

## Original Article

# Acinetobacter Blood Stream Infection in a Teaching Hospital - Riyadh, Saudi Arabia

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**ABSTRACT**

**Objective:** To study the clinical conditions of patients with blood stream infection (BSI) due to *Acinetobacter* species, the predisposing factors, the antimicrobial susceptibilities and the outcome of infection by these organisms.

**Settings:** The study took place at King Khalid University Hospital, Riyadh, Saudi Arabia.

**Methods:** Forty patients with blood stream infection due to *Acinetobacter* species were prospectively studied. *Acinetobacter* isolates were identified by API 20E. Antimicrobial susceptibility to 13 antimicrobial agents was performed by a disc comparative Stoke's method. For 23 isolates, susceptibility was also tested by minimum inhibitory concentration using E test (Ab biodisk, Solna, Sweden).

**Results:** The predominant *Acinetobacter* isolate was *Acinetobacter baumannii* 24 (60%) followed by *Acinetobacter baumannii* complex 10 (25%) and six (15%) were other *Acinetobacter* species. Patients with *Acinetobacter baumannii* blood stream infection were more frequently managed in intensive care units. Nineteen of them (47.5%) had serious underlying illnesses predisposing to *Acinetobacter* blood

stream infections including, cardiac, renal diseases, prematurity and severe burns with six (25%) having a fatal outcome. Risk factors for *Acinetobacter baumannii* blood stream infection included: intravascular catheters, mechanical ventilation, prior antibiotic use and colonization at other body sites. These factors were independently associated with *Acinetobacter baumannii* acquisition in these patients ( $P = > 0.05$ ). The results of antimicrobial susceptibility tested by disc diffusion method were comparable to those of E test. Among the 13 antimicrobial agents tested, imipenem was the most active agent against *Acinetobacter baumannii* as well as other *Acinetobacter* species.

**Conclusion:** We concluded that *Acinetobacter baumannii* is the most common *Acinetobacter* species causing significant blood stream infections among patients in intensive care units with serious underlying illnesses. Risk factors studied were independently associated with the disease process of these patients. Imipenem is the most active antimicrobial agent against clinically significant *Acinetobacter baumannii* blood stream infection.

**KEYWORDS:** *Acinetobacter baumannii*, *Acinetobacter calcoaceticus-baumannii* complex, blood stream infection, intensive care units, risk factors

**INTRODUCTION**

*Acinetobacter* species are a heterogenous group of gram negative coccobacilli, which have been grown from numerous human sources and are widespread in the environment<sup>[1-3]</sup>. Although these organisms are normally considered to be of low virulence, they have been increasingly implicated as a cause of a wide spectrum of infections including community and hospital acquired infections associated with intravenous catheters and contaminated respiratory therapy equipment among patients with impaired host defences in intensive care units<sup>[3-9]</sup>. *Acinetobacter baumannii* (*A. baumannii*) is the main species with clinical significance in nosocomial context<sup>[3,7,9]</sup>.

To our knowledge, this is the first report on blood stream infections (BSI) due to *Acinetobacter* species, from the Kingdom of Saudi Arabia, in patients seen at King Khalid University Hospital (KKUH),

Riyadh, Saudi Arabia. We investigated the clinical conditions of these patients, the predisposing factors, the presence of colonization, the antimicrobial susceptibilities and the outcome of infections caused by these organisms.

**PATIENTS AND METHODS**

This prospective study was carried out during the period from 1st of April 2000 to 31 December 2001 on 40 patients managed at (KKUH) Riyadh, Saudi Arabia. KKUH is a 850-bed providing primary, secondary and tertiary health care. It has five different intensive care units (ICUs) including medical, surgical, cardiac, pediatric and neonatal intensive care units in addition to haemodialysis and burn units.

The clinical data collected from each patient included age, sex, in-patient/out-patient, underlying

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diseases, date of onset of BSI after admission, other specimens yielding *Acinetobacter* species, risk factors including intravenous devices, urinary catheters, hemodialysis shunts, endotracheal tubes, ventilator support, previous antibiotic given within the last month, and outcome of infection. Five to ten milliliters of peripheral venous blood were collected from each patient and divided equally and inoculated into BacT/Alert 3D bottles (Organon Teknika, USA). Blood from bottles showing positive growth index was gram stained, and those with gram negative rods or coccobacilli were subcultured on sheep blood and McConKey agar plates and incubated aerobically for 24 hours at 37° C. Isolates were identified as *Acinetobacter* by a negative oxidase test and API 20E system updated profile (Biomérieux, SA, France). Multiple blood cultures yielding the same organism from the same patient were considered to be a single infection. Antimicrobial susceptibility was performed by disc comparative Stoke's method using *Pseudomonas aeruginosa* (ATCC TM 27853) as a control organism<sup>[10]</sup>. Minimum inhibitory concentration (MIC) of antimicrobial agents for 23 isolates was done using E test (AB Biodisk, Solna, Sweden) according to the manufacturer's instructions. The following antimicrobial agents were tested: ampicillin, tetracycline, cefuroxime, ceftriaxone, ceftazidime, cefotaxime, aztreonam, gentamicin, amikacin, ciprofloxacin, imipenem, piperacillin and cotrimoxazole.

### Statistical analysis:

Results were analyzed by the X<sup>2</sup> Mann-Whitney tests. The P value of < 0.05 was set as significant.

### RESULTS

Forty patients with BSI due to *Acinetobacter* species were reported during the study period. No outbreak was noticed during this period. Patients' demographics, underlying illnesses, predisposing factors and outcome of infections are shown in Table 1. Twenty-one (52.5%) of patients were females. Mean age was 46 years, and male patients were younger than female patients. Ten patients (25%) were children and five were premature infants. Thirty-eight (95%) were inpatients, two were outpatients. Clinically significant blood stream infection was defined as a bacteraemic episode associated with clinical findings such as fever or leucocytosis that lasted at least eight hours<sup>[11]</sup>. Such *Acinetobacter* infection occurred in 37 (92.5%) patients and in three (7.5%), *Acinetobacter* isolates were considered as contaminants. In 27 (67.5%) patients, blood stream infection developed after a mean interval of 19 days of hospital admission. For the remaining patients, this information could not be obtained. While *A.*

**Table 1**

Patient demographics, underlying diseases, predisposing factors and outcome of BSI caused by *Acinetobacter*

Patients Data	Total N=40 (%)	<i>A. baumannii</i> 24 (60) N (%)	<i>A. calcoaceticus</i> <i>s-baumannii</i> complex 10 (25) N (%)	Other <i>Acinetobacter</i> species 6 (15) N (%)	P-Value
Sex:					
Females	21 (52.5)	12 (50)	4 (40)	5 (83.3)	**NS
Males	9 (47.5)	12 (50)	6 (60)	1 (16.7)	NS
Mean age (years)	46	44	41	25	NS
ICU Care	19 (47.5)	11 (45.8)	5 (50)	3 (50)	NS
Underlying disease:					
Cardiac	12 (30)	6 (25)	5 (50)	1 (16.7)	NS
Pulmonary	4 (10)	3 (12.5)	1 (10)	0 (0)	NS
Renal	8 (20)	7 (29.1)	0 (0)	1 (16.7)	NS
Trauma	4 (10)	3 (12.5)	1 (10)	0 (0)	NS
*Others	12 (30)	5 (20.8)	2 (20)	5 (83.3)	NS
Predisposing factors:					
Intravascular catheters	34 (85)	22 (91.7)	8 (80)	4 (66.7)	NS
Urinary Catheters	9 (22.5)	6 (25)	3 (33.3)	0 (0)	NS
Endotracheal tube	10 (25)	7 (29.1)	3 (30)	0 (0)	NS
Ventilator support	19 (47.5)	14 (58.3)	3 (30)	2 (33.3)	NS
Haemodialysis	8 (20)	6 (25)	1 (10)	1 (16.7)	NS
Prior antibiotic use	26 (65)	20 (83.3)	6 (60)	0 (0)	NS
Colonization at other body site	6 (15)	4 (16.7)	1 (10)	1 (16.7)	NS
Other bacteria isolated	8 (20)	6 (25)	1 (10)	1 (16.7)	NS
Fatal Outcome	7 (17.5)	6 (25)	0 (0)	1 (16.7)	NS

\*Others = Including cases of: Systemic lupus erythematosus, hepatocellular carcinoma, hepatic encephalopathy, post-laminectomy, post-herniorrhaphy, burn, goiter, deep vein thrombosis and diabetic ketoacidosis.

\*\*NS = Not significant (p = 0.05)

*baumannii* constituted 60% of the isolates, *A. calcoaceticus-baumannii* complex accounted for 25%. These two isolates accounted for 34 (85%) of *Acinetobacter* BSI. Six (15%) isolates were other *A. species* among which were two, *A. lwoffii* and one *A. junii*, and three had no profile, all six were considered as other *A. species*. The majority of patients infected by *Acinetobacter* species were females with five (83.3%), compared to one male patient (16.7%) (P => 0.05). No such difference between the number of sexes infected with *A. baumannii* or *A. calcoaceticus-baumannii* complex was found. Intensive care management was required for 47.5% of patients and the majority 11(45.8%) had *A. baumannii* BSI. Cardiac illness was the commonest underlying condition among patients with *A. baumannii* and *A. calcoaceticus-baumannii* complex, six (25%) and five (50%) respectively. Renal disease was the next commonest and this was seen in seven (29.1%) patients with *A. baumannii* infections. Pulmonary disease and trauma due to road traffic accident were present in four patients, (10%) of each. Five patients (20.8%), yielded *A. baumannii*. The underlying illnesses included systemic lupus erythematosus, hepatocellular carcinoma, hepatic encephalopathy,

post-laminectomy, diabetic ketoacidosis and a patient with 90% burns, were seen in single patients (Table 1). Patients with *A. baumannii* more frequently had risk factors such as intravascular catheters in 22 (91.7%), prior antibiotic use in 20 (83.3%) (mainly including second and third generation cephalosporins, gentamicin and ciprofloxacin), and ventilatory support in 14 (58.3%), when compared with patients with *A. calcoaceticus*-*baumannii* complex and *Acinetobacter* species. None of these results reached statistical significance ( $P = 0.05$ ). Concomitant organisms were isolated with *Acinetobacter* in eight (20%) patients, six of these (25%) were simultaneously isolated from blood stream with *A. baumannii*. They included: *Pseudomonas aeruginosa*, *Enterococcus faecalis*, Methicillin Resistant *Staphylococcus aureus*, and *Enterobacter cloacae*. Viridans streptococci were isolated with *A. calcoaceticus*-*baumannii* complex from one patient and *Pseudomonas aeruginosa* was also isolated from one patient with *Acinetobacter* species. Colonization at other body sites was documented in four (16.7%) patients having *A. baumannii* BSI. These were isolated from urine and foley's catheter samples as well as swabs from axilla, nose, and throat. *A. calcoaceticus*-*baumannii* complex colonized the intravenous catheter of one patient and another *Acinetobacter* species colonized the endotracheal tube of another patient with BSI. A fatal outcome associated with BSI occurred in six patients (25%) with *A. baumannii* and one (16.7%) elderly patient with lung disease with another *Acinetobacter* species. Those with *A. baumannii* were two preterm quadruplets in the neonatal ICU, one 90% burns patient in surgical ICU, and the other three; elderly patients with pulmonary edema, post coronary artery bypass surgery and one with renal failure ( $P = 0.05$ ).

Results of antimicrobial susceptibility tested by disc diffusion method were comparable to those tested by MIC. Antimicrobial susceptibility of *A. baumannii* and *A. calcoaceticus*-*baumannii* complex isolates tested by MIC are listed in Table 2. *A. baumannii* was more resistant than *A. calcoaceticus*-*baumannii* complex. All species were susceptible to imipenem. Amikacin was active against all isolates of *A. calcoaceticus*-*baumannii* complex and 11 (84.6%) isolates of *A. baumannii*. Aztreonam, ceftriaxone, cefotaxime, cefuroxime and ampicillin were the least active against both species. All *Acinetobacter* species tested were susceptible to most antimicrobials, but some species (33.3%) were resistant to aztreonam, tetracycline and cotrimoxazole.

## DISCUSSION

It has been difficult to characterise the different species of *Acinetobacter*. However, it can be

**Table 2**

Antimicrobial susceptibility of 13 antimicrobial agents tested by MIC against *A. baumannii* and *A. calcoaceticus* - *baumannii* complex isolates. (Total No. = 23)

Antimicrobial Agents	<i>A. baumannii</i> No = 13 (56.5%)	<i>A. calcoaceticus</i> <i>baumannii</i> complex N = 7 (39.4%)	*Range µg/ml	
	N (% Susceptible)	N (% Susceptible)	**S	R***
Ampicillin	2 (15.4)	0 (0)	8	32
Piperacillin	8 (61.5)	2 (28.6)	16	128
Ceftazidime	9 (69.2)	2 (28.6)	8	32
Ceftriaxone	3 (23.1)	1 (14.3)	8	64
Cefotaxime	3 (23.1)	1 (14.3)	8	64
Cefuroxime	1 (7.7)	0 (0)	8	32
Azactam	2 (15.4)	0 (0)	8	32
Imipenem	13 (100)	7 (100)	4	16
Gentamicin	9 (69.2)	6 (85.7)	4	8
Amikacin	11 (84.6)	7 (100)	16	64
Ciprofloxacin	9 (69.2)	5 (71.4)	1	4
Tetracycline	6 (46.2)	4 (57.1)	4	16
Trimethoprim / Sulphamethoxazole	10 (76.9)	5 (71.4)	2/38	476

\*National Committee for clinical Laboratory standards MIC interpretation standards for *Acinetobacter*. \*\*S = susceptible, \*\*\*R = Resistant

delineated into different genomic species by DNA-DNA hybridization<sup>[3,11,12]</sup>. Important species include *A. calcoaceticus* (genospecies 1) and *A. baumannii* (genospecies 2). The unnamed genospecies 3 and 13 have a close relationship and similar clinical significance to genomic species 1 and 2 and were included in one complex (*A. calcoaceticus*-*baumannii* complex)<sup>[3,11,12]</sup>. The latter contain isolates that acidify glucose<sup>[3,11,12]</sup>. This new taxonomy that is found in the updated API 20 E profile, has been used in this study and is helpful in the diagnostic laboratory for identifying clinically-significant isolates.

*A. baumannii* is the commonest *Acinetobacter* species isolated from clinical specimens in hospitals and is associated with most hospital outbreaks<sup>[3,7,8,13]</sup>. In a large population study by Wisplinghoff *et al* involving 49 United States hospitals over three years, 166 episodes of *Acinetobacter* BSI were reported<sup>[8]</sup>. Eighty-six percent of *Acinetobacter* isolates were *A. baumannii*. No outbreak similar to our study was noticed<sup>[8]</sup>. Tilley and Roberts reported 52 episodes of *Acinetobacter* BSI during a six-year period, 41 of which were clinically significant<sup>[14]</sup>. Although our study sample was small, it seems comparable to other studies since during the 21-month study period, 40 episodes of *Acinetobacter* BSI have occurred in our hospital with 60% being due to *A. baumannii*, of which 37 (92.5%) were clinically significant. In our study BSI occurred after 19 days of hospital admission in 67.5% of the patients. Several reports agree on the

number of days after which BSI occurs which ranged between 12 to 14 days<sup>[3,7,14]</sup>, although others reported a range between 2-195 days<sup>[8]</sup>.

Many investigators have studied risk factors predisposing to nosocomial acquisition of *A. baumannii*<sup>[3,7,8,14]</sup>. These included male patients prior stay in ICU, malignancy, multiorgan system failure, mechanical ventilation, prior antibiotic use (in particular third generation cephalosporins and aminoglycosides), enteral hyperalimentation and invasive procedures such as tracheostomy, intravenous and urinary catheters. Our results showed that males were slightly more infected by *A. baumannii* and *A. calcoaceticus-baumannii* complex than female patients<sup>[8]</sup>. In contrast more females than males had *Acinetobacter* species ( $p = 0.05$ ). Prior ICU management was more frequently observed among our patients with *A. baumannii* BSI as was shown in other reports also<sup>[3,7,8]</sup>.

In this study, the source of the organism could not be identified in most patients since septic screening at admission to ICU did not recognize specific sites. This is in agreement with the Wisplinghoff *et al* study where in 49% of patients the source of *Acinetobacter* was not known<sup>[8]</sup>. However, the same author found prior colonization with *A. baumannii* as the most significant independent risk factor for *A. baumannii* BSI<sup>[8]</sup>. Intravascular catheters and the respiratory tract were the most common portal of entry for this organism<sup>[8]</sup>. In our study, since 34 (85%) patients had intravascular catheters, it was difficult to evaluate the role of this procedure as an independent risk factor for BSI. However, the role of intravascular catheters in the pathogenesis of *A. baumannii* BSI may be underestimated, if quantitative cultures of catheters are not performed and the clinical criteria for the diagnosis of catheter-related BSI are not employed<sup>[15]</sup>. Catheter-related BSI was defined as occurring in a clinically ill patient when one or more blood cultures taken at least 48 hours after admission yielded a pathogenic organism in the presence of an intravascular catheter, antimicrobial therapy and either a temperature of  $> 38^{\circ} \text{C}$  or  $< 36^{\circ} \text{C}$  with chills, or a systolic blood pressure of  $< 90 \text{ mmHg}$ <sup>[16]</sup>. This is probably true for the 19 mechanically ventilated patients, in whom the role of this factor in the development of *Acinetobacter* infection was not statistically confirmed in this study ( $P = 0.05$ ).

Many investigators have reported on different sources of *A. baumannii* BSI<sup>[3,16-18]</sup>. Urinary and gastrointestinal tracts seemed unimportant sources of *A. baumannii* BSI, since these sites were identified as the portal of entry of other gram-negative pathogens but not *A. baumannii*<sup>[8]</sup>. In contrast, Timsit *et al* reported the digestive tract as an important

reservoir for *A. baumannii* in ICU patients. Strategies to decrease the number of *A. baumannii* in colonized patients in order to decrease the rate of sepsis are needed<sup>[17]</sup>. They also reported on the importance of careful asepsis and improved hand washing to prevent transmission of *A. baumannii* from patient to patient<sup>[17]</sup>. Other studies disregarded the implication of endogenous sources in causing *A. baumannii* BSI<sup>[8]</sup>. Al-Khoja *et al* isolated *Acinetobacter* species from 16 (22.8%) of 70 hospitalized patients compared to six (20%) of 30 healthy staff and stated that when these organisms are isolated from the blood of patients with no clinical evidence of bacteremia, they should be considered as skin contaminants, whilst recognizing the occurrence of occasional bacteremic episodes in compromised patients<sup>[18]</sup>. In our study, *A. baumannii* colonization was noticed in a child in the pediatric ICU following a road traffic accident. Weernink *et al* reported that *A. baumannii* and genomic species 13 (which correspond to *A. calcoaceticus-baumannii* complex) and *A. radioresistance* are commonly found in feather pillows and play a role in nosocomial outbreak of *Acinetobacter* infections<sup>[19]</sup>. There are many reports of hospital epidemics where *Acinetobacter* species were commonly isolated from respiratory equipments, contaminated bedding materials, bath, hospital sink traps, hospital floor swabs and other<sup>[3,4,5,20]</sup>. Therefore, the majority of reports focussed on the ability of *Acinetobacter* to survive and persist at different temperatures and pH values in hospitals<sup>[21]</sup>. Some reports focussed on the seasonal variation in different *Acinetobacter* infections with reports of higher occurrence during July-October compared to November-June periods<sup>[22]</sup>. This could be explained by seasonal variation in humidity and the installation of air-conditioning<sup>[22]</sup>. Our results were similar to those of Tilley and Roberts and Wisplinghoff where no such seasonal variation of *Acinetobacter* BSI was noticed<sup>[8,14]</sup>.

Seven (17.5%) patients in our study ended with fatal outcome although they received imipenem. Six (25%) out of seven were infected with *A. baumannii* and all had serious illnesses as well as being highly immune compromised as mentioned earlier. These patients were elderly females except one male and one premature female infant ( $P = 0.05$ ). Other studies reported 19-37% mortality due to *A. baumannii* and 11 - 14% among non-*A. baumannii* BSI<sup>[7,8,23]</sup>. Death in patients with burns infected with *A. baumannii* BSI reflect severe burn injuries rather than intrinsic virulence of the organism, and this is similar to that seen in 90% of patients with burns in our study<sup>[7]</sup>. Tilley and Roberts reported that patients with malignancy and those with burns do poorly while

trauma patients and patients with other illnesses did relatively well<sup>[14]</sup>. Prognosis also depends on polymicrobial bacteremia<sup>[14]</sup>. This is in accordance with our findings where death occurred in two premature infants who had methicillin resistant *Staphylococcus aureus* in addition to *A. baumannii* BSI and in one patient who had *Pseudomonas aeruginosa* and *A. baumannii* BSI.

*Acinetobacter* have the ability to develop resistance extremely rapidly due to long term exposure to antibiotic-producing organisms in soil environments<sup>[3]</sup>. Conjugation plays a significant role in antibiotic resistance gene transfer between *Acinetobacter* species<sup>[24]</sup>. As seen in this study, 26 (65%) of our patients were exposed to antibiotics including cephalosporins, aminoglycosides and ciprofloxacin. *Acinetobacter* spp. were resistant to most antibiotics tested and there were similar reports of increasing resistance of *Acinetobacter* to aminoglycosides and ciprofloxacin from Germany and France respectively<sup>[3,25]</sup>. The most worrying is the emergence of *A. baumannii* resistant to imipenem<sup>[26]</sup>. Fortunately most reports, including ours, showed low resistance to imipenem<sup>[8,27]</sup>.

## CONCLUSION

Our study has documented that *A. baumannii* is the most common *Acinetobacter* species causing BSI observed among patients in ICUs. Serious underlying illnesses predisposing to *Acinetobacter* BSI include cardiac and renal diseases, premature infants and severe burn patients. These patients had a high mortality. Lack of significant statistical association among the various risk factors studied does not underestimate their importance as most of these factors overlapped in these patients. Extensive in-vitro susceptibility testing of -lactam antibiotics, aminoglycosides, and quinolones is needed for *Acinetobacter* isolates from BSI to monitor the emergence of resistance to commonly used antibiotics. Control of antibiotic use and intense surveillance of *A. baumannii* at high-risk areas in the hospital and effective infection control measures are strongly needed to counter nosocomial BSI.

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