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Article

Virulence Determinants and Antimicrobial Susceptibility Profiles of Clinical Acinetobacter baumannii Isolates

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Abstract: Acinetobacter baumannii is an opportunistic pathogen that causes mainly nosocomial infections and/or a global threat due to its ability to acquire resistance against almost all known antimicrobial agents . Antimicrobial susceptibility pattern and virulence attributes of clinical A. baumanniiIsolates were retrieved from different types of hospital specimens.. Methods A combination of 60 non-duplicated clinical isolates from wound swabs, sputum, urine, blood, endotracheal aspirates and burn exudates were collected. Identification of isolates was performed using standard microbiological procedures and an automated identification system. The disk diffusion method was used for antimicrobial susceptibility testing and specific minimum inhibitory concentrations were confirmed by broth microdilution. Polymerase chain reaction was used to monitored virulence genes while phenotypic sterol, motility and hemolysin production were tested on bacterial strains.

Keywords: Acinetobacter baumannii, Antimicrobial resistance, Virulence factors, Biofilm formation, Clinical isolates

Introduction

Due to its exceptional persistence in hospital environments, ability to colonize susceptible patients and rapid acquisition of multi-drug resistance, Acinetobacter baumannii has become one of the most problematic opportunistic pathogens encountered in modern clinical practice. It has now emerged as a significant contributor to healthcare associated infections, especially in the settings of intensive care units where it is often implicated in ventilator associated pneumonia, bloodstream infection, urinary tract infection, wound infection and post-surgical complications [1], [2]. The clinical significance of these infections is further highlighted by their associated high morbidity and mortality, particularly in critically ill, immunocompromised, mechanically ventilated or long-term hospitalized patients [3], [4]. High resistance rates in Gram-negative organisms, particularly from ICU isolates emphasise the need for integrated stewardship, infection control and surveillance strategies. A. baumannii is more than just an antimicrobial-resistant nosocomial pathogen [5]. Instead, pathogenicity is the result of a delicate balance between resistance mechanisms and an impressive arsenal of virulence factors that promote survival, colonization, evasion of innate immune response (IIR), persistence in a host and tissue damage. Research conducted recently showed that A. baumannii has a number of virulence factors that are cell-associated and/or secreted, including outer membrane proteins, capsular

polysaccharides, lipooligosaccharide, phospholipases, pili, quorum-sensing systems, iron-acquisition systems, secretion systems as well as biofilm-associated proteins [1], [2], [4]. These factors allow the bacterium to survive hostile environments in both host and hospital surroundings including desiccation, oxidative stress, nutrient-limited states, exposure to disinfectants and pressure from antibiotics [2], [3].

Among the well characterized virulence determinants, outer membrane protein A (OmpA) plays a central role during adherence of epithelial cells, biofilm formation, invasion and in induction of host-cell apoptosis [6]. Besides, other Omp associated proteins like Omp33-36, CarO and OmpW have also been implicated in adhesion, cytotoxicity, nutrient uptake and alteration of the antimicrobial susceptibility [1], [4]. In pathogenicity, polysaccharides at the cell surface also play a major role [7], [8]. Capsule protects A. baumannii from complement-mediated killing, phagocytosis, desiccation and some antimicrobial agents while lipooligosaccharide contributes to immune activation, membrane stability and survival under environmental stress [1], [2], [9]. Different capsule compositions have also been correlated with virulence differences among clinical strains [2], [9].

Biofilm formation is one of the other key features that cause A. baumannii to persist in the clinical environment. Biofilms enable attachment to both abiotic and biotic surfaces, such as in catheters, ventilator tubing, and epithelial tissues. They afford significant protection against antibiotics, disinfectants, or host immune defenses [10], [11]. For instance, Csu pili, Bap and Bap-like proteins [12], poly-N-acetylglucosamine production, quorum-sensing regulators including AbaI/AbaR (Table 1) and two-component regulatory systems such as BfmRS [2]–[4]. This phenotype is associates with chronic colonization, recurrent infection and therapeutic failure because biofilm-forming isolates are easily to be eradicated. Moreover, the pathogenic traits help to extremely potentiate the virulence potential of this microorganism [13]. Phospholipases and proteases mediate membrane disruption, tissue invasion and host-cell injury whereas acinetobactin-mediated iron acquisition systems help the organism to grow under growth conditions imposed by iron-deprived environment presented by the host. Emergence of protein secretion systems—type I, II, V and VI—as major bacterial adhesion–mediated colonization factors that contribute to an increased capacity for nutrient exploitation, interbacterial competition [1]–[4], and virulence during infection have been the topic of focused studies in recent years (Shadan et al., in add, outer membrane vesicles are also involved in cyto toxicity, inflammatory stimulation with potentially horizontal transfer from resistance-associated material [93], emphasizing the multifactorial manner in which A. baumannii pathogenesis takes place [1], [2].

Concurrently with its virulence armamentarium, A. baumannii has acquired a worrisome level of antibiotic resistance [14]. Clinical isolates many times are multidrug-resistant, extensively drug-resistant and sometimes even pandrug-resistant, dramatically reducing the options for treatment. Mechanisms of resistance in Klebsiella pneumoniae such as production of beta-lactamases including OXA-type carbapenemases, overexpression of efflux pumps, decreased outer membrane permeability, aminoglycoside-modifying enzymes, alteration of target site and adaptive stress responses [1], [3], [4]. Extensive data is now available from systematic reviews; with continued high levels of carbapenem resistance, however, much higher rates are seen in some regions than others and although colistin still appears active against a large proportion of isolates, recently acquired resistance patterns to last-resort agents has been extensively reported [14], [15]. It is especially troubling that virulence determinants and antimicrobial resistance coexist because they could cooperate in a clinical setting. Production of biofilm, expression of capsule, outer membrane remodelling and efflux activity can possibly promote survival while simultaneously reducing susceptibility to antibiotics thus supporting the persistence in a hospital environment and dissemination. In addition, dominant epidemic lineages frequently co-associate strong biofilm-forming ability, desiccation tolerance with broad resistance profiles helping to explain the long-term success of this pathogen in outbreaks and endemic hospital transmission [2], [16]. Therefore, the entire characterization of virulence factors and antimicrobial susceptibility patterns in clinical isolates is necessary for profiling local epidemiology together with effective therapy and prediction of infection-control measures.

Considering the burden that multidrug-resistant A. baumannii imposes globally, studies investigating the association between virulence mechanisms and antimicrobial resistance are especially important. The study of these traits in clinical isolates will enable greater understanding of their role in

pathogenicity, persistence and treatment outcome, as well as generate locally relevant data for surveillance and improve antimicrobial stewardship. Hence, the aim of the current study was to investigate virulence factors and antimicrobial susceptibility patterns among clinical isolates of A. baumannii in order to gain better insight into its pathobiology and improve management strategies against this resilient pathogen.

Materials and Methods

Study design and setting

A laboratory-based cross-sectional study was developed to check the virulence factors and or antimicrobial susceptibility patterns of clinical Acinetobacter baumannii isolates. Design: An interventional study conducted in the microbiology laboratory of a tertiary care teaching hospital and university research laboratory over 8months.. After approval from the relevant institutional authorities, clinical specimens were collected from hospitalized patients with suspected bacterial infections. This study used only one isolate per patient to avoid duplication and for the microbiological results to be reliable.

Collection of clinical specimens

A total of 60 unique clinical specimens were collected from a variety of sources including wound swabs, sputum, urine, blood, endotracheal aspirates and burn exudates. Specimens were collected from patients admitted to the emergency Department and required admission other departments including intensive care units (ICU), surgical wards, internal medicine wards, or burn units. All samples were taken IMMEDIATELY to the laboratory in aseptic conditions and processed within 02 hours of collection. Isolation was performed using standard microbiological protocols.

Isolation & Identification of Bacterial Isolates

Methods Data Collection Clinical samples were inoculated onto MacConkey agar, blood agar and CHROMagar (where applicable) then incubated aerobically for 18–24 hours at 37°C. Routine testing suspected colonies of Acinetobacter species were recognized by characterizing epidermis and morphology, Gram's discoloration, oxidase negativity, catalase positivity, non-lactose fermentation on MacConkey agar and motility

. VITEK 2 compact system was used to confirm the final identification of A. baumannii. Isolates that tested positive were held at −20°C in tryptic soy broth with glycerol for further phenotypic and molecular characterization.

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed via the Kirby–Bauer disk diffusion method with Mueller–Hinton agar. An overnight culture was prepared, and a fresh bacterial suspension approx 0.5 McFarland standard was obtained from it, then evenly inoculated onto the agar surface with sterile swabs. Antibiotic disks widely used for treatment of Gram-negative infections were utilized including amikacin, gentamicin, ciprofloxacin, levofloxacin, ceftazidime, cefepime, piperacillin–tazobactam in addition to imipenem and meropenem (for Enterobacterales), trimethoprim–sulfamethoxazole. tetracycline as well as colistin where applicable. Plates were incubated at 37°C for 18–24 hr and the diameters of the inhibition zones were measured in millimeters. The results were interpreted based on current CLSI criteria. Multidrug-resistant isolates were defined as those resistant to at least one agent in three or more antimicrobial classes.

To obtain more reliable results for selected last-line agents, the minimum inhibitory concentrations (MICs) of imipenem, meropenem, and colistin were additionally determined for a subset of resistant isolates using the broth microdilution method. Escherichia coli ATCC 25922 and Pseudomonas aeruginosa ATCC 27853 were used as quality control strains throughout susceptibility testing.

Phenotypic detection of virulence factors

Microtiter plate assay for biofilm formation. In brief, overnight cultures in tryptic soy broth with 1% glucose (ThermoFisher Scientific) were diluted and inoculated into sterile 96-well flat-bottom microplates and incubated at 37°C for 24 hours. The wells were washed with phosphate-buffered saline (PBS), fixed with methanol, stained with crystal violet, and the absorbance was determined at 570 nm using a microplate reader. The isolates were classified as non-biofilm

producers, weak, moderate or strong biofilm producers according to the optical density values. Intracellular chemical invasion and intracellular competitive growth were assessed. Fresh overnight culture diluted in 10%LB was inoculated at the center of the plate, followed by incubation for 24h at 37°C. Bacterial spreading was measured in millimeters, and isolates were classified as non-motile, moderately motile, or highly motile .

Hemolytic activity was determined by streaking the isolates onto 5% sheep blood agar and incubating them aerobically at 37°C for 24–48 hours. The presence of a clear or greenish zone surrounding the colonies was recorded as beta- or alpha-hemolysis, respectively, while the absence of a hemolytic zone was considered negative. Protease production was examined on skim milk agar by observing clear zones around the colonies after incubation.

Molecular detection of selected virulence genes

DNA extraction Genomic DNA was extracted from fresh bacteria-colonies using a commercial kit for extraction according to the manufacturer instructions. To detect some virulence-associated genes (ompA, bap, plcN and lasB), conventional polymerase chain reaction (PCR) was carried out. PCR was performed in 25 μL of reaction mixture containing template DNA, forward and reverse primers, PCR master mix and nuclease-free water. Amplifications were carried out in a thermal cycler using optimized cycling conditions for each primer set. Electrophoresis was performed on a 1.5% agarose gel (previously stained with ethidium bromide) and the bands were visualized under ultraviolet light after electrophoresis of amplified products. The absence or presence of each gene was documented according to the predicted band size.

Results

A total of 60 biofilm-forming clinical isolates were obtained from various hospitals specimens where Acinetobacter baumannii was detected. The most common source of isolation was wound swabs (33.4%), followed by sputum (32.1%), urine specimens (24.5%) and blood specimen (10%). The importance of this pathogen in hospital-acquired severe infections is reflected in the fact that most (72%) of the isolates studied were recovered from patients hospitalized for intensive care unit admission. Discovery via aptitude testing of, in the main, antibiotics indicated admirable resistance to maximum ordinary medicines amongst the isolates. The highest resistance rates were seen towards ceftazidime, ciprofloxacin, levofloxacin and piperacillin–tazobactam. Gentamicin and amikacin showed moderate resistance and carbapenem resistance occurred in a substantial number of isolates. Colistin exhibited the lowest resistance rate and continued to be most active agent against most of isolates.

Our phenotypic analysis of virulence factors demonstrated that biofilm formation was very commonly distributed among the isolates. Many of the isolates were high biofilm producers and only few non or weak biofilm producers. Most of the isolates also demonstrated motility on semi-solid agar and hemolytic activity was identified in many of them. This data reflects that the isolates possessed several pathogenic characteristics which might aid in being more persistant and prevent past its time in the hospital environment. In molecular screening, selected virulence genes were detected at different frequencies. Biofilm, outer membrane related genes were the most and phospholipase- and elastase-related genes were the least detected. Multi-virulence gene carrying isolates was mostly associated with increased biofilm forming ability and improved resistance of a few antibiotics. This indicates possible correlation of virulence potential and antimicrobial resistance. Conclusions: The clinical isolates of A. baumannii were multidrug-resistance; they produced robust biofilm and possessed multiple virulence determinants. These results reinforce the notion that this organism serves as a serious nosocomial pathogen and has both therapeutic and epidemiological importance.

Table 1. Distribution of clinical isolates according to specimen source and hospital ward

Source / Ward	No. of isolates	Percentage (%)
Wound swab	18	30
Sputum	14	23.3

Source / Ward	No. of isolates	Percentage (%)
Urine	10	16.7
Blood	8	13.3
Endotracheal aspirate	6	10
Burn exudate	4	6.7
ICU	28	46.7
Surgical wards	14	23.3
Internal medicine	10	16.7
Burn unit	8	13.3

Table 2. Antimicrobial susceptibility pattern of clinical A. baumannii isolates

Antibiotic	Resistant n (%)	Intermediate n (%)	Sensitive n (%)
Ceftazidime	53 (88.3)	4 (6.7)	3 (5.0)
Ciprofloxacin	50 (83.3)	5 (8.3)	5 (8.3)
Levofloxacin	48 (80.0)	6 (10.0)	6 (10.0)
Piperacillin–tazobactam	46 (76.7)	7 (11.7)	7 (11.7)
Gentamicin	38 (63.3)	9 (15.0)	13 (21.7)
Amikacin	35 (58.3)	10 (16.7)	15 (25.0)
Imipenem	42 (70.0)	7 (11.7)	11 (18.3)
Meropenem	40 (66.7)	8 (13.3)	12 (20.0)
Trimethoprim–sulfamethoxazole	44 (73.3)	6 (10.0)	10 (16.7)
Colistin	6 (10.0)	4 (6.7)	50 (83.3)

Table 3. Phenotypic and molecular virulence characteristics of clinical A. baumannii isolates

Virulence trait	Positive n (%)
Strong biofilm formation	29 (48.3)
Moderate biofilm formation	18 (30.0)
Weak biofilm formation	9 (15.0)
Non-biofilm producer	4 (6.7)
Motility positive	47 (78.3)
Hemolysin production	31 (51.7)
ompA gene	44 (73.3)
bap gene	39 (65.0)
plcN gene	26 (43.3)
lasB gene	21 (35.0)

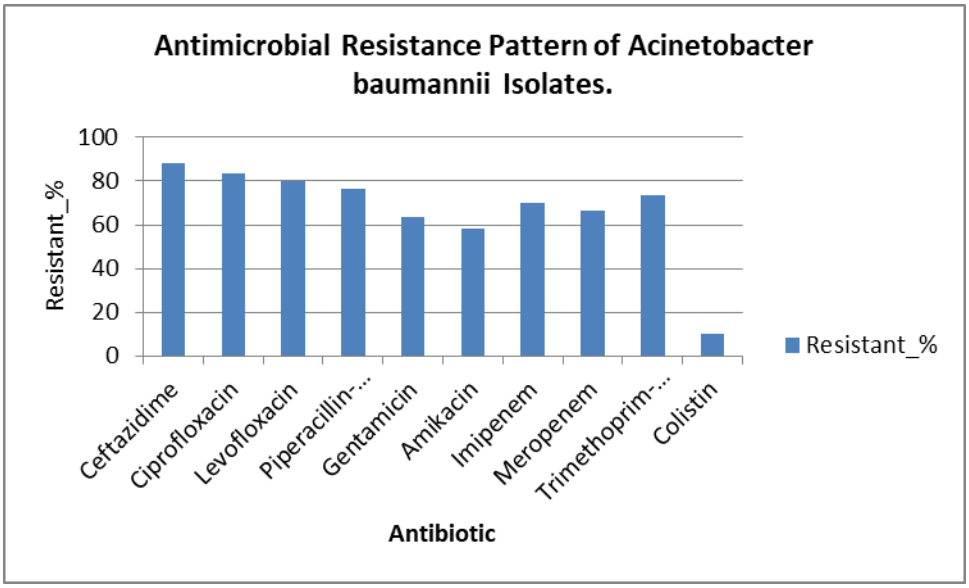

Figure 1. Antimicrobial resistance pattern of clinical Acinetobacter baumannii isolates against the tested antibiotics.

Figure 1 shows the antimicrobial resistance pattern of clinical Acinetobacter baumannii isolates against the tested antibiotics. The highest resistance rates were observed against ceftazidime, ciprofloxacin, levofloxacin, and piperacillin tazobactam, whereas colistin showed the lowest resistance rate and remained the most active agent. This pattern indicates a high level of multidrug resistance among the isolates.

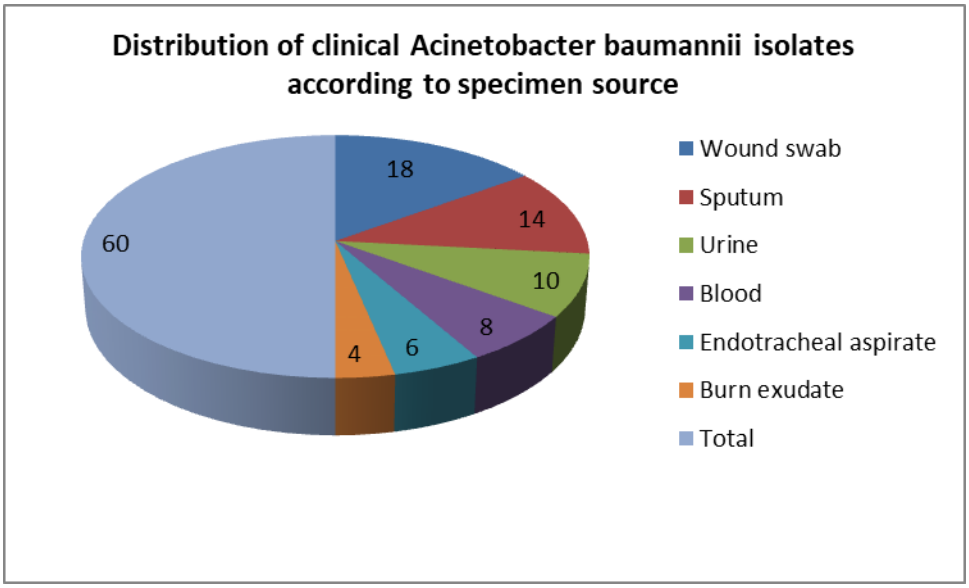

Figure 2. Distribution of clinical Acinetobacter baumannii isolates according to specimen source.

Figure 2 shows the distribution of clinical Acinetobacter baumannii isolates according to specimen source. Wound swabs represented the highest proportion of isolates, followed by sputum, urine, and blood samples. Lower frequencies were recorded in endotracheal aspirates and burn exudates. This distribution indicates that A. baumannii was more frequently isolated from infection sites associated with severe hospital-acquired infections.

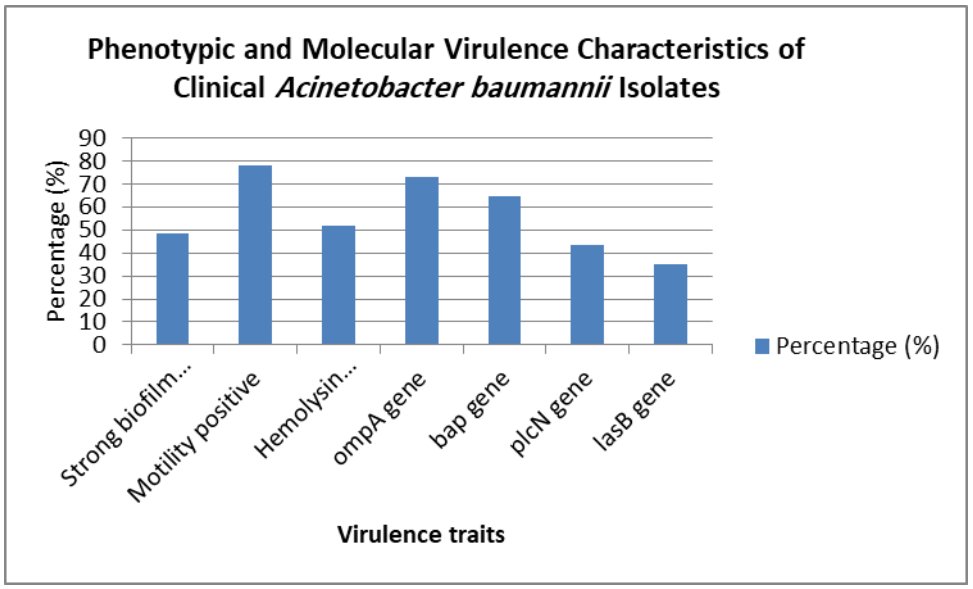

Figure 3. Phenotypic and molecular virulence characteristics of clinical Acinetobacter baumannii isolates.

Figure 3 illustrates the distribution of phenotypic and molecular virulence characteristics among clinical Acinetobacter baumannii isolates. Motility and the presence of the ompA gene were the most frequent traits, followed by bap and hemolysin production, while lasB showed the lowest prevalence. This pattern indicates that the isolates possessed multiple virulence determinants with potential relevance to persistence and pathogenicity.

Discussion

In this regard, the current study showed that clinical Acinetobacter baumannii isolates were mainly obtained from wound swabs and sputum specimens with a high proportion from intensive care unit patients. This distribution aligns with the recognized epidemiology of A. baumannii as a hospital-adapted opportunistic pathogen that preferentially targets fragile patients and colonizes respiratory and wound habitats in nursing homes, ICUs or surgical wards . The relative abundance of isolates from invasive and device-associated infections additionally mirrors the selection pressure for persistence on abiotic surfaces and evasion of compromised host barrier, a significant enhancer of nosocomial transmission success. High frequency of resistance to frequently used antimicrobial classes, especially cephalosporins and fluoroquinolones and carbapenems was detected based on unique mechanisms confirmed by whole genome sequencing; however, colistin was the most active drug against most isolates. This pattern correlates with multiple reports, which describe A. baumannii as multidrug-resistant, with a lack of treatment options and an increased dependence on last-resort agents. The resistance to carbapenems is particularly concerning as these agents are generally first-line therapy for serious Gram-negative infections. The majority of isolates retained susceptibility to colistin, with a small subset also resistant to the antimicrobial and serves as an early warning sign for more treatment-restriction. One of the most interesting results from this work was the high biofilm production (48%) obtained with almost all strains producing strong biofilms. This result is particularly relevant because biofilm formation is one important virulence mechanism of A. baumannii: it enables bacteria to adhere to tissue and medical devices, escape from host clearance mechanisms, and show high tolerance against antibiotics [21]. It has been shown over and over that biofilm-forming strains can be associated with chronic colonisation, ventilator-assisted pneumonia, bloodstream infection and worse outcomes in hospitalised patients. Thus, the high prevalence of biofilm-producing isolates found in this study further supports the concept that virulence may no longer be viewed solely as an accessory trait within A. baumannii pathogenesis and more as a facet of clinical success for this species overall. At the molecular level, virulence gene detection showed

that ompA and bap were the most commonly detected markers (51 to 100% positive), followed by plcN and lasB. This pattern is in line with the old study showed that omitting ompA and bap may be very important for A. baumannii activity with regard to adherence, biofilm formation and host-cell interaction[1]. The impressive number of isolates carrying these genes shows that the clinical strains present within the studied environment are correctly adapted to survival and persistence in it. The absence of plcN and lasB may therefore be indicative of strain specific adaptation or genetic diversity and reflect virulence factor content heterogeneity among local isolates. This variability is a characteristic of A. baumannii, a species recognized for its genomic plasticity and the non-uniform presence of virulence factors for certain clones (Shupon et al., 2018).

One of the most clinically significant conclusions drawn from this study is the positive correlation observed between biofilm production and antimicrobial resistance. The isolates forming biofilms had a significantly higher resistance rate than non-biofilm-forming isolates to cephalosporins, fluoroquinolones and carbapenems. Several studies have noted this relationship, which is postulated to be a combination of protection by physical removal, altered metabolic activity, limitation of antibiotic penetration and enhanced transferability of resistance elements between biofilm communities. This means that in practice a avirulent isolate would be seen as resistant from an in vitro perspective but the ability of this isolate to form a biofilm could contribute to therapeutic failure in vivo. Thus, resistance testing alone may misleadingly downplay the actual therapeutic obstacle that these strains present. Evidence for motility and hemolytic activity in a large proportion of isolates also indicates their potential pathogenicity. While A. baumannii is not classically defined as motile to the degree that other Gram-negative bacteria are, surface-associated motility can play a role in colonisation and dissemination over moist or semi-solid surfaces. Likewise, hemolytic and cell-destructive activity could facilitate both tissue penetration and the consequence inflammatory damage. In combination with biofilm formation and virulence genes carriage, these phenotypes suggest a multi-factorial nature of the virulence mechanism in isolates. Conclusion The concurrent high multidrug resistance with tight biofilm formation and wide distribution of virulence genes is concerning in terms of epidemiology. These strains are more prevalent within the ward environment, endure in the presence of disinfectant, contaminate equipment and lead to persistent colonization or infection patients. These data underscore the importance of rigorous infection prevention practices (eg contact precautions, environmental decontamination, targeted surveillance cultures as indicated and judicious antimicrobial stewardship). Virulence and resistance trends should also be locally evaluated to inform empirical therapy and document emergence of high-risk clones in communities at an early stage. In conclusion, the volunteer study reports that clinical A. baumannii isolates in circulation from a tertiary hospital are at an alarming link between multidrug-resistant virulence potential. Since biofilm formation is commonly linked with adaptation and persistence in the clinical environment , the high rate of biofilm positives obtained from clinical samples and perpetuity of these strains to bear concurrent important virulence factors, particularly ompA and bap, suggests a synergistic interaction between pathways of isolate survival into human internal reserves during infection. These data underline the need for continued surveillance, diagnostic improvements and stronger policies to restrict infection-control measures of this important pathogen.

Conclusion

Clinical Acinetobacter baumannii isolates have been rare in clinical practice, but we report here that a clinical isolate simultaneously achieves high-level antibiotic resistance and systemic invasion. In addition, the high ability of biofilm production, occurrence of major virulence genes as well as reduced sensitivity to several first-line antibiotics are serious challenges in therapeutic management of infections caused by this pathogen. This underscores the need for your continued surveillance, implementation of effective infection control measures and rational antibiotic use to prevent and contain A. baumannii in hospitals endemic settings.

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