Prevalence and antimicrobial resistance of *Acinetobacter* isolates from King Fahd General Hospital, Saudi Arabia

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Abstract: Multi-drug resistant (MDR) *Acinetobacter baumannii* has emerged as an important nosocomial pathogen and many Hospital outbreaks have been described from various geographic areas. The resistance patterns of 104 isolates from King Fahad General Hospital; Jeddah, Saudi Arabia was studied. These *Acinetobacter* 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. Regardless of the specimen, there was a high rate of MDR *Acinetobacter* spp. in ICU isolates. Of the 104 isolates of *Acinetobacter*, 88.5% were *A. baumannii* and 11.5% Non *A. baumannii*. All of the strains were susceptible to colistin ($MIC_{50} \leq 0.5$ mg/l; $MIC_{90} \leq 1$ mg/l) and higher MICs were recorded for all tested antibiotics. Twenty antibiotypes were observed among the *Acinetobacter* spp. The largest predominate antibiotype contained a total of 32 MDR strains including 29 *A. baumannii* (Ab) and 3 non *A. baumannii* isolates. The isolates gelonging to the predominate antibiotype was resistant to the tested antimicrobial agents such as amikacin, cefepime, cefotaxime, ceftazidime, ciprofloxacin, gentamicin, imipenem, meropenem, piperacillin/ tazobactam, trimethoprim/ sulfamethoxazole and was sensitive to colistin and tigecycline. This study help taking effective measures for controlling *Acinetobacter* and data could be used in future as medical reference.

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1. Introduction

Acinetobacter baumannii is the most medically significant *Acinetobacter* spp. and their clinical impact was increasing morbidity or mortality and their infections are responsible for the increase in patient mortality that occurs in critically ill patients (Doughari *et al.*, 2011). These organisms are most often associated with nosocomially rather than community- acquired infections (Jain and Danziger, 2004). Indeed, European Prevalence of Infection in Intensive Care study reported that *Acinetobacter* spp. were the seventh most common isolate recovered from critically ill patients (Beggs *et al*., 2006). *A. baumannii* may be of low virulence except when isolated in critically ill or immunocompromised patients. The ability of *A. baumannii*is to develop multidrug resistance and to persist in harsh environmental conditions makes infections by *Acinetobacter* is very dangerous specially in those who have recently undergone major surgery, with malignant diseases or burns or immunosuppressed patients such as the elderly, neonates with low birth weights, and patients with prolonged illnesses (Doughari *et al.*, 2011). Moreover, *A. baumannii* is one of the "red alert" pathogens that greatly threaten the utility of our current antibacterial armamentarium (Peleg *et al*., 2008). Unfortunately, as resistance has increased, few antimicrobials can be reliably used for effective treatment of MDR *Acinetobacter* infections. Since few antimicrobials remain consistently effective in the treatment of nosocomial *Acinetobacter* infections, the search for new drugs and the reevaluation of older agents have become a priority (Jain and Danziger, 2004).

Several studies determined the increased risk factors for the acquisition of multidrug-resistant outbreak isolates which include support with mechanical ventilation, prolonged duration, longer hospital, ICU stay, exposure to infected or colonized patients, greater disease severity and administration of broad-spectrum antimicrobial agents were found particularly third-generation cephalosporins, carbapenems and fluoroquinolones (Karageorgopoulos and Falagas, 2008, Villalon *et al*., 2011).

Antibiotic resistance in *Acinetobacter* species has increased dramatically in the resent years (Lockhart *et al*., 2007). *Acinetobacter* resistance may be due to the impermeability of the outer membrane and/or environmental exposure to resistance genes (Bonomo and Szabo, 2006). Multidrug-resistant *Acinetobacter* species identified as carbapenem

resistance or resistance to ⩾3 classes of antibiotics (Falagas *et al.,* 2006). Multidrug-resistant in *Acinetobacter* from deep wound infections, osteomyelitis, respiratory infections, and bacteremia have been reported among military personnel with traumatic injuries during the conflicts in Iraq and Afghanistan (Davis *et al*., 2005). Carbapenems (Imipenem and Meropenem) are the mainstay of treatment for MDR *Acinetobacter* and increasing carbapenem resistant makes their infections difficult to treat (Rice, 2006; Maragakis and Perl, 2008). Resistance *Acinetobacter* isolates to all antibiotics including polymyxins has also been documented and treatment of these infections is very difficult and may be impossible (Urban *et al*., 2001, Gales *et al*., 2006). At King Fahd general Hospital (KFGH), it has been a particular *Acinetobacter* infection control problem especially in the most vulnerable patients. Information about *Acinetobacter* sensitivity in KFGH is lacking**.** The aim of this study was to determine the antimicrobial susceptibility of some clinical *Acinetobacter* isolates from KFGH to different classes of antibiotics. MICs to different antibiotics for each isolate were also studied.

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2. Material and Methods

Bacterial isolation and identification

From clinical specimens of patients at different service unit in King Fahd General Hospital (KFGH), 104 isolates of *Acinetobacter* spp. were collected during 6 months, from April to September 2010 (Al Massoudi *et al.*, 2013). The isolates were identified at Clinical Microbiology Laboratory, KFGH. All isolates were maintained on slopes of Nutrient agar at 4°C and regenerated every six months (Dadgar *et al.*, 2006), and on Glycerol Broth medium (20% glycerol) at -70°C for a long period storage (Zarrilli *et al.*, 2007).

Identification and susceptibility test by Phoenix System

The bacterial inoculum was prepared from nutrient agar slant of the pure culture, grown at 35°C for 24 hours. The panel was inoculated with the prepared ID Broth in less than 60 minutes after that prepared ID Broth. After vertox, the absorbance was adjusted to 0.50 – 0.60 McFarland (Standard inoculum) by using the Phoenix Spec™ Nephelometer (BD Diagnostic Systems). After that, 25 µl of the prepared ID Broth with one drop from the indicator were inoculated, placed closure securely on the panel to seal, then panels were loaded into Phoenix System. After 24h of incubation, the identification of the bacterial isolate and sensitivity to some antibiotics in addition to MIC were determined.

Sensitivity to tigecycline (15 mg, Oxoid Ltd, wade Road, Basingstoke, Handmade in UK) was determined according to Kirby-Bauer Disk Diffusion Susceptibility Test Protocol approved by CLSI guidelines. Inhibition zone diameter of ≥19 mm was considered as the breakpoint for tigecycline sensitive and the diameter of inhibition zone <19 mm considered resistant (R) as recorded by Hudzicki (2010) and CLSI guidelines.

3. Results

Acinetobacter isolates (104 isolates) were obtained from 6 different sites and the clinical distribution of these isolates was as the following (table 1): isolates from sputum (n=41, 39.42%); isolates from wound (n= 32, 30.8%); isolates from urine (n=19, 18.3%), isolates from blood, (n=6, 5.8%), isolates from cerebrospinal fluid (n=1, 1%) and from other locations (n=5, 4.8%). Out of 104 isolates of *Acinetobacter* spp., 92 isolates was identified as *A. baumannii* and 12 isolates were belonging to other

species thus named non *A. baumannii* isolates (table 1).

Table 1. Counts and percentage of *Acinetobacter* spp. isolated from different sites

Site of isolation	Count (%)	*Acinetobacter baumannii* (%)	Non *Acinetobacter baumannii* (%)
Sputum	41 (39.42)	39 (95)	2(5)
Wound	32 (30.8)	26 (81)	6(19)
Urine	19 (18.3)	16 (84)	3(16)
Blood	6 (5.8)	6 (100)	0
Cerebrospinal fluid	1 (1)	1 (100)	0
Others	5 (4.8)	4 (80)	1 (20)
Total	104 (100)	92 (88.5)	12 (11.5)

Antimicrobial susceptibility of the 104 clinical isolates of *Acinetobacter* was determined using panel of 12 antibiotics (Amikacin; Cefepime; Cefotaxime; Ceftazidime; Ciprofloxacin; Colistin; Gentamicin; Imipenem; Meropenem; Piperacillin-Tazobactam; Trimethoprim- Sulfamethoxazole and Tigecycline) and microdilution method.

Table 2 showed the susceptibility of the different isolates for each antimicrobial agent. Seventy two isolates (69.2%) were resistant for amikacin and 32 (30.8%) were sensitive. For imipenem and meropenem, 92 isolates (88.5%) were resistant and 12 isolates (11.5%) were sensitive. Moreover, 23 isolates (22.1%) were resistant for tigecycline and 81 isolates (77.9%) were sensitive. Finally, all of isolates were sensitive for colistin as shown in table 2 and figure 1.

Amikacin resistance was common in genus *Acinetobacter*, out of 72 resistant isolates, 64 (88.9%) were belonging to *A. baumannii* and 8 isolates (11.1%) were non *A. baumannii* (table 3). Also, out of 92 imipenem and meropenem resistant isolates, 82 (89.1%) were *A. baumannii* and 10 isolates (10.9%) were non *A. baumannii*. Moreover, 23 isolates were resistant for tigecycline, 22 isolates (95.7%) were *A. baumannii* and 1 isolate (4.3%) was non *A. baumannii* (table 3). According to sensitivity to the 12 tested antibiotics, the tested 104 *Acinetobacter* isolates were classified into twenty Antibiotypes patterns (antibiotic susceptibility profiles) and these antibiotypes give a designated code for patterns numerals (P1 to P20), as shown in table 4. Antibiotype P1 were resistance for all the broad spectrum antimicrobials tested excepted colistin and tigecycline and was the largest predominate antibiotype contained a total of 32 MDR strains including 29 *Ab* strains and 3 non *Ab*. Antibiotype P2 showed resistant to all tested antimicrobial agents except colistin and is the second predominate antibiotype. Isolates of antibiotype P3 were resistance for all the broad spectrum antimicrobials tested excepted amikacin, colistin and tigecycline and is the third predominate antibiotype, contained a total of 14 MDR strains including 13*Ab* and one non *Ab*. The isolates of antibiotype P4 were susceptible to all the antibiotics tested excepted cefotaxime where they were intermediate. This pattern contained seven isolates which were fully sensitive strains including six*Ab* strains and one non *Ab*. Antibiotype P5 isolates were susceptible to colistin, gentamicin and tigecyclineand showed resistance to cephalosporins, amikacin, imipenem, meropenem, piperacillin/ tazobactam and trimethoprim/ sulfamethoxazole which contain a total of 7 multidrug resistant strains, 5 isolates were *Ab* and 2 non *Ab*. Isolates of antibiotype P6 had sensitive to colistin, trimethoprim/ sulfamethoxazole and tigecycline which contain 5 multidrug resistant strains, 4 isolates were *Ab* and 1 non *Ab*. Antibiotypes P7, P8, P9 and P10 were contained 3 isolates multidrug resistance. Antibiotypes P11 to P20 were contained one multidrug resistant isolate except P13 and P19 which were fully sensitive.

MICs were determined for 12 antibiotics against 104 *Acinetobacter* clinical isolates using microdilution method (table 5). All isolates were classified to sensitive, intermediate or resistance. All *Acinetobacter* isolates (0.0 %) were resistant to Colistin which is the drug of choice, while 95 isolates (91.3%) were resistant to Piperacillin/ Tazobactam and 92% were risisyance to either Meropenem, Imipenem or Ciprofloxacin. Moreover, 93 isolates (89.4%) were resistant to Cefotaxime and 80-89% of the isolates were resistant to Trimethoprim/ Sulfamethoxazole, Gentamicin, Ceftazidime or Cefepime. In addition, 72 isolates (69.2 %) resist Amikacin. Lower resistance was recorded by 23 isolates (22.1%) against Tigecycline.

The results in table 6 showed that *Acinetobacter* spp. were highly resistant to amikacin and cefotaxime with $MICs_{50}$>32 mg/l. They were also resistant to cefepime and ceftazidime, with MIC_{50} and MIC_{90}>16. Concerning, imipenem and meropenem, the isolates that had MIC_{90}>8mg/l, considered resistant. From the previous results all tested antibiotics had weak antibacterial activities against *Acinetobacter* with high MIC_{50} and MIC_{90} values except colistin (MIC_{50}≤0.5 mg/l; MIC_{90}≤1 mg/l) which was the most effective antibiotic. Statistical analysis showed that there is a significant difference between antimicrobial agents and type of susceptibility of *Acinetobacter* spp. ($P < 0.05$ as measured by Kruskal-Wallis test, ANOVA) which means that each antimicrobial agent had special activity and different impact.

Table 2. Percentage of antimicrobial susceptibility of 104 *Acinetobacter* spp. to different antibiotics.

Antimicrobial agent	Percentage of susceptibility to antibiotics		
	Resistance (%)	Intermediate (%)	Sensitive (%)
AK	72 (69.2)	0(0)	32(30.8)
FEP	86(82.7)	6(5.8)	12(11.5)
CFM	93(89.4)	11(10.6)	0(0)
CAZ	89(85.6)	4(3.8)	11(10.6)
CIP	92(88.5)	0(0)	12 (11.5)
CL	0(0)	0(0)	104(100)
GEN	80(76.9)	3(2.9)	21(20.2)
IMP	92(88.5)	0(0)	12(11.5)
MEM	92(88.5)	0(0)	12(11.5)
TAZ	95(91.3)	2(1.9)	7(6.7)
TRI	89(85.6)	0(0)	15(14.4)
TGC	23(22.1)	0(0)	81 (77.9)

AK: Amikacin; FEP: Cefepime; CFM: Cefotaxime; CAZ: Ceftazidime; CIP: Ciprofloxacin; CL: Colistin; GEN: Gentamicin; IMP: Imipenem; MEM: Meropenem; TAZ: Piperacillin/ Tazobactam; TRI: Trimethoprim/ Sulfamethoxazole; TGC: Tigecycline.

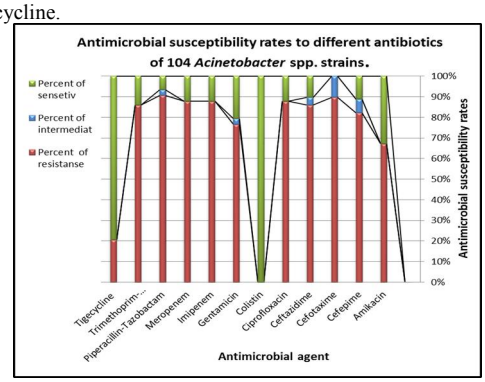

Figure 1. Antimicrobial susceptibility rates of 104 *Acinetobacter* spp. to different antibiotics

Table 3. Distribution of antimicrobial resistance of *Acinetobacter* spp. to different antibiotics.

Antimicrobial agent	Resistance species		Total
	Ab (%)	Non *Ab* (%)	
AK	64 (88.9%)	8 (11.1%)	72
FEP	77 (89.5%)	9 (10.5%)	86
CFM	83 (89.2%)	10 (10.8%)	93
CAZ	79 (88.8%)	10 (11.2%)	89
CIP	82 (89.1%)	10 (10.9%)	92
CL	0 (0%)	0 (0%)	0
GEN	74 (92.5%)	6 (7.5%)	80
IMP	82 (89.1%)	10 (10.9%)	92
MEM	82 (89.1%)	10 (10.9%)	92
TAZ	85 (89.5%)	10 (10.5%)	95
TRI	81 (91%)	8 (9%)	89
TGC	22 (95.7%)	1 (4.3%)	23

Ab: Acinetobacter baumannii; *Non Ab: Non Acinetobacter baumannii*; AK: Amikacin; FEP: Cefepime; CFM: Cefotaxime; CAZ: Ceftazidime; CIP: Ciprofloxacin; CL: Colistin; GEN: Gentamicin; IMP: Imipenem; MEM: Meropenem; TAZ: Piperacillin/Tazobactam; TRI: Trimethoprim/ Sulfamethoxazole; TGC: Tigecycline.

Table 4. Antimicrobial susceptibility patterns of *Acinetobacter* spp., collected from different service units at KHGH.

type	Antimicrobial susceptibility												No. and type of resistance	Biotype	
														Ab	Non *Ab*
P1	R	R	R	R	R	S	R	R	R	R	R	S	32 MDR	29	3
P2	R	R	R	R	R	S	R	R	R	R	R	R	17 MDR	17	0
P3	S	R	R	R	R	S	R	R	R	R	R	S	14 MDR	13	1
P4	S	S	I	S	S	S	S	S	S	S	S	S	7 FS	6	1
P5	R	R	R	R	R	S	S	R	R	R	R	S	7 MDR	5	2
P6	R	R	R	R	R	S	R	R	R	R	S	S	5 MDR	4	1
P7	R	I	R	R	R	S	S	R	R	R	R	S	3 MDR	2	1
P8	R	R	R	R	R	S	I	R	R	R	R	S	3 MDR	2	1
P9	S	R	R	R	R	S	R	R	R	R	R	R	3 MDR	3	0
P10	R	S	R	I	R	S	S	R	R	R	R	S	3 MDR	3	0
P11	R	I	R	R	R	S	R	R	R	R	R	S	1 MDR	1	0
P12	R	R	R	R	S	S	R	S	S	R	R	S	1 MDR	1	0
P13	S	S	I	I	S	S	S	S	S	I	S	S	1 FS	1	0
P14	S	R	R	R	S	S	R	S	S	R	R	S	1 MDR	1	0
P15	S	I	R	R	S	S	R	S	S	R	R	S	1 MDR	1	0
P16	S	I	I	S	R	S	R	R	R	R	R	R	1 MDR	1	0
P17	S	R	I	S	R	S	R	R	R	R	R	S	1 MDR	1	0
P18	S	R	R	S	R	S	R	R	R	R	R	R	1 MDR	1	0
P19	S	S	I	S	S	S	S	S	S	I	S	R	1 FS	0	1
P20	S	R	R	R	R	S	R	R	R	R	S	S	1 MDR	0	1
P-Value														0.0	0.15

AK: Amikacin; FEP: Cefepime; CFM: Cefotaxime; CAZ: Ceftazidime; CIP: Ciprofloxacin; CL: Colistin; GEN: Gentamicin; IMP: Imipenem; MEM: Meropenem; TAZ: Piperacillin/ Tazobactam; TRI: Trimethoprim/ Sulfamethoxazole; TGC: Tigecycline. MDR: Multidrug resistance; FS: Fully sensitive; *Ab: Acinetobacter baumannii*; Non *Ab:*Non *Acinetobacter baumannii*.

Table 5. MICs of 11 antibiotics used against 104 clinical isolates of *Acinetobacter* spp., as determined by microdilution method.

Antibiotics	Count of strains	MIC (mg/l)	Case	Antibiotics	Count of strains	MIC (mg/l)	Case
AK	21	≤8	S	GEN	9	≤2	S
	11	16	S		12	4	S
	72	>32	R		3	8	I
FEP	1	≤2	S		80	>8	R
	5	4	S	IMP	11	≤1	S
	6	8	S		1	2	S
	6	16	I		92	>8	R
	86	>16	R	MEM	11	≤1	S
CFM	6	16	I		1	2	S
	5	32	I		92	>8	R
	93	>32	R	TAZ	3	≤4/4	S
CAZ	2	4	S		3	4/8	S
	9	8	S		1	4/16	S
	4	16	I		2	4/32	I
	89	>16	R		95	>4/64	R
CIP	12	0.5	S	TRI	12	≤19/1	S
	92	>2	R		3	38/2	S
CL	66	≤0.5	S		1	76/4	R
	32	≥1	S		88	>76/4	R
	6	1	S				

MIC: Minimum inhibitory concentration; AK: Amikacin; FEP: Cefepime; CFM: Cefotaxime; CAZ: Ceftazidime; CIP: Ciprofloxacin; CL: Colistin; GEN: Gentamicin; IMP: Imipenem; MEM: Meropenem; TAZ: Piperacillin/Tazobactam; TRI: Trimethoprim/ Sulfamethoxazole; R: Resistance; S: Sensitive and I: Intermediate

Table 6. Antimicrobial resistance of 104 *Acinetobacter* spp. from KFGH

Antibiotics	MIC_{50}	MIC_{90}	Rang
AK	>32	>32	8 ≥ Rang >32
FEP	>16	>16	2 ≥ Rang >16
CFM	>32	>32	16 Rang >32
CAZ	>16	>16	4 Rang >16
CIP	>2	>2	0.5 ≥ Rang >2
CL	≤0.5	≤1	0.5 ≥ Rang 1
GEN	>8	>8	2≥ Rang >8
IMP	>8	>8	1≥ Rang >8
MEM	>8	>8	1≥ Rang >8
TAZ	>4/64	>4/64	4/4≥ Rang >4/64
TRI	>76/4	>76/4	19/1≥ Rang >76/4

MIC: Minimum inhibitory concentration; AK: Amikacin; FEP: Cefepime; CFM: Cefotaxime; CAZ: Ceftazidime; CIP: Ciprofloxacin; CL: Colistin; GEN: Gentamicin; IMP: Imipenem; MEM: Meropenem; TAZ: Piperacillin/Tazobactam; TRI: Trimethoprim/ Sulfamethoxazole

4. Discussions

Drug-resistant *Acinetobacter baumannii* is a Gram-negative bacterium found primarily in hospital settings, where it frequently dwells on catheter lines of ICU patients (Thomson and Bonomo, 2005; Maragakis and Perl, 2008). Because of *Acinetobacter's* low virulence, few colonized patients develop a disease. However, when an infection does occur, it often results in hospital-wide outbreaks and relatively high rates of mortality (Jawad *et al.*, 1996; Fournier and Richet, 2006). In the outpatient setting, the pathogen has been associated with wound infection (www.eurofins.com). National-level rates of multidrug-resistant *A. baumannii* (defined as simultaneously resistant to three classes from the following: antipseudomonal penicillins, ceftazidime, carbapenems, fluoroquinolones, aminoglycosides, sulbactams) grew significantly over the period, going from 32.1% in 1999 to 51% in 2010 and the largest and most consistent increase came from the Midwest, followed by South Atlantic states (www.eurofins.com). In this study, 104 *Acinetobacter* isolates were recovered from 102 patients at KFGH in six months and clinically, *Acinetobacter baumannii* was the main species. Many authors reported that *Acinetobacter* can be isolated from different clinical samples and can cause a variety of diseases, ranging from pneumonia to blood or wound infections (Towner *et al.*, 2008, Dent *et al.*, 2010). It may also "colonize" or live in a patient without causing infection (Bergogne-Berezin and Towner, 1996). All strains were susceptible to colistin ($MIC_{50} \leq 0.5$ mg/l; $MIC_{90} \leq 1$ mg/l) and higher MICs were recorded for all other tested antibiotics. In a Surveillance Network study, a large number of *A. baumannii* were recorded (Lee *et al.*, 2011). Similarly in a retrospective Saudi study, cohort investigation was performed and the resistance rates for *A. baumannii* were for amikacin (86%), colistin (0%) and tigecycline (43%). which is

almost similar to our results (Saeed *et al*., 2010). In previous study, tigecycline was the most active agent against *A. baumannii* isolates followed by amikacin where 80.6% and 29.6% of the isolate were sensitive, respectively (Akinci *et al*., 2008). This result differed with our obtained results where colistin was not tested but the activity of the other antibiotics consistent with our results. Pachon-Ibanez *et al*. (2004) reported 38 isolates were imipenem resistant (78%), an isolate showed intermediate susceptibility to imipenem (2%), and 10 isolates were imipenem susceptible (20%). Gurung et al. (2013) reported that out of 176 *Acinetobacter* spp., 57 isolates were identified as *Acinetobacter baumannii* and none of the isolates were resistant to colistin, levofloxacin, imipenem, cefepime, meropenem or ciprofloxacin but resistance to amikacin, gentamicin, piperacillin, and cefotaxime was 2.3, 7.4, 2.3, and 4.0%, respectively.

Definitions of multidrug-resistant *Acinetobacter* species vary, referring to a wide array of genotypes and phenotypes (Flages *et al.,* 2006). Resistant isolates to all antimicrobial agents making treatment of *Acinetobacter* infections extremely difficult and in some cases impossible (Gales *et al*., 2006). The definitions of multidrug resistance is resistance to 3 classes of antimicrobials and in this study 20 different antibiotic susceptibility profiles were recorded for the tested 104 *Acinetobacter* isolates. In contrast to our study, six different antibiotic susceptibility profiles (patterns) were observed among the 43 *Acinetobacter* isolates when tested for five broad spectrum antibiotics. These antibiotypes were designated using Roman numerals I-VI (Prashanth and Badrinath, 2005). In another study, Sadeghifarda *et al*. (2010) reported that *A. baumannii* isolates revealed nine different patterns of antibiotic resistance designated arbitrarily from A to I and these differences may due to types and number of the tested antibiotics. Gurung *et al*. (2013) added that *A. baumannii* strains were more susceptible to most of the antimicrobial agents tested compared with other *Acinetobacter* spp. which showed 17 different patterns of antimicrobial resistance.

MICs were recorded for the 12 tested antibiotics and all the *Acinetobacter* isolates were susceptible to colistin ($MIC_{50} \leq 0.5$ mg/l; $MIC_{90} \leq 1$ mg/l) and higher MICs were recorded for other tested antibiotics. Twenty antibiotypes were observed among the *Acinetobacter* spp. Lower level of resistant was recorded by Pachon-Ibanez *et al*. (2004) where three imipenem-susceptible strains (MIC, 1 µg/ml), three with intermediate susceptibility to imipenem (MIC, 8 µg/ml for one strain and MIC, 16 µg/ml for two strains), and three resistant to imipenem (MIC, 32 µg/ml) were obtained. Conversely, 45 strains were tigecycline susceptible (92%), with a MIC range of 1 to 4 mg/liter.

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