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Major Article

Is airborne transmission of *Acinetobacter baumannii* possible: A prospective molecular epidemiologic study in a tertiary care hospital

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Key Words:
Intensive care unit
Health care-associated infection
Aerial spread
Environmental sampling

Background: Understanding the dynamics of aerial spread of *Acinetobacter* may provide useful information for production of effective control measurements. We investigated genetic relationships between air and clinical isolates of *Acinetobacter baumannii* in an intensive care unit (ICU) setting.
Methods: We conducted a prospective surveillance study in a tertiary care hospital for 8 months. A total of 186 air samples were taken from 2 ICUs. Clonal characteristics of air isolates were compared with the prospective clinical strains and the previously isolated strains of ICU patients over a 23-month period.
Results: Twenty-six (11.4%) air samples yielded *A baumannii*, of which 24 (92.3%) isolates were carbapenem-resistant. The *Acinetobacter* concentration was the highest in bedside sampling areas of infected patients (0.39 CFU/m^3). Air isolates were clustered in 13 genotypes, and 7 genotypes (including 18 air strains) were clonally related to the clinical strains of 9 ICU patients. One clone continued to be cultured over 27 days in ICU air, and air isolates could be clonally related to 7-week retrospective and approximately 15-week prospective clinical strains.
Conclusions: The results of this study suggest that infected patients could spread significant amounts of *Acinetobacter* to ICU air. These strains could survive in air for some weeks and could likely still infect new patients after some months. Special control measurements may be required against the airborne spread of *Acinetobacter* in ICUs.
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Acinetobacter baumannii is a gram-negative nonfermentative coccobacillus responsible for various life-threatening infections in health care settings. Due to the considerable ability of this bacterium to develop resistance for many classes of antimicrobials, the infections caused by *Acinetobacter* frequently result in significant mortality and morbidity.[1] Studies have reported that more than 80% of *Acinetobacter*-infected patients in an intensive care unit (ICU) may die,[2] and such infections can be associated with prolonged ICU stays of 15 days or longer and hospital stays of 30 days or longer.[3]

In hospitals, infections due to *Acinetobacter* generally develop as a result of the acquisition of this bacterium by handborne transmission from a source,[4] by using contaminated medical tools or devices,[5] and prior colonization of the patients at admission.[6] However, despite extensive infection control efforts, the incidence of *Acinetobacter* has increased all over the world during the past decade.[7] This is partly due to the excellent ability of this genus to adapt to different physical and chemical environments; it is also likely due to incomplete understanding of the spreading dynamics of *Acinetobacter*.[8] Hence, increasing interest has been focused on the airborne transmission of this bacterium.[9]

Some authors have reported that indoor air may be contaminated by *A baumannii* in hospital settings, and a genetic link between air isolate and an infecting strain has been demonstrated in a few studies.[10,11] However, no data exist about how long an aerial *Acinetobacter* can survive in ICU air, and whether an airborne strain can cause an infection in a prospective patient.

During early 2007, the Hospital Infection Control Committee (HICC) of our medical center developed an initiative to reduce health care-associated infections in our hospital, including a number of actions, such as active surveillance of nosocomial pathogens,

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implementation of standard infection control measurements, hand hygiene campaigns, frequent staff education, and standardization and enforcement of sterilization and disinfection procedures. Consequently, the rate of health care-associated infections was reduced by more than 5-fold in some clinics, and the incidence of many common nosocomial pathogens was substantially reduced. However, the incidence of *Acinetobacter* increased by more than 2-fold in our ICUs during the same period. Therefore, we conducted this study to understand the dynamics of *Acinetobacter* spread by air in 2 ICUs being actively used throughout an 8-month prospective and a 23-month retrospective period. We think that the results of this study may provide useful information for infection preventionists to consider particular measurements against possible airborne *Acinetobacter* threat.

MATERIALS AND METHODS

Setting and study design

A prospective surveillance study was conducted in Turgut Ozal Medical Center, an 1,140-bed teaching hospital with 255 ICU beds in 15 different wards. Two medical ICUs (ICU-I and ICU-II) (20 beds total) were selected as the cohort area for this study. All ICUs of the facility were being ventilated by a high- efficiency particulate arresting air conditioning system complying with the requirements of Deutsches Institut für Normung 1946-4:1999 standards. No human subject was used in this study.

Air sampling

Active air sampling was done by using an air IDEAL 3P device (BioMérieux, Marcy-l'Étoile, France) according to the manufacturer's instructions. This device is an impactor-type instrument that aspirates indoor air through a grid perforated with a pattern of 286 calibrated holes. The resulting airstreams containing microbial particles are directed onto the surface of a 100-mm agar plate. Use of the air IDEAL 3P device was validated by a third-party institution to meet International Organization for Standardization 14698-1 requirements for the control of clean rooms; it was shown to efficiently collect 100% of particles above 5 μm using the reference air sampling method of the UK Health Protection Agency.[12]

Air samples were taken from 4 previously defined points in ICU-I and ICU-II at 7- to 10-day intervals. Additionally, when a patient in these units was diagnosed with *Acinetobacter* infection, further air sampling was done on the same day from patient's bedside and from the previously defined sampling areas in the ICU. We collected 2 samples at a distance of 1 m from the bed, and 1 sample each at a distance of 2 m and 3 m, as indicated in Figure 1. Before each sampling session, control air samples were also taken under the blower vents.

Identification and antimicrobial susceptibility

Air-inoculated plates were incubated at 35°C for 24-48 hours. Any growing microorganisms were then counted, and colony-forming units were calculated per meter^3 air and identified with classic bacteriologic procedures and the Vitek II System (BioMérieux). Antimicrobial susceptibility of any *A baumannii* strains isolated was assessed using Vitek II susceptibility cards, and the results were evaluated according to Clinical and Laboratory Standards Institute criteria.[13]

Genotyping

All *A baumannii* strains were molecularly typed with DiversiLab System (BioMérieux), a repetitive sequence-based polymerase chain

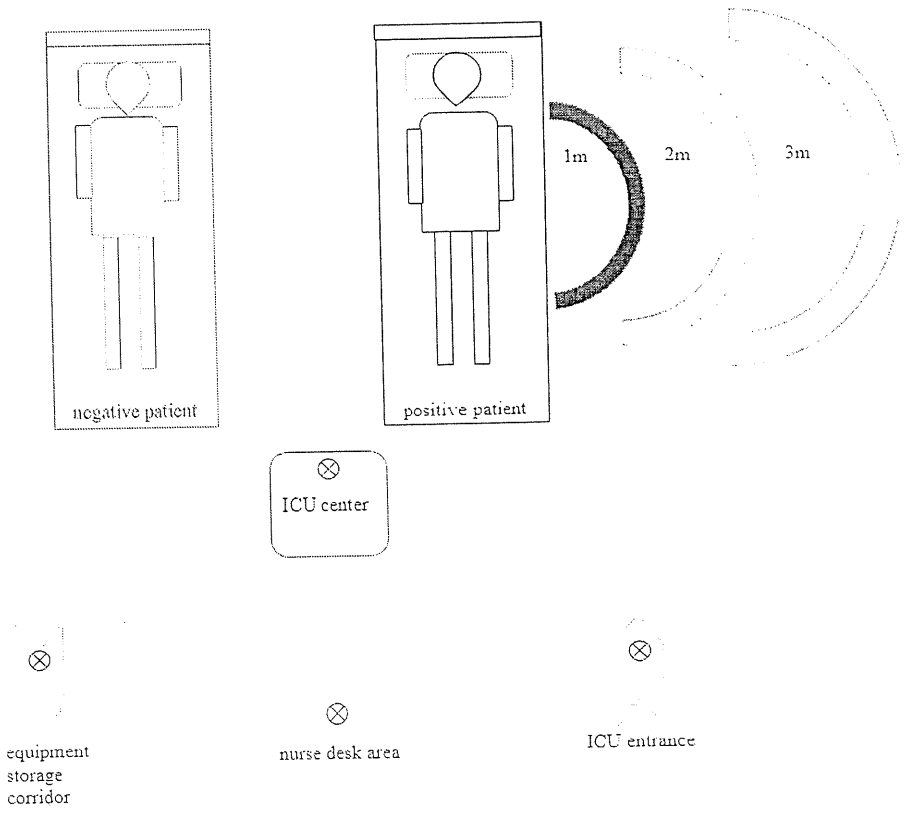


Fig 1. Air sampling areas in intensive care units (ICUs).

reaction-based rapid molecular typing method, which was able to study high numbers of strains within 4-6 hours, with excellent agreement with pulsed-field gel electrophoresis for discrimination of *Acinetobacter* strains.[14] Briefly, DNA of the isolates was extracted using the Ultra Clean Microbial DNA isolation kit (MoBio Laboratories, Carlsbad, CA), then the repetitive sequence-based polymerase chain reaction typing method was performed using a Geneamp PCR System 9700 Thermocycler (Applied Biosystems, Foster City, CA) and DiversiLab *Acinetobacter* kit. The amplified fragments, ranging from 100-1,000 bp in size, were separated electrophoretically with the microfluid labchip. Electrophorograms were downloaded and automatically analyzed using DiversiLab software (version 3.4). Finally, the software created a customized report presenting a dendogram, electrophorograms, virtual gel images, and scatter plots. Clonal relationships of the isolates were determined according to previously described criteria by Deplano et al.[15] Pulsed-field gel electrophoresis was performed to confirm the genetic relationships between the *A baumannii* strains that were found to be clonally related using the DiversiLab method.

Genotype database and evaluation of the clonal relationships

In 2011, the HICC of our hospital and the Medical Microbiology Department collaborated in a study for early determination of possible outbreaks in our hospital. According to this survey, each newly isolated nosocomial *Acinetobacter* strain was molecularly typed with the DiversiLab system, and genotypic characteristics of this new strain were compared with the previously isolated strains. The HICC was informed when a new isolate was included in any cluster. The epidemiologic links between clonally related strains were analyzed by HICC, routes of transmission were investigated, and precautions were taken as required. Thus, a genotype database was developed over a 23-month period before the initiation of this study in early 2013. Finally, the genotypic characteristics of a total of 315 clinical *Acinetobacter* isolates were collected in this database, of which 82 were from the included medical ICUs.

In this study, we prospectively investigated the clonal relationships between *A baumannii* isolates collected from air samples and the prospective clinical strains detected throughout the study period, and the prospective isolates described in this database.

RESULTS

A total of 186 air samples were taken during the study period, including 118 from ICU areas and 68 from patients' bedsides. *A baumannii* was isolated from 26 (13.9%) of these samples. Of these isolates, 24 were carbapenem-resistant, and the remaining 2 strains were susceptible to most of the antimicrobial agents tested. Antimicrobial susceptibility of the air isolates is shown in Table 1.

Of the collected air samples, 16 of 118 (13.5%) from the ICU sampling areas and 10 of 68 (14.7%) from patients' bedsides yielded *Acinetobacter*. *A baumannii* existed at 0.39 CFU/m^3 in the bedside air of infected patients (range, 0-6 CFU/m^3), and in other ICU areas it was present at 0.27 CFU/m^3 air (range, 0-4 CFU/m^3). All control air samples taken from under the blower vents were negative for any microorganism.

Genotyping identified 13 different genetic clones in all 26 of the air isolates, as indicated in Table 2. The DiversiLab system identified clonal relationships between 7 air genotypes, including 18 air isolates and 9 clinical isolates of ICU patients. Out of these, 17 air isolates were clonally related to the clinical isolates of 6 patients still hospitalized in the units. Additionally, 1 air genotype was detected in the ICU-I air cultures over 27 days, and this genotype was also found to be genetically related to a clinical isolate from a patient treated about 53 days earlier in the same unit. Furthermore, 1 air clone isolated from the central ICU-I sample was found to be clonally related with 2 prospective clinical strains. One of these clinical strains was isolated from a patient in the ICU-I about 20 days later, and another strain was isolated from a patient in ICU-II about 102 days later.

Table 1
Antimicrobial resistance of air isolates

Antimicrobial agent	Resistance	
	n	%
Ampicillin-sulbactam	25	96.1
Piperacillin	25	96.1
Piperacillin-tazobactam	24	92.3
Ceftazidime	24	92.3
Cefoperazone-sulbactam	22	84.6
Cefepime	24	92.3
Imipenem	24	92.3
Meropenem	23	88.4
Amikacin	17	65.3
Gentamycin	20	76.9
Netilmycin	12	46.1
Ciprofloxacin	24	92.3
Levofloxacin	24	92.3
Tigecycline	9	34.6
Colistin	0	0
Cotrimoxazole	21	80.7

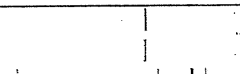
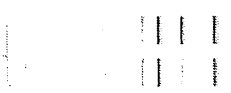
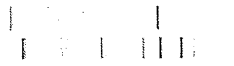
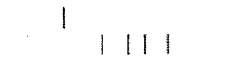
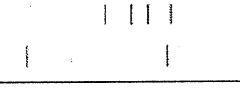
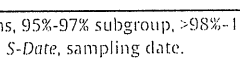
NOTE. Colistin and tigecycline were the most effective antimicrobials against *Acinetobacter* air isolates. More than 75% of air strains were multidrug-resistant.

DISCUSSION

Acinetobacter is a relatively resistant bacterium to extreme physical and chemical conditions. Studies have reported that this bacterium can survive in antiseptics[16] and in tap, distilled, and normal saline waters for more than 20 days, within the pH range of 4.5-8, up to 45°C, and can form biofilm.[17] Therefore, hospitals may be favorable environments for *Acinetobacter* colonization. This bacterium was isolated from various hospital surfaces, including patients' mattresses, bed railings, curtains, stethoscopes, computers, and telephones.[18,19] Hence, it is not surprising that hospital outbreaks are being increasingly reported, indicating the source of the epidemic clones as areas of the physical environment, such as hand hygiene sinks,[20] milk pumps,[21] headboards of beds,[22] and bag valve mask bags.[23] Baang et al[24] initiated a longitudinal molecular epidemiologic study after an outbreak of a multidrug-resistant (MDR) *Acinetobacter* clone following the admittance of an *Acinetobacter*-infected patient to a US hospital. In that prospective survey, the incidence of MDR *Acinetobacter* rapidly increased from 0.36 cases (per 1,000 patient-days) to 0.86 cases within a few months, despite immediate implementation of enhanced infection control measures. Moreover, waves of epidemiologically relevant MDR *Acinetobacter* continued to be documented for 3 years in that facility.[24]

All of the above studies indicate that *Acinetobacter* can survive in various physical environments and can cause infections and outbreaks. Additionally, it is understood that *Acinetobacter* has excellent ability to spread rapidly among patients and in the environment, and can survive for long periods of time, possibly as a result of settling in the patients', employees' and environment's microbial flora. Moreover, standard infection control measurements cannot limit the spread of *Acinetobacter*; most probably, this bacterium can even colonize environments other than surfaces that are frequently disinfected in ICUs. Therefore, air may be an alternative ecosystem for reserving *Acinetobacter*, and there is no further barrier for this organism to spread by air in the ICU environment other than single rooms, isolation rooms, or negative-pressure rooms. However,

Table 2
Genetic relationships of air and clinical isolates

ID	Air Sampling Area	Genotype ICU	Genotype No	Genotype S-Date	DNA Band Pattern: Air Isolates	DNA Band Pattern: Patient Isolate	S-Date	Relationship
1	Patient-A-1m	II	1	3/9/2013			3/8/2013	Clonal relation with Patient-A's clinical isolate (DL>95%)
2	ICU center							
3	Patient-C Bs-1m						1/23/2013	Clonal relation with Patient-B's clinical isolate (DL>96%)
4	Patient-C Bs-2m		2	3/19/2013				
5	Patient-C Bs-3m							
6	ICU center	I		3/19/2013				
7	Nurse desk		2a	4/16/2013			3/17/2013	Clonal relation with Patient-C's clinical isolate (DL>99%)
8	Storage corridor							
9	ICU entrance							
10	Patient-D Bs-3m	II	3	3/20/2013			3/19/2013	Clonal relation with Patient-D's clinical isolate (DL>98%)
11	Nurse desk							
12	Patient-E Bs-2m	II	4	4/19/2013			4/17/2013	Clonal relation with Patient-E's clinical isolate (DL>99%)
13	Patient-E Bs-1m							
14	ICU center	II	5	5/20/2013		None		No clonal relation
15	Nurse desk		5a					
16	ICU center	I	6	6/20/2013			7/10/2013	Clonal relation with Patient-F's clinical isolate (in ICU-I; DL>98%)
							10/1/2013	Clonal relation with Patient-G's clinical isolate (in ICU-II; DL>97)
17	ICU entrance	II	7/C-S	6/24/2013		None		No clonal relation
18	Nurse desk	I	8	7/10/2013		None		No clonal relation
19	ICU center	I	9	8/7/2013		None		No clonal relation
20	ICU entrance		9a					
21	Patient-H Bs-1m	I	10	9/3/2013			8/31/2013	Clonal relation with Patient-H's clinical sample (DL>99%)
22	Patient-H Bs-2m							
23	Nurse desk		10a					
24	ICU center	II	11	9/11/2013		None		No clonal relation
25	Storage room	II	12/C-S	11/20/2013		None		No clonal relation
26	Patient-I Bs-1m	II	13	11/20/2013			11/18/2013	Clonal relation with Patient-I's clinical sample (DL>96%)

NOTE. Similarity cut off value >95% clonally related strains, 95%-97% subgroup, >98%-100% same genotype, and <95% no relation.
Bs, bedside; *C-S*, carbapenem-susceptible; *DL*, DL pattern; *S-Date*, sampling date.

most modern ICUs are structured as wide rooms housing many patients in the same physical environment.

In this study, we determined that *Acinetobacter*-infected patients could discharge significant amounts of the bacteria into the air of the ICU. The air closest to patients contained higher concentrations of the organism. Additionally, we found epidemiologic links between strains isolated from the air and strains isolated from clinical samples taken from both patients who were discharged weeks earlier and patients who were hospitalized more than 3 months later (Table 2). Therefore, we think that it is most likely for this bacterium to establish a circulation chain among positive patients and the environment (including air)/employees and new patients. Hence, concurrent control of these 3 components may be essential to reduce the spread of this pathogen. Because our ICUs were ventilated according to current quality standards, we were surprised to find clinical strains in the air. Ideally, the air in ICUs should be free of organisms.

Our results indicate that ICU air is contaminated by mostly carbapenem-resistant *Acinetobacter* originating from infected patients hospitalized in the unit. We believe that because positive patients remained untreated due to antimicrobial resistance of the infecting *Acinetobacter* strain, these patients could continue to spread the pathogen. On the other hand, we did not detect any clonal relationship between the remaining 8 air strains (2 were carbapenem-susceptible) and any clinical strain. Because we only included clinical isolates from patients with laboratory-confirmed infection, these unrelated strains might originate from colonized patients/employees or undiagnosed patients in the units. Furthermore, such strains could be transferred from outside the ICU by health care staff working in more than 1 ward, or directly via the air circulation when doors are open.

We showed that *Acinetobacter* could remain in ICU air for approximately 4 weeks, and could infect the prospective patients admitted some months later. We believe that these results provide some insight into the dynamics of airborne *Acinetobacter* transmission. However, the further study is still required regarding the issue of airborne *Acinetobacter*. Therefore, comprehensive studies will be helpful to better understand the aerial spread of *Acinetobacter*. We performed this study in a limited cohort within a limited time period. Future studies should include all related medical wards, with simultaneous samplings in shorter periods, not only from patients with confirmed infection, but also from the surfaces, indoor air, and patients' and employees' body floras. Additionally, according to our experiences from this survey, following particular suggestions may be beneficial for the next researchers. Specifically, relatively lower numbers of *Acinetobacter* colonies generally grow on air-inoculated plates. Therefore, it will not be difficult to perform molecular epidemiologic studies for each colony grown on the plate, because it is sometimes problematic to discriminate *Acinetobacter* colonies from different genetic clones by direct visual evaluation. Second, we

performed air sampling at a 1-m height from the ICU floor. Therefore, taking vertical samples at different heights on the same point will be beneficial to determine whether *Acinetobacter* drops down or fluctuates in the air. This may also provide important knowledge for planning environmental precautions intended to prevent aerial *Acinetobacter* spread.

According to our results, we can predict the potential consequences of *Acinetobacter* air contamination. Firstly, the presence of a pathogen of which the human respiratory tract is among its main target areas for infection development poses a strong risk for patients in ICUs (also for health care workers). Secondly, air circulation makes it very possible for this bacterium to reach more convenient physical environments to multiply, including a number of areas that are shown to be outbreak sources. Thirdly, by airborne routes, this bacterium can contaminate skin, wounds, and catheter entry sites, potentially resulting in autoinoculation. Therefore, because contact precautions are not effective to prevent aerial transmission, it is better to isolate a positive patient in a single room, or preferably in a negatively pressurized room. Additionally, at least 1 *Acinetobacter*-effective air disinfection method should be included in standard room preparation procedures to prevent longitudinal airborne spread, particularly after an infected or colonized patient is discharged from the room. Because the air surrounding a patient contains a higher number of organisms, health care workers should wear air masks during care delivery. Lastly, the airflow dynamics of ICU ventilating systems can be revised to reduce the low- or nonvented spaces in a room where *Acinetobacter* may persist.

CONCLUSIONS

In this study, we determined that carbapenem-resistant clinical strains of *Acinetobacter* can survive in ICU air for weeks, possibly causing further nosocomial infections. Therefore, novel strategies should be sought to reduce potential health risks related to this potential transmission route.

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