Review Article

A Review of Vancomycin-Resistant *Staphylococcus aureus* Prevalence in Egypt and Saudi Arabia

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ABSTRACT

Staphylococcus aureus is considered an important and dangerous pathogen worldwide. Vancomycin was extensively used for treatment of the emerging methicillin-resistant *Staphylococcus aureus* (MRSA), but this led to the emergence of vancomycin-resistant *Staphylococcus aureus* (VRSA). Epidemiological and molecular data on VRSA are still scarce in both Egypt and Saudi Arabia. We conducted the present study to review the emergence of VRSA in Egypt and Saudi Arabia until December 2018. Among 220 VRSA isolates reported, only 10 of them were detected in Saudi Arabia and the remaining 210 were detected in Egypt. Nearly, all the reported VRSA isolates were multidrug resistant. Many factors contribute to differences in the prevalence of VRSA between Egypt and Saudi Arabia. Accurate diagnostic techniques, stringent infection control, rationale antibiotic use, environmental hygiene and improving the knowledge of the healthcare workers about VRSA in Saudi Arabia are proved to be effective in limiting its spread.

Key words: MDR, MIC, MRSA, *Staphylococcus aureus*, Vancomycin, VRSA.

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INTRODUCTION

Staphylococcus aureus (SA) is a common cause of infections; both nosocomial and community acquired. In humans, the skin and nasal cavity commonly harbor these bacteria.[1] SA can cause a wide range of diseases ranging from wound, skin and bone infections to bloodstream infection, endocarditis and devastating septicemia.[2-4] The emergence of methicillin-resistant *Staphylococcus aureus* (MRSA) in the early 1960s[5] enforced the use of Vancomycin (VA) as a first therapeutic option, but due to its non-optimal use, reduced susceptibility and enhanced resistance to VA ended with the emergence of vancomycin-resistant *Staphylococcus aureus* (VRSA).[2,3] The first report of decreased susceptibility of SA to VA was in 1997. The so-called Vancomycin intermediate-resistant *Staphylococcus aureus* (VISA) and the hetero-VISA (hVISA) isolates were reported in Japan. VA Minimal inhibitory concentrations (MIC) were 8 mg/L for the former and 2mg/L for the later,[3,6] Later on, serious concerns were issued when VRSA with VA MIC of 32 mg/L was reported in Michigan, USA.[7] Since then, VRSA had become an increasing major health concern due to the improper choices of antibiotics.

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Mechanisms of Vancomycin Resistance

The resistance of SA to VA has been identified in 2 forms; the 1st one depends on target overproduction and the 2nd depends on target changing.[8] With a MIC to VA of 4-16 µg/ml, the first form of VISA strains has been identified. It seems that repeated exposure to VA resulted in emergence of VISA isolates from VA heterogeneously resistant subpopulations.[9] VISA strains showed a significant increase in the synthesis of peptidoglycan which makes the cell wall looks irregularly shaped, thickened and with reduced cross-linking.[8] Consequently, the excess D-Ala-D-Ala residues, presenting "target overproduction", bind and trap VA preventing it from interacting with their target on the bacterial cytoplasmic membrane.[9]

The second form of VA resistance has resulted from the plausible conjugal transfer of the vanA operon from a VA-resistant *Enterococcus faecalis*. The Enterococcal plasmid containing vanA also encodes a sex pheromone which is synthesized by SA, to facilitate the conjugal transfer of the plasmid.[10] These VRSA isolates were completely VA resistant, with MICs of ≥16 µg/ml, due to replacement of D-Ala-D-Ala by D-Ala-D-Lac "target changing". Synthesis of D-Ala-D-Lac occurs only on exposure to low levels of VA.[11] The bacteria are characterized by ecological fitness in which there is a high possibility that the plasmid containing vanA is exchanged as there is an increasing possibility of colonization with both MRSA and vancomycin-resistant enterococci (VRE). The resistance of these strains to both β-lactam antibiotics and glycopeptides will increase the likelihood of VRSA strains to become more prevalent rapidly.[9]

There are 6 phenotypes of VA resistance (VanA, VanB, VanC, VanD, VanE and VanG) that have been identified.[12] Phenotype VanD, VanE and VanG are uncommon. Both VanA and VanB phenotypes are common and could be transferred. The VanA phenotype exhibits high-level resistance to VA and teicoplanin, while the VanB phenotype confers variable degrees of resistance to VA, but not teicoplanin. VanB phenotype has no resistance to teicoplanin. The VanC phenotype is cannot be transferred. It is limited to *Enterococcus casseliflavus* (*Enterococcus flavescens*) and *Enterococcus gallinarum*.[13]

Resistant VISA isolates adopt mechanisms, which evolve on exposure to VA and are not readily transferrable to other strain, so in the absence of VA therapy, there is a low possibility of spread of these isolates. On the contrary, VRSA isolates consistently contain the vanA gene acquired from Enterococcus. Of note, The vanA phenotype could be transferred to other MRSA isolates and even across species, with greater possibility of spread, even without VA.[13] According to Moravvej *et al.*[15] thirty-three VRSA isolates were reported worldwide till the end of 2012, including 16 strains from India, 13 from the USA, 3 from Iran and one from Pakistan.

Ability of Various Methods to Detect Levels of VA Susceptibility/Resistance in SA

The disc diffusion testing cannot differentiate VA-susceptible isolates of SA from VA-intermediate or resistant isolates. Strains of SA producing VA zone of inhibition ≥ 7 mm in diameter may have MICs ranging from ≤ 2 up to 16 µg/mL. Thus, isolates of SA for which the VA zones of ≥ 7 mm should not be reported as susceptible without confirmation by a VA MIC test, even if showing zones of ≥15 mm. The reporting of resistant isolates showing zones of inhibition < 7 mm in diameter should be confirmed by a VA MIC test (Table 1).[16]

The aim of the present review is to report the total number of VRSA isolates reported from Egypt and Saudi Arabia until December 2018 depending on the clinical and laboratory standards institute (CLSI) guidelines. Pubmed and other related databases were reviewed and VRSA isolates were included in this review depending on the guidelines of CLSI 2014 (Table 1).

VRSA Reports in Egypt and Saudi Arabia

Nine reports of VRSA from Egypt and Saudi Arabia were found until December 2018. All the demographic and laboratory characteristics of these reported VRSA isolates are illustrated in (Table 2). The 9 reports reported 220 isolates of VRSA, only 10 of them were detected in Saudi Arabia and the remaining 210 were detected in Egypt. Nearly, all the reported VRSA isolates were multidrug resistant (MDR) as illustrated in (Table 3).

The present study described the burden of VRSA in Egypt and Saudi Arabia until December 2018. However, some reports might be misleading and we should highlight that the actual number of VRSA isolates might be more or less than the reported number of resistant isolates in these reports because not all the studies have the definitive molecular approach to properly detect VA-resistant strains and also, some studies failed to utilize the guidelines and standards of the CLSI for laboratory procedures. Accordingly, articles 7, 17, 19, 22 and 25 can be considered as credible articles because they performed the definitive molecular and CLSI guidelines. As demonstrated in Table 3, Nearly, all the reported VRSA isolates were MDR.

Table 1: Ability of disc diffusion/MIC methods to detect levels of VA susceptibility/resistance in SA according to CLSI 2014.[16]

Vancomycin MIC (µg/mL)	MIC method	Disk diffusion (DD) method
≤2 (S)	Yes	No
4 (I)	Yes	No
8 (I)	Yes	No
16 (R)	Yes	No
≥32 (R)	Yes	Yes (zone of inhibition < 7 mm)

- SA with VA MICs ≥ 32 µg/mL can be detected by either DD or MIC.
- Strains with VA MICs < 32 µg/mL are not detected by DD, even with 24 hr incubation.
- In order to recognize strains of *Staphylococci* for which the VA MICs < 32 µg/mL, MIC testing must be done and the tests must be incubated for 24 hrs at 35°C.

Table 2: Demographic and laboratory characteristics of reported VRSA and associated VISA isolates in Egypt and Saudi Arabia till December 2018.

Reference	Year of isolation/ publication	Location of isolation	Samples	Number of isolates	Vancomycin MIC (µg/mL)	PCR for vanA	Sensitive to	Others
[17]	2008	Egypt (Mansoura)	Blood (1), bed sore (1)	VRSA (2)	33.5 (Mean)	+ve in 100%	Daptomycin, Quinipristin/ dalfopristin, Tigecyclin	All represent 9.23% of isolated MRSA. Risk factors for all : DM, liver cirrhosis, unhealed chronic wounds, patients received immunosuppressive therapy, prolonged hospital stay and previous use of antibiotics.
			Skin ulcer (5), Surgical wound (5), LRTI (2)	VISA (12)	11.33 (Mean)	+ve in 83.3%		
			Blood (8), skin ulcer (8), surgical wound (6), urine (2) others (3)	hVISA (27)	4.15 (Mean)	+ve in 66.7%		
[18]	2010	Saudi Arabia (Riyadh)	Blood	VISA (1)	4	NR	Linezolid Rifampin Tigecycline	The case was on VA/ Meropenem empirical therapy.
[19]	2012	Egypt (Zagazig)	Pus (6), abscess (1), blood (1), urine (1), catheter (1)	VRSA (10)	32	+ve in 50%	Trimethoprime Amikin Rifampin	Represent 13.16% of isolated MRSA. Risk factors; administration of multiple prophylactic and post-operative antibiotics with prolonged hospitalization
[20]	2009-2010/ 2012	Saudi Arabia (Qassim)	Skin swabs from children with atopic dermatitis	VRSA (9)	≥32	NR	Linezolid Levofloxacin Gentamycin Erythromycin	MIC detected by Vitek system
				VISA (7)	8			
[7]	2011-2013/ 2014	Egypt (Minia)	Skin swab	VRSA (1)	16	+ve	• Ciprofloxacin • Amikacin	All represent 37.5% of isolated MRSA
			Skin swabs (2), throat swabs (2), urine (1)	VISA (5)	4-8	NR	• Ciprofloxacin • Amikacin Levofloxacin • Gentamycin	
[21]	2010-2012/ 2014	Egypt (Menoufia)	Different	VRSA (30)	NR	+ve in 51.9%	• Linezolid (100%) • Rifampin (50%) • Amikacin (43%) • Clindamycin (43%) • Tigecycline (40%)	• VRSA prevalence in the National Liver Institute is 20.68%. • Identified by Vitek system.
				VISA (30)	NR	NR	NR	VISA prevalence in the National Liver Institute is 20.68%.
[22]	2011-2012/ 2015	Egypt (Tanta)	Wound pus, sputum, blood, nasal swabs, urine.	VRSA (88)	≥32	+ve in 5 isolates out of 9 selected isolates	• Linezolid (63%) • Cotrimoxazole (56%)	VRSA prevalence is 20.13%. The relatively high rates of VRSA isolates in this study could be partially explained by a selection pressure induced by an inadequate use of antimicrobials.
				VRSA (55)	≥ 16	NR		

[23]	2014/2015	Egypt (Qalyubia)	Nasal swabs	VRSA (3)	NR	NR	• Ciprofloxacin • Tobramycin • Moxifloxacun	Detected by disc diffusion method. Represent 9% of isolated MRSA
		Saudi Arabia (Qassim)	Nasal swabs	VRSA (1)	NR	NR		Detected by disc diffusion method. Represent 4% of isolated MRSA.
[24]	2005-2013/ 2016	Egypt (12 hospitals in Cairo and Alexandria)	Pus (12), wound swabs (23), urine (19), blood (135), bronchoalveolar lavage (123), endotracheal tube (6), sputum (56), others (74)	VISA (4)	4	NR	NR	Identification was done in the US Naval Medical Research Unit No. 3 (NAMRU-3) laboratories. Represent 1.2% of isolated HA-MRSA.
[25]	2014-2016/ 2017	Egypt (Zagazig)	Different	VRSA (10)	≥ 16	+ve	• Tigecyclin (100%) • Linezolid (60%) • Quinipristin/ dalfopristin (40%) • Nitrofurantion (30%)	Represent 11.1% of isolated MRSA. Identified by Vitek2 system.
				VISA (12)	4-8	-ve	• Tigecyclin (100%)	Represent 13.3% of isolated MRSA. Identified by Vitek2 system.
[26]	2013-2014/ 2018	Egypt (Kafreldawar)	Wound swabs, skin swabs, nasal swabs	VRSA (3) VRSA (6) VRSA (2)	≥64 ≥32 ≥16	NR	• Linezolid (36.4%) • Clindamycin (36.4%) • Sulfamethoxazole-trimethoprim (36.4%),	Represent 13.75% of SA clinical isolates. The relatively high prevalence of VRSA isolates in this study may be partially due to a selection pressure induced by the improper use of antimicrobials.
			Nasal swabs	VISA (1)	4-8		NR	Represents 0.5% of isolated SA.

Before VRSA strains were reported, the use of VA was increasing in order to treat MDR SA, MRSA strains, nosocomial infections and infections caused by coagulase-negative staphylococci.[27] Non-optimal use of VA in treating MRSA strains led to the emergence of VRSA.[28] Glycopeptide resistance was first reported in enterococci[29] and caused a great concern as the vanA gene could be transferred from enterococci to form VRSA strains. The first VRSA strain sequencing and identification took place in the United States in 2002; drawing great attention to potential health problem.[30]

Many genes are accused in resistance mechanism of VRSA strains to VA.[31,32] While vanA resistance gene and other resistance genes are co-transferred from *Enterococcus faecalis* to SA,[33] resistance gene (Tn1546) is transferred from glycopeptide-resistant enterococci to some MRSA strains.[31]

The emergence of multidrug-resistant bacteria is a major challenge in medical practice and put an additional burden on healthcare sectors.[34] Several factors were accused of the emergence of VRSA; inadequate MRSA or VRE strains treatment, careless antibiotics prescription with unnecessary exposure to VA, the availability of antibiotics without prescription, prolonged hospitalization, extensive surgical procedures and underlying disease as diabetes, renal failure or malignancy.[2,7,31]

In Egypt and Saudi Arabia, the development of VRSA might be related to the selective VA pressure in treating MRSA infections in addition to the irrational use of such valuable antibiotic.[7,17,18,22,26] Other factors as extensive surgical procedures, long hospital stay and misuse of antibiotics in clinical practice also contributed to the emergence VRSA.[7,19] Al-Mustafa *et al.*[35] reported that 29 antimicrobial agents were involved in poultry use in Saudi Arabia. About 22 (75.9%) of these antimicrobials were crucial for treating many human infections. The most frequently used drugs were doxycycline, enrofloxacin, sulfamethoxazole, oxytetracycline, ampicillin, neomycin, colistin and erythromycin. The authors warned against the uncontrolled use of these antimicrobial agents in food-producing animals and agriculture as such practice might result in life-threatening MDR infections in human.

Table 3: Multidrug resistance Pattern of reported VRSA isolates in Egypt and Saudi Arabia until December 2018.

Reference	VRSA Number	Reported antibiotic Resistance Pattern
[17]	1-2	Vancomycin, linezolid, ciprofloxacin, erythromycin
[19]	3-12	Vancomycin, amoxicillin\clavulanic acid, cefoxitin, cefazolin, oxacillin, tetracycline, cefixime
[20]	13-21	Vancomycin, streptomycin, benzylpenicillin, ampicillin, third generation cephalosporins, quinipristin/ dalfopristin
[7]	22	Vancomycin, penicillin, amoxicillin\clavulanic acid, ampicillin\sulbactam
[21]	23-52	Vancomycin (100.00%), Ampicillin (100.00%) Ampicillin-sulbactam (100.00%) Amoxicillin (100.00%) Amoxiclav (100.00%) Erythromycin (86.67%) Azithromycin (80.00%) Ciprofloxacin (80.00%) Gentamicin (70.00%) Levofloxacin (66.67%)
[22]	53-140	Vancomycin (100%), ampicillin (100%), cefotaxime (100%), ampicillin/sulbactam (88.8%), Sulphamethoxazole/trimethoprime (43.8%)
	141-195	Vancomycin (100%), ampicillin (100%), cefotaxime (100%), ampicillin/sulbactam (88.8%), Sulphamethoxazole/trimethoprime (43.8%)
[23]	196-199	Vancomycin, erythromycin, clindamycin, gentamycin, chloramphenicol, trimethoprim-sulfamethoxazole, rifampicin.
[25]	200-209	Vancomycin, tetracyclin, ciprofloxacin, erythromycin, penicillin, rifampin, levofloxacin, trimethoprim, clindamycin
[26]	210-220	Vancomycin (100%), ciprofloxacin (90.9%), erythromycin (81.8%), sulfamethoxazole-trimethoprim (63.6%), clindamycin (63.6%) and linezolid (63.6%).

CONCLUSION

To our knowledge, this is the first review of the literature highlighting the increased risk of VRSA strains in clinical settings in Egypt and Saudi Arabia. Many factors contribute to the lower prevalence of VRSA in Saudi Arabia than that in Egypt. The first factor is the use of more accurate diagnostic techniques depending on the CLSI guidelines with performing the definitive molecular techniques. The other factors are stringent infection control practices, appropriate antibiotic prescription in hospitals, agriculture and livestock, environmental hygiene and improved knowledge of the healthcare workers about VRSA in Saudi Arabia. Finally, treatment of VRSA should depend on the antibiotic susceptibility testing of isolates and considers linezolid as an alternative to vancomycin.

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CONFLICT OF INTEREST

Author declares that there is no conflict of interest associated with this study.

Authors' Contribution

Author has made substantial, direct and intellectual contribution to the work and approved it for publication.

Data Availability

All datasets generated or analyzed during this study are included in the manuscript.

Ethics Statement

This article does not contain any studies with human participants or animals performed by the author.

ABBREVIATIONS

CLSI: Clinical and Laboratory Standard Institute; **DD:** Disk diffusion; **MDR:** Multidrug resistant; **MIC:** Minimum inhibitory concentration; **MRSA:** Methicillin-resistant *Staphylococcus aureus*; **PCR:** Polymerase chain reaction; **SA:** *Staphylococcus aureus*; **VA:** Vancomycin; **VISA:** Vancomycin intermediate-resistant *Staphylococcus aureus*; **VRE:** Vancomycin resistant enterococci; **VRSA:** Vancomycin resistant *Staphylococcus aureus*.

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