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Editorial

Acinetobacter Species as Nosocomial Pathogens

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Acinetobacter is a gram-negative coccobacillus that is strictly aerobic, nonmotile, catalase positive, and oxidase negative. It is widespread in nature, being found in soil and water. *Acinetobacter*, like other nonfermentative gram-negative bacteria, has been recognized as a nosocomial pathogen since the 1970s. The introduction of new broad-spectrum antibiotics in hospitals has been one of the main factors responsible for this development.

The key features of nosocomial infections with *Acinetobacter* spp. have been described as the propensity of *Acinetobacter* to affect severely ill patients, especially those on artificial ventilation, the limited therapeutic options due to multiple-antibiotic resistance, and the contamination of the patients' environment, creating a potential reservoir for patient-to-patient transmission [1].

Taxonomy of *Acinetobacter* and Clinically Important Species

The taxonomy of *Acinetobacter* spp. has changed frequently. Up to now 21 genomic species have been described [2]. Some of the species have formal names: *A. baumannii*, *A. johnsonii*, *A. calcoaceticus*, *A. haemolyticus*, *A. junii*, *A. Iwoffii*, and *A. radioresistens*. There is a close relationship between the genomic species 1 (*A. calcoaceticus*), 2 (*A. baumannii*), 3, and 13 TU, which are sometimes named as the *A. calcoaceticus-A.baumannii complex*, corresponding quite well to the species previously named as *A. calcoaceticus* var. *anitratus* [3].

For typing methods the reader is referred to the *Acinetobacter* review of Bergogne-Bérézin and Towner [3] and the study of Ehrenstein et al. [2]. Clinically, the most important species is *A. baumannii*. In one study that included 584 clinical *Acinetobacter* isolates, 72.9% of the isolates were identified as *A. baumannii* [4]. Of these *A. baumannii* strains, 48.8% were isolated from respiratory tract specimens, 26.5% from blood cultures and from central venous lines, 16.4% from wound swabs, and 8.3% from other sites. The remaining 27.1% of the strains isolated belonged to other species, of which the most frequent were *Acinetobacter* genomic species 3 (9.4%), *A. johnsonii* (5.0%) and *A. Iwoffi* (3.6%).

In general, identification rates of *A. baumannii* must be interpreted cautiously, especially those in older reports, because phenotypic identification methods for *Acinetobacter* spp. are not totally reliable. Identification of the clinically important species 2 (*A. baumannii*), 3, and 13 TU is particularly difficult [3].

Nosocomial Infections due to *Acinetobacter* Species

The most common site of nosocomial infections is the respiratory tract, especially in ventilated patients, followed by the bloodstream. Less frequently, the urinary tract, surgical wounds, the central nervous system, the peritoneum, the skin, and the eye are involved.

According to data from the National Nosocomial Infections Surveillance (NNIS) system from 1990–1992, *Acinetobacter* spp. were isolated in 1% of all nosocomial infections [5]. The true frequency of nosocomial infection caused by *Acinetobacter* spp. is difficult to assess because isolation of *Acinetobacter* spp. in clinical specimens may reflect colonization rather than infection.

Acinetobacter spp. were isolated in 4% of all cases of nosocomial pneumonia in NNIS hospitals [5]. In mechanically ventilated patients, however, *Acinetobacter* occurs more frequently. *Acinetobacter*, like *Pseudomonas aeruginosa* and *Stenotrophomonas maltophilia*, is a typical pathogen in late-onset ventilator-associated pneumonia [6]. According to studies in which diagnosis was supported by analysis of protected brush specimens, *Acinetobacter* spp. are involved in 4–26% of ventilator-associated pneumonia cases [6–11]. *Acinetobac-*

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ter is thus one of the leading pathogens that cause ventilator-associated pneumonia, the prognosis of which may be as poor as that of ventilator-associated pneumonia caused by *Pseudomonas* spp. In one study by Fagon et al. [12], mortality associated with *Acinetobacter* or *Pseudomonas* pneumonia in ventilated patients was 71%, with an attributable mortality of 43%. *A. baumannii* is also one of the main causative agents of sinusitis in mechanically ventilated patients [13], a condition that is associated with an increased risk of pneumonia in these patients.

According to NNIS data, *Acinetobacter* spp. are found in 2% of all nosocomial bloodstream infections [5]. *Acinetobacter baumannii* bacteremia is usually acquired in the intensive care unit (ICU). Mortality rates in patients with *Acinetobacter baumannii* bacteremia range from 17%–52% [14, 15]. In two larger recent studies, the mortality rate among patients with *A. baumannii* bacteremia was 44% and 52%, respectively [14, 15]. In the study of Cisneros et al. [15], death was related to the bacteremia in 65% of the patients who died.

Septic shock is a common complication in these patients, occurring in 25–30% of patients. In the two studies mentioned above [14, 15], polymicrobial bacteremia was observed in 35% and 19% of patients, respectively. Multivariate logistic regression analysis in the study of Seifert et al. [14] revealed the following independent predictors of mortality: rapidly or ultimately fatal disease, septic shock, and mechanical ventilation. Cisneros et al. [15] identified disseminated intravascular coagulation and inappropriate antimicrobial treatment as independent risk factors for mortality in patients with *Acinetobacter* bacteremia.

The major portals of entry of *Acinetobacter* into the bloodstream are the respiratory tract and vascular devices. Other less frequently involved portals of entry are the urinary tract, the abdomen, the skin and soft tissue, the central nervous system, and burn wounds [14]. Multiple sources of bacteremia are possible, but the source frequently cannot be determined. Intestinal translocation as a possible source in these cases has been discussed [15].

Patients with *A. baumannii* bacteremia associated with intravascular catheters seem to have a good prognosis, probably because the source of infection can be eliminated by removing the catheter. The mortality among 72 patients with epidemic *Acinetobacter baumannii* bacteremia associated with contaminated pressure transducers used for arterial catheters was not significantly higher than that among controls [16]. In the study of Seifert et al. [14], catheter-related infection was also associated with an increased chance of survival, whereas all eight patients with pneumonia as the primary site of infection died. Moreover, mortality in preterm neonates [14, 17, 18] and in children with malignancies [19] was found to be lower than that in the general population. In two of these studies, the majority of bacteremic episodes were catheter-related [14, 19]. In catheter-related bloodstream infections due to *Acinetobacter johnsonii,* a similarly good outcome has been described [20].

Secondary meningitis is the most frequent type of *Acinetobacter* meningitis, the mortality rate being around 25%. Risk factors include the presence of ventriculostomies, cerebrospinal fluid fistulas, indwelling ventricular catheters (especially if present more than 5 days), and overuse of antimicrobial agents in neurosurgical ICUs [3, 21].

Acinetobacter spp. are isolated in 1% of the cases of nosocomial urinary tract infections and surgical site infections [5]. Moreover, *Acinetobacter* can be a cause of peritonitis in patients undergoing continuous ambulatory peritoneal dialysis and of choloangitis and sepsis following percutaneous transhepatic cholangiography or biliary drainage [22]. Other infections, such as ophthalmic infections and osteomyelitis, seem to be very rare [3]. Severe underlying diseases, extended ICU stay, prolonged respiratory therapy, and previous antibiotic therapy have been identified as predisposing risk factors for the acquisition of resistant *Acinetobacter* spp. in the ICU [23, 24].

Epidemiology of *Acinetobacter* Species

In 1979 the Centers for Disease Control described a seasonal pattern of *Acinetobacter* infections with an infection rate that was twice as high in late summer as in the early winter for each year in the study period from 1974 to 1977 [25]. This pattern was seen in all services and all major infection sites for *A. calcoaceticus* var. *anitratus*. Temperature and humidity changes were discussed as the possible reasons for this finding [3, 21, 26].

Acinetobacter spp. are present on the skin of at least one-fourth of healthy male individuals [27], particularly in moist regions such as the axillae, the groin, and toe webs [3]. Oropharyngeal or rectal carriage is rare. In a recent study that included more than 300 patients admitted to a surgical ICU, less than 4% of the patients were colonized with *A. baumannii* on admission to the unit when screening cultures of stool samples, nasal and pharyngeal swabs, tracheal secretions, and urine were performed [28]. *Acinetobacter* spp. were found on the hands of 19% of hospital personnel and may be regarded as a part of the nontransient flora [29]. Nontransient *Acinetobacter* and other gram-negative bacilli on the hands of hospital personnel are thus a potential reservoir of hospital strains. Patient contact has not been found to be an obvious source for the acquisition of nontransient gram-negative bacilli [29].

Since *Acinetobacter* is a ubiquitous organism, it is not surprising that *Acinetobacter* is also frequently present in the hospital environment. In one study, *Acinetobacter* spp. were recovered from approximately 25% of sink traps and from roughly 25% of hospital floor swab cultures [30].

In outbreak situations colonization rates of the respiratory tract [28, 31], the pharynx [28, 32], the skin [32], the urinary tract [33, 34], and the gastrointestinal tract of patients may be high, depending on the specific features of the outbreak [3]. Besides the respiratory tract, the digestive tract is one of the major sites of *Acinetobacter* colonization in outbreak situations [17, 32, 35]. Almost all patients involved in an outbreak may be colonized at multiple sites [28].

In one study the epidemic *Acinetobacter* strain was isolated from 29% of the hands of hospital personnel by using the handwashing technique [34]. In another study reporting high carriage rates among staff members (33%), multiple strains were isolated [36]. Hand carriage in outbreaks was reported to be transient [34, 36], except in one pulmonary therapist with chronic dermatitis [36] and in two microbiologists investigating the reported outbreak [34]. Transmission of an epidemic strain from patients' skin to staff members' hands was demonstrated experimentally [8]. In studies using less sensitive methods such as streaking fingers across the surface of an agar plate or culturing swabs from hands, and, in studies without description of the sampling technique, the isolation rate of epidemic *Acinetobacter* strains from the hands of staff members was in the range of 0–15% [17, 23, 26, 32]. Nose or throat carriage among staff members is uncommon. It should be noted, however, that precise typing methods were not established in these older studies, thus high rates of hand colonization may be misleading without typing the isolates to determine whether isolates from the staff, the inanimate environment, and patients belong to the same genotype [31]. In two recent studies, typing methods demonstrated hand carriage of an epidemic *A. baumannii* strain in 4% and 11% of staff members [31], respectively [37]. In the study of Go et al. [31], a decline in the incidence of hand carriage occurred after reinforcement of strict hand washing procedures [14].

As in outbreaks with other nosocomial pathogens, cross-contamination of *Acinetobacter* via hands of staff members was found to be the main or most likely source of an outbreak in many studies [26, 31–34, 36]. A considerable number of outbreaks with *Acinetobacter* due to contaminated medical equipment and materials have been reported. The majority of published outbreaks have described *Acinetobacter* respiratory tract infections associated with contaminated respiratory equipment [38–41]. In one outbreak respiratory infection and colonization of ICU patients was attributed to contaminated, reusable ventilator circuits and resuscitation bags that were only pasteurized between patients [39]. The outbreak was controlled when terminal ethylene oxide sterilization of the equipment after sterilization was instituted. Another outbreak was associated with contaminated disinfected temperature probes and ventilator circuits. Multifaceted control measures, including sterilization of temperature probes, terminated the outbreak [42].

One outbreak of respiratory tract infections could be traced to a pulmonary therapist with chronic dermatitis who had persistent hand colonization with the epidemic strain and contaminated respiratory therapy equipment [36]. One small outbreak of *Acinetobacter* pneumonia in an ICU was associated with contaminated gloves used without routine changing between patients [43]. The review of Wendt and Herwaldt [44] provides a complete survey of reported *Acinetobacter* outbreaks.

Allen and Green [32] investigated the possibility of an airborne mode of spread in an outbreak of multiresistant *Acinetobacter anitratus* in three neurosurgical wards, one medical ward, and the ICU of a general hospital. Sputum was the most frequent source of *Acinetobacter anitratus* from colonized or infected patients, the majority of whom were intubated or had tracheostomies. During the outbreak, settle plates placed within 3 m of three colonized patients (all colonized in multiple skin sites, one patient with a colonized respiratory tract was ventilated) were positive for *Acinetobacter anitratus*. The authors concluded that airborne *Acinetobacter* can produce extensive environmental contamination. However, "hand-carriage from contact with infected patients was the most likely mode of transmission, although staff hand contamination from inanimate objects... may have been an important factor" [32].

Transmission of *Acinetobacter* strains between patients is facilitated by the propensity of *Acinetobacter* to persist for a considerable time on the hands of personnel and in the inanimate environment, the latter which serves as a reservoir for continuous contamination of hands. In outbreak studies using typing methods, epidemic *Acinetobacter* strains were found in variable quantities: from about 3% [31, 44] to 18% [45] of environmental samples. After experimental contamination of fingers, *A. calcoaceticus* var. *anitratus* survived 60 min; longer survival times were not examined [46]. On dry Formica surfaces, *A. calcoaceticus* survived up to 13 days compared to other gram-negative bacteria, which survived only up to 3 days [47].

In a recently published study, Wendt et al. [48] could demonstrate that the ability of strains of *A. baumannii* to survive under dry conditions varies greatly. Some strains were able to survive for several months (more than 4) without any reduction in colony count, whereas other strains hardly survived the process of drying. Surprisingly, this feature of the strain correlated well with

the source from which the strain was isolated. Strains isolated from wet sources did not survive as well as those isolated from dry sources [48]. The reasons for this difference are unclear.

Conclusion

In summary, *Acinetobacter* colonization or infection may originate from the patients' own flora under the pressure of antimicrobial selection, the hands of staff members, or contaminated equipment. Transmission of *Acinetobacter* strains between patients occurs primarily via the hands of health care workers. In outbreak situations, colonized or infected patients and the inanimate environment, which can be secondarily contaminated, are the main reservoirs in the hospital setting for cross-transmission. However, colonized or infected patients seem to be the most important source of cross-contamination, as epidemic strains spread easily throughout different wards. Especially in prolonged outbreaks in which control efforts such as proper hand washing, glove changing, and restriction of antimicrobial agents are ineffective and specific sources such as contaminated equipment are not identified, the source of the epidemic strain is likely the patients' inanimate dry environment [45, 48].

In outbreak situations it is necessary that isolated *Acinetobacter* strains are identified to the genomic species level and then typed before epidemiological conclusions can be drawn, because *Acinetobacter* spp. are ubiquitous organisms [3, 31].

Note added in proof

A new study by Seifert et al. has recently made an important contribution to the epidemiology of *Acinetobacter* spp. Seifert et al. examined the distribution of *Acinetobacter* spp. on the skin and mucous membranes of 40 patients hospitalized in a cardiology ward and of 40 healthy controls using phenotypic and genotypic identification methods. They found 75% of patients and 42.5% of the healthy controls to be colonized with *Acinetobacter* spp. *A. lwoffii* (47%), *A. johnsonii* (21%), *A. radioresistens* (12%) and DNA group 3 (11%) were the most frequently isolated species. However, the clinically most important species, *A. baumannii* DNA group 13 TU, were found on the skin in only 0.5% and 1% of the patients respectively. This means, as the authors point out, that the natural habitat of these species remains to be defined.

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