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Antibiotic Sensitivity of *Staphylococcus aureus* Isolated from Patients Attending Ruiru District Hospital, Kenya August to November 2012

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Authors' contributions

This work was carried out in collaboration between all authors. Author EM collected the data and did the tests, author ZO the design and the protocol, author SM the statistical analysis and author MM the literature searches and wrote the paper. All authors read and approved the final manuscript.

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ABSTRACT

Aim: To determine the sensitivity of *Staphylococcus aureus* to commonly used antibiotics in patients with skin, soft tissue and upper respiratory tract infections.
Study Design: Cross sectional.
Place and Duration of Study: Samples were obtained from Ruiru District Hospital, Kenya between August and November 2012. The antibiotic sensitivity tests were done at the Laboratories of Jomo Kenyatta University of Agriculture and Technology at Juja, Kenya.
Methodology: The study included 100 in and outpatients with infections on clinical diagnosis, cellulitis, wound infection, ulcers, septic bruises, abscess (including furuncle / boil / superficial skin abscess). A questionnaire was used to collect patient demographic data from patient records and cultures from the hospital laboratory collected and transported to the Jomo Kenyatta University Laboratories for identification of staphylococcal colonies by culture and biochemical tests. Disc

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diffusion test was used to determine *in vitro* antibiotic sensitivities of the *S. aureus* isolates as per the Kirby-Bauer diffusion technique.
Results: The results indicate that the isolates were very resistant (10% sensitivity) to methicillin and gentamycin, moderately resistant (less than 40% sensitivity) to morepenem, erythromycin, oxacilin, ampicilin, penicillin, trimethoprime/sulfamethoxazole and amoxicillin/clavulanic acid but sensitive to minocycline (83.3%). The isolates were also sensitive to cefuroxime, ciprofloxacin, chlorophenicol and lincomycin (75, 58.3, 50 and 41.6% sensitivity respectively). In this regard, antibiotics such as gentamycin, chloramphenical, trimethoprime / sulfamethoxazole and ampicillin, on the WHO and Kenyan essential drugs list are unlikely to offer help to the patients in Ruiru District hospital. Minocycline would thus be the antibiotic of choice against *Staphylococcus* infections followed by cefuroxime (75%) and ciprofloxacin (58.3%) as the next alternative drugs of choice.

Keywords: Antibiotic; bacteria; sensitivity; resistance; S. aureus.

1. INTRODUCTION

The use penicillin as antibiotic was first described by Sir Alexander Fleming [1] and since then, antibiotics have been important therapeutic agents for mankind. In the past years there has been marked increase in the number of antibiotics prescribed as well as evolution of new antibiotics. Simultaneously there has been irrational and excessive use of antibiotics for chemotherapy in both humans and livestock has led to development of antibiotic resistance and even multiple drug resistance bacteria.

In most case, the initial antimicrobial prescribed for treatment of an infection is chosen on the basis of clinical impression after the physician is convinced that an infection exists and has made a tentative etiological diagnosis on clinical grounds. Rarely in developing countries such as Kenya are specimens obtained for laboratory isolation of the causative agent and sensitivity testing to inform the antibiotic of choice. However, the identification microorganisms that are known to be susceptible to certain antibiotics in an area permit the selection of optimally effective drugs. The commonly performed tests are disks diffusion susceptibility tests [2].

Staphylococcus, a member of the Firmicutes, was first identified in 1880 in Aberdeen, United Kingdom, by the surgeon Sir Alexander Ogston in pus from a surgical abscess in a knee joint [3]. One species, *Staphylococcus aureus* is frequently found as part of normal bacteria flora and is estimated that between 20-40% of the human population are long-term carriers in reservoirs in the anterior nares and thus produce nasal secretions and can infect others [4-6].

Although *S. aureus* is not always pathogenic, disease-associated strains cause a range of illnesses, from minor skin infections, boils (furuncles), cellulitis, folliculitis, carbuncles, scalded skin syndrome, and abscesses, to life-threatening diseases such as pneumonia, meningitis, osteomyelitis, endocarditis, toxic shock syndrome (TSS), bacteremia, and sepsis. Its incidence ranges from skin, soft tissue, respiratory, bone, joint, endovascular to wound infections. *S. aureus* is one of the five most common causes of nosocomial infections and is often the cause of postsurgical wound infections especially in people with weakened defense systems or in patients undergoing major surgery. Each year, some 500,000 patients in American hospitals contract a staphylococcal infection [7].

Bacteria either have intrinsic mechanisms for antibiotic resistance which they develop through mutation and selection processes or acquires resistance through genetic transfer from other bacteria in the vicinity. Joshua and Esther Lederberg showed penicillin and streptomycin resistant bacteria existed before penicillin treatment and are believed that this emerged as a defense mechanism [8]. The prevalence of antibiotic resistant bacteria has not only increased with time but also multi resistance in regard to antibiotics of similar or different mechanism of action.

Widespread use of antibiotics both inside and outside of medicine has however played a significant role in the emergence of resistant bacteria, development of multi drug resistant strains and the spread of resistance between bacterial species [9]. This has been facilitated by misuse, underuse and overuse of antibiotics by physicians as well as patients. Of particular importance is “over the counter” dispensing of

antibiotics believed to be rampant and thrives due to poor regulatory implementation as well as patients taking less than the recommended dosage or failing to complete their doses. It has been reported that a large number of people do not finish a course of a once daily antibiotic, 10% to 44% saying that this is because they felt better [9]. These low or incomplete dosages result in decreased concentration of antibiotics in the blood stream and tissues allowing the more resistant bacteria present to survive and be selected leading to more resistant strains and a possible relapse with a more severe infection that is more difficult to treat.

Despite the importance of antibiotic resistance, no comprehensive data base is available regarding the status of clinical isolates in certain areas due to the dynamic nature of emergence and distribution of resistance.

There are various mechanisms of antibiotic resistance for instance; penicillin resistance mediated by penicillinase (a form of β-lactamase) production is renders β-lactam antibiotics, such as methicillin, nafcillin, oxacillin, cloxacillin, dicloxacillin, and flucloxacillin ineffective; or mediated via the mec operon, lowering affinity for binding β-lactams (penicillins, cephalosporins, and carbapenems). Glycopeptide resistance on the other hand is mediated by acquisition of the vanA gene while resistance to aminoglycoside antibiotics (kanamycin, gentamycin) is by aminoglycoside modifying enzymes, ribosomal mutations, and active efflux of the drug out of the bacteria [10,11].

There are *S. aureus* strains resistant to many commonly used antibiotics. The β-lactamase-resistant penicillins (methicillin, oxacillin, cloxacillin, and flucloxacillin) were developed to treat penicillin-resistant *S. aureus,* and are still used as first-line treatment. Methicillin, the first antibiotic in this class introduced in 1959, was ineffective only two years later, as the first case of methicillin resistant *S. aureus* (MRSA) was reported in England [12]. Despite this, MRSA generally remained an uncommon finding, even in hospital settings, until the 1990s, when there was an explosion in MRSA prevalence in hospitals, where it is now endemic. In the UK, only 2% of all *S. aureus* isolates are sensitive to penicillin, with a similar picture in the rest of the world [13].

MRSA infections in both the hospital and community settings are commonly treated with non-β-lactam antibiotics, such as clindamycin and trimethoprime-sulfisoxazole. Resistance to these antibiotics has also led to the use of new, broad-spectrum anti-Gram-positive antibiotics, such as linezolid. First-line treatment for serious invasive infections due to MRSA is currently glycopeptide antibiotics (vancomycin and teicoplanin). In Kenya, this is restricted for use in level 4 hospitals [14].

Vancomycin-resistant *S. aureus* (VRSA) also emerged the first case of vancomycin-intermediate *S. aureus* (VISA) reported in Japan in 1996 [15] and the first case of *S. aureus* truly resistant to glycopeptide antibiotics reported in 2002 [16]. Three cases of VRSA infections had been reported in the United States as in 2005 [17]. MRSA is the most frequently identified antimicrobial drug resistant pathogen in hospitals in the United States, with such hospital acquired *S. aureus* infections increasing from 2% in 1974 to 22% in 1995, and 50% in 1997 [18].

Antibiotic resistant pathogens including *S. aureus* have been observed in Africa. For instance, in a hospital study in Mogadishu, Somalia, multidrug *S. aureus* strains were observed [19] while in Nigeria, 40% of ear infections and bronchitis were shown to be resistant [20]. Although *S. aureus* is responsible for illnesses ranging from bacteremia to infections of the central nervous system and respiratory tract, the prevalence of resistant isolates has not been documented for the majority of African countries [21].

In developing countries, the need for antibiotics is driven by high incidence of infectious diseases. There are also complex socioeconomic and behavioral factors such as irrational use of antibiotics by health professionals, unskilled practitioners and lay persons, poor drug quality, unhygienic conditions, poor drug regulation and inadequate surveillance [9]. This bacterial exposure culminates in development, selection and spread of resistant organisms contributing to escalating problem of antibiotic resistance worldwide.

Of all surgical infection cases in Kenya, 30-40% were shown to be due to *Staphylococcus* approximately 50% being abscesses which culminated in mortalities in 30% of those untreated [4,22]. Patients with purulent wounds may infect others by invisible droplets containing bacteria Staphylococci through flies, equipment and fingers and clothing of the nursing and medical staff [22]. Resistant strains of

Staphylococci are common in Kenyan hospitals and may cause very serious infections particularly in hospitalized patients. It is believed that the excessive use and misuse of antibiotics has contributed towards antimicrobial resistance leading treatment failure and relapses with more severe infections that are more difficult to treat. This is particularly among chronically ill, surgical and hospitalized patients.

Determination of antibiotic sensitivity profiles of *S. aureus* in respect to commonly used antibiotics may vary in different hospitals from time to time. An institution specific study on efficacy of antibiotics in treatment of infections caused by *S. aureus* is thus necessary to inform treatment plans that will achieve successful treatment outcomes. Furthermore, nationwide information from such studies can then be used to inform national policy such as the essential drug list. Overall, the emergence and spread of resistance to antibiotics used to treat infections caused by *S. aureus* will be minimized by rational use of antibiotics by health professionals.

To mitigate this resistance, it is important for laboratory identification of *Staphylococcus aureus* and antibiotic sensitivity of isolates from the site of infection or blood culture through commonly used methods which include coagulase [23], agglutination [24] and hybridization tests [25]. This will provide comprehensive information on antibiotic sensitivities of *S. aureus* which is lacking in Kenya [26]. The aim of the present study was to determine, among the most commonly dispensed antibiotics, which were the most efficacious in the treatment of *Stapylococcus aureus* infections in patients attending Ruiru District Hospital, Kenya.

2. MATERIALS AND METHODS

This was a cross sectional study done at Ruiru District hospital located in Kiambu County of Kenya using specimens from the hospital laboratory and demographic data from patient records. The population studied was both in and outpatient adults (18 years ad above) attending the Ruiru District Hospital in the period between August and November 2012. This is a public hospital serving patients with middle to lower social economic status from a rural and peri urban area. Eligible patients for enrolment were those with skin, soft tissue and upper respiratory tract infections on clinical diagnosis, cellulitis, wound infection, ulcers, septic bruises, abscess (including furuncle / boil / superficial skin abscess). A sample size of 100 was determined using the following formula according to Fisher [27].

After obtaining ethical consent from the hospital administration, specimens were taken with sterile Bacron swabs from the hospital laboratory, put on Armies transport media (Oxoid, Unpath, Hemisphere England), transported to the Jomo Kenyatta University Laboratories, cultured on Blood Agar media or Mannitol Salt Agar, and then incubated at 37°C for 18-24 hours. The plates are then examined for typical staphylococcal colonies and gram staining, culture, biochemical tests (catalase, coagulase). The demographic data of the patients from patient records was captured in a questionnaire without including patient details that would jeopardize their privacy and confidentiality.

After identifying pathogenic *Staphylococcus aureus*, the disc diffusion test was used to determine i*n vitro* sensitivities of the isolates to commonly used and easily available antibiotics (morepenem, erythromycin, oxacilin, ampicilin, penicillin, trimethoprime / sulfamethoxazole, amoxicillin / clavulanic acid, cefuroxime, ciprofloxacin, chlorophenicol and lincomycin) as per the Kirby-Bauer diffusion technique [28] in accordance with CLSI (formerly, NCCLS) criteria, 2006 [29].

3. RESULTS AND DISCUSSION

Samples were obtained from a total of 100 patients of mean age 18-69 years; 32 of which were male and 68 females. Among 100 patients sampled, boils, abscesses, cellulitis and ulcers were the major conditions (Fig. 1). The frequency distribution of ages of the patients ranged from 50-59 (12%) to 30-39 (29%). Although abscesses and cellulitis were most frequently found in patients aged 30-39 and boils in the 60-69 age group, these differences were not statistically significant (Fig. 2). Cellulitis and boils were more prevalent in females than males ($p < 0.001$ and $p < 0.05$). It may be that cellulitis and boils have a pre-direction for specific population based upon demographic factors such as sex.

The morphological and biochemical characterization showed that all 100 samples isolated were identified as pathogenic *Staphylococcus aureus* for they yellow medium sized colonies of gram positive cocci with catalase and coagulase activity.

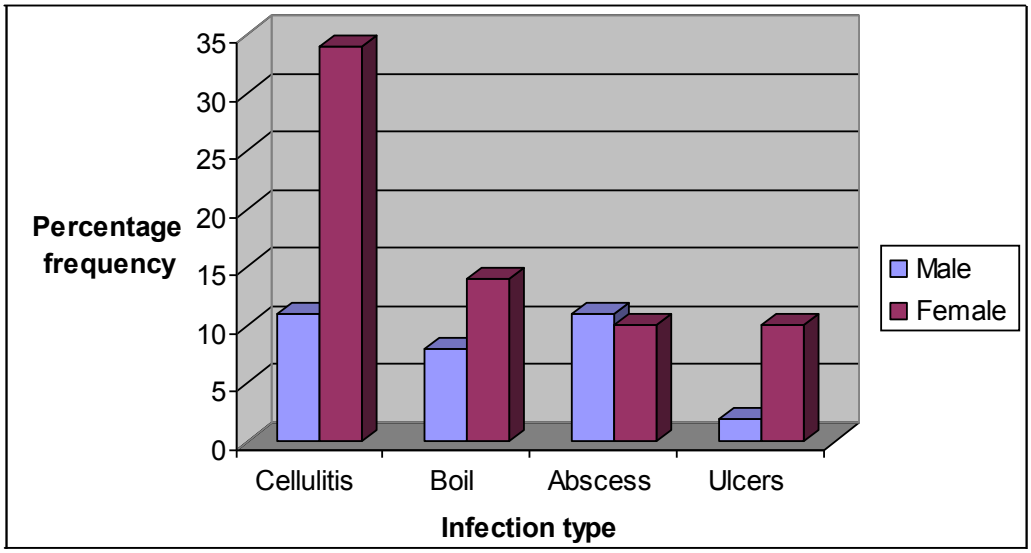

Fig. 1. Sex and infection of the patients from whom *Staphylococcus aureus* was isolated

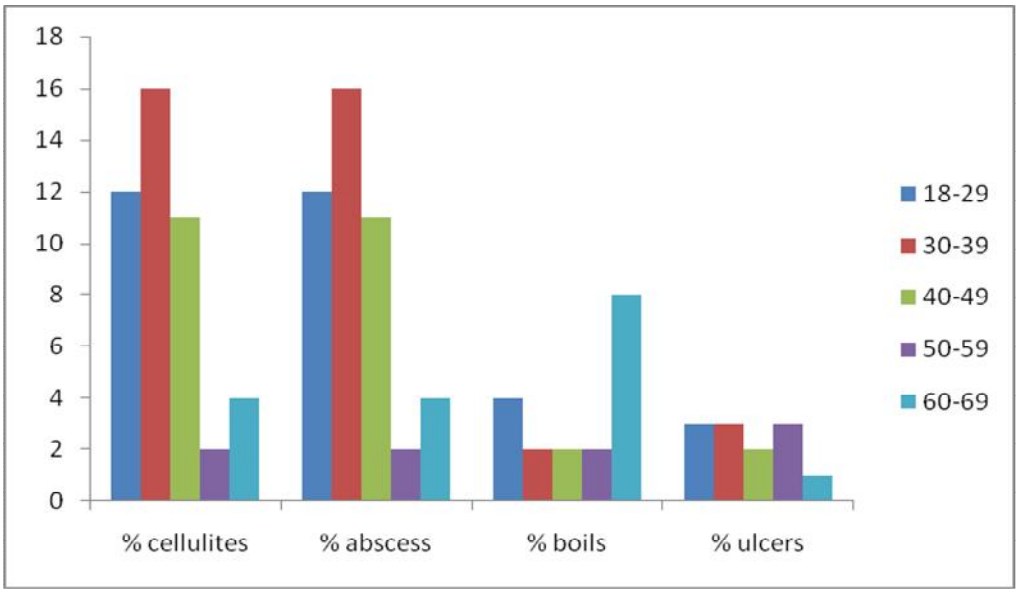

Fig. 2. Source of *Staphylococcus aureus* according to age of patients

As shown in Fig. 3 the *S. aureus* isolated was most sensitive to minocycline (83.3%) while the least sensitive was the aminoglycoside Gentamycin and the β-lactam methicilin (8.3%). Other antibiotics had varying sensitivities; 75% sensitivity to the β-lactam cefuroxime; 58.3% sensitivity to the quinolone ciprofloxacin; 50% to the bacteriostatic chloramphenicol; 41.6% sensitivity to the lincosamide lincomycin; 33.3% to the β-lactam morepenem; 25% the macrolide erythromycin as well as the β-lactams oxacilin, ampicillin, penicillin; trimethoprime sulfamethoxazole and amoxicillin / Clavulanic acid 16.6%. There was no relationship between resistance to the antibiotics tested and sex or age of the patient nor the source of the isolate be it cellulitis, abscess, boil or ulcer.

All isolates were identified as *Staphylococcus aureus*, the most commonly isolated human bacterial pathogen found in skin and soft-tissue infections as well as sepsis [30]. In 2009, Omuse [31] reported that 34% blood stream infections between 2003 and 2008 at the Aga Khan

University hospital in Nairobi, Kenya were due to *S. aureus*. Maina et al. [32] reported that of all skin and soft tissue infections from patients in Nairobi hospitals, 65% of the boil isolates were *S. aureus* and so were 23.3% of abscess cultures, 52.9% of cellulitis cultures and 44% of ulcer cultures.

Unlike the UK where only 2% of all *S. aureus* isolates were reported in 2001 to be sensitive to penicillin (13), in this study the prevalence of sensitive isolates was 25%. In respect to methicillin, the 91.7% methicillin resistance (8.3% susceptibility) observed in *Staphylococcus aureus* in this study is much higher than the 24.2% found in hospital associated *S. aureus* in Texas in 2001 [33]. Earlier studies in American hospitals had tracked the increase of methicillin resistant *S. aureus* from 2.4% in 1975; 29% in 1991; to 50% in 1997 [34,18]. In addition to resistance to methicillin, the resistance patterns of *S. aureus* observed in the present study to the aminoglycoside gentamycin (8.3% susceptibility), the β-lactam / clavulaninic acid combination amoxicillin / clavulanic acid (16.6% susceptibility) and other β-lactams morepenem (33.3% susceptibility) oxacilin, ampicilin, penicillin (25% susceptibility) can be compared with studies in varying countries. Boyee (1998) earlier reported that methicillin resistant bacteria are usually resistant to macrolides such as erythromycin, clindamycin, and frequently to gentamycin and ciprofloxacillin. Unlike this observation [35] however, the isolates in this study were moderately resistant to ciprofloxacillin (58.3%) as well as lincomycin a lincosamine similar to clindamycin (41.6%).

In 2009, 49%, 44%, 39% and 50% of isolates *S. aureus* from chronic wounds in a study in India were found to be resistant to ciprofloxacin, methicillin, oxacillin and vancomycin respectively [36]. Later in 2011, in another study [37], resistance of MRSA to penicillin (100%), amoxicillin / clavulanic acid (70.83%), erythromycin (66%), tetracycline (62.5%), cefotaxime (66%), cephalexin (58%) was reported. Unlike the study reported from India, all MRSA strains in the present study were sensitive to vancomycin.

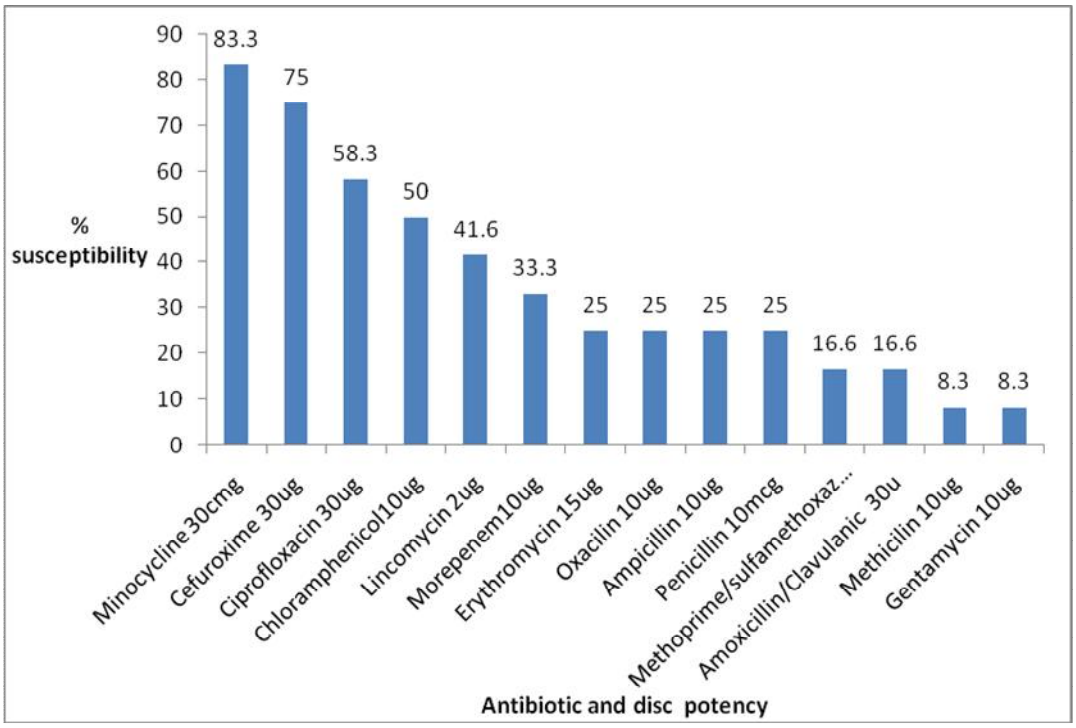

Fig. 3. Sensitivity profiles of *Staphylococcus aureus* isolates to selected antibiotics

There are limited studies on antibiotic resistance in Africa in general and Kenya in particular. Vlieghe et al. [38] systematically reviewed published literature on bacterial resistance in Central African countries; Cameroon, Chad, Gabon, São Tomé e Príncipe, Congo-Brazzaville, Democratic Republic of the Congo, Central African Republi, Angola and Equatorial Guinea. What was observed was methicillin-resistant *S. aureus*, high-level penicillin-resistant *Streptococcus pneumoniae* and extended-spectrum beta-lactamases among Gram-negative pathogens. In another analysis, Kimang'a [39], observed there were resistant bacteria to ampicillin, amoxycillin, chloramphenicol, streptomycin, spectinomycin, rrimethoprim / sulfamethoxazole, kanamycin, tetracycline and gentamycin in different parts of Africa.

An investigation of eight African hospitals noted comparatively higher levels of MDR MRSA in Kenya, Nigeria and Cameroon, with more than 60% of isolates exhibiting resistance to at least three antibiotics [21]. In Kenya, there was MRSA (28%) of all *S. aureus* tested in Nairobi city hospitals in 1997 with MRSA susceptibility to gentamicin and tetracycline extremely low (15 and 8% respectively). In addition, 2006, MRSA was found in 33 percent of *S. aureus* isolates in nosocomial infections at Kenyatta National Hospital in Kenya [40]. A study on the public health threat of bacterial infections and antibiotic resistance at Kenyatta National Hospital during the same year found the prevalence of MRSA to be 40% of all *S. aureus* infections [31]. Later, a study in 2009 reported that *S. aureus* isolated from healthcare facilities in western Kenya showed 25% resistance to commonly used antibiotics [41]. A situational analysis of 2011 documented that at the Kenyatta National hospital *S. aureus* resistance to methicillin was 40%, 85% to gentamycin and 94% to tetracycline [26]. Even more recently, a study of skin and soft tissue isolates from patients attending Nairobi hospitals reported in 2013 that 84.1% were MRSA [32].

In Kenya, the bacterial infections that contribute most to human disease are often those in which resistance is most evident to antibiotics most frequently used [26,42]. Increased use of antibiotics thus appears to correlate with increased antibiotic resistance. In Kenya, penicillins are the most prescribed antibiotic class at 31% [43,44]. Tetracyclines were the second most frequently consumed antibiotic class of which doxycycline was the most popular. Consumption of trimethoprime-sulfamethoxazole increased from 1997 to 1999 possibly due to its prophylactic use among an increasing HIV positive population. Aminoglycoside consumption also rose steadily after 1999, of which gentamicin accounted for 78 percent of the mean annual amount. While macrolide use remained stable after 1999, Cephalosporin use from 1999 increased in 2000 but later dropped in 2001.

Multi-drug resistant (MDR) *S. aureus* towards routinely used antibiotics as observed in this study is similar to Kimang'a, (39) who reported that 71% of *S. aureus* isolates from Kenya demonstrated multiple drug resistance. This phenomenon may be attributed to widespread availability for gentamycin, trimethoprime / sulfamethoxazole, and erythromycin (25%) observed in this study to be least effective are recommended for use as a first level antibiotic thus together with methicillin and oxacillin are available in the numerous health centres categorized as level 2 [45,14]. In addition, the most effective cefuroxime (75%) and chloramphenicol (50%) are only found as second level drugs in level 4 district hospitals. However, ciprofloxacin with bacterial sensitivity of 58.3% was also widely available as a first level drug in health centres and amoxicillin/clavulanic acid which is available as a first level drug only in district hospitals was not effective (16.6% sensitivity).

Other factors that could be associated with antibiotic resistance of the *S. aureaus* isolated could be improper use and patients not taking complete antibiotic course of treatment as prescribed. These habits are prevalent where there is dispensing of antibiotics over the counter (OTC) without the requisite prescriptive precautions. Such dispensing is particularly common among low and middle income such as found in the population likely to attend Ruiru District Hospital.

It has been noted that such indiscriminate use of antibiotics by over-the-counter access, not only gives rise to resistant strains of bacteria, but also provide opportunity for conjugative transfer of resistant plasmids to susceptible bacteria as has been observed in β-lactams and aminoglycosides [46-48]. It may be possible that there may be sub-standard and counterfeit antibiotics in the market that may also work towards development of resistance as in the

case reported in antimalarial drugs circulating in the markets of developing countries [49]. Furthermore, there are suggestions that antibiotic resistance observed in pathogens isolated from humans may emanate from the livestock where the most popular antibiotic use reported was tetracycline (55%) followed by sulfonamides (21%) aminoglycosides (7%), β-lactam (6%), quinolones (0.6 %) macrolides (0.2 percent) and tiamulin [43]. The association between sex of the patient with cellulitis and boils is difficult to explain except as it may relate to low socio-economic status and thus hygiene standards of the people in this area. Such associations of antibiotic resistance with socio-economic status have been observed in the previous studies [4,50].

4. CONCLUSION

This study has confirmed that as in others parts of Kenya, there is *S. aureus* isolated at Ruiru District hospital that are resistant to commonly used antibiotics. In this regard, gentamycin, chloramphenical, trimethoprime / sulfamethoxazole and ampicillin some of which are on the WHO and Kenyan essential drugs list are unlikely to offer help to the patients in Ruiru District hospital. Overuse, misuse and underuse through lack of access, inadequate dosing, poor adherence, and substandard antimicrobials may have contributed to its emergence and spread. Furthermore, comprehensive local data on usage and antibiotic resistance needed to give a true reflection of the situation in Kenya and to form policy such as the essential drug list is lacking [26].

There is thus need for further research including establishment of prevalence and resistance patterns of *Staphylococcus aureus* in other geographic locations of Kenya. Since the isolates were also resistant to trimethoprime / sulfamethoxazole and lincomycin, the antibiotic of choice would be minocycline and cefuroxime (75%) and ciprofloxacin (58.3%) as the next alternative drugs of choice. These should be used carefully to avoid development of resistant *Staphylococcus aureus* strains. it is thus recommended and the essential drug list be subsequently reviewed.

Noting that currently sensitive microbes could also develop resistance to effective antibiotics, there is need for strictly enforcing quality and regulatory strategies, regularly updating prescription guidelines and emphasizing continued medical and public education to alleviate irrational use of antibiotics by health care professional, unskilled practitioners and patients. Where resources and expertise is available, the ultimate would be determination of bacterial antibiotic sensitivity prior to selection of treatment after isolation from a patient.

ETHICAL APPROVAL

Ethical approval for the study was obtained from the Medical Superintendent at Ruiru District Hospital.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Fleming A. On the antibacterial action of cultures of a Penicillium, with special reference to their use in the isolation of *B. influenzae*. Brit J of Exp Path. 1929;10:226-236.
2. Mackie, McCarteney. Practical medical microbiology edited by G. Collee, A.G. Fraser, P. Marlton, A. Simmons 14th Edition; 1996.
3. Ogston A. On abscesses classics in infectious diseases. Rev Infect Dis. 1984; 6(1):122–28.
4. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections: A modification of CDC definitions of surgical wound infections. Infect Control Hosp Epidemiol. 1992;17:780–785.
5. Kluytmans J, van Belkum A, Verbrugh H. Nasal carriage of *Staphylococcus aureus*: Epidemiology, underlying mechanisms, and associated risks. Clin. Microbiol. Rev. 1997;10(3):505–20.
6. Cole AM, Tahk S, Oren A, Yoshioka D, Kim YH, Park A, Ganz T. Determinants of *Staphylococcus aureus* nasal carriage. Clin Diagn Lab Immunol. 2001;8(6):1064–9
7. Bowersox J. Experimental staph vaccine broadly protective in animal studies. NIH. Archived from the original on 5 May 2007; 1999.
8. Lederberg J, Lederberg E. Replica plating and indirect selection of bacterial mutants. J of Bact. 1952;63(3):399–406.
9. Stepanovic S, Dakic I, Djukic S, Lozuk B, Svabic-Vlahovic M. Surgical wound

infection associated with *Staphylococcus sciuri*. Scand J Infect Dis. 2002;34:685–686.
10. Carter AP, Clemons WM, Brodersen DE, Morgan-Warren RJ, Wimberly BT, Ramakrishnan V. Functional insights from the structure of the 30S ribosomal subunit and its interactions with antibiotics. Nature. 2000;407(6802):340–8.
11. Pantosti A, Sanchini A, Monaco M. Mechanisms of antibiotic resistance in *Staphylococcus aureus*. Future Microbiol. 2007;2(3):323–34.
12. Jevons MP. Celbenin-resistant staphylococci. Brit Med J 1961;1(5219):124–5.
13. Johnson AP, Aucken HM, Cavendish S, Ganner M, Wale MC, Warner M, Livermore DM. An, Cookson BD. Dominance of EMRSA-15 and -16 among MRSA causing nosocomial bacteraemia in the UK: Analysis of isolates from the European Antimicrobial Resistance Surveillance System (EARSS). J Antimicrob Chemother. 2001;48(1):143–4.
14. National Standard Treatment Guidelines Last revised in; 2010.
15. Hiramatsu K, Hanaki H, Ino T, Yabuta K, Oguri T, Tenover FC. Methicillin-resistant *Staphylococcus aureus* clinical strain with reduced vancomycin susceptibility. J Antimicrob Chemother. 1997;40(1):135–6.
16. Chang S, Sievert DM, Hageman JC, Boulton ML, Tenover FC, Downes FP, Shah S, Rudrik JT, Pupp GR, Brown WJ, Cardo D, Fridkin SK. Infection with vancomycin-resistant *Staphylococcus aureus* containing the vanA resistance gene. N Engl J Med. 2003;348(14):1342-7.
17. Menichetti F. Current and emerging serious Gram-positive infections. Clin Microbiol Infect. 2005;11(Suppl 3):22–8.
18. CDC. Centers for Disease Control and Prevention. "MRSA Surveillance" April 8, 2011
19. Nur YA, Vandenbergh MFQ, Yusuf MA. Van Belkum A, Verbrugh HA. Nasal carriage of multiresistant *Staphylococcus aureus* among health care workers and pediatric patients in two hospitals in Mogadishu, Somalia. Int. J. Infect. Dis. 1996;1:186–91.
20. Cisse MF, Sow AI, Adjovi DR, Samb A. Bacteriological study of purulent otitis media in children in CHU in the tropical zone. Arch Pediatr. 1995;2:29–33.
21. Kesah C, Ben Redjeb S, Odugbemi TO, Boye CS, Dosso M, Ndinya Achola JO. (2003) Prevalence of methicillin-resistant *Staphylococcus aureus* in eight African hospitals and Malta. Clin Microbiol Infect. 2003;9:153–156
22. Gakuu LN. Review of methicillin resistant *Staphylococcus aureus* with special reference to handling of surgical patients. East Afr Med J. 1997;74:198–202.
23. Croize J, Gialanella P, Monnet D, Okada J, Orsi A, Voss A, Merlin S. Improved identification of *Staphylococcus aureus* using a new agglutination test. Results of an international study. APMIS. 1993;101:487–491.
24. Wilkerson M, McAllister S, Miller JM, Heiter BJ, Bourbeau PP. Comparison of five agglutination tests for identification of *Staphylococcus aureus*. J Clin Microbiol. 1997;35:148–151.
25. Davis TE, Fuller D. Direct identification of bacterial isolates in blood cultures by using a DNA probe. J Clin Microbiol. 1991;29(10): 2193–2196.
26. Kariuki S. Situational analysis and recommendations. Antibiotic Use and Resistance in Kenya the Global Antibiotic Resistance Partnership-Kenya Working Group; 2011.
27. Fisher LD. Self-designing clinical trials. Stat. Med. 1998;17:1551-1562.
28. Bauer AW, Kirby WMM, Sherris JC, Turck M. Antibiotic susceptibility testing by standardized single disk method. Am. J. Clin. Pathol. 1966;45:493-496.
29. CLSI. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing; twentieth informational supplement. M100-S20. Wayne, PA: CLSI; 2010;29(3).
30. Lowy FD. *Staphylococcus aureus* infections. N. Engl. J. Med. 1998;339:520-532.
31. Omuse G. Methicilin Resistant *Staphylococcus aureus* (MRSA): The aga khan experience. global antibiotic resistance partnership inaugural meeting, Nairobi, Kenya. Unpublished Conference Proceedings; 2009.
32. Maina EK, Kiiyukia C, Wamae CN, Waiyaki PG, Kariuki S. Characterization of methicillin-resistant *Staphylococcus aureus* from skin and soft tissue infections in patients in Nairobi, Kenya. Int J Infect Dis. 2013; 17(2):115-9.
33. Diekema DJ, Pfaller MA, Schmitz FJ, Smayevsky J, Bell J, Jones RN. Survey of infections due to *Staphylococcus* species:

Frequency of occurrence and antimicrobial susceptibility of isolates collected in the United States, Canada, Latin America, Europe, and the Western Pacific region for the SENTRY Antimicrobial Surveillance Program, 1997–1999. Clin Infect Dis. 2001;32:114–132.

34. Panlilio AL, Culver DH, Gaynes RP, Banerjee S, Henderson TS, Tolson JS, Martone WJ. Methicillin-resistant *Staphylococcus aureus* in U.S. hospitals, 1975-1991. Infect Control Hosp Epidemiol. 1992;13(10):582–586
35. Boyee JM. Are the epidemiology and micro-biology of methicillin resistant *Staphylococcus aureus* changing. JAMA. 1998;8:623-624.
36. Karthy ES, Ranjitha P, Mohankumar A. Performance of CHROM Agar and Oxacillin Resistant Screening Agar Base Media for Detection of Methicillin Resistant Staphylococcus aureus (MRSA) from Chronic Wound Modern App Sci. 2009;3(5).
37. Shazia PS, Jyothsna K. Resistance among Isolates of *Staphylococcus aureus*: Antibiotic Sensitivity Pattern and Phage Typing Ann Biol Res. 2011;2(4):57-61.
38. Vlieghe E, Phoba MF, Tamfun JJ, Jacobs J. Antibiotic resistance among bacterial pathogens in Central Africa: A review of the published literature between 1955 and 2008. Int J Antimicrob Agents. 2009;34(4):295-303.
39. Kimang'a AN. A situational analysis of antimicrobial drug resistance in africa: are we losing the battle? Ethiop J Health Sci. 2012;22(2):135–143.
40. Ngumi ZWW. Nosocomial infections of Kenyatta National Hospital intensive care unit in Nairobi, Kenya. Dermatology. 2006; 212(Suppl 1):4-7.
41. Kakai R. Antibiotic resistance in Western Kenya, global antibiotic resistance partnership inaugural meeting, Nairobi, Kenya, Unpublished conference proceedings; 2009.
42. Karambu I. Abuse of 'prescription only drugs on the rise. Corporate news; 2011. Available:http://www,businessdailyafrica.com/corportate+news/-/539550/-/rlfi4x/-/index.html
43. Mitema ES, Kikuvi GM, Wegener HC, Stohr K. An assessment of antimicrobial consumption in food producing animals in Kenya. J of Vet Pharm Ther. 2001;24:385-390
44. Mitema ES. The role of unregulated sale and dispensing of antimicrobial agents on the development of antimicrobial resistance in developing countries. Antimicrobial resistance in developing counties, A Sosa, Dk Byarugaba, CF Amabile-Cuevaset al Springer; 2010.
45. Kenya Essential Medicines List Last revised in 2010.
46. Davies J. Inactivation of antibiotics and the dissemination of resistance genes. Science. 1994;264:375-378.
47. Waters VL. Conjugative transfer in the dissemination of beta-lactam and aminoglycoside resistance. Front. Biosci. 1999;4:416-439.
48. Grohmann E, Muth G, Espinosa M. Conjugative plasmid transfer in gram-positive *Bacteria.* Microbiol Mol Biol Rev. 2003;67(2): 277–301.
49. Amin A, Kokwaro G. Antimalarial Drug Quality in Africa. J Clin Pharm Ther. 2007; 32(5):429–440.
50. Vandenesch F, Naimi T, Enright MC, Lina G, Nimmo GR, Heffernan H, Liassine N, Bes M. Greenland T. Surgical wound infections. Inf Contr and Hosp Epi. 2003;13(10):606-8.

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