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ANTIBIOTIC SUSCEPTIBILITY PATTERN AND DETECTION OF *mec*A GENE IN METHICILLIN RESISTANT *STAPHYLOCOCCUS EPIDERMIDIS* ISOLATED FROM WARDS SURFACES OF KAMPALA INTERNATIONAL UNIVERSITY TEACHING HOSPITAL, UGANDA

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ABSTRACT

Introduction: *Staphylococcus epidermidis* is a Gram positive, coagulase-negative staphylococcus that frequently causes device- or surgery-associated nosocomial infections worldwide. Drug-resistant strains such as methicillin resistant *S. epidermidis* (DR-SE) have been reported with serious clinical implications.

Objectives: This study determined the percentage of *S. epidermidis* from wards surfaces of Kampala International University-Teaching Hospital (KIU-TH), the drug susceptibility patterns of the isolates and searched for *mec*A gene among Cefoxitin resistant isolates.

Materials and Methods: A total of three hundred sixty-three (363) swab samples were collected from floors, door knobs and walls from different wards. *S. epidermidis* was identified after subjecting the samples to five tests including growth on mannitol salt agar, catalase, coagulase, Desferrioxamine and Fosfomycin tests. Susceptibility patterns of all the *S. epidermidis* isolates identified were tested against Amikacin, Cefazolin, Trimethoprim-Sulfamethoxazole, Ciprofloxacin, Gentamycin and Cefoxitin using the disc diffusion method. All the isolates resistant to Cefoxitin were analysed for the presence of *mecA gene* using the conventional polymerase chain reaction (PCR) method.

Results: One hundred and twelve 112 (30.8%) strains of *S. epidermidis* were isolated from 363 samples collected. Out of 112 *S. epidermidis* isolates, 11 (9.8%) were found resistant to Cefoxitin and all Cefoxitin resistant isolates (100%) were found to have the *mec*A gene, while 89.3% of the strains were found non-susceptible to Trimethoprim-Sulphamethoxazole.

Conclusion: This study found that *S. epidermidis* is present on wards surfaces of KIU-TH. *S. epidermidis* isolates harboured Trimethoprim-Sulfamethoxazole resistance in a high percent. All the isolates resistant to Cefoxitin were positive for the *mec*A gene. Taking into consideration the high rate of Trimethoprim-Sulphamethoxazole non-susceptibility, Cefazolin, Cefoxitin, Gentamycin and Amikacin are recommended as a better prescription for managing infections caused by *S. epidermidis* resistant to commonly used antibiotics in the studied area.

Keywords: S. epidermidis, ward surfaces, cefoxitin, *mec*A gene, Trimethoprim-Sulphamethoxazole

REZUMAT

Introducere: *Staphylococcus epidermidis* este o bacterie Gram pozitivă, coagulazo-negativă, care produce frecvent infecţii nosocomiale asociate dispozitivelor medicale sau intervenţiilor chirurgicale, la nivel mondial. Tulpini rezistente (DR-SE), precum cele rezistente la meticilină, au fost raportate ca având implicaţii clinice importante.

Obiective: Acest studiu a determinat procentul de tulpini de *S. epidermidis* de pe suprafeţe din secţii ale

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Spitalului Internaţional Universitar Kampala (KIU-TH), profilele de susceptibilitate ale acestora şi a investigat prezenţa genei *mec*A la tulpinile rezistente la cefoxitin.

Materiale și metode: Trei sute şaizeci şi trei (363) de probe au fost colectate cu ajutorul tampoanelor, de pe podele, mânerele uşilor şi pereţi din diferite secţii. *S. epidermidis* a fost identificat utlizând cinci teste: creşterea pe manitol agar, testul catalazei, testul coagulazei, testul deferoxaminei şi testul fosfomicinei. S-a testat susceptibilitatea tuturor izolatelelor la amikacină, cefazolin, trimetoprim-sulfametoxazol, ciprofloxacin, gentamicină şi cefoxitin, utilizând metoda disc difuzimetrică. Toate izolatele rezistente la cefoxitin au fost analizate pentru prezenţa genei *mec*A, utilizând o metodă bazată pe tehnica convenţională de reacţie în lanţ a polimerazei (PCR).

Rezultate: Au fost izolate o sută doisprezece tulpini (112) de *S. epidermidis* din 363 probe colectate (30,8%). Din 112 izolate de *S. epidermidis,* 11 (9,8%) au fost rezistente la cefoxitin, iar din acestea toate (100%) au fost pozitive pentru gena *mec*A, în timp ce 89,3% din tulpini au fost rezistente la trimetoprim-sulfametoxazol.

Concluzie: Acest studiu a arătat că *S. epidermidis* este prezent pe suprafeţe din secţii ale KIU-TH. Izolatele de *S. epidermidis* au prezentat rezistenţă la trimetoprim-sulfametoxazol într-un procent mare. Toate izolatele rezistente la cefoxitin au fost pozitive pentru gena *mec*A. Având în vedere rata mare de rezistenţă la trimetoprim-sulfametoxazol, se recomandă ca o alternativă mai bună, prescrierea cefazolinului, cefoxitinului, gentamicinei şi a amikacinei pentru tratarea infecţiilor cauzate de *S. epidermidis* rezistent la antibioticele utilizate în mod frecvent în zona studiată.

Cuvinte-cheie: *S. epidermidis*, suprafeţe din secţii clinice, cefoxitin, gena *mec*A, trimetoprim-sulfametoxazol

INTRODUCTION

Despite the fact that *Staphylococcus epidermidis* is usually non-pathogenic, it is one of the frequent causes of nosocomial infections worldwide to which hospitalised patients undergoing invasive procedure are prone [1, 2]. Among the nosocomial infections, respiratory and surgical site infections, urinary tract infections, meningitis, blood stream infections, gastroenteritis and endocarditis are considered life-threatening with prosthetic valve endocarditis being of the highest risk and causing 25% mortality worldwide [3]. Two million people are affected by nosocomial infections annually, and 5% to 15% of them result in hospitalization globally [2, 4]. The United States nationwide Surveillance and Control of Pathogens of Epidemiological Importance (SCOPE) database reported *S. epidermidis* as the most common pathogens recovered from nosocomial bloodstream infections (31%) within a 7-year period, followed by *S. aureus* (20%) [5]. According to Azeez [6], the rates of nosocomial staphylococcal infection range from 2 to 49% in Sub-Saharan Africa and varied with the environment, with intensive care units (ICUs) having had the highest occurrence rates of 21.2 - 35.6% [7]. Recently Ehlers *et al.* [8] reported 31% *S. epidermidis* from catheter-related bloodstream infections at an Academic Hospital in Pretoria, South Africa. In Uganda, the percentage of *S. epidermidis* is approximately 15%, according to a study conducted in Mulago Teaching and National Referral Hospital, Kampala [9]. A study by Nalwoga *et al.* [10], showed 13% prevalence of coagulase negative staphylococcus including *S. epidermidis* from wound samples in the surgical ward patients of KIU-TH. However, the source of contamination had not been established. Another study conducted in Makerere revealed a 27% out of 187 isolates of coagulase-negative staphylococci of which 60% were resistant to the common antibiotics tested [11].

Drug-resistant strains of *S. epidermidis* (DR-SE) have become a very serious clinical problem, due to the difficulties in eradicating infections from colonized devices [12]. *S. epidermidis* is resistant against commonly used antibiotics, including methicillin, which is mediated by the *mec*A gene encoding a penicillin binding protein with reduced affinity to beta-lactam antibiotics, similar to that in *S. aureus* [13]. However, a study on susceptibility patterns of *S. epidermidis* to antibiotics commonly used in KIU-TH have not yet been done.

Compared to methicillin-resistant *S. aureus* (MRSA), little attention is paid to methicillin

resistant *S. epidermidis* (MRSE) in Karolin health care settings [14]. However, a high incidence (80%) of *S. epidermidis* isolates resistant to both methicillin and other common antibiotics is recorded worldwide [15]. A study by Milisavljevic *et al.* [16] showed antibiotic resistance genes to be widespread among *S. epidermidis*, the most common being the *mec*A gene, known to confer resistance to Oxacillin and some other antibiotics carried by staphylococcal cassette chromosome *mec* (SCC*mec*) of various sizes and structure, with biofilm as their major virulence factor [17-18]. The *mec*A gene encodes for the binding protein known as penicillin binding protein (PBP2a), which mediates resistance to all β-lactam antibiotics in staphylococci [18-19].

Information on the gene responsible for resistance among resistant isolates from KIU-TH ward surfaces was not available. Therefore, the present study aimed at determining the antibiotics susceptibility patterns and detecting the *mec*A gene in methicillin resistant *Staphylococcus epidermidis* isolated from wards surfaces of (KIU-TH) in Uganda.

MATERIALS AND METHODS

Study area

This study was carried out in KIU-TH, located in Ishaka Municipality, Bushenyi District, Western Uganda, GPS location 00˚ 32'19"S; 30˚ 08'40"E. This hospital is approximately 330 kilometres (210 miles) Southwest of Kampala [20]. It is a well-established referral hospital with several wards which include: Medical, Surgical, Paediatric, Maternity, Psychiatric, Accident and Emergency, Private and Semi-Private Wards. In addition, there are a wide range of "Specialist" departments and clinics, including: General Surgery, Orthopaedics, Obstetrics & Gynecology, Internal Medicine, Ophthalmology, Dentistry, Paediatrics, and Physiotherapy. This hospital has 700 beds occupancy (unpublished data).

Study design, sampling strategy and Sample collection

A cross-sectional study using purposive sampling was conducted to determine the percentage of *S. epidermidis*, its susceptibility patterns and genes associated with Cefoxitin resistance. Three hundred and sixty three swab samples were collected from floors, door knobs, walls, and bedrails from seven wards: Medical, Surgical, Paediatric, Maternity, Psychiatric, Accident and Emergency, private and semi-private wards with approval from the hospital management. The samples were collected from the surfaces using sterile swabs soaked in normal saline (0.8%), according to the method described by Valle *et al.* [21]. The swabs were placed into a falcon tube (15ml) containing 2 ml of Stuart transport media (Oxoid-CM0111, UK) and the samples were kept in an ice cooler box during collection process, after which the samples were transported to the Microbiology Laboratory, Department of Microbiology and Immunology of KIU-Western Campus for analysis.

Isolation and identification of *S. epidermidis*

The swabs were inoculated on mannitol salt agar – MSA (Oxoid-CM0085, UK) plates and streaked with a sterile wire loop to obtain discrete colonies, in a bio-safety cabinet to avoid contamination. The plates were then incubated at 37°C for 24h [22]. The isolates were identified by Gram-staining method. To differentiate between coagulase negative and coagulase positive staphylococci, the coagulase test was performed by placing 50µl of rabbit plasma onto a sterile glass slide followed by emulsifying a loopful of the bacterial colony on the slide using a sterile wire loop and agglutination observed [23]. Further confirmation of *S. epidermidis* was done using Desferrioxamine and Fosfomycin. Solutions containing 1mg/mL each were prepared and impregnated on 6mm diameter paper discs made from Whatman filter paper number 1 and tested against the coagulase-negative isolates to differentiate *S. epidermidis* from other species of coagulase-negative staphylococci [24].

Antibiotics susceptibility patterns of *S. epidermidis strains*

The antibiotic susceptibility testing was per-

formed using the Kirby-Bauer disc diffusion method [25]. Colonies of a 24h pure culture of *S. epidermidis* strains isolated from the selected surfaces of KIU-TH were picked up from nutrient agar (HiMedia M001, India) plates, transferred into tubes containing tryptic soy broth media (Sigma-Aldrich), and turbidity adjusted to 0.5 McFarland standards. Freshly prepared Sterile Mueller Hinton agar plates (Oxoid-CM0337, UK) were inoculated with the standardized suspension (0.5 McFarland standards) of the isolates using sterile cotton swabs. Antibiotic discs of commonly used antibiotics in the wards of KIU-TH (Amikacin 30µg, Cefazolin 30µg, Cefoxitin 30µg, Trimethoprim-Sulfamethoxazole 25µg, Ciprofloxacin 30µg and Gentamicin 30µg) were placed on the surfaces of inoculated agar plates using sterile forceps and then incubated at 37°C for 24h. The diameters of the zone of inhibitions were measured in millimeters and results interpreted according to the guidelines of Clinical and Laboratory Standard Institute [26].

Detection of *mec*A gene among Cefoxitin resistant *S. epidermidis strains*

Bacterial DNA extraction was performed using the standard protocol as described by Prasad *et al.* [27]. Briefly, 5 ml overnight culture of *S. epidermidis* was centrifuged for 10 minutes to harvest the cells. The supernatant was discarded and 875 µL of TE buffer was added to the pellet. The cells were suspended in the buffer by gentle mixing. 100 µL of Sodium dodecyl sulphate and 5 µL of proteinase K were added to the cells. One millilitre (1 mL) of phenol-chloroform mixture was added to the content and mixed well by inverting the tubes and incubated at room temperature for 5 minutes. The tubes were centrifuged at 10,000 rpm for 10 minutes at 4°C. The supernatant was collected using cut tips and transferred to a fresh tube. The process was repeated once again by using phenol-chloroform mixture and the supernatant was collected in a fresh tube. One hundred microliters (100 µL) of sodium acetate 5M was added to the tubes and mixed gently. Two millilitres (2 mL) of isopropanol was added and mixed gently by inversion till a white precipitate of DNA was formed from the mixture. Ninety microliters (90µL) of the supernatant containing the deoxyribonucleic acid (DNA) were transferred into a new clean tube and put in an ice box for amplification [28, 29].

Polymerase chain reaction (PCR)

The presence of *mec*A gene in Cefoxitin resistant *S. epidermidis* isolates was detected using conventional PCR conducted by the Molecular Laboratory, College of Veterinary Animal Resources and Biosecurity, Makerere University. A known methicillin resistant *S. epidermidis* and distilled water were used as positive and negative controls respectively as described by Arefi *et al.* [28]. The DNA extracts of the methicillin resistant *S. epidermidis* strains were submitted to amplification with the *mec*A-Forward (5′-AAA ATC GAT GGT AAA GGT TGG C-3′) and *mec*A-Reverse (5′-AGT TCT GCA GTA CCG GAT TTG C-3′) primers. PCR was performed in a 25µl volume reaction mix containing: 3.5µL of reaction buffer (50mM KCl, 10mM Tris-HCl) (pH 8.0), 2.5 mM MgCl2), 0.5µl of Deoxyribonucleotide triphosphates (dNTP's) (10mM), 1.5µl forward primer, 1.5µl reverse primer, 2.5µl of Taq DNA polymerase, 2.5µl (0.4mM) of DNA template and 13µl of RNase free water. Amplification was achieved through denaturation performed at 94°C for 5 minutes, followed by lowering the temperature of the thermocycler to 55°C for 30 sec to allow the annealing of primers and extension at 72°C for 2 minutes, with a total of 35 cycles and an additional extension at 72°C for 10 minutes. The amplified products were visualized on a 1.5% agarose gel containing 0.5% ethidium bromide under U.V. illumination and analysed against a molecular ladder for an expected amplification product of (500bp) which indicates the presence of *mec*A gene [30].

Data analysis

Descriptive data were uploaded in Ms excel and imported into the Statistical Package for Social Sciences (SPSS) version 21 software (developer/make). Univariate and one way

ANOVA test were used to obtain proportion of the occurrence of *S. epidermidis* and compare its percentage between different wards. Statistical significance was considered at p value ≤0.05.

Ethical approval

The study was approved by the research and ethics committee of KIU-WC (Ref: 2017/06).

RESULTS AND DISCUSSION

Out of the 363 swabs samples collected from the wards surfaces of KIU-TH, 112/363 (30.8 %) samples were positive for *S. epidermidis* as shown in Table 1. The percentage of *S. epidermidis* observed in this study was lower compared to the 43.7% which was reported in the study by Aloma *et al.* [30] in a tertiary health care hospital, Brazil and 39.2% reported by Amenu [31] in the hospital environment of Ethiopia. However, this study showed a higher percentage of *S. epidermidis* than in a study by Ochie and Ohagwu [32] in Nigeria and Wojtyczka *et al.* [33] in Ghana which reported 12.7% and 17.2% respectively. The percentage reported by this study was in line with a study by Boyce *et al.* [34] in Poland, where the percentage of *S. epidermidis* reported in hospital environment was 26.2%.

This study found the highest distribution of *S. epidermidis* in surgical ward 25/112 (22.3%) Table 1. This was in line with the findings of Hammuel *et al.* [35] who conducted a similar study in the surgical ward of KIU-TH which showed that the percentage of Coagulase negative staphylococci isolated from wounds swab was 13.3%. The higher percentage in the surgical ward in this study could probably be the result of the high number of cases referred to this ward as compared to the other wards, thereby increasing the opportunity of disseminating this pathogen on this ward's surfaces.

The higher distribution rate of *S. epidermidis* on bedrail 44/112 (39.2%) (Fig. 1) was in disagreement with the 100% percentage on bedrail as reported by Carvalho *et al.* [36] from Nigeria. Similarly, the distribution rate of 53.8% and 38% of staphylococci on door knobs/handles reported by Carvalho *et al.* [36], Allegranzi and Pittet [37] respectively was relatively higher than the percentage of *S. epidermidis* found on door knobs/handles in our study 30/112 (26.7%). The distribution rate of *S. epidermidis* on door knobs/handles may probably be attributed to the fact that they are the most frequently touched surfaces by visiting patients and attending clinicians. Additionally, the knobs and handles were not being mopped with disinfectant after cleaning in all the seven selected wards as reported by interviewed cleaners (data not shown), Carvalho *et al.* [36], Allegranzi and Pittet [37] reported that environmental contamination in health care settings arise when healthcare workers touch the surfaces with their bare hands or gloves, particularly after attending to the patients or when the patients come in direct contact with the surfaces.

The distribution rate of 27/112 (24.1%) on the floor was higher than 8.6% and 16.7% reported by Allegranzi and Pittet [37] in two different hospital environment in Zaria. However, the percentages of 30.8% reported by Carvalho *et al.* [36] and 50.0% by Hulya and Dilek [38] in Brazil were higher if compared to those in this study. Aloma [30] reported a distribution rate of *Staphylococcus aureus* on floors of 87.5, 75, 100, and 100% each from Gambo Sawaba hospital, St. Gerard, Barau Dikko Specialist hospital, Nigeria and Yusuf Dantsoho memorial hospital, Nigeria which were higher than the finding of this study. Perhaps the variations of the contamination between these hospitals could be due to the difference in hygiene practices within the hospitals. In our study, walls had the least distribution rate of *S. epidermidis* (9.8%) among other surfaces; this may be due to the fact that patients and health workers rarely get in contact more with the walls as compared to other surfaces [39-40].

The percentage of resistant (R), intermediate (I) and susceptible (S) strains of *S. epidermidis* isolated from the selected wards surfaces of KIU-TH against the different antibiotics commonly used at KIU-TH are shown in Table 2 (a, b) below. Cefazolin and Cefoxitin were the most efficient (92.2% susceptible strains each), while Trimethoprim-Sulfa-

methoxazole was the least efficient (10.7% susceptible strains) among the antibiotics tested. The highest resistance rate was registered with Trimethoprim-sulfamethoxazole (80.4%) while Amikacin and Gentamycin showed the least resistance with 4.5% each.

The highest percentage of resistance of *S. epidermidis* against Trimethoprim-Sulfamethoxazole (80.4%) was in agreement with the findings of Muhammad *et al.* [41], Meriem *et al.* [42], Hadadi *et al.* [43] and Rodríguez *et al.* [44] who reported 62.5%, 82%, 58.5% and 87% of *S. epidermidis* resistance to Trimethoprim-Sulfamethoxazole respectively. However, it was higher than 33.33% of *S. epidermidis* resistance to Trimethoprim-Sulfamethoxazole reported by Bayram and Balci [45]. Resistance to Ciprofloxacin (18.8%) in this study was in disagreement with the results reported by Meriem *et al.* [42], Bayram and Balci [45], with 79% and 66.67% resistance respectively. Moreover, a set of different results were reported from Iran and Argentina, ranging between 56% - 77% and 80% respectively [46-47]. However, our results were close to the results reported in Turkey and Brazil by Madhusudhan *et al.* [49] and Akindele *et al.* [50] with 20 to 59.2% and 25.5% resistance respectively. The highest percentage of resistance of *S. epidermidis* to Trimethoprim-Sulfamethoxazole in our study could be a result of its frequent use in the hospital because of being cheaper and thus considered a first line drug.

The percentage of Cefoxitin resistance (9.8%) in our study was very low if compared to the previous studies by Meriem *et al.* [42], Ibrahim *et al.* [50], Bilal and Srikanth [51] who reported 58%, 50.4% and 33.3% of Cefoxitin resistance of *S. epidermidis* in their studies, respectively. Resistance to Cefoxitin by disc diffusion can be used for the detection of methicillin resistant *S. epidermidis* (MRSE) strains in routine testing because Cefoxitin is a potential inducer of the system that regulates *mecA* gene [52]. The low percentage of Cefoxitin resistance by *S. epidermidis* in this study could be the result of the insignificant number of MRSE distribution in the hospital through which increased chances of transferring resistance genes from one strain to another would occur.

The low percentage of Gentamycin resistance (4.5%) in our study was similar to the study by Allegranzi and Pittet [37] who reported 0.00% of the pathogens to be resistant to Gentamycin but different to the finding of Iorio *et al.* [53] with 39% resistance. Moreover, the percentage of Amikacin resistance in *S. epidermidis* (4.5%) according to our study is similar with the findings of Bayram and Balci [45] showing 2.78% of *S. epidermidis* resistant to Amikacin. Cefazolin resistance (9.8%) in this study is similar with percentages found by Rodríguez *et al.* [44], of 7.2% *S. epidermidis* resistant to Cefazolin, while, Andrea *et al.* [54] reported that 82.8% of *S. epidermidis* were resistant to Cefazolin in their study, which was higher than our findings. However, Cefazolin and Cefoxitin remained the most effective antibiotics against *S. epidermidis* isolated from KIU-TH and this could be as the result of it being a rarely used drug, which is lowering the probability of selecting bacterial resistant strains and resistance transmission.

The percentage of susceptibility to Cefazolin, Gentamycin, Amikacin and Ciprofloxacin (90.2%, 86.7%, 89.3%, and 61.6%) in our study was slightly higher than findings of Andrea *et al.* [54] who reported 50.8%, 44.3% and 17.2% for Gentamycin, Ciprofloxacin and Cefazolin respectively. Moreover, in other studies by Rodríguez *et al.* [44] and Amita *et al.* [55], Amikacin, Cefazolin and Gentamycin were effective against *S. epidermidis* with 93.1%, 91.8%, and 96.2%, respectively which were higher than the results of the present study. The high susceptibility rates to Cefazolin, Gentamycin, Amikacin and Ciprofloxacin (90.2, 86.7%, 89.3%, and 61.6%)) in our study could be the result of them being second line antibiotics and a bit more expensive, which decreases their use in our patients and thus they are less exposed to resistant bacteria. These variations in antibiotic susceptibility patterns indicate that regional differences may play a role in the resistance profiles of bacteria and further justifies the necessity to embark on antibiotic susceptibility studies

on bacterial isolates from different hospitals on a regular basis [37]. Though in a low percent, it was observed that 11 strains of *S. epidermidis* (9.8%) isolated from our hospital's wards were resistant to Cefoxitin and Cefazolin.

All the isolates resistant to Cefoxitin in this study were positive for the *mec*A gene. The presence of the *mec*A gene associated with methicillin resistance among the Cefoxitin resistant *S. epidermidis* isolated from different selected wards surfaces is shown in Table 3 and Fig. 2.

The presence of *mec*A gene in all the 11 isolates in this study indicated that the isolates were resistant to all the β-lactam group of antibiotics. The presence of *mec*A gene in all the 11 isolates resistant to Cefoxitin in this study was in line with the study by Samah *et al.* [56], where *mec*A gene was found in all the 300 isolates resistant to Cefoxitin (100%). The percentage obtained from this study was higher compared to studies by Peacock *et al.* [57] and Natalia *et al.* [58] who reported percentages of 95.12% and 93.75% respectively of *mec*A positive *S. epidermidis* resistant to Cefoxitin.

Table 1. The percentage distribution of *Staphylococcus epidermidis* in ward surfaces of Kampala International University-Teaching Hospital

Sample source (wards)	Surfaces	Sample collected	No. of *S. epidermidis* isolated (%)
Medical ward	Wall	12	1 (0.89)
	Bedrail	14	8 (7.14)
	Floor	12	5 (4.46)
	Doorknob	14	3 (2.67)
Subtotal	**-**	**52**	**17 (15.18)**
Surgical ward	Wall	12	2 (1.78)
	Bedrail	14	9 (8.03)
	Floor	12	6 (5.36)
	Doorknob	14	8 (7.14)
Subtotal	**-**	**52**	**25 (22.32)**
Maternity ward	Wall	12	1 (0.89)
	Bedrail	14	5 (4.46)
	Floor	12	5 (4.46)
	Doorknob	14	5 (4.46)
Subtotal	**-**	**52**	**16 (14.29)**
Accident and Emergency ward	Wall	12	3 (2.67)
	Bedrail	14	5 (4.46)
	Floor	12	5 (4.46)
	Doorknob	14	2 (1.78)
Subtotal	**-**	**52**	**15 (13.39)**
Pediatrics ward	Wall	12	2 (1.78)
	Bedrail	14	7 (6.36)
	Floor	12	3 (2.67)
	Doorknob	14	3 (2.67)
Subtotal	**-**	**52**	**15 (13.39)**
Private ward	Wall	11	0 (0.00)
	Bedrail	14	4 (3.57)
	Floor	12	1 (0.89)
	Doorknob	14	5 (4.46)
Subtotal	**-**	**51**	**10 (8.93)**
Semi-private ward	Wall	12	2 (1.78)
	Bedrail	14	6 (5.36)
	Floor	12	2 (1.78)
	Doorknob	14	4 (3.57)
Sub total	**-**	**52**	**14 (12.5)**
Total	**-**	**363**	**112 (30.85)**

Table 2a. The antibiotic susceptibility pattern of *Staphylococcus epidermidis* isolates from the selected wards surfaces of Kampala International University-Teaching Hospital

Isolates per ward	Antibiotics (%)							
	Amikacin			Cefazolin*		Trimethoprim-sulfamethoxazole		
	R	I	S	R	S	R	I	S
SGW (N=25)	1 (4.0)	3 (12.0)	21 (84.0)	3 (12.0)	22 (88.0)	15 (60.0)	3 (12.0)	7 (28.0)
MDW (N=17)	3 (17.6)	1 (5.9)	13 (76.5)	2 (11.8)	15 (88.2)	11 (64.7)	4 (23.5)	2 (11.8)
AEW (N=15)	1 (6.7)	0 (0.0)	14 (93.3)	2 (13.3)	13 (86.7)	11 (73.3)	3 (20.0)	1 (6.7)
PDW (N= 15)	1 (6.7)	0 (0.0)	14 (93.3)	2 (13.3)	13 (86.7)	14 (99.3)	0 (0.0)	1 (6.7)
MTW (N=16)	0 (0.0)	1 (6.2)	15 (93.7)	2 (12.5)	14 (87.7)	15 (93.7)	0 (0.0)	1 (6.2)
PRW (N=10)	0 (0.0)	0 (0.0)	10 (100.0)	0 (0.0)	10 (100.0)	10 (80.0)	0 (20.0)	0 (0.0)
SPW (N=14)	0 (0.0)	1 (7.1)	13 (92.9)	0 (0.0)	14 (100.0)	14 (100.0)	0 (0.0)	0 (0.0)
Total (N=112)	**6(5.4)**	**6(5.4)**	**100(89.3)**	**11(9.8)**	**101(90.2)**	**90(80.4)**	**10(8.9)**	**12(10.7)**

*Susceptibility to Cefazolin is mentioned because this is the therapeutic alternative in Cefoxitin susceptible strains.

Key: R= Resistance, I= Intermediate, S= Susceptible, SGW= Surgical ward, MDW= Medical ward, AEW= Accident and Emergency ward, PDW= Pediatrics ward, MTW= Maternity ward, PRW= Private ward, SPW= Semi-private ward.

Table 2b. The antibiotic susceptibility pattern of *Staphylococcus epidermidis* isolates from the selected wards surfaces of Kampala International University-Teaching Hospital

Isolates per ward	Antibiotics							
	Ciprofloxacin			Gentamycin			Cefoxitin	
	R	I	S	R	I	S	R	S
SGW (N=25)	6 (24.0)	7 (28.0)	12 (48.0)	1 (4.0)	3 (12.0)	21 (84.0)	3 (12.0)	22 (88.0)
MDW (N=17)	1 (5.9)	6 (35.3)	10 (58.8)	2 (11.8)	4 (23.5)	11 (64.7)	2 (11.8)	15 (88.2)
AEW (N=15)	2 (13.3)	3 (20.0)	10 (66.7)	1 (6.7)	0 (0.0)	14 (93.3)	2 (13.3)	13 (86.7)
PDW (N= 15)	2 (13.3)	1 (6.7)	12 (80.0)	1 (6.7)	0 (0.0)	14 (93.3)	2 (13.3)	13 (86.7)
MTW (N=16)	3 (18.7)	3 (18.7)	10 (62.5)	0 (0.0)	1 (6.2)	15 (93.7)	2 (12.5)	14 (87.7)
PRW (N=10)	4 (40.0)	1 (10.0)	5 (50.0)	0 (0.0)	1 (10.0)	9 (90.0)	0 (0.0)	10 (100.0)
SPW (N=14)	3 (21.4)	1 (7.1)	10 (71.4)	0 (0.0)	1 (7.1)	13 (92.9)	0 (0.0)	14 (100.0)
Total (N=112)	**21(18.8)**	**22(19.6)**	**69(61.6)**	**5(4.5)**	**10(8.9)**	**97(86.6)**	**11(9.8)**	**101(90.2)**

Key: R= Resistance, I= Intermediate, S= Susceptible, SGW= Surgical ward, MDW= Medical ward, AEW= Accident and Emergency ward, PDW= Pediatrics ward, MTW= Maternity ward, PRW= Private ward, SPW= Semi-private ward.

Table 3. Methicillin resistant *Staphylococcus epidermidis* isolated from wards surfaces of Kampala International University-Teaching Hospital

Isolates	Wards Surfaces	Resistance in other antibiotics	*mec*A gene
SG11B	Bedrail	SXT,CIP	+
SG03D	Doorknob	SXT,CIP,CZ	+
SG17D	Doorknob	SXT	+
MD11B	Bedrail	SXT,GEN	+
MD53F	Floor	SXT,AK	+
AE28B	Bedrail	SXT,CIP	+
AE48F	Floor	SXT,AK	+
PD23B	Bedrail	SXT	+
PD19B	Bedrail	SXT,GEN	+
MT05W	Wall	SXT	+
MT13B	Bedrail	SXT,CIP	+

Key: SXT= Sulfamethoxazole-Trimethoprim, **CIP**= Ciprofloxacin, **CZ**= Cefazolin, **AK**= Amikacin, **GEN**= Gentamicin, **SGB**= Surgical (Bedrail), **SGD**= Surgical (Doorknob), **MDB**= Medical (Bedrail), **MDF**= Medical (Floor), **AEB**= Accident and Emergency (Bedrail), **AEF**= Accident and Emergency (Floor), **PDB**= Pediatrics (Bedrails), **MTW**= Maternity (Wall), **MTB**= Maternity (Bedrail), + represents positive.

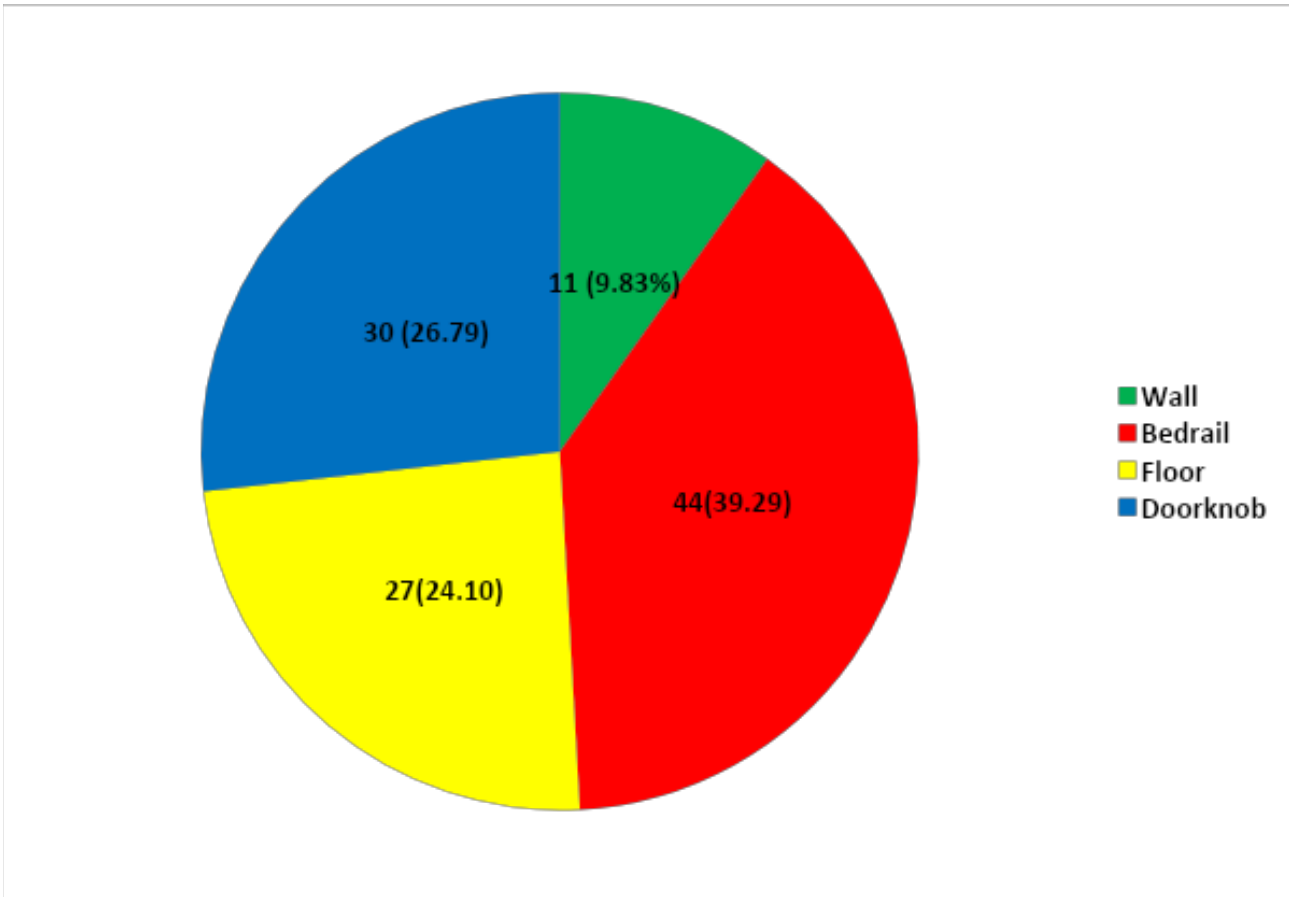

Fig. 1 - Percentage distribution of *Staphylococcus epidermidis* according to the environmental sources

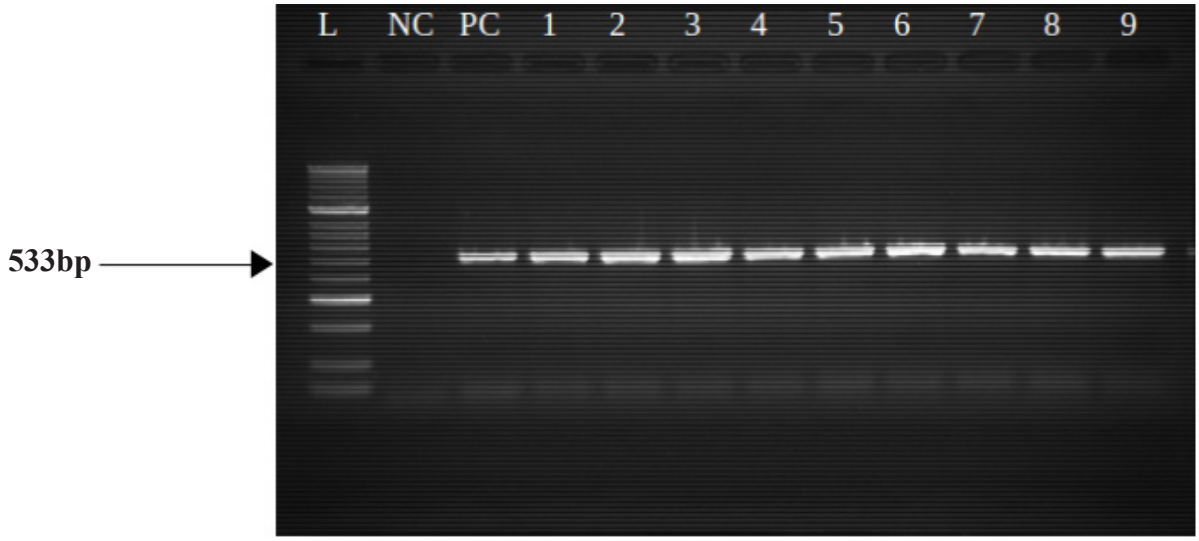

Fig. 2 - Typical amplicons of *mec*A genes for 9 representatives of Methicillin resistant *S. epidermidis* isolates
L: 533bp ladder, **NC:** Negative control (Distilled water), **PC:** Positive control (*mec*A positive of *S. epidermidis*), **sample 1:** SG11B, **sample 2**: SG3D, **sample 3:** SG17D, **sample 4:** MD11B, **sample 5:** MD57F, **sample 6:** AE28B, s**ample 7:** AE48F, **sample 8:** PD23B, **sample 9:** PD19B.

However, our results were different if compared with other studies [59-60] which reported only 80% and 74.02% of *S. epidermidis* harbouring *mec*A gene among the Cefoxitin resistant isolates respectively, which is suggesting other mechanisms of methicillin resistance.

CONCLUSION

This study showed a percentage of positivity of 30.8% *S. epidermidis* from different wards surfaces in KIU-TH with door knobs and bedrails being more contaminated. Trimethoprim-Sulfamethoxazole was the least effective against *S. epidermidis* isolates, while Cefazolin was the most effective. All the 11 (9.8%) Cefoxitin resistant *S. epidermidis* isolates harboured the *mec*A gene.

Based on our findings, our recommendation is that door knobs and bedrails should be mopped and decontaminated using a strong disinfectant that contains phenol, biguanides or halogens. Cefazolin, Gentamycin and Amikacin could be better prescriptions than Trimethoprim-Suphamethoxazol for the management of infections caused by *S. epidermidis* in the studied area. The presence of these resistant bacteria (*S. epidermidis*) from the wards of KIU-TH is dangerous as this organism is associated with transfer of SCC*mec* to methicillin-susceptible *S. aureus* (MSSA). Our study provided evidence that MR-CoNS may act as a reservoir of SCC*mec* for MSSA and this may increase the risk of treatment failure especially among immunocompromised patients. Taking into consideration our findings, there is a need to take measures on this bacteria (*S. epidermidis*) by the management of the KIU-TH hospital and the ministry of health, including hospital hygiene procedures and prescribing of the appropriate antibiotics, as this may fuel the spread of these resistant bacteria within the hospital and the community in general.

Conflict of Interests: The author declares that there is no conflict of interest regarding the publication of this paper.

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