Original Article

Bacteriology of the burn wound at the Bai Jerbai Wadia Hospital for children, Mumbai, India—a 13-year study, Part I-Bacteriological profile

Shankar Srinivasan, Arvind M. Vartak, Aakanksha Patil, Jovita Saldanha

Department of Burns and Plastic Surgery, B.J. Wadia Hospital for Children, Parel, Mumbai, India

Address for correspondence: Dr. Shankar Srinivasan, 91/22, Bhaveshwar Vijay, Wadala, Mumbai-400 031, India.

E-mail: plastic_shankar@hotmail.com

ABSTRACT

Aim: To study which organisms were prevalent in our burn unit and their antibiotic sensitivity pattern in brief. **Method:** Microbiological data of 1534 patients admitted to the burns unit of the Bai Jerbai Wadia Hospital for Children, Mumbai over a period of 13 years (1994-2006) was reviewed retrospectively. A total of 9333 swabs were cultured and antibiotic sensitivities to the isolated organisms determined. The age group of patients admitted to our facility ranged from one month to 15 years. **Result:** *Klebsiella* was the predominant organism in our set-up (33.91%), closely followed by *Pseudomonas* (31.84%). The antibiotic sensitivities of the isolated organisms are discussed in detail in the text. **Conclusion:** Every treatment facility has microorganisms unique to it and these change with time. It is therefore of paramount importance to have an in-depth knowledge of the resident organisms and their antibiotic sensitivity pattern so that infection-related morbidity and mortality are improved.

KEY WORDS

Bacteriology in burns; burn wounds; paediatric burns

INTRODUCTION

Burn injury is a major problem in many parts of the world. It has been estimated that 75% of all deaths following burns are related to infection. Thermal injury destroys the skin barrier that normally prevents invasion by microorganisms. This makes the burn wound the most frequent origin of sepsis in these patients.^[1]

Initially, the burnt area is considered free of microbial contamination. But gram-positive bacteria in the depth of sweat glands and hair follicles heavily colonize the

wounds within 48 h of the injury.^[2,3]

Topical antimicrobials decrease microbial overgrowth but seldom prevent further colonization with other potentially invasive bacteria and fungi. These are derived from the patient's gastrointestinal and upper respiratory tract and the hospital environment.^[4,5]

Following colonization, these organisms start penetrating the viable tissue depending on their invasive capacity, local wound factors and the degree of the patient's immunosuppression.^[5] If sub-eschar tissue is invaded,

disseminated infection is likely to occur.^[3] Great emphasis must therefore be placed on early identification of local signs of invasive burn wound infection.

The causative infective microorganisms in any burn facility change with time.^[6,7] Individual organisms are brought into the burns ward on the wounds of new patients. These organisms then persist in the resident flora of the burn treatment facility for a variable period of time, only to be replaced by newly arriving microorganisms. Introduction of new topical agents and systemic antibiotics influence the flora of the wound.^[6,7]

Thus, it is just not sufficient to be aware of the microorganisms that pose a problem for burn patients. To have an in-depth knowledge of the organisms that are predominant in that particular treatment facility during the particular period along with their sensitivity pattern is vital as many septic burn patients need to be treated with antibiotics before the results of microbiological cultures are available. This would be crucial to reduce the overall infection-related morbidity and mortality.

In the present study, we determined the nature of microbial wound colonization in 1534 patients. The major objectives were to determine:

- Which microorganisms were prevalent in our treatment facility,
- Their antibiotic sensitivity pattern.

MATERIAL AND METHODS

Patients

This is a retrospective analysis of the study of isolates from the burns unit of Bai Jerbai Wadia Hospital for children, Mumbai. The hospital caters exclusively to a paediatric population. In our study, the youngest child was a month old and the oldest, 15 years old. Between 1994 and 2006, a total of 9333 samples were processed. The sex distribution of the patients and the aetiology of burns are presented in Tables 1 and 2. It is interesting to note that in our series, male children outnumbered females by 13.2%. Mortality figures are presented in Table 3. This study focuses exclusively on the microbiological profile and no attempt has been made to correlate this with clinical data. We desire to do this as a separate study.

Wound treatment

Closed dressings using silver sulphadiazine ointment

were used in all patients without exception. The burn wounds were washed daily to remove necrotic tissue and the remnants of the previous day's ointment.

Procedure for wound sampling

Microbial colonization of all wounds was studied from the time of admission to discharge. On admission, the sampling procedure included swabs that were taken from clinically deep areas of the burn wound prior to any cleansing. Swabs were taken twice weekly. The bandages were removed, the remnants of the previous day's ointment were washed away and the wounds were swabbed and cultured as follows: A sterile cotton swab is moistened with sterile normal saline. This swab is rubbed onto the burn wound surface. Swabs are taken from areas which appear deep, areas with discharge, thick eschar, etc. The swabs are then sent for culture.

Microbiology

The swabs are transported to the laboratory for processing immediately. They are streaked onto a differential medium (e.g.; Mac Conkey agar) and an enriched medium (e.g.; blood agar). Isolation is carried out by the conventional T-method using sterile nichrome loop. These plates are incubated at 37 °C for 16-18 h. The basic aim was to isolate the organisms predominant on the burn wound and determine their sensitivity to various antibiotics for clinical purposes.

Antibiotic sensitivity of isolates obtained from the burn wound was carried out by filter paper disc diffusion method (Kirby Bauer method). Sterile commercially available filter paper discs, onto which a definite amount of antibiotic has been absorbed, are used. Since the antibiotic in the disc tends to diffuse more onto the surface of the agar than into the deeper layers, the plate is surface spread with the organisms. A broth culture of the isolate is prepared using sterile peptone water comparable to 0.5 Macfarlands turbidity standard (i.e. 1×10^7 to 1×10^8 organisms/ml). Approximately 0.2 ml of this broth culture is surface spread onto sterile Mac Conkey agar plate (for gram-negative organisms)/sterile blood agar plate (for gram-positive organisms), so as to get a matt growth.

Sterile antibiotic discs are equidistantly placed on these plates and gently pressed onto the medium with the help of sterile forceps to ensure complete contact with the agar surface. The plates are incubated at 37°C for 16 to 18 h.

Table 1: Age sex distribution age - 1 month - 15 years

Year	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	Total															
No. of Patients	112	129	153	144	157	132	135	122	92	96	77	73	112	1534															
	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	*Tested for															
Male	58	51.8	75	58.1	84	54.9	70	48.6	92	58.6	80	60.6	81	60.0	72	59.0	56	60.9	58	60.4	44	57.1	42	57.5	50	44.6	1534	868	56.6
Female	54	48.2	54	41.9	69	45.1	74	51.4	65	41.4	52	39.4	54	40.0	50	41.0	36	39.1	38	39.6	33	42.9	31	42.5	62	53.9	1534	666	43.4

Table 2: Aetiology

Year	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	Total															
Total	112	129	153	144	157	132	135	122	92	96	77	73	112	1534															
	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	No.	*Tested for															
Scalds	82	73.2	98	76.0	105	68.6	108	75.0	124	79.0	100	75.8	83	61.5	96	78.7	65	70.7	75	78.1	62	80.5	56	76.7	94	83.9	1534	1152	75.1
Flame	15	13.4	18	13.9	25	16.4	22	15.3	15	9.6	20	15.1	32	23.7	18	14.8	14	15.2	14	14.6	8	10.3	9	12.3	9	8.0	1534	221	14.4
Crackers	3	2.7	3	2.3	16	10.4	4	2.8	5	3.2	4	3.0	7	5.2	0	0.0	2	2.2	3	3.1	0	0.0	1	1.4	4	3.6	1534	52	3.4
Contact	9	8.0	5	3.9	5	3.3	5	3.5	2	1.3	5	3.7	4	3.0	3	2.5	4	4.3	2	2.1	2	2.6	2	2.7	1	0.9	1534	49	3.2
Electrical	1	0.9	4	3.1	2	1.3	3	2.1	5	3.2	1	0.8	6	4.4	2	1.6	4	4.3	2	2.1	4	5.2	3	4.1	3	2.7	1534	40	2.6
Chemical	2	1.8	1	0.8	-	-	0	0.0	4	2.5	1	0.8	3	2.2	0	0.0	0	0.0	0	0.0	1	1.3	2	2.7	1	0.9	1534	15	1
Tar	0	0.0	0	0.0	0	0.0	1	0.7	2	1.3	1	0.8	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	1534	4	0.3
Frostbite	0	0.0	0	0.0	0	0.0	1	0.7	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	0	0.0	1534	1	0.1

Zones of inhibition are measured in millimetres and the organisms classified as sensitive or resistant according to the zone size interpretation chart. It must be noted that our antibiotic sensitivities were not carried out on Mueller Hinton agar as advocated by some authors [20,21]. Subsequently, we have carried out a comparative study and tested antibiotic sensitivities for 10 different burn wound isolates on Mueller Hinton and Blood agar/ Mac Conkey agar and found no significant difference in the [Figure 1 & 2] results.

RESULTS

In the present study, a total of 9333 samples were processed from patients admitted to the burns unit; 1281 samples (13.72%) showed absence of bacterial pathogens [Table 4].

The most common isolate was *Klebsiella* (33.91%) followed by *Pseudomonas aeruginosa* (31.84%). A detailed break-up is given in Table 5.

Figure 1: Comparative study with mueller hinton agar and blood agar

Figure 2: Comparative study with mueller hinton and mac conkey agar

Table 3: Mortality statistics

Year	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	Total
Total no. of Patients	112	129	153	144	157	132	135	122	92	96	77	73	115	1534
No. of Deaths	No. 11, % 9.8	No. 8, % 6.2	No. 13, % 8.0	No. 12, % 8.3	No. 14, % 8.9	No. 13, % 9.8	No. 20, % 14.8	No. 18, % 14.8	No. 12, % 13.0	No. 13, % 13.5	No. 7, % 9.1	No. 6, % 8.2	No. 8, % 7.0	*Tested for No. 1534, % 10.1

Table 4: Bacteriological studies

Year	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	Total
Patients	112	129	153	144	157	132	135	122	92	96	77	73	112	1534
Total	No. 336, % 17.3	No. 524, % 14.1	No. 782, % 22.4	No. 1184, % 20.2	No. 1244, % 17.1	No. 1080, % 16.5	No. 949, % 13.2	No. 879, % 11.9	No. 472, % 12.7	No. 519, % 6.0	No. 422, % 29	No. 383, % 47	No. 559, % 63	*Tested for No. 1534, % 9333
No	58	74	175	202	205	107	125	105	60	31	29	47	63	1281
Growth	278	450	607	982	1039	973	824	774	412	488	393	336	496	8052
Isolates	379	696	1016	1931	2059	1557	1530	1362	689	972	805	509	634	1534

A detailed analysis on individual microorganisms and their antibiotic sensitivities, along with changing trends over this 13-year period is presented. What follows is a bird's eye view of the microorganism and its dominant sensitivity pattern.

Klebsiella was sensitive to Gatifloxacin (86.3%)

Cefaperazone+Sulbactam (82.8%)

Piperacillin+Tazobactam (77.4%)

Meropenem (72.4%)

Amikacin (66.9%)

Azithromycin (60.4%).

Pseudomonas was sensitive to Cefoperazone+Sulbactam (73.9%)

Piperacillin+Tazobactam (72.2%)

Amikacin (62.3%)

Azithromycin (56.3%)

Meropenem (55.8%)

Gatifloxacin (49.9%).

S. aureus was sensitive to Sparfloxacin (90.4%)

Cefpirome (80.9%)

Piperacillin+Tazobactam (78.4%)

Netilmicin (77.2%)

Imipenem (64%)

Erythromycin (51.1%).

E. coli was sensitive to Ticarcillin+Clavulanic acid (67.2%)

Meropenem (63.6%)

Amikacin (42.7%)

Azithromycin (27.4%)

Gatifloxacin (61.9%)

Cefoperazone+sulbactam (69.1%).

Proteus was sensitive to Piperacillin+Tazobactam (97.1%)

Meropenem (82.9%)

Ceftriaxone and Ceftizoxime (64.6%)

Gatifloxacin (62.9%)

Amikacin (55.8%)

Azithromycin (47.8%).

DISCUSSION

Thermal injury destroys the barrier function of skin, allowing microbial colonization of wounds and even with the use of topical antimicrobials, contamination of wounds is unavoidable.

The type and amount of microorganisms on and in the injured tissue influence wound healing,^[7] the frequency

incidence of *Staphylococci* is on the decline from 2002 to 2005.

CONCLUSION

It may be concluded that the composition of bacterial flora in burns is dependent not only on the depth and extent of the burn but also on the site of burn, the duration of burn, the age of the patient and his/her co-morbidities.^[15] Burn wound monitoring requires the study of changing bacterial flora and the antibiotic sensitivity reports. Repeated swab cultures and antibiograms are advised for proper selection of antibiotics to control sepsis.^[18] The development of resistance to a particular antibiotic is dependent on the use of that antibiotic in society at large. Overuse of any antibiotic predisposes to development of resistance. Our unit gets patients from all over Mumbai, other parts of the state of Maharashtra and at times, from other states too. Due to this huge diversity, we have a particular microorganism predominant at a particular point in time, but then, it is also difficult to comment on the source of the changing trends.

REFERENCES

- Vindenes, Jerknes. Microbial colonization of large wounds. *Burns* 1995;21:575-9.
- Luterman A, Dasco CC, Curreri PW. Infection in burn patients. *Am J Med* 1986;81:45-52.
- Mooney DP, Gamelli RL. Sepsis following thermal injury. *Comp Ther* 1989;15:22-9.
- Monafo WW, Freedman B. Topical therapy for burns. *Surg Clin North Am* 1987;67:133-45.
- Hansbrough JF. Burn wound sepsis. *J Intensive Care Med* 1987;2:313-27.
- Pruitt BA Jr, McManus AT. Opportunistic infections in severely burnt patients. *Am J Med* 1984;76:146-54.
- Manson WL, Pernot PC, Fidler V, Sauer EW. Colonisation of burns and duration of hospital stay of severely burned patients. *J Hosp Infect* 1992;22:55-63.
- Revathi G, Puri J, Jain BK. Bacteriology of burns. *Burns* 1998;24:347-9.
- Mehta M, Dutta P, Gupta V. Bacterial isolates from burn wound infections and their antibiograms. *Indian J Plast Surg* 2007;40:25-8.
- Agnihotri N, Gupta V, Joshi RM. Aerobic bacterial isolates from burn wound infections and their antibiograms: A 5 year study. *Burn* 2004;30:241-3.
- Ahmad M, Shahid Hussain S, Ibrahim Khan M, Malik SA. Pattern of bacterial invasion in burn patients at the Pakistan institute of medical sciences, Islamabad. *Ann Burns Fire Disasters* 2006;19:18-21.
- Pezzino T, Cucchiara B, Vitale R, Benigno A, Cucchiara P, D'Arpa N, *et al.* Analysis of bacteriological monitoring in patients at the Palermo Burns Centre-A 5 yr experience. *Annals of the MBC-vol 2-n2-June 1989*. p. 72-8.
- Ozumba UC, Jiburum BC. bacteriology of burn wounds in Enugu, Nigeria. *Burns* 2000;26:178-80.
- Kehinde AO, Ademola SA, Okesola AO, Oluwatosin OM, Bakare RA. Pattern of bacterial pathogens in burn wound infections in Ibadan, Nigeria. *Ann Burns Fire Disasters* 2004;18:12-5.
- Thomsen M. The Burns unit in Copenhagen. *Scand J Plast Reconstr Surg* 1970;4:126-39.
- Edward-Jones V, Greenwood JE. What's new in burn microbiology? James laing Memorial Prize Essay 2000. *Burns* 2003;29:15-24.
- Steer JA, Papini RP, Wilson AP, McGrouther DA, Parkhouse N. Quantitative microbiology in the management of burn patients, I: Correlation between quantitative and qualitative burn wound biopsy culture and surface alginate swab culture. *Burns* 1996;22:173-6.
- Bairy I, Shivananda PG. Aerobic bacterial flora of burn wound infection. *Indian J Surg* 1997;59:215-8.
- Nasser S, Mabrouk AM, Maher A. Colonisation of burn wounds in Ain Shams Univ Burns Unit. *Burns* 2003;29:229-33.
- Bauer, Kirby, Sherris and Turck, 1996, *Am. J. Clin. Path.*, 45:493.
- Performance Standards for Antimicrobial Disk Susceptibility Test, CLSI Vol. 28 No. 1, Jan. 2008.

Source of Support: Nil, **Conflict of Interest:** None declared.