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Research article

Antibiotic sensitivity pattern of bacterial isolates in burns wound infection in a tertiary care hospital

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Abstract

Infection is an important cause of mortality in burns. Rapidly emerging nosocomial pathogens and the problem of multi-drug resistance necessitates periodic review of isolation patterns and antibiogram in the burns ward. Aim: The present retrospective study from wounds of patients admitted to burns unit was undertaken to determine the sensitivity pattern from the burns ward over a period from June – November 2014. Methods: The antibiotic sensitivity pattern of bacterial isolate from burn patients admitted in Microbiology Laboratory, Govt. Kilpauk Medical College were studied. 1378 patients samples were collected which comprised of burn wound swabs. All samples were cultured on Nutrient agar, Mac conkey agar and Blood agar at 37ºC for 24 hrs. The isolates were identified by culture, and their antibiotic sensitivity determined. Results: The most common isolate was Staphylococcus aureus. All Staphylococcus aureus isolates were sensitive to Vancomycin (100%), followed by Doxycycline (67%) and Amikacin (62%). Most of the Klebsiella pneumoniae isolates were resistant to third generation Cephalosporins (62%) but sensitive to Imipenem (100%), Levofloxacin (100%), followed by Piperacillin and Tazobactam (87%), amikacin(51%). Imipenem (100%), Levofloxacin (100%), followed by Piperacillin and Tazobactam (98%) were sensitive in most of the isolates of Pseudomonas aeruginosa.

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1. Introduction

In Burns patient, infections are the major cause of morbidity and mortality. Burns wound provide a suitable site for bacterial multiplication and are more persistent sources of infection than surgical wound, mainly because of larger area involved and longer duration of stay in hospital. Infection is a major cause of morbidity and mortality in hospitalized burn patients [1]. It is now estimated that about 75% of the mortality following burn injuries is related to infections rather than osmotic shock and hypovolemia [2]. The pattern of infection differs from hospital to hospital; the varied bacterial flora of infected wound may change considerably during the healing period [3].

In every hospital set up the incidence and organisms isolated will be distinct. Hence these studies are important for providing adequate and effective treatment of the cases, thereby reducing the morbidity and mortality. Burns provide a suitable site for bacterial multiplication and are more persistent richer sources of infection than surgical wounds, mainly because of the larger area involved and longer duration of patient stay in the hospital [4].

Burns are one of the most common and devastating forms of trauma and a major public health concern in all around the world [5]. The burn patients have unique predisposition to different infections which are linked to impaired resistance from disruption of the skin's mechanical integrity and generalized immune suppression. The skin barrier is replaced by a protein rich, a vascular environment that provides a favourable niche for microbial colonization and proliferation. Additionally migration of immune cells is hampered, which contributes to septic process [6-10].

In spite of considerable advances in the last 60 years in antimicrobial treatment, infection still continues to pose the greatest danger to burn patients. It was shown that approximately 73 per cent of all death within the first five days post-burn has been caused by sepsis [11-13]. Also the worldwide emergence of antimicrobial resistance among bacterial pathogens, limits the available therapeutic options for effective treatment of infections [14, 15].

The rate of nosocomial infections are higher in burn patients due to various factors like nature of burn injury itself, immune compromised status of the patient, invasive diagnostic and therapeutic procedures and prolonged ICU stay [16]. Complicating this high rate of infection is the fact that the spectrum of bacterial isolates varies with time and geographical area [17, 18]. In various countries, including India, the importance of Acinetobacter species, as a rapidly emerging nosocomial pathogen, has been documented [19] and these bacteria are predominantly isolated from ICUs, burn units and surgical wards. In addition, the problem of multi-drug resistance in gram-negative bacilli due to extended spectrum beta lactamases (ESBL) production is becoming a serious threat to the healthcare worker, who are likely to contract the infection, as the therapeutic options to these organisms are limited [20]. This necessitates periodic review of the isolation pattern and antibiogram of the burn ward, which forms the basis for modification of drug regimen strategy.

Despite the advances in patient care and the use of a large number of antimicrobial agents, infections which complicate the clinical course of patients who had sustained severe thermal injures continue to be a major unsolved problem. The present study was an ongoing outbreak of multiple drug resistant pathogens in the burn patients. Thus, the aim of the current study was to determine the antibiotic sensitivity pattern of bacterial isolates in burns wound infection in tertiary care hospital.

Aim and objectives:

- To determine the common bacterial isolates in burns wound infection in our hospital.
- To find out suitable antimicrobial agent against the

2. Materials and methods

This is a study of 1133 bacterial isolates from 1378 patients admitted in Microbilogy Labaratory, Govt. Kilpauk Medical College, between June–November 2014. The Pus swab samples were obtained before cleaning of the wounds and were processed for isolation and identification of bacterial pathogens according to the standard microbiological techniques [4]. Bacterial isolates from burns wound infections were cultured and identified. Antibiotic susceptibility testing was done by Kirby Bauer disc diffusion method as per CLSI Guidelines.

Phenotypic confirmatory test for ESBL producers among Klebsiella pneumonia were done by double disc potentiation test using Ceftazidime and Ceftazidime plus Clavulanic acid (CAZ/CAC) as per CLSIguidelines 2014. The clinical samples comprised burn wound swabs of the patient. All samples were collected from wound infections and immediately transferred under aseptic conditions to Microbilogy Labaratory where they were processed.

Samples were cultured on Nutrient agar, Mac conkey agar, Blood agar at 37ºc for 24 hrs. The isolates were identified by culture and antibiotic sensitivity determined [21].

3. Result

In the present study 1378 patients were enrolled. The most common isolate was Staphylococcus aureus followed by Klebsiella pneumoniae 354 (31.24%), Pseudomonas aeruginosa 219 (19.33%), Acinetobacter baumanii 68 (6%). Incidence of isolates in burn patients were listed in Table 1. List of antibiotics tested and the sensitivity pattern of gram positive and gram negative organisms were presented in Table 2 and 3.

Table 1 Incidence of isolates in burn patients

Organisms Isolated	No. of Organisms Isolated	%
Staphylococcus aureus	381	34
Enterococcus faecalis	17	2
Coagulase negative Staphylococcus	49	4
Escherichia coli	20	2
Klebsiella pneumoniae	354	31
Pseudomonas aeruginosa	219	19
Acinetobacter baumannii	68	6
Proteus mirabilis	25	2

- Out of 1378 samples cultured, 1133 bacterial growth isolated. Among which 224 (16%) were Polymicrobial growth and remaining were monomicrobial.

- The most common isolate was Staphylococcus aureus 381(33.63%). Other isolates were Klebsiella pneumoniae 354(31.24%), Pseudomonas aeruginosa 219(19.33%), Acinetobacter baumanii 68(6%). ESBL producers among Klebsiella pneumonia were 221(62.42%).

Table 2 Antibiotics tested and the relative sensitive pattern of gram positive organisms

Antibiotic sensitivity pattern of gram positive organisms in burns wound infection										
S.N	**Organisms**	**No. of Organisms**	**Ampicillin**	**Doxycycline**	**Erythromycin**	**Amikacin**	**Gentamicin**	**Cephalhexin**	**Ciprofloxacin**	**Vancomycin**
1	Staphylococcus aureus	381	2%	67%	27%	62%	23%	7%	27%	100%
2	Coagulase negative Staphylococcus	49	27%	64%	45%	64%	55%	9%	55%	100%
3	Enterococcus faecalis	17	33%	67%	33%	67%	NT	NT	47%	100%

Table 3 Antibiotics tested and the relative sensitive pattern of Gram negative organisms

Antibiotic sensitivity pattern of gram negative organisms in burns wound infection												
S.N	**Organisms**	**No. of Organisms**	**Ampicillin**	**Ciprofloxacin**	**Amikacin**	**Gentamicin**	**Cefotaxime**	**Ceftazidime**	**Cephalhexin**	**Piperacillin + Tazobactam**	**Imipenem**	**Levofloxacin**
1	Klebsiella pneumoniae	354	10%	15%	51%	15%	37%	38%	3%	87%	100%	100%
2	Pseudomonas aeruginosa	219	0	19%	58%	23%	32%	64%	3%	98%	100%	100%
3	Acinetobacter baumannii	68	0	10%	20%	10%	9%	50%	0%	80%	93%	100%
4	Escherichia coli	20	15%	20%	100%	65%	40%	75%	20%	100%	NT	NT
5	Proteus mirabilis	25	17%	33%	58%	25%	33%	64%	17%	100%	NT	NT

All Staphylococcus aureus isolates were sensitive to Vancomycin(100%), followed by Doxycycline (67%) and Amikacin(62%). Most of the Klebsiella pneumoniae isolates were resistant to third generation Cephalosporins (62%) but sensitive to Imipenem (100%), Levofloxacin (100%), followed by Piperacillin and Tazobactam (87%), amikacin(51%). Imipenem (100%), Levofloxacin (100%), followed by Piperacillin and Tazobactam (98%) were sensitive in most of the isolates of Pseudomonas aeruginosa.

Conclusion:

It was observed that bacteria, which were isolated from burn patients, were multidrug resistant. Furthermore Staphylococcus aureus found to be most common 381(33.63%) isolate from burn patients where as Pseudomonas spp. was highly sensitive to Imipenem and Levofloxacin (100%). This study reveals the common bacterias causing burns wound infection as Staphylococcus aureus followed by Klebsiella pneumoniae and Pseudomonas aeruginosa and their antibiotic sensitivity pattern. In Klebsiella, ESBL producers were 62.2% This study aid us in selecting appropriate antibiotics for therapy and control of infections and development of resistance strains in the

burns wound infection. In conclusion, present observations seem to be helpful in providing useful guidelines for choosing effective therapy against isolates from burn patients.

References

[1] Mc Manus A.T. MADj MWF, PBA. (1994) A decade of reduced Gram -negative infections and mortality improved isolation of burned patients. *Arch. Surg.* p.; 129: 1306-1309.

[2] Donati I Sfgmmasbff.I. (1993) Infection and antibiotic therapy in 4000 burned patients treated in Milan, Italy, between 1976 and 1988. *Burns*; p.19: 345-348.

[3] Kumar.V. BS, SA, KS, MR. (2001) Burn Wound Infection: A study of 50 cases with special reference to antibiotic resistance. *Indian Journal of Bio Research*; p.; 46: 66-69.

[4] Agnihotri N Gvjr. (2004) Aerobic bacterial isolate from burn wound infections and their antibiograms- a five- year study. *Burns*; p. 30: 241-243.

[5] Qader AR MJ. (2011) Nosocomial infection in sulaimani burn hospital, *Ann Burns Fire Disasters. Iraq*. p.; 23(4):177-81.

[6] Soares de Macedo JL SJ. (2006) Nosocomial infections in a Brazilian Burn Unit. *Burn* p.; 32(4):477-81.

[7] Ahuja RB Gagr. (2009) A prospective double-blinded comparative analysis of Framycetin and Silver Sulphadiazine as topical agents for burn. *A pilot study. Burns*. p.; 35(5):672-6.

[8] Vindenes HA UEBR. (1998) Conce0ntrations of cytokines in plasma of patients with large burns: their relation to time after injury, burn size, inflammatory variables, infection and outcome. *Eur J Surg.* p.; 164(9):647-56.

[9] Alexander M CISM (2002) Relationships between burn size, immunosuppresion, and macrophage hyperactivity in a murine model of thermal injury. *Cell Immunol.* p.; 220(1):63-9.

[10] Church D Esrowblr. (2006) Burn wound infections. *Clin Microbiol Rev*; p.; 19(2):403-34.

[11] Macedo JLS SJ. (2005) Bacterial and fungal colonization of burn wounds. *Mem Inst Oswaldo Cruz*. p.; 100(5):535-39.

[12] Taneja N Ercpsm. (2004) A prospective study of hospital-acquired infections in burn patients at a tertiary care referral centre in North India. *Burns*; p. 30(7):665-9.

[13] Vindenal H BR. (1995) Microbial colonization of large wounds. *Burns.* p.; 21(8):575-9.

[14] Singh D Sasasl. (1998) Burn mortality in Chandigarh zone: 25 years autopsy experience from a tertiary care hospital of India. *Burns*; p.; 24(2):150-6.

[15] Sharma BR Hdsvbs (20056). Septicemia as a cause of death in burns: an autopsy study. *Burns.* 2006; p.; 32(5):545-9.

[16] Pruitt Jr BA Maksgc. (1998) Burn Wound infections: current status. *World J Surg.*p.; 22:135-45.

[17] Gupta M Goyruj (1993) Burn epidemiology: The Pink City scene. *Burns,* p.; 19:47-51.

[18] Ananthakrishanan AN Krkkbs. (2002) Detection of extended spectrum beta Lactamase producers among surgical wound infections and burn patients in JIPMER. *Indian J Med Microbiol*. p. 18:160-5.

[19] Sengupta S Hpcasp. (2001) Acinetobacter bac: An emerging nosocomial pathogen in the burns unit. Manipal, India. *Burns*; p.; 27:140-4.

[20] Singh NP Grmvdskztv. (2003) Changing Trends in bacteriology of burns in the burns units, Delhi, India. *Burns.* 2003; p; 29:129-32.

[21] Standards. NcfCL (1993) Methods for Determining Bactericidal Activity of Antimicrobial Agents, Tentative Guidelines, M-26-T. NCCLS, Vilanova, PA..