Original article

Association of tsst-1 gene and phenotypic antibiotic resistance among clinical Staphylococcus aureus isolates in a tertiary healthcare center

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ABSTRACT

Background: The toxic shock syndrome toxin (TSST-1) is important in the pathology of toxic shock syndrome. However, little data exist on its prevalence among clinical isolates of S. aureus in Nigeria. Hence, this study was carried out to detect the tsst-1 gene and associate it with phenotypic antibiotic resistance in clinical isolates of S. aureus.

Methods: Staphylococcus aureus isolates were presumptively identified by Gram’s staining and conventional biochemical tests while confirmatory identification was through the detection of the thermonuclease (nuc) gene. Antibiotic sensitivity testing was carried out using the modified Kirby-Bauer disc diffusion method while phenotypic detection of methicillin resistance was carried out using the cefoxitin disc sensitivity assay. The tst gene was detected within the genome of the bacterial isolates using Uniplex polymerase chain reaction (PCR).

Results: Of the 152 S. aureus isolates identified in this study, 103 (67.76%) encoded the tst gene. Of these 103 tst-positive isolates, 63 (61.16%) were methicillin-resistant while 40 (38.84%) were methicillin-sensitive. The tst-positive isolates (n=103) were resistant to tetracycline (39.81%), erythromycin (24.27%), gentamicin (22.33%), ciprofloxacin (22.33%), fusidic acid (16.5%), fosfomycin (10.68%), and clindamycin (8.16%). Comparatively, tst-negative isolates (n=49) were resistant to tetracycline (69.39%), cotrimoxazole (56.06%), gentamicin (53.06%), ciprofloxacin (51.02%), erythromycin (46.94%), fusidic acid (28.57%), fosfomycin (26.53%), and clindamycin (8.16%). Phenotypic antibiotic resistance is significantly associated with the presence of the tst gene (p<0.05) except for clindamycin and fusidic acid (p>0.05).

Conclusion: Hence, the high prevalence of the tst gene and its association with antibiotic resistance in S. aureus is a cause for worry.

Introduction

Staphylococcus aureus (S. aureus) is a normal flora of the skin and nasal cavity of healthy immunocompetent individuals [1]. However, the organism causes diseases of immense clinical significance due to its vast array of virulence factors, which includes superantigen toxins [2]. Staphylococcus aureus is a clinically significant pathogen whose diseases range from mild superficial infections to severe and life-threatening systemic infections [3,4]. In tropical countries, S. aureus remains a significant cause of morbidities and mortalities in clinical settings.
Pathogenic organisms possess genetic factors that make up their virulence arsenal and enhance their fitness in proliferating host cells and tissues to cause diseases [5]. These virulence factors grade the pathogenicity of microbial pathogens and play important roles in determining the pathology of the vast arrays of microbial diseases. These virulence factors are genetically encoded and translated into proteins under inducible conditions. Toxic shock syndrome toxin (TSST-1) is a virulence factor produced by lysogenic converted strains of *S. aureus* [6].

Toxic shock syndrome toxin induces excessive T cell proliferation and cytokines release by inducing the cross-linking of class II major histocompatibility complex (MHC) molecules on antigen-presenting cells (APCs) with the variable region of the β-chain of T cell receptors (TCRs) on naive T lymphocytes [7,8]. This excessive T cell proliferation and cytokine release induce an acute phase of host immune excitation that culminates in a cytokine storm which cripples the host’s physiology and leads to death in extreme cases [9]. The TSST-1 is associated with toxic shock syndrome (TSS) and produced by lysogenic converted *S. aureus* strains encoding the *tst* gene, a gene carried on *S. aureus* pathogenicity islands (SaPIs) [6,10].

Toxic shock syndrome is a highly fatal multisystemic disease. It is mostly associated with the usage of tampons by menstruating women (menstrual TSS), although non-menstrual TSS has also been elucidated [11,12]. It is characterized by pathological hallmarks such as rapid fever onset, erythematous skin rash, hypotension, hemodynamic shock, multiorgan failure, and ultimately death [1,7].

Despite the enormous clinical significance of TSST-1 as a virulence factor, there is inadequate available data on its prevalence among clinical isolates of *S. aureus* in Nigeria and Africa at large. Furthermore, little research has sought to associate phenotypic antibiotic resistance with superantigen toxin genes such as tst. Hence, this study was carried out to associate phenotypic antibiotic resistance with the possession of the tst gene among clinical isolates of *S. aureus*.

**Methods**

**Study setting and ethical approval**

University of Ilorin Teaching Hospital (UITH) is a tertiary healthcare center located at Oke-Oyi, Ilorin East, Kwara State, Nigeria. The services of the hospital are rendered to patients from several states including, but not limited to, Kwara, Oyo, Ekiti, Osun, Lagos, Kogi, Niger, Kebbi, and the Federal Capital Territory (FCT) [13]. Ethical approval for the study was obtained from the Ethical Review Board (ERB) of the UITH.

**Study design**

The study adopted a laboratory-based cross-sectional design that used clinical isolates of *S. aureus* recovered from clinical specimens submitted to the Department of Medical Microbiology and Parasitology of UITH.

**Culture, isolation, and identification of *S. aureus***

Clinical specimens, including wound specimens, aspirates, eye swabs, and abscesses were inoculated directly on sheep blood agar (Oxoid, UK) and MacConkey agar (Oxoid, UK) plates. Bact/Alert-positive blood specimens were cultured on sheep blood, chocolate, and MacConkey agar plates. Inoculated plates were incubated aerobically while chocolate agar plates were incubated in a microaerophilic environment in a candle extinction jar. All culture plates were incubated at 37°C for 18-24 hours. Isolates on culture plates were identified morphologically by Gram's stain reaction and standard biochemical tests that included catalase, coagulase, DNase, and mannitol fermentation tests (Oxoid, UK). Isolates that were Gram-positive cocci in clusters, catalase-positive, coagulase-positive, DNase-positive, and mannitol-fermenters were identified as *S. aureus* [14].

**Antibiotic sensitivity test (AST) of *S. aureus***

Antibiotic sensitivity testing was carried out on each *S. aureus* isolate using the modified Kirby-Bauer disc diffusion method. Bacterial inoculum was standardized to 0.5 McFarland standard before inoculating the surface of freshly prepared Mueller-Hinton agar (MHA) plates. The isolates were tested against the following antibiotics (Oxoid, UK); erythromycin (15µg), clindamycin (2µg), tetracycline (30µg), cotrimoxazole (1.25/23.75µg), mupirocin (5µg), linezolid (30µg), tigecycline (15µg), fusidic acid (10µg), fosfomycin (50µg), ciprofloxacin (5 µg), rifampin (5µg), and gentamicin (10µg). *S. aureus* ATCC 25923 was used as a control strain. Diameters of the zone of inhibition were measured with a calibrated ruler and interpretation of each isolate as sensitive, intermediate, or resistant to the antibiotics was done
were classified as methicillin-resistant (MRSA) and methicillin-sensitive (MSSA) [15].

Phenotypic detection of methicillin resistance was carried out using a cefoxitin (30µg) disc diffusion test. Diameters of inhibition of the zone of inhibition ≤ 21 mm were classified as methicillin-resistant (MRSA) and those with a diameter ≥ 22 mm as methicillin-sensitive (MSSA) [15].

**Molecular detection of tst gene**

To detect the presence of tst (306bp) and nuc (279bp) genes among S. aureus isolates, DNA extraction was carried out using the conventional boiling method with proteinase K (Inqaba Biotec, Ibadan, Nigeria). The thermonuclease (nuc) gene is a target nucleotide sequence that is used to selectively identify and differentiate S. aureus from other Staphylococcus spp [16]. Uniplex PCR was carried out using the primers tst-F: AGCCCTGCTTTTCAAAAAGGGAAAAA and tst-R: CCAATAACCACCCGTTTATCGCTTG and nuc-F: GCGATTGATGGTGATACGGTT and nuc-R: AGCCAGCCTTGAACAGAACTTAAAGC (Inqaba Biotec, South Africa) and 5X master mix (New England Biolabs Ltd, UK). The PCR conditions implemented for the amplification of the tst gene included, DNA template denaturation at 94°C (60 seconds), annealing at 60°C (60 seconds), and extension at 72°C (2 minutes) [17]. The PCR amplification of the nuc gene was carried out at 94°C for 60 seconds (denaturation), 56°C for 2 minutes (annealing), and 72°C for 2 minutes (extension) [18]. PCR products were thereafter run on gel electrophoresis using 1.2% agarose gel using 100 bp uncut DNA ladder as the standard.

**Statistical analysis**

Statistical analyses were computed using IBM SPSS (version 21). Fisher exact test (with Odds ratio and 95% CI) was used to determine the association between methicillin resistance and possession of the tst gene as well as between possession of the tst gene and antibiotic resistance. p-values were computed at a 95% confidence interval to determine the significance of difference. Computed p-values less than 0.05 (p < 0.05) were considered to be statistically significant.

**Results**

A total of 152 clinical isolates of S. aureus were recovered from clinical specimens. Of these, 103 (67.76%) possessed the tst gene. Sixty-three (61.16%) of these tst-positive S. aureus were methicillin-resistant (MRSA) while 40 (38.84%) were methicillin-sensitive (MSSA). The possession of the tst gene was higher among MRSA strains than among MSSA isolates (Table 1). The prevalence of the tst gene in different clinical infections is shown in table (2). Staphylococcus aureus harboring the tst gene was more prevalent in sepsis (29.13%), diabetic foot syndrome (21.36%), and eye infection (10.68%). Conversely, tst-negative S. aureus was more prevalent in pyomyositis (26.54%), sepsis (18.37%), and otitis media (12.25%). Furthermore, pyomyositis was more associated with tst-negative strains of S. aureus (p < 0.05) while diabetic foot syndrome was more associated with S. aureus strains harboring the tst gene (p < 0.05). For other clinical infections, there was no significant association with the presence of the tst gene (p > 0.05). All S. aureus isolates were sensitive to linezolid, tigecycline, mupirocin, and rifampin. The tst-positive MRSA isolates were more resistant to tetracycline (47.62%), erythromycin (31.75%), and cotrimoxazole (26.98%) while tst-positive MSSA isolates were more resistant to tetracycline (27.5%), gentamicin (20%), ciprofloxacin (17.5%), and cotrimoxazole (15%). There was no association between methicillin sensitivity among tst-positive S. aureus and antibiotic resistance except for tetracycline and erythromycin (Table 3). Staphylococcus aureus isolates encoding the tst gene were more resistant to tetracycline (39.81%), erythromycin (24.27%), and fusidic acid and gentamicin (22.33% each) while tst-negative S. aureus isolates were more resistant to tetracycline (69.39%), cotrimoxazole and gentamicin (53.06% each), and ciprofloxacin (51.02%). However, S. aureus isolates harboring the tst gene were significantly more resistant to erythromycin, tetracycline, cotrimoxazole, fosfomycin, ciprofloxacin, and gentamicin than tst-negative strains. Hence, a positive association between antibiotic resistance and possession of the tst gene was observed against all tested antibiotics, except clindamycin and fusidic acid (Table 4).
Table 1. Prevalence of \textit{tst} gene among \textit{S. aureus} strains.

<table>
<thead>
<tr>
<th>\textit{S. aureus} strain</th>
<th>\textit{tst} positive \textit{n} (%)</th>
<th>\textit{tst} negative \textit{n} (%)</th>
<th>\textit{Total} \textit{n} (%)</th>
<th>\textit{OR}</th>
<th>95% CI</th>
<th>\textit{p}-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRSA</td>
<td>63 (61.16)</td>
<td>7 (14.28)</td>
<td>70 (46.05)</td>
<td>9.45</td>
<td>3.87-23.08</td>
<td>&lt;0.0001*</td>
</tr>
<tr>
<td>MSSA</td>
<td>40 (38.84)</td>
<td>42 (85.72)</td>
<td>82 (53.95)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>103 (67.76)</td>
<td>49 (32.24)</td>
<td>152 (100.0)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

MRSA: methicillin-resistant \textit{Staphylococcus aureus}; MSSA: methicillin-sensitive \textit{Staphylococcus aureus}; \textit{tst}: toxic shock syndrome toxin gene; \textit{OR}: odds ratio; CI: confidence interval; \textit{n}: number of isolates; *: statistically significant.

Table 2. Prevalence of \textit{S. aureus} from clinical infections.

<table>
<thead>
<tr>
<th>Clinical infection</th>
<th>\textit{tst} positive \textit{n} (%) (\textit{n}=103)</th>
<th>\textit{tst} negative \textit{n} (%) (\textit{n}=49)</th>
<th>\textit{Total} \textit{n} (%) (\textit{n}=152)</th>
<th>\textit{OR}</th>
<th>95% CI</th>
<th>\textit{p}-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye infection</td>
<td>11 (10.68)</td>
<td>5 (10.20)</td>
<td>16 (10.53)</td>
<td>1.052</td>
<td>0.345-3.213</td>
<td>0.464430</td>
</tr>
<tr>
<td>Otitis media</td>
<td>8 (7.77)</td>
<td>6 (12.25)</td>
<td>14 (9.21)</td>
<td>0.604</td>
<td>0.197-1.846</td>
<td>0.188022</td>
</tr>
<tr>
<td>Wound sepsis</td>
<td>5 (4.85)</td>
<td>3 (6.12)</td>
<td>8 (5.26)</td>
<td>0.782</td>
<td>0.179-3.415</td>
<td>0.372012</td>
</tr>
<tr>
<td>Pyomyositis</td>
<td>10 (9.71)</td>
<td>13 (26.54)</td>
<td>23 (15.13)</td>
<td>0.298</td>
<td>0.12-0.74</td>
<td>0.004528*</td>
</tr>
<tr>
<td>Surgical site infection</td>
<td>8 (7.77)</td>
<td>5 (10.20)</td>
<td>13 (8.55)</td>
<td>0.741</td>
<td>0.229-2.395</td>
<td>0.308295</td>
</tr>
<tr>
<td>Mastitis</td>
<td>4 (3.88)</td>
<td>2 (4.08)</td>
<td>6 (3.95)</td>
<td>0.949</td>
<td>0.168-5.369</td>
<td>0.476624</td>
</tr>
<tr>
<td>Sepsis</td>
<td>30 (29.13)</td>
<td>9 (18.37)</td>
<td>39 (25.66)</td>
<td>1.826</td>
<td>0.789-4.226</td>
<td>0.079622</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>5 (4.85)</td>
<td>3 (6.12)</td>
<td>8 (5.26)</td>
<td>0.782</td>
<td>0.179-3.415</td>
<td>0.372012</td>
</tr>
<tr>
<td>Diabetic foot syndrome</td>
<td>22 (21.36)</td>
<td>3 (6.12)</td>
<td>25 (16.45)</td>
<td>4.164</td>
<td>1.182-14.673</td>
<td>0.013201*</td>
</tr>
</tbody>
</table>

\textit{tst}: toxic shock syndrome toxin gene; \textit{OR}: odds ratio; CI: confidence interval; \textit{n}: number of isolates; *: statistically significant.

Table 3. Analysis of antibiotic resistance among \textit{tst}-encoding \textit{S. aureus} strains.

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>MRSA (%) (\textit{n}=63)</th>
<th>MSSA (%) (\textit{n}=40)</th>
<th>Total (%) (\textit{n}=103)</th>
<th>\textit{OR}</th>
<th>95% CI</th>
<th>\textit{p}-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythromycin</td>
<td>20 (31.75)</td>
<td>5 (12.5)</td>
<td>25 (24.27)</td>
<td>3.2558</td>
<td>1.11-9.56</td>
<td>0.026394*</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>5 (7.94)</td>
<td>1 (2.50)</td>
<td>6 (5.82)</td>
<td>3.3621</td>
<td>0.38-29.89</td>
<td>0.25093</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>30 (47.62)</td>
<td>11 (27.50)</td>
<td>41 (39.81)</td>
<td>2.3967</td>
<td>1.02-5.62</td>
<td>0.042129*</td>
</tr>
<tr>
<td>Cotrimoxazole</td>
<td>17 (26.98)</td>
<td>6 (15.0)</td>
<td>23 (22.33)</td>
<td>2.0942</td>
<td>0.75-5.87</td>
<td>0.154221</td>
</tr>
<tr>
<td>Fusidic acid</td>
<td>13 (20.63)</td>
<td>4 (10.0)</td>
<td>17 (16.50)</td>
<td>2.34</td>
<td>0.71-7.767</td>
<td>0.156265</td>
</tr>
<tr>
<td>Fosfomycin</td>
<td>8 (12.70)</td>
<td>3 (7.50)</td>
<td>11 (10.68)</td>
<td>1.7939</td>
<td>0.45-7.208</td>
<td>0.40511</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>15 (23.81)</td>
<td>7 (17.5)</td>
<td>22 (21.36)</td>
<td>1.4732</td>
<td>0.54-4.007</td>
<td>0.446312</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>15 (23.81)</td>
<td>8 (20.0)</td>
<td>23 (22.33)</td>
<td>1.25</td>
<td>0.48-3.29</td>
<td>0.654721</td>
</tr>
</tbody>
</table>

MRSA: methicillin-resistant \textit{Staphylococcus aureus}; MSSA: methicillin-sensitive \textit{Staphylococcus aureus}; \textit{OR}: odds ratio; CI: confidence interval; \textit{n}: number of isolates; *: statistically significant.
Table 4. Analysis of antibiotic resistance among *tst*-positive and *tst*-negative *S. aureus*

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th><em>tst</em>-positive (%) (n = 103)</th>
<th><em>tst</em>-negative (%) (n = 49)</th>
<th>Total (%) (n = 152)</th>
<th>OR</th>
<th>95% CI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erythromycin</td>
<td>25 (24.27)</td>
<td>23 (46.94)</td>
<td>48 (31.58)</td>
<td>0.3623</td>
<td>0.176-0.744</td>
<td>0.004943*</td>
</tr>
<tr>
<td>Clindamycin</td>
<td>6 (5.82)</td>
<td>4 (8.16)</td>
<td>10 (6.58)</td>
<td>0.6959</td>
<td>0.187-2.588</td>
<td>0.586834</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>41 (39.81)</td>
<td>34 (69.39)</td>
<td>75 (49.34)</td>
<td>0.2917</td>
<td>0.141-0.602</td>
<td>0.000652*</td>
</tr>
<tr>
<td>Cotrimoxazole</td>
<td>23 (22.33)</td>
<td>26 (53.06)</td>
<td>49 (32.24)</td>
<td>0.2543</td>
<td>0.123-0.527</td>
<td>0.000152*</td>
</tr>
<tr>
<td>Fusidic acid</td>
<td>17 (16.50)</td>
<td>14 (28.57)</td>
<td>31 (20.39)</td>
<td>0.4942</td>
<td>0.22-1.11</td>
<td>0.084299</td>
</tr>
<tr>
<td>Fosfomycin</td>
<td>11 (10.68)</td>
<td>13 (26.53)</td>
<td>24 (15.79)</td>
<td>0.3311</td>
<td>0.136-0.807</td>
<td>0.01228*</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>22 (21.36)</td>
<td>25 (51.02)</td>
<td>47 (30.92)</td>
<td>0.2607</td>
<td>0.125-0.542</td>
<td>0.000217*</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>23 (22.33)</td>
<td>26 (53.06)</td>
<td>49 (32.24)</td>
<td>0.2543</td>
<td>0.123-0.527</td>
<td>0.000152*</td>
</tr>
</tbody>
</table>

*tst*: toxic shock syndrome toxin gene; OR: odds ratio; CI: confidence interval; n: number of isolates; *: statistically significant.

Figure 1. Uniplex PCR gel electrograph for thermonuclease (*nuc*) gene- 279 bp. L: DNA ladder (100bp); P: Positive control; N: Negative control; 1-8: *nuc*-positive samples.

Figure 2. Uniplex PCR gel electrograph for *tst* gene (306 bp). L: DNA ladder (100bp); P: Positive control; N: Negative control; 1-8: *tst*-positive samples.
Discussion

Toxic shock syndrome toxin is a very potent superantigen toxin in the virulence arsenal of *S. aureus* and strains encoding the *tst* gene can cause toxic shock syndrome, a multisystemic disease. From our current study 103 (67.76%) isolates possessed the *tst* gene of the 152 *S. aureus* isolates studied. This prevalence is comparable to 60.6% reported in Egypt [19]. However, Vandendriessche et al. [20] reported 19.4% among blood culture isolates in the Democratic Republic of Congo, Ezeamagu et al. [21] reported 14% in Southwest Nigeria, and Omar et al. [22] reported 46.7% in Baghdad, Iraq. In Iran, Derakhshan et al. [23] reported 22.8%, Goudarzi et al. [24] reported 25.43%, Houri et al. [25] reported 33.7%, and Alni et al. [26] reported 22.5%. Furthermore, Senon et al. [27] reported 43.3% in Malaysia, Bhowmik et al. [28] reported 50.79% in Southern Assam, India and Costa et al. [29] reported 26.31%. Also, the presence of the *tst* gene was significantly associated with diabetic foot syndrome, a significant condition associated with neuropathy, ischemia, and infection [30]. Hence, the high prevalence of the *tst* gene among clinical isolates, as reported in our study is a significant cause of alarm. However, variations in the prevalence of the *tst* gene for other geographical locations can be associated with the fact that the *tst* gene is harbored on *S. aureus* pathogenicity island (SaPIs) whose dissemination is not constant within different ecological niches. Also, as a superantigen, TSST can play a significant role in the pathogenesis of several *S. aureus* diseases including diabetic foot syndrome and sepsis. However, TSST-1 production is a factor of environmental variables such as partial oxygen and carbon dioxide gases, pH, temperature, and iron concentration [25].

Our study reported a higher prevalence of the *tst* gene among MRSA than MSSA. This finding is in tandem with reports made in similar studies where an association was observed between methicillin resistance and the prevalence of the *tst* gene [20,31]. Other similar studies have reported an insignificant association between methicillin sensitivity and prevalence of the *tst* gene [19,22,23]. The positive association between the prevalence of the *tst* gene and phenotypic methicillin resistance is a cause for worry. Hence, it becomes imperative to take caution to curb the spread of *tst*-harbouring MRSA strains within hospital and community settings. Furthermore, *tst*-harbouring MRSA strains within this center have higher odds of being resistant to tetracycline and erythromycin compared to other antibiotics.

In our present study, the presence of the *tst* gene was associated with resistance to erythromycin and tetracycline while the absence of the *tst* gene was associated with resistance to cotrimoxazole, fosfomycin, ciprofloxacin, and gentamicin. Also, no association was observed between the presence of the *tst* gene and resistance to clindamycin and fusidic acid. Derakhshan et al. [23] also reported an association between resistance to ciprofloxacin and the absence of the *tst* gene in Iran. However, Sultan et al. [19] and Omar et al. [22] did not report any correlation between possession of the *tst* gene and phenotypic antibiotic resistance. All *S. aureus* isolates were susceptible to linezolid, mupirocin, tigecycline, and rifampin. However, caution should be taken to prevent the development of resistant strains due to antibiotic pressure.

Conclusion

Toxic shock syndrome toxin is a superantigen toxin of immense public health importance due to its ability to excite the human immune system, ultimately leading to a cytokine storm that culminates in the pathological signs of TSS. Hence, this study was carried out to determine the prevalence of the *tst* gene in clinical isolates and to also associate the presence of this gene with phenotypic sensitivity to antibiotics. The prevalence of the *tst* gene was 67.76%, with a significantly higher prevalence among MRSA isolates than MSSA isolates. Hence, in the study, the presence of the *tst* gene was associated with methicillin resistance in *S. aureus*. The study also reported higher resistance to erythromycin and tetracycline among *tst*-harbouring *S. aureus* and higher resistance to cotrimoxazole, fosfomycin, ciprofloxacin, and gentamicin among *tst*-negative *S. aureus* strains. The high prevalence of the *tst* gene among clinical *S. aureus* isolates is a cause for concern in this study area. However, of more concern is the higher prevalence of this gene amongst MRSA isolates and its association with higher resistance rates to antibiotics.

Conflict of interest

We declare that we have no conflict of interest.

Financial disclosure: None.
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