutical Microbiology and Biotechnology, Federal University Oye-Ekiti, Ekiti State, [2]Department of Pharmaceutical Microbiology and Biotechnology, Ahmadu Bello University, Zaria, [3]Department of Pharmaceutical Microbiology and Biotechnology, Faculty of Pharmaceutical Sciences, Kaduna State University, Kaduna State, Nigeria

For correspondence: **Email: olatunji.aremu@fuoye.edu.ng; **Tel:** 08033116140*

Sent for review: 24 March 2026 *Revised accepted: 15 August 2026*

Abstract

Purpose: *To evaluate the presence of genes associated with vancomycin resistance among S. aureus isolates recovered from clinical specimens collected in a hospital setting and to assess the possibility of S. aureus harboring genes that encode resistance to vancomycin in that setting.*

Methods: *Clinical samples of S. aureus were obtained at the Department of Medical Microbiology Laboratory of Ahmadu Bello Teaching Hospital, Shika, Zaria. Determination of vancomycin resistance and the confirmatory test were done using the broth dilution method and Brain Heart Infusion (BHI) agar plates incorporated with 4 and 8 µg/mL vancomycin, respectively. Confirmation of the presence of the vancomycin resistance genes was carried out and serial culturing in an antibiotics free media was used to determine the phenotypic characteristics of the isolates.*

Results: *The study showed that 25 % (36/144) of the S. aureus isolates were resistant to 30 µg vancomycin disc. Sixty-five (45.1 %) vancomycin-resistant Staphylococcus aureus (VRSA) were observed and the VRSA were hetero-resistant, having grown in BHI agar incorporated with 4 µg/mL and 8 µg/mL of vancomycin. Genes that are responsible for vancomycin resistance were not found, but it was observed that with increasing serial dilution, greater proportion of the previously resistant isolates became susceptible and the minimum inhibitory concentrations (MICs) of those that were still within the resistant range were discovered to be lower compared to the pre-serial dilution values.*

Conclusion: *This study isolated S. aureus that exhibited intermediate and resistant phenotypic characteristics to vancomycin in an environment where vancomycin is not commonly prescribed.*

Keywords: *Antibiotic resistance, Incidence, Vancomycin, VISA, VRSA*

Tropical Journal of Pharmaceutical Research is indexed by Scopus, Chemical Abstracts, Embase, Index Copernicus, EBSCO, African Index Medicus, JournalSeek, Directory of Open Access Journals (DOAJ), African Journal Online, Bioline International, Open-J-Gate and Pharmacy Abstracts

INTRODUCTION

The emergence and spread of antimicrobial resistance among bacterial pathogens have become a major global public health challenge. This is largely driven by the overuse and misuse of antibiotics in human medicine, their routine use as growth promoters in animal feed, and the rapid movement of resistant strains across regions through travel, trade, and inadequate infection control practices [1].

Staphylococcus aureus is a major opportunistic pathogen owing to its virulence factors and ability

to adapt to diverse environments. It causes a wide spectrum of serious infections, including skin and soft tissue infections, osteomyelitis, pneumonia, and bacteremia [2]. Mortality rates from *S. aureus* bacteremia remain high, ranging from 20 to 40 %, despite available antimicrobial therapy [3].

MRSA has risen dramatically worldwide. Initially limited to healthcare settings, these strains are currently widespread in communities, making treatment and control more difficult [4].

Vancomycin, a glycopeptide that inhibits cell wall biosynthesis, has served as the primary agent for serious MRSA infections. However, isolates with reduced susceptibility have emerged and are classified as vancomycin-intermediate *S. aureus* (VISA, MIC 4 – 8 µg/mL) or vancomycin-resistant *S. aureus* (VRSA, MIC ≥16 µg/mL) [5]. These phenotypes develop through stepwise genetic changes in genes involved in cell envelope biosynthesis [6].

The first VRSA strain was reported in the United States in 2002, with resistance conferred by the *vanA* gene cluster on a conjugative plasmid, which was transferred from vancomycin-resistant enterococci to MRSA [7]. The VRSA infections are uncommon but difficult to treat and often multidrug-resistant. VISA strains are encountered more frequently and are associated with treatment failure, recurrent infections, and poor outcomes [8]. Key phenotypic features include increased cell wall thickness, reduced autolysis, altered teichoic acids, and decreased lysostaphin susceptibility [9].

These changes arise under antibiotic pressure through mutational selection or horizontal gene transfer via transformation, conjugation, or transduction. Despite growing international reports, data on VRSA in many African countries, including Nigeria, remain limited [10,11].

The present study therefore determined the incidence of VRSA among clinical isolates in a tertiary health facility in Northern Nigeria. The objectives were to isolate and characterise *S. aureus* from clinical specimens using standard methods, identify resistant isolates by MIC determination, and assess whether resistance is due to genotypic or phenotypic variations.

EXPERIMENTAL

Culture media

Mannitol Salt agar, DNase agar, Nutrient agar, Nutrient broth, Mueller Hinton agar, Peptone water, Mueller Hinton broth, all from Oxoid Ltd, UK.

Chemicals

Sodium chloride, Analar grades of hydrochloric acid, sulphuric acid, methylene blue, acetone, crystal violet, dilute carbol fuchsin solution.

Collection of clinical samples

One hundred and seventy-seven (177) *S. aureus* isolates were obtained from various specimens submitted to the Medical Microbiology Laboratory of ABUTH, Shika, Nigeria over a cumulative period of eight months. The isolates identified as *S. aureus* were collected on nutrient agar slants. The slants were incubated for 18 h at 37 °C and kept refrigerated until needed. The isolates were then purified, identified and confirmed to be *S. aureus* by Gram staining and growth on mannitol salt agar. The biochemical tests that were done include: catalase test, coagulase slide test and deoxyribonuclease (DNase) test according to Cheesbrough [12].

Determination of methicillin and vancomycin resistance

The antimicrobial susceptibility pattern of each *S. aureus* isolate was determined using Kirby-Bauer-NCCLS modified disc diffusion technique [13]. Discrete colonies of isolates on nutrient agar plates were emulsified in 3 mL of saline solution and the turbidity was adjusted to 0.5 McFarland standard. Using sterile swab sticks, the surface of Mueller-Hinton Agar (MHA) in a 90-mm diameter plate was inoculated with the bacterial suspension by streaking the surface of agar in three directions, rotating the plate approximately 60° to ensure even distribution. The inoculated plates were left to dry for 10 minutes before the antibiotic discs were applied aseptically to the surface of the agar. After 30 minutes of applying the discs, the plates were inverted and incubated at 35°C. The same treatment was extended to standard *S. aureus* ATCC 25923, which was used as control. The following antibiotics were tested: cefoxitin (5 µg), vancomycin (30 µg) and oxacillin (10 µg). The diameter of the zone of inhibition produced by each antibiotic disc was measured, recorded and the isolates were classified as “resistant”, “intermediate” and “sensitive” based on the standard interpretative chart updated according to the current NCCLS standard [13,14] and Fluka zone interpretative chart in accordance with WHO requirements (flukatec@eurnotes.sial.com).

Determination of minimum inhibitory concentration (MIC) of vancomycin

The MICs of vancomycin for each isolate were determined by the broth dilution method as described by Cheesbrough [12]. Seventy-eight of the isolates that showed multidrug resistance (MDR; i.e., those that were resistant to three or more classes of antibiotics using disc agar diffusion) were selected for the vancomycin MIC determination.

Confirmatory test for VRSA

Suspect VRSA isolates were sub-cultured on Brain Heart Infusion (BHI) agar plates incorporated with 4 and 8 µg/mL vancomycin. The presence of VRSA is evident when there is growth.

Determination of nature of vancomycin resistance among the isolates

In order to test if the resistance is due to genotypic or phenotypic factors, the method of Cui *et al* [14] was adopted. Isolates that expressed phenotypic vancomycin resistance were selected for determination of the resistance profile. The isolates were sub-cultured into antibiotic-free brain heart infusion (BHI) broth daily for 10 days using three test tubes per isolate. Four and half milliliters (4.5 mL) of BHI broth were measured into each test tube labeled 1, 2, and 3 and sterilized. Half a milliliter (0.5 mL) of the prepared culture was then added to test tube 1 and transferred serially to test tubes 2 and 3; thereafter, the serially diluted test tubes were incubated. This procedure was done for 10, 20 and 30 days for each of the isolates.

Molecular characterization of vancomycin-resistant *S. aureus*

Bacterial cell preparation

Preparation of the isolates was carried out using the method described by Dubey *et al* [15]. Single colonies were picked from freshly streaked isolates on mannitol salt agar and inoculated into 5 mL Luria-Bertani (LB) broth medium, incubated at 37 °C for 24 h.

Genomic DNA extraction

Genomic DNA extraction was carried out using the method described in the DNeasy Blood and Tissue Handbook [16]. It was then centrifuged for 1 minute at 10,000 rpm. The purification column was discarded and the purified DNA was used as template to detect the gene of interest. The DNA was confirmed by agarose gel electrophoresis.

Agarose gel electrophoresis

The eluent was subjected to agarose gel electrophoresis to ascertain that genomic DNA was extracted. When electrophoresis was completed, the gel was removed from the buffer (Tris) and viewed under a Trans-illuminator UV light of wavelength 302 nm. Afterward, the band pattern of the DNA fragments was then photographed with a Polaroid camera and documented using an electrophoresis gel documentation system.

Detection of resistance gene by PCR

Amplification of resistance DNA fragments was carried out on the extracted genomic DNA by PCR, as reported by Brakstad *et al* [17], using specific primers for *vanA, vanH, vanX* and the two selected antitoxins (*hlg* and PVL). The primer sequences specific for *vanHAX*, *vanH, vanA* and *vanX* are as presented in Table 1.

RESULTS

Antibiotics susceptibility

Significant incidence: 70.3 % (101/144) of the coagulase-positive *S. aureus* (144) was observed to show resistance to both oxacillin and cefoxitin, an indication of phenotypic resistance to methicillin, while 25 % (36/144) of these isolates also expressed resistance to vancomycin (Figure 1).

Minimum inhibitory concentrations of vancomycin

Sixty-five (45.1 %) of the isolates were discovered to be Vancomycin-Resistant *Staphylococcus aureus* (VRSA**;** with MICs ≥ 8 µg/mL)

Confirmatory for isolated VRSA

All Brain Heart Infusion (BHI) agar plates supplemented with 4 and 8 µg/mL vancomycin showed growth, confirming the isolates as vancomycin-resistant S. aureus (VRSA) and indicating hetero-resistance.

Table 1: Primer sequences and the expected base pairs of Vancomycin genes analyzed [18]

Specific gene for amplification	Primer sequence (5′-3′)		Expected size of amplicon (bp)
*van*HAX	Forward	ATGAATAACATCGGCATTAC	2,600
	Reverse	TTATTTAACGGGGAAATC	2,300
*Van*H	Forward	ATGAATAACATCGGCATTAC	969
	Reverse	CTATTCATGCTCCTGTCTCC	
*Van*A	Forward	ATGAATAGAATAAAAGTTG	1,032
	Reverse	TCACCCCTTTAACGCTAATA	
*Van*X	Forward	ATGGAAATAGGATTTACTTT	609
	Reverse	TTATTTAACGGGGAAATC	

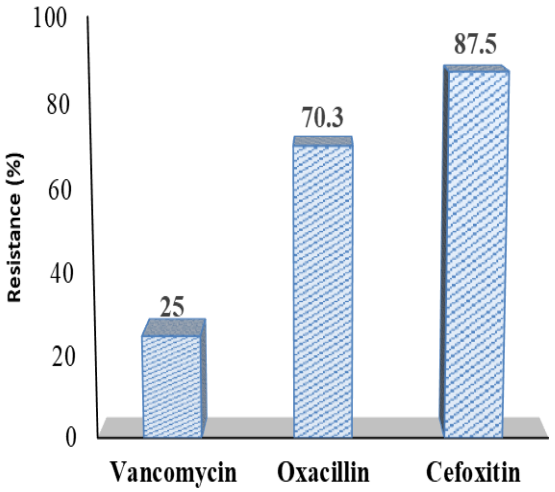

Figure 1: Percentage resistance of *S. aureus*

Table 2: Minimum inhibitory concentrations of vancomycin against the *S. aureus* isolates

MIC (µg/mL)	Number of Isolates (n=78)	Distribution (%)
4	13	16.7
8	0	0
16	0	0
32	1	1.2
62.5	2	2.5
125	3	3.8
250	14	17.9
500	17	21.8
1000	15	19.2
2000	13	16.7
4000	0	0

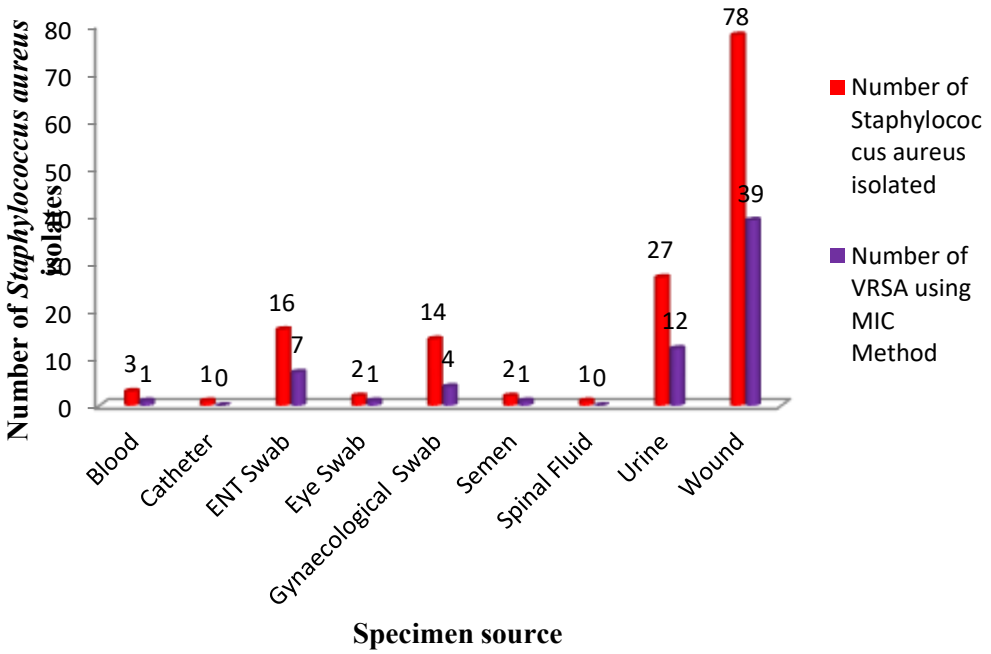

Figure 2: Distribution of VRSA isolates obtained using MIC in relation to specimens' source

Nature of isolates

Following serial dilution for 10 and 20 days, only 12 of the 36 isolates remained resistant. This number further decreased to 8 isolates by 30 days of serial dilution in an antibiotic free media. The highest MIC after 30 days of dilution obtained was 62.5 µg/mL compared to 500 µg/mL before the procedure. It was observed that with increasing serial dilution, greater proportion of the previously resistant isolates might become susceptible, while the MICs of those that were still resistant continued to decrease (Table 3).

Table 3: Comparison of percentage distribution of vancomycin MIC against the isolates

MIC value (µg/mL)	Percentage Distribution			
	Day 0	**Day 10**	**Day 20**	**Day 30**
4	36.1 (13)	0.0 (0)	0.0 (0)	5.6(2)
8	0(0)	0.0 (0)	11.1(4)	2.7(1)
16	0(0)	2.8(1)	5.6(2)	5.6(2)
32	2.8(1)	5.6(2)	13.9(5)	5.6(2)
62.5	5.6(2)	30.6(11)	2.7(1)	2.7(1)
125	8.3(3)	8.3(3)	0.0 (0)	0.0 (0)
250	38.9(14)	0.0 (0)	0.0 (0)	0.0 (0)
500	8.3(3)	0.0 (0)	0.0 (0)	0.0 (0)
Total	**100 (36)**	**47.2(17)**	**33.3(12)**	**22.2(8)**

Molecular analysis

Genomic DNA extraction

All 8 isolates that still had ≥ 4 µg/mL MIC after 30 days of serial dilution in an antibiotic-free medium were subjected to genomic DNA extraction, as shown in Figure 2.

Multiplex polymerase chain reaction

The amplification at 609 base pairs essentially signified that VanA and VanH are not too clear in this research work, so the presence of these genes cannot be substantiated categorically (Figure 3).

DISCUSSION

The emergence of *S. aureus* with intermediate resistance (VISA) and high resistance profile (VRSA) in clinical samples, especially in a setting where vancomycin is not actively being prescribed, is a serious global public health concern that affects nations irrespective of their level of development. This study elucidates the presence of vancomycin-resistant strains among *S. aureus* isolated from different clinical samples in a tertiary institution in Northern Nigeria.

The reduced susceptibility of *S. aureus* to vancomycin observed in this study is alarming since vancomycin is a reserved drug for life-threatening *S. aureus* infections that are recalcitrant to multiple antibiotics [19]. A similar percentage of resistance has been widely reported within and outside Nigeria [19]. This resistance may be due to the acquisition of resistance-determining genes like mec A and Van A, B, C, or as a result of the thickening of the cell wall, as reported by some authors [20]. A review of available biochemical studies by Nikolic and Mudgil [9] also suggested that *S. aureus* resists glycopeptides primarily by limiting drug access to its site of action, that are located on the cell membrane.

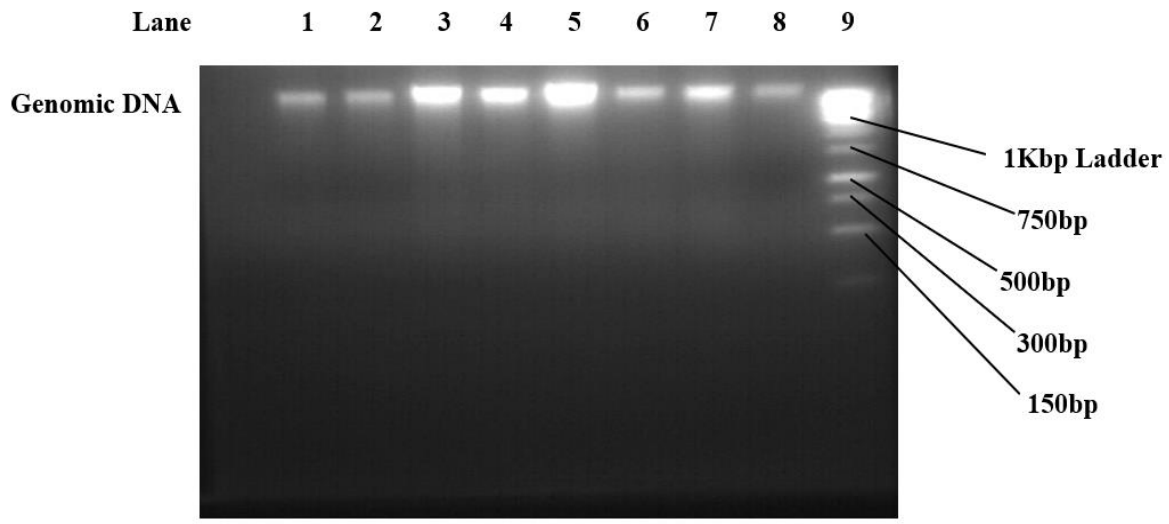

Figure 2: Electrophoregram of genomic DNA extraction on 1.5 % agarose gel**. Keys:** Lane 1 – 8: isolates with vancomycin resistance profile, Lane 9: 1 kb ladder

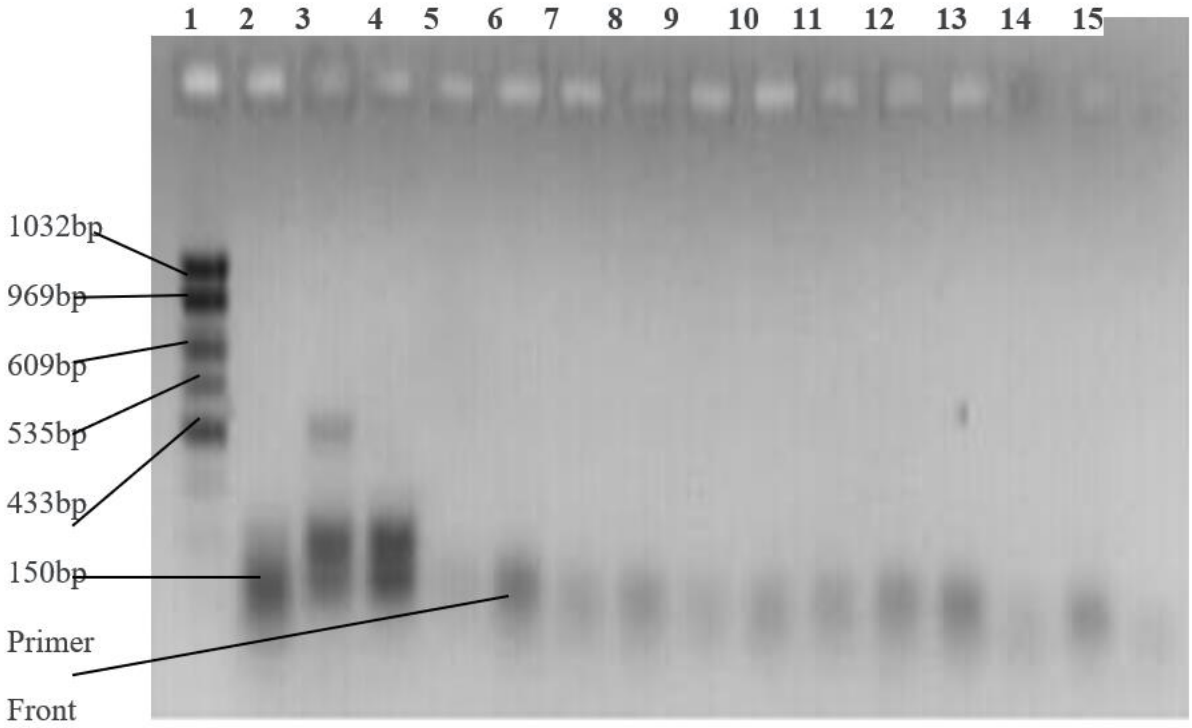

Figure 3: Electropherogram of vancomycin resistance genes. **Keys:** Lane 1: 1kb ladder, Lane 2: Negative Control- Contains all other components of the PCR mix but no template, Lane 3: Positive Control-Contains all the components of the PCR mix and a standard MRSA strain, Lane 4: Multiplex Control – The standard that came with a control primer, Lanes 5 - 12: VRSA isolates

This occurs through cell wall thickening and alterations in cell wall composition, instead of drug inactivation or modification of the target site. A significant number (34) of the *S. aureus* isolates in this study grew on 4 and 8 µg/mL of Vancomycin incorporated into Brain Heart Infusion screening agar plates within 24 hours. Nikolic and Mudgil [9] stated that growth on BHI agar with 4 µg/mL Vancomycin is one of the criteria for classification of VRSA, provided that vancomycin broth microdilution MIC is greater than or equal to 8 µg/mL. Growth on 8 µg/mL agar is used to confirm whether the isolate has hetero-resistant ability. It is important to detect both VRSA and h-VRSA in the clinical laboratory since the two phenotypes have been associated with treatment failure with glycopeptides in deep-seated infections [8,18]. The following factors

have been cited to be responsible for VRSA: Increased thickness of cell wall material; Reduced peptidoglycan cross-linkages; Inactivation of penicillin-binding protein 4 (PBP 4) and other cell wall alterations such as decreased glycan chain length and absence of PBP2 [8,19].

In view of these reports, several different mutations can cause vancomycin resistance in *S. aureus*. These alterations, however, do not affect the structure of the vancomycin binding target (D-Ala-D-Ala residue of peptidoglycan). Although the precise role of these individual factors in the reported resistance mechanism remains unclear, they seem to create an obstacle that prevents vancomycin from reaching its target on the cytoplasmic membrane where nascent peptidoglycan biosynthesis is ongoing [8,19]. Other studies indicate that structural or metabolic alterations in cell wall teichoic acids can contribute to vancomycin resistance by slowing the rate of cell wall degradation, rather than accelerating cell wall synthesis/ This mechanism helps maintain the link between cell wall thickening and decreased susceptibility to vancomycin [8].

Some studies have reported increased empirical use of vancomycin since the 1980s due to the emergence of MRSA in health-care institutions, which has established selective pressure that may have eventually led to the emergence of *S. aureus* and other *Staphylococcal* species with decreased susceptibility to vancomycin [3,9]. However, in the present environment, Vancomycin selective pressure is relatively low because it is not frequently used, yet a high percentage of resistance was still recorded. David and Daum supported this finding by reporting that prior or recent exposure to vancomycin is not required for the emergence of VRSA. Rather, frequent use of other antimicrobial agents can generate enough selective pressure to promote colonization or infection with VRE and MRSA, which may ultimately lead to the development of VRSA [2].

The evolution of antimicrobial-resistant bacterial species is driven by the widespread and often inappropriate use of these drugs in human medicine, their extensive application as growth promoters in livestock feed, and the increasing movement of people and goods across regions. These factors have greatly facilitated the spread of resistant strains across geographic boundaries [1,9]. The phenotypic stability of vancomycin resistance in *S. aureus* was assessed in this study by serial subculturing of selected VRSA isolates in antibiotic-free medium for 30 days. At the end of this period, only eight (23.5 %) of the tested isolates retained their resistant phenotype. This finding is consistent with previous observations on the instability of the vancomycin-resistant phenotype [8,18]. In a similar study, Cui *et al.* observed complete reversion to susceptibility after 84 days of serial passages in antibiotic-free medium. The authors suggested that such phenotypic reversion may explain the relatively low frequency of VRSA isolation observed in both clinical and laboratory settings [8].

There is growing concern about the possible emergence of VRSA in multidrug-resistant *S. aureus* strains, particularly after laboratory studies successfully demonstrated the transfer of the vanA gene from enterococci to *S. aureus* [19]. Another study, however, reported that acquisition of the enterococcal vancomycin resistance mechanism by staphylococci was observed in clinical isolates [19]. However, cases of reduced vancomycin susceptibility have been documented in both Japan and the United States, often linked to failure of vancomycin treatment of MRSA infection [21]. These observations have been attributed to the presence of a thickened cell wall in VRSA strains, resulting from excessive peptidoglycan accumulation. This thickened cell wall appears to be a consistent feature observed in VRSA clinical isolates reported from different countries [14].

PCR screening of the tested isolates using vanA, vanHAX, vanH, and vanX gene-specific primers did not detect any of the targeted vancomycin resistance genes. The absence of these genes suggests that the observed resistance phenotype may not be associated with the acquisition of the van operon, which is commonly implicated in high-level vancomycin resistance. Similar observations have been reported in some vancomycin-intermediate Staphylococcus aureus (VISA) strains, where resistance occurs in the absence of transferable van genes [9].

The resistance observed in the present study may therefore be attributed to intrinsic physiological adaptations of the bacterial cell wall. Previous studies have shown that vancomycin resistance can arise through progressive thickening of the cell wall, resulting in the sequestration of vancomycin molecules within the outer peptidoglycan layers before they reach their target sites. Alterations in peptidoglycan composition, including an increased proportion of non-amidated muropeptides and reduced peptidoglycan cross-linking, have also been associated with decreased susceptibility to vancomycin [5.9]. These structural modifications reduce the

efficiency of antibiotic penetration and binding, thereby enabling bacterial survival despite exposure to the drug.

The findings of this study suggest that the vancomycin-resistant isolates may possess resistance mechanisms that are independent of the vanA gene cluster. This observation highlights the complexity of vancomycin resistance in Staphylococcus aureus and underscores the importance of investigating alternative genetic and physiological determinants responsible for the resistance phenotype.

CONCLUSION

The preponderance of *S. aureus* among the *Staphylococcus* specimens showed that *S. aureus* could be isolated from most clinical samples in ABUTH, Shika, and is a common nosocomial agent in the study environment. Though vancomycin is not commonly used in the study area, the high level of vancomycin resistance among the *S. aureus* isolates is worrisome. The finding that phenotypic adaptation is most likely the major factor responsible for the prevalence of vancomycin-resistant *S. aureus* in the environment where this study was conducted indicates that mutations in the genome for adaptation purposes are largely responsible. The non-detection of the common resistance genes (*van*A, *van*HAX, *van*H and *van*X) and the antitoxins (hlg and Panton Valentine Leucocidin) in the VRSA isolates further buttress the fact that the major factor responsible for the high incidence of VRSA in the environment is phenotypic and not genotypic. This, however, does not exclude the fact that other resistance plasmids outside the ones tested for in this study may be involved.

DECLARATIONS

Acknowledgement/Funding

None.

Ethical approval

Not applicable.

Use of Artificial intelligence/Large language models

The authors confirm that AI-based tools were used solely for language editing and manuscript polishing purposes (e.g., grammar, phrasing, and clarity). No AI tools were used to generate content, interpret data, or influence study design, results, or conclusions. All intellectual contributions, study analyses, and interpretations reported in the manuscript are the original work of the authors.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Conflict of interest

No conflict of interest is associated with this work.

Contribution of authors

We declare that this work was done by the author(s) named in this article, and all liabilities pertaining to claims relating to the content of this article will be borne by the authors.

REFERENCES

1. *Muteeb G, Rehman MT, Shahwan M, Aatif M. Origin of antibiotics and antibiotic resistance, and their impacts on drug development: A narrative review. Pharmaceuticals 2023; 16(11): 1615. https://www.mdpi.com/1424-8247/16/11/1615#*
2. *David MZ, Daum RS. Community-associated methicillin-resistant Staphylococcus aureus: epidemiology and clinical consequences of an emerging epidemic. Clin Microbiol Rev 2010; 23(3): 616-687. https://doi.org/10.1128/cmr.00081-09*
3. *Van Hal SJ, Jensen SO, Vaska VL, Espedido BA, Paterson DL, Gosbell IB. Predictors of mortality in Staphylococcus aureus bacteremia. Clin Microbiol Rev 2012; 25(2): 362-386. https://doi.org/10.1128/cmr.05022-11*
4. *Li J, Cheng F, Wei X, Bai Y, Wang Q, Li B, et al. Methicillin-resistant Staphylococcus aureus (MRSA): Resistance, prevalence, and coping strategies. Antibiotics 2025; 14(8): 771. https://www.mdpi.com/2079-6382/14/8/771#*
5. *Wang Y, Sun Y, Chen G, Cheng X. Emerging antimicrobial strategies against heterogeneous and vancomycin-intermediate Staphylococcus aureus. Int J General Med 2025: 5329-5344. https://doi.org/10.2147/IJGM.S532471*
6. *Daffé M, Crick DC, Jackson M. Genetics of capsular polysaccharides and cell envelope (glyco) lipids. Molecular Genetics of Mycobacteria 2014: 559-609. https://doi.org/10.1128/9781555818845.ch28*
7. *Haas W, Singh N, Lainhart W, Mingle L, Nazarian E, Mitchell K, et al. Genomic analysis of vancomycin-resistant Staphylococcus aureus isolates from the 3rd*

case identified in the United States reveals chromosomal integration of the vanA locus. Microbiol Spec 2023; 11(2): e04317-4322. https://doi.org/10.1128/spectrum.04317-22

8. Cui R, Song Z, Suo B, Tian X, Xue Y, Meng L, et al. Correlation analysis among genotype resistance, phenotype resistance and eradication effect of Helicobacter pylori. Infection Drug Resist 2021: 1747-1756. https://doi.org/10.2147/IDR.S305996
9. Nikolic P, Mudgil P. The cell wall, cell membrane and virulence factors of Staphylococcus aureus and their role in antibiotic resistance. Microorganisms 2023; 11(2): 259. https://doi.org/10.3390/microorganisms11020259
10. Bamigboye BT, Olowe OA, Taiwo SS. Phenotypic and molecular identification of vancomycin resistance in clinical Staphylococcus aureus isolates in Osogbo, Nigeria. European Journal of Microbiology and Immunology. 2018 Mar;8(1):25-30.
11. Anie CO, Ibezim EC, Esimone CO, Arhewoh MI. Molecular characterization of selected nasal isolates of methicillin-resistant Staphylococcus aureus (MRSA) from healthy students of a tertiary institution. Trop J Pharm Res 2022; 21 (4): 825-831. http://dx.doi.org/10.4314/tjpr.v21i4.20
12. Cheesbrough M. District laboratory practice in tropical countries (Part 2, 2nd ed.). Cambridge University Press. 2006. https://doi.org/10.1017/CBO9780511543470
13. Abubakar U, Adisa JO, Mohammed U, Tsamiya RI, Mohammed MO, Muhammad AT, et al. Chronic toxicity study of aqueous stem bark extract of Khaya senegalensis on the histology of the liver and its biochemical parameters in Wistar rats. Asian J Res Botany 2020; 3(3): 1-9.
14. Cui R, Song Z, Suo B, Tian X, Xue Y, Meng L, et al. Correlation analysis among genotype resistance, phenotype resistance and eradication effect of Helicobacter pylori. Infection Drug Resist 2021: 1747-1756. https://doi.org/10.2147/IDR.S305996
15. Dubey KK, Haque S, Jawed A, Singh BP, Behera BK. Construction of recombinant Escherichia coli for enhanced bioconversion of colchicine into 3-demethylated colchicine at 70 L bioreactor level. Process Biochem 2010; 45(7): 1036-1042. https://doi.org/10.1016/j.procbio.2010.03.014
16. Qiagen. DNeasy Blood & Tissue Handbook. Hilden, Germany: Qiagen; 2020
17. Brakstad OG, Maeland JA, Tveten Y. Multiplex polymerase chain reaction for detection of genes for Staphylococcus aureus thermonuclease and methicillin resistance and correlation with oxacillin resistance. APMIS 1993; 101(9): 681-688. https://doi.org/10.1111/j.1699-0463.1993.tb00165.x.
18. Donabedian S, Hershberger E, Thal LA, Chow JW, Clewell DB, Robinson-Dunn B, et al. PCR fragment length polymorphism analysis of vancomycin-resistant Enterococcus faecium. J Clin Microbiol 2000; 38(8): 2885-2888. https://doi.org/10.1128/JCM.38.8.2885-2888.2000
19. OG B. Multiplex polymerase chain reaction for detection of genes for Staphylococcus aureus thermonuclease and methicillin resistance and correlation with oxacillin resistance. APMIS 1993; 101: 681-688.
20. Li G, Walker MJ, De Oliveira DM. Vancomycin resistance in Enterococcus and Staphylococcus aureus. Microorganisms 2022; 11(1): 24. https://doi.org/10.3390/microorganisms11010024
21. Blechman SE, Wright ES. Vancomycin-resistant Staphylococcus aureus (VRSA) can overcome the cost of antibiotic resistance and may threaten vancomycin's clinical durability. PLOS Pathogens 2024; 20(8): e1012422.