Acinetobacter in the intensive care unit

TJJ Inglis • DM MRCPath DTM&H FRCPA
Medical Microbiologist
PathCentre, Western Australia

Abstract

Acinetobacter baumannii, *an increasingly common hospital pathogen, is notable for its ability to colonise and infect the more vulnerable among hospital patients. The species also has the capacity to acquire antibiotic resistance determinants and thus restrict antibiotic options. Survival and persistence on inanimate environmental surfaces assists its spread within the hospital.* A. baumannii *has caused several reported outbreaks in intensive care units, in several of which respiratory support equipment was implicated as a vehicle or reservoir. Aspects of ventilator circuit design provide a potential portal of entry to the patient's lower respiratory tract that* A. baumannii *is able to exploit. Recognition of these critical microbial entry points, particularly the temperature probe and its socket, may provide a means of curtailing* Acinetobacter *outbreaks in intensive care patients.*
[AIC Aust Infect Control 1999; 4(2):8-10]

Introduction

In recent years, *Acinetobacter baumannii* (formerly *calcoaceticus* var. *anitratus*) has become a common nosocomial pathogen in many hospitals and in some intensive care units (ICUs) and high-dependency units, has replaced *Pseudomonas aeruginosa* as the dominant antibiotic-resistant, Gram-negative isolate. Several time-space clusters of *Acinetobacter* infection have been reported from geographically separated ICU settings. In some, there was a substantial attributable mortality, adding to concerns about spread of the species. A knowledge of the hospital epidemiology of *Acinetobacter* can help direct outbreak investigations and attempts at control.

The genus *Acinetobacter*

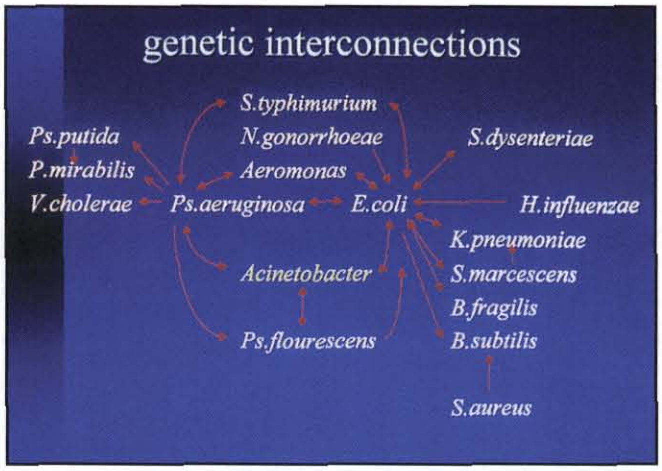
Recognition of the importance of *Acinetobacter* species as hospital pathogens has been hampered by changes of name throughout this century. Indeed, naming of the *Acinetobacters* was only recently settled upon after completion of detailed genetic homology studies. The list of species includes 17 genomo-species, of which *Acinetobacter baumannii* is the most important cause of hospital infection (Table 1). Since the majority of isolates belonging to this species were formerly *A. calcoaceticus* var. *anitratus*, older references to *A. calcoaceticus* var. *anitratus* probably refer to *Acinetobacter baumannii.*

A. baumannii has a remarkable ability to acquire resistance to a variety of commonly used, injectable antibacterial agents. In the last two decades, hospital isolates have become increasingly resistant to aminoglycosides and third-generation cephalosporins [1], with quinolone and even carbapenem resistance now being reported [2, 3]. It is clear one of the factors that contributes to the survival of this species in a hospital setting is its ability to act as an acceptor of antibiotic-resistance-conferring genes. Reference to a map of possible gene transfer routes between hospital pathogens shows how *Acinetobacter* lies at a genetic crossroads between a range of commonly encountered species (Figure 1).

Table 1. *List of* **Acinetobacter** *species.*

Strain	Name(s)
1	*Acinetobacter calcoaceticus*
2	*Acinetobacter baumannii*
3, 6 ,9-11, 13-17	*Acinetobacter spp.*
4	*Acinetobacter haemolyticus*
5	*Acinetobacter junii*
7	*Acinetobacter johnsonii*
8	*Acinetobacter lwoffii*
12	*Acinetobacter radioresistans*

Figure 1. ***Genetic interconnections: potential routes for the transfer of genes between bacterial pathogens.***



In addition to antibiotic resistance, *A. baumannii* has acquired an ability to survive on inanimate surfaces that is more like that of staphylococci than other Gram-negative bacilli [4]. *A. baumannii* has been shown to be more tolerant of drying, particularly when suspended in organic material [5].

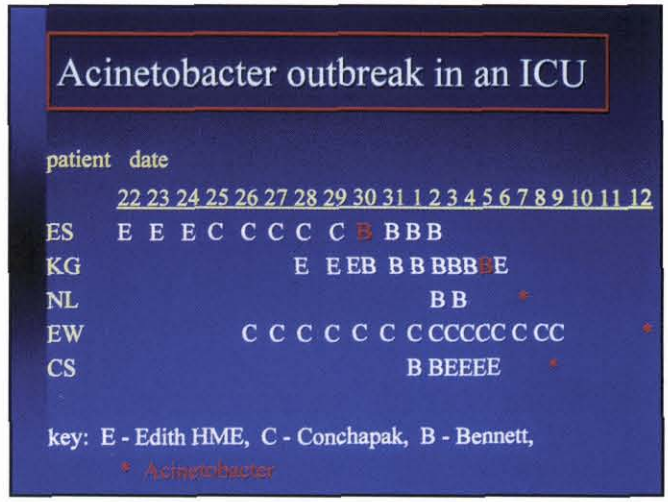
Epidemiology

A. baumannii infections are more likely in high-dependency or critically ill patients; in particular, those requiring mechanical ventilation [6]. Antibiotic therapy, especially with cephalosporins, has also been associated with *Acinetobacters* [7]. An interesting observation has been an association between *Acinetobacter* infection and warmer weather [8], but the significance of this is not known. For the most part, infections appear sporadic or endemic, but several time-space clusters have been well-documented. One involved contamination of a spirometer [9]. Another, which affected five ICUs, was linked to the respiratory therapy equipment, including the humidifier temperature probes [10]. However, in several documented outbreaks, no single point source has been identified and multiple contaminated environmental sites were found [1, 11].

Respiratory equipment

In late 1993, there was a small cluster of *Acinetobacter* infections in the ICU of a teaching hospital in the north of England (Figure 2) [12]. Conventional and molecular epidemiology established that contamination of the tip of a humidifier temperature probe had occurred. Use of the specific type of humidifier system requiring that probe was linked to *Acinetobacter* infection, with the plug-in port for the temperature probe in the ventilator tubing providing a point of access for bacteria. Moreover, the design of the temperature probe ensured that the manufacturer's recommended

Figure 2. ***Acinetobacter** outbreak in an English ICU: the key indicates the type of humidifier used. A red highlighted letter or asterisk indicates the date of first isolation of **Acinetobacter** spp. The isolate from patient EW was subsequently shown to belong to a different subtype than the other four.*

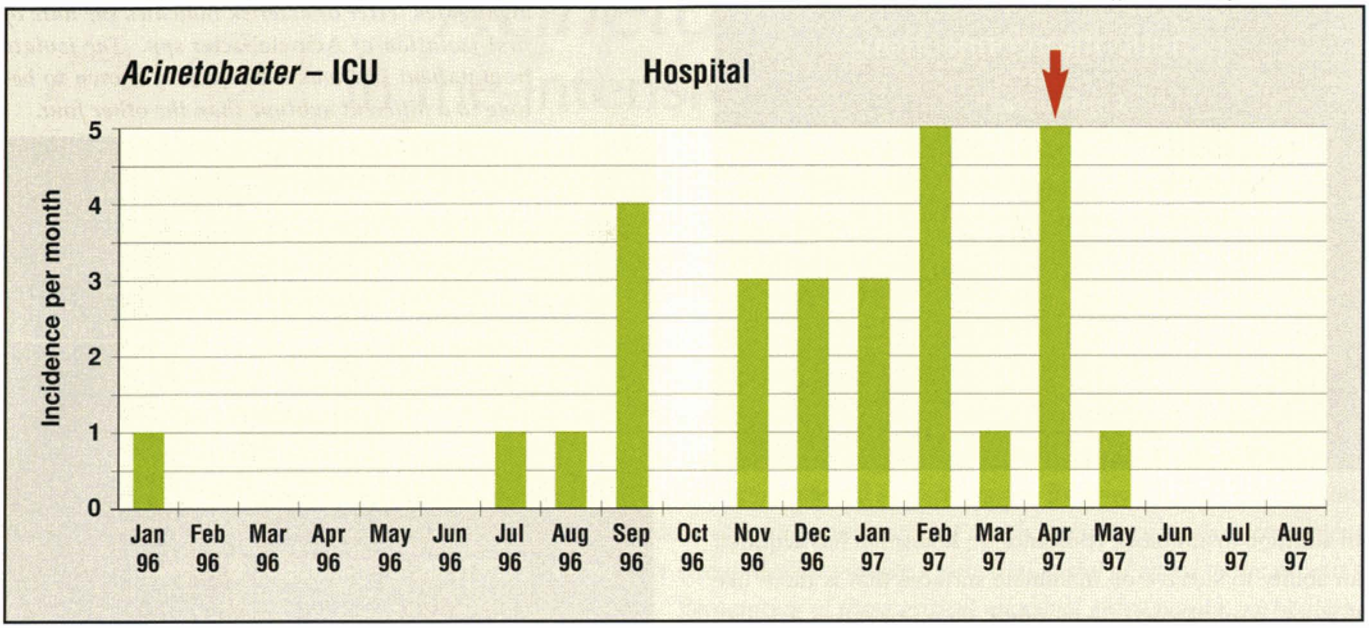


method of decontamination (wiping with isopropyl alcohol) could not be fully effective. A similar cluster of *Acinetobacter* infections occurred in a Western Australian ICU in 1997 and was brought to an end only after a visiting microbiologist had suggested pasteurising the temperature probes (Figure 3). However, this is not to say that all *A. baumannii* respiratory infections in ventilated patients can be explained by this one flaw in the design of widely-used equipment.

Other control measures

As most *Acinetobacter* infections are sporadic or endemic, a number of measures can be routinely employed to help restrict their spread within ICUs. Clearly, a high level of compliance with standard precautions should deal with spread on hands and clothing after close contact with patients and their body fluids. This will also help prevent contamination of surfaces, including charts near the patient's bed-space. Handling of respiratory equipment should take place with careful attention to hygiene principles, since *Acinetobacter* can persist for long periods on dry surfaces. Procedures likely to generate aerosol of respiratory secretions or ventilator-tubing condensate should be carried out with care; if necessary, a disposable drape can be used to prevent contamination of the external surfaces of the proximal respiratory circuit. Disposable tracheal suction catheters should be wound up during withdrawal and the glove peeled off to enclose the catheter. Medical staff should make every attempt to reduce their reliance on injectable cephalosporins in units where *A. baumannii* has become a problem and must also be careful to maintain a

Figure 3. **Acinetobacter** ***outbreak in a Western Australian ICU. The red arrow indicates the introduction of interventions suggested by a visiting microbiologist [data courtesy of the infection control officer at the affected hospital].***



high standard of hand hygiene between patients. Where the pathogen has got out of hand, infection control practitioners do need to think carefully about additional control measures. The ability of *Acinetobacter* species to exploit every weakness in the delivery of hospital inpatient care will test the imagination of infection control staff to the limits, and it is for this reason that genetic fingerprinting methods can help confirm suspicions.

Conclusion

A. baumannii has become an increasingly important nosocomial pathogen over the last decade. Resistance to antibiotics and an ability to survive on inanimate surfaces may have contributed to its prominence in some ICUs. Because of these factors, its challenge to hospital infection control programs is likely to increase in years to come.

References

1. Riley TV, Webb SA, Cadwallader H, Briggs BD, Christiansen L & Bowman RA. Outbreak of gentamicin-resistant *Acinetobacter baumanii* in an intensive care unit: clinical, epidemiological and microbiological features. Pathol 1996; **28**:359-63.
2. Cox TR, Roland WE & Dolan ME. Ventilator-related *Acinetobacter* outbreak in an intensive care unit. Mil Med 1998; **163**: 389-91.
3. Lyytikäinen O, Köljalg S, Härmä M & Vuopio-Varkila J. Outbreak caused by two multi-resistant *Acinetobacter baumannii* clones in a burns unit: emergence of resistance to imipenem. J Hosp Infect 1995; **31**:41-54.
4. Wendt C, Dietze B, Dietz E & Rüden H. Survival of *Acinetobacter baumannii* on dry surfaces. J Clin Microbiol 1997; **35**:1394-97.
5. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM & Hawkey PM. Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. J Clin Microbiol 1996; **34**:2881-87.
6. Seifert H, Strate A & Pulverer G. Nosocomial bacteremia due to *Acinetobacter baumannii*: clinical features, epidemiology, and predictors of mortality. Medicine 1995; **74**:340-09.
7. Courcol RJ, Pinkas M & Martin GR. A seven year survey of antibiotic susceptibility and its relationship with usage. J Antimicrob Chemother 1989; **23**:441-51.
8. Christie C, Mazon D, Hierholzer W Jr & Patterson JE. Molecular heterogeneity of *Acinetobacter baumanii* isolates during seasonal increase in prevalence. Infect Control Hosp Epidemiol 1995; **16**: 590-94.
9. Ahmed J, Brutus A, D'Amato RF & Glatt AE. *Acinetobacter calcoaceticus anitratus* outbreak in the intensive care unit traced to a peak flow meter. Am J Infect Control 1994; **22**:319-21.
10. Kaul R, Burt JA, Cork L, Dedier H, Garcia M, Kennedy C, Brunton J, Krajden M & Conly J. Investigation of a multi-year multiple critical care unit outbreak due to relatively drug-sensitive *Acinetobacter baumannii*: risk factors and attributable mortality. J Infect Dis 1996; **174**:1279-87.
11. Crowe M, Towner KJ & Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. J Med Microbiol 1995; **43**:55-62.
12. Snelling AM, Gerner-Smidt P, Hawkey PM, Heritage J, Parnell P, Porter C, Bodenham AR & Inglis T. Validation of use of whole-cell repetitive extragenic palindromic sequence-based PCR (REP-PCR) for typing strains belonging to the *Acinetobacter calcoaceticus-Acinetobacter baumannii* complex and application of the method to the investigation of a hospital outbreak. J Clin Microbiol 1996; **34**:1193-1202.