Impact of 4% chlorhexidine whole-body washing on multidrug-resistant Acinetobacter baumannii skin colonisation among patients in a medical intensive care unit

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Summary The prevalence of skin colonisation with Acinetobacter baumannii (ACBA) on admission to the medical intensive care unit (MICU) was studied in an institution endemic for ACBA bloodstream infections (BSIs). The impact of 4% chlorhexidine gluconate (4% CG) whole-body washing on the patients' ACBA skin colonisation was also determined. A prospective cohort trial in a MICU during March 2002 to December 2003 was performed, with a comparison between the prevalence and incidence of ACBA-BSIs obtained after intervention and retrospectively. During the intervention period, ACBA skin-screening swabs were taken from all patients on admission and periodically until discharge. Patients underwent whole-body disinfection with 4% CG immediately after obtaining the initial cultures. Disinfection was carried out on a daily basis until discharge, regardless of colonisation status. Of the 320 patients at ward admission, 55 (17%) yielded ACBA. The prevalence of ACBA colonisation among the remaining MICU patients was 5.5% at 24 h and 1% at 48 h following the disinfection regimen (P = 0.002, OR: 2.4). Following a second screen, 80% of colonised patients were decolonised. Prevalence of ACBA-BSIs decreased

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Introduction

Spread of multidrug-resistant (MDR) micro-organisms is a great concern worldwide.1–3 Pandrug-resistant Acinetobacter baumannii (ACBA) has already been reported, posing serious treatment limitations in intensive care units (ICUs).4–6 In addition, the decreased susceptibility of ACBA to diluted disinfectants, together with its long survival on dry surfaces, could lead to breakdown of strict 'classic' infection control measures.7

Unsuccessful control and acquisition of nosocomial MDR-ACBA infections in ICUs could give rise to an imminent threat of untreatable infections.8,9 Infection control programmes with emphasis on the particular epidemiology of MDR-ACBA are lacking. Prolonged uncontrolled epidemic situations are now occurring in some institutions. Clinical and microbiological epidemiology of ACBA infections in such epidemic situations are complex and on occasion remain obscure.

Our aims were to determine the prevalence of ACBA skin colonisation, the association of ACBA skin decolonisation and bloodstream infections (BSIs), and the effectiveness of whole-body washing with 4% chlorhexidine gluconate (CG) to reduce ACBA contamination of patients’ skin.

Methods

Study design

The study was conducted at the Soroka University Medical Centre, a 1000-bed tertiary care hospital in southern Israel. ACBA is endemic in our institution, with over half of the isolates exhibiting multidrug resistance, including strains resistant to all antibiotics.10

The medical intensive care unit (MICU) is an eight-bed unit with an average of 450 patient admissions per year. There are two main bay areas each with four beds, including two isolation rooms. There is a ratio of one nurse for every two patients, alcohol hand rub is available in each bed, and there are infection control protocols. Approximately 40% of admissions involve patients from internal medicine wards, and 60% of admissions involve patients from an emergency room or another ICU.

Before intervention

From February 2001 to February 2002, monthly ACBA rate data were collected retrospectively from blood cultures for the 12 months prior to introduction of the intervention in March 2002. Routine whole-body washing was done with a liquid plain soap (Fisher Pharmaceuticals Ltd) on a daily basis. During this period, ACBA infections were identified only from clinical cultures, and patients were isolated and barrier-nursed upon confirmation that they had an ACBA infection.

Intervention

All patients admitted to MICU from March 2002 to December 2003 were screened for ACBA skin colonisation within 2 h of admission before introduction of CG and then periodically until discharge. One cotton swab was obtained from each patient by the nursing staff. The culture specimens were obtained from right and left inguinal and axillary areas before the morning wash. Swabbing was performed mostly daily up to 3 days, because few patients remained in the ICU for longer than this time.

Following the collection of skin cultures, the skin disinfection was carried out once a day.

The protocol consisted of using 4% CG (Septal Scrub, Teva Medical, Ashdod Israel) sponges, towels, portable wash basin with warm tap water and gloves. Four-percent CG was poured on a sponge previously moistened with warm water to make suds. The patient was sponge-bathed with a whole-body wash from clean to dirty area and excluding the face and scalp. The 4% CG remained on the skin for at least 2 min to receive maximum contact time. It was then removed by warm tap water and the patient was towel-dried. Stool from incontinent patients was removed and the genital area washed with 4% CG and completely dried before diapering and sheet changing. Patients were examined daily for localised or
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body-wide 4% CG skin reactions. Exclusion criteria to a whole-body washing on admission were history of allergy to chlorhexidine or patients with a clinically unstable condition requiring urgent resuscitation. Catheter-related BSI was defined in a patient with an intravascular catheter with at least one ACBA-positive blood culture obtained from a peripheral vein, clinical manifestation of infection, no apparent source for the BSI except the catheter, and positive semiquantitative (>15 cfu/catheter segment) ACBA culture from the catheter tip.11

**Bacteriology**

Soaked swabs containing neutralising solutions (0.5% sodium thiosulphate and 2.5% Tween 20) were placed in transport medium (COPAN Innovation, Brescia, Italy) and transferred to the clinical microbiology laboratory.12,13 Bacteria were cultured in a MacConkey medium according to standard bacteriological techniques and identified as ACBA by the ID20NE system (bioMérieux, Marcy l'Etoile, France). Antibiotic susceptibility was determined routinely for 15 different antibiotic agents, including aminoglycosides, penicillins, cephalosporins, aztreonam, carbapenems, fluoroquinolones, tetracyclines, as well as polymyxin E and ampicillin/sublactam, using the disc-diffusion method of Bauer and Kirby, and in accordance with guidelines from the National Committee for Clinical Laboratory Standards (now the Clinical and Laboratory Standards Institute).14 MDR was defined as resistance to at least three different antibiotic classes. The E-test method was used for the detection of colistin-resistant strains (AB BIODISK, Solna, Sweden); the susceptibility break point was considered to be ≥4 μg/ml.15

**Molecular typing**

Genotypic analysis was performed using pulsed-field gel electrophoresis (PFGE) in order to determine the genetic relatedness of ACBA strains isolated from the patient’s skin and bloodstream. Chromosomal DNA fragments, generated by digestion with Apal, were analysed using a contour-clumped homogeneous electric field apparatus (CHEF-DRIII, Bio-Rad Laboratories, Hercules, CA, USA). Interpretation of strain relatedness according to PFGE banding pattern was performed according to current consensus.16

**Statistical analysis**

Data were analysed using SPSS software (version 12, SPSS Inc., Cary, NC, USA). Differences between categorical variables were analysed by the Chi-squared test and two-tailed Fisher's exact test, as appropriate. Odds ratios (ORs) are presented for significant variables. *P* < 0.05 was considered statistically significant.

**Results**

Thirty-seven (10.5%) out of 357 patients were excluded. They were all patients with severely unstable clinical condition on ward admission. Of the 320 patients admitted to MICU in the study, 47% (150/320) came from the emergency room, 33% (105/320) from internal medicine, 12% from other ICUs and 8% from other wards. From March 2002 to December 2003, there was a mean of 32 ± 4 patient admissions to MICU per month compared with 34 ± 6 during the 12 month period prior to the study. The mean incidence of the ACBA skin colonisation rate on admission was 55/320 (17%), being lowest [2/35 (6%) in October 2002] and highest [8/33 (24%)] in May 2002 (Figure 1). Of the ACBA-colonised patients, 27/55 (48%) were admitted from the emergency room, 16/55 (29%) from internal medicine wards, 11/55 (21%) from other ICUs and one patient from the neurology ward. At 24 h, 71 (22%) of the patients were discharged or had died, and four (5.5%) patients out of the 71 were colonised with ACBA on ward admission.

The prevalence of ACBA colonisation among the remaining patients in the unit was 13/249 (5.2%) at 24 h and 1/117 (0.9%) at 48 h (*P* = 0.002, OR: 2.4) (Figure 2). By the third culture (48 h), 116 de-colonised patients remained in the ICU, and 18 ACBA-positive patients had been discharged to other wards. Altogether, there were 203 patients discharged from the unit at 48 h, including 185 (91.1%) with negative cultures and 18 (8.9%) with positive cultures. New acquisition of ACBA skin colonisation occurred in the unit in 15 (4.7%) of the 320 patients, 8/249 (3.2%) during the first 48 h and 0/116 (0%) on culture 3 (*P* < 0.01). At 72 h and beyond, there were no new cases colonised with ACBA in the ICU, but there was one patient who developed ACBA colonisation on discharge to another ward (Figure 2).

Fifteen out of 329 patients (4.6%) developed ACBA-BSIs prior to intervention, compared with 2/320 (0.6%) following intervention (*P* ≤ 0.001; OR: 7.6). The incidence of ACBA-BSIs was reduced from 7.8 to 1.25 cases per thousand patient-days (85% reduction). Forty-two (60%) out of 70 ACBA strains were resistant to more than three antibiotic classes and 6/70 (8.5%) were only susceptible to polymyxin E. Twenty-six out of 50 strains
are represented by PFGE. Apal digestion of DNA from Acinetobacter spp. after PFGE demonstrated four different biotypes isolated from patients' skin and matching the invasive isolates causing BSIs. Two of these strains belonged to infected patients with an overlapping hospital stay in the MICU. One strain was cultured during the same hospital stay, but on admission to the MICU from an internal...

![Figure 1](image1.png)

**Figure 1** Monthly prevalence of *Acinetobacter baumannii* patient skin colonisation on admission to the medical intensive care unit.

![Figure 2](image2.png)

**Figure 2** Patient population changes and *Acinetobacter baumannii* (ACBA) skin colonisation by time of culture. In-ward population: colonised and non-colonised ACBA patient population in the medical intensive care unit. Out-of-ward population: patients discharged from the unit either colonised or non-colonised at time of discharge.
Discussion

The genus *Acinetobacter* spp. has been isolated from the skin of healthy humans and in acute care settings from both healthcare personnel and hospitalised patients. Acinetobacter may lead to nosocomial infection, being one of the leading pathogens causing ventilator-associated pneumonia in intensive care units.\(^\text{17,18}\) Whereas nosocomial infection is mainly caused by ACBA, this species has only rarely been reported as a human skin commensal. Thus little is known about the natural and/or persistent reservoirs of ACBA in acute care settings.\(^\text{19}\) Primary BSI is invariably caused by, or is related to, contamination of vascular catheters, mainly of central venous or arterial origin.

Our study demonstrated that ACBA strains colonised the skin of the patients in our institution corresponding to the species causing primary BSIs. Both clone A and clone B showed strains found in the blood and skin of the patients in our MICU. The recognition of clone C, the same strain being found in both an internal medicine unit and MICU, could be explained by the cross-transmission of these strains among wards in our institution, contributing to the endemicity of MDR-ACBA infection.\(^\text{20}\) Clone D belonged to a patient admitted from another institution. This finding may reflect the dissemination among different acute care settings in our country.\(^\text{21}\) The prevalence of MDR-ACBA strains and of strains susceptible only to polymyxin E was 60 and 8.5%, respectively.

Our data indicate that skin decolonisation of resistant ACBA strains could be an important additional control measure, especially in an outbreak situation involving BSI.

Patient disinfection and skin hygiene by bathing, showering and body washing with antimicrobial agents has been reported to significantly reduce the rate of cutaneous infection.\(^\text{22}\) Whole-body washing with chlorhexidine-containing detergent has been shown to reduce infections among neonates.\(^\text{23}\) Chlorhexidine-containing detergent is used in the control of outbreaks mainly caused by meticillin-resistant *Staphylococcus aureus*.\(^\text{24}\)

![Whole-body disinfection for A. baumannii](image)

**Figure 3** Pulse-field gel electrophoresis of *Acinetobacter baumannii* (ACBA) strains. Lanes 1, 5, 13 and 21 represent strains recovered from blood of patients with ACBA bloodstream infections. Lanes 2–4, 6–12, 14–20 and 22–24 represent strain isolates from patients with positive skin colonisation in the study period. Four clones were found. Clones A and B represent strains isolated from infected patients (lanes 1 and 5) and strains isolated from skin of patients who had an overlapping hospital stay in the medical intensive care unit (MICU) (lanes 2–4, 6–12). Clone C represents a strain isolated from an infected patient (lane 13) at 2 h after admission to the unit, and lanes 14–20 show closely related strains isolated from the skin of patients already in the unit. Clone D (lane 21) represents an isolate recovered from blood of an infected patient from another hospital at the same study period that shows an indistinguishable pattern compared with that of the strains isolated from the skin in our MICU (lanes 22–24).
Although preoperative showering with chlorhexidine-containing detergent seemed to reduce the frequency of surgical wound infections, a more recent paper has disputed this finding.\textsuperscript{25,26} The effectiveness of patient-source control to reduce the bio-burden of vancomycin-resistant enterococci (VRE) with cloth-saturated 2\% CG has been recently described as a simple method to decrease patient acquisition of VRE.\textsuperscript{27}

We believe that patients with skin colonisation of MDR-ACBA within ICUs would benefit from the method of decolonisation described in this paper, with monitoring for the development of chlorhexidine resistance and alertness to any allergic reactions. The aim of controlling untreatable invasive nosocomial infection caused by MDR-ACBA through reducing colonisation, while posing little or no risk to the patient, appears to be an appealing strategy. Of our patients, 17\% had ACBA on admission to the unit. Maximal reduction was achieved by day 3 (third culture). Three days of whole-body bathing appears to be adequate and could reduce and perhaps prevent, subsequent unnecessary skin decontamination.

Our study has some shortcomings. Firstly, our use of CG was not controlled against other agents. However, previous studies have shown that CG is a more effective skin disinfectant than other agents, excluding alcohol-based products.\textsuperscript{27–30} Secondly, we did not take environmental cultures and periodic personnel hand cultures. These cultures could have indicated the presence of other potential sources of new acquisition of ACBA in the unit. Further studies are needed to determine the impact of CG body washing on the patient skin colonisation acquired in the unit from hands of personnel and/or the environment. Nevertheless, the fact that 17\% of the patients were colonised in the MICU at the time of their admission suggests the impact of ACBA skin decolonisation in avoiding further dissemination into this unit. Third, we did not assess the development of CG resistance during the study period. We therefore believe that whole-body bathing should only be considered in highly endemic situations and high-risk settings when totally drug-resistant microorganisms are involved. Only two of our patients developed a skin rash, but we did not pursue a definitive aetiology in these cases.

In conclusion, skin colonisation with multidrug-resistant ACBA appears to be substantial during the first 2 h of admission among adults admitted to the MICU in our institution. Repeated whole-body bathing with 4\% CG significantly reduced patients’ skin colonisation. This method could be considered in institutions with a high endemic rate of nosocomial infection caused by MDR-ACBA, in addition to current infection control measures. We are of the opinion that the impact of 4\% CG whole-body washing on BSIs caused by ACBA in ICUs requires further investigation.

**Conflict of interest statement**
None declared.

**Funding sources**
None.

**References**
