Incidence and Prevalence of *Acinetobacter baumannii* in King Fahd General Hospital, Saudi Arabia

Saad B. Al Masoudi[1], Magda M. M. Aly[1,2*], Noha Q. Al humidi[1], Muhammad A. Halwani[3]

[1]Biology Department, Faculty of Science, King Abdulaziz University, P.O. Box 80203, Jeddah 21589, Saudi Arabia,
[2]Botany Department, Faculty of Science, [3]Microbiology Department, Faculty of Medicine, Al Baha University
magdammali@hotmail.com

Abstract: The prevalence of multi-drug resistant (MDR) *Acinetobacter* that is causing patient infections at the King Fahad General Hospital, Jeddah, Saudi Arabia was studied. 104 bacterial isolates were collected in six month period, from April to December 2010 from 102 patients with various specimens including sputum, wound, urine; blood; cerebrospinal fluid and other locations (sites). Regardless of the specimen, there was a high rate of nosocomial MDR *Acinetobacter* spp. isolated from Intensive Care Unit (ICU). Of the 104 isolates of *Acinetobacter* spp., 88.5% were identified as *A. baumannii* and 11.5% were other genospecies. The largest predominate antibiotype contained a total of 32 MDR isolates (resistant to quinolones, cephalosporins and carbapenems), including 29 *A. baumannii* (Ab) and 3 isolates were other genospecies. Respiratory tract specimens (sputum) represented nearly 72.2% of all the specimens collected in the ICU. Generally, *Acinetobacter* was isolated from various types of infection, specially respiratory tract infection (27.8%), followed by urinary tract infections (20.8%), *Acinetobacter* ventilator-associated pneumonia (11.1%), blood stream infections, burn wound infection and surgical site infection (9.7%), skin and soft tissues infection (6.9%), meningitis (1.4%) and other infection (6.2%).This study help taking effective measures for controlling *Acinetobacter* and data could be used in future as medical reference.
[Al Masoudi SB, Aly MM, Al humidi NQ, Halwani M. **Incidence and Prevalence of *Acinetobacter baumannii* in King Fahd General Hospital, Saudi Arabia.** *Life Sci J* 2013;10(4):1702-1710] (ISSN:1097-8135). http://www.lifesciencesite.com. 225

Keywords: *Acinetobacter*, genospecies, multi-drug resistant, King Fahd General Hospital

1. Introduction

Nowadays, the genus *Acinetobacter* is increasingly being implicated in many outbreaks in hot and humid climates and has become a growing concern in many hospitals (Bergogne-Berezin and Towner, 1996). It recognized to be among the most difficult antimicrobial-resistant Gram-negative bacilli to control and treat. The most frequently reported species of the genus *Acinetobacter* in the literature *are Acinetobacter baumannii, A. calcoaceticus,* and *A. lwoffii*. It is difficult to differentiate among the previous species on the basis of phenotypic characteristics, thus the term *A. calcoaceticus–A. baumannii* complex can be used (Schreckenberger *et al.,* 2007).

Acinetobacter can survive under a wide range of environmental conditions and can persist for extended periods of time on many surfaces make it a frequent cause of outbreaks of infection and health care–associated pathogen (Fournier and Richet, 2006, Jawad et al., 1996). Outbreaks of infection have been traced to respiratory care equipment, wound care procedures, humidifiers and patient care items. In humans, *Acinetobacter* can colonize skin, wounds, and respiratory and gastrointestinal tracts causing infections, particularly in intensive care units (ICUs), and sometimes cause community-acquired pneumonia (Munoz-Price and Weinstein, 2008). Wilks *et al.* (2006) reported a recent outbreak of multidrug-resistant *Acinetobacter* infection, with environmental contamination found on curtains, laryngoscope blades, patient lifting equipment, door handles, mops, and keyboards. Further investigation of the efficacy and cost-effectiveness of various infection control strategies to prevent transmission of multidrug-resistant *Acinetobacter* infection are needed.

Resistance to multiple antibiotics is a frequent finding with *A. baumannii,* which is now recognized to be of great clinical importance. However, many other *Acinetobacter* spp. are also responsible for nosocomial infections, numerous reports implicates *A. baumannii* as a major pathogen involved in nosocomial infections causing endemic and epidemic infections. It survives for prolonged periods under a wide range of environmental conditions (Munoz-Price et al., 2006) and caused many infections and health care–associated infections including, pneumonia, bacteremia, meningitis, urinary tract and wound infection. Antimicrobial resistance greatly limits the therapeutic options for patients who are infected with this organism, especially if isolates are resistant to carbapenem group of antibiotics. Because therapeutic options are limited for multidrug-resistant *Acinetobacter* infection, the development or discovery of new therapies, well-controlled clinical trials of existing antimicrobial regimens and combinations, and greater

emphasis on the prevention of health care–associated transmission of multidrug-resistant *Acinetobacter* infection are essential.

Risk factors for colonization or infection with multidrug-resistant *Acinetobacter* species include prolonged length of hospital stay, exposure to an intensive care unit (ICU), receipt of mechanical ventilation, colonization pressure, exposure to antimicrobial agents, recent surgery, invasive procedures, and underlying severity of illness (Fournier and Richet, 2006, Playford et al., 2007).

King Fahd general Hospital (KFGH) is one of the most important hospitals in Jeddah, serves a large number of population of the region. It was opened in 1973 and had bed capacity of 600 beds, distributed over different sections. In KHGH. *A. baumannii* increasingly recognized as a cause of hospital-acquired outbreaks, particularly in the ICU and represents a particular infection control problem, in the most vulnerable in-patients. Information on epidemiological investigation of outbreaks caused by *Acinetobacter* in KFGH is lacking**.** The aim of this study was to study the occurrence of *A. baumannii* from KFGH, Jeddah, Saudi Arabia.

2. Material and Methods

The study was approved by Research Ethics Committee, Health Ministry, Jeddah, in June 2010. Patient consents were not obtained because data were analyzed anonymously.

Bacterial isolates

Isolates of *Acinetobacter* spp. were collected from KFGH, Jeddah; Saudi Arabia during 6 months from April to September 2010 at Clinical Microbiology Laboratory. The isolates were obtained from clinical specimens of the different units and were maintained on both slopes of Nutrient agar at 4°C (Dadgar *et al.*, 2006) and in 20% Glycerol Broth at -70°C (Zarrilli *et al.*, 2007).

Culturing of the isolates

The isolates were cultured on 5% Sheep blood and LAM agar which is a selective medium for *Acinetobacter* isolation and differentiation. Incubated was carried out at 35°C for 24 h.

Patient data collection

Patient data including age, gender, diagnosis, date of admission, date of acquisition of *Acinetobacter* spp. and previous infected were collected from patient files, Hospital Electronic Data Bases and Infection Control Department.

Identification and susceptibility test by Phoenix System

The bacterial inoculums were prepared from nutrient agar slant of the pure culture, grown at 35 °C for 24 hours. Identification of the bacterial isolate and sensitivity to antibiotics were obtained using Phoenix Spec™ Nephelometer (BD Diagnostic Systems) at Microbial Laboratory, KFGH. Identification was also confirmed after growth on LAM (Hardy Diagnostics, USA, catalog No. G261).

Nosocomial infections

Nosocomial infections were classified according to standard CDC.

Statistical analyses

All statistical analyses were performed using SPSS version 20.0. Analysis was performed by non-parametric test for categorical variables, Chi-square, Fisher's exact, Mann-Whitney, Kruskal-Wallis, Student t test and ANOVA for continuous variables were used. All variables test with 0.05 of significance level (P value $\leq$ 0.05) were considered significant.

3. Results

From April to September 2010, out of 102 patients admitted to the hospital, 104 *Acinetobacter* isolates were identified at Microbiology Lab. from different samples, obtained from different units.

On blood agar medium, the isolates appeared as non haemolytic mucoid colonies with diameter of 2 - 3 mm (Fig. 1A). On MacConkey agar, they had light lavender color and were non- lactose ferments (figure 1B). However, they produced a pink diffused pigment and mucoid colonies on Leeds *Acinetobacter* medium (LAM) as shown in Figure 1C. *Acinetobacter* isolates are Gram negative bacteria and examination under scanning electron microscope, they are short rod during logarithmic phase and more coccoid in the stationary phase (Fig. 2). Out of 104 isolates of *Acinetobacter* spp., 92 isolates were identified as *Acinetobacter baumannii* (Ab) and 12 isolates were belonging to other species (non Ab).

The highest *Acinetobacter* isolates were obtained from sputum (n=41, 39.42%) followed by wound (n= 32, 30.8%); urine (n=19, 18.3%), blood, (n=6, 5.8%), and cerebrospinal fluid (Table 1). About 95%, 85 % and 84% of the isolates from sputum, wound and urine, respectively were identified as *Acinetobacter baumannii*.

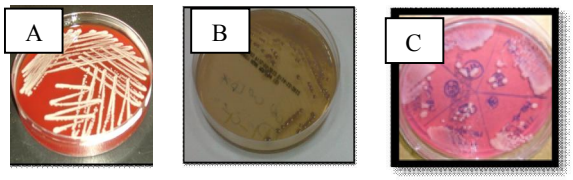

Figure 1. *Acinetobacter baumannii* cultured on blood agar (A), MacConkey agar (B) and Leeds *Acinetobacter* medium (LAM).

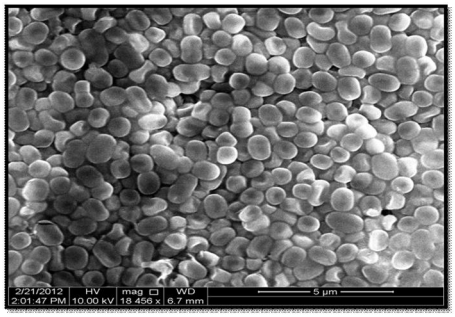

Figure. 2. *Acinetobacter baumannii* grown on Blood agar for 24 h. and examined under scanning electron microscope

Table 1. Distribution and percentages of *Acinetobacter* spp, isolated from different sites of patient at KFGH

Site	Biotype		Count (%)
	Ab (%)	Non *Ab* (%)	
Sputum	39 (95)	2(5)	41
Wound	26 (81)	6(19)	32
Urine	16 (84)	3(16)	19
Blood	6 (100)	0	6
Cerebrospinal fluid	1 (100)	0	1
Others	4 (80)	1 (20)	5
Total	92 (88.5)	12 (11.5)	104

Ab: Acinetobacter baumannii; *Non Ab:* Species other than *baumannii*.

Acinetobacter isolates were from patients at different Service Units including ICU, Burn unit, General surgery, orthopedic unit, Medical unit, Urology and Special Ward unit (Table 2). The highest incidence of *Acinetobacter* spp. was recorded in ICU (Table 2) where 36 isolates were recovered, followed by Burn unit and Medical unit, where 18 and 16 isolates were obtained, respectively. At ICU, 97% of the isolated were identified as *A. baumannii*. Table 3 showed that most of *Acinetobacter* isolates (26 isolates) were recovered from the respiratory tract samples, collected from ICU, followed by wound samples collected from Burn unit (13 isolates). Statistical analysis showed that there is a highly significant difference between Service Units and site of isolation using Fisher's Exact test ($P \leq 0.05$).

Table 2. Distribution and percentage of the identified *Acinetobacter sp.* obtained from different Service Units at KFGH.

Service units	Biotype (*Acinetobacter sp.*)		Count (%)
	Ab (%)	Non *Ab* (%)	
ICU	35 (97.2)	1 (2.8)	36
Burn unit	13 (72.2)	5(27.8)	18
General surgery	9 (81.8)	2(18.2)	11
Orthopedics	11 (84.6)	2(15.4)	13
Medical	15 (93.75)	1(6.25)	16
Urology	2 (66.7)	1(33.3)	3
MRC	1 (100)	0	1
ENT	5 (100)	0	5
Special Ward	1 (100)	0	1
Total	92	12	104

ICU: Intensive care unit; MRC: Medical rehab center; CSF: Cerebrospinal fluid; ENT: Ear, nose and throat; *Ab: Acinetobacter baumannii*; *Non Ab:* Species other than *baumannii*

Antimicrobial susceptibility test was determined of the *Acinetobacter* isolates for the panel of 12 antibiotics (Amikacin; Cefepime; Cefotaxime; Ceftazidime; Ciprofloxacin; Colistin; Gentamicin; Imipenem; Meropenem; Piperacillin-Tazobactam; Trimethoprim- Sulfamethoxazole; Tigecycline) were determined using microdilution method. *Acinetobacter* isolates would consider multidrug resistant if they resist three or more antibiotics. Out of 104

Acinetobacter spp., 92 isolates was identified as *A. baumannii* of them 85 were multidrug resistance isolates, especially to quinolones, cephalosporins and carbapenems and 7 fully sensitive isolates. Species other than *baumannii* were 12 isolates including 10 multidrug resistance and 2 fully sensitive isolates (tables 4). Statistical analysis showed that there is no significant association between type of resistance and biotype using Chi-Square test ($P \geq 0.05$).

Table 3. Distribution of *Acinetobacter* spp., isolated from different specimen collected from different Service Units at KFGH.

Service Units	Sputum	Wounds	Urine	Blood	CSF	Other	Total
ICU	26 (72.2%)	0	5 (13.95)	3 (8.3%)	0	2 (5.6%)	36
Burn unit	3 (16.7%)	13 (72.2%)	2 (11.1%)	0	0	0	18
General surgery	2 (18.2%)	6 (54.5%)	3 (27.3%)	0	0	0	11
Orthopedics	1(7.7%)	7 (53.8%)	4(30.8%)	0	0	1 (7.7%)	13
Medical	6 (37.5%)	3 (18.8%)	4(25.0%)	2 (12.5%)	0	1 (6.2%)	16
Urology	1 (33.3%)	1 (33.3%)	1(33.3%)	0	0	0	3
MRC	1 (100%)	0	0	0	0	0	1
ENT	1 (20.0%)	1 (20.0%)	0	1 (20.0%)	1 (20.0%)	1 (20.0%)	5
Special Ward	0	1 (100%)	0	0	0	0	1
Total	41(99.8)	32	19(100.1)	6	1	5	104

ICU: Intensive care unit; MRC: Medical rehab center; CSF: Cerebrospinal fluid; ENT: Ear, nose and throat.

Table 4. Resistance and sensitivity (MDR and FS) of *Acinetobacter* isolates, obtained from clinical samples from KFGH.

Biotype (n)	MDR (%)	FS (%)
Ab (92)	85 (89.5)	7(77.8)
Non Ab (12)	10 (10.5)	2 (22.2)
Total (104)	95	9

MDR: Multidrug resistant; FS: Fully sensitive; Ab*:* *Acinetobacter baumannii*; Non Ab*:* Species other than *baumannii*.

The results in table 5 showed that 41 isolates were from sputum including 39 multidrug resistant (MDR) isolates and 2 fully sensitive (FS); 32 isolates were from wound including 31 MDR and one isolate FS. The isolates from urine were 19 including 15 MDR isolates and 4 FS while the isolates from blood were 6 including 4 MDR and 2 isolates FS. Only one isolate was from cerebrospinal fluid and was MDR. The isolates from other locations were 5 including only MDR. Conclusion, 95 bacterial isolates were MDR and 9 isolates were FS. Statistical analysis showed that there is no association between isolation site and antimicrobial susceptibility of *Acinetobacter* ($P > 0.05$).

Infections or colonization of the patients was evaluated (Table 6). Majority of the collected cases were from patients admitted to ICU. The patients age ranged from 12 months to 92 years (mean age 48 ± 20 years). Among these, 61 were male (58.7%) and 43 were female (41.3%). About infection and age (Category), there is no significant association ($P > 0.05$, using Chi-Square test) but in fact there is low significant different between infection and age using ANOVA test. The different comes between 2 subgroup (Infection & Colonization) with $P = 0.041$ which means that there is very low impact of infection on age.

Out of 61 male patients, 37 patients were infected and 13 colonized. Moreover, 35 female patients were infected and one colonized. A total of 65 infections were with MDR *Acinetobacter* and 7 infections were with fully sensitive *Acinetobacter*. There is significant different between male and female infection (Table 6).

The mean day of isolation (DOI) was 19 ± 21 days. There is significant association between day of isolation and infection which means that most of infections were obtained in early days ($DOI \leq 19$). Majority of the collected cases were from patients admitted at ICU. Twenty nine admitted patients at ICU were infected but 2 admitted patients at ICU were colonized. In addition, 10 admitted patients at Burn unit and Medical unit were infected.

The results showed that thirty five diabetic and 37 non diabetic patients were infected in addition to 7 diabetic patients were colonized. Moreover, thirteen patients had previous *Acinetobacter* infection while 59 were with no previous infection. Unfortunately, 54 patients died, mortality rate 52%, during their stay at the hospital (Table 6). From previous data, there is no significant association or difference between infection and the previous variables.

Table 7 showed the type of infection in each Service unit and biotype of *Acinetobacter* (either Ab or non Ab). Seventy two *Acinetobacter* spp. was isolated from various types of infections, 20 (27.8%)

isolates were from Respiratory tract infections, 7 (9.7%) were from either blood stream, burn wound or surgical site infection, one (1%) from meningitis infection, 15 (20.8%) from urinary tract infections, 8 (11%) were from *Acinetobacter* ventilator-associated pneumonia (VAP), 5 (6.9%) from skin & soft tissues infection (SSTI) and 3 (4%) were from other infection (Table 7). *A. baumannii* was the main species responsible for most of the infections where 64 *A. baumannii* and 8 other species were obtained from different infection type. Species other than *A. baumannii* (non Ab) were also responsible for many infections. Concerning *A. baumannii*, there is significant association between type of infection and service unit (P = 0.000, Fisher's Exact test) but in case of non Ab, there is no significant association (P > 0.05, Fisher's Exact test).

Table 5. Antimicrobial susceptibility of *Acinetobacter* spp, isolated from different sites

Sensitivity / Site	MDR (%)	FS (%)	Total isolates
Sputum	39 (95)	2 (5)	41
Wound	31 (97)	1 (3)	32
Urine	15 (79)	4 (21)	19
Blood	4 (67)	2 (33)	6
CSF	1 (100)	0	1
Other	5 (100)	0	5
Total	95 (91)	9 (9)	104

CSF: Cerebrospinal fluid; MDR: Multidrug resist; FS: Fully sensitive

Table 6. Demographic data of 104 *Acinetobacter* isolates, collected from King Fahd General Hospital

Prognostic factors		Infection (n=72)	Colonization (n= 14)	Community (n=14)	P value
Age	0 to ≤ 15	1	1	2	0.17
	>15 to ≤ 30	14	4	1	
	>30 to ≤ 45	15	5	5	
	>45 to ≤ 65	24	3	4	
	> 65	18	1	2	
Gender	Male	37	13	8	0.016*
	Female	35	1	6	
Biotype	Ab	64	13	13	0.83
	Non Ab	8	1	1	
Antibiotype	MDR	65	14	13	0.46
	FS	7	0	1	
Day of isolation (DOI)	≤19	43	9	14	0.01*
	>19	29	5	0	
Hospital Service	ICU	29	2	4	0.21
	Burn unit	10	5	2	
	General surgery	9	1	1	
	Orthopedics	7	4	2	
	Medical	10	1	4	
	Urology	2	0	0	
	ENT	4	0	1	
	MRC	1	0	0	
	Special Ward	0	1	0	
Diabetic patient	Yes	35	7	9	0.56
	No	37	7	5	
Previous *Acinetobacter* infection	Yes	13	0	3	0.20
	No	59	14	11	
Outcome	Expired	41	4	7	0.14
	Survived	31	10	7	

Ab: Acinetobacter baumannii; Non *Ab:* Species other than *baumannii*, ; MDR: Multidrug resist; FS: Fully sensitive; ICU: intensive care unit; MRC: medical rehab center**;** ENT: ear, nose and throat.* Significant association

Table 7. Types of *Acinetobacter* infection in different Service units at KFGH.

Type of infection	*Acinetobacter baumannii* (Ab)			(non Ab)			Total isolates
	Total no.	Number	Unit	Total no.	Number	Unit	
***Acinetobacter* ventilator-associated pneumonia**	8	6	ICU	0	0	0	**8 (11. 1)**
		1	Med				
		1	Uro				
Respiratory Tract Infection	19	13	ICU	1	1	ICU/	**20 (27.8)**
		1	G.S				
		1	MRC				
		2	Med				
		1	Ortho				
		1	B.U				
Burn wound infection	5 (71.4)	-	B.U	2	BU	B.U	**7 (9.7)**
Surgical site Infection	5 (71.4)	2	G.S		1	G.S	**7 (9.7)**
		1	ENT				
		1	Ortho		1	Orth o	
		1	Uro				
Skin and Soft Tissues Infection	4 (80)	2	Med	2	1	G.S	**5 (6.9)**
		1	GS		1	Orth o	
		1	Ortho				
Urinary tract infection	13	4	ICU	2(13.3)	2	BU	**15 (20.8)**
		3	G.S				
		3	Ortho				
		3	Med				
Blood stream infection (BSI)	6	3	ICU	0	0	0	**7 (9.7)**

4. Discussions

Over the last decade, *Acinetobacter* has emerged to become an important cause of nosocomial infections in many parts of the world (Peleg *et al*., 2008). Recently, resistance problem has been relatively much worsened in Gram-negative bacilli specially *Acinetobacter* which have increasingly been resist many antimicrobial agents (Fournier and Richet, 2006). *Acinetobacter* is typical nosocomial pathogens causing infections and high mortality, almost exclusively in compromised hospital patients. They are intrinsically less susceptible to antibiotics than the members of the family Enterobacteriaceae, and have propensity to acquire resistance (Lee *et al*., 2011). Moreover, *A. baumannii* causes a significant number of nosocomial outbreaks worldwide, which commonly occur in settings with high antibiotic selective pressures (Mak *et al*., 2009). In this study, 104 *Acinetobacter* isolates were recovered from 102 patients at KFGH in six months. *Acinetobacter baumannii,* clinically the most important of *Acinetobacter* genospecies, was the main species (92 isolates). More isolates were obtained by Dent *et al* (2010) where 247 isolates of *Acinetobacter baumannii* were obtained from 164 patients at USA Hospital. In another studies, 96 isolates of *A. baumannii* were obtained from 25 hospitals in 17 European countries from 2001-2004 (Towner *et al*., 2008) and 35 clinical isolates of MDR *A. baumannii* were collected from 3 Korean University hospitals over a 2 years period (Koo *et al*., 2010).

Leeds *Acinetobacter* medium was used as a specific medium for *A. baumannii* growth and identification. Many previous studies have employed Leeds *Acinetobacter* medium for environmental surveillance of *A. baumannii* (Barbolla, *et al*., 2008, Jawad, *et al*., 1994, Webster *et al*., 1998). The most isolates of *A. baumannii* were recovered from respiratory specimens (sputum) followed by wound specimens. These findings were in accordance with the results reported by other investigators from Italy and Finland (Esposito and Leone, 2007; Ylipalosaari *et al*., 2006). Also, this findings consistent with another Saudi study (Saeed *et al*., 2010), Hong Kong study (Ho *et al*., 2010), Taiwan study (Chang *et al*., 2009). Moreover, Park *et al*. (2010) showed that the most common *Acinetobacter* isolates were the respiratory isolates which may due to the tools and instruments used or respiratory infections. In addition, *A. baumannii* has the ability to live on dry environmental surfaces for up to 13 days which were more than that of other Gram-negative bacteria (Getchell-White *et al.,* 1989). On contrast, another study conducted between 2003 and 2005 in Warsaw in Poland, reported that the predominantly *A. baumannii* isolates were obtained from blood samples (Wroblewska *et al*., 2007).

In this study, the majority of *Acinetobacter* spp. was isolated from the Intensive Care Unit (ICU). These results are in consistent with that reported by Saeed *et al*. (2010), Prashanth and Badrinath (2005) and Hawkey and Finch (2007). *A. baumannii* outbreaks have been reported previously, particularly in ICU by many authors (Corbella *et al*., 2000; Pournaras *et al*., 2006). Severe underlying diseases, invasive diagnostic and therapeutic procedures used in ICUs have been demonstrated to predispose patients to severe infections with *A. baumannii* (Husni *et al*., 1999; Tejada *et al*., 2001; Raka *et al*., 2009). Lower numbers of *A. baumannii* were obtained from ICU in Poland by Wroblewska *et al*., (2007) which may due to the lower number of beds found in the ICU at this hospital. Our results were in accordance with studies in Indian that showing *Acinetobacter* isolation from different types of infection in hospitals and the highest infection was respiratory infection specially at ICU (Prashanth and Badrinath, 2005). Also, respiratory infection due to *Acinetobacter* in mechanically ventilated patients in ICU was also high at JIPMER hospital (Prashanth and Badrinath, 2007). However, Van Looveren *et al*. (2004) reported that multi-resistant strains of *Acinetobacter* spp. were isolated from hospitalized patients with bacteraemia, pneumonia, meningitis, urinary tract infections and surgical wound infections. Our results showed that out of 92 *A. baumannii*, 85 isolates were MDR isolates and 7 were fully susceptible to antimicrobial agents, while 95-97 % of the *Acinetobacter* isolates from sputum or wound were MDR. Shakibaie *et al*. (2012) found that *Acinetobacter* spp. have acquired resistance to almost all currently available antimicrobial agents, including aminoglycosides, quinolones, and broad-spectrum β-lactams. The spectrum of antibiotic resistance of these organisms, together with their survival capabilities, makes them a threat in hospital environments. Rates of carbapenem resistance in 3601 isolates of *A. baumannii*, clinically the most important of 25 *Acinetobacter* genospecies, increased from 9% in 1995 to 40% in 2004 (Carey *et al*., 2006). The nosocomial *Acinetobacter* infections were about 70 % whereas 15% were colonization and 15% were community acquired infection. In Spain, one-third of patients were colonized during their stay, with the trachea (43%), rectum (31%), and skin (35%) being the most frequent sites and 92% of cases, colonization was established within the first 9 days after admission (Martínez-Pellús et al., 2002).

Almost all cases of MDR *A. baumannii* in our study were hospital acquired, 88% were acquired in KFGH during the index hospitalization and 10% were imported into the hospital by patients with recent exposure to the healthcare system. The highest rate of *Acinetobacter* spp. on respiratory tract infection was 27.8%, most of them occurred in ICU. In Spain, Martínez-Pellús *et al.* (2002) found significant risk factors included mechanical ventilation ($p < 0.01$) and *Acinetobacter* was recovered from thermometers (35%), respirator switches (43%), and damp surfaces (54%). Falagas et al. (2008) reported that according to a meta-analysis, *Acinetobacter* was a more common cause of ICU acquired pneumonia in studies originating from European countries (0–35%) and Asian (range 4-44%) than those of United States (6-11%). A gradient in the proportion of ICU acquired pneumonias caused by *Acinetobacter* in various European countries was apparent where the rates were very low in Scandinavia and became gradually higher in Germany and United Kingdom and the highest rates were reported for Turkey, France, Spain, Italy and Greece (Falagas *et al*., 2008). During March - July 2006, *A. baumannii* was isolated from 30 patients, of whom 22 were infected and 8 were colonized (Raka *et al*., 2009). These results are in consistent with our result. However, *A. baumannii* was isolated from 74 patients, out of them 38 were infected (33 had ventilator-associated pneumonia) and 36 were colonized (Zarrilli *et al*., 2007).

In conclusion, high incidence rate of *Acinetobacter* was reported in KFGH and colonization appeared in 15 % of patients, this event generally precedes infection. The use mechanical ventilation and day of isolation are the main factors affect isolation. The main bacterial reservoirs are the colonized patient in addition to environmental elements.

Acknowledgements:

Thanks and appreciation to King Abdul-Aziz City for Science and Technology, Saudi Arabia, for financial support to carry out this work.

Corresponding Author:
Dr Magda M. Aly
Department of Biology, Faculty of Scince, King Abdulaziz University, Saudi Arabia
E-mail: magdammali@hotmail.com

References

1. Stirilng G, Wilsey B. Emprical relationships Barbolla R. E., et al. 2008. Molecular epidemiology of *Acinetobacter baumannii* spread in an adult intensive care unit under an endemic setting. Am. J. Infect. Control 36: 444–452.
2. Bergogne-Berezin E and Towner KJ. (1996). *Acinetobacter* spp. as Nosocomial pathogens: Microbiological, clinical, and epidemiological features. Clin. Microbiol. Rev., 9(2):148-165.
3. Carey RB, Banerjee SN, Srinivasan A. (2006). Multidrug-resistant *Acinetobacter* infections,

1995-2004. Presented at the 46th Interscience Conference on Antimicrobial Agents and Chemotherapy, San Francisco, September 27–30.
4. Corbella X, Montero A, Pujol M, Dominguez A, Ayats J, Argerich J, Garrigosa F, Ariza J, and Gudiol F. (2000). Emergence and rapid spread of carbapenem resistance during a large and sustained hospital outbreak of multiresistant *Acinetobacter baumannii*. J Clin Microbiol, 38: 4086–4095.
5. Dadgar T, Asmar M, Saifi A, Mazandarani M, Bayat H, Mordi A, Bazueri M, and Ghaemi E. (2006). Antibacterial Activity of Certain Iranian Medicinal Plants against Methicillin-Resistant and Sensitive *Staphylococcus aureus*, Asian Journal of Plant Sciences., 5(5): 861-866.
6. Dent L L, Marshall D R, Pratap S and Hulette R B (2010). Multidrug resistant Acinetobacter baumannii: a descriptive study in a city hospital. Infectious Diseases, 10:196.
7. Esposito S, Leone S. (2007). Antimicrobial treatment for intensive care unit (ICU) infections including the role of the infectious diseases specialist, Int. J. Antimicrob. Agents, 29: 494–500.
8. Falagas ME, Karveli EA, Siempos I, and Vardakas KZ. (2008). *Acinetobacter* infections: a growing threat for critically ill patients. Epidemiol. Infect., 136:1009–1019.
9. Fournier PE, Richet H. (2006). The epidemiology and control of *Acinetobacter baumannii* in health care facilities. Clin Infect Dis; 42:692-9.
10. Getchell-White SI, Donowitz LG, Gröschel DH. (1989). The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of Acinetobacter calcoaceticus. Infect Control Hosp. Epidemiol., 10 (9):402-407.
11. Hawley JS, Murray CK, Griffith ME, McElmeel ML, Fulcher LC, Hospenthal DR, and Jorgensen JH. (2007). Susceptibility of *Acinetobacter* strains isolated from deployed U.S. Military Personnel. Antimicrob. Agents Chemother., 51(1): 376–378.
12. Ho PL, Ho AY, Chow KH, Lai EL, Ching P, Seto WH. (2010) Epidemiology and clonality of multidrug-resistant *Acinetobacterbaumannii* from a healthcare region in Hong Kong. J. Hospital. Infect., 74: 358–364.
13. Husni RN, Goldstein LS, Arroliga AC, Hall GS, Fatica C, Stoller JK, Gordon SM. (1999). Risk factors for an outbreak of multi-drug-resistant *Acinetobacter* nosocomial pneumonia among intubated patients. Chest, 115(5):1378-1382.
14. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM, Hawkey PM (1996). Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. J Clin Microbiol; 34: 2881-2887.
15. Koo S H, Kwon K C, Cho H H, and Sung J Y. (2010). Genetic Basis of Multidrug-resistant *Acinetobacter baumannii* Clinical Isolates from Three University Hospitals in Chungcheong Province, Korea. *Korean J Lab Med*; 30:498-506.
16. Lee K, Yong D, Jeong SH, Chong Y. (2011). Multidrug Resistant *Acinetobacter* spp.: Increasingly problematic Nosocomial pathogens, Yonsei Med J, 52(6):879-891.
17. Mak JK, Kim M, Pham J, Tapsall J and Peter A. (2009). Antibiotic resistance determinants in nosocomial strains of multidrug-resistant *Acinetobacter baumannii*. J. Antimicrobial Chemotherapy, 63: 47–54.
18. Martínez-Pellús A, Ruiz Gómez J, Jaime Sánchez F, Simarro Córdoba E, Fernández Lozano JA. (2002). Incidence of colonization and infection by *Acinetobacter baumannii* in an endemic seting (ICU). Analysis of risk factors by means of a surveillance study. Enferm Infecc Microbiol Clin., 20 (5):194-199.
19. Munoz-Price L S and. Weinstein R A (2008). *Acinetobacter* infection. The New England Journal o f Medicine; 358:1271-1281.
20. Munoz-Price LS, Baig MO, Lavin MA, (2006). Clinical features and outcomes of Imipenem resistant (Imi-R) *Acinetobacter baumannii* (Ab) bloodstream infections (BSI). Presented at the 46th Interscience Conference of Antimicrobial Agents and Chemotherapy, San Francisco, September 27–30.
21. Peleg AY, Seifert H, and David L. Paterson DL. (2008). *Acinetobacter baumannii*: Emergence of a successful pathogen. Clinical Microbio. Rev., 21(3): 538-582.
22. Playford EG, Craig JC, Iredell JR. Carbapenem-resistant *Acinetobacter baumannii* in intensive care unit patients: risk factors for acquisition, infection and their consequences. J Hosp Infect 2007; 65: 204-211.
23. Pournaras S, Markogiannakis A, Ikonomidis A, Kondyli L, Bethimouti K, Maniatis AN, Legakis NJ, and Tsakris A. (2006). Outbreak of multiple clones of imipenem-resistant *Acinetobacter baumannii* isolates expressing OXA-58 carbapenemase in an intensive care unit. J. Antimicrob Chemother., 57: 557–561.
24. Prashanth K, and Badrinath S. (2005). Epidemiological investigation of nosocomial *Acinetobacter* infections using arbitrarily primed

PCR & pulse field gel electrophoresis. Indian J Med Res, 122: 408–418.
25. Prashanth K, and Badrinath S. (2007). Nosocomial infections due to *Acinetobacter* species: clinical findings, risk and prognostic factors.Indian J. Medical Microbiol., 24 (1):39–44.
26. Raka L, Kalenc S, Bosnjak Z, Budimir A, Katic S, Sijak D, Mulliqi-Osmani1 G, Zoutman D, and Jaka A. (2009). Molecular Epidemiology of *Acinetobacter baumannii* in Central Intensive Care Unit in Kosova Teaching Hospital. The Brazilian J. Infectious Diseases, 13(6):408-413.
27. Saeed NK, Kambal AM, El-Khizzi NA. (2010). Antimicrobial-resistant bacteria in a general intensive care unit in Saudi Arabia. Saudi Med. J., 31 (12):179-187.
28. Schreckenberger PC, Daneshvar MI, Weyant RS, Hollis DG. (2007). *Acinetobacter, Achromobacter, Chryseobacterium, Moraxella*, and other non fermentative gram negative rods. In: Murray PR, Baron EJ, Jorgensen JH, Landry ML, Pfaller MA, eds. Manual of clinical microbiology. 9th ed. Washington, DC: ASM Press,:770- 802.
29. Shakibaie M R, Adeli S and Salehi MH. (2012). Antibiotic resistance patterns and extendedspectrum b-lactamase production among *Acinetobacter* spp. isolated from an intensive care Unit of a hospital in Kerman, Iran. *Antimicrobial Resistance and Infection Control*, 1:1 http://www.aricjournal.com/1/1/1
30. Tejada AA, Bello DS, Chacon VE, Munoz MJ, Villuendas UMC, Figueras P, Suarez FJ, Hernandez A. (2001). Risk factors for nosocomial pneumonia in critically ill trauma patients. Crit. Care Med., 29(2):304–309.
31. Towner KJ, Levi K and Vlassiadi M. (2008). Genetic diversity of carbapenem-resistant isolates of *Acinetobacter baumannii* in Europe. Clin Microbiol Infect; 14(2): 161–167.
32. Van Looveren M, Goossens H; ARPAC Steering Group. (2004). Antimicrobial resistance of *Acinetobacter* spp. in Europe. Clin Microbiol Infect. Vol. 10 (8): 684-704.
33. Webster C. A., Crowe M., Humphreys H., Towner K. J. 1998. Surveillance of an adult intensive care unit for long-term persistence of a multi-resistant strain of *Acinetobacter baumannii*. Eur. J. Clin. Microbiol. Infect. Dis. 17: 171–176
34. Wilks M, Wilson A, Warwick S, Price E, Kennedy D, Ely A, Millar MR (2006). Control of an outbreak of multidrug-resistant *Acinetobacter baumannii*-calcoaceticus colonization and infection in an intensive care unit (ICU) without closing the ICU or placing patients in isolation. Infect Control Hosp Epidemiol. 27(7):654-658.
35. Wroblewska MM, Towner KJ, Marchel H, and Luczak M. (2007). Emergence and spread of carbapenem-resistant strains of *Acinetobacter baumannii* in a tertiary-care hospital in Poland. Clin Microbiol Infect., 13: 490–496.
36. Ylipalosaari P, Ala-Kokko TI, Laurila J, Ohtonene P, Syrjala H. (2006). Epidemiology of intensive care unit (ICU)-acquired infections in a 14 month prospective cohort study in a single mixed Scandinavian University Hospital ICU. Acta Anaesthesiol Scand., 50: 1192–1197.
37. Zarrilli R, Casillo R, Popolo AD, Tripodi MF, Bagattini M., Cuccurullo S, Crivaro V, Ragone E, Mattei A, Galdieri N, Triassi M and Utili R. (2007). Molecular epidemiology of a clonal outbreak of multidrug-resistant *Acinetobacter baumannii* in a university hospital in Italy. Clin. Microbiol. Infect., 13: 481–489.

10/4/2013