Original Article

# Clinical and antimicrobial profile of *Acinetobacter* spp.: An emerging nosocomial superbug

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## Abstract

**Background:** Recently, *Acinetobacter* has emerged as significant hospital pathogen, notoriously known to acquire antibiotic resistance to most of the commonly prescribed antimicrobials. Many risk factors are associated with *Acinetobacter* infections, especially in patients in intensive care unit (ICU). This study aims to isolate *Acinetobacter* from various clinical specimens and to determine its antimicrobial sensitivity pattern.

**Materials and Methods:** Identification, speciation and antimicrobial sensitivity testing were performed using the standard microbiological techniques. Slime production was also tested by microtiter plate and tube method.

**Results:** From the processed clinical specimens, 107 *Acinetobacter* strains (1.02%) were isolated of which 76 (0.74%) isolates were from general wards and 31 (11.96%) were from ICU. Significantly higher percentage of *Acinetobacter* strains was found in ICU compared with general wards ($P < 0.05$). Most common *Acinetobacter* infection was abscess. Infections were more common in males and were associated with major risk factors such as post-surgical, diabetes mellitus, catheterization, extended hospital stay and prolonged antibiotic usage. *Acinetobacter baumanii* was the most common species isolated to cause abscess, wound infection, etc. 62.61% and 28.97% isolates produced slime by microtiter plate and tube method. Imipenem was most sensitive drug followed by amikacin. Ceftazidime, cefotaxime, piperacillin were most resistant. 43.00% isolates were IPM resistant. *A. baumanii* was more resistant to commonly used antimicrobials.

**Conclusion:** *Acinetobacter* nosocomial infections resistant to most antimicrobials have emerged, especially in ICU. Early identification and continued surveillance of prevalent organism will help prevent the spread of *Acinetobacter* in hospital environment.

**Key Words:** *Acinetobacter*, antimicrobial resistance, nosocomial pathogen

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## INTRODUCTION

*Acinetobacter* are Gram-negative *Coccobacilli*, strictly aerobic, non-motile, catalase positive, oxidase negative and lack pigmentation.[1] They are ubiquitous[2] free living saprophytes in soil and water.[3]

Up to 25% of healthy ambulatory adults exhibit cutaneous colonization by *Acinetobacter* and are the



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most common Gram-negative bacteria carried on the skin of hospital personnel.[4] They are usually opportunistic pathogens reported to cause a number of outbreaks of nosocomial infections such as septicemia, pneumonia, wound sepsis, endocarditis, meningitis, urinary tract infections and peritonitis,[5] but their predominant role is in ventilator associated pneumonia (VAP), in intensive care units (ICUs).[1]

Predisposing factors for *Acinetobacter* infections include the presence of prosthesis, endotracheal intubation, intravenous (I.V.) catheters and prior antibiotic therapy in a seriously ill-patient in hospital.[3] Such infections are often extremely difficult to treat because of widespread resistance to the major groups of antibiotics and long-term survival of bacteria in the hospital environment.[1]

Resistance to all known antibiotics has now emerged in *Acinetobacter* spp. with the majority of strains still being susceptible to carbapenems.[6] Multidrug-resistant (MDR) *Acinetobacter* infections are associated with increased time on mechanical ventilation, in the ICU and in the hospital. Treatment options are severely limited; carbapenems and colistin are the agents of choice. More research and greater emphasis on the prevention of health-care associated transmission of MDR *Acinetobacter* infection are essential.[7]

The aim of this study was to isolate *Acinetobacter* species from clinical specimens and to study the antimicrobial susceptibility pattern of *Acinetobacter* isolates.

## MATERIALS AND METHODS

The study was carried out in the Department of Microbiology from August 2008 to September 2010. Relevant clinical specimens (sputum, blood, pus, urine, cerebrospinal fluid, peritoneal fluid etc.) were collected from inpatient and out-patient departments by standard collection procedures. No specific exclusion criteria envisaged. Specimens were processed by standard microbiological techniques.[3] Non-fermenters were initially separated and further identified as *Acinetobacter* spp. In Gram stain of direct smears *Acinetobacter* appeared as tiny, Gram-negative coccobacillary cells often appearing as diplococci.[5] All specimens were inoculated on 10% sheep blood agar and MacConkey agar and incubated at 37°C for 18-24 h.[3] Colonies on blood agar were 0.5-2 mm diameter, translucent to opaque (never pigmented), convex and entire. On MacConkey agar a faint pink tint was produced.[5] Gram stain, catalase, oxidase and motility tests were performed. *Acinetobacter* are Gram-negative *Coccobacilli*, non-motile, strictly aerobic, catalase positive and oxidase negative. Rapid utilization of 10% glucose was seen with O-F medium. *Acinetobacter* isolates were differentiated from other oxidase negative, non-motile organisms such as Centers for Disease Control and Prevention NO-1, *Bordetella holmessii* by nitrate reduction test and presence of brown soluble pigment.[5]

*Acinetobacter* isolates confirmed by the above standard microbiological tests were further speciated as per the following scheme of identification[3,5] [Table 1].

All *Acinetobacter* spp. were tested for slime production, an important virulence factor by two methods viz. microtiter plate method[8] and tube method.[9] In microtiter plate method, optical density (OD) of stained adherent bacteria was determined at 570 nm wavelength. If OD value is >0.240 then it was strong slime producer.[8] In tube method, biofilm formation was considered positive when a visible film lined the wall and bottom of the tube.[9]

Antimicrobial susceptibility testing[3] was performed by modified Kirby Bauer method[10] as per the Clinical and Laboratory Standards Institute guidelines.[11] Antibiotics tested were ceftazidime (CAZ), ciprofloxacin (CIP), imipenem (IPM), gentamicin, tobramycin (TOB), amikacin (AK), piperacillin-tazobactam (P/T), cefepime (CPM), cefotaxime (CTX), tetracycline, piperacillin (PIP), trimethoprim-sulfamethoxazole (COT), gatifloxacin (GAT).

### Statistical analysis

*P* value was reported and a value of $P < 0.05$ was considered as a significant. The statistical analysis was performed using the Chi-square test.

## RESULTS

In total, 107 *Acinetobacter* strains (1.02%) were isolated from the processed clinical specimens (10,453). Out of these 107 *Acinetobacter* isolates, 76 (0.74%) isolates were from general wards and 31 (11.96%) were from ICU. Significantly higher percentage of *Acinetobacter* strains were found in ICU compared with general wards ($P < 0.05$) [Table 2]. The

**Table 1: *Acinetobacter* species identification**

| *Acinetobacter* spp. | Growth at 37°C | Growth at 44°C | Hemolysis | Gelatin hydrolysis | OF glucose | Arginine |
|---|---|---|---|---|---|---|
| *A. baumannii* | + | + | − | − | + | + |
| *A. calcoaceticus* | + | − | − | − | + | + |
| *A. haemolyticus* | + | − | + | + | V | + |
| *A. lwoffii* | + | − | − | − | − | − |
| *A. junii* | + | − | − | − | − | + |
| *A. johnsonii* | − | − | − | − | − | V |

most common *Acinetobacter* infection was abscess (28.03%), followed by pneumonia (23.86%), septicemia (17.75%), wound infection (16.84%) and urinary tract infection (12.14%) [Table 3]. *Acinetobacter* infections were more common in males (54.20%) as compared with females (45.80%). Major risk factor associated with *Acinetobacter* infection were post-surgical (37.50%), followed by diabetes mellitus (8.33%), I.V. catheterization (21.05%), extended hospital stay (10.52%) and mechanical ventilation (84.00%) [Table 3]. Most common *Acinetobacter* species isolated was *Acinetobacter baumannii* (79.43%) [Table 3]. *A. baumannii* was the most common species responsible for abscess (90.00%), wound infection (88.88%), septicemia (47.36%), urinary tract infection (61.54%) and pneumonia (92.00%). Out of 107 *Acinetobacter* isolates, slime production can be detected in 62.61% isolates by microtiter plate method, but in only 28.97% by tube method. Though laborious, microtiter plate method is the reliable and reproducible method for demonstration of slime production. The maximum sensitivity of *Acinetobacter* was seen to IPM (57.00%), AK (55.14%), followed by GAT (44.87%) and TOB (41.12%). Maximum resistance was observed to CAZ (100%), CTX (100%), PIP (100%) and P/T (86.92%). IPM resistance was seen in 46 (43.00%) *Acinetobacter* strains [Figure 1]. In general wards and in ICU, *A. baumannii* was more resistant to commonly used antimicrobials. *Acinetobacter junii* was more susceptible to the majority of the drugs used.

**Table 2: Distribution of specimens and *Acinetobacter* isolates**

| Place of collection | No. of specimens | No. of *Acinetobacter* isolates (%) |
|---|---|---|
| General wards | 10,194 | 76 (0.74)* |
| ICU | 259 | 31 (11.96)* |
| Total | 10,453 | 107 (1.02) |

*Chi-square test, $P < 0.05$. Table 2 shows that out of the total 10,453 samples processed 107 (1.02%) *Acinetobacter* strains were isolated. Of the 107 isolates, 76 (0.74%) isolates were from general wards and 31 (11.96%) were from intensive care units. Significantly higher percentage of *Acinetobacter* strains were found in ICU compared with general wards ($P < 0.05$), ICU: Intensive care unit

## DISCUSSION

*Acinetobacter* spp. is Gram-negative *Coccobacilli* that contribute profoundly to the burden of modern medicine. *Acinetobacter* spp. is the second most commonly isolated non-fermenter in human specimens (after *Pseudomonas aeruginosa*). They rank fourth (after *P. aeruginosa, Stapylococcus aureus* and *Klebsiella pneumoniae*)

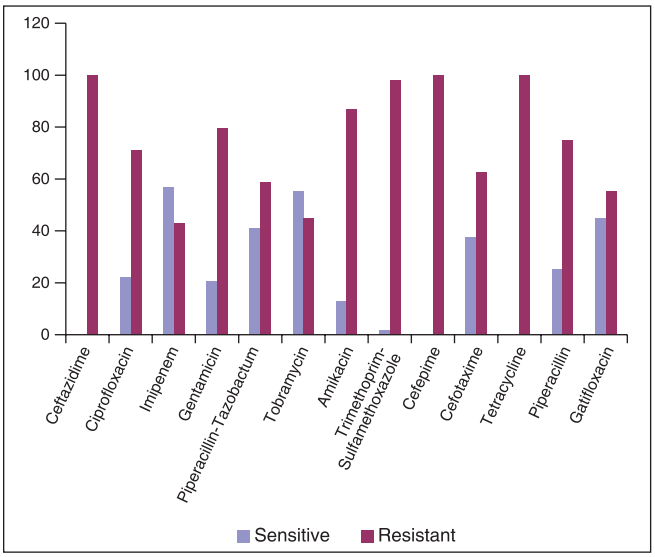


**Figure 1:** Antimicrobial sensitivity pattern of *Acinetobacter* isolates ($n = 107$)

**Table 3: Distribution of *Acinetobacter* species, major risk factors and various infections ($n = 107$)**

| *Acinetobacter* infections | Associated risk factor (%) | *A. baumannii* $n = 85$ (%) | *A. calcoaceticus* $n = 13$ (%) | *A. haemolyticus* $n = 4$ (%) | *A. lwoffii* $n = 3$ (%) | *A. junii* $n = 2$ (%) | Total $n = 107$ (%) |
|---|---|---|---|---|---|---|---|
| Abscess | Post-surgical (37.5) Diabetes mellitus (8.33) | 27 (90.00) | 2 (6.66) | 1 (3.34) | – | – | 30 (28.03) |
| Pneumonia/ventilator associated pneumonia | Mechanical ventilation (84) Chronic obstructive pulmonary disease (4) | 23 (92.00) | 2 (8.00) | – | – | – | 25 (23.36) |
| Septicemia | IV catheter (21.05) Hospital stay (>7 days) (10.52) Surgery (5.26) Parental nutrition, anemia (10.52) | 9 (47.36) | 3 (15.79) | 2 (10.53) | 3 (15.79) | 2 (10.53) | 19 (17.75) |
| Wound infection | Trauma (6.25) Previous infection (10.52) | 16 (88.88) | 1 (5.56) | 1 (5.56) | – | – | 18 (16.84) |
| Urinary tract infection | Catheterization (38.46) Prolonged antibiotic use* and hospital stay (>7 days) (15.38%) | 8 (61.54) | 5 (38.46) | – | – | – | 13 (12.14) |
| Pleural effusion | – | 1 (100.00) | – | – | – | – | 1 (0.94) |
| Meningitis | – | 1 (100.00) | – | – | – | – | 1 (0.94) |
| Total | | 85 (79.43) | 13 (12.14) | 4 (3.73) | 3 (2.81) | 2 (1.89) | 107 |

*Antibiotics such as third generation cephalosporins, fluoroquinolones

among the most frequent hospital acquired infectious agents.[12] *Acinetobacter* spp. have emerged as a cause of ICUs infection. Multiresistant *Acinetobacter* spp. have become established as "alert" pathogens, particularly in ICUs and are associated with outbreaks of infection.[13] Their ubiquitous nature in the ICU environment and inadequate infection control practice have continuously raised the incidence of *Acinetobacter* infections over the past two decades. The understanding and recognition of *Acinetobacter* infections in the ICU is critically needed.[14]

In our study, a total number of 107 (1.02%) *Acinetobacter* strains were isolated from processed clinical specimens. Houang *et al*.[15] reported a total of 1.32% *Acinetobacter* isolates from all clinical specimens, which was well comparable with our study. In our study, 31 (11.96%) *Acinetobacter* strains were isolated from clinical specimens from ICUs and *Acinetobacter* infections were more common in ICU as compared with general wards ($P < 0.05$) [Table 2]. Prashanth and Badrinath[16] reported 10.00% *Acinetobacter* infections in ICU. Patwardhan *et al*.[17] isolated 13.23% *Acinetobacter* isolates. Our findings are comparable with Patwardhan *et al*. Occurrence of *Acinetobacter* is contributed by several factors including immunosuppressed hosts, patients with severe underlying disease, previous use of antibiotics, duration of hospital stay and more frequent use of antibiotics in ICU. Patients in ICU are sicker and require more invasive monitoring and therapeutic procedures to survive. ICU environmental contamination appears to be another important source of *Acinetobacter* infection.[14] The development of ICU-acquired infections is strongly related to prolonged ICU stay and is associated with worse outcomes including increased morbidity and mortality.[18] In the present study, most common infection was abscess (28.03%), followed by pneumonia (23.86%), septicemia (17.75%), wound infection (16.84%) and urinary tract infection (12.14%) [Table 3]. Joshi *et al*.[19] reported that 27.50% of wound infection were caused by *Acinetobacter*. *Acinetobacter* ICU-acquired infections during the last decade represent a growing concern among clinicians and researchers. These infections most frequently involve the respiratory tract of intubated patients.[18] In our study, out of the 31 *Acinetobacter* isolates from ICU, 21 (67.74%) *Acinetobacter* were isolated from patients on mechanical ventilation causing VAP. Bennani *et al*.[20] reported 68.18% VAP ranging from 9% to 68% *Acinetobacter* infections. Our findings are comparable with Bennani *et al*.

In the present study, *Acinetobacter* infections were more common in males (54.20%) as compared with females. This may be due to the fact that the males report more frequently to the hospitals compared with females. Prashanth and Badrinath[16] reported the infections to be more common in males (58.00%) compared with females (42.00%). Joshi *et al*.[19] reported 50.20% infection in males.

In the present study, out of 107 *Acinetobacter* cases major predisposing and associated risk factors were evident in many cases [Table 3]. Joshi *et al*.[19] reported existing debilitating chronic illness (20.20%), post-operative surgical (18.50%), trauma (3.30%), urinary catheterization (4.10%) as risk factors associated with *Acinetobacter* infections.

Currently at least 31 *Acinetobacter* genomospecies have been described. *Acinetobacter johnsonii, Acinetobacter lwoffii* and *Acinetobacter radioresistant* seem to be natural inhabitants of human skin and commensals in human oropharynx and vagina.[5] The digestive tract of patients within ICUs often serve as reservoirs for multiresistant *A. baumannii* strains involved in hospital outbreaks.[2] The most common site for *A. baumannii* infection is the respiratory tract and the most common manifestation is VAP and bloodstream infections. *A. lwoffii* has been more commonly associated with meningitis, *A. junii* rarely causes ocular infection and bacteremia.[5] In our study, out of the 107 *Acinetobacter* isolates, *A. baumannii* (79.43%) was the most common species to cause *Acinetobacter* infection [Table 3]. From 140 *Acinetobacter* isolates, Joshi *et al*.[19] isolated 70.00% *A. baumannii*, 1.40% *Acinetobacter calcoaceticus*, 6.40% *Acinetobacter haemolyticus*, 8.60% *A. junii* and 1.40% *A. johnsonii*. Prashanth and Badrinath[16] isolated 71.42% *A. baumannii*, 10.02% *A. lwoffii,* 4.08% *A. haemolyticus* and 2.04% strains of *A. junii*.

The ability of *Acinetobacter* strains to adhere to surfaces is an important mechanism in the pathogenicity. It frequently causes infections associated with medical devices, e.g., vascular catheters, cerebrospinal fluid shunts or Foley catheters. Biofilm formation is a well-known pathogenic mechanism in such infections.[21] Biofilms have clinical and therapeutic implications, because biofilms preserve bacteria from the action of hosts defensive mechanisms and antimicrobial activity against bacteria in biofilms might be substantially diminished.[21] In the present study, out of total 107 *Acinetobacter* isolates, 67 (62.61%) *Acinetobacter* isolates produced slime by microtiter plate method, but only 31 (28.97%) isolates by Tube method. Rodríguez-Baño *et al*.[21] reported 63.00% biofilm production in *Acinetobacter* isolates. Our findings are comparable with Rodríguez-Baño *et al*.

As noted by the Infectious Disease Society of America, *Acinetobacter* is "a prime example of mismatch between unmet medical need and the current antimicrobial research and development pipeline." *Acinetobacter* spp. are notorious for their ability to acquire antibiotic resistance.[22] Antimicrobial

resistance among *Acinetobacter* spp. has increased substantially in the past decade and has created a major public health dilemma. The most potent antibiotic drug class currently available are the carbapenems, but resistant strains have emerged.[7] We have studied the antimicrobial resistance pattern among *Acinetobacter* isolates by Kirby-Bauer disc diffusion method. In our study, *Acinetobacter* isolates showed resistance to most of the antibiotics available. Maximum sensitivity was observed to IPM (57.00%), AK (55.14%), followed by GAT (44.87%) and TOB (41.12%). Maximum resistance was observed to CAZ (100%), CTX (100%), PIP (100%), CPM (98.13%) and P/T (86.92%). IPM resistance was seen in 46 (43.00%) *Acinetobacter* strains [Figure 1]. Sinha *et al*.[23] reported maximum sensitivity to meropenem (86.00%), CIP (36.00%), AK (33.00%), CPM (26.00%), CAZ (26.00%) and maximum resistance was reported to PIP (90.00%) and CTX (87.00%). *Acinetobacter* spp. is universally resistant to penicillin, ampicillin and cephalothin. Various susceptibility to second and third generation cephalosporins have been reported.[5] *Acinetobacter* species possess a wide array of β-lactamases that hydrolyze and confer resistance to penicillins, cephalosporins and carbapenems. AmpC cephalosporinases are chromosomally encoded and confer resistance to broad-spectrum cephalosporins. Class D oxacillin-hydrolyzing-type enzymes, Class B metallo β-lactamases (MBLs), hydrolyze a broad array of antimicrobial agents, including carbapenems. Increasing antimicrobial resistance leaves few therapeutic options for MDR *Acinetobacter* infection. The Meropenem Yearly Susceptibility Test Information Collection surveillance program has documented discordance that favors IPM as the more potent agent, compared with meropenem, for treatment of MDR *Acinetobacter* infection.[7] In the present study, 43.00% of *Acinetobacter* were IPM resistant. Out of these, 60.71% were imipenem resistant *A. baumannii* (IRAB) compared with 16.66% *A. calcoaceticus* in general wards and 34.48% IRAB in ICU. Sinha *et al*.[23] reported 35.00% IPM resistant *Acinetobacter*. Lee *et al*.[24] reported 21.18% IRAB. Corbella *et al*.[25] reported 36.00% carbapenem resistant *A. baumannii* from the patients admitted to ICU.

## CONCLUSIONS

*Acinetobacter* are the "superbugs" of the modern hospital environment causing significant proportion of infections in specific patient populations, especially in critically-ill patients in the ICU. As ubiquitous organisms (fortunately of low virulence), with few requirements for growth and survival, *Acinetobacter* spp. are prone to persist indefinitely in the hospital environment and to cause infections periodically when iatrogenic factors are present, i.e., overuse of broad spectrum antibiotics and high-risk patients. This situation, together with the fact that *Acinetobacter* isolates have inherent and/or easily acquired mechanisms of resistance against many of the available antimicrobial agents, makes this pathogen one of the most significant microbial challenges of the current era. Antibiotic resistance is attributed to production of extended spectrum beta-lactamase, MBL, loss of outer membrane proteins, efflux pumps and biofilm formation. Are there ways to control or limit the spread of these multiresistant strains? Is it still possible to treat *Acinetobacter* infections? First, it is necessary to improve microbiological techniques for early and more accurate identification and laboratory vigilance to prevent inappropriate empirical treatment. Second, newer strategies for antibiotic use should be employed to reduce selection pressure, including more frequent rotation of antibiotic groups or sequential use of antibiotic classes. The development of totally new antibiotics with novel bacterial molecular target sites may constitute therapeutic alternatives within the next few years. Nevertheless, continued surveillance of prevalent organisms in ICUs, combined with preventive measures (e.g., isolation precautions, hand disinfection, efficient sterilization of instruments) remains absolutely essential in efforts to prevent or limit the spread of *Acinetobacter* infection. Continued awareness to maintain good housekeeping, control of the environment including equipment decontamination, strict attention to hand washing, isolation procedures and control of antibiotic usage, especially in high-risk areas, appear most likely measures to control the spread of *Acinetobacter* spp. in hospitals.

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