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Original Research Article

Antimicrobial susceptibility profiles of bacterial isolates from burn wound infections: experience at a tertiary care hospital teaching institution

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ABSTRACT

Background: This study was conducted to know, understand and document the current bacterial isolates and their antimicrobial susceptibility profile in our tertiary healthcare facility. Aim was to isolate, identify and study the current antimicrobial susceptibility profiles of bacterial isolates from patients with burn wound infections admitted in our tertiary care hospital.
Methods: Wound swabs from burn patients admitted in burn ICU and burn wards were collected on day 1, day 3, day 5 and day 10. Samples were processed using standard microbiologic procedure and bacterial isolates that grew on culture was tested for their antimicrobial sensitivity pattern using Kirby Bauer disk diffusion method.
Results: Majority of patients in this study were of the age group of 21 to 30 years (32%). Total body surface area (TBSA) of the study subjects were collected and it was found that the mean TBSA was 39.59±11.6. Total number of bacterial isolates identified during this study was 226 and of those most common was *Klebsiella pneumoniae* (n=62). Aminoglycoside resistance were at 61% and among carbapenems, ertapenem showed 90% resistance in *Klebsiella pneumoniae* isolates. Cefoxitin resistance indicating the presence of MRSA were seen in 31% of *Staphylococcus aureus* isolates. Acinetobacter showed 100% resistance to ceftazidime.
Conclusions: The results indicate the predominance of drug resistant gram-negative bacterial isolates in burn wounds. *Klebsiella pneumoniae* came out to be the most common bacterial isolate in our study. Because of increasing resistance and decreasing availability of newer antibiotics, active microbial surveillance and judicious antibiotic usage is the way forward.

Keywords: Antimicrobial susceptibility profiles, Bacterial isolates, Burn wound infections

INTRODUCTION

Burn wound infections are one of the most common cause burn mortality and morbidity especially in post initial resuscitation.[1,2] Add to this, development of multidrug resistant bacterial organisms and changing spectrum of bacteria colonizing and infecting burn wound make it even more challenging for clinicians to effectively treat burn wound infections. Burn injury causes coagulative necrosis of the skin and the underlying tissue.[2] The amount of damage caused by burn is directly proportional to the energy that the causative agent (like fire, hot liquid, and other offending substance) imparts on the skin.[2] Skin to an extent decreases the transfer of heat to internal structures, but the damage to underlying tissue occur anyway due to local tissue responses.[2,3] Burn injury on the skin causes three zones of cutaneous injury known as zone of coagulation, stasis and hyperemia.[2] The risk of subsequent wound infection and systemic infection in a patient of burn correlates with the amount of skin

involved.[3] This is usually represented as total body surface area (TBSA).[1,3] Burn of significant TBSA can lead to immunosuppression that predisposes the patient to wound and systemic infections. Even though local inflammation is a necessary factor for wound healing burn of significant TBSA can lead to a systemic inflammatory response that at first is proinflammatory but later become anti-inflammatory to preserve homeostasis.[3] Both these phases are mediated by cytokines and other cell signalling molecules. So, immune modulation may be the way forward when it comes to burn management.[2] This anti-inflammatory state causes increased risk of colonization and infection of burn wound. The usual pattern of burn wound infection and colonization is from gram positive (commensals) to gram negative organism which are thought to arise from the endogenous enteric flora.[4] In hospital setting, exogenous microorganism also colonizes burn wounds via direct contamination from the hands of health care workers and other sources like untreated water, soil, and other articles of daily use and worsen the wounds.[5] Coupled with this, emergence of wide spread antimicrobial drug resistance have added to the challenge of managing burn wound infections which has direct correlation with burn mortality.[3] The current Indian studies have also shown emerging drug resistance among a wide variety of human burn wound pathogens (bacteria and fungus) particularly the nosocomial isolates. This further limit the available therapeutic options for effective treatment of burn wound infections. On this aspect there are limited studies that reflect the current profiles of bacterial isolates and their antimicrobial susceptibility pattern, clearly showing gaps in our present knowledge. It is strongly felt that these lacunae must be overcome by dedicated, judicious, scientifically designed and carefully monitored studies. The antibiotic sensitivity profile will help with developing an institute based antibiograms and an antibiotic policy. A good antibiotic policy will go a long way when it comes to antibiotic rationing and antibiotic stewardship. Use of prophylactic systemic antibiotics in burn wound is controversial as of now and therefore when burn wound infection do occur, starting the patient on right antibiotic even before the susceptibility report can make a big difference in patient morbidity and mortality.[5,6] For that regular analysis of antibiotic susceptibility pattern and making antibiograms based on it and further improving the antibiotic policy of the institute based on this information is critical.

Considering all this factors we thus designed this study to understand the isolation rates of various microbial organisms from burn wounds and the antibiotic profile of these isolates.

METHODS

The study was carried out in the Department of Microbiology and Department of Burns and Plastics, Vardhman Mahavir Medical College and Safdarjung Hospital after obtaining hospital ethical committee approval. For this study, patients admitted in burn emergency, burn intensive care unit (BICU) and burn wards were recruited and informed consent was taken. The study was a hospital based observational study from November 2018 to April 2020 (18 months).

Inclusion criteria

Adult patients with burns ranging from 20% to 60% total body surface area (TBSA). Pediatric patients with burns ranging from 10% to 60% total body surface area (TBSA).

Exclusion criteria

Adult and pediatric patients with second degree burns.

Calculation of sample size

Burn wound infections are polymicrobial in nature and prevalence of bacteria in burn wound infections in different Indian studies are in the range of 50-70%.[7-9] Taking prevalence as 60% (allowable error of 10%) and applying the formula $N=4PQ/E^2$, the number of samples required is 96, therefore a total of 100 patients were recruited in the study.[10]

History was taken from patient or their reliable attendant using predesigned questionnaires to determine socio-demographic data, type of burn injury, TBSA, length of hospital stay, antibiotics given etc. Wound swabs of the burn patients were taken with a sterile cotton swab moistened with normal saline and transported to lab in less than 30 minutes as such or in brain heart infusion (BHI) broth (if pus is scanty) within 24 hours of patient admission. Swabs were taken on day 1 of admission and then on 3rd, 5th and 10th day of hospital stay.[3] Only patients, whose day 1, day 3 and day 5 samples were processed was included in the study. Patients who were lost to follow up after day 5 sample collection, were also included in the study. All samples were processed according to standard microbiological procedures. Isolated colonies were first identified using conventional test. And Biomérieux VITEK 2® was used were conventional method failed to identify an organism. Antimicrobial sensitivity testing was done using the Kirby Bauer disc diffusion method. And the test was interpreted according to the 28th edition of performance standard for microbial susceptibility testing, CLSI supplement M100.[11] Supplementary for inducible clindamycin resistance and ESBL (extended spectrum beta lactamases) was done. Inducible clindamycin resistance indicating a MLS_B phenotype was tested for in *Staphylococcus aureus* and CoNS, using erythromycin (15 µg) disc placed at a distance of 15 mm (edge to edge) from clindamycin (2 µg) disc on a Mueller-Hinton agar plate, previously inoculated with 0.5 McFarland standard bacterial suspension.[11,12] Inducible clindamycin resistance were detected for isolates with erythromycin zone diameter ≤21 mm diameter giving D-shaped zone of

inhibition around clindamycin with flattening towards erythromycin disc.[11,13] Extended spectrum beta lactamase (ESBL) production was tested on *Klebsiella pneumoniae*, *Klebsiella oxytoca*, *Escherichia coli* and *Proteus mirabilis* using CLSI recommendations.[11,14] Screening for ESBL production was done by looking for zone diameter of ≤27 mm for cefotaxime disk of 30 µg concentration on Mueller-Hinton agar plate, previously inoculated with 0.5 McFarland standard bacterial suspensions. The isolates that screened positive were confirmed using clavulanate inhibition test. A zone diameter of ≥5 mm difference between cefotaxime 30 µg and cefotaxime clavulanate 30/10 µg was taken as ESBL positive.[11,14]

Statistical analyses was done, and categorical variables were presented in number and percentage (%) and continuous variables were presented as mean±SD and median. The data was entered in MS EXCEL spreadsheet and analysis was done using Statistical Package for Social Sciences (SPSS) version 21.0.

RESULTS

Age and sex of study subjects were taken along with other relevant history. Majority of patients in this study were of the age group of 21 to 30 years (32%). Followed by age group of 31 to 40 years (27%). The mean age group of patients was 33.42. 55% patients were males and 45% were females (Table 1).

Total body surface area (TBSA) of the study subjects were collected and it was found that the mean TBSA was 39.59±11.61 with a median of 40 and IQR between 30-47.75. The range of TBSA in patients was 15-60%. Of the 270 swabs samples, single isolates were grown in 103 samples (38.15%) and 167 swabs (61.85%) grew multiple isolates. Based on gram staining, 77% were gram-negative isolates and 23% were gram-positive isolates. Total number of bacterial isolates identified during this study was 226. Most common was *Klebsiella pneumoniae* (62) followed by *Pseudomonas aeruginosa* (50). The rest of the isolates were *Proteus mirabilis* (24), *Staphylococcus aureus* (32), *Acinetobacter spp.* (23), CoNS (19), *Escherichia coli* (8), *Providencia stuartii* (3), *Enterobacter spp.* (3), *Klebsiella oxytoca* (1) and *Citerobacter koseri* (1) respectively (Table 2).

Table 1: Age and gender distribution of study subjects.

Age (years)	Number of patients (%)	
≤20	16 (16)	
21-30	32 (32)	
31-40	27 (27)	
41-50	13 (13)	
51-60	6 (6)	
>60	6 (6)	
Mean±SD	33.41±14.7	
Gender distribution w.r.t. frequency (percentage)	**Male**	**Female**
	55 (55%)	45 (45%)

Antimicrobial sensitivity testing was performed on all the bacterial isolates using the predetermined antibiotic panels. The test was then interpreted according to the 28th edition of performance standard for microbial susceptibility testing, CLSI supplement M100.[11] Resistance of organisms to different antibiotics are tabulated below (Table 3, Table 4). Phenotypic detection of ESBL using clavulanate inhibition test and inducible clindamycin resistance detection done using D test results are tabulated below (Table 5 and 6).

Table 2: Distribution of total bacterial isolates.

Bacterial Isolates	Day 1	Day 3	Day 5	Day 10	Total	
					Frequency	**Percentage**
Klebsiella pneumoniae	0	8	23	31	62	27.43
Pseudomonas aeruginosa	0	4	28	18	50	22.12
Proteus mirabilis	0	5	11	8	24	10.61
Staphylococcus aureus	2	15	8	7	32	14.15
Acinetobacter spp.	1	7	9	6	23	10.17
CoNS	6	10	3	0	19	8.40
Escherichia coli	0	3	3	2	8	3.53
Enterobacter spp.	0	0	1	2	3	1.32
Providencia stuartii	0	0	2	1	3	1.32
Klebsiella oxytoca	0	0	0	1	1	0.44
Citerobacter koseri	0	0	0	1	1	0.44
Total	9	52	88	77	Total =226	

Table 3: Antibiotic resistance seen in Gram positive cocci.

Gram positive cocci	Cefoxitin (30 µg)	Gentamicin (10 µg)	Ciprofloxacin (5 µg)	Cotrimoxazole (1.25/23.7 µg)	Penicillin (10 Units)	Erythromycin (15 µg)	Clindamycin (2 µg)	Vancomycin (30 µg)	Linezolid (30 µg)
***Staphylococcus aureus* (total number of isolates 32)**	31% (10)	34% (11)	69% (22)	28% (9)	100% (32)	41% (13)	41% (9)	0% (0)	0% (0)
other *Staphylococcus spp.* (total number of isolates 19)	26% (5)	16% (3)	16% (3)	0% (0)	100% (19)	16% (3)	11% (2)	0% (0)	0% (0)

Table 4: Antibiotic resistance seen in Gram negative bacilli.

Gram negative bacilli	Ceftazidime (30 µg)	Cefotaxime (30 µg)	Amikacin (30 µg)	Piperacillin tazobactam (100/10 µg)	Netilmicin (30 µg)	Ertapenem (10 µg)	Meropenem (10 µg)	Imipenem (10 µg)	Ciprofloxacin (5 µg)	Cotrimoxazole (1.25/23.75 µg)	Colistin (10 µg)	Ampicillin (10 µg)	Amoxicillin-clavulanate (20/10 µg)
***Acinetobacter spp.* (Total 23)**	100% (23)	-	78% (18)	82% (19)	65% (15)		61% (14)	48% (11)	96% (22)	39% (9)	0% (0)	-	-
***Citrobacter koseri* (Total 1)**	-	100% (1)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)	100% (1)	0% (0)	0% (0)	-	-
***Enterobacter spp.* (Total 3)**	-	100% (3)	100% (3)	33% (1)	67% (2)	67% (2)	33% (1)	33% (1)	100 % (3)	67% (2)	0% (0)	-	-
***Escherichia coli* (Total 8)**	-	25% (2)	13% (1)	50% (4)	25% (2)	50% (4)	0% (0)	0% (0)	75% (6)	25% (2)	0% (0)	-	-
***Klebsiella pneumoniae* (Total 62)**	-	98% (61)	61% (38)	94% (58)	42% (26)	90% (56)	69% (43)	58% (36)	90% (56)	66% (41)	0% (0)	-	-
***Pseudomonas aeruginosa* (Total 50)**	78% (39)	-	56% (28)	20% (10)	38% (19)	-	28% (14)	26% (13)	52% (26)	-	0% (0)	-	-
***Proteus mirabilis* (Total 24)**	-	50% (12)	12% (3)	8% (2)	4% (1)	42% (10)	0% (0)	0% (0)	46% (11)	25% (6)	-	80% (19)	54% (13)
***Providencia stuartii* (Total 3)**	-	100% (3)	33% (1)	33% (1)	33% (1)	33% (1)	33% (0)	-	33% (1)	33% (1)	-	100% (3)	100% (3)
***Klebsiella oxytoca* (Total 1)**	-	100% (1)	0% (0)	100% (1)	0% (0)	0% (0)	0% (0)	0% (0)	100% (1)	0% (0)	0% (1)	-	-

Table 5: Distribution of ESBL producers among family *Enterobacteriaceae*.

ESBL	*Escherichia coli* (Total 8)	*Klebsiella oxytoca* (Total 1)	*Klebsiella pneumoniae* (Total 62)	*Proteus mirabilis* (Total 23)
Negative	8 (100%)	1 (100%)	58 (93%)	23 (100%)
Positive	0	0	4 (7%)	0

Table 5: D test showing MLSB phenotypes among CoNS and *Staphylococcus aureus.*

D test	CoNS (n=19)	*Staphylococcus aureus* (n=32)
Negative	94% (18)	81% (26)
Positive	6% (1)	19% (6)

DISCUSSION

Burn patients are at increased risk of acquiring burn wound infections caused by highly drug resistant nosocomial bacterial strains. In this hospital based observational study, 100 patients who were admitted in burn ICU or burn ward were studied for their burn wound isolates and its susceptibility pattern. Majority of the patients in this study were in the age group of 21 to 30

years (32%) which is similar to the study findings by Mundhada et al and Priyadarshini et al having 70% and 42% respectively in this age group.[9,15] Patients in this study were mostly males (55%) which was the observation made in the study conducted by Jauhari et al with 60.7% of the patient population as males.[16] But other Indian studies such as Mundhada et al, Priyadarshini et al and Gupta et al had female predominance.[9,15,16] The mean total body surface area (TBSA) in the study were 39.59±11.61% with a median of 40 and range of 15% to 60%. This study was done in both burn ward and burn ICU, with 78% of patients coming from burn wards. A total of 375 swabs were cultured for this study of which 28% had no isolates, rest of swabs had bacterial growth indicating an isolation rate of 72%. This observation was corroborated by the finding of Jauhari et al, with 70.6% isolation rate and Gupta et al, with isolation rate of 61.87%.[16,17] There were other studies that showed higher isolation rate such as Priyadarshini et al and Mundhada et al with 96% and 89.60% respectively.[9,15] Of the 270 swabs with bacterial isolates, single isolates were seen in 38.15% swabs and 61.85% had multiple isolates. A similar study done by Mundhada et al, which had a much smaller sample size showed a different result.[9] In that study swabs cultured on day 4, day 10 and day 16 of admission in burn ward were analysed, and single isolates were seen in 71.28% swabs and multiple in 18.31%.[9]

Most of the bacterial isolates in the study were gram-negative in nature. Of the 226 bacterial isolates, 175 bacterial isolates were gram-negative (77%) and 51 isolates were gram-positive (23%). Similar findings were seen in Mundhada et al, Jauhari et al and Priyadarshini et al with gram-negative constituting 72.4%, 60.2% and 59.26% respectively.[9,15,17] *Klebsiella pneumoniae* constituting 62 (27.43%) of the 226 isolates were the most common organism followed by *Pseudomonas aeruginosa* constituting 50 (22.12%) of the 226 isolates. Similar study by Mundhada et al showed *Klebsiella pneumoniae* (34.4%) as the most common isolate followed by *Pseudomonas aeruginosa* (23.94%).[9] However, in Gupta et al and Chauhan et al, *Klebsiella pneumoniae* was the second most common organism isolated.[16,18] In case of Bhatt et al *Staphylococcus aureus* was the second common isolate.[19] Comparing this with western reviews and studies (Norbury et al and De Macedo et al) *Staphylococcus aureus* is quoted as the chief causative agent of burn wound infection.[4,20] Other isolates in our study, in the descending order of representation are *Staphylococcus aureus* (14.15%), *Proteus mirabilis* (10.61%), *Acinetobacter spp.* (10.17%), CoNS (8.40%), *Escherichia coli* (3.53%), *Providentia stuartii* (1.32%), *Enterobacter* spp (1.32%), *Klebsiella oxytoca* (0.44%) and *Citerobacter koseri* (0.44%). This cluster of isolates are more or less similar to other studies like Bhatt et al, Gupta et al and Chauhan et al showing a common pattern of infection in burn patients.[16,18,19]

After analysing day wise swab culture results, day 1 sample of 100 patients had 87% sterile swabs and rest with isolates. This indicates that majority of the burn patients have sterile wounds at the time of presentation. The isolates that were obtained on day 1 consisted mainly of CoNS which is a known commensal of skin. Day 1 isolates mainly constituted of gram-positive organisms (89%). Sample collected on Day 3 also had a significantly high number of sterile swabs (53%) and gram-positive organisms like *Staphylococcus aureus* and CoNS. This finding is similar to the study of Mundhada et al, where day 4 swabs had *Staphylococcus aureus* as the most common isolate.[9] Samples of day 5 had only 27% sterile swabs with *Pseudomonas aeruginosa* (23%) as the most common isolate followed by *Klebsiella pneumoniae*. Day 5 samples indicated a shift in pattern, with gram-negative organisms (87%) representing the bulk of isolates. Samples collected on Day 10 had the least number of sterile swabs (22.78%) with *Klebsiella pneumoniae* (30.69%) predominating in the number of bacterial isolates. Gram-negative (91%) was the predominant isolate in Day 10 samples. *Acinetobacter spp*. have emerged as a significant nosocomial pathogen resulting in considerable morbidity and mortality in burn patients. In our study *Acinetobacter spp*. was the fifth most common isolate (10.17%) having more frequency than *Escherichia coli* (3.53%). This is similar to Gupta et al and Bhatt et al who had an isolation rate of 14.83% and 17.27% for *Acinetobacter spp*. respectively.[16,19] Other studies like Mundhada et al, and De Macedo et al recorded a very low isolation rate, 2.75% and 3.9% respectively.[9,20]

During the study, all the bacterial isolates were tested for their antimicrobial susceptibility pattern using predetermined antibiotic panel. Analysing the most common isolate *Klebsiella pneumoniae*, it was seen that resistance to cephalosporins were 98%. This was corroborated by findings of Gupta et al (100%) and Lunawat et al (91%).[16,21] Study by Priyadarshini et al showed 50% resistance to cephalosporins.[15] Aminoglycoside resistance were at 61% and among carbapenems, ertapenem showed 90% resistance in *Klebsiella pneumoniae* isolates. Colistin showed 100% sensitivity followed by netilmicin 56% (Table 3). Most common sensitivity pattern of *Klebsiella pneumoniae* from our antibiotic panel was strains that were only sensitive to colistin. Colistin resistance were noted in study done by Gupta et al which was not seen in our study.[16] *Pseudomonas aeruginosa* resistance to ceftazidime was 78% with high sensitivity towards piperacillin tazobactam, carbapenems and colistin, showing similar patterns to what suggested by Jauhari et al.[17] But higher antimicrobial resistance was showed by Gupta et al and Lunawat et al.[16,21] Colistin resistance was again seen in the study conducted by Gupta et al in *Pseudomonas aeruginosa* but in our study colistin resistance were not recorded.[16] Third most common isolate *Staphylococcus aureus* showed 100% sensitivity to vancomycin and linezolid. Mundhada et al, *C*hauhan et al and Priyadarshini et al also showed 100% sensitivity to both vancomycin and linezolid.[9,15,18] But resistance to

vancomycin and linezolid was noted in study done by Lunawat et al.[21] Cefoxitin resistance indicating the presence of MRSA were seen in 31% of isolates in our study which is similar to studies conducted by Mundhada et al and Priyadarshini et al with 34% and 22.2% cefoxitin resistance respectively.[9,15] *Acinetobacter* showed 100% resistance to ceftazidime and high resistance to quinolones, piperacillin tazobactam and carbapenems, sensitivity was maximum for colistin (100%). *Enterobacter spp*. showed high resistance to aminoglycosides, cephalosporins, ertapenem and quinolones, and better sensitivity was seen for meropenem, imipenem and colistin. *Escherichia coli* isolation was exceptionally low (3.53%) in our study which was similar to results of Priyadarshini et al.[15] In *E. coli* cephalosporins, quinolones, and ertapenem showed high resistance but other carbapenems, aminoglycosides and colistin were highly sensitive. ESBL detection showed that 4 *Klebsiella pneumoniae* isolates out of 62 were producers of this enzyme. D test for inducible clindamycin resistance indicated 1 out of 19 CoNS and 6 out of 32 *Staphylococcus aureus* as positive.

Some of the studies indicate resistance to colistin among gram-negative and to vancomycin among gram-positive organisms, both of which were absent in our study. Since the CLSI recommended method of antimicrobial testing for colistin and vancomycin was not performed during our study, further testing is required to rule out such resistance patterns in our hospital settings. Test for beta lactamase using chromogenic cephalosporins were not performed which was a CLSI recommended supplementary test. Production of carbapenemases is a common method of resistance among *Enterobacteriaceae* which was not tested for in our current study. Anaerobic bacterial isolates in burn wound infection could not be documented as the present study only covered the aerobic burn wound isolates. Also, burn wound histopathologic evaluation for invasiveness of the infective agents would have shed more light on to the significance of all the bacterial isolates tested. Fungal agents are also a known etiologic agent for burn wound infections, which was not covered. These were some of the limitations of our current study.

CONCLUSION

The results indicate the predominance of drug resistant gram-negative bacterial isolates in burn wounds with *Klebsiella pneumoniae* as the most common isolate. A surge in isolation rate of *Acinetobacter spp*. was seen. Resistance among quinolones and cephalosporins was widespread among all bacterial isolates. Carbapenems have also shown decreased sensitivity in many of our isolates. Due to increasing resistance and decreasing availability of newer antibiotics, active microbial surveillance and strict implementation of guidelines and awareness on rational use of antibiotics is the way forward.

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REFERENCES

1. Miles RS, Amyes SGB. Laboratory control of antimicrobial therapy. In: Colle JG, Fraser AG, Marmion BP, Simmons A, eds, Mackie and McCartney Practical Medical Microbiology. 14th edn. Churchill Livingstone: New York; 1996; 151-177.
2. Ansermino M, Hemseley C. Intensive care management and control of infection. BMJ. 2004;329:220-3.
3. Kabboura S. Burn injury: review of pathophysiology and therapeutic modalities in major burns. Ann Burns Fire Disast. 2017;2:97-102.
4. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev. 2006;19:403-34.
5. Norbury W, Herndon D, Tanksley J, Jeschke M, Finnerty C. Infection in Burns. Surg Infect. 2016;17:250-5.
6. Ramos G. Antibiotic prophylaxis in burn patients: a review of current trends and recommendations for treatment. J Infectiol. 2018;1:1-5.
7. Ramos G, Cornistein W, Cerino GT, Nacif G. Systemic antimicrobial prophylaxis in burn patients: systematic review. J Hosp Infect. 2017;97:105-14.
8. Diederen B, Wardle C, Krijnen P, Tuinebreijer W, Breederveld R. Epidemiology of clinically relevant bacterial pathogens in a burn center in the Netherlands between 2005 and 2011. J Burn Care Res. 2015;36:446-53.
9. Vinitha CT, Tiwari P, Singh S, Rasania S, Khokkar A, Talwar R. Pattern and extent of hospital acquired wound infections in burns patients in a Delhi tertiary Care hospital. Indian J Prev Soc Med. 2011;42:79-81.
10. Mundhada SG, Waghmare PH, Rathod PG, Ingole KV. Bacterial and fungal profile of burn wound infections in tertiary care center. Indian J Burns. 2015;23:71-5.
11. Banerjee B. Mahajan's Methods in biostatistics for medical students and research workers. 9th edn. New Delhi: Jaypee Brothers; 2018:124-125.
12. CLSI. M100 Performance standard for antimicrobial susceptibility testing. 28th edn. Wayne, PA: Clinical and Laboratory Standards Institute; 2018.
13. Prabhu K, Rao S, Rao V. Inducible clindamycin resistance in *Staphylococcus aureus* isolated from clinical samples. J Lab Phys. 2011;3:25-7.
14. Lewis J, Jorgensen J, Inducible clindamycin resistance in *Staphylococci*: should clinicians and microbiologists be concerned. Clin Infect Dis. 2005;40:280-5.

15. Rawat D, Nair D. Extended-spectrum β-lactamases in Gram Negative Bacteria. J Glob Infect Dis. 2010;2:263-74.
16. Priyadarshini M, Kumar M, Sharma AK, Prashad A, Seema K. Bacteriological profile and antibiogram of burn wound infections from burn patients at RIMS, Ranchi. Int J Med Res Prof. 2018;4:203-6.
17. Gupta M, Naik AK, Singh SK. Bacteriological profile and antimicrobial resistance patterns of burn wound infections in a tertiary care hospital. Heliyon. 2019;5;1-4.
18. Jauhari S, Shalabh P, Goyal M, Prakash R, Juyal D. Bacteriological and antimicrobial sensitivity profile of burn wound infections in a tertiary care hospital of Uttarakhand. Int J Curr Res 2020;12:30-6.
19. Chauhan JR, Khare S, Lal P, Kunhikatta V, Thunga G, Nair S, et al. An appraisal of antibiotic sensitivity pattern and drug utilization in burn patients. Indian J Burns. 2016;24:69-73.
20. Bhatt P, Rathi K, Hazra S, Sharma A, Shete V. Prevalence of multidrug resistant pseudomonas aeruginosa infection in urn patients at a tertiary care center. Indian J Plast Surg. 2015;23:56-9.
21. De Macedo JL, Santos JB. Nosocomial infections in a Brazilian Burn Unit. Burns. 2006;32:477-81.
22. Lunawat A, Sharma R, Kolla V, Patel S. Emerging resistance of higher antimicrobials and growing sensitivity of old antimicrobials against existing infections in burns. Int Surg J. 2015;2:385-91.

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