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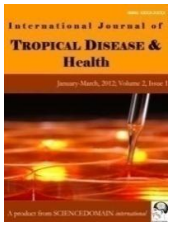

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Clinical Description, Bacterial Causes and the Association of HIV with Pyodermas Presenting at a Skin Clinic of a Tertiary Hospital in Rural South Western Uganda

Ogwang Edward[1], Joel Bazira[2*] and Mulyowa Grace[1]

[1]Department of Dermatology, Mbarara University of Science and Technology, Mbarara, Uganda.
[2]Department of Microbiology, Mbarara University of Science and Technology, Mbarara, Uganda.

Authors' contributions

Author OE participated in the planning of the study, conduct of the study, data entry and drafting of manuscript; author JB participated in the planning the study, supervised the laboratory work and drafting of manuscript, author MG participated in the planning of the study and drafting and critical review of the manuscript. All authors read and approved the final manuscript.

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ABSTRACT

Background: Diagnosis of pyoderma is clinical and antibiotic therapy in low resource setting is largely empirical. At MRRH Skin clinic repetitive visits by some patients with pyoderma led to speculation of antibiotic resistance. This indicated a need to survey the bacterial aetiology of pyoderma, their antibiotic susceptibility, clinical presentation, and look for association with HIV if any.
Methods: We consecutively enrolled 216 study participants with clinical diagnosis of pyodermas in a descriptive cross sectional study. Consenting participants had photographic documentation of pyoderma lesions, HIV counselling and testing. Skin swabs were cultured and sensitivity performed on the isolates. Pearson's chi-square and Fischer's exact test were performed to determine association between HIV status and bacterial causes of pyoderma.

**Corresponding author: Email: jbazira@gmail.com;*

Results: Non-bullous impetigo was the commonest clinical presentation, followed by folliculitis/perifolliculitis and lastly ecthyma. The major aetiological agents for the pyodermas were *Staphylococcus aureus* 77.78% and *Streptococcus pyogenes* 7.41% respectively. Other bacteria isolated were *Klebsiella* species 2.31% (5/216), *Proteus* species 1.85% (4/216) and *Pseudomonas* species 0.93% (2/216). Resistance of *Staphylococcus aureus* isolates to the anti-staphylococcal penicillin oxacillin was at 19.53% (33/169). The bacteria isolated from HIV positive participants were no different from that of HIV negative participants; for *Staphylococcus aureus* (82.35% versus 77.47% or p-value =0.527), *Streptococcus pyogenes* (5.88% versus 9.34% or p-value = 0.513) and for *Klebsiella* species (2.94% versus 2.75% or p-value = 0.647). Antibiotic susceptibility to most antibiotics was not significantly different between HIV positive and HIV negative participants except susceptibility to the cephalosporin cefalexin (p-value 0.039) which was much lower in the HIV positive pyoderma patients (83.87% versus 95.12%).
Conclusion: Non-bullous impetigo is the most common pyoderma in this population with *Staphylococcus aureus* and *Streptococcus pyogenes* being the major causes of pyoderma and pyoderma aetiology was not affected by HIV status.

Keywords: Pyoderma; HIV/AIDS; bacteria; Uganda; Staphylococcus aureus; Streptococcus pyogenes.

1. INTRODUCTION

Bacterial skin infections (pyodermas) are infections of the skin by pyogenic bacteria [1]. The primary pyoderma consists of staphylococcal and streptococcal impetigo contagiosa, surface and deep folliculitis, furuncles and carbuncles, Paronychia and ecthyma. Secondary bacterial infections complicate existing skin diseases. Examples are super infections of burns, eczema, scabies, ulcers, wounds and diseases such as varicella.

Pyodermas occur worldwide, the prevalence of which varies in different parts of the world [2]. Worldwide prevalence is not known. A 4% prevalence of pyodermas has been documented among primary school children in Tanzania [3].

In Uganda, the national prevalence is not known. Locally, in Mbarara pyodermas occur with an estimated annual incidence of 169/1000 cases (Unpublished data).

Therapy of pyodermas consists of incision and drainage of pus, use of antiseptics and antibiotics targeting the organisms [4]. Bacterial skin infections are encountered in most fields of clinical medicine. *Staphylococcus aureus* and group A streptococci are common invaders of eczematous, traumatised or immuno-compromised skin. These bacteria have several virulence factors that enable them attach to host tissues, evade immune response and spread by penetrating host tissue layers [5]. Advances in pharmacology have introduced a wide array of new antibiotics into the physician's armamentarium, but the rising incidence of bacterial resistance continues to be a problem [5].

The use of antibiotics in Mbarara just as in most developing countries is largely empirical. At Mbarara skin clinic antibiotic therapy of pyoderma is by empirical use of the penicillinase stable penicillin cloxacillin, erythromycin for penicillin allergic patients and a cephalosporin (cefixime and cephalexin) for persistent cases. It is however recommended that empiric therapy be based on local data of culture and sensitivity; and that specific therapy be given for every individual patient when microbiological services are available.

HIV prevalence among patients attending Mbarara skin clinic is at 20% (unpublished data). It is also known that HIV positive patients are at increased risk of bacterial skin and soft tissue infection especially with methicillin-resistant strains of *Staphylococcus aureus* [6]. The proportion of pyoderma cases because of MRSA infections at Mbarara University skin clinic was not known. Recommended choice of antibiotic therapy for MRSA skin infections in outpatient's setting is from clindamycin, linezolid, amoxicillin and doxycycline; while for in-patient settings would be from vancomycin, linezolid, daptomycin and clindamycin [7]. These are expensive antibiotics and not readily available at MRRH pharmacy.

It was therefore a necessity to carry out active surveillance to identify the bacteria causing pyoderma locally and their sensitivity to antibiotics. This study was set to describe the

clinical presentation of pyodermas in South-western Uganda, identify the causative organisms and their antibiotic susceptibility and determine if the bacterial causes differ by HIV status.

2. MATERIALS AND METHODS

The study was a descriptive cross sectional study conducted at Mbarara Regional Referral Hospital (MRRH) Skin clinic between January to June 2013. The study subjects were both adults and children with a clinical diagnosis of pyoderma.

2.1 Sampling Proceddure

The study subjects were consecutively enrolled until the target number was attained. The recruitment was through the dermatology clinic, of Mbarara Regional Referral Hospital. The staffs within the hospital were informed about the study to enhance referral of eligible participants to the researcher. To avoid the same participant being included more than once, each participant had a unique identification number and an identification sticker placed on their card as an indicator of participation.

2.2 Data Collection

The data was collected by use of a pretested questionnaire which included demographics, clinical type and location of pyoderma, HIV status, and a record of underlying dermatologic disease. Patients were asked about their HIV status and were required to provide evidence of having tested within the past three months. Counselling and testing was done for those who didnot know their HIV status and accepted to be tested. A pus swab was taken from the patients' skin for microbiological examination. The swab was transported to the laboratory in a special sterile container.

2.3 Laboratory Procedures

In the Laboratory, the pus was innoculated on McConckay and Chocolate Agar and incubated for 24 hours. Colony characteristics were recorded and gram staining was perfomed. Identification was performed using standard biochemical techiques. Drug susceptibility testing was performed using the Kirby Bauer Techniques according to CLSI guidelines [8]. Six different antibiotics were tested; penicillin, ampicillin, oxacillin, ciprofloxacin, erythromycin and cephalexin. The standard organisms used were S.aureus ATCC 25923 for gram positive bacteria and *E. coli* ATCC 25922 for gram negative bacteria according to protocol CLSI 100 [8]. The drug susceptibility test results were recorded as sensitive, intermediate or resistant as per zones of inhibition measured.

2.4 Quality Control

All questionnaires were pretested and administered by a trained research assistant. They were reviewed at the end of the day for any errors. All the microbiological techniques were carried out by qualified personel and were performed according to establised standard operating procedures. All media used for culture was freshly prepared and we used standard antibiotic discs. Standard Organisms were used during the drug susceptiblity testing.

2.5 Data Analysis

The data was double entered using Excel and analysed using STATA 11 (StataCorp, 2009). The data was then coded and cleaned to have a dataset ready for analysis. The statistical analysis involved a descriptive analysis of the demographics of the study subjects and of the bacterial isolates and susceptibility pattern to antibiotics. Data was summarised in form of tables, pie charts and frequency distributions. To determine the relationship between HIV and bacterial causes of pyodermas, the Pearsons chi square test was done for association between HIV status and *Staphylococcus aureus*; and between HIV status and *Streptococcus pyogenes*. The Fisher's exact test was done in circumstances where the number of events was small such as the determination of the association between HIV status and *Klebsiella* species as a bacterial cause of pyoderma.

3. RESULTS

3.1 Demographics of the Study Participants

The demographics of the study participants is summarised in the Table 1.

It could be seen in Table 1, that most of the pyoderma cases, 37.5% (81/216), occurred in children less than 10 years. The mean age of the study participants was 18.60 years and the

median 16.00 years. Males were more affected that females with a ratio of 1.3:1, and majority of the participants lived in rural areas. HIV prevalence among the patients with pyoderma was 15.74% (34/216).

Table 1. Demographics of the study population

Variable	Frequency
Age (yrs)	
0-10	37.50% (n=81)
11-20	25.90% (n=56)
21-30	16.20% (n=35)
31-40	10.20% (n=22)
41-50	6.50% (n=14)
>50	3.80% (n=8)
Tribe	
Munyankole	83.80% (n=181)
Muganda	5.60% (n=12)
Mukiiga	4.60% (n=10)
Other	6.00% (n=13)
Residential area	
Rural	62.90% (n=136)
Urban	37.10% (n=80)
Education	
None	38.40% (n=83)
Primary	31.50% (n=68)
Secondary	19.40% (n=42)
Tertiary	10.70% (n=23)
Sex	
Male	56.48% (n=122)
Female	43.52% (n=94)
HIV status	
Positive	15.74% (n=34)
Negative	84.26% (n=182)

3.2 Clinical Presentation of Pyoderma

From data in Table 2, it is conspicuous that the non-bullous form of impetigo is the most common pyoderma seen in the study population, followed by follicular pyodermas and lastly ecthyma. The erythema surrounding the lesions appeared as shiny bluish black discoloration and no overt redness was observed as in Caucasian skin.

It can be seen from the data in Table 3 that most pyodermas in the study population were primary and the existing dermatoses most commonly complicated with pyoderma were; eczema, tinea infections and scabies. These secondary dermatoses compromise the skin barrier making it easy for bacteria to gain entrance and cause infection

3.3 Bacteriology of Pyoderma at Mbarara Regional Referral Hospital

Staphylococcus aureus and *Streptococcus pyogenes* are the major causes of pyoderma.

Susceptibility test results in Table 4, shows that the broad spectrum cephalosporin cefalexin offers the best empiric therapy for both gram positive and gram negative bacteria. The choice for a narrow spectrum antibiotic for *S. aureus* infection would be oxacillin with sensitivity of 79.75% and that for *Streptococcus pyogenes* infection would be ampicillin with 94.74% sensitivity.

The MRSA prevalence was 7.1% (12/168), using resistance to oxacillin and cephalexin as indicators.

Table 2. Clinical types of pyodermas seen at mbarara skin clinic

Disease	Proportion	Lesion morphology
Impetigo	57.85% (n=125)	
Bullous impetigo	6.90% (n=15)	Widespread thin roofed cloudy bullae on erythematous base.
Non-bullous impetigo	50.90% (n=110)	Patches of crusted erosions and erythematous/skin colored papules
Folliculitis & Perifolliculitis	31.48% (n=68)	Skin colored and erythematous follicular papules and pustules. There was coalescing to form furuncles and carbuncles in some cases
Ecthyma	10.65% (n=23)	Well demarcated ulcers with surrounding erythema. Honey colored crusting over the ulcers

Table 3. Occurrence of primary pyoderma and dermatoses complicated by pyoderma (secondary pyoderma)

Pyoderma	Proportion
Primary pyoderma	67.59% (n=146)
Secondary pyoderma	32.41% (n=70)
Eczema	15.28% (n=33)
Tinea infections	5.09% (n=11)
Scabies	4.68% (n=10)
Insect bite reactions	2.78% (n=6)
Drug eruptions	2.78% (n=6)
Keloids	0.93% (n=2)
Papular Pruritic eruption	0.93% (n=2)

3.4 HIV and Bacteriology of Pyoderma

The prevalence of HIV among the patients with Pyoderma was 15.74% (34/216). Half of the HIV positive patients presented with Impetigo, followed by Ecthyma with 29.41% (10/34) and follicular pyodermas accounted for 20.59% (7/34).

The bacteria isolated from HIV positive patients were; *Staphylococcus aureus* 82.35% (28/34), *Streptococcus pyogenes* 5.88% (2/34) and Klebsiella species 2.94% (1/34).

There was no association between the type of bacteria and HIV status. Antibiotic susceptibility was significantly different for the cephalosporin cefalexin in which lower susceptibility was registered among the HIV positive pyoderma patients (chi square 6.5026, p-value 0.039). A detailed report of the association between HIV status and bacteriology of pyodermas is shown in Table 5.

Table 4. Antibiotic sensitivity of bacteria isolated from Pyoderma lesions in MRRH

Antibiotic	Sensitivity	Resistance
Oxacillin		
	Staphylococcus aureus 79.75% (n=130)	20.25% (n=33)
	Streptococcus pyogenes 89.47% (n=17)	10.53% (n=2)
ciprofloxacin	*Staphylococcus aureus*	
	82.21% (n=134)	16.56% (n=27)
	Streptococcus pyogenes 63.16% (n=12)	31.58% (n=6)
Erythromycin		
	Staphylococcus aureus 69.33% (n=113)	29.45% (n=48)
	Streptococcus pyogenes 73.68% (n=14)	26.32% (n=5)
Cefalexin		
	Staphylococcus aureus 92.02% (n=150)	7.36% (n=12)
	Streptococcus pyogenes 100.00% (n=19)	
Ampicillin		
	Staphylococcus aureus 20.86% (n=34)	79.14% (n=129)
	Streptococcus pyogenes 94.74% (n=18)	5.26% (n=1)
Penicillin		
	Staphylococcus aureus 12.27% (n=20)	87.73% (n=143)
	Streptococcus pyogenes 94.74% (n=18)	5.26% (n=1)
Trimethoprime-Sulphamethoxazole		
	Staphylococcus aureus 12.00% (n=6)	88.00% (n=44)
	Streptococcus pyogenes	100.00% (n=5)

Antibiotic	Sensitivity	Resistance
Ciprofloxacin		
	E. coli 100.00% (n=2)	
	Klebsiella species 60.00% (n=3)	40.00% (n=2)
	Pseudomonas species 50.00% (n=1)	50.00% (n=1)
	Proteus species 100.00% (n=4)	
Cefalexin		
	E.coli 100.00% (n=2)	
	Klebsiella species 100.00% (n=5)	
	Pseudomonas species 100.00% (n=2)	
	Proteus species 100.00% (n=4)	
Ampicillin		
	E. coli 50.00% (n=1)	50.00% (n=1)
	Klebsiella species	100.00% (n=5)
	Pseudomonas species	100.00% (n=2)
	Proteus species 25.00% (n=1)	75.00% (n=3)

Table 5. Association between HIV status and bacterial causes of pyoderma

Bacteria isolated	Proportion among HIV positive	Proportion among HIV negative	P-value
S. aureus	82.35% (n=28)	77.47% (n=141)	0.527
S. pyogenes.	5.88% (n=2)	9.34% (n=17)	0.513
Klebsiella species	2.94% (n=1)	2.74% (n=5)	0.647

4. DISCUSSION

This study set out to describe the clinical presentation of pyodermas, determine the bacterial aetiology & antibiotic susceptibility; and examine its association with HIV at Mbarara Hospital in South-western Uganda.

4.1 Clinical Presentation of Pyoderma

The commonest clinical type of pyodermas was impetigo affecting 57.9% (n=125) of study participants, followed by folliculitis & perifolliculitis at 31.5% (n=68) and lastly ecthyma which is considered an ulcerated form of non-bullous impetigo accounted for 10.6% (n=23). Non-bullous Impetigo being the most common pyoderma is in agreement with findings of Tan et al. [9] at a tertiary dermatology facility in Singapore. Similarly in Fiji, Steer et al. [10] found impetigo to be the most common bacterial skin infection. Non-bullous impetigo caused by *S. aureus* was also the most prevalent pyoderma in a north Indian population [11]. Knowing the clinical presentation has an impact on management and most importantly on expected complications in a patient suffering from pyodermas. For example non-bullous impetigo caused by *Streptococcus pyogenes* (serotypes 1, 4, 12, 25 and 49) can result in acute post streptococcal glomerulonephritis (APSG). Hypertrophic scars and Keloids can result from ulcerative lesions [12].

In our study impetigo was more common in children. Just as in most parts of the world impetigo is more common in children especially of school going age. The contagious nature of impetigo explains why it is more common in school children.

Contrary to the type of lesion seen in Caucasians where erythema is seen as red, the erythema in the indigenous population studied appeared as shiny bluish-black hue. The non-bullous impetigo lesions therefore presented as bluish-black or skin colored papules with subsequent vesiculation, crusting and ulceration. This is important for any physician in recognising erythema in the people of skin of color.

The most of pyodermas lesions in the population studied presented on the lower limbs. The

presentation on the lower limbs suggest initial trauma to the skin as a predisposition to colonisation by bacteria. Majority of the patients were also peasants from rural south-western Uganda who easily get trauma to their limbs while gardening. Likewise, looking at some of the underlying dermatoses for secondary pyodermas like tinea, scabies, these represent the "dermatoses of poverty" which will only decrease with improved living standards.

4.2 Bacteriology of Pyoderma

The most prevalent bacteria isolated from the study participants is *S. aureus* 75.78% (n=168) and *streptococcus pyogenes* 8.52% (n=16). This is in agreement with Kakar et al. [13] who had similar findings. Mixed infections were found in isolates from 2.3% (5/216) of the study participants. The other bacterial isolates include; *Klebsiella* species, *proteus* species, *Escherichia coli* and *pseudomonas* species. These, as did Brook et al. [14] elucidates the polymicrobial aetiology of pyodermas especially in secondary pyodermas. Brook et al. [14] however did not find Klebsiella species which was found in this study. However in addition to the above bacteria Brook et al. [14] isolated bacteroides species, pigmented prevotella, porphryomona species and fusobacterium species which this study did not find.

As noted 84.47% (184/216) of the study participants had *Staphylococcus aureus* and/or *Streptococcus pyogenes* isolated from their pyodermas lesions. Empiric antibiotic therapy would therefore aim to cover both these micro organisms.

Resistance to oxacillin a beta-lactam antibiotic and marker of Methicillin-resistant *Staphylococcus aureus* was found in 19.53% (n=33) of the staph aureus isolates. Using oxacillin and cephalexin resistance to screen for MRSA, prevalence for MRSA was 7.1% (12/168). Since the isolates were from patients in an out-patient's setting, this conforms to the rate of community-acquired MRSA in this community. However CA-MRSA is noteworthy in their tendency to be susceptible to most beta-lactam antibiotics, and is most often only resistant to Methicillin [15]. This finding suggests presence of community acquired strains of MRSA. Nyambura et al. [16] found MRSA prevalence of 15% in Tanzania. These are low prevalence compared to findings in developed countries where more than 25% of *Staphylococcus aureus* strains have been found to be MRSA. Prevalence data from South Africa of MRSA ranges from 4-74% [17]. The marked variations in prevalence of MRSA between regions suggest evolution of resistant clones. With increasing prevalence of such strains of MRSA in the community there will be increase in morbidity associated with diseases it causes and also on health expenditure due costs incurred on newer medication.

4.3 HIV and Bacteriology of Pyoderma at MRRH

The proportion of study participants who were HIV positive is 15.74% (34/216). This prevalence is much higher than the national HIV prevalence of 7.3% [18]. This suggests there is an increase in occurrence of HIV among patients with pyoderma; however that is not the focus of this study.

Our study shows that the aetiology of Pyoderma is not affected by HIV status, as opposed to Bar et al. [19] who found increased *E. coli* associated pyoderma in HIV positive patients.

Susceptibility to most antibiotics was not significantly different between HIV positive and HIV negative participants; except susceptibility to the cephalosporin cefalexin which was much lower among HIV positive participants (p-value 0.039). The susceptibility of *Staphylococcus aureus* to oxacillin, an anti-staphylococcal penicillin was not significantly different between HIV positive and HIV negative participants (p-value 0.207). MRSA prevalence (using resistance to oxacillin and cephalexin as a screen) was higher in HIV positive patients with pyoderma (16.13% versus 4.27%). This is in agreement with Ramsetty et al. [20] and Bar et al. [19] who registered higher MRSA among HIV positive patients. MRSA infections have a huge socioeconomic impact in terms of morbidity and increased costs of treatment.

5. CONCLUSION

The study has shown that non-bullous form of impetigo was the most common clinical presentation, followed by follicular pyoderma and lastly ecthyma.

Staphylococcus aureus and/or *Streptococcus pyogenes* were the most frequent isolate from pyodermas lesions in patients attending MRRH skin clinic. There was no significant difference in bacterial isolates between HIV positive and HIV

negative participants; and antibiotic susceptibility to cefalexin was much lower in the HIV positive group.

CONSENT

Written informed consent for participation in the study was obtained from participants and in case the participants were children consent was sought from their parents or care takers

ETHICAL APPROVAL

The study was approved by the Institutional review committee of Mbarara University of Science and Technology. Counselling services were provided to participants before and after HIV testing. Participants who were found to be HIV positive upon testing were referred to the HIV clinic Mbarara Regional Referral Hospital for further care.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Burgdorf W.H.C, et al. Staphylococcal and streptococcal diseases, braun-falco's dermatology 3rd edition springer medicin verlag Heidelberg. 2009:114-139.
2. Bernard P. Management of common bacterial infections of the skin. Curr Opin infect Dis. 2008;(2):122-8.
3. Komba EV, Mgonda YM. The spectrum of dermatological disorders among primary school children in Dar es Salaam. BMC Public Health. 2010;16(10):765.
4. Jones H. Systemic antibiotic treatment of skin, skin structure and soft tissue infections in the outpatient setting. Adv Skin wound care. 2012;25(3):132-40.
5. Webb SAR, Kahler CM. Bench to bedside review: Bacterial virulence and subversion of host defences. Crit Care. 2008;12:234.
6. Kyle J Popovich, Robert A Weinstein. Community Associated Methicillin-Resistant *Staphylococcus aureus* and HIV: Intersecting epidemic. Clin Infect Dis 2010;50(7):979-987.
7. John G Bartlett. Treating MRSA skin and soft tissue infections- new guidelines. Medscape HIV/AIDS expert reviews; 2011. Accessed august 2013.
   Available:www.medscape.com/viewarticle/741303_2
8. Hudzicki Jan. Kirby-bauer disk diffusion susceptibility test protocol; 2009. Available online at:www.microbelibrary.org/component/resource/laboratory-test/3189-kirby-bauer-disk-diffusion-susceptibility-test-protocol
9. Tan HH, Tag YK, et al. Bacterial skin infections at a tertiary dermatologic care. Singapore Med J. 1998;39(8):353-6.
10. Steer AC, Jenney AWJ, Kado J, Batzloff MR, La Vincente S, et al. High burden of impetigo and scabies in a tropical country. PLoS Negl Trop Dis. 2009;3(6):e467. DOI:10.1371/journal.pntd.0000467.
11. Suresh K Malhotra, Sita M alhotra, Gurit S Dhaliwal. Bacteriological study of pyodermas in a Tertiary care dermatological center. Indian J Dermatol. 2012;57(5):358-361.
12. Cole C, Gazewood J. Diagnosis and treatment of impetigo. Am Fam Physician. Mar 15. 2007;75(6):859-64.
13. Kakar N, Kumar V et al. Clinico-bacteriological study of pyoderma in children. J Dermatol. 1999;26(5):283-93.
14. Brook I, Frazier EH, Yeager JK. Microbiology of infected atopic dermatitis. Int J Dermatol. 1996;35(11):791-3.
15. HF Chambers. The changing epidemiology of Staph aureus. Emerg Infect Dis 2001;7:178-182.
16. Nyambura M oremi, Stephen E Mshana, et al. Predominance of methicillin-resistant *Staphylococcus aureus* ST88 and new ST1797 causing wound infections and abscesses. J Infect Dec Ctries. 2012;6(8): 620-625.
17. Jan MB, Turnidge JD, SENTRY APAC Participants. High prevalence of oxacillin-resistant *Staphylococcus aureus* isolates from hospitalised patients in Asia-Pacific and South Africa: Results from SENTRY Antimicrobial Surveillance Program 1998-1999. Antimicrob Agents and chemother. 2002;46:879-881.
18. Ministry of Health(MOH) Uganda. Press release of key results of the 2011 Uganda AIDS Indicator Survey. Ministry of Health Uganda; 2011.

19. Bar A, Hantschke D, Mirmohammad-sadegh A, Hengge UR. Spectrum of bacterial isolates in HIV positive patients with skin and soft tissue Infections: Emergence of methicillin-resistant staphylococci. AIDS. 2003;17(8):1253-1256

20. Ramsetty SK, Stuart LL, Blake RT, Parsons CH, Salgado CD. Risks for Methicillin-resistant *Staphylococcus aureus* colonisation or infection among patients with HIV Infection. HIV med. 2010;11(6):389-394.

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