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ORIGINAL RESEARCH ARTICLE

The most frequent bacterial infections in burn injuries at burn units of two hospitals in Baghdad

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Abstract

In addition to the nature and extent of the thermal injury influencing infections, the type and quantity of microorganisms that colonize the burn wound appear to influence the risk of invasive wound infection. A total of 80 patients with second degree burns were enrolled in this study with a gender ratio of 1.22:1 (44 females and 36 males). They were at both Imam Ali and Al-kindy hospitals in Baghdad. Culturing of the burn areas and antibiotic sensitivity were tested at three different times (48 h, 5th day and 10th day) of the patients entry to the burn units in hospitals to detect the most frequent microorganisms isolated from burn injuries and then determine the antibiotic they are sensitive to. The most frequent microorganisms were *Pseudomonas aeruginosa,* followed by *Klebsiella pneumoniae* and then *S. aureus*, while *Acinetobacter baumannii* has a clear percentage. Mixed culture have been also found in both survivor and non-survivor, especially *P. aeruginosa* and *K. pneumoniae*. A high percentage of resistance was found among clinical isolates in general to the commonly used antibiotics.

Keywords: burn, burn injuries, burn infection, mixed culture, burn unit infection

Percentage mortality of burns is depending on different factors. The most important factor is burn degree. Table 2 and Figure 1 show the relation between the degree of the burn and mortality. As most of the living and the dead groups are second-degree burns, Moore et al. has found that there is a relationship between the degrees of burn injury and mortality[12].

After a burn injury, most of the patients suffer from serious consequences of the bacterial infection. which are the most common challenge to fight morbidity and mortality. The rupture of the skin barrier by burn injury leads to local and systemic immune responses and share in a complication of microorganism infection[13].

The results in Table 3 show that 10 (66.6%), 2 (14.3%), and 6 (40%) of the patients in the (48 h, 5th day and 10th day, respectively) have no detectable microorganisms while microorganisms are detected in 5 (44.3%), 13 (86.6%), 9 (60%) in (48 h, 5th day, and 10th day, respectively) in non-survivor patients, respectively. Table 3 also shows that the 14 (40%), 5 (14.3%), 9 (25.7%) patients have no detectable microorganisms and 21 (60%), 30 (85.7%), 26 (74.3%) have detectable microorganisms throughout the three times samples after burn injuries in surviving patients, respectively. And positive cultures were significantly more frequent among patients in the 5th day of burn injury in two groups.

Table 4 and Figure 2 show that the most frequent microorganisms cultured from burn injuries which are *Pseudomonas aeruginosa*, followed by *K. pneumoniae* and then *S. aureus*, while *Acinetobacter baumannii* has a clear percentage.

P. aeruginosa was the most frequent, conforms to many published studies, which have reported *P. aeruginosa* as the commonest microbe cultured from burn wounds[7,14–17].

In contrast to our finding, some published studies have reported *S. aureus* as their predominant microbe of bacterial burn wound infections[18]. Mixed culture have been also found in both survivor and non-survivor, especially *P. aeruginosa* and *K. pneumoniae*, which is a common result as it has been shown in many other studies[19–21]. There is other bacteria species which are found in excluded patients like *Streptococcus pyogenes, Staphylococcus epidermidis, Proteus mirabilis.*

In general, the common cause of death in severely burning injuries is sepsis or complications of inhalation injury. Burn injuries cause anti-inflammatory response, which leads to serious consequences as an immunosuppression

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Table 1 Distribution of mortality according to gender and age

Age group	Male dead %	Female dead %	Dead total %	Male live %	Female live %	Live total %	Total %
>1	0	0	0	4.75	0	4.75	4.75
1–10	4.75	4.75	7	11.25	12.5	24.75	30.75
11–20	2.5	6.25	8.75	7.5	8.75	16.25	25
21–30	5	4.75	8.75	2.5	10	12.5	22.5
31–40	0	2.5	2.5	2.5	4.75	6.25	10
41–50	0	1.25	1.25	2.5	1.25	4.75	5
50<	1.25	0	1.25	2.5	1.25	4.75	5
Total	12.5	17.5	29.5	32.5	37.5	70	99.5

Table 2 Mortality and burn degree

Burn degree	Dead subjects no.(%)	Live subjects no. (%)	Total no.(%)
2nd & 3rd degree	11 (14.75%)	15 (18.75%)	26 (42.5%)
2nd degree	12 (15%)	40 (50%)	52 (65%)
3rd degree	1 (1.25%)	1 (1.25%)	2 (2.5%)
	24 (40%)	56 (70%)	80 (100%)

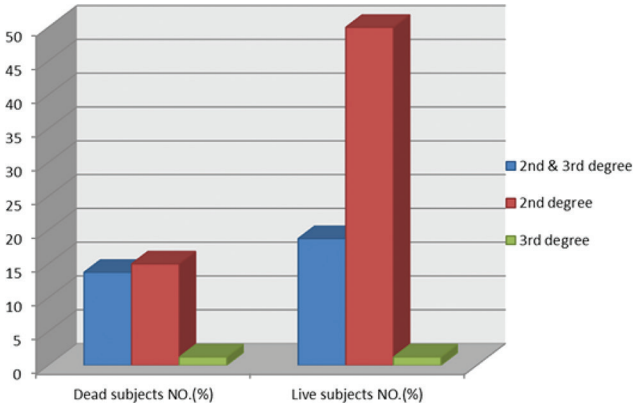

Figure 1 Mortality and burn degree.

Table 3 Percentage of bacterial culture from burn injury

Group (no)		Time of sampling		
		48 h (%)	5th day (%)	10th day (%)
Non-survivor (15)	Sterile	10 (66.6)	2 (13.3)	6 (40)
	Positive	5 (33.3)	13 (86.6)	9 (60)
Survivor (35)	Sterile	14 (40)	5 (14.3)	9 (25.7)
	Positive	21 (60)	30 (85.7)	26 (74.3)

that leads to infectious complications[1], which are characterized by the decrease of monocytes/macrophages after burn injury and sepsis[22]. But the increase of releasing of ProstaglandinE2 (PGE2) by inhibitory macrophages plays a remarkable role in immunosuppression of burn patients[1], but the real events which follow the burn injury and which lead to immunosuppression are remaining unknown.

The burn skin injury provides a rich environment of avascular necrotic tissue that supplies microorganism with a rich medium of nutrient which cause suppuration. Sometimes, the formation of scars leads to reduce migration of immune cells into the burned area and impair local host immune responses which lead to limit distribution of antimicrobial agents which are produced by the host to the burned area[23].

Normally, the human skin has a mixture of normal flora and pathogenic bacteria like *Micrococcus* spp., *Corynebacterium* spp., *Staphylococcus* spp., *Enterococcus viridans, Propionibacterium acnes, Neisseria* spp., *Peptococcus* spp. and *Brevibacterium* spp.[24], so wound swab cultures help to distinguish between infection and colonized bacteria, including gram-negative bacteria, gram-positive bacteria, and yeasts which are found normally in gastrointestinal and upper respiratory flora and/or from the hospital environment or that are transferred via a health care worker's hand. *Staphylococci* exists normally within hair follicles and sweat glands, so within 48 hr after burning, it increases in colonization unless antimicrobial drugs are used[1].

The two most general infections of burn injuries are *Pseudomonas areuginosa* and *S. aureus,* which become difficult to be treated because of multidrug-resistant. Clark et al. find out that Gram-negative bacteria are considered the most common invasive infectious agent due to having large factors of virulence and an antimicrobial resistant characteristic like we have seen in the present study[25].

The present study shows that *P. aeruginosa* has the largest percentage of burn injury infections as many studies found out that *P. aeruginosa* from the gastrointestinal flora and/or an environmental source is the most common cause of burn injury infections[5,20,21,26,27].

A Chinese study[28] reveals that the percentage of burn bacterial infection is 75.75% gram negative bacilli, 19.25% gram positive cocci and 4.99% are fungi. They have discovered a high detection rate of *A. baumannii* which are found in 1 out of 7 burn injury infection patients, while other research[29] has found that the percentage of *A. baumannii* is (12.5%) in burned patients comparing to our study which shows an approach percentage of *A. baumannii* (8.5, 5.7, 8.5%) for the three times samples of survivor groups.

Table 2 Percentage of the isolated bacteria according to different types

Groups	Survivors group (35)			Non-survivor group (15)		
Time of sampling	48 hr (%)	5th day (%)	10th day (%)	48 hr (%)	5th day (%)	10th day (%)
No growth	40	14.285	25.714	66.666	13.333	40
K. pnumoniae	22.857	25.714	14.285	6.6666	13.333	20
A. baumannii	8.571	5.714	8.571	0	6.666	0
P. aeruginosa	8.571	22.857	31.428	6.6666	26.666	20
S. aureus	8.571	5.714	2.857	6.6666	6.666	0
E. faecalis	2.857	0	0	6.6666	13.333	6.666
Proteus vulgaris	0	0	0	6.6666	13.333	6.666
Escherichia coli	0	0	0	0	0	6.666
K. pneumoniae + *E. faecalis*	2.857	0	0	0	0	0
P. aeruginosa + *K. pneumoniae*	5.714	20	17.142	0	6.666	0
P. aeruginosa+ *S. aureus*	0	2.857	0	0	0	0
K. pneumoniae + *A. baumannii*	0	2.857	0	0	0	0

Table 4 Antibiotics susceptibility of isolated bacteria

Bacterial spp.	Antibiotic resistance	Antibiotic sensitive
K. pneumoniae	Gentamicin, Ampicillin, Piperacillin, Cefotaxime, Aztreonam	Tigecycline, Imipenem
A. baumannii	Ciprodar, Piperacillin, Aztreonam, Tetracycline	Amikacin, Gentamicin, Imipenem
P. aeruginosa	Piperacillin, Ciprodar, Netilmicin, Gentamicin, Aztreonam,	Imipenem, Amikacin
S. aureus	Erythromycin, Cefotaxime, Ciprodar	Tigecycline, Tetracycline
Enterococcus faecalis	Ceftriaxone, Clindamycin, Erythromycin	Tigecycline, Ampicillin, Vancomycin, Penicillin
Proteus vulgaris	Tetracycline, Ciprodar	Cefotaxime, Piperacillin
Escherichia coli	Piperacillin, Aztreonam, Ceftriaxone, Gentaiin	Tigecycline, Imipenem
S. epidermidis	Ciprodar, Azithromycin, Trimethoprim	Vancomycin, Clindamycin, Gentamicin
Streptococcus pneumoniae	Ceftriaxone, Tetracycline, Erythromycin	Tigecycline, Vancomycin
S. pyogenes	Amoxicillin, Amikacin	Clindamycin, Vancomycin, Imipenem
P. mirabilis	Aztreonam, Tigecycline, Ciprodar, Gentamicin	Amikacin, Piperacillin, Imipenem

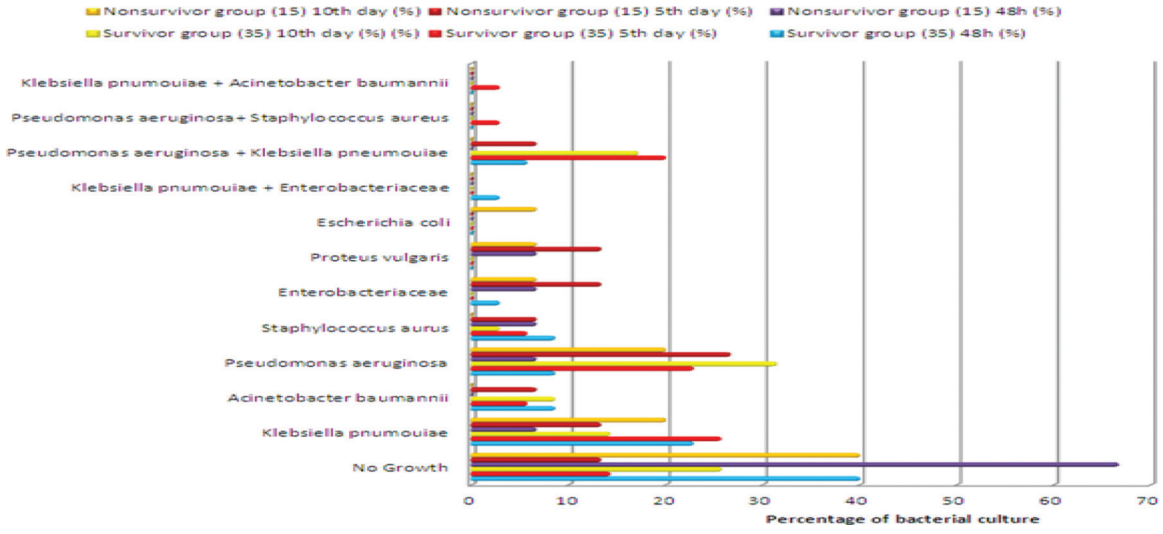

Figure 2 Comparison of all the isolated bacteria according to different types.

But still the hospital environment and multidrug-resistant bacteria are a greater risk factor for morbidity and mortality. Bacteria, which is transferred from the environment of hospitals, tend to be more resistant to antimicrobial agents than those originating from the patient's normal flora.

Table 4 shows the bacterial susceptibility to different types of antibiotics. Our results observed a variety of antibiotic resistance and susceptibility among the bacterial culture. Further studies should be done documenting the antimicrobial resistance prevalence in burn injury patients of our country in recent years. A high percentage of resistance was found among clinical isolates in general to the commonly used antibiotics[21].

Conclusions

Our study concluded that the most frequent microorganism infections in burn injuries were *P. aeruginosa*, followed by *K. pneumoniae* and then *S. aureus*, while *A. baumannii* has a clear percentage. Mixed culture have been also found in both survivor and non-survivor, especially *P. aeruginosa* and *K. pneumoniae*. A high percentage of resistance was found among clinical isolates in general to the commonly used antibiotics.

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References

1. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev. 2006;19:403–434.
2. Shahzad MN, Ahmed N, Khan IH, Waheed ABMF. Bacterial profile of burn wound infections in burn patients. Ann Pakistan Inst Med Sci. 2012;8:54–57.
3. Sharma BR. Infection in patients with severe burns: causes and prevention thereof. Infect Dis Clin North Am. 2007;21:745–759, ix.
4. Azimi L, Motevallian A, Ebrahimzadeh Namvar A, Asghari B, Lari AR. Nosocomial infections in burned patients in Motahari Hospital, Tehran, Iran. Dermatol Res Pract. 2011;2011:436952.
5. Othman N, Babakir-Mina M, Noori CK, Rashid PY. Pseudomonas aeruginosa infection in burn patients in Sulaimaniyah, Iraq: risk factors and antibiotic resistance rates. J Infect Dev Ctries. 2014;8:1498–1502.
6. van Duin D, Strassle PD, DiBiase LM, Lachiewicz AM, Rutala WA, Eitas T, et al. Timeline of health care-associated infections and pathogens after burn injuries. Am J Infect Control. 2016;44:1511–1516.
7. Raz-Pasteur A, Fishel R, Hardak E, Mashiach T, Ullmann Y, Egozi D. Do wound cultures give information about the microbiology of blood cultures in severe burn patients?. Ann Plast Surg. 2016;76:34–39.
8. Belba MK, Petrela EY, Belba AG. Epidemiology of infections in a burn unit, Albania. Burns. 2013;39:1456–1467.
9. Girerd-Genessay I, Bénet T, Vanhems P. Multidrug-resistant bacterial outbreaks in burn units: a synthesis of the literature according to the ORION statement. J Burn Care Res. 2016;37:172–180.
10. Ruth A Bryant, Denise P Nix. Acute and chronic wounds: current management concepts. Elsevier Health Sciences, USA. 5th ed. 2016;288.
11. Quinn P J, Markey BK, Leonard FC, Fitzpatrick ES, Fanning S, Hartigan P. Veterinary microbiology and microbial disease. Wiley. 2nd ed, 2011;145.
12. Moore EC, Pilcher DV, Bailey MJ, Stephens H, Cleland H. The burns evaluation and mortality study (BEAMS). Predicting deaths in Australian and New Zealand burn patients admitted to intensive care with burns. J Trauma Acute Care Surg. 2013;75:298–303.
13. Lederer JA, Rodrick ML, Mannick JA. The effects of injury on the adaptive immune response. Shock. 1999;11:153–159.
14. Saaiq M, Ahmad S, Zaib MS. Burn wound infections and antibiotic susceptibility patterns at Pakistan institute of medical sciences, Islamabad, Pakistan. World J Plast Surg. 2015;4:9–15.
15. Coyle RM, Harrison HL. Emergency care capacity in Freetown, Sierra Leone: a service evaluation. BMC Emerg Med. 2015;15:2.
16. Hussien IA, Habib KA, Jassim KA. Bacterial colonization of burn wounds. J Baghdad Sci. 2012;9:623–631.
17. Japoni A, Kalani M, Farshad Sh, Ziyaeyan M, Alborzi A, Mehrabani D, et al. Antibiotic-resistant bacteria in hospitalized patients with bloodstream infections: analysis of some associated factors. Iran Red Crescent Med J. 2010;12:164–172.
18. Pasalar M, Mohammadi AA, Rajaeefard AR, Neghab M, Tolidie HR, Mehrabani D. Epidemiology of burns during pregnancy in southern Iran: effect on maternal and fetal outcomes. World Appl Sci J. 2013;28:153–158.
19. Dryden MS. Complicated skin and soft tissue infection. J Antimicrob Chemother. 2010;65:35–44.
20. Saha SK, Muazzam N, Begum SA, Chowdhury A, Islam MS, Parveen R. Study on time-related changes in aerobic bacterial pattern of burn wound infection. Faridpur Medical College J. 2011;6:3–5.
21. Elmanama AA, Laham NA, Tayh GA. Antimicrobial susceptibility of bacterial isolates from burn units in Gaza. Burns. 2013;39:1612–1618.
22. Gamelli RL, He LK, Liu H. Marrow granulocyte-macrophage progenitor cell response to burn injury as modified by endotoxin and indomethacin. J Trauma. 1994;37:339–346.
23. Nasser S, Mabrouk A, Maher A. Colonization of burn wounds in Ain Shams university burn unit. Burns. 2003;29:229–233.
24. Wysocki AB. Evaluating and managing open skin wounds: colonization versus infection clinical issues. American Association of Critical-Care Nurses. 2002;13:382–397.
25. Clark NM, Patterson J, Lynch JP. Antimicrobial resistance among gram-negative organisms in the intensive care unit. Curr Opin Crit Care. 2003;9:413–423.
26. Altoparlak U, Erol S, Akcay MN, Celebi F, Kadanali A. The time-related changes of antimicrobial resistance patterns and predominant bacterial profiles of burn wounds and body flora of burned patients. Burns. 2004;30:660–664.
27. Hammoudi AA. Association of pathogenic bacterial isolates in burn wound infections. Med J Babylon. 2013;11:52–57.
28. Chen B, Li X, Zhang Z, Zhang X, Deng Z, Zhong X, et al. Drug resistance and status of infection of Acinetobacter baumannii in burn intensive care unit during 3 years. Zhonghua Shao Shang Za Zhi. 2015;31:21–24.
29. Shaykh Baygloo N, Bouzari M, Rahimi F, Abedini F, Yadegari S, Soroushnia M, et al. Identification of genomic species of Acinetobacter isolated from burns of ICU patients. Arch Iran Med. 2015;18:638–642.