EPIDEMIOLOGICAL TYPING

The epidemiology of acinetobacter infections in Hong Kong

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A retrospective survey was conducted of the characteristics of acinetobacter infections in Hong Kong — seasonal and geographic distributions, frequency of isolation from various body sites, antimicrobial susceptibility and molecular epidemiology. Most (80%) isolates of *Acinetobacter* spp. belonged to DNA groups 2 (*A. baumannii*) or 13, as defined by growth at 44°C. An increased isolation rate in summer was related to higher ambient temperatures. The notion that acinetobacters are opportunist nosocomial pathogens was supported by the body site- and ward-specific distributions, which were similar to those of *Pseudomonas aeruginosa* and in marked contrast to those of coagulase-negative staphylococci and *Escherichia coli*. Typing of *Acinetobacter* isolates by arbitrary-primed polymerase chain reaction revealed extensive genotypic polymorphism, suggesting that numerous unrelated strains were circulating between patients. In view of the association with a high incidence of polymicrobial bacteraemia and multiresistance to antibiotics, a careful selection of appropriate antibiotics in combination is necessary for empirical therapy of infections caused by *Acinetobacter* spp.

Introduction

The genus *Acinetobacter* — which comprises non-fermentative gram-negative bacteria of low pathogenicity — has attracted recent clinical and research interest as members of the genus are implicated increasingly as nosocomial pathogens. Ubiquitous in soil and water, *Acinetobacter* spp. are also part of the indigenous flora of human skin. The remarkable resistance of some of these micro-organisms to prolonged desiccation and to multiple antibiotics has allowed them to adapt successfully to the hospital niche [1, 2]. The genus comprises at least 17 DNA groups (genomic species) [3–5], of which *A. baumannii* (DNA group 2) — a glucose oxidiser that is able to grow at 44°C — is involved most frequently in hospital outbreaks [6–9].

Several studies on the epidemiological and clinical features of acinetobacter infections have been published [10–15]. Unlike the low incidence of *Acinetobacter* spp. isolated from in-patients in the West [10], an endemic presence of these organisms has been observed in this hospital [16]. As comprehensive data about the prevalence of these organisms amongst

hospitalised patients in this locality are lacking, a retrospective survey was conducted to elucidate the role of *Acinetobacter* spp. as opportunist nosocomial pathogens. The annual incidence of isolation and seasonal prevalence during the 5-year period 1990–1994 was determined. The relative distribution of *Acinetobacter* spp. with respect to isolation site and geographic location within the hospital, either in the general ward or intensive care unit (ICU), was compared to those of three other established nosocomial pathogens (*Pseudomonas aeruginosa*, coagulase-negative staphylococci and *Escherichia coli*). As the spectrum of infection caused by these organisms differs — *P. aeruginosa* is involved mainly in wound, respiratory and urinary tract infections; coagulase-negative staphylococci are linked usually to foreign device-related infections; and *E. coli* is related generally to intra-abdominal and urinary tract infections — they served as references for comparison. The epidemiological relationships between some of the *Acinetobacter* isolates were investigated by arbitrary-primed polymerase chain reaction (AP-PCR).

Materials and methods

This study was performed at the Queen Mary Hospital, a tertiary teaching hospital with 1350 beds that serves a population of 1.5 million in Hong Kong. Bacteriologi-

cal data from 1 Jan. 1990 to 30 Nov. 1994 were retrieved from the microbiology laboratory database and reviewed. When multiple isolates of the same organism from the same site were reported for one patient, only a single entry was included. Likewise, only the first positive blood culture from the same patient was included in the enumeration of bacteraemic episodes.

Bacteriology

Acinetobacter spp. were identified routinely as non-motile, oxidase-negative gram-negative coccobacilli that grew only in aerobic incubation. The ability of each isolate to oxidise glucose in Hugh and Leifson's medium was tested. The isolates were reported as glucose-oxidising or non-glucose-oxidising strains of *Acinetobacter* spp. Since early 1995, non-haemolytic glucose-oxidising isolates from blood, wound, respiratory and urinary tract specimens were tested for growth at 44°C. These temperature-tolerant isolates were identified presumptively as *A. baumannii* (DNA group 2) or the unnamed DNA group 13 [5]. There was no further attempt to differentiate the isolates by DNA hybridisation or extensive substrate assimilation tests. Isolates of *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* were identified according to standard criteria [17]. When isolated from respiratory tract and wound specimens, unless Gram's stain of the specimen smear showed bacteria and a significant increase in the number of white blood cells, *Acinetobacter* spp., *P. aeruginosa* and coagulase-negative staphylococci were regarded as contaminants and thus not reported. Similarly, for urinary tract specimens, these organisms were reported only in instances of significant bacteriuria ($>10^5$ cfu/ml). In addition, they were also reported in catheter culture-positive specimens (>15 cfu).

Antimicrobial susceptibility testing

In-vitro susceptibility testing was performed routinely in this laboratory on all significant *Acinetobacter* isolates by the disk diffusion method recommended by the National Committee for Clinical Laboratory Standards [18]. Antibiotic disks were obtained from Becton Dickinson and contained ampicillin (10 µg), cephalothin (30 µg), cefuroxime (30 µg), ceftriaxone (30 µg), ceftazidime (30 µg), amoxycillin + clavulanic acid (20 and 10 µg, respectively), imipenem (10 µg), gentamicin (10 µg), netilmicin (30 µg), amikacin (30 µg), trimethoprim + sulphamethoxazole (1.25 and 23.75 µg, respectively) or ofloxacin (5 µg). For quality control, the antibiotic susceptibility of the standard *P. aeruginosa* strain ATCC 27853 was tested routinely.

Arbitrary-primed PCR (AP-PCR)

In total, 60 clinical strains isolated from blood (28), respiratory tract (14), urinary tract (10) and wound (8)

specimens — presumed to be *A. baumannii* or the unnamed DNA group 13 because of growth at 44°C — were retrieved from stock cultures for AP-PCR. *A. baumannii* strain ATCC 19606 served as an internal control in every experiment. Each isolate was streaked first on blood agar and incubated at 37°C overnight. One colony of each isolate was suspended in 1 ml of sterile water. A crude DNA extract was obtained by heating the bacterial suspension at 95°C for 15 min. After centrifugation at 12 000 *g* for 15 min, 2 µl of the supernate were added to 48 µl of PCR reaction mixture containing 10 mM Tris-HCl (pH 8.0), 50 mM KCl, 1.5 mM MgCl₂, 200 µM (each) of the four deoxynucleoside triphosphates (Pharmacia), 20 µM (GTG)₅ primer (Department of Molecular Medicine, King's College, University of London) and 2.5 U of *Taq* polymerase (Perkin-Elmer Cetus, Emeryville, USA). A negative control containing the reaction mixture without any added DNA sample was included in all experiments. In preliminary studies, (GTG)₅ yielded better banding patterns than two other primers tested: T₇ (5'-GTA ATA CGA CTC ACT ATA G-3') and LD (5'-CCA CAC GCG CAC ACG GGA-3'). PCR was performed in a Gene Cyclor (BioRad) with the following programme: 30 cycles of denaturation at 94°C for 1 min, low stringency annealing at 45°C for 2 min and extension at 72°C for 1 min. The reaction was terminated with a final extension cycle at 72°C for 6 min. The PCR products were separated by electrophoresis at 100 V for 1.5 h in an agarose 2% w/v gel, and detected by staining with ethidium bromide.

Results

Incidence

Data were available for 7475 strains of *Acinetobacter* isolated between Jan. 1990 and Nov. 1994. The average annual incidence of isolation was 298/10 000 hospital admissions (Table 1). *Acinetobacter* spp. accounted for 34.9, 11.3 and 7.4% of non-fermenters, gram-negative bacilli and all bacteria, respectively. *Acinetobacter* spp. capable of oxidising glucose made up 94.5% of all isolates and 83.8% of the isolates tested (202 of 241 isolates) were able to grow at 44°C. Hence, with regard to genotype, most isolates were presumed to belong to the temperature-tolerant genospecies *A. baumannii* (DNA group 2) or the unnamed DNA group 13 [5]. Unless stated otherwise, the following sections focus specifically on the glucose-oxidising strains of *Acinetobacter* spp.

Seasonal prevalence

A seasonal variation in the isolation rate of *Acinetobacter* spp. was observed in the general ward (Fig. 1). The largest number of isolates was obtained during July–Oct., 1990–1993. The peak period corresponded with an increased number of isolates from all body sites. However, in the ICU, apart from a peak in Nov.

Table 1. The incidence and glucose oxidation capability of *Acinetobacter* spp. isolated during 1990–1994

Annual incidence (per 10 000 hospital admissions):	298	
<i>Acinetobacter</i> spp. isolated as a percentage of:		
total non-fermenters (n = 21 442)	34.9%	
total gram-negative bacilli (n = 66 251)	11.3%	
all bacteria (n = 101 114)	7.4%	
Subdivision of total <i>Acinetobacter</i> spp. (n = 7 475) by area of isolation*:	GW	ICU
capable of glucose oxidation	72.6%	21.9%
not capable of glucose oxidation	5.2%	0.3%

*GW, general ward; ICU, intensive care unit.

Fig. 1. Seasonal distribution of (a) *Acinetobacter* spp. and (b) *P. aeruginosa* isolated from patients in the general ward (—) and the intensive care unit (····).

1991, the isolation rate was constant with an average of 36 isolates/month. This increase in the number of *Acinetobacter* spp. in Nov. 1991 was caused by an increased number of positive surveillance cultures from the patients and the environment, obtained as part of an outbreak investigation. As suitable molecular typing methods were unavailable at that time, it was not possible to conduct a full-scale epidemiological investigation.

The observed seasonality prompted an examination of the monthly distribution of *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* during the same period. A similar ward-dependent seasonal isolation rate of *P. aeruginosa* was seen. In the general ward, the largest number of isolates of *P. aeruginosa* was collected during June–Sept., 1990–1994 (Fig. 1), while in the ICU, the isolation rate remained steady with an average of 34 isolates/month. Unlike *Acinetobacter* spp. and *P. aeruginosa*, the incidence of coagulase-negative staphylococci and *E. coli* isolates was unaffected by seasonal changes (data not shown).

Comparative distribution of Acinetobacter spp. according to isolation site in patients in the general ward and ICU

In the general ward, 32.8, 25.8, 14.6 and 11.4% of *Acinetobacter* isolates were from wound, urinary tract, lower respiratory tract and sputum specimens, respectively (Table 2). In the ICU, the isolates were mainly from lower respiratory tract specimens (75.5%).

The distribution of *P. aeruginosa* amongst general ward patients was similar to that of *Acinetobacter* spp., with 22.4% of isolates from wound, 27.9% from urinary tract, 11.1% from lower respiratory tract and 28.3% from sputum specimens (Table 2). Likewise, the major isolation site in ICU patients was the lower respiratory tract (61.6%).

The relative distributions of coagulase-negative staphylococci and *E. coli* differed from those of *Acinetobacter* spp. and *P. aeruginosa* (Table 2). Coagulase-negative staphylococci were found mainly in urinary tract, wound and blood specimens from patients in the general ward (31.3, 25.9 and 20.4%, respectively), and from CSF and i.v. catheters and lower respiratory tract specimens of ICU patients (31.0 and 29.9%, respectively). In contrast, *E. coli* was isolated primarily from the urinary tract of patients in both wards (73.0 and 32.9%, respectively).

Comparative distribution of Acinetobacter spp. in bacteraemic patients in the general ward and ICU

In total, there were 9104 bacteraemic episodes in hospitalised patients admitted between Jan. 1990 and Nov. 1994. *Acinetobacter* spp. accounted for 14.8 and 4.8% of all bacteraemic episodes in the ICU and general ward, respectively (Table 3). *Acinetobacter* spp. were the second most frequently encountered organisms, after coagulase-negative staphylococci, amongst all bacteria isolated from bacteraemic ICU patients.

The incidence of polymicrobial bacteraemic episodes amongst the four bacteria was highest with *Acinetobacter* spp. (25.2%) (Table 3). The frequency of other organisms isolated simultaneously with *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* was 15.5, 13.7 and 14.6%, respectively. When the types of organism isolated concurrently with each of the four groups were compared, similarity between *Acinetobacter* spp. and coagulase-negative staphylococci was observed (Table 4). More than half of the organisms isolated in conjunction with each of these two groups were skin colonisers and other non-fermenters likely to be acquired ectogenously by patients. This was in contrast to *P. aeruginosa* and *E. coli*, where at least half of the co-existing organisms were probably of

Table 2. Distribution of *Acinetobacter* spp., *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* according to isolation site in patients in the general ward and intensive care unit

Isolation site	Percentage of isolates from							
	General ward				ICU			
	<i>Acinetobacter</i> spp. (n = 5416)	PA (n = 7253)	CNS (n = 8059)	<i>E. coli</i> (n = 15 401)	<i>Acinetobacter</i> spp. (n = 1630)	PA (n = 941)	CNS (n = 802)	<i>E. coli</i> (n = 550)
Sputum	11.4	28.3	3.1	1.0	5.2	13.3	3.2	2.3
LRT*	14.6	11.1	3.3	0.8	75.5	61.6	29.9	20.9
Urinary tract*	25.8	27.9	31.3	73.0	2.3	4.5	5.9	32.9
Wound*	32.8	22.4	25.9	10.1	4.9	6.8	11.3	11.7
Blood	6.9	3.3	20.4	8.8	3.8	0.8	9.2	2.0
Body fluid*	5.8	5.4	5.7	6.0	5.3	11.1	9.4	28.9
Others*	2.7	1.7	10.4	0.3	3.0	1.9	31.0	1.4

PA, *P. aeruginosa*; CNS, coagulase-negative staphylococci; LRT, lower respiratory tract.

*LRT specimens included bronchoalveolar lavage, endotracheal and bronchial aspirates; urinary tract specimens included mid-stream, catheterised and nephrostomic urine; wound specimens included superficial and deep wound swabs; body fluid included bile, pleural, peritoneal, peritoneal dialysis and drain fluids; others included cultures from CSF and i.v. catheters.

Table 3. The incidence of bacteraemia associated with *Acinetobacter* spp., *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* in patients in the general ward and intensive care unit

Location	Percentage of bacteraemic episodes*			
	<i>Acinetobacter</i> spp.	PA	CNS	<i>E. coli</i>
Bacteraemic episodes in general ward (n = 8799)	4.8	2.7	18.7	14.7
Bacteraemic episodes in ICU (n = 305)	14.8	2.6	28.0	4.2
Polymicrobial bacteraemic episodes (both locations)	25.2	15.5	13.7	14.6

PA, *P. aeruginosa*; CNS, coagulase-negative staphylococci.

*Other organisms causing bacteraemia included *Staphylococcus aureus*, other members of the Enterobacteriaceae (e.g., *Salmonella* and *Klebsiella* spp.), non-fermenters (e.g., *Xanthomonas* and *Pseudomonas* spp.) and yeasts.

Table 4. Organisms isolated in conjunction with *Acinetobacter* spp., *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* from episodes of polymicrobial bacteraemia

Organisms	Percentage of polymicrobial episodes			
	<i>Acinetobacter</i> spp. (n = 106)	PA (n = 46)	CNS (n = 222)	<i>E. coli</i> (n = 187)
CNS	25.5	15.2	...	4.6
Other skin colonisers*	9.4	4.3	30.2	5.5
<i>Acinetobacter</i> spp.	...	6.5	9.6	2.5
<i>Xanthomonas maltophilia</i>	9.4	2.1	8.9	0.0
Other non-fermenters*	11.4	2.3	11.0	6.4
<i>E. coli</i>	5.7	10.9	3.2	...
<i>Klebsiella</i> spp.	7.5	10.9	2.8	30.0
Other enteric colonisers*	8.5	13.0	2.1	5.5
<i>Streptococcus</i> and <i>Enterococcus</i> spp.	16.9	17.4	12.4	27.4
Others*	5.7	17.4	33.3	17.4

PA, *P. aeruginosa*; CNS, coagulase-negative staphylococci.

*Other skin colonisers included species of *Bacillus* and *Corynebacterium*; other non-fermenters included species of *Pseudomonas* and *Flavobacterium*; other enteric colonisers included other members of the Enterobacteriaceae; Others included *S. aureus* and species of *Clostridium*, *Bacteroides*, *Aeromonas*, *Rhodotorula* and *Candida*.

endogenous origin (e.g., members of the Enterobacteriaceae and streptococci). Furthermore, the genus *Acinetobacter* was unique in that it existed with organisms of ectogenous and endogenous sources in similar ratio.

Antimicrobial susceptibility

The susceptibility of *Acinetobacter* spp. to 11 antimicrobial agents has remained unchanged over the past 5 years. At least half of the glucose-oxidising isolates were insusceptible to most β -lactam agents (ampicillin, cephalothin, cefuroxime, ceftriaxone and amoxycillin + clavulanic acid), gentamicin and trimethoprim + sulphamethoxazole (Table 5). However, 55.4% of these isolates were sensitive to ceftazidime. Susceptibility to other aminoglycosides (netilmicin and amikacin) and the quinolone ofloxacin ranged between 66.4 and 72.7%. Most of these isolates were susceptible to imipenem, with resistance detected in only 5.8% of the isolates. In contrast, the majority of non-glucose-oxidising isolates were susceptible to most antimicro-

bial agents tested, except cephalothin which was active against only 14.1% of the isolates.

Genotypic polymorphism

Examples of amplification products obtained by PCR with (GTG)₅ as the arbitrary primer are shown in Fig. 2; 19 unique banding patterns were observed amongst the 60 clinical isolates identified presumptively as *A. baumannii* or the unnamed DNA group 13. Each PCR profile contained one to six DNA fragments with sizes of 0.18–1.01 kb. These profiles differed amongst isolates derived from patients residing in the same wards.

Discussion

Acinetobacter spp. have been isolated commonly from clinical specimens in this locality over the past 5 years, comprising 35% of non-fermenters, 11% of gram-negative bacilli and 7% of all bacteria isolated (Table 1). These values are similar to the corresponding

Table 5. The antimicrobial susceptibility of *Acinetobacter* spp. isolated during 1990–1994

Antimicrobial agent	Percentage of isolates susceptible*	
	Glucose-oxidising strains (n = 7061)	Non-glucose-oxidising strains (n = 410)
Ampicillin	0.3	75.9
Cephalothin	0.1	14.1
Cefuroxime	3.5	77.9
Ceftriaxone	4.7	ND
Ceftazidime	55.4	65.0
Amoxycillin + clavulanic acid	22.3	88.9
Imipenem	94.2	100.0
Gentamicin	47.9	95.3
Netilmicin	66.4	96.9
Amikacin	72.7	75.7
Trimethoprim + sulphamethoxazole	49.1	86.1
Ofloxacin	66.7	87.2

ND, not determined.

*Susceptibility of isolates was determined by the disk diffusion method [18].

Fig. 2. Examples of amplified DNA fragments of glucose-oxidising *Acinetobacter* isolates (presumed to be *A. baumannii* or DNA group 13) generated by AP-PCR with (GTG)₅ as the primer. Lane M, DNA molecular size markers (ϕ X174 DNA-*Hae*III); 1 and 9, negative control; 4, internal control (*A. baumannii* strain ATCC 19606); 2 and 8, isolates from different ICU patients; 3, 4, 7 and 10, isolates from different general ward patients; 5 and 6, isolates from patients admitted to the same general ward at different dates; 11–13, isolates from patients admitted to another general ward at different dates; all isolates were obtained from blood specimens.

figures of 37, 12 and 7% reported in Taiwan and Singapore [19, 20].

The increase in the prevalence of nosocomial *Acinetobacter* spp. during summer has also been observed in the USA by the Centers for Disease Control [12]. In investigating invasive acinetobacter infections, it was found that 53% of 32 meningitis cases and 41% of 27 bacteraemic episodes occurred in summer [13, 21]. The higher temperature and humidity

of the ambient air in summer has been postulated to contribute to this peak incidence [22]. We suggest that if the warm weather is a contributing factor to the seasonality of acinetobacter infections, then a similar pattern for other organisms from environmental sources should be observed. Indeed, an annual increase of *P. aeruginosa* isolates during the summer months was evident (Fig. 1). The fact that the seasonal variation was observed in the general ward, but not in the ICU, further supported the proposition. In the early

1990s, the ICU was the only air-conditioned ward. Hence it is not unexpected that the seasonal pattern was absent in the ICU as a constant room temperature was maintained. Moreover, central air-conditioning was extended to all general wards in early 1994, which coincided with and may account for the lack of any seasonal increase of *Acinetobacter* isolates in the same year. Therefore, the observation of seasonality in some reports, and its absence in others [14], could be explained by the variation in ambient temperature, or the lack of it in air-conditioned wards. However, we are unable to explain why the peak incidence of *P. aeruginosa* isolates persisted in 1994 after the installation of air-conditioning in all wards. It is possible that the organism's predilection for moist environments has contributed to its increased isolation during Hong Kong's humid summers.

The characteristics of acinetobacter infections were studied with a comparative approach. An attempt was made to correlate laboratory data with those of established nosocomial pathogens whose ecological reservoir was either related strongly to the hospital environment (*P. aeruginosa*), skin (coagulase-negative staphylococci) or the gastrointestinal tract (*E. coli*). In comparing the relative distribution of *Acinetobacter* spp. with those of the reference organisms, it was hoped to draw a parallel to the type of infection with which it is associated. The relative distributions of *Acinetobacter* spp. and *P. aeruginosa* amongst the various isolation sites and the two patient populations were similar (Table 2). This suggests that the role played by *Acinetobacter* spp. in causing hospital-acquired infections in this region may be as prominent as that of *P. aeruginosa*.

The prevalence of acinetobacter infections differed amongst patients in different wards. In the general ward, almost 60% of *Acinetobacter* isolates were from wound and urinary tract specimens. In the ICU, isolates were cultured mainly from lower respiratory tract specimens. It seems likely that the bacterial reservoir differed between the two populations, probably with skin and urinary tract catheters in the former, and ventilators in the latter. This difference may reflect the overwhelming number of patients requiring mechanical ventilation in the ICU. *Acinetobacter* spp. are associated with endotracheal intubation and ventilation, and are the predominant pathogens causing ventilator-associated pneumonia [23, 24].

The prevalence of acinetobacter-positive blood cultures in this locality was higher than published data in which these organisms accounted for <1% of total blood isolates [14, 25]. *Acinetobacter* spp. were the second most common micro-organisms isolated from bacteraemic ICU patients (Table 3). They were isolated about three times more frequently from ICU patients than from patients in the general ward. In

addition, these organisms were associated with the highest rate of polymicrobial bacteraemia (25.2%). Polymicrobial bacteraemia associated with *P. aeruginosa*, coagulase-negative staphylococci and *E. coli* represented 13.7–15.5% of total bacteraemic episodes. These figures were in agreement with those reported elsewhere [14, 21, 26–30]. The high polymicrobial bacteraemic rate of *Acinetobacter* spp. may reflect the potential polymicrobial sources of infection. The organisms isolated in conjunction with *Acinetobacter* spp. could be ectogenous or endogenous in origin (Table 4). Therefore, direct or indirect contact with skin or foreign devices, as well as trauma that damages membrane integrity, are possible sources of bacteraemia. Seifert and Baginski [31] isolated glucose-oxidising *A. baumannii* from blood and vascular catheter-tip cultures in 45% of 31 bacteraemic ICU patients. Similarly, catheter-associated bacteraemia by the non-glucose-oxidising *A. johnsonii* was documented in 13 patients [32]. Peacock *et al.* [33] reported that 27% of 22 ICU patients developed acinetobacter bacteraemia following the appearance of this organism in sputum.

It was of interest that, in terms of the body site- and ward-specific distribution, *Acinetobacter* spp. were similar to *P. aeruginosa*. However, in terms of the types of organism co-existing in blood specimens, *Acinetobacter* spp. were more comparable with coagulase-negative staphylococci. Yet unlike any of the reference organisms, *Acinetobacter* spp. existed with similar proportions of organisms that may have been acquired by the patient ectogenously or endogenously. Therefore the genus *Acinetobacter* was unique in that it had no dominant apparent portal of entry into the bloodstream.

As in previous reports, in-vitro resistance of glucose-oxidising strains of *Acinetobacter* spp. to multiple antibiotics was observed, with imipenem having the greatest likelihood of activity against these bacteria (Table 5). The antimicrobial susceptibility patterns of the glucose-oxidising and non-glucose-oxidising isolates were similar to those reported by Vila *et al.* [34] and Seifert *et al.* [35]. PCR with primers of a random sequence has been applied successfully to the molecular typing of *A. baumannii* [25, 36, 37]. When a simple tandem repeat, (GTG)₅, was used as an arbitrary primer, 19 distinct profiles were obtained (Fig. 2), and no major epidemiological relationship was detected amongst the isolates, although some clusters of isolates were obtained simultaneously from patients in the same ward. Hence the presence of a dominant clone circulating in this hospital was not detected.

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