Vancomycin Resistant *Staphylococcus aureus* Reported from Tertiary Care Hospital in Nepal

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ABSTRACT

Objectives: The study was conducted to assess the rate of Methicillin-resistant *Staphylococcus aureus* (MRSA) among patients and healthcare personnel at Mannohan Memorial College and Teaching Hospital, Kathmandu, Nepal and to evaluate the minimum inhibitory concentration of Vancomycin to MRSA isolates.

Methods: A total of 1433 different clinical specimens from patients and 33 nasal swabs from healthcare personnel were subjected to bacteriological investigation following standard protocol. *S. aureus* were isolated and identified by using standard Microbiological tools. Those isolates were subjected to Antimicrobial susceptibility testing using modified Kirby-Bauer’s disc diffusion method following CLSI guidelines.

Results: The rate of *S. aureus* carriage was found to be 65 (18.9%) in the samples from clinical patients and 24 (72.7%) in the samples from healthcare personnel. The rate of MRSA was found to be 57(85.1%) in patients and 24 (100%) in healthcare personnel. The high distribution of MRSA was found in female of age group 21-30 years (patients: 10.4%; healthcare personnel: 70.8%). Amikacin was found to be most effective antimicrobial. All *S. aureus* isolates were found to be multidrug resistant (100%). On performing D-test, 10 (17.5%) and 22 (38.6%) of MRSA from clinical specimens showed inducible and constitutive Clindamycin resistance respectively. Whereas, 11 (45.8%) and 4 (16.7%) of MRSA from nasal swabs were found to be inducible and constitutive Clindamycin resistance respectively. Upon performing minimum inhibitory concentration (MIC) test for clinical isolates, 3.5% (2) of MRSA were found to be Vancomycin resistant (VRSA), 54.4% (31) were Vancomycin intermediate (VISA) and 42.1% (24) were found to be Vancomycin sensitive (VSSA). All of the nasal swab MRSA isolates were found sensitive to Vancomycin. Congo red agar method was done for biofilm production. For clinical isolates, 32 (47.8%) were found to be strong, 6 (8.9%) moderate and 29 (43.3%) were non biofilm producer. For nasal swab isolates, 66.7% (16) and 33.3% (8) were found as strong and non-biofilm producer respectively.

Conclusion: This study reported the case of VRSA which hasn’t been reported in Nepal. Though present study showed that Vancomycin remains the main choice of treatment of MRSA infection. Therefore, to preserve its value, use of vancomycin should be limited only to those cases where there are clearly needed.

Key words: *S. aureus*, MRSA, D-test, Inducible Clindamycin resistance, VRSA

INTRODUCTION

*Staphylococcus aureus* is one of the common human pathogens capable of causing a wide range of infections. A great deal of virulence from the organism occurs through cross infection by patient to patient in hospitals and other institutional settings. In contrast, healthy individuals have a small risk of invasive infection caused by *S. aureus*, but they can
be carriers of the organism (Foster 2004). Infection
due to Methicillin-Resistant S. aureus (MRSA) are an
increasing problem worldwide in community as well
as hospital environment (Boyce et al. 2005; Skoy et
al. 2006). The incidence of community-acquired and
hospital-acquired S. aureus infections has been rising
with increasing emergence of drug-resistant strains
called Methicillin resistant S. aureus (MRSA) (Steinberg
et al. 1996).

The resistance of S. aureus to Methicillin is caused by
the meca gene which codes the low affinity
78-Kda penicillin-binding protein (PBP2a). Beta-
lactam antibiotic normally binds to PBPs in the cell
wall, resulting in the disruption of synthesis of the
peptidoglycan layer and death of bacterium. Since
the beta-lactam antibiotics cannot bind to low affinity
PBP2a, synthesis of peptidoglycan layer and cell wall are
able to continue (Duerenberg 2007). MRSA infections
often require systematic antibiotic therapy. The
spread of MRSA can also be potentially minimized by
prevention of the risk factors such as previous antibiotic
use, contact with the healthcare workers or nursing
home resident, daycare attendance, hospitalization,
admission to an intensive care unit, intravenous drug
use, invasive indwelling devices, haemodialysis or
peritoneal dialysis, immunosuppression, chronic
illness, and previous isolation of MRSA (Cohen 2007).

Following the spread of MRSA, glycopeptides (usually
Vancomycin and more recently Teicoplanin) have
become the mainstay of treatment for MRSA infections
(CDC 2013). Vancomycin is the choice of drug for MRSA
isolates. Patients unable to tolerate vancomycin have
been treated with fluoroquinolones, Trimethoprim-
Sulfomethoxazole, Clindamycin or Minocycline (Shah
2008). As Vancomycin is commonly used for the
treatment of MRSA infections, which has resulted into
development of Vancomycin-Intermediate S. aureus
(VISA) and Vancomycin-Resistant S. aureus (VRSA).

Clindamycin, a lincosamide drug, has been used to
treat serious infections caused by susceptible S. aureus
in children for more than 30 years (Woods 2009). It is
also an alternative choice in case of Penicillin allergic
patients. Clindamycin is recommended in some
European countries for suppression of panton-valentine
leukocidin (PVL) toxin, along with Linezolid and
Rifampin (Adaleti et al. 2010). In vitro, S. aureus isolates
with constitutive resistant are resistant to Erythromycin
and Clindamycin while isolates with inducible resistant
are resistant to Erythromycin but appear susceptible
to Clindamycin (Steward et al. 2005). Inducible
MLSB (Macrolide, Lincosamide and Streptogramin B)
resistant can be detected by disapproxiation test (D-
test) by placing Erythromycin and Clindamycin discs
in adjacent positions (Fiebelkorn et al. 2003).

Biofilms are communities of microorganisms embedded in
extracellular polymeric substances (EPS) matrix. Bacteria
in biofilms demonstrate distinct features from their free-
living planktonic counterparts, such as different physiology
and high resistance to immune system and antibiotics that
render biofilm a source of chronic and persistent infections.
Extracellular polymeric matrix plays various roles in
structure and function of different biofilm communities.
Adhesion to the surface provides considerable advantages
such as protection against antimicrobial agent, acquisition of
new genetic traits and the nutrients availability and metabolic
co-operability. Bacterial biofilms cause chronic infections
because they show increased tolerance to antibiotics and
disinfectant chemicals as well as resisting phagocytosis and
other components of the body’s defense system (Donlan and
Costerton 2002).

The study was thus done to determine the rate of
Methicillin resistant, emergence of Vancomycin
resistant and inducible Clindamycin resistant S. aureus
among biofilm producing and non-producing isolates
of S. aureus.

MATERIALS AND METHODS

Study design: A hospital based cross sectional
descriptive study was conducted.

Study period, site and population: The study was
conducted at Mannoham Memorial Medical College
and Teaching Hospital, Swoyammbhu, Kathmandu,
Nepal in collaboration with Kantipur College of Medical
Science, Sitapaila, Kathmandu, Nepal from April 2014 to
October 2014. All the clinical specimens obtained from
individuals of all ages and sexes visiting hospital during
the study period were included in the study.

Sample size: A total of 1344 clinical specimens
including blood, urine, sputum, vaginal swab, eye
swab, ear swab, throat swab, wound swab, clavical
swab, body fluids like pus, synoval fluid, pleural fluid,
asiatric fluid, peritoneal fluid and catheter swabs and
urethral discharge; were processed in the study. For
the study of hospital acquired MRSA, a total of 33 nasal
swabs were collected from the hospital personnel.
Laboratory diagnosis

Sample collection: Sterilized sample collection container was used for the collection of all clinical specimens. Blood was collected with sterile syringe and then poured in leak proof, dry and sterilized container.

Sample processing and bacterial identification: All the clinical samples were inoculated onto blood agar, chocolate agar and mac-conkey agar plates. Blood samples were inoculated into brain heart infusion broth and incubated at 37°C for 7 days and then further inoculated into agar media. All the culture plates were then incubated at 37°C for overnight. The plates showing growth of bacterial were processed for identification of S. aureus using standard microbiological procedures by inoculating the organism on mannitol salt agar and performing specific biochemical tests catalase test, coagulase test and oxidative-fermentative test.

Antimicrobial susceptibility testing and screening of multi drug resistant (MDR) S. aureus: All the identified isolates of S. aureus were undertaken in-vitro antibiotic susceptibility test by using modified Kirby-Bauer’s disc diffusion method (CLSI 2013). The antibiotics used were Cefoxitin (5mcg), Ciprofloxacin (5mcg), Cefixime (5mcg), Tetracycline (30mcg), Amikacin (30mcg), Azithromycin (30mcg), Vancomycin (30mcg), Cloxacillin (5mcg), Cefotaxime (30mcg), Clindamycin (10mcg), Ceftriaxone (30mcg), Erythromycin (15mcg), Gentamicin (10mcg), Penicillin (10mcg), Co-trimoxazole (25mcg), Mupirocin (5mcg), and Chloramphenicol (50mcg). The organism resistant to three or more antibiotics of different classes were classified as MDR isolates (Magiorakos et al. 2012). Intrinsic resistance to any of the employed antibiotics was not counted.

Screening of methicillin resistant S. aureus: Screening for Methicillin resistant S. aureus was carried out by Cefoxitin disc diffusion method and interpreted according to CLSI (2013) guidelines. The growth of S. aureus with zone of inhibition around Cefoxin disc (ZOI) ≥ 22mm were identified as Methicillin sensitive S. aureus and that of ZOI ≤ 21 were identified as Methicillin resistant S. aureus.

Detection of Inducible clindamycin resistance (ICR): In this assay, two discs namely Erythromycin and Clindamycin were placed 18mm away edge-to-edge on Muller Hinton agar plates that were previously inoculated with 0.5 McFarland bacterial suspensions. Plates were observed after 18 hours of incubation at 35±2°C. Flattening of the zone of inhibition adjacent to the Erythromycin disc (referred to as D-zone) or hazy growth within the zone of inhibition around Clindamycin (even if no D- zone is apparent) is regarded as positive test, i.e. Inducible Clindamycin resistance (CLSI 2013).

Determination of minimum inhibitory concentration of vancomycin: Minimum inhibitory concentration (MIC) technique was performed to determine the Vancomycin intermediate and resistant strains of S. aureus isolates MIC to Vancomycin in isolated MRSA was done by agar dilution method following CLSI guidelines (CLSI 2013). Different concentrations ranging from 0.06-32µg/ml of Vancomycin incorporated plates was prepared. Positive growth controls were kept for each isolates and S. aureus (ATCC 25923) of known MIC was also included in each test as control for antibiotic potency.

Biofilm production: Biofilm detection was carried out by Congo Red Agar method (CRA): CRA medium was prepared with brain heart infusion broth 37 g/L, sucrose 50 g/L, agar 10 g/L and Congo Red indicator 8 g/L. Congo Red stain was prepared as a concentrated aqueous solution and autoclaved separately from the other medium constituents. Then it was added to the autoclaved brain heart infusion agar with sucrose. CRA plates was inoculated with test organisms and incubate at 37°C for overnight aerobically. Black colonies were considered as biofilm producing isolates (Freeman et al. 1989).

RESULTS
Out of total 1433 clinical specimens, S. aureus was isolated from 67 specimens (4.7%), among them 57 (85.1%) were found to be MRSA. Whereas from 33 nasal swab specimens, 24 (72.7%) S. aureus were isolated and all of them were found to be MRSA (100%). All of the S. aureus isolates from clinical as well as nasal swab specimens were multi-drug resistant (MDR) (Figure 1).
Among total of 67 *S. aureus* isolates, 27 (40.3%) from male and 30 (44.8%) from female. High rate of MRSA was 12 (17.9%) from age group 21-30 year (Table 1).

**Table 1: Age and sex wise distribution of MRSA from clinical specimens**

<table>
<thead>
<tr>
<th>Age group (year)</th>
<th>Total No. of samples</th>
<th>Male Number (%)</th>
<th>Female Number (%)</th>
<th>Total no. of MRSA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Samples</td>
<td><em>S. aureus</em></td>
<td>MRSA</td>
<td>Samples</td>
</tr>
<tr>
<td>&lt;10</td>
<td>109</td>
<td>56</td>
<td>3(4.5)</td>
<td>2(2.9)</td>
</tr>
<tr>
<td>11-20</td>
<td>204</td>
<td>99</td>
<td>7(10.4)</td>
<td>4(5.9)</td>
</tr>
<tr>
<td>21-30</td>
<td>382</td>
<td>121</td>
<td>6(9)</td>
<td>5(7.5)</td>
</tr>
<tr>
<td>31-40</td>
<td>202</td>
<td>86</td>
<td>3(4.5)</td>
<td>2(2.9)</td>
</tr>
<tr>
<td>41-50</td>
<td>124</td>
<td>65</td>
<td>6(9)</td>
<td>6(9)</td>
</tr>
<tr>
<td>51-60</td>
<td>128</td>
<td>40</td>
<td>1(1.5)</td>
<td>1(1.5)</td>
</tr>
<tr>
<td>61-70</td>
<td>108</td>
<td>55</td>
<td>3(4.5)</td>
<td>3(4.5)</td>
</tr>
<tr>
<td>71-80</td>
<td>87</td>
<td>37</td>
<td>2(2.9)</td>
<td>2(2.9)</td>
</tr>
<tr>
<td>&gt;80</td>
<td>89</td>
<td>51</td>
<td>2(2.9)</td>
<td>2(2.9)</td>
</tr>
<tr>
<td>Total</td>
<td>1433</td>
<td>610</td>
<td>33(49.2)</td>
<td>27(40.3)</td>
</tr>
</tbody>
</table>

Among 24 nasal swab MRSA isolates, 2 (8.3%) were from male and 22 (91.7%) were from female. High rate of MRSA was 19 (79.2%) obtained from age group 21-30 year (Table 2).

**Table 2: Age and sex wise distribution of MRSA from nasal swabs**

<table>
<thead>
<tr>
<th>Age group (year)</th>
<th>Total no. of samples</th>
<th>Male Number (%)</th>
<th>Female Number (%)</th>
<th>Total no. of MRSA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Samples</td>
<td><em>S. aureus</em></td>
<td>MRSA</td>
<td>Samples</td>
</tr>
<tr>
<td>&lt;10</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11-20</td>
<td>4</td>
<td>1</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>21-30</td>
<td>22</td>
<td>3</td>
<td>2(8.3)</td>
<td>2(8.3)</td>
</tr>
<tr>
<td>31-40</td>
<td>7</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>4</td>
<td>2</td>
<td>8.3</td>
</tr>
</tbody>
</table>
MRSA from clinical specimens were 100% resistant to Penicillin G, Co-trimoxazole and Cefixime, followed by Cloxacillin (94.7%), Ceftriaxone (93%), Vancomycin (92.2%), and Cefotaxime (84.2%). Whereas MSSA isolates were 100% resistant to Cefixime, followed by Cefotaxime (90%), Ceftriaxone (90%), Penicillin G (80%) and Co-trimoxazole (80%). For nasal swab MRSA isolates, 100% showed resistance towards Cefixime, Cefotaxime, Penicillin G and Co-trimoxazole, followed by Cloxacillin (75%), Clindamycin (54.2%), and Tetracycline (50%). Whereas no MSSA isolates were obtained (Table 3).

<table>
<thead>
<tr>
<th>Antibiotics</th>
<th>Clinical specimens</th>
<th>Nasal swab specimens</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Antibiotic profile</td>
<td>Antibiotic profile</td>
</tr>
<tr>
<td></td>
<td>of MRSA (n=57)</td>
<td>of MSSA (n=10)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antibiotic profile</td>
</tr>
<tr>
<td></td>
<td></td>
<td>of MRSA (n=24)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>profile of MSSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antibiotic (n=0)</td>
</tr>
<tr>
<td>Cefoxitin</td>
<td>57(100%)</td>
<td>-</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>15(26.3%)</td>
<td>3(30%)</td>
</tr>
<tr>
<td>Cefixime</td>
<td>57(100%)</td>
<td>10(100%)</td>
</tr>
<tr>
<td>Tetracyclin</td>
<td>32(56.1%)</td>
<td>-</td>
</tr>
<tr>
<td>Amikacin</td>
<td>8(14%)</td>
<td>-</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>25(43.9%)</td>
<td>1(10%)</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>53(92.9%)</td>
<td>4(40%)</td>
</tr>
<tr>
<td>Cloxacillin</td>
<td>54(94.7%)</td>
<td>1(10%)</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>48(84.2%)</td>
<td>9(90%)</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>42(73.7%)</td>
<td>4(40%)</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>53(93%)</td>
<td>9(90%)</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>32(56.1%)</td>
<td>-</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>13(22.8%)</td>
<td>-</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>57(100%)</td>
<td>8(80%)</td>
</tr>
<tr>
<td>Co-trimoxazole</td>
<td>57(100%)</td>
<td>8(80%)</td>
</tr>
<tr>
<td>Mupirocin</td>
<td>43(75.4%)</td>
<td>3(30%)</td>
</tr>
<tr>
<td>Cloramphenicol</td>
<td>11(19.3%)</td>
<td>-</td>
</tr>
</tbody>
</table>

Upon performing D-test, 10 (17.5%) and 22 (38.6%) of MRSA from clinical specimens showed inducible and constitutive Clindamycin resistance respectively. Whereas, 11 (45.8%) and 4 (16.7%) of MRSA from nasal swabs were found to be inducible and constitutive Clindamycin resistance respectively (Table 4).

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Erythromycin</th>
<th>Clindamycin</th>
<th>D-test</th>
<th>MRSA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>iMLSB</td>
<td>Resistance</td>
<td>Sensitive</td>
<td>Positive</td>
<td>10 (17.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>11 (45.8)</td>
</tr>
<tr>
<td>cMLSB</td>
<td>Resistance</td>
<td>Resistance</td>
<td>Negative</td>
<td>22 (38.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4 (16.7)</td>
</tr>
</tbody>
</table>

From clinical specimens, 47.8% (32), 8.9% (6) and 43.3% (29) of S. aureus isolates were found to be strong, moderate and non-biofilm producer respectively. Among nasal swab specimens, 66.7% (16) and 33.3% (8) of S. aureus isolates were found to be strong and non-biofilm producer respectively (Table 5).

<table>
<thead>
<tr>
<th>Biofilm production by S. aureus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serial no.</td>
</tr>
<tr>
<td>1. clinical specimen (n=67)</td>
</tr>
<tr>
<td>2. Nasal swab (n=24)</td>
</tr>
</tbody>
</table>

Table 3: Antimicrobial resistance profile of MRSA and MSSA

Table 4: D-test of MRSA isolates

Table 5: Biofilm production by S. aureus
On performing MIC of MRSA from clinical specimens, 42.1% (24) of isolates were reported as VSSA (showed MIC value of 2 $\mu$g/ml), 54.4% (31) as VISA (showed MIC value of 4-8 $\mu$g/ml) and 3.5% (2) of MRSA isolates were reported as VRSA (showed MIC value of $\geq 16$ $\mu$g/ml) (Figure 2). Whereas all MRSA isolates from nasal swabs were found to be Vancomycin sensitive (VSSA).

**DISCUSSION**

*S. aureus* has remained a versatile and potent pathogen in humans, since it is one of the most common causes of nosocomial and community acquired infections (Rajbhandari et al. 2003). *S. aureus* is a major cause of infectious morbidity and mortality around the world, causing a wide variety of clinical manifestations ranging from localized infection to toxin mediated diseases and invasive blood stream infections (Vandecasteele et al. 2008).

In this study, the rate of MRSA isolation was found to be 85.1% from clinical specimens. This result is higher than the many other studies conducted by Kumari et al. (26.14%), Shakya et al. (12.5%) and Tiwari et al. (69.1%) (Tiwari et al. 2006; Kumari et al. 2008; Shakya et al. 2010). All of the *S. aureus* isolates from nasal swab specimens were found to be MRSA i.e. 100%. Nasal carriage rate of MRSA among health care workers in hospital setting ranges from 6-17.8% (Cesur and Cokca 2004; Pant and Rai 2007). Nasal carriage rate of 43.8% has been reported among the healthcare personnel of a Medical College Teaching Hospital in Kathmandu (Pant and Rai 2007). The nasal carriage rate of *S. aureus* in this study i.e. 72.7% was found to be greater than the study conducted by Shakya et al. i.e. 12.5% (Shakya et al. 2010). The result is also in agreement with the study by Gonsu et al. (Gonsu et al. 2013).

Regarding the sex wise distribution of MRSA clinical specimens, the study showed high incidence of MRSA from female patients (44.8%) than males (40.3%). The present study showed the opposite variation with the study conducted by Boucher and Corey (Boucher and Corey 2008) showing males (64.4%) were more predisposed than females (35.6%). The highest distribution of MRSA was found within the age group of 21-30 years (17.9%) and the lowest in the age group below 10 years (4.5%). However, the study conducted by Arch et al. (Arch et al. 2006) and Lucet et al. (Lucet et al. 2003) showed high rate of MRSA colonization among the population with age group 60 years and above.

The nasal carriage rate of MRSA reported in present study was found to be higher (72.7%) than the previous studies conducted in Nepal by Shakya et al. and Rijal et al. (Rijal et al. 2008; Shakya et al. 2010). Penicillin was found resistant to all of MRSA isolates.
i.e. 100%. This result is higher than that of Shrestha et al. who reported 91.94% (Shrestha et al. 2009). In present study, clinical MRSA isolates showed rate of resistance to antibiotics Co-trimoxazole (100%), followed by Cloxacillin (94.7%), Ceftriazone (93%), Vancomycin (92.9%), Cefotaxime (94.2%), Mupirocin (75.4%), Clindamycin (73.7%), Tetracycline (56.1%) and Erythromycin (56.1%). Rijal et al. reported the rate of resistance to Cloxacillin (68.8%), followed by Tetracycline (15.6%) and Erythromycin (9.4%) (Rijal et al. 2008). Resistance to Erythromycin is seen to be greater than the finding disseminated by study conducted by Mishra i.e. 14.29% (Mishra 2008) and lower than the finding disseminated by Tiwari et al. i.e. 68.7% (Tiwari et al. 2006).

All isolates were found to be multi drug resistant (MDR) in this study. The rate of MDR-MRSA (100%) is higher than that of the result reported in the studies conducted by Tiwari et al. i.e. 40.1% and Pandey et al. i.e. 75.86%. Though this study is in accordance with the previous studies from Nepal and other countries showing high percentage of MDR among MRSA; 65% by Kumari et al., 93% by Rahimi et al. and 63% by Salah et al. (Kumari et al. 2008; Salah et al. 2012; Rahimi et al. 2013).

In this study, 17.5% and 38.6% isolates were found to be inducible and constitutive Clindamycin resistance respectively. Among nasal swab specimens taken from hospital staffs, 45.5% and 16.7% were found to be inducible and constitutive Clindamycin resistance respectively. In the study conducted by Ujwol et al. (Bhomi et al. 2016), D-test positive isolates were found to be 18.03% and study also reported constitutive resistance in 36.06% of isolates.

Upon performing MIC, 3.5% (Skoy et al. 2006)) of clinical MRSA isolates were reported as VRSA, 54.4% (31) as VISA and 42.1% (24) were as VISA and VSSA respectively. Whereas all nasal swab isolates were sensitive to Vancomycin. From this study, it could be concluded that all of the MRSA isolates i.e. 100% were multi drug resistant (MDR), which is the significant public health problem in context of Nepal, indicating the high risk of staphylococcal infections in our context. This high load of MDR organisms provokes the necessity of strictly performing susceptibility testing before starting antibiotic therapy, or there may be chance of clinical failure. Thus determination of MIC of Vancomycin is crucial. Inducible Clindamycin resistance test cannot be observed in routinely done antibiotic susceptibility testing by Kirby Bauer method hence specific D-test should be performed before treatment with Clindamycin. There are various methods for detection of biofilm production and both tube test and agar plate methods can be carried out for comparative study.

CONCLUSION

Higher rate of MRSA was found in female in age group 21-30 years. Amikacin was found to be most effective drug, whereas penicillin G was found as the least effective drug. Upon performing MIC test of MRSA isolates from clinical specimens, 3.5% (2) of MRSA isolates were found as VRSA and 54.4% (31) and 42.1% (24) were as VISA and VSSA respectively. Whereas all nasal swab isolates were sensitive to Vancomycin. From this study, it could be concluded that all of the MRSA isolates i.e. 100% were multi drug resistant (MDR), which is the significant public health problem in context of Nepal, indicating the high risk of staphylococcal infections in our context. This high load of MDR organisms provokes the necessity of strictly performing susceptibility testing before starting antibiotic therapy, or there may be chance of clinical failure. Thus determination of MIC of Vancomycin is crucial. Inducible Clindamycin resistance test cannot be observed in routinely done antibiotic susceptibility testing by Kirby Bauer method hence specific D-test should be performed before treatment with Clindamycin. There are various methods for detection of biofilm production and both tube test and agar plate methods can be carried out for comparative study.

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