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Characterization of a Hospital Outbreak of Imipenem-Resistant *Acinetobacter baumannii* by Phenotypic and Genotypic Typing Methods

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During a 13-month period, 31 patients hospitalized primarily in two intensive care units (ICUs) were either colonized or infected by imipenem-resistant *Acinetobacter baumannii*. Typing of the isolates by three methods (antibiotyping, biotyping, and pulsed-field gel electrophoresis) revealed that two distinct strains were involved in the first 9 cases of the outbreak and that one of these strains, which had acquired a higher level of imipenem resistance as well as resistance to all aminoglycosides, accounted for 21 of 22 cases in the second part of the outbreak. ICU environmental contamination was recognized as an important reservoir of this epidemic strain. The outbreak ceased only after the ICUs were closed for complete cleaning and disinfection.

Bacteria of the genus *Acinetobacter* are nonmotile, nonfermentative, aerobic, and gram-negative coccobacilli. They are widely dispersed in nature, present in the hospital environment, and also commonly part of the normal skin flora (6, 23, 27). In recent years, increasing numbers of nosocomial infections occurring either as isolated cases or as outbreaks have been caused by these bacteria, especially in patients with severe underlying diseases hospitalized in intensive care units (ICUs) (7, 12, 13, 24, 32). The genus *Acinetobacter* includes at least 17 DNA hybridization groups (genomic species) (8, 10, 28), but one species, *A. baumannii* (DNA group 2), is responsible for most human *Acinetobacter* infections.

A variety of phenotypic methods have been used to discriminate *A. baumannii* isolates, including determination of antibiotic susceptibility patterns (antibiotyping) (20), serotyping (29), phage typing (11, 20), biotyping (9), whole-cell and cell envelope protein electrophoresis (2, 11), and esterase electrophoresis (22). Recently, genomic DNA typing methods, such as ribotyping (15), analysis of macrorestriction fragments by pulsed-field gel electrophoresis (PFGE) (16, 17, 26), and PCR fingerprinting (17, 26), have also been used.

Clinical strains of *A. baumannii* are often resistant to numerous antibiotics, but fluoroquinolones, aminoglycosides (tobramycin, netilmicin, and amikacin), ticarcillin, and ceftazidime can remain active (21). Although imipenem susceptibility is almost universal, imipenem-resistant isolates causing nosocomial infections have been reported since 1986 (21, 30).

In 1991 and 1992, our hospital experienced an outbreak of colonizations and infections due to imipenem-resistant *A. baumannii*. The possibility of nosocomial transmission of one or more epidemic strain(s) was investigated by means of three typing methods, antibiotyping, biotyping, and PFGE analysis.

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MATERIALS AND METHODS

Bacterial strains. From February 1991 to March 1992, imipenem-resistant *A. baumannii* organisms were isolated from 31 patients, 26 of them hospitalized in two ICUs. The first isolate from each patient and ten imipenem-resistant *A. baumannii* environmental isolates were included in the study. Five isolates of *A. baumannii* susceptible to imipenem and collected from the two ICUs during the outbreak period were also studied. All isolates had the properties that define the genus *Acinetobacter*: they were gram-negative, strictly aerobic, oxidase-negative, and nonmotile coccobacilli. They were identified to the species level by the simplified identification scheme described by Bouvet and Grimont (9), including growth at 37, 41, and 44°C; production of acid from glucose; gelatin hydrolysis; and assimilation of 14 different carbon sources.

Environmental samples. 225 samples (room surfaces, 168; air, 12; hands and gowns of personnel, 45) were collected in January 1992 from the medical ICU (MICU) and the general surgical ICU (SICU). During the same period, 157 samples (room surfaces, 128; air, 9; hands and gowns of personnel, 20) were collected from the dermatology and urology medical units. The direct contact method (18) was used with Count-Tact agar plates (bioMérieux, Marcy l'Etoile, France) that were applied to room surfaces (windowsills, furniture, soil, handwashing sinks, patient care items, and miscellaneous locations) and the hands and gowns of ICU personnel. Air samples were collected with the RCS air sampler and agar strips (Biotest, AG, Dreieich, Germany) with a sample volume of 160 liters. Count-Tact plates and agar strips were incubated at 37°C for 24 h, suspected colonies of *A. baumannii* were identified, and susceptibility to imipenem was determined by disk diffusion.

Antibiotic susceptibility testing. For antibiotyping, disk-agar diffusion tests (Diagnostics Pasteur, Marnes-la-Coquette, France) were performed on Mueller-Hinton agar (bioMérieux) with an inoculum of 2.10^6 CFU/ml. Sixteen antibiotics were tested: ticarcillin (75 μg), ticarcillin-clavulanate (75 and 10 μg, respectively), piperacillin (75 μg), imipenem (10 μg), cefotaxime (30 μg), ceftazidime (30 μg), streptomycin (10 IU), kanamycin (30 IU), tobramycin (10 μg), amikacin (30 μg), gentamicin (15 μg), netilmicin (30 μg), polymyxin B (50 μg), tetracycline (30

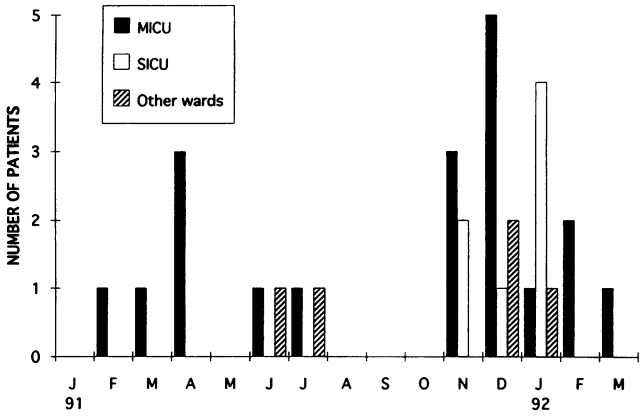

FIG. 1. Time course of the outbreak, showing the number of patients of the MICU, the SICU, and other wards colonized or infected by imipenem-resistant isolates of *A. baumannii* from February 1991 to March 1992.

IU), trimethoprim-sulfamethoxazole (1.25 and 23.75 μg, respectively), and ciprofloxacin (5 μg). MICs of imipenem (Merck Sharp & Dohme, Munich, Germany) were determined by the agar dilution technique with Mueller-Hinton agar and an inoculum of 10^4 CFU per spot. All cultures were incubated aerobically at 37°C for 24 h. The breakpoints used were those from published recommendations (1).

Biotyping. The biotyping system developed for *A. baumannii* by Bouvet and Grimont was used (9). This assay tests the ability of the strains to utilize six carbon sources (levulinate, citraconate, L-phenylalanine, phenylacetate, 4-hydroxybenzoate, and L-tartrate) and differentiates 19 biotypes (11).

Macrorestriction analysis by PFGE. Isolation, deproteinization, and restriction of DNA were performed as described by Allardet-Servent et al. (3). Restriction fragments were separated on 1% agarose gels run at 200 V in 0.5× Tris-borate-EDTA buffer (44.5 mM Tris · HCl [pH 8.0], 44.5 mM boric acid, and 1 mM EDTA) by using a CHEF-DR II system (Bio-Rad Laboratories, Nazareth, Belgium). Optimal separation was achieved by using a 5- to 13-s pulse-time linear gradient for 20 h. Dice coefficients of similarity [(number of shared fragments × 200)/total number of fragments in the two samples] were determined for each pair of isolates by visual comparison of restriction patterns (19), and results were the means of two independent determinations. Isolates with Dice coefficients of greater than 90% were assigned the same type.

RESULTS

Description of the outbreak. The susceptibility of gram-negative bacilli to imipenem has been routinely tested in our laboratory since 1985. Before the epidemic, our laboratory had detected only one isolate of *A. baumannii* with decreased susceptibility to imipenem (March 1990). From February 1991 to March 1992, 31 patients were either infected (10 patients) or colonized (21 patients) by imipenem-resistant *A. baumannii* (Fig. 1). The outbreak could be divided in two waves, from February to July 1991 (9 patients) and from November 1991 to March 1992 (22 patients). Twenty-six (84%) of these patients were hospitalized in two ICUs: 19 in the MICU and 7 in the SICU. The remaining five patients were located in various wards. The sources of first isolation were the lower respiratory tract (11 cases), blood (7 cases), intravascular catheters (5 cases), abdomen-related sites (4 cases), urine (3 cases), and wound infection (1 case). During the outbreak, imipenem-resistant isolates accounted for 11% of the total number of *A. baumannii* clinical isolates detected by our laboratory. Rates of infection and/or colonization by *A. baumannii* at our hospital increased 23% between 1990 and 1991 (from 5.6 to 6.9 per 1,000 admissions). Annual imipenem consumption at our hospital was stable between 1988 and 1990 (approximately 3.5 kg) but increased by 55% in 1991.

TABLE 1. Results of environmental culture in the MICU and SICU

Location	No. of samples	No. (%) positive for *A. baumannii*	
		Imipenem-susceptible	Imipenem-resistant
Room surfaces	168	25 (15)	31 (18)
Windowsills	40	7 (17)	7 (17)
Furniture	50	9 (18)	8 (16)
Patient care items	24	2 (8)	6 (25)
Handwashing sinks	21	2 (9)	2 (9)
Soil	17	4 (23)	7 (41)
Miscellaneous	16	1 (6)	1 (6)
Air	12	1 (8)	0 (0)
ICU personnel	45	4 (9)	2 (4)
Gowns	28	2 (7)	2 (7)
Hands	17	2 (12)	0 (0)

Environmental samples. Imipenem-resistant *A. baumannii* organisms were isolated from 18 and 7% of the samples collected from room surfaces of the two outbreak-related ICUs and from gowns of ICU personnel, respectively (Table 1). Samples collected from the dermatology and urology wards, where two cases of colonization or infection by imipenem-resistant *A. baumannii* had been observed, were negative.

Imipenem resistance. All nine clinical isolates of the first epidemic wave and a single one of the second (HM148) had intermediate susceptibility to imipenem (zone diameters, 17 to 21 mm; MIC, 8 μg/ml). All but 1 of the 22 clinical isolates of the second wave and all 10 environmental isolates were resistant (zone diameters, 6 to 14 mm; MICs, 32 to 64 μg/ml).

Typing methods. Among the 41 imipenem-resistant clinical and environmental isolates studied, two antibiotypes (designated S and R), two biotypes, and two types of *Apa*I DNA patterns (designated X and Y) were identified. Antibiotype S was characterized by resistance to all the antibiotics tested except polymyxin B, kanamycin, tobramycin, amikacin, and netilmicin, whereas isolates of antibiotype R were susceptible to polymyxin B only. Isolates belonged either to biotype 1 (absence of utilization of citraconate) or to biotype 2 (absence of utilization of citraconate and L-tartrate). *Apa*I patterns assigned to types X and Y showed similarity values of greater than 91 and 95%, respectively. Several subtypes were individualized within each type (Fig. 2), subtypes Xa and Ya being the most frequently observed. Similarity values of less than 72% were obtained when the patterns of type X were compared with those of type Y.

The five isolates of *A. baumannii* susceptible to imipenem were resistant to all the other antibiotics tested except polymyxin B and were of biotypes 1, 2, 6 (two isolates), and 9. Their *Apa*I patterns differed markedly from one another and from those of types X and Y (range of similarity values, 42 to 78%) (data not shown).

Associations of markers. Among imipenem-resistant iso-

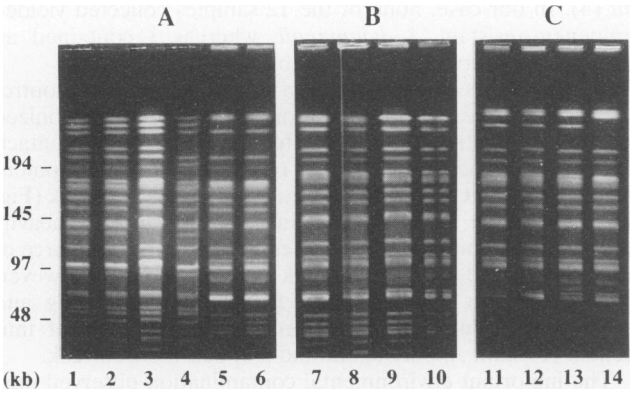

FIG. 2. PFGE patterns of *Apa*I-digested genomic DNA obtained from imipenem-resistant *A. baumannii* isolates. (A) First period of the outbreak (February to July 1991). Lanes: 1 to 4, isolates of type X (subtype Xa [lanes 1 and 2], subtype Xb [lane 3], and subtype Xc [lane 4]); 5 and 6, isolates of type Y (subtype Ya). (B) Second period of the outbreak (November 1991 to March 1992); all isolates are type Y. Lanes: 7, 8, and 10, subtype Ya; 9, subtype Yb. (C) Environmental isolates of type Y. Lanes: 11 and 13, subtype Yb; 12, subtype Ya; 14, subtype Yc. Size markers (lambda DNA concatemer) are shown on the left.

lates, three different associations of markers were observed (Table 2): (i) antibiotype S, biotype 1, and *Apa*I pattern X; (ii) antibiotype S, biotype 2, and *Apa*I pattern Y; and (iii) antibiotype R, biotype 2, and *Apa*I pattern Y. The latter two types differed only by susceptibility or resistance to kanamycin, tobramycin, amikacin, and netilmicin. Moreover, the PFGE-generated *Sma*I patterns of isolates of these two types were similar but differed markedly from those of isolates of the first type (data not shown). Thus, the outbreak was caused by two distinct strains designated A (antibiotype S, biotype 1, and *Apa*I pattern X) and B (antibiotype S or R, biotype 2, and *Apa*I pattern Y). These strains were responsible for a similar number of cases in the first epidemic wave, but strain B accounted for 21 of the 22 cases in the second wave. The 10 environmental isolates studied also belonged to strain B (Table 2).

Control measures. In November 1991, colonized and infected patients were housed in single rooms with contact isolation precautions. Use of a disinfectant (chlorhexidine) for handwashing after patient contact was recommended. Following patient discharge, rooms and room equipment were cleaned and extensively decontaminated with formaldehyde. Since these measures had no effect on the outbreak (Fig. 1), the two ICUs were closed (February 1992) for complete cleaning and disinfection after colonized and infected patients had been transferred to other wards with isolation precautions. These patients were not reintroduced into the disinfected ICUs. Afterwards, environmental samples taken on five occasions from each ICU over 18 months of follow-up remained negative. During the same period, susceptibility to imipenem was determined by disk diffusion for all isolates of *A. baumannii* detected from clinical samples of patients of the two ICUs. No new cases of colonization or infection with imipenem-resistant *A. baumannii* were recorded. The overall incidence of *A. baumannii* colonizations and infections at our hospital decreased from 6.9 per 1,000 admissions in 1991 to 4.7 in 1992 (31.8% reduction).

DISCUSSION

Isolates from an outbreak of infections and colonizations by imipenem-resistant *A. baumannii* were investigated by three methods, antibiotyping, biotyping, and PFGE analysis. The first two techniques are easy to perform and are largely used by clinical microbiology laboratories for epidemiological typing of *A. baumannii*. Their discriminatory power is low because the majority of clinical *A. baumannii* isolates are resistant to multiple antibiotics and belong to one of only four biotypes (1, 2, 6, and 9) (9, 20). However, biotyping alone or in combination with antibiotyping has been shown to be a valuable method of screening (11, 20). In our study, two biotypes (1 and 2) and two antibiotypes were distinguished but the two markers did not always correlate (Table 2); a third method was needed to delineate this outbreak.

Other powerful phenotypic typing techniques, such as serotyping (29), phage typing (11, 20), whole-cell or cell envelope electrophoresis (2, 11), and esterase electrophoresis (22), have been developed for *A. baumannii*, but their use is limited to a few specialized centers. Among genotypic techniques, ribotyping has been shown to have a high discriminatory power for *A. baumannii* but is rather labor-intensive (15). PCR fingerprint-

TABLE 2. Phenotypic and genotypic characteristics of outbreak-related isolates of *A. baumannii*

Period	Location	Source	Antibiotype[a]	Biotype	PFGE (*Apa*I)[b]
February to July 1991	MICU	5 patients	S	1	X
	MICU	1 patient	S	2	Y
	MICU	1 patient	R	2	Y
	Gastroenterology	1 patient	S	2	Y
	Nephrology	1 patient	S	2	Y
November 1991 to March 1992	MICU	1 patient	S	1	X
	MICU	11 patients	R	2	Y
	SICU	7 patients	R	2	Y
	Dermatology	1 patient	R	2	Y
	Neurosurgery	1 patient	R	2	Y
	Urology	1 patient	R	2	Y
January 1992	MICU	5 environmental isolates	R	2	Y
	SICU	5 environmental isolates	R	2	Y

[a] Antibiotype S is defined as resistance to all the antibiotics tested except polymyxin B, kanamycin, tobramycin, amikacin, and netilmicin. Antibiotype R is defined as resistance to all the antibiotics tested except polymyxin B.

[b] Macrorestriction genotype as determined by PFGE after digestion by restriction enzyme *Apa*I. The two patterns observed were designated X and Y.

ing with arbitrarily selected primers is a rapid and simple method that has been successfully used for strain typing of *A. baumannii* (17, 26). However, a lack of reproducibility has been reported for PCR fingerprinting (5). PFGE analysis has proven to be a stable, reproducible, and discriminating technique for typing a wide variety of bacterial species, including *A. baumannii* (16, 17, 26). Two studies have established the diversity of PFGE-generated *Apa*I profiles obtained with this species (16, 17). Furthermore, a significant DNA polymorphism was found among strains belonging to biotype 2 (25 of the 31 cases of the outbreak were due to isolates belonging to biotype 2) (16). In our study, the results of PFGE analysis confirmed those of biotyping and showed that two distinct strains, designated A and B, were involved in the outbreak, with strain B predominating during the second epidemic wave. Concurrent transmission of two epidemic strains of *A. baumannii* has been observed previously (26, 31). In our case, the almost simultaneous appearance in the same unit of two different imipenem-resistant strains suggests the transfer of resistance from one strain to the other.

Although antibiotyping alerted us to the emergence of an *A. baumannii* outbreak, it did not allow us to distinguish between the two strains responsible. Two antibiotypes were observed (S and R), differing only in susceptibility or resistance to several aminoglycosides. Whereas all type A isolates belonged to antibiotype S, type B isolates were of antibiotype S or R. These results illustrate not only the lack of discriminatory power of antibiotyping as an epidemiological marker for *A. baumannii* but also its instability, which was, in our case, probably due to the presence or absence in type B isolates of a plasmid or another mobile genetic element carrying an aminoglycoside resistance gene.

All type A and type B isolates of the first epidemic wave had intermediate susceptibility to imipenem (MIC, 8 μg/ml), whereas all the type B isolates of the second wave were resistant (MICs, 32 to 64 μg/ml). It is possible that one type B isolate acquired an additional mechanism of resistance to imipenem, which gave it a selective advantage, thus permitting its spread in the second part of the outbreak. An increase in imipenem resistance and the acquisition of resistance to all aminoglycosides (antibiotype R) seem to be unlinked since one isolate of antibiotype R, HM129, had intermediate susceptibility to imipenem. Sporadic isolations of imipenem-resistant *A. baumannii* have been reported since 1986 (21), but our observation as well as that of Urban et al. (30) shows that hospital outbreaks can be due to such strains.

In most previous reports, an environmental source for the epidemic strain could be identified, e.g., ventilators (12), ventilator tubing (13), resuscitation bags (25), arterial pressure transducers (7), or mattresses (24). During this epidemic, we did not recognize an environmental source with a significantly higher frequency of isolation of imipenem-resistant *A. baumannii* but found that all ICU room surfaces were extensively contaminated (Table 1). Contaminated hands and gloves of the ward staff seem to have an important role in patient-to-patient transmission of *A. baumannii* (14). In our study, 7% of the samples collected from the gowns of ICU personnel were positive for imipenem-resistant *A. baumannii* but those collected from their hands were negative (Table 1). However, the technique used (direct contact plates) has limited sensitivity for detecting hand carriage compared with that of the broth-bag method (26). A transient colonization of the hands of the ICU staff by the epidemic strains was, nonetheless, most probably present, and we think that both hands and gowns were involved in transmission. Airborne dispersal has been proposed as a mode of indirect contamination in an outbreak of *A. baumannii* (4). In our case, none of the 12 samples collected yielded imipenem-resistant *A. baumannii*, whereas 1 contained an imipenem-susceptible isolate (Table 1).

The measures initially taken in November 1991 to control the outbreak, i.e., contact isolation precautions for colonized and infected patients, hand disinfection after patient contact, and cleaning and disinfection of rooms and room equipment following patient discharge, had no effect on the outbreak (Fig. 1). This failure was probably due to the fact that heavily contaminated room surfaces represented an indirect source of contamination for both patients and personnel. However, closing the two ICUs involved for complete cleaning and disinfection eliminated the environmental reservoir of imipenem-resistant *A. baumannii* and stopped the outbreak.

The important environmental contamination observed during the outbreak led us to improve the routine practices of room cleaning and disinfection following patient discharge. This procedure now includes the especially difficult-to-reach (hidden and/or elevated) room surfaces which were previously cleaned and disinfected only once a month. Moreover, emphasis has been placed on the need for physicians to limit the use of imipenem to a few well-defined indications. As a consequence, annual consumption of imipenem at our hospital in 1992 and 1993 decreased by approximately 20% compared with that in 1991.

Thus, this hospital outbreak presented several peculiar features. Isolates were imipenem-resistant, and PFGE analysis permitted us to determine that two distinct strains were involved. One strain acquired a higher level of imipenem resistance as well as resistance to all aminoglycosides and predominated during the second part of the outbreak. Room surfaces of the two outbreak-related ICUs were extensively contaminated, and the outbreak ceased only after closing these ICUs for complete cleaning and disinfection.

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