

Epidemiology of burn wound bacterial infections at a Meknes hospital, Morocco

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Abstract

This study aimed to determine the prevalence of burn wound infection in the ward of Burns and Plastic Surgery at Mohammed V Hospital, Meknes, Morocco, and to determine the pathogenic bacterial species responsible for this infection as well as the susceptibility of these isolates to various antibiotics. Over the 1-year study period, 126 patients were admitted. The main sources of burns were flames (52.38%) and hot water (28.57%); 71% had burns with 11% to 40% burn surface and 48.41% had burns between 11% and 20% total burn surface. The mean \pm SD duration of hospitalization was 22.15 \pm 13.84 days after injury. Eighty-six patients were found to have at least one positive culture requiring treatment and were thus included in this study. The predominant bacteria isolated were *Staphylococcus aureus* (33.85%), followed by *Pseudomonas* spp. (18.46%), *Acinetobacter baumannii* (15.38%), *Klebsiella pneumoniae* (13.85%), *Escherichia coli* (8.46%) and *Proteus mirabilis* (4.42%). Disc-diffusion susceptibility testing indicated a high prevalence of resistance to various antimicrobial agents. Among the *Staphylococcus aureus* and *Enterobacteriaceae* strains isolated, 86.36% were methicillin resistant and 48.64% were extended-spectrum β -lactamase producers respectively.

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Introduction

Nosocomial bacterial infections in both developed and developing countries continue to pose challenges for healthcare providers and trouble the healthcare industry [1]. These infections in burn patients are a major cause of morbidity and mortality [2]. Factors that predispose patients to increased risk of infection and complications include the nature of the burn, increased risk of infectious complications due to the burn, lengthy hospital stay, use of catheters and other invasive devices and immunocompromising effects of burns [2–6]. The great challenges in treating these bacterial infections is their resistance to antimicrobial treatments. Many studies have been performed in developed and developing countries to establish the most prevalent multidrug-resistant (MDR) bacteria in burn units [7–11]. Prevalent pathogenic bacteria isolated from infected burn patients include *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, *Staphylococcus aureus*, *Klebsiella pneumoniae* and various coliform bacilli [4,5,8,12,13].

The small number of publications discussing the epidemiologic status of burn wound infections in Moroccan hospitals means that this topic remains understudied. Our aims were therefore to determine the prevalence of burn wound infections at the Service of Burns and Plastic Surgery in Mohammed V Hospital in Meknes city, Morocco; to determine the pathogenic bacterial species responsible of this infection along with antibiotic susceptibility studies; and to determine risk factors related to bacterial infections at the Service of Burns and Plastic Surgery.

Materials and methods

Setting

The study was performed at the Service of Burns and Plastic Surgery ward located at Mohammed V Hospital (378 beds), Meknes city, Morocco. It has seven beds with an operating room. Major and moderate burns are referred to this service. Local patients may be admitted directly without first receiving primary care, and patients from other districts come after receiving first aid at regional hospitals.

Ethics

Ethical approval was obtained from the hospital's human research and ethics committee.

Clinical data collection

All burn patients admitted to the Service of Burns and Plastic Surgery ward from January 2015 to January 2016 were included in the study. The patients were divided into two groups. The first group was composed of patients diagnosed with healthcare-associated infections according to the criteria of the US Centers for Disease Control and Prevention [1]. The second group was composed of patients who had not developed bacterial infections. We excluded burn patients with less than 24 hours' hospitalization.

The medical records of included patients were retrieved and reviewed. Information was obtained about basic demographic characteristics (age, gender and sex), burn surface area (BSA), burn degree and cause, invasive devices used, polymicrobial infection, duration of hospitalization and mortality rate. The analysis was limited to the first episode.

Microbiologic sampling procedures

Diagnosis of burn wound infection was based on clinical signs and microbiologic surveillance by surface swabs taken routinely twice a week. Microbial colonization of all wounds was studied from the time of admission to discharge.

At admission, the sampling procedure included swabbing clinically deep areas of the burn wound before any cleansing. Later swabbing was performed twice per week in the morning, while dressing burn wounds. Swab samples were taken from the wound area where the degree of burn was highest. When samples were collected, special attention was paid to the areas where infection was most evident before the dressings were changed. Wound samples were collected for culture after several clinical assessments from the leading edge of wound sites showing signs of infection, such as discolouration, bad odour and rapid separation of the eschar or the presence of pus. The oral, genital, scalp and anal regions were never used for sample collection. 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 was moistened with sterile normal saline, and the centre was swabbed for 30 seconds according to Levine's technique. After ensuring that the swabs were saturated with wound exudates, the swabs were placed in appropriate containers. The swabs were transported to the laboratory for immediate processing. In patients with bacteraemia, blood samples were taken for culture; in all patients, we routinely checked for catheter tip colonization, including patients with central venous catheters.

Upon arrival at the laboratory, the wound swab samples were immediately cultured using nutrient agar (Bio-Rad, Marnes-la-Coquette, France), MacConkey agar (Bio-Rad) and blood agar (Bio-Rad), then incubated at 37°C for 18 to 72 hours. After incubation, colony morphology, including colour, shape and general appearance of the individual colonies on each of the plates, was examined. Eighty-six representative single colonies were Gram stained and biochemically tested to identify the bacteria species present.

After identification of the bacterial strains, the antibiotic susceptibility of all cultured pathogens was determined.

Antibiotic susceptibility testing

Antimicrobial drug susceptibility was determined using the disc-diffusion method on Müller-Hinton agar (Bio-Rad) plates with interpretation according to the recommendations of the antibiogram committee of the French Microbiology Society (CA-SFM-2013). The antibiotics (Bio-Rad) were tested, including amoxicillin (25 µg), amoxicillin/clavulanic acid (20/10 µg), ticarcillin (75 µg), piperacillin (75 µg), ceftazidime (30 µg), ceftazidime (30 µg), cefotaxime (30 µg), imipenem (10 µg), gentamicin (15 µg), tobramycin (10 µg), amikacin (30 µg), ciprofloxacin (5 µg), sulfamethoxazole/trimethoprim (1.25/23.75 µg), ticarcillin (75 µg), colistin (50 µg), penicillin G (1 unit), ampicillin (2 µg), erythromycin (15 µg), vancomycin (5 µg), teicoplanin (30 µg), ceftazidime (30 µg), ofloxacin (5 µg), chloramphenicol (30 µg), ticarcillin/clavulanic acid (75/10 µg), piperacillin/tazobactam (30/6 µg), rifampicin (5 µg), chloramphenicol (30 µg) and fusidic acid (10 µg) [14].

Phenotypic detection of extended-spectrum β-lactamase production

Müller-Hinton agar plates were inoculated with standardized inoculums (corresponding to 0.5 McFarland standard) to form a lawn culture. With a sterile forceps, a disc of clavulanic acid/ticarcillin (20/10 µg) was placed in the centre of the plate. Then, ensuring a centre-to-centre distance of 20 mm around the clavulanic acid/ticarcillin disc, the ceftazidime (30 µg) and aztreonam (30 µg) discs were applied. The plates were incubated at 37°C in an incubator overnight [7,15].

Data analysis

To identify variables associated with burn wound bacterial infection, a risk factor analysis was performed using a case-control study format. Demographic and hospitalization variables from patients infected were compared with noninfected patients. Data were entered into a database using SPSS 20.0 for Windows (IBM, Armonk, NY, USA). The chi-square test and independent-sample *t* test were used for categorical and continuous variables respectively.

Results

Over the 1-year period, 126 patients were admitted, of whom 64 (50.8%) were male and 62 (49.2%) were female. The mean ± SD age was 22.5 ± 18.84 years. Flames were the cause of injury for 66 patients (52.38%), hot water was responsible for 36 injuries (28.57%) and ten patients each (7.93%) were injured by electricity and hot tea. According to BSA, 71% of patients had a BSA between 11% and 40%, and 48.41% had a BSA between 11% and 20%. Only 11.1% of patients had a burn size of >41% total BSA. The mean ± SD duration of hospitalization was 22.15 ± 13.84 days after injury. Eighty-six (68.25%) of 126 patients were found to have been infected with pathogens and needed treatment. Table I summarizes the characteristics of the infected patient group (n = 86) vs. the noninfected patient group (n = 40). Age was similar between groups, with a mean age of 22.94 years in infected patients and 21.57 years in noninfected patients. In the multivariate analysis, none of the risk factors for infected patients was identified. The mortality of infected patients was 6.98% and was 15% for noninfected patients, a result that was not statistically significant. (The patients whose burns were uninfected had burns closer to relevant physiologic sites (respirator) and had a very high degree of burn.)

In the 86 infected patients, ten different bacterial species were identified, with the predominant bacteria being *Staphylococcus aureus* (33.85%). It was followed by *Pseudomonas* spp. (18.46%), *Acinetobacter baumannii* (15.38%), *Klebsiella pneumoniae* (13.85%), *Escherichia coli* (8.46%) and *Proteus mirabilis* (4.42%) (Table 2).

Disc-diffusion susceptibility testing indicated a high prevalence of resistance to various antimicrobial agents. Isolates of *Staphylococcus aureus* were found to be resistant to ampicillin (88.63%), penicillin (86.36%), amoxicillin/clavulanic acid (84.09%), ofloxacin (63.63%), cefoxitin (56.81%), erythromycin (54.54%), ciprofloxacin (54.54%) and fusidic acid (52.27%). Among the 44 *S. aureus* strains isolated, 38 (86.36%) were methicillin-resistant *S. aureus*. Methicillin resistance was detected in the five *Staphylococcus epidermis* isolates.

Pseudomonas aeruginosa isolates were resistant to ticarcillin (100%), ticarcillin/clavulanic acid (100%), amoxicillin/clavulanic acid (95%), piperacillin (80%), cotrimoxazole (75%) and rifampicin (65%). All isolates of *Acinetobacter baumannii* were resistant to ticarcillin, piperacillin, ticarcillin/clavulanic acid, amoxicillin/clavulanic acid, piperacillin/clavulanic acid, ciprofloxacin, levofloxacin, tobramycin and cotrimoxazole. The *Klebsiella pneumoniae* and other *Enterobacteriaceae* species isolates were resistant to amoxicillin, ticarcillin, cephalothin, amoxicillin/clavulanic acid, ciprofloxacin, ceftriaxone, ceftazidime, cefotaxime and

TABLE I. Patient demographic and clinical data

Characteristic	Infected (n = 86)	Not infected (n = 40)	All (n = 126)	p
Age				
Mean ± SD	22.94 ± 19.21	21.57 ± 18.24	22.50 ± 18.84	0.860
Gender				
Female	44 (51.16)	18 (45)	62 (49.20)	0.519
Male	42 (48.84)	22 (55)	64 (50.80)	0.519
Cause of burn				
Flame	45 (52.32)	21 (52.5)	66 (52.38)	0.985
Electrical	6 (6.97)	4 (10)	10 (7.93)	0.724
Hot water	25 (29.07)	11 (27.5)	36 (28.57)	0.855
Hot tea	7 (8.13)	3 (7.5)	10 (7.93)	1
Hot coffee	2 (2.32)	0	2 (1.58)	1
Burn surface area				
0–10%	6 (6.97)	6 (15)	12 (9.52)	0.194
11–20%	42 (48.83)	19 (47.5)	61 (48.41)	0.888
21–40%	25 (29.07)	10 (25)	35 (27.77)	0.634
41–60%	7 (8.14)	2 (5)	9 (7.14)	0.717
61–80%	1 (1.6)	4 (10)	5 (3.96)	0.034
81–100%	0	0	0	—
Mean ± SD	21.82% ± 12.50	24.2% ± 20.86	22.76% ± 15.57	0.076
Burn degree				
Second degree (deep)	8 (9.30)	3 (7.5)	11 (8.73)	1
Second + third degree (deep)	77 (89.53)	36 (90)	113 (89.68)	0.936
Third degree (deep)	1 (1.16)	1 (2.5)	2 (1.58)	0.535
Duration of hospitalization, mean ± SD	26.05 ± 12.82	13.75 ± 12.22	22.15 ± 13.84	0.076
Invasive devices				
Intubation	7 (8.14)	1 (2.5)	8 (6.35)	0.434
Intubation + urinary catheter	1 (1.16)	0	1 (0.8)	1
Intubation + central venous catheter	29 (33.72)	19 (47.5)	48 (38.09)	0.138
Urinary catheter + central venous catheter	49 (56.97)	20 (50)	69 (54.76)	0.463
Polymicrobial infection				
Yes	42 (48.84)	—	—	—
No	44 (51.16)	—	—	—
Mortality				
Yes	6 (6.98)	6 (15)	12 (9.52)	0.194
No	80 (93.02)	34 (85)	114 (90.47)	0.153

Data are presented as n (%) unless otherwise indicated.

cotrimoxazole. Among the 47 *Enterobacteriaceae* strains isolated, 18 (48.64%) were extended-spectrum β-lactamase producers (Table 3).

Discussion

The present study aimed to describe the epidemiologic characteristics of patients with infected burns hospitalized at the ward of the Service of Burns and Plastic Surgery, Mohammed V Hospital, Meknes city, Morocco. We further identified the common bacterial pathogens implicated in burn wound infections and studied their antibiotic resistance patterns in order to improve our therapeutic approach and to prevent burn wound infections at Moroccan hospitals because there are few studies on this subject.

In the present study, the rate of burn injuries was equal in female and male subjects (50.80% vs. 49.20%). This is in contradiction to a study conducted in 2014 in Morocco by Essayagh et al. [16], which reported a higher incidence of burn

TABLE 2. Bacteria isolates recovered from burn wound infections

Species	n (%)
<i>Citrobacter freundii</i>	1 (0.77)
<i>Escherichia coli</i>	11 (8.46)
<i>Klebsiella pneumoniae</i>	18 (13.85)
<i>Proteus mirabilis</i>	6 (4.62)
<i>Proteus morganii</i>	1 (0.77)
<i>Staphylococcus aureus</i>	44 (33.85)
<i>Staphylococcus epidermidis</i>	5 (3.85)
<i>Pseudomonas putida</i>	4 (3.08)
<i>Pseudomonas aeruginosa</i>	20 (15.38)
<i>Acinetobacter baumannii</i>	20 (15.38)

injuries in male (64%) than in female (36%) subjects. The mean \pm SD age was 22.5 ± 18.84 years, a result which correlates with the results reported by other studies [6,16]. This could be because the 16- to 59-year age group is the most active and is exposed to hazardous environments at both work and home [11,13]. Flames were the cause of injury in 66 patients (52.38%), hot water was responsible for 36 (28.57%) injuries and ten each (7.93%) were injured by electricity and hot tea. This correlates with the Essayagh et al. study, which reported the predominant burn agent to be gas flame, followed by scalding liquid and contact with an electrical source.

We found a prevalence of burn infections of 68.25%. Some studies report a higher rate [17,18], whereas Wurtz et al. [19] reported a percentage of infected patients which was comparable to that of our study.

In a prospective analysis of nosocomial infections in the burn population, one study found that length of hospital stay was 5.3 ± 5.6 days in patients with infection and 12.8 ± 16.2 days in patients without infection [20]. Another study found the mean length of hospital stay was 12.0 ± 9.4 days [2].

According to several studies, infections result from prolonged hospital stays. However, a prolonged hospital stay was a risk factor for infections with MDR microorganisms [2,7,10,11,15]. In our study, length of hospital stay was found to be 36.5 ± 19.5 days in patients with MDR-resistant *Acinetobacter baumannii* (MDR-AB) infection and 14.3 ± 9.1 days in patients without infection. Our study revealed that length of stay was a risk factor for MDR-AB infections.

In our study, there was no significant association between patient sex and development of an infection; there was also no significant association with the type of burn injury and the development of MDR-AB infection. To our knowledge, there are no available data about the effect of burn type on the development of wound infections caused by MDR-AB.

TABLE 3. Antibiotic resistance pattern of bacteria species isolated from burn patients

Antibiotic	SA (n = 44)	SE (n = 5)	EC (n = 11)	KP (n = 1)	Other (n = 8) ^a	PA (n = 20)	AB (n = 20)
Ampicillin	39 (88.63)	5 (100)	ND	ND	ND	ND	ND
Amoxicillin	ND	ND	10 (90.9)	18 (100)	8 (100)	ND	ND
Penicillin	38 (86.36)	5 (100)	ND	ND	ND	ND	ND
Ticarcillin	ND	ND	11 (100)	18 (100)	8 (100)	20 (100)	20 (100)
Piperacillin	ND	ND	ND	ND	ND	16 (80)	20 (100)
Amoxicillin/ clavulanic acid	37 (84.09)	5 (100)	11 (100)	18 (100)	8 (100)	19 (95)	20 (100)
Piperacillin/ tazobactam	ND	ND	ND	ND	ND	12 (60)	20 (100)
Ticarcillin/ clavulanic acid	ND	ND	ND	ND	ND	20 (100)	20 (100)
Cephalothin	ND	ND	6 (54.54)	16 (88.88)	7 (87.5)	ND	ND
Cefoxitin	25 (56.81)	5 (100)	ND	ND	ND	ND	ND
Cefotaxime	ND	ND	2 (18.18)	15 (83.34)	5 (62.5)	ND	ND
Ceftazidime	ND	ND	2 (18.18)	15 (83.34)	5 (62.5)	8 (40)	13 (65)
Ceftriaxone	ND	ND	(36.36)	15 (83.34)	5 (62.5)	ND	ND
Imipenem	ND	ND	(12.5)	9 (50)	3 (37.5)	6 (30)	18 (90)
Ertapenem	ND	ND	2 (18.18)	9 (50)	5 (62.5)	ND	ND
Amikacin	13 (29.54)	1 (20)	2 (18.18)	5 (27.77)	2 (25)	7 (35)	9 (45)
Gentamycin	17 (38.63)	3 (60)	4 (36.36)	5 (27.77)	5 (62.5)	11 (55)	18 (90)
Tobramycin	20 (45.45)	2 (40)	ND	ND	ND	8 (40)	20 (100)
Vancomycin	2 (4.54)	0	ND	ND	ND	ND	ND
Ciprofloxacin	24 (54.54)	4 (80)	8 (72.72)	14 (77.77)	7 (87.5)	16 (80)	20 (100)
Levofloxacin	ND	ND	3 (27.27)	13 (72.23)	6 (75)	11 (55)	20 (100)
Ofloxacin	23 (52.27)	4 (80)	ND	ND	ND	ND	ND
Erythromycin	24 (54.54)	4 (80)	ND	ND	ND	ND	ND
Teicoplanin	2 (4.54)	1 (20)	ND	ND	ND	ND	ND
Rifampicin	ND	ND	ND	ND	ND	13 (65)	19 (95)
Chloramphenicol	12 (27.27)	2 (2)	ND	ND	ND	8 (40)	17 (85)
Colistin	ND	ND	1 (12.5)	2 (11.11)	3 (37.5)	0	4 (20)
Sulfamethoxazole/ trimethoprim	22 (50)	5 (100)	7 (63.63)	15 (83.34)	6 (75)	15 (75)	20 (100)

Data are shown as n (%). Species abbreviations are as follows: SA, *Staphylococcus aureus*; SE, *Staphylococcus epidermidis*; EC, *Escherichia coli*; KP, *Klebsiella pneumoniae*; PA, *Pseudomonas aeruginosa*; AB, *Acinetobacter baumannii*.

ND, not determined.

^aOther Enterobacteriaceae included: *Citrobacter freundii* (n = 1), *Proteus mirabilis* (n = 6) and *Proteus morganii* (n = 1).

The mean age of patients with infected burns varied depending on sociocultural level and the distribution of the population living in the region. In a study of nosocomial infection, the mean age of patients was found to be 38.4 ± 22.1 years [20].

Nosocomial infections are common in burn patients with large burn areas. In the present study, the mean total BSA of burns was $13.5\% \pm 10.9$ in uninfected patients and $34.7\% \pm 16.2$ in infected patients ($p < 0.001$). Different studies in the literature support our results. In one particular study, the mean of total BSA of burns was 10.75% (6–18%) in uninfected patients and 26.25% (17.75–32%) in infected patients [11]. According to our results and the data in the literature, we can say that an increase in BSA led to an increased risk of development of MDR-AB infections. In studies of nosocomial infections, the Abbreviated Burn Severity Index (ABSI) score was found to be a risk factor for nosocomial infections. A higher ABSI score was found to be a risk factor for MDR-AB infections in the present study.

Our study showed *S. aureus* to be a common cause of nosocomial infection, similar to previously published results [6,13]. Essayagh et al. [16] showed that nosocomial infection caused by *A. baumannii* and *P. aeruginosa* was a major risk in burn patients in Morocco. In our study, 86.6% of *S. aureus* were methicillin resistant. This pathogen has been reported to be a major cause of nosocomial infection in many countries [5,8,11,12].

It has been reported that burn wards harbour MDR bacterial isolates, which can colonize burn patients and establish infection [4]. Our study showed a high prevalence of MDR bacterial infections among burn patients compared to studies from other countries, such as Tunisia [11], Egypt [3], Iran [5] and India [10]. This difference might be attributed to antibiotic misuse, different hospital strategies for the management of infections, hygiene protocols and geographic climate variation [5,6,8,11,13].

Treatment of MDR bacterial infection is frequently complicated by limited susceptibility to antimicrobial drugs and the spread of antibiotic resistance during therapy. In the last few years, Morocco has identified as a country with one of the highest rates of antimicrobial resistance [6,20–22]. In our study, there were high rates of resistance to all available antimicrobial drugs among bacteria isolated from Mohammed V Hospital. The risk factors we identified for acquiring MDR bacteria are supported by the results of previous studies demonstrating that the host's condition, infection control practices and antimicrobial use can be related [3,4,8,12].

Conclusions

Moroccan burn patients were most commonly infected with *S. aureus* followed by *P. aeruginosa* and *A. baumannii*, with the

microorganisms resistant to most of the antibiotics tested. Nosocomial infection surveillance and management systems must be implemented to reduce the rate of morbidity and mortality of patients with infected burns and to find better therapeutic options for this patient population.

Conflict of interest

None declared.

References

- [1] Rosenthal VD. International Nosocomial Infection Control Consortium (INICC) resources: INICC multidimensional approach and INICC surveillance online system. *Am J Infect Control* 2016;44:11.
- [2] Litt JS. Evaluation and management of the burn patient: a case study and review. *Mo Med* 2018;115:443–6.
- [3] AbdelWahab ME, Sadaka MS, Elbana EA, Hendy AA. Evaluation of prognostic factors affecting length of stay in hospital and mortality rates in acute burn patients. *Ann Burns Fire Disasters* 2018;31:83–8.
- [4] Aurora A, Le TD, Akers KS, Blyth DM, Graybill JC, Clemens MS, et al. Recurrent bacteremia: a 10-year retrospective study in combat-related burn casualties. *Burns* 2019;45:579–88.
- [5] Ekrami A, Kalantar E. Bacterial infections in burn patients at a burn hospital in Iran. *Indian J Med Res* 2007;126:541–4.
- [6] Rafik A, Chabbak H, Jouhri K, Diouri M, Bahechar N, Chlihi A. Nosocomial infections in a Morocco burn unit. *Open Access Lib J* 2015;2:e1394.
- [7] Hakemi Vala M, Hallajzadeh M, Hashemi A, Goudarzi H, Tarhani M, Sattarzadeh Tabrizi M, et al. Detection of Ambler class A, B and D β -lactamases among *Pseudomonas aeruginosa* and *Acinetobacter baumannii* clinical isolates from burn patients. *Ann Burns Fire Disasters* 2014;27:8–13.
- [8] Ozumba UC, Jiburum BC. Bacteriology of burn wounds in Enugu, Nigeria. *Burns* 2000;26:178–80.
- [9] Ranjbar R, Owlia P, Saderi H, Mansouri S, Jonaidi-Jafari N, Izadi M, et al. Characterization of *Pseudomonas aeruginosa* strains isolated from burned patients hospitalized in a major burn center in Tehran, Iran. *Acta Med Iran* 2011;49:675–9.
- [10] Srinivasan S, Saldanha J, Abhyankar S, Patil A, Vartak AM. Bacteriology of the burn wound at the Bai Jerbai Wadia hospital for children, Mumbai, India—a 21 year study of predominant *Pseudomonas* species. *Int J Burns Trauma* 2018;8:98–105.
- [11] Thabet L, Turki A, Ben Redjeb S, Messadi A. [Bacteriological profile and antibiotic resistance of bacteria isolates in a burn department]. *Tunis Med* 2008;86:1051–4.
- [12] Agnihotri N, Gupta V, Joshi R. Aerobic bacterial isolates from burn wound infections and their antibiograms—a five-year study. *Burns* 2004;30:241–3.
- [13] Siah S, Belefqih R, Elouennass M, Fouadi FE, Ihray I. L'infection nosocomiale en réanimation des Brûlés. *Ann Burns Fire Disasters* 2009;22:72–8.
- [14] Clinical and Laboratory Standards Institute (CLSI). Performance standards for antimicrobial susceptibility testing. Twenty-second informational supplement. Wayne, PA: CLSI; 2012.
- [15] Touati M, Diene SM, Dekhil M, Djahoudi A, Racherache A, Rolain JM. Dissemination of a class I integron carrying VIM-2 carbapenemase in *Pseudomonas aeruginosa* clinical isolates from a hospital intensive care unit in Annaba, Algeria. *Antimicrob Agents Chemother* 2013;57:2426–7.

- [16] Essayagh M, Essayagh T, Essayagh S, El Hamzaoui S. [Epidemiology of burn wound infection in Rabat, Morocco: three-year review]. *Med Sante Trop* 2014;24:157–64.
- [17] Wenzel RP, Thompson RL, Landry SM, Russell BS, Miller PJ, Ponce de Leon S, et al. Hospital-acquired infections in intensive care unit patients: an overview with emphasis on epidemics. *Infect Control* 1983;4:371–5.
- [18] Chandrasekar PH, Kruse JA, Mathews MF. Nosocomial infection among patients in different types of intensive care units at a city hospital. *Crit Care Med* 1986;14:508–10.
- [19] Wurtz R, Karajovic M, Dacumos E, Jovanovic B, Hanumadass M. Nosocomial infection in a burn intensive care unit. *Burns* 1995;21:181–4.
- [20] Barguigua A, El Otmani F, Lakbakbi El Yaagoubi F, Talmi M, Zerouali K, Timinouni M. First report of a *Klebsiella pneumoniae* strain coproducing NDM-1, VIM-1 and OXA-48 carbapenemases isolated in Morocco. *APMIS* 2013;121:675–7.
- [21] Barguigua A, El Otmani F, Talmi M, Bourjilat F, Haouzane F, Zerouali K, et al. Characterization of extended-spectrum beta-lactamase-producing *Escherichia coli* and *Klebsiella pneumoniae* isolates from the community in Morocco. *J Med Microbiol* 2011;60:1344–52.
- [22] Barguigua A, El Otmani F, Talmi M, Reguig A, Jamali L, Zerouali K, et al. Prevalence and genotypic analysis of plasmid-mediated β -lactamases among urinary *Klebsiella pneumoniae* isolates in Moroccan community. *J Antibiot (Tokyo)* 2013;66:11–6.