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Review

Acinetobacter baumannii: a universal threat to public health?

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Abstract

Acinetobacter spp. are non-fermentative, strictly aerobic, Gram-negative microorganisms with a confusing taxonomic history. The *Acinetobacter baumannii–Acinetobacter calcoaceticus* complex is the species most commonly isolated from clinical specimens. It is ubiquitous in nature and has been found as part of the normal skin, throat and rectal flora as well as in food and body lice. It colonises patients in Intensive Care Units and contaminates inanimate hospital surfaces and devices as well as wounds, including war injuries. Although a frequent coloniser, *Acinetobacter* can be the cause of severe and sometimes lethal infections, mostly of nosocomial origin, predominantly ventilator-associated pneumonia. Bacteraemic infections are rare but may evolve to septic shock. *Acinetobacter* also emerges as a cause of nosocomial outbreaks and is characterised by increasing antimicrobial multiresistance. Antibiotic use, especially carbapenems and third-generation cephalosporins, is recognised as the most important risk factor for multiresistance. Described resistance mechanisms include hydrolysis by β-lactamases, alterations in outer membrane proteins and penicillin-binding proteins, and increased activity of efflux pumps. Today, *Acinetobacter* resistant to carbapenems, aminoglycosides and fluoroquinolones presents a challenge to the clinician. However, sulbactam, tigecycline and colistin represent the current therapeutic approaches, which are associated with satisfactory efficacy.
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1. Introduction

Acinetobacter spp. have emerged in recent years as a major cause of nosocomial infections associated with significant morbidity and mortality [1,2]. Considered to be a commensal of low-grade pathogenicity, i.e. an opportunistic microorganism, *Acinetobacter* was frequently ignored in the 1970s whenever isolated from clinical specimens [3]. However, interest in *Acinetobacter* has grown rapidly in the last 20 years, a fact attributed to: (i) the worldwide expansion of Intensive Care Units (ICUs) that led to a change in the type of infections caused by *Acinetobacter*; and (ii) the emergence of multidrug-resistant (MDR) strains, some of which nowadays are pan-resistant to antibiotics with the exception of colistin [4–8]. Developing resistance patterns have prompted the suggestion that we are closer to the end of the antibiotic era with *Acinetobacter* than with meticillin-resistant *Staphylococcus aureus* (MRSA) [9].

This review focuses on the epidemiology and resistance surveillance of *Acinetobacter baumannii* as well as current mechanisms of resistance and their clinical impact. In addition, current strategies in therapeutic efforts and control of this universal threat to public health are reviewed.

2. Taxonomy

Acinetobacter spp. are non-fermentative, strictly aerobic, non-motile, non-pigmented, catalase-positive and oxidase-negative Gram-negative coccobacilli, usually occurring in diploid formation or in chains of variable length that grow on usual laboratory media [10]. The taxonomic history of *Acinetobacter* spp. appears confusing since they have been classified variously, moving from the family Neisseriaceae to the family Moraxellaceae under the names *Moraxella*, *Herellea*, *Mima*, *Achromobacter* and *Alcaligenes* [9,10]. Even today the situation is not clarified, undergoing continuous change. Studies based on DNA/DNA hybridisation have resulted in the description at least of 32 ‘genomic species’, 17 of which have been assigned species names [7]. Application of traditional methods of identification is in general

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not very satisfactory [11]. *Acinetobacter baumannii*, *Acinetobacter calcoaceticus* and the unnamed genospecies 3 and 13 sensu Tjernberg and Ursing (13TU) are difficult to distinguish phenotypically and therefore they are often given the name *A. baumannii–A. calcoaceticus* complex [1,7,11]. This group represents the strains of *Acinetobacter* most commonly associated with hospital-acquired infections, accounting for 75% of the *Acinetobacter* spp. isolated from clinical specimens [12].

3. Global epidemiology

Acinetobacter baumannii has been recovered from soil, water, animals and humans, being ubiquitous in nature [13]. *Acinetobacter* spp. are normal inhabitants of the human skin in the community but are also frequently isolated from the respiratory tract of hospitalised patients [14]. Throat carriage may occur in up to 10% of community residents with excessive alcohol consumption [15]. It has been suggested that human skin could be the source of severe *A. baumannii* infections such as bacteraemia [14]. A study by Berlau et al. [16] of 192 healthy volunteers revealed that 40% carried various *Acinetobacter* spp. (with *Acinetobacter lwoffii* predominating in 60%), while only 1 volunteer carried a strain of *A. baumannii*. In contrast, in recent studies *A. baumannii* was isolated in unsuspected sources such as food and arthropods [14]. In a study where the distribution and frequency of *Acinetobacter* genospecies in a variety of purchased or harvested fresh fruit and vegetables was investigated, *Acinetobacter* grew in 17%, with *A. baumannii* complex accounting for 56% of all strains isolated from apple, melon, bean, cabbage, cauliflower, carrot, potato, radish, lettuce, cucumber, pepper, mushroom and corn [17]. Therefore, according to the latter study, hospital food could be a potential source for *A. baumannii* acquisition and subsequent colonisation of the digestive tract of hospitalised patients. However, *Acinetobacter* colonisation rates of almost 41% in ICU patients contradict vegetables and fruit as an important vector [18]. Nevertheless, it is of interest that *A. baumannii* strains were isolated from body lice collected from homeless persons in Marseille (France), indicating that epidemic *A. baumannii* infections among human body lice could be a source of human infection [19]. Recently, *A. baumannii* infections have been identified in patients with traumatic injuries, suggesting environmental wound contamination [2]. It is of interest that for almost 3 years (1 January 2002 to 31 August 2004) *A. baumannii* was isolated from blood cultures obtained from 102 patients hospitalised at military medical facilities treating service members injured in Afghanistan and the Iraq–Kuwait region [2,14,20]. During the Vietnam war, *A. baumannii* was the most common Gram-negative bacillus recovered from traumatic injuries of the extremities [14]. In addition, MDR *Acinetobacter* ventilator-associated pneumonia (VAP) was described in 2006 in critically injured Canadian forces soldiers who returned from Afghanistan [21]. However, in both cases the sources of the pathogen are still unknown, only suggesting prolonged environmental contamination since *Acinetobacter* spp. can survive both in moist and dried environments [22,23].

Inanimate hospital sources have been found to be colonised with dried *Acinetobacter* spp. for prolonged periods up to 5 months [9,14]. The most common sources have been ventilators, suctioning equipment, mattresses, pillows, humidifiers, bed rails, bedsides, containers of distilled water, urine collection jugs, intravenous (i.v.) nutrition equipment, potable water, reusable arterial pressure transducers, the knobs of electrocardiographs, wash basins, infusion pumps, sinks, hygroscopic bandages, showers, stainless-steel trolleys, resuscitation equipment and tables, i.v. access devices, portable radiology equipments, bed linen, soap dispensers, spinometers, temperature probes and multidose nebulisers [13].

Acinetobacter baumannii is distinguished by its propensity to cause outbreaks, which are probably related to its multidrug resistance patterns as well as to its resistance to desiccation [14]. There have been many reports of *Acinetobacter* spp. surviving on abiotic surfaces such as hospital bed rails [22] as well as persisting as contaminants of the hands of healthcare workers [9]. *Acinetobacter lwoffii*, *Acinetobacter johnsonii* and *Acinetobacter radioresistens* are usually the most frequently isolated species. However, in ICU outbreaks of *Acinetobacter* infections, epidemic *A. baumannii* strains have been isolated from patients' skin and rectal samples [24]. Locations of healthcare outbreaks caused by *A. baumannii* include the neonatal ICU, burn unit, neurosurgery unit, surgical unit, internal medicine and oncology unit, whereas until 2004 adult ICUs predominated in almost 60% of reports in the English literature [14]. *Acinetobacter baumannii* is also implicated in multifacility outbreaks described in France and the USA, very probably as a result of the transfer of colonised and infected patients from one facility to another [14].

Recently, an outbreak of MDR *Acinetobacter* spp. infection associated with moist-site contamination occurred during pulsatile lavage wound treatment, a high-pressure irrigation treatment used to debride wounds [25]. In one of the most strange recent *Acinetobacter* outbreaks, the source of the microorganism appeared to be the curtains surrounding the patients' beds [26]. Therefore, environmental decontamination with hypochlorite solutions has been reported as being of great importance in controlling such outbreaks [27].

Three major epidemic European clones have been recognised. Clones I and II were responsible for outbreaks in hospitals of countries of northwestern Europe. Clone I has also been recovered from Spain, South Africa, the Czech Republic, Poland and Italy, whereas clone II has been recovered from Spain, Portugal, South Africa, France, Greece and Turkey. Clone III was identified in France, Italy, Spain and The Netherlands. The latter data suggest that these clones are very fit, being virulent and MDR, causing outbreaks that are difficult to control and thus establishing endemicity in hospitals [28].

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