Indian J Med Res 130, December 2009, pp 758-764

Prospective analysis of nosocomial infections in a Burn Care Unit, Turkey

O. Oncul, E. Ulkur* A. Acar*, V. Turhan, E. Yeniz**, Z. Karacaer & F. Yildiz*

*Gulhane Military Medical Academy, Haydarpasa Training Hospital, Department of Infectious Diseases & Clinical Microbiology, *Department of Plastic & Reconstructive Surgery, Uskudar-Istanbul & **Sarikamis Military Hospital, Kars, Turkiye*

Received March 10, 2008

***Background & objectives*: Prevention of infection in burned patients poses a great challenge as infection is the most common cause of mortality after burn injury. An analysis of burned patients, admitted and treated between January 2004 and December 2005 in a nine-bed burn unit in Turkey, was performed prospectively to identify the common pathogens and incidence of nosocomial infection in these patients.**

***Methods*: Of the 182 burn cases admitted to Burn Care Unit during the study period, 169 met the inclusion criteria. Information related to nosocomial infection (NI) was collected. Samples were collected for culture and microorganisms isolated were tested for antimicrobial sensitivity.**

***Results*: Of the 169 burn patients, 127 acquired 166 nosocomial infection (NI) (15.7% pneumonia, 56.0% burn wound infection, 8.4% urinary tract infection and 19.9% blood stream infection) with an overall NI rate of 18.2 per 1000 patient-days. The mean age (38 ± 21 yr), the mean length of hospitalization (45.06 ± 11.67 days) and the total burned surface area (TBSA) (34.58 ± 18.46%) of the patients with NI were higher than those of the patients with non NI (23 ± 17 yr), (16.38 ± 11.14 days) and (12.44± 8.69%) (P=0.03, P=0.001, P=0.01) respectively. By multiple logistic regression analysis, TBSA co-morbidities, broad spectrum antibiotic usage and invasive devices usage were significantly related to acquisition of NI. *Pseudomonas aeruginosa* (57%), *Acinetobacter baumannii* (21%) and *Staphylococcus aureus* (14%) were the most common resistant organisms isolated.**

***Interpretation & conclusion*: Our findings emphasize the need for careful disinfection and more strict infection control procedures in areas that serve immunosupressed individuals, such as burn patients.**

Key words Antibacterial resistance - burn - nosocomial infection

Infection is the most common cause of death following burn injury. Burn patients are at a high risk for infection as a result of the nature of the burn injury itself, the immunocompromising effects of burns, prolonged hospital stays, and intensive diagnostic and therapeutic procedures[1]. In addition, the control and prevention of infectious diseases among burned patients present a greater and more specialized problem, because the skin barriers are disrupted, the environment in burn units can become contaminated with resistant organisms,

and these organisms can be transmitted easily from one patient to another. Thus, burn care units (BCU) can be the site of explosive and prolonged outbreaks caused by resistant organisms[2,3].

Although eradication of infection in burn patients is impossible, a well conducted surveillance, infection control and prevention programme can help reduce the incidence. It is known that effective surveillance and infection control may reduce infection, mortality rates, length of hospitalization and associated costs. The objective of this prospective study was to identify the most common pathogens and their antibacterial patterns, the incidence of nosocomial infection (NI) related to invasive device usage in A Turkish BCU. Also the risk factors for acquisition of NI in burn patients were also assessed.

Material & Methods

Study setting: The study was conducted at Gulhane Military Medical Academy, Haydarpasa Training Hospital, a 1000-bed hospital with a nine-bed BCU. This is one of the referral centres for thermal injuries for patients in Istanbul, Turkey. Data were obtained during a two years period (January 01, 2004 to December 31, 2005). Four trained investigators participated in this study, including specialist of Infectious Diseases and Clinical Microbiology who served as the head of the team, specialist of plastic and reconstructive surgery, a physician and a nurse. Medical records for all patients, as well as incidence of NI were reviewed during the study period. All the patients admitted to the BCU were eligible for the study, if they met the following criteria: total burn surface area (TBSA) >10 per cent, length of stay in hospital more than 48 h, survival more than 48 h, age ≥ 12 yr and infected as per the criteria of the National Nosocomial Infections Surveillance (NNIS) System[4]. The parameters extracted were: age, gender, transfer from other hospital, invasive devices usage, broad-spectrum antibiotic (beta-lactam/beta-lactamases, quinolones, 3rd generation cephalosporins, glicopeptides and carbapenems) usage in last month, the mean admission days (the mean time between burn trauma and hospital admission), co-morbidities (including diabetes mellitus, malignancy, ischaemic heart disease, hypertension, asthma, chronic obstructive airways disease, autoimmun diseases, previous cerebrovascular accident, Parkinson's disease, psychiatric illness), total burn surface area (TBSA), presence of full-thickness burns, length of stay, duration of ventilation on admission, antibiogram of microorganisims.

Any infections manifested during the management of a burn victim were followed carefully. In this study, NIs were classified into four main groups: pneumonias, bloodstream infections (BSI), burn wound infections (BWI) and urinary tract infections (UTI); and diagnosis was estabilished by the definitions given by the Centers for Disease Control (CDC), Atlanta[5]. According to this criteria, NI was defined as one that was neither present, nor incubating at the time of the patient's admission but had its onset during hospitalization[6]. Rates of wound colonization not attributable to infection, and bacteraemia were also noted for all patients. Burn wound infections were defined as Table I[7].

Assesment and management of burns patients: Extent and severity of burns was calculated in admission days by assessing total percentage TBSA and the acute physiology, age, chronic health evaluation (APACHE) III score[8].

Table I. Classification of burn wound infections

Burn wound cellulitis:
Localized pain, tenderness, oedema, erythema, and heat
Involves unborned and undamaged skin at margin of burn or skin graft donor site
May be associated with lympangitis and systemic signs of infection
Diagnosis by clinical signs, wound culture, or both
Burn wound infections:
Invasive infections of unexcised eschar –
Modest to profound changes in appearance of burn wound
Diagnosis by histologic examination of viable tissue in wound biopsy
Invariably associated with systemic signs of infection
Infection of excised burn wound or donor site –
Formation of neoeschar or focal necrosis on wound surface (or both)
Diagnosis by histologic examination of viable tissue in wound biopsy
Infection of "grafted" wound –
Histologic examination of wound bed biopsy may be required to differentiate microbial colonization of nonviable graft from infection of wound bed
Culture of graft and wound to identify β-hemolytic streptococci
Burn wound impetigo:
Loss of epithelium (initially focal and may become generalized) from a previously grafted or healed burn wound or skin graft donor site
May be associated with systemic signs of infection
Diagnosis by wound culture (most often caused by *Staphylococcus*)

As per our BCU practices, early removal of necrotic tissue, topical burn wound creams, wound closure, aggressive surgical approach, autograft if donor sites were available or allograft if donor sites were not available, blood transfusion, use of nutrition, routine isolation of patients and strictly infection control procedures were introduced.

Skin grafting was introduced in BCU as soon as the cases were surgically fit, and repeated till all areas of the body were grafted. Superficial and mid dermal burns not requiring surgery were dressed with silver-containing dressing (Acticoat, Smith and Nephew, Istanbul, Turkey) or 0.5 per cent chlorhexidine acetate (CA) (Bactigrass, Smith and Nephew Istanbul, Turkey) or Acticoat™ (Smith and Nephew, Istanbul, Turkey) to permit moist wound healing.

Prophylactic antibiotics were given to the burn patients with inhalation injury, poor immunestatus and an open burn wound that did not close within a few days. It was also used, if broad excisional surgery was planned in the cases[9].

In order to control of catheter-related bloodstream infection, evidence-based procedures recommended by the CDC were implemented[10]. The recommended procedures were hand washing, using full-barrier precautions during the insertion of central venous catheters, cleaning the skin with chlorhexidine, avoiding the femoral site if possible, and removing unnecessary catheter[11].

Microbiological procedures: For patients meeting the inclusion criteria, subeschar tissue biopsy cultures were taken twice per week and two consecutive blood cultures, urine and sputum cultures were drawn during fever or clinical features of sepsis. Endotracheal aspirates were taken if the patients had inhalation injury, entubation and pneumonia is suspected. Subescar tissue biopsy was taken for quantitative culture, and specimens were processed according to standard histologic methods[5]. In viable tissue of groin, axilla and nares swab were also taken from all the patients for culture on admission days and were evaluated for methicillin resistant *Staphylococcus aureus* (MRSA) and vancomicin resistant enterococci (VRE)[12,13].

Blood samples for culture were incubated at 37^0C in a BACTEC 9240 cabinets and monitored on a continuous basis. Identification was performed by convantional method[14]. The antimicrobial susceptibility of bacteria was determined using the disc diffusion method[14]. Isosensitest agar and antibiotic discs were obtained from Oxoid Limited (Basingstoke, UK).

The Gram positive isolates were tested in µg against penicillin (10), erythromycin (15), gentamicin (10), amikacin (30), netilmicin (30), ciprofloxacin (5), moxifloxacin (5), chloramphenicol (30), trimethroprim-sulphamethoxazole (1·25/23·75), tetracycline (30), doxycycline (30), vancomycin (30) and fusidic acid (10). The Gram negative isolates were tested and sensitivity, intermediate sensitivity and resistance were determined by the zone of complete growth inhibition around each disc according to the reference standard[15].

Swab samples in Amies' transport medium (Copan, Italy) were inoculated on a commercial selective enterococcal agar containing 6 mg/l of vancomycin (Novamed, Jerusalem) and incubated aerobically at 35°C. VRE (*Enterococcus faecium* and *E faecalis*) were isolated and identified by standard methods[15]. *Staphylococcus aureus* ATCC 25923 and *E. faecalis* ATCC 29212 were used as reference standards to check quality of routine and high content discs respectively.

The isolates of *P. aeruginosa* and *Acinetobacter* spp. were defined as 'panresistant' if they are resistant to all six available anti-pseudomonal classes of antimicrobial agents, namely anti-pseudomonal penicillins, cephalosporins, carbapenems, monobactams, quinolones and aminoglycosides and polymyxins except from polymixin B[16].

The protocol of this study was approved by the Ethical Committee of the Institution.

Statistical analysis: Risk factors [age, gender, APACHE III score and Multiple Organ Dysfunction Score (MODS) at the admission day, TBSA, the mean length of hospital stay, the mean admission days, operation time, the mean white blood cells (WBC), co-morbidities, transfer from other hospital, invasive device usage and prior broad spectrum antibiotic usage] for NIs were determined using Chi square test and univariate analysis. All significant variable and risk factors were then entered into a multivariate model. An explanatory model was opted, backward conditional was used as the selection method, and a relatively high significance level (P<0.5) was applied for each variable retained in the model[17]. Statistical analyses were performed using SAS 10.0 packet programme (SAS Institute, Cary, NC). All data were expressed as mean ± standard deviation (mean ± SD).

Results

Of the 182 admitted patients, 13 died during the first 48 h because of non infectious (delayed admission, osmotic shock and hypovolemia) causes, remaining 169 (92.8%) presented no infection within the first 48 h of admission, and included in the study.

Of the 169 burned patients, 108 (63.9%) were male and rest female. The mean age was 33 ± 6 yr (range: 12-90 yr), 27 (16.0%) were older than 60 yr. In study group, 21 (12.4%) were admitted from another hospital. The most common cause of burn was exposure to flame, 147 (87%), exposure the boiling water (7%) and electrical injury (2%), the remaining patients had injury with boiling liquids. The mean length of hospital stay was 26 ± 17 days (range: 3-79 days). The mean TBSA of 169 burned cases was 29.62 ± 13.27 per cent (range: 10-97%). A total of 152 patients (89.9%) underwent surgical intervention during the hospitalization (Table II).

Among 169 burned patients, 127 acquired 166 NI (15.7% pneumonia, 56.0% BWI, 8.4% UTI and 19.9% BSI for an overall NI rate of 18.2 per 1000 patient-days). Of all the BSI, 69.7 per cent were accompanied with BWI and 30.3 per cent of that related to catheter usage. The rates of central vascular catheter, urinary catheter and ventilator usage per 1000-hospitalization days were 0.52, 0.85 and 0.31, respectively. On the other hand, the NI rates related to central vascular catheter, urinary catheter and ventilator usage were 8.3, 2.16 and 11.0 per 1000-invasive device usage days, respectively. The mean age (38 ± 21 yr), the mean length of hospitalization (45.06 ± 11.67 days) and the TBSA (34.58 ± 18.46%) of the patients with NI were higher than those of the patients without NI (23 ± 17 yr), (16.38 ± 11.14 days) and (12.44% ± 8.69%) (P=0.03, P=0.001, P=0.01) respectively. There were no significant differencies in the mean admission days, the mean WBC, co-morbidities between the groups. Totally, 143 (84.6%) patients used antibacterial therapy and 34 (20.1%) received prophylactic antimicrobials. Thirty nine patients received antimicrobial therapy by day 3, 47 by days 4-7 and 57 by day 8. Twenty six patients (15.4%) who had lesser than 10 per cent TSBA did not receive any antimicrobial therapy, whereas, 16 patients who had 23 per cent TSBA and did not have NI received antibiotics in the first three days prophylactically. All the treatments were curative after the third days. The most frequently administered prophylactic antibiotics were amoxicillin-clavulanic acid (62%) and sulbactam-ampicillin (38%). All but one of the 15 patients who died had infections.

Table II. Results of univariate analysis of potential risk factors for acquisition of nosocomial infection in burned patients

Factor	Infected patients (n=127)	Non infected patients (n=42)	*P*
Age (yr)	38.29±21.67	23.36±17.72	0.04
Gender:			
Male	83	25	0.02
Female	44	17	0.06
APACHE III	12±1	5±1	0.001
MODS	3.6±0.2	1.2±0.3	0.02
TBSA (%)	34.58±18.46	12.44±8.69	0.001
The mean length of hospital stay (days)	45.06±11.67	14.38±11.14	0.001
The mean admission days	8±2	2±1	0.06
The mean WBC	13300±1200	16200±1700	0.17
Operation time (h)	2.58	0.58	0.026
Co-morbidities	40 (32%)	6 (14%)	0.12
Transfer from other hospital	42 (33%)	4 (10%)	0.03
IDU	119 (94%)	22 (52%)	0.001
Prior broad spectrum antibiotic usage	49 (39%)	7 (17%)	0.01

APACHE, acute physiology and chronic health evaluation; MODS, multiple organ dysfunction score; TBSA, total burn surface area; WBC, white blood cell; IDU, invasive device usage. The mean admission days: The mean time between burn trauma and hospital admission.

Risk factors: By using univariate analysis, age, gender (male), APACHE III score, MODS, TBSA, the mean length of hospital stay, operation time, transfer from other hospital, IDU and prior broad spectrum antibiotic usage were found as the risk factors for NI (Table II).

In the step-wise forward logistic regression undertaken to control the effect of confounding variables, the variables found to be significantly associated with NI were TBSA (odds ratio (OR: 3.1; 95% CI 1.8-6.5), co-morbidities (OR: 2.3; 95% CI 1.4-4.6), broad spectrum antibiotic usage (OR: 1.9; 95% CI 0.8-3.8) and usage of invasive devices (OR: 2.2; 95% CI 1.0-5.5) were significantly related to acquisition of NI. On the other hand, APACHE III scores (≥10) on admission were significant independent predictors of NI (Table III).

A total of 182 microorganisms were isolated from 127 patients: *Ps. aeruginosa* (104, 57%), *Acinetobacter baumannii* (38, 21%) and *Staph. aureus* (25, 14%) were the most common resistant organisms. Frequency of *P. aeruginosa* and *Staph. aureus* on admission day were 9 and 87 per cent, and on seven day of the admission

were 79 and 11 per cent, respectively. Methicillin-resistant strains accounted for 40 per cent of isolated *Staph. aureus*. In groin, axilla and nares cultures, MRSA was isolated in 18 (10.7%) of the cases and 15 of those were transferred from another hospital. That was 60 per cent of all the isolated MRSA from burn cases. No vancomicin resistant enterococci was isolated. The most effective antibacterial agents for *Ps. aeruginosa* and *A.baumannii* were meropenem (65 and 62%) and cefaperazon-sulbactam (63 and 74%) respectively (Table IV). Eight (7.7%) *Ps. aeruginosa* and five (13.2%) *A. baumannii* isolates were resistant to all antibiotics.

Discussion

Our investigation revealed NI rate of 18.2 per 1000 patient-days between 2004 and 2005. BWI was the most common infection (56%) in contrast to other reports in which there was a predominance of pneumonia[18,19] and primary BSI[18]. Data from the NNIS[4], from 1995 to 2000 involving nearly 800 intensive care units (ICUs), of which 17 were burns units, demonstrated that BSI rates were higher in burns ICUs compared with others types of ICUs[20]. In our study, the rate of BSI was 19.9 per cent similar to a recent study from Turkey reporting 18.6 per cent BSI[21].

The most frequently used invasive devices were urinary catheter, whereas the least was ventilator. The rate of invasive device usage in BCU was accounted as 1.68 per 1000 hospitalization-days. Compared with the NNIS results[4], our results showed that the rates of central vascular catheter, ventilator and urinary catheter usage were in 50, 75, and 25 per cent percentile, respectively. However, the NIs rates related to those were in 90, 25, and 90 per cent percentile, respectively. Our results showed that, the rate of invasive device usage was low, however, the rate of NIs related to those especially for vascular catheter and ventilator was high in our BCU. For this reason, more strictly infection control procedures should be implemented.

In this study, similar to a previous a Turkish study[22], univariate analysis suggested the following risk factors for acquisition of NI: APACHE III scores, TSBA, the number of co-morbidities, and invasive device usage. Thus, decreased usage of invasive devices, better infection control procedures and improved aseptic technique while inserting devices could decrease the rates of NI on burn units, thereby, decreasing use of antimicrobial agents and, in turn, decreasing the risk of selecting antimicrobial resistant organisms. Broad-spectrum antibacterial usage was one of the most well known risk factor for developing of multiresistant strains in burned patients. Panresistance strains of *P.aeruginosa* and *A. baumannii* were not defined in previous studies of our burn care unit[23]. It could be related to antibacterial usage policy. Mozingo *et al*[24] demonstrated that bacteraemia secondary to

Table III. Risk factors of NI in burned patients, results of multivariate logistic regression

	Adjusted Odds ratio	CI 95%	*P* value
TBSA (%)			
10-20	1.0	0.3-1.2	NS
21-40	2.3	1.2-2.6	0.01
>40	3.1	1.8-6.5	0.001
Co-morbidities			
No	1.0	0.6-1.3	NS
Yes	2.3	1.4-4.6	0.002
Broad spectrum antibiotic usage			
No	1.0	0.7-1.2	NS
Yes	1.9	0.8-3.8	0.01
Invasive devices usage			
No	1.0	0.8-1.4	NS
Yes	2.2	1.0-5.5	0.01
APACHE III Score ≥10 at admission	1.7	0.9-3.5	0.03

TBSA, total burn surface area; APACHE, acute physiology and chronic health evaluation; NS, no significant

Table IV. Antimicrobial susceptibility (%) of microorganisms causing NI in the Burn Care Unit of Gulhane Military Medical Academy, Haydarpasa Training Hospital, Turkey

Microorganism (n)	MEM	CSF	PIP/TAZ	CIP	AK	OX
P. aeruginosa (104)	65	63	65	54	72	ND
A. baumannii (38)	62	74	52	65	68	ND
S. aureus (25)	ND	ND	ND	76	100	60
E. coli (6)	100	86	82	84	66	ND
Proteus spp. (2)	100	78	72	78	100	ND
Enterococcus spp. (2)	ND	ND	ND	65	100	ND
Candida spp. (2)	ND	ND	ND	ND	ND	ND
Other (3)						

MEM, Meropenem; CSF, cephaperazon/sulbactam; PIP/TAZ, piperacillin/tazobactam; CIP, ciprofloxacin; AK, amikacin; OX, oxacillin; ND, not done

surgical debridment is related to the size (> 45%) and duration of the burn injury (greater than 10 days old) and that prophylaxis is not needed for patients with small, acute burns. In our study, 16 burn patients without NI received prophylactic antimicrobial agents because of poor immunostatus and inhalation injury. Antimicrobial usage in our BCU could be further reduced if only prophylaxis was given to patients with poor immunostatus and an open burn wound that did not close within a few days. Prophylactic antibacterials were also used, if broad excisional surgery was planned. Due to fear of developing antibacterial resistance, some investigators suggested no antibiotic usage in burn patients during initial few days[25]. An open wound that does not close within a few days is likely to get infected at any time[9]. Full therapeutic dose of antibiotics with debridement of dead tissue and slough will ensure penetration of optimal concentration of the drug up to the surface of viable tissue and, hence, will minimize the risk of developing infection.

In our study, *S. aureus* was the most prevalent microorganism on burn patients especially on the first day. Further, 40 per cent of the isolates were methicillin resistant. On the 7th day, the rate of *P. aeruginosa* increased whereas that of *S. aureus* strongly decreased. It could be because *S. aureus* originated from the patient's non-burned cutaneous area. In our burn units, prophylactic antibacterials are introduced in burn patients with inhalation injury. Recently, Maybauer *et al*[26] showed that gentamicin improves haemodynamics in ovineseptic shock after smoke inhalation injury. They further demonstated that the administration of gentamicin 6 h after smoke inhalation injury and bacterial challenge improved cardiovascular function, but not at such a late time point after injury. In our study, prophylactic antibiotic was introduced to all 34 cases, haemodynamic and laboratory parameters stayed in normal ranges. But during the subsequent days, it served as suitable areas for colonization of panresistant Gram negative bacteria in 12 of those. These patients, with a average TBSA of 60 per cent on admission, and seven of those had a past medical history of smoking-related lung disease, died. For this reason, prophylactic antibiotics in burned patients could be helpfull but potantial risks of this application should be considered.

In summary, considering the high incidence of burn wound infection and panresistant strains in our burn care unit, more strict infection control policies are required, and a comrehensive education campaign for all health measures and more restricted antibacterial prophylaxis should be introduced for burn patients.

Acknowledgment

Authors thank the personnel of Burn Care Unit and Infectious Diseases and Clinical Microbiology at the GMMA Haydarpasa Training Hospital, Turkey and Ozlem Koksal for statistical support.

References

1. Lari AR, Alaghehbandan R. Nosocomial infections in an Iranian burn care center. *Burns* 2000; *26* : 737-40.
2. Falk PS, Winnike J, Woodmansee C, Desai M, Mayhall CG. Outbreak of vancomycin-resistant enterococci in a burn unit. *Infect Control Hosp Epidemiol* 2000; *21* : 575-82.
3. Roberts SA, Findlay R, Lang SD. Investigation of an outbreak of multi-drug resistant *Acinetobacter baumannii* in an intensive care burns unit. *J Hosp Infect* 2001; *48* : 228-32.
4. National Nosocomial Infections Surveillance System. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992-June 2002, issued August 2002. *Am J Infect Control* 2002; *30* : 458-75.
5. Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM. CDC definitions for nosocomial infections. In: Olmsted RN, editor. *APIC infection control and applied epidemiology*: *Principles and practice*. St. Louis: Mosby; 1996. p. A1-20.
6. Garner JS, Jarvis WR, Emori TG, Horan TC, Hughes JM. CDC definitions for nosocomial infections, 1988. *Am J Infect Control* 1988; *16* : 128-40.
7. Pruitt BA Jr, McManus AT, Kim SH, Goodwin CW. Burn wound infections: Current status. *World J Surg* 1998; *22* : 135-45.
8. Tanaka Y, Shimizu M, Hirabayashi H. Acute physiology, age, and chronic health evaluation (APACHE) III score is an alternative efficient predictor of mortality in burn patients. *Burns* 2007; *33* : 316-20.
9. Kumar P. Prophylactic antibiotics in burns. *Burns* 2006; *32* : 655-6.
10. Mermel LA. Prevention of intravascular catheter-related infections. *Ann Intern Med* 2000; *132* : 391-402.
11. Pronovost P, Needham D, Berenholtz S, Sinopoli D, Chu H, Cosgrove S, *et al*. An intervention to decrease catheter-related bloodstream infections in the ICU. *N Engl J Med* 2006; *355* : 2725-32.
12. Hori S. Hospital infection control measures for methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococci. *Nippon Rinsho* 2002; *60* : 2144-9.
13. Evans RS, Lloyd JF, Abouzelof RH, Taylor CW, Anderson VR, Samore MH. System-wide surveillance for clinical encounters by patients previously identified with MRSA and VRE. *Stud Health Technol Inform* 2004; *107* : 212-6.
14. National Committee for Clinical Laboratory Standards: *Performance standards for antimicrobial disk susceptibility tests*. Approved standard-Eighth edition. NCCLS Document M2-A8, Wayne PA.: NCCLS; 2003.

15. Clinical and Laboratory Standards Institute. *Performance standards for antimicrobial susceptibility testing,* 17th Informational Supplement. M100-S17, vol. 27, no. 1. Wayne, PA: 2007.

16. Falagas ME, Kasiakou SK. Correct use of the term 'pan-drug-resistant' (PDR) Gram-negative bacteria. *Clin Microbiol Infect* 2005; *11* : 1049-50.

17. Derksen S, Keselman HJ. Backward, forward and stepwise automated subset selection algorithms: frequency of obtaining authentic and noise variables. *Br J Math Stat Psychol* 1992; *45* : 265-82.

18. Taylor GD, Kibsey P, Kirkland T, Burroughs E, Tredget E. Predominance of staphylococcal organisms in infections occurring in a burns intensive care unit. *Burns* 1992; *18* : 332-5.

19. Wurtz R, Karajovic M, Dacumos E, Jovanovic B, Hanumadass M. Nosocomial infections in a burn intensive care unit. *Burns* 1995; *21* : 181-4.

20. Santucci SG, Gobara S, Santos CR, Fontana C, Levin AS. Infections in a burn intensive care unit: experience of seven years. *J Hosp Infect* 2003; *53* : 6-13.

21. Ekrami A, Kalantar E. Bacterial infections in burn patients at a burn hospital in Iran. *Indian J Med Res* 2007; *126* : 541-4.

22. Ozkurt Z, Ertek M, Erol S, Altoparlak U, Akcay MN. The risk factors for acquisition of imipenem-resistant *Pseudomonas aeruginosa* in the burn unit. *Burns* 2005; *31* : 870-3.

23. Oncul O, Yuksel F, Altunay H, Acikel C, Celikoz B, Cavuslu S. The evaluation of nosocomial infection during 1-year-period in the burn unit of a training hospital in Istanbul, Turkey. *Burns* 2002; *28* : 738-44.

24. Mozingo DW, McManus AT, Kim SH, Pruitt BA Jr. Incidence of bacteremia after burn wound manipulation in the early postburn period. *J Trauma* 1997; *42* : 1006-11.

25. Ugburo AO, Atoyebi OA, Oyeneyin JO, Sowemimo GO. An evaluation of the role of systemic antibiotic prophylaxis in the control of burn wound infection at the Lagos University Teaching Hospital. *Burns* 2004; *30* : 43-8.

26. Maybauer MO, Maybauer DM, Traber LD, Westphal M, Enkhbaatar P, Morita N, *et al*. Gentamicin improves hemodynamics in ovine septic shock after smoke inhalation injury. *Shock* 2005; *24* : 226-31.

Reprint requests: Dr Oral Oncul, Gulhane Military Medical Academy, Haydarpasa Training Hospital, Department of Infectious Diseases & Clinical Microbiology, 34668 Uskudar-Istanbul, Turkiye
e-mail: oraloncul@yahoo.com