IOSR Journal Of Pharmacy *www.iosrphr.org*
(e)-ISSN: 2250-3013, (p)-ISSN: 2319-4219
Volume 6, Issue 10 Version. 2 (Oct 2016), PP. 42-54

Topical Use of Silver Sulfadiazine In The Prevention of Burn Wound Infection At University Teaching Hospital, Lusaka, Zambia

MWAPE MATILDAH*, Bipharm, MD, Yassa Pierre**,Mmed, PhD, Munogo L.T***, Bpharm, MD,PhD*,Christopher Sakala, Bpharm, MD,PhD**, Myer Glickman****,PhD.

Lusaka Apex Medical University University of Kamina at Republic Democratic of Congo*
****University of Zambia**** NHS UK*

ABSTRACT:- The primary challenge in dealing with the colonization of burn wound is to find a mean of prophylaxis that minimizes the establishment of resistant organisms at the burn wound site. The use of topical prophylaxis is important in reducing the wound's microbial load thereby facilitating wound healing.
Objective:- The aim of this clinical research was to find out if the use of prophylactic topical SSD prevents the onset of bacteria colonization and wound infection in burn patients at University Teaching Hospital.
Methods:- The study design was a quasi-experimental using a single group pre- test post- test design. A single group design demonstrated that a treatment lead to a particular effect in the presence and absence of the SSD. Observation and self-administered tool were used to capture the variable such as Age, gender, TBSA, burn wound swabs post burn, culture and sensitivity test results. The study was conducted over five months.
The study was conducted in the female surgical wards in the department of surgery at the University Teaching Hospital. A sample size of 46 patients was studied: 15 had minor burns, 16 had moderate burns and 15 had severe burns.Outcome measures were pus swabs in presence of SSD and absence of SSD, culture and sensitivity results in presence of SSD and absence of SSD.
Results:- The study revealed that SSD has a role in the prevention of bacteria on the burn wound colonization at UTH. We found that of the 46 burn patients studied 42 (91.3%) has positive swabs with no SSD and 32 (69.57%) had positive pus swabs with SSD. The study also found out that *staphylococcus spp* most predominant colonizing organism isolated, 31% and 37% with SSD and with no SSD respectively.
Conclusion:- It can be concluded from the finding of this study that SSD has a role in prevention of bacterial wound colonization in burn patients at UTH.

I. INTRODUCTION

Burn injuries are a common form of trauma worldwide and Infections in burn patients continue to be the primary source of morbidity and mortality. According to the data provided by the American Burn Association 2008, more than 450,000 American people sustained burn injuries. It is also estimated that 5000 deaths are related to burn injury each year with a 1 in 70 chance of an American being burned seriously enough to require hospitalization in his /her lifetime. (Herdon DN, 4th Ed, 1996)A burn is a type of injury to flesh or skin caused by heat, electricity, chemical, friction or radiation (Herndon .4th Ed.1996). According to the American burn association, 2012**)** burn severityis classified as follow (table1):

Table 1. American Burn Association severity classification

Minor	**Moderate**	**Major**
Adult <10% TBSA	Adult 10-20% TBSA	Adult >20% TBSA
Young or old < 5% TBSA	Young or old 5-10% TBSA	Young or old >10% TBSA

The management of microbial contamination of burns is a routine requirement; this practice has led to the development of a variety of therapeutic agent for topical use. (Monafo WW 1990). The goal of topical agent in burn wound management is to reduce the onset and density of bacterial contamination which usually occur by the first week after injury and thereby preventing invasive wound infection. (Lawrence J.C 1992).In the burn arena, before the advent of topical antibacterial agents the overall mortality rate in a typical burn would be reported at 38% to 45 %. However after the use of topical antimicrobial therapy the overall mortality was reduced to 14% to 25 %.(Hangnga ,Vu et .al 2002).Silver sulfadiazine cream has been the standard of treatment for burns over the past decades. Silver is toxic to microbes, it poisons the respiratory enzymes and components

of the microbial electron transport system resulting in the impairment of microbes DNA functions. (O'sulivan S, et al. 2001). Topical Silver sulfadiazine in the management of burns may be defined as the external application of silver sulfadiazine cream started immediately after injury with the aim of preventing bacterial colonisation the burn wound. This is usually done before the results for wound swabs and cultures are available. SSD has a wide spectrum of bactericidal activity against both gram-positive and gram-negative organisms. Organisms that are susceptible to topical silver sulphadiazine include Staphylococcus aureus, S. epidermidis, beta-hemolytic streptococci, Klebsiella, Escherichia coli, Enterobacter (including E. cloacae), Citrobacter, Proteus, Pseudomonas, Morganella morganii, Providencia, Serratia, and Candida albicans (Shuck J, et al 1975).

The standard of care at UTH involves the use of isotonic saline wet soak therapy. Isotonic saline wet soaks therapy (ISWST) is the use of 0.9% NaCl to irrigate the wound and provide a moist environment allowing the dead cells on the wound surface to swell up and eventually peel off**.** ISWST does not prevent the wound from bacterial contamination (*Winter G.D. 1963)* The use of topical prophylaxis is important in reducing the wound's microbial load thereby facilitating wound healing (Madson SM, et.al 1996).The use of SSD in the treatment of burn at UTH is not as a standard of care as Isotonic saline wet soaks. Its use on burn depends on whether the patient can afford it or not. This has resulted in SSD not being used routinely in the management of burn patients.A sequential analysis of burn wound swabs carried out at the University Teaching Hospital, Lusaka by Lameck ,Z 2000 revealed that the common organisms isolated in the first week from the wound swabs were staphylococcus aureus 68%, streptococcus pyogens (6.25%) and pseudomonas aeuroginosa (6.25%).The deficiency in this study is that it did not relate to whether the SSD topical was used or not.

This study was therefore designed to find out if the early initiation (post burn day 0) of topical Silver sulfadiazine will be effective in preventing or delaying the onset of bacterial wound colonization on the burn wound surfaces in burn patients at UTH.

II. METHODOLOGY

Introduction:

The methodology that was chosen for this study was based on objective 1 and 2 (section 1.4)

2.1 Research design

The research design chosen for this enquiry was based on the specific objective and the questions to be answered in this research. The study design that best fitted this inquiry is the quasi experimental, controlled intervention study using interrupted time series analysis; the intervention consisted of using topical silver sulfadiazine cream (Bradley E. H ,2011, 2nd edition). The type of quasi experimental design that was be used is singe group pretest-posttest design:

Group Time -----

Single Group Tx Obs 1 ----- Obs 2

In the singe group pretest-posttest design –sample design an intervention (TX) was implemented, and post-test measurements were made and then Tx was removed then post-test measurements were made. This type of Quasi experimental design was chosen for this study because it is an on – off design that shows in a single group that a treatment lead to a particular effect in the presence and in the absence of the experimental variable.

2.2 Sampling Technique

Stratified randomisation was used as the sampling technique in the study. The study population of 46 participants was broken into strata, and each strata was allocated the following participants :15 participants for minor burns , 16 participants for moderate burns and 15 participants for severe burns and the participants in each group were systematic randomly selected (using sampling interval of 2) within each group to ensure a good representation of the three groups**.** This type of sampling ensured that the results were more accurate and participants were chosen on the basis of chance and biasness was eliminatedfrom the study.

2.3 Study site and population

The study site for this research was the University Teaching Hospital at the G-block wards G01, G02, G12, G21, G22, Male surgical wards and female surgical ward.

Target population – in this study the target population included burn patients

Study population- included burn patients day 0 post burn or old burns (more than 24hrs) with minor, medium and major sized burns.

2.4 Sample size determination

The sample size for this non randomized study was calculated in order for the study to have a power of the sample and quantify the association. the following formula was used to estimate the number of study participants (N) needed in this study .The N calculated was not multiplied by 2 because the study only had one single group that received the intervention (Nick et al .,2009 **):**

The sample size for each of the two groups, N was given by

$$N = \frac{K\{P_1(1-P_1) + P_2(1-p_2)\}}{(P_1-P_2)^2}$$

- P1= expected population to develop bacterial colonization control group =60 percent (based on the surgery department records)
- P2= expected population to develop bacterial colonization from experimental group=30 percent
- alpha= 0.05= significant level ***(p***)=5%
- beta= 0.2= power of study = 80%
- K = constant which is a function of beta and alpha=7.8

$$n = \frac{7.8\{0.6(1-0.6)+ 0.3(1-0.3)\}}{(0.6-0.3)^2}$$

$$= \frac{7.8(0.24+0.16)}{0.09} = \frac{7.8(0.40)}{0.09} = \frac{3.12}{0.09} = 39$$

Loss to follow-up at 30%= n x 1/ 1-0.3 =39 x1.25=55.7= 60 participants

Only 46 was studied were studied because the loss to follow up was adjusted to 10% thus gave the total number of participant equals 43 = 45 participants but in our study 46 participants were studied.

2.5 Inclusion-exclusion criteria

2.5.1 Inclusion

- Admitted with medium sized burns or minor sized burns or major sized burn
- Consented by patient (above age of 18)
- consented by guardian or parents (below age of 18)
- Clean wounds (if dirty wounds were cleaned)
- patients with less than 24 hours burn injury were included without doing wound swab
- Old burns or more than 24 hours burns were only be included if a wound swab is done before intervention phase and if no topical antibiotic was used

2.5.2 Exclusion

- Patients sensitive to silver or sulfa drugs
- Any patient developing clinical evidence of wound infection
- Pregnant and nursing mothers
- Diabetic patients with burns (poor control of glucose is a predisposing factor to infection)

2.6 Data collection

A structured self-administered tool with demographic variable and a burn follow up sheet was used (Appendix C). The data collected from observational and a self administered tool was used to capture the variables such as age, gender, TBSA affected by the burns, burn wound swabs post burn and culture and sensitivity test results.
Surface wound swabsfor bacteria isolation and culture and sensitivity tests were collected as follow:

- 3rd day of post burn (OBS 1)
- 6th day of post burn (OBS 2)

There were two phases in this study, therefore two post test measurements (at day 3,7) for burn wound swabs results and culture and sensitivity tests were recorded in the burn wound follow up sheet. The daily entry of the data was done by the researcher on the burn wound follow up sheet according to the phases of the study.
For fresh burn (burns less than 24hrs old) on presentation the burns were thoroughly washed and a 1mm thin layer of 1 % SSD cream was applied and was changed every after 8 hourly every day. After 3 days of SSD use a pus swab was taken and was considered as OBS 1.From the 4th day up to 6th day only wet soaks were used and a second pus swab was taken at the end of the 6th day and considered as OBS 2.For old burns (burns more than 24hrs old) only burn where no topical antibiotic cream had been applied were considered. Before applying SSD a swab was taken and was considered as OBS 2 and then the burns were washed and a thin layer of 1mm was applied on the burns and changed every 8 hourly everyday for three days. After three days of SSD use a pus was taken and was considered as OBS 1All Surface wound swabs were taken using pus swabs lot no 110308 and introduced in a tube containing amies and charcoal medium. A swab was taken by rubbing a swab on the entire burnt surface and a swab was introduced in the tube containing Amies and charcoal mediums then taken to the laboratory for culture and sensitivity. One type of the brand of SSD cream (*Flazine*^{Tm} *Mumbai,India,*) was used throughout the research to avoid issues of bioequivalence.

2.6.1 Surface wound swabs processing

Day 1:

- The pus swabs were inoculated on the blood agar, chocolate and mackonkey.

Day 2:

- Growths were checked on all the three plates mentioned above.
- If there lactose fomenters or non lactose fermenters were grown on the mackonkey the biochemicals with citrate,SIM (triple sugar iron and LIA (lysine iron agar) were done.
- To do susceptibility and sensitivity testing McFarland's standard on muller Hintoin agar was used
- If either streptococcus or staphylococcus were suspected, the culture and drug susceptibility testing was done using catalase test
- If the catalase test was +ve the staphylococcus was the isolate and if the catalase test was –ve then the streptococcus was isolated.

Day 3:Name of the organism and drug susceptibility were reported.

The following classification of the data was used: types of bacterial that colonize the burn wound in presence of the intervention , types of bacterial that colonize the burn wound in the absence of the intervention , extent of bacterial colonization in presence of the intervention , extent of bacterial colonization in the absence of the intervention , relationship between the culture and resistant profile of the microorganism in the presence of the intervention and in absence of the intervention and relationship between the culture and sensitivity profile of organism in the presence of the intervention phases and absence of intervention phases

2.7 Data analysis

The was data collected and coded on sheet and kept in the electronic software .The analysis of the data was based on the objective and was entered using Stastical Package for Social Sciences Version 16 then converted to STATA 10 and analysed using both STATA 10 and SPSS vs. 16 then presented using Microsoft excel.

2.8 Research data analysis plan for the variables

Variables were selected and defined (table 2)

Table 2: Variables with their definitions and scales of measurements

VARIABLE NAME	DEFINATION OF VARIABLES	SCALE OF MEASUREMENT	STATISTICAL METHOD
Dependent variable (bacterial colonisation present Vs not present)	Pus swab positive or negative	Categorical	Frequency
Independent variable-SSD applied on the burn wound from day 0-day 3	Outcome (observations for bacterial colonisation Day0-3)	Continuous variable	Mean
Independent variable- SSD not applied on the bun wound from day 4-7	Observations for bacterial colonisation day 4-7	Continuous variable	Mean

A bivariable analysis was used to analyse the data because the study had two independent variables (phases). From the table above bacterial colonisation as the dependent variable is a categorical variable therefore the association between the extent of bacterial colonisation in presence of intervention and absence of intervention was done using a Pearson chi-square test.

2.9 ETHICAL CONSIDERATION

Ethical issues are an integral component of this study as it among other things involves human being. To address this issue, participation in this study was purely voluntary and participants were free to withdraw at any point without any consequences. Approval was sought from the UNZA Biomedical Research Ethical Committee (UNZABREC). Permission to conduct the research was granted by UNZABREC .The following specific standards were upheld:

- In order to maintain high level of confidentiality, no names were used in this study instead ID numbers will be used and for data analysis ID numbers will be coded.

- The participants were given education on signs and symptoms of allergic reaction to the SSD. In stances where the participant suffered from allergic reaction to the SSD, a medical doctor was on hand to do assessment and therapy was given.
- In instances where a participant developed clinical evidence of wound infection during the study period, a medical doctor was on hand to do assessment and therapy was given.
- The samples were only used for the purpose of the study and after the study the sample have been discarded.
- The principal investigator discussed and agreed with the supervisors with regard to research data ownership.
- To ensure sound ethical consideration, the researcher ensured that the participants were fully informed about the purpose, methods and intended possible use of the research and a written informed consent was obtained from participants or guardian of the participants that were included in the study.

III. RESULTS

Introduction

This chapter gives data of the characteristics:, types of bacterial that colonize the burn wound in presence of the intervention , types of bacterial that colonize the burn wound in the absence of the intervention , extent of bacterial colonization in presence of the intervention , extent of bacterial colonization in the absence of the intervention , relationship between the culture and resistant profile of the microorganism in the presence of the intervention and in absence of the intervention and relationship between the culture and sensitivity profile of organism in the presence of the intervention phases and absence of intervention phases . This data was studied and analyzed repeatedly in order to develop the answer for the research questions. Data collection and analysis resulted in vivid understandings of the research.The study population was 46 burn patients. The study population of 46 participants was broken into strata, and each strata was allocated the following participants :15 participants for minor burns , 16 participants for moderate burns and 15 participants for severe burns. The participants were chosen using Stratified randomisation and the participants in each group were systematic randomly selected (using sampling interval of 2).The following research questions bounded and guided my research:

1. What are the types of bacterial that colonize the burn wound in presence of the intervention and in the absence of the intervention in burn patients at UTH?
2. What is the extent of bacterial colonization in presence of the intervention and in the absence of the intervention in burn patients at UTH?
3. What is the relationship between the culture and resistant profile of the microorganism in the presence of the intervention and in absence of the intervention in burn patients at UTH?
4. What is the relationship between the culture and sensitivity profile of organism between the intervention phases and the non-intervention phases in burn patients at UTH?

3.1 Types of bacteria isolated with SSD and without SSD

The types of bacteria isolated were more with no SSD than with SSD (Table 4). However the highest bacterium isolated in the two phases was Staphylococcus *spp* 31% and 37.5% with SSD and with NO SSD respectively.

Table 4: Relationship between types of Bacteria isolated with SSD and with no SSD

Bacterial Name	Frequency with SSD	Percent	Frequency with no SSD	Percent	Change*
E.coli	3	9.4	7	16.7	-4
Staphylococus	12	37.5	13	31.0	-1
Pseudomonas	4	12.5	8	19.0	-4
Kiebsiella	6	18.8	5	11.9	1
Acenibacteria	1	3.1	1	2.4	0
Streptococcus	2	6.3	2	4.8	0
Enterobacter Spp	2	6.3	3	7.1	-1
Mixed Enterobacter/Dipheroids	1	3.1	1	2.4	0
Hafnia	1	3.1	0	0.0	1
Proteus	0	0.0	2	4.8	-2

Total	32	100.0	42	100.0	-10

In table 3 above the negative values imply that the bacterium increased with no SSD.

3.2 Relationship between extent of bacteria colonisation with SSD and no SSD

We found the proportion of positive cases from swabs with SSD greater than the proportion of positive cases from swabs with no SSD thus it was statistically significant with regard to onset of burn wound colonisation. (P value =0.0043)(Table 3)

Table 3: SWABS with SSD and with no SSD

	SWAB with no SSD		SWAB with SSD	
Result	Actual obs	Percent Actual obs		Percent
Positive	42	91.3	32	69.57
Negative	4	8.7	14	30.43
Total	46	100	46	100

Table 3: Two-sample test of proportion for SWABS results with and with no SSD

Variable	Proportion	Std. Err.	z	P > |z|	[95% Conf. Interval]
x	0.913	0.04155			0.831555 0.994445
Y	0.6957	0.0678			0.5627369 0.8286631
Diff	0.2173	0.0795548			0.0613754 0.3732246
	under Ho:	0.0827477	2.63	0.009	

Number of observations for x and y are 46 respectively. Diff = prop(x) - prop(y)

Where:

X is the proportion of patients whose swabs with SSD results were positive;

Y is the proportion of patients whose swabs without SSD results were positive.

Null hypothesisHo: diff = 0 (there is no difference)

Alternative hypothesesHa$_1$: diff < 0: states that the proportion of positive cases from swabs with SSD is less than the proportion of positive cases from swabs with no SSD Pr (Z < z) = 0.9957

Ha$_2$: diff ≠ 0 states that the proportion of positive cases from swabs with SSD is not equal to the proportion of positive cases from swabs with no SSD. Pr (|Z| < |z|) = 0.0086

Ha$_3$: diff > 0 states that the proportion of positive cases from swabs with SSD is greater than the proportion of positive cases from swabs with no SSD Pr (Z > z) = 0.0043

The p – values 0.0086 and 0.0043 for alternative hypothesis Ha$_2$ and Ha$_3$ respectively are less than the 5 percent level of significance thus significant.

3.3 The culture, sensitivity profile and resistant profile of the microorganism in absence with no SSD in burn patients at UTH

The study found that the most organisms isolated with no SSD were Staphylococcus spp 37.35%, pseudomonas 21.43 %, E.coli 19.05%, and kiebsialla spp 11.90% (Table 4)

The sensitivity and resistant profile: Among the staphylococcus (37.50%) isolated in our study, 46% were sensitive to ciprofloxacin, 23.07% were resistant to ciprofloxacin and 53% were sensitive to chloramphenicol and 7.69% were resistant to chloramphenicol.Among pseudomonas (21.43 %) isolated in our study 50% were sensitive to ciprofloxacin and 37.50% were resistant to ciprofloxacin and 75% were sensitive to ceftazidime and 25% were resistant to ceftazidime. Among the E.coli (19.05%) isolated in our study, 85.71% were sensitive to

cefotaxime and none was resistant and 71.42% were sensitive to chloramphenicol and 14.29% were resistant to chloramphenicol. Among the kiebsialla spp (11.90%) isolated in our study ,80% were sensitive to ciprofloxacin ,60% sensitive to ceftazidime and chloramphenicol and 20% resistant to it.(Table 4)

Table 4: The culture, Sensitivity profile and resistant profile of Bacteria with no SSD

Bacteria isolated	Sensitivity	Nitrofurantoin	Ciprofloxacin	Gentamycin	Cotrimoxazole	Oxacillin	Erythromycin	Cefotaxime	Ceftazidime	Penicillin	Tetracycline	Imipenem	Chloramphenicol	Total
E.coli Total= 7	Sensitive		5	3				6(85.71%)		1	1		5(71.42%)	21
	Intermediate			1	4			1	1	2				9
	Resistant												1(14.29%)	1
Staphylococus Total= 13	Sensitive	1	6(46.15%)		1	5	4	3		4			7(53.85%)	31
	Intermediate		1(7.69%)		8	4	1	4		2			2(15.38)	22
	Resistant		3(23.07%)										1(7.69)	4
Pseudomonas Total = 8	Sensitive		4(50%)	3					6(75%)			1		14
	Intermediate													0
	Resistant		3(37.50%)						2(25%)					5
Kiebsiella spp Total= 5	Sensitive		4(80%)					1	3(60%)				3(60%)	11
	Intermediate			1	3					1	1		1	7
	Resistant								1(20%)					1
Acenib	Sensit													0

acteria Total= 1	ive													
	Inter media te			1				1						2
	Resist ant		1						1					2
Strepto coccus Total= 02	Sensit ive						1	2	1			1		5
	Inter media te									1				1
	Resist ant						1						1	2
Entero bacter Spp Total= 03	Sensit ive		2					2	1				2	7
	Inter media te			1	2					1	1			5
	Resist ant													0
Mixed Entero bacter/ Dipher oids Total= 4	Sensit ive		1	1									1	3
	Inter media te							1						1
	Resist ant													0
Proteus Total =02	Sensit ive		1	1				1					1	4
	Inter media te												1	1
	Resist ant													0

∗Only the drugs that were subjected to all the bacteria isolates or 80% isolates in the respective bacteria type were considered. ∗Only the bacteria isolates that were more than or equal to five were reported in our study

3,4 The culture, sensitivity profile and resistant profile of the microorganism with SSD in burn patients at UTH

The study found that the most organisms isolated with SSD were Staphylococcus spp 30.95%, and kiebsialla spp 18.75% (Table 5)

The sensitivity and resistant profile: Among the staphylococcus (30.95%) isolated in our study, 50% were sensitive to ciprofloxacin, 33.33% sensitive to oxacillin and 16.66% were resistant to erythromycin and 33.33% to cefotaxime. Among the kiebsialla spp (11.90%) isolated in our study, 50% were sensitive to ciprofloxacin and chloramphenicol, 16.66% sensitive to ciprofloxacin (Table 5).

Table 5: The culture resistant profile and sensitivity profile of organism with SSD

Bacteria Isolated	Sensitivity	Nitrofurantoin	Ciprofloxaci	Gentamycin	Cotrimoxazol	Oxacillin	Erythromycin	Cefotaxime	Ceftazidime	Penicillin	Tetracycline	Imipenem	Chloramphenicol	Total

			n		e									
E.coli Total =3	Sensitive		1	1				2					1	5
	Intermediate			1	1					1			1	4
	Resistant		1					1						2
Staphylococcus Total=12	Sensitive		6(50%)		1	4(33.33)	5	3(25%)		3			6(50%)	28
	Intermediate				7	7	1			1			2	18
	Resistant						2(16,66%)	4(33.33%)						6
Pseudomonas Total=4	Sensitive		1	2				1	2			1		7
	Intermediate			1					2					3
	Resistant		2											2
Kiebsiella spp Total =6	Sensitive		3(50%)	2				2				1	3(50%)	11
	Intermediate		1	3	2				1				3	10
	Resistant		1(16.66)					1			1			3
Aceniba	Sensitiv													0

cteria Total=1	e													
	Intermediate			1					1					2
	Resistant		1											1
Streptococcus Total=2	Sensitive							2	2					4
	Intermediate									2			2	4
	Resistant						1							1
Enterobacter Spp Total=2	Sensitive		1					1						2
	Intermediate							1		2	1		1	5
	Resistant													0
Mixed Enterobacter/ Diphoreids Total=01	Sensitive		1	1									1	3
	Intermediate													0
	Resistant							1						1
Hafnia Total =1	Sensitive										1			1
	Intermediate			1									1	2
	Resistant							1						1

∗Only the drugs that were subjected to all the bacteria isolates or 80% isolates in the respective bacteria type were considered. ∗Only the bacteria isolates that were more than or equal to five were reported in our study

IV. DISCUSSION

Types of bacteria isolated with SSD and without SSD

We found that the types of bacteria isolated with SSD was less than with no SSD evidenced by 42 (91.3%) different types of bacteria isolated with no SSD and 32 (69.57%) different type of bacteria isolated with SSD. The most predominant colonising organisms isolated from the surface swabs revealed *Staphylococcus spp*, 31% and 37.5% with SSD and with no SSD respectively. These findings are comparable to the findings of A.O. Ugburo, et.al 2004 conducted in Nigeria .In their study they used systemic antibiotics to evaluate their role in the prevention of onset of burn wound colonisation and they found that the predominant

isolate for the three groups was S*taphylococcus spp*, 47.6% of the cases with ampicillin and cloxacillin, 38.9% of the cases with gentamycin and erythromycin and 65% of the cases in the control of the *staphylococcus spp* respectively.Our findings in this study give more satisfactory findings compared to the A.O. Ugburo, et.al 2004 findings because the SSD was used topically compared to the systemic antibiotic which were given systemically in the prevention of onset of burn wound colonisation. The Systemic antibiotic did not play a role in bacteria wound colonisation due to presence of necrotic a vascular tissue around the burn wound which does not allow systemic antibiotics to reach the area of concern. (Kapoor. N, et.al 1989)

Relationship between extent of bacteria colonisation with SSD and no SSD

In this study, we found the proportion of positive cases from swabs with SSD greater than the proportion of positive cases from swabs with no SSD. The evidence is that out of the 46 burn patients studied 42 (91.3%) had positive swabs with no SSD and 32 (69.57%) had positive swab with SSD. We found that there was statistically significant difference between the phases with regard to onset of wound colonisation. (P value =0.0043). Our study findings on the extent of bacterial colonisation are comparable to the findings of Barry Wright, et.al 2009 in Canada. Their study found that SSD, silver nitrate and chlorhexidine have broad excellent activity against all the bacterial pathogens and concluded that they could be used empirically for prevention of bacterial wound colonisation.

Relationship between the culture, resistant profile and sensitivity profile of organism with SSD and no SSD

In this study we found that the most organisms isolated with SSD were Staphylococcus spp 30.95% and kiebsialla spp and with no SSD were Staphylococcus spp 37.35%, pseudomonas 21.43 %, E.coli 19.05%, and kiebsialla spp 11.90%.

Demographic characteristics of study patients

The study has revealed that there were more male 70% participants than female 30%, the age of participants were more of those between the age of 0-5years 73.91% and the least was above 18years 2.17%.Burning agent profile for participants, the burning agents profile revealed that most of the participants were burnt from hot liquids representing 67.39% and the least was frame 8.7%. The study revealed that most patients used eggs as the mode of first 52.17% and mud was least with 2.17%

Limitations

The following limitations to the study were identified:

- The relationship between the sensitivity and resistant profile of the bacteria isolated with SSD and with no SSD could not be done because the number of bacteria isolates with regard to the types of bacteria in both phases were too small to draw conclusions on
- There was no uniformity in the drugs that were subjected to each type of bacteria isolated thus a comparison could not be made on the sensitivity and resistant profile between the SSD and no SSD.

V. CONCLUSION AND RECOMMENDATIONS

The study investigated the role of SSD in the prevention of onset of burn wound surface colonisation on the in the burn patients at UTH, Lusaka in Zambia.It was noted that the most predominant colonising surface organisms isolated from the surface swabs revealed *Staphylococcus spp*, as can be seen from the 31% and 37.5% with SSD and with no SSD respectively .

From the study findings, it could be concluded that SSD has a role in the prevention of burn wound surface bacterial colonisation in burn patients at UTH (p-value =0.0043) evidenced by the significant difference between the two proportions ,91.3% had positive swabs with no SSD and 69.57% had positive swab with SSD.

As the researcher went into this study, he approached it with the belief that what was important was understanding the role of silver sulfadiazine in the prevention of onset of bacterial surface wound colonization. This resulted into lack of standardization in the susceptibility tests that were done on the bacteria isolated with SSD and with no SSD. Therefore there is need for a susceptibility test study to be done on the bacterial isolates of burn wound surface in burn patients.

VI. RECOMMENDATIONS

1. There need to do Periodic studies of these micro-organisms quarterly in burn patients because the ecology of microbes in the burn wound is always changing.
2. There is need for the hospitals to enforce adherence to treatment protocols and and improve the availability of the SSD drug.
3. These finding may not be extrapolated to other hospital in our country because the spectrum of microbiota causing infection in burn patients varies with geographical location therefore, there is need to carry out a similar survey at all provincial centres to determine the prescribing patterns

REFERENCES

[1] American Association Society (2012)

[2] Albertyn .R., Bickler S.W and Rode.H. (2006).Burns 32:Pediatrics burn injuries in Sub Saharan Africa—an overview

[3] Barry Wright, Kan Lam, Robert and E. Burrell(1998). Wound management in an era of increasing bacterial antibiotics: a role for topical silver treatment. Fort Saskatchewan, Alberta, Canada.

[4] Bang ,R.L., Gang, R.K., Sanyal ,S.C., Mokaddas ,E and Ebrahim ,M.K. (1998) Burns; 24.Burn septicemia: an analysis of 70 patients.

[5] Bone, R .C., Sibbald, W.J and Sprung, C .L. (1992). The ACP-SCCM consensus conference on sepsis and organ failure: 1481-1490

[6] Bowen-Jones., J.R, Coovadia ,Y.M and Bowen-Jones EJ. (1990) Infection control in a third world burn facility: **16**:445–452

[7] Garmel, (2012) edited by S.V. Mahadevan, Gus M. *An introduction to clinical emergency medicine* (2nd ed.). Cambridge: Cambridge University Press. pp. 216–219

[8] Guggenheim, M.,Zbinden R.,Handschin AE., Gohritza A.,Lintas MA and giovanoli P (2009) change in bacterial isolate from burn wound and their antibiogram : A 2o year study burn. The spectrum of microbes causing infection in burn patient varies with geographical location: **35**:553-560

[9] Hangnga VU. Miccoy L. Carino E et al (2002) burn wound infection susceptibilities to topical agents: the Nathan"s agar well diffusion technique P & T 2002 :**27**: 390-6

[10] Herndon D (2002). "Chapter 4: Prevention of Burn Injuries". *Total burn care* . Edinburgh: Saunders. p. 46.

[11] Hussain, M. T and Tajuri, S. (1989). Analysis of infection in burn ward.**15**:299-302

[12] Jones WG, Minei JP, Barber AE, et al. (1990) Bacterial translocation and intestinal atrophy after thermal injury and burn wound sepsis. Ann Surg **211**:399–405.

[13] Kapoor N, chibber S, Vadebra DV (1989) Susceptibility of multidrug resistant isolates of kiebsiela pneumonia to silver nitrates :**34**: 94-8

[14] Katara Gunjan, Chamania Shobha, Chitnis Sheetal, Hemvani Nanda, Chitnis Vikrant, and Dhananjay Sadashiv ChitnisIndian J Plast Surg. 2012 May-Aug; **45**(2): 374–37

[15] Lameck Z (2000) Possible aetiological factors that cause pyrexia in children with burns at UTH, Zambia

[16] Lawrence JC. (1985) The bacteriology of burns. J Hosp Infect

[17] Lawrence JC. (1992) Burn bacteriology during the last 50 years. Burns ;2 (Suppl):523–9.

[18] Lansdown AB.(2002) Silver. 2. Toxicity in mammals and how its products aid wound repair. J Wound Care ,**11**:173–7

[19] Madson SM, west H. danielsen L. Rosdah V (1996) Bacterial colonisation and healing of venous ulcers APMIS **104**:895-9

[20] Munford RS, Suffredini AF. (2009) Sepsis, severe sepsis, and septic shock. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and Practice of Infectious Diseases*. 7th ed. Philadelphia, Pa: Churchill Livingstone Elsevier; chapter 70.

[21] Monafo WW, west WA. (1990) Current treatment recommendation for topical therapy.Drug 1990: **40** : 364-73

[22] Mooney D P and Gamelli R L(1989) sepsis following thermal injury: **15**: 22- 31

[23] Nick,F,Amanda H, Nigel M 2009: Sampling and sample size calculation;National Institute Of health Research

[24] O'sulivan S, Schmhz T, physical rehabilitation assessment and treatment 4th ed. Philadelphia PA.Fa Davis Company: 2001

[25] Onuba O, Udoidiok E. (1987). Hospital management of massive burns in the developing countries. Burns; **13**:386–90

[26] Parish RA, Novach AH, Heimbach DM, Engrav LR.(1987) Fever as a predictor of infection in burned patients Journal of trauma :**27** :67-71.

[27] Pandit DV, Gore MA, Saileshwar N, Deodhar LP.(1993) Laboratory data from the surveillance of a burns ward for the detection of hospital infection. Burns ; **19**:52–5

[28] Price WR. Wood M. Silver nitrate burn dressing. Treatment of seventy burned person .Am J Surg .1966 : **112** :674-80

[29] Pruitt Jr B.A McManus A. T, Kim SH and Goodwin CW. (1998): Burn Wound Infection. Current status. Word journal of surgery **22**; 153 -45

[30] Pandit DV, Gore MA, Saileshwar N, Deodhar LP.(1993) Laboratory data from the surveillance of a burns ward for the detection of hospital infection. Burns ; **19**:52–5

[31] Pruitt BA, Lindberg RB, McManus WF and Mason AD. (1983) Current approach to prevention and treatment of Pseudomonas aeuroginosa infections in burned patients. Rev Infect Dis, **5**:S889–97.

[32] Pruitt BA, McManus AT. (1984) Opportunistic infections in severely burned patients. Am J Med 1984**; 76:**146–54.

[33] Walter CJ, Dumville JC, Sharp CA, 2012. Systematic review and meta-analysis of wound dressings in the prevention of surgical-site infections in surgical wounds healing by primary intention. *T.Br J Surg*.; **99**(9):1185-94.

[34] Wurtz R. Karajovic M. Dacumos E. Jovanovic B. Hanumadass M(1995) Nosocomial infection in a burn intensive care unit: 875—881

[35] Wasserman D. (2001) Systemic complications of extended burns. Ann Chir Plast 2001; **46**:196–209.

[36] Sharma B.R D. Harish, Virendar Pal Singh, Sumedha Bangar (2006) Burn 32: Septicemia as a cause of death in burns: An autopsy study

[37] Sowemimo GOA. (1993)Burn care in Africa: reducing the misery index. The 1993 Everett Idris Evans Memorial lecture. J Burn Care Rehabilitation ;**14**: 589–94

[38] .Ugburo.A.O, O.A. Atoyebi, J.O. Oyeneyin, G.O.A. Sowemimo .Burn 30 (2004) An evaluation of the role of systemic antibiotic prophylaxis in the control of burn wound infection at the Lagos University Teaching Hospital.