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ORIGINAL ARTICLE

Characterization of Antimicrobial Resistance and Prevalence of OXA Genes in the Emerging Threat *Acinetobacter baumannii* Causing Blood Stream Infection in ICU Patients

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Key words:
Acinetobacter baumannii, antimicrobial resistance, OXA genes, blood stream infections, ICU patients

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ABSTRACT

Background*: Acinetobacter baumannii became one of the emerging life-threatening hospital acquired infection pathogens with marked antibiotic resistance due to multiple resistance mechanisms. One of these mechanisms is production of carbapenemase enzymes like Ambler Class B and Class D enzymes that hydrolyze carbapenems the last resort antimicrobial drug for treating multidrug resistant organisms.* ***Objectives:*** *To study the antimicrobial resistance of A. baumannii with detection of* bla_{OXA} *carbapenemase genes responsible for resistance to carbapenems. Also to evaluate effectiveness of newly issued commercial Rapidec Carba NP kit test for detection of carbapenemases production and to assess the effect of implementing infection prevention and control (IPC) practices in decreasing rate of ICU blood stream infections.* ***Methodology:*** *This study was conducted from May 2017 to September 2018 by collecting blood cultures from ICU patients. A. baumannii species were identified and antibiotic susceptibility was run by VITEK 2Compact automated ID/AST instrument. Carbapenemase production was tested phenotypically by using Rapidec Carba NP kit test. A. baumannii species were tested for carbapenemase genes* bla_{OXA-51}, bla_{OXA-23}, bla_{OXA-24} *and* bla_{OXA-58} *by multiplex PCR.* ***Results****: there was a significant decrease in number of ICU blood stream infections after implementing IPC practices. Isolated A. baumannii species were 100 % resistant to Ampicillin/Sulbactam, Cefipime, Ceftriaxone, Imipenem and Trimethoprim/Sulphamethoxazole, while highest sensitivity was to Amikacin (27.4 %). All isolates A. baumannii species gave positive results with Rapidec Carba NP kit test. Only* bla_{OXA-23} *(82.4 %) and* bla_{OXA-51} *(88.2 %) were detected in isolated A. baumannii species but* bla_{OXA-24} *and* bla_{OXA-58} *were not detected.* ***Conclusion****: A. baumannii is a great life-threatening hospital acquired pathogen with marked drug resistant and easy spread. Rapidec Carba NP kit test is an easy, non-labor phenotypic test for carbapenemases production detection which can replace old cumbersome, difficult and labor Carba NP test.*

INTRODUCTION

Bloodstream infection (BSI) was defined as positive isolate of blood cultures associated with clinical findings [1]. Blood stream infections are potentially life-threatening health care–associated infections especially in immunosuppressed patients, Most of health care–associated BSIs occur due to the presence of a central venous catheter [2]. It is costly with a potential fatal fate [3]. In the United States, central venous catheters (CVPs) cause approximately 80,000 catheter-associated bloodstream infections with up to 28,000 deaths from patients in intensive care units (ICUs). Every patient with such infection costs nearly $45,000and totally it may reach $2.3 billion annually. The National Nosocomial Infections Surveillance (NNIS) system of the Centers for Disease Control and Prevention (CDC), announced that the median rate of catheter-associated bloodstream infection in ICUs may range from 1.8 to 5.2/1000 catheter-days [4].

Antimicrobial-resistance in Gram-negative bacteria is considered a great health threat, increasing in prevalence globally in both community and health care settings. The Antimicrobial Availability Task Force formed by the Infectious Diseases Society of America (IDSA) pointed three Gram-negative bacteria of high importance, they are, extended-spectrum β-lactamase (ESβL)-producing *Enterobacteriaceae*, *Acinetobacter baumannii* (*A. baumannii*) and *Pseudomonas aeruginosa* [5].

Acinetobacter baumannii is one of the most serious Gram-negative pathogens not easy to control or treat in

the intensive care units, causing morbidity and increased costs. Also it causes serious infections in critically ill patients with impaired host defenses and expanding their length of stay [6]. This infection is becoming increasingly common worldwide. Isolating *A. baumannii* colonized patients is the main infection control measure to control its spread [7].

A. baumannii has been rapidly growing over the last two decades, mainly due to emergence of multidrug resistant (MDR) *A. baumannii* representing an important cause of hospital acquired infections like blood stream infection, pneumonia, urinary tract infections, skin and soft tissue infections[8]. Bloodstream infection caused by *A. baumannii* is associated with marked morbidity and mortality rate may be as high as 58% [9,10].

Risk factors associated with *A. baumannii* colonization and infection include but not limited to: major surgeries, major trauma, burn, premature birth, previous hospitalization, length of hospital or ICU stay, mechanical ventilation, indwelling foreign devices, number of invasive procedures and previous antimicrobial therapy[11].

Virulence of *A. baumannii* is due to many factors including: its multidrug resistance, acquisition of several genetic elements as plasmids, insertion sequences and resistance islands; ability to resist dryness, which permits the pathogen to stick on abiotic surfaces in healthcare facilities and ability to asymptomatically colonize human host, assisting its prompt spread. One of the strongly suggested virulence factors and thoroughly studied is biofilm formation on abiotic surfaces due to expression of various proteins, extracellular polysaccharide and type IV pili. Although *A. baumannii* is non-motile, but it shows twitching and swarming-like motility mediated by type IV pili. Additionally, *A. baumannii* is naturally competent, means it can take up antibiotic resistance encoding genetic elements from the environment where Type IV pili play a significant role in DNA uptake[12].

One of the most important mechanisms of drug resistance in *A. baumannii* is production of beta-lactamases enzymes. Beta-lactamases having carbapenemase activities are divided into: serine oxacillinase (OXA) or Ambler Class D and Metallo-Beta-Lactamase (MβL) or Ambler Class B. OXA type carbapenemase, was first isolated from *A. baumannii* clinical isolate. It is plasmid encoded, transferable and previously calledbla_{OXA-23}.It has two closely related enzymes namedbla_{OXA-27} and bla_{OXA-49}. Now there are two more gene clusters contributing to carbapenem resistance, bla_{OXA-24}-like and bla_{OXA-58}-like have been reported. It is found thatbla_{OXA-51}-like genes are intrinsic in *A. baumannii* and is chromosomally encoded; that is why it is used as an identification marker for that species [13].

The International Network for the Study and Prevention of Emerging Antimicrobial Resistance described the development of infections caused by carbapenem resistant in *A. baumannii* as a "global sentinel event" necessitating immediate epidemiological and microbiological investigations and stated that the most effective measure to reduce MDR *A. baumannii* infections in hospitals is to implement infection control measures as strict contact precautions, cohorting, implementation of routine surveillance program to detect silent carriers, deep cleaning of the environmental surfaces and proper management of infected patients [14].

MEHTHODOLOGY

This study was conducted from May 2017 to September 2018 by collecting blood cultures from blood stream infection suspected patients admitted in National Liver Institute ICU, Menoufia University, Egypt. The study protocol was approved by ethics committee of the National Liver Institute. Blood culture bottles were incubated in BACT/ALERT system. Positive culture bottles were subcultured on routine microbiology media including blood agar and MacConkey agar (Oxoid, UK) and incubated at 37^{o}C for 24 hours. Bacterial isolates were identified using standard microbiological methods. Cultures were considered negative and discarded after 5 days incubation without giving positive alarm.

***Acinetobacter baumannii* species:**

Isolated 34 *Acinetobacter baumannii* species were identified and antibiotic susceptibility was run by VITEK 2Compact automated ID/AST instrument. Antibiotic susceptibility was done against the following antibiotics: Ampicillin/sulbactam (10/10 µg), piperacillin-tazobactam (100/10 µg), cefepime (30 µg), ceftriaxone (30µg), imipenem (10 µg), meropenem (10 µg), amikacin (30 µg), gentamicin (10 µg), ciprofloxacin (5 µg), levofloxacin (5 µg) and trimethoprim/sulphamethoxazole (1.25/23.75 µg) (CLSI, 2018).

Rapidec Carba NP kit test

All the 34 *A. baumannii* isolates were tested for carbapenemase production by using Rapidec Carba NP kit test (*bioMérieux*) according to manufacturer instructions. This test is a commercially available, easy to use test, considered as a modification of Carba NP test and modified Carba NP test with the same sensitivity and specificity [15].

Procedure:

According to manufacturer's instructions: **a.** Adding 100 µl of suspension supplied with the kit in wells "a", "b" and "c" for well rehydration and leave for 5–10 min at room temperature with lid covered. **b.** Mixing content of well "b" properly. **c.** Adding bacterial colonies into well "c" with turbidity similar to that of well "b" and then place the cover lid for 30 min at room temperature. **d.** Transferring 25 µl suspension from well "c" to wells

"d" and "e"(well "d" as control well and well "e" as test well). **e.** Transferring 25 µl suspension from well "a" to wells "d" and "e". **f.** Placing cover lid and incubation for 30 min at 37°C.

Results interpretation:

First reading should be taken after 30 min. If positive for carbapenemase production, well "d" changes to red color and well "e" yellow to orange color. If any other colors are detected, second reading should be taken after 30 min. If still colors different so report as negative for carbapenemase production[16].

Genomic DNA Isolation

Genomic Bacterial DNA was extracted by using QIAamp ® DNA Mini Kit (Qiagen, USA).

DNA amplification for detection of Carbapenemase (OXA) Genes by multiplex-PCR:

Detection and amplification of *bla*$_{OXA}$ genes was performed using multiplex PCR method. Sequences of forward and reverse primers used for detection of *bla*$_{OXA-51}$, *bla*$_{OXA-23}$, *bla*$_{OXA-24}$ and *bla*$_{OXA-58}$ genes and their respective sizes are presented in Table 1.

Table 1: Primers used for detection and amplification of *bla*$_{OXA}$ Genes

Bla$_{OXA}$ Gene	Sequence (5´→3´)	Size, bp	Reference
OXA-51	F: 5-TAA TGC TTT GAT CGG CCT TG-3	324	16
	R: 5-TGG ATT GCA CTTCATCTT GG-3		
OXA-23	F:GAT CGG ATT GGA GAACCAGA	501	16
	R:ATT TCT GAC CGC ATT TCC AT		
OXA-24	F:GGT TAG TTG GCC CCC TTA AA	246	16
	R:AGTTGAGCGAAA AGGGGATT		
OXA-58	F:AAG TAT TGG GGC TTG TGC TG	599	16
	R:CCCCTCTGCGCTCTACATAC		

PCR conditions:

Contents of the reaction included: 10 µL master mix containing, 0.9 µL primer for the studied *bla*$_{OXA}$ like genes,OXA-51, OXA-23, OXA-24,OXA-58, to which added 2 µL extracted genomic DNA, 5.5 µL DDW, 1U Taq polymerase, 1 × PCR buffer which contained 1.5 µM MgCl2 (25mM) (QIAGEN Inc., Valencia, CA, USA) and 0.7 µM of every deoxynucleoside triphosphate (dNTPs). Multiplex-PCR amplification was done by a thermal cycler (ThermoFisher, USA) where the first denaturation temperature was 94°C for 5 minutes, then 30 amplification cycles consisted of three phases denaturation step at 94°C for 35 seconds, then annealing step at 60°C for 35 seconds followed by extension step at 72°C for 40 seconds. The final extension step was done at 72°C for 6 minutes [17]. DNA ladder consisted of a plasmid double digest with the size range 100-1200 bp. Amplified DNA product was separated by 2% agarose gel electrophoresis at 80V for 2h.

Infection control practices

Due to importance of implementing infection control practices and guidelines along with patient care bundles, infection control campaign was conducted for doctors and nurses for orientation and follow up of health care personnel implementation of institutional infection control policies like hand hygiene, appropriate use of PPEs, standard precaution, transmission based precautions with using visual reminding signs.

RESULTS

Throughout the study for sixteen months 452 blood cultures were collected from ICU patients, 116 blood cultures were positive (80 cultures before and 36 after implementing infection control practices) and 336blood cultures were negative (176 before and 160 after implementing infection control practices) as shown in table 2.

Table 2: percentage of positive and negative blood cultures from ICU patients

No & % of blood cultures	Positive blood cultures		Negative blood cultures		X^2	P Value
	No	%	No	%		
Before IPC Practices implementation	80	68.9	176	52.4	9.65	0.001
After IPC Practices implementation	36	31.1	160	47.6		
Total (n=452)	116	25.7	336	74.3		

Bacterial isolates

Bacterial species isolated from blood cultures in this study were totally 116 isolates, 52 isolates (44.9 %) were *S. aureus*, 34 isolates (29.3 %) were *A. baumannii*, 28 isolates (24.1 %) were *K. pneumoniae* and 2 isolates (1.7 %) were *E. coli*.

Antimicrobial susceptibility

Antibacterial susceptibility of isolated *A. baumannii* species; done by VITEK 2Compact automated ID/AST instrument; were as follow: highest resistance was to Ampicillin/Sulbactam, Cefipime, Ceftriaxone, Imipenem and Trimethoprim/Sulphamethoxazole (100 % for each), then Piperacillin/Tazobactam and Levofloxacin (97.1 % for each), then Meropenem and Gentamycin (94.1 % for each), followed by Ciprofloxacin (91.2 %) and the highest sensitivity was to Amikacin (27.4 %).Results illustrated in table 3.

Table 3: antimicrobial susceptibility of isolated *A. baumannii* species

Antibiotic	*A. baumannii* species (n= 34)	
	Sensitive (No. & %)	Resistant (No. & %)
Ampicillin/Sulbactam	0 (0)	34 (100)
Piperacillin/Tazobactam	1 (2.9)	33 (97.1)
Cefipime	0 (0)	34 (100)
Ceftriaxone	0 (0)	34 (100)
Imipenem	0 (0)	34 (100)
Meropenem	2 (5.9)	32 (94.1)
Amikacin	4 (27.4)	30 (72.6)
Gentamycin	2 (5.9)	32 (94.1)
Trimethoprim/ Sulphamethoxazole	0 (0)	34 (100)
Ciprofloxacin	3 (8.8)	31 (91.2)
Levofloxacin	1 (2.9)	33 (97.1)

Rapidec Carba NP kit test

All the isolated *A. baumannii* species were positive to Rapidec Carba NP kit test. Positive test result is illustrated in figure 1.

Fig. 1: Rapidec Carba NP test kit, showing red color in well "d" and yellow color in well "e" interpreted as positive *A. baumannii* for carbapenemase production

Detection of OXA like genes by multiplex PCR

None of $bla_{\text{OXA-24}}$ or $bla_{\text{OXA-58}}$ was detected in the 34 isolates, $bla_{\text{OXA-51}}$was detected in 30 isolate (88.2 %) while $bla_{\text{OXA-23}}$ was detected in 28 isolate (82.4 %) (figure2).

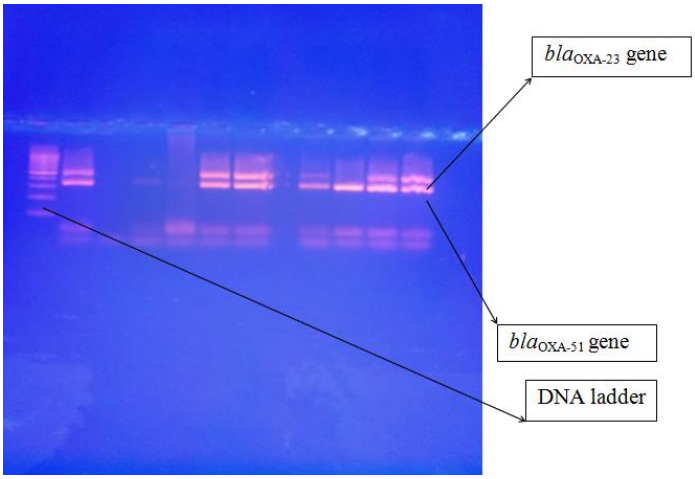

Fig. 2: Multiplex-PCR for $bla_{\text{OXA-51}}$, $bla_{\text{OXA-23}}$, $bla_{\text{OXA-24}}$ and $bla_{\text{OXA-58}}$ for *A. baumannii* isolated from ICU patients; $bla_{\text{OXA-24}}$ and $bla_{\text{OXA-58}}$was absent in all the tested isolates.

DISCUSSION

Hospital acquired infections are major problem for hospitalized patients in intensive care units and associated with significant morbidity, mortality and increased costs. Infections and related septicemia are the chief cause of death in non-cardiac ICUs, with mortality rates reaching 60% and accounting for almost 40% of total ICU expenses. Outstandingly, the incidence of sepsis is increasing, along with the number of subsequent infection-related mortalities [18].

There was no data available for the incidence of blood stream infections and incidence of *A. baumannii* species causing blood stream infection in ICU patients in National Liver Institute, Menoufia University. So in this study we investigated the incidence of *A. baumannii* blood stream infection in ICU patients admitted to National Liver Institute, Menoufia.

At least 32 named and unnamed *Acinetobacter* spp. have been defined. Genospecies 1 (*Acinetobacter calcoaceticus*), genospecies 2 (*Acinetobacter baumannii*), genospecies 3 and genospecies 13 are genetically closely related and difficult to be distinguished phenotypically by routine laboratory methods. So, these species have been referred to as a group, the *A. calcoaceticus–A. baumannii* complex (ACB complex).*A. calcoaceticus* is hardly isolated from clinical samples. The other three species (*A. baumannii*, *Acinetobacter* genospecies 13 and *Acinetobacter* genospecies 3) are common causes of community-acquired as well as nosocomial infections [19].

Being a life-threatening pathogen strict infection control programs are required to control spread of infection as per our study implementing infection control practices along with patient care bundles through campaign stressing on hand hygiene, appropriate use of PPEs, standard precaution, transmission based precautions with visual reminding signs followed by auditing for compliance; we noticed significant decrease in number of positive blood cultures taken from ICU patients. Same findings were illustrated by Barsuk et al.[20] who concluded from their study that implementation of patient care bundles can be used to reduce central-line associated blood stream infection (CLABSI) rates in a teaching-hospital ICU setting. Also Tsakris et al.[21] showed marked decrease in infections caused by multidrug resistant *A. baumannii* after reducing prescriptions of carbapenems with implementation of other infection control measures, however later on carbapenem consumption returned again almost to previous levels, which subsequently lead to the recurrence of multidrug resistant *A. baumannii* infections in the ICU.

In our study the most commonly isolated bacterial species causing ICU BSI were firstly *S. aureus* (44.9%), followed by *A. baumannii* (29.3%) and*K. pneumoniae* (24.1 %) then *E. coli* (1.7 %) so isolated Gram negative species were mora than 50 % of all species isolated. These results are generally in agreement with Vincent et al.[17], where 62 % of isolated species in ICU were Gram negative; they were mainly *E. coli* & *Pseudomonas aeruginosa*; and 47 % were Gram positive species mainly *S. aureus.* Results of Marra et al.[22] are strongly matching with our results where the most commonly isolated species from ICU BSIs were *S. aureus*, followed by *A. baumannii* and then *K. pneumoniae.* Results of Garmendia et al.[23] are almost in agreement with our results where Staphylococcal species (both *S. aureus* and *Coagulase negative Staphylococci*) were the most frequently isolated species from BSI infections in critically ill patients (45.9 %), followed by *A. baumannii* (18 %), then *Pseudomonas aeruginosa*(7.3 %) and *K. pneumoniae* (5.1 %). In a study conducted in 3 hospitals in Cairo, Egypt, by Fouad et al. [24], where only Gram negative species were isolated from ICU BSI infections. The most frequently isolated species were *K. pneumoniae* (9 isolates), *E. coli* (2 isolates) &*A. baumannii* (1 isolate).

Regarding antimicrobial resistance of *A. baumannii* species isolated from ICU bacteremic patients in the current study, it was noticed that there was higher resistance with 100 % resistance to one of the carbapenems (imipenem), third and fourth generation cephalosporins (Ceftriaxone & Cefipime) and also to Ampicillin/Sulbactam and Trimethoprim/Sulphamethoxazole. Also resistance was high to quinolones (97.1 % for levofloxacin and 91.2 % for ciprofloxacin). Resistance to Meropenem and Gentamycin was 94.1 % for each. *A. baumannii* species were more susceptible to amikacin (sensitivity was 27.4 %). This high resistance rate especially to carbapenems is mainly cause by maluses and over prescription of carbapenems to ICU patients.

We have compared our results with that of multiple studies conducted in Egypt. Our results highly matched with a study conducted in Kasr El Aini Hospital, Cairo and Dar Al Fouad, 6th October City, Egypt by Al-Agamy et al.[25], where the isolated *A. baumannii* species were 100 % resistant to amoxicillin/clavulanic, ceftriaxone, ceftazidime and cefipime. Resistance to ciprofloxacin was 85 % and to imipenem was 70 %. Least resistance was to amikacin (45 %). Results were mostly matching with results of Fattouh and Nasr El-din [26] in Sohag University Hospital where highest resistance was to Ampicillin/Sulbactam (86%), followed by Imipenem, Meropenem, Piperacillin and Gentamycin (72% of isolates); then Ceftazidime and Amikacin (67 %) with least resistance was to Ciprofloxacin (47% of isolates). However, resistance rate was lesser than in our study as they didn't record 100 % resistance to any of antibiotics.

The study done by Ahmed et al. [27] in multiple University Hospitals in Upper Egypt, similar to our results they documented 100 % resistance to ciprofloxacin and third generation cephalosporins and amikacin (76.9 %). They had lesser resistance incidence to imipenem (96 %), fourth generation cephalosporin (80 %) and meropenem (76 %).

Also, in a study conducted in Mohammed Military Teaching Hospital of Rabat, Morocco, by Lachhab et al. [28], the results were in agreement with our results where the reported 100% resistance to pipercillin/tazobactam, ceftazidime, imipenem and ciprofloxacin. Resistance to cotrimoxazole was 92 % and for gentamycin was 83 % and the least resistance was to amikacin (50 %).

In Turkey, a study done by Ciceket al. [29], the resistance rates agreed with our study where they reported resistance rates 97 % for Ampicillin-sulbactam, Piperacillin-tazobactam, Ceftazidime and Cefepime, For Imipenem, Meropenem, Ciprofloxacin and Levofloxacin (95, 94, 94 and 84 % respectively). Resistance to Trimethoprim-sulphamethoxazole was only 69 %. Least resistance was to Amikacin, Gentamicin (63 & 48 % respectively).

In contrast Lee et al., [19] reported much less resistance rates than ours. They reported resistance rate to Ampicillin/ sulbactam, Piperacillin/ tazobactam, Cefepime, Meropenem, Imipenem, Amikacin, Gentamicin, Ciprofloxacin and Levofloxacin (37.4, 38.3, 35.7, 36.5, 36.5, 40, 37.6, 35.7 and 36.5 % respectively). Also, Marra et al.[22] reported less resistance rates, where resistance rates to Ampicillin/ sulbactam, Piperacillin/ tazobactam, Ceftriaxone. Ceftazidime, Cefepime, Ciprofloxacin and Gentamicin were 54.5, 33.5, 55.4, 54.4, 50.2, 36.2 and 30.7 % respectively, with very high sensitivity to imipenem (99.7 %) and meropenem (98.7 %).

The detection of carbapenemase enzymes is challenging as phenotypic tests like the modified Hodge test and combined-disc tests are time consuming with false results probabilities. Molecular techniques, UV spectrophotometry and MALDI-TOF MS assays have good sensitivity and specificity but need well-trained personnel and good infrastructure and costs. So *bioMérieux* introduced Rapidec Carba NP kit for carbapenemases detection from bacterial colonies grown on selective or non-selective agar plates. The manufacturer claimed that sensitivity and specificity is 97.8% in comparison with molecular methods [15].

Regarding testing of *A. baumannii* species against Rapidec Carba NP kit test, all the isolated *A. baumannii* species in our study were positive to Rapidec Carba NP kit test. These results agreed with Garg et al., [15] who reported 92 % sensitivity for the test, Poirel and Nordman [30] who concluded that the test has very high sensitivity and specificity and Monica et al., [31] who reported 88 % sensitivity of the test to detect carbapenemase producing isolates.

For molecular workup to detect OXA carbapenemase genes in *A. baumannii* isolated in our study, none of $bla_{\text{OXA-24}}$ or $bla_{\text{OXA-58}}$ were detected in the 34 isolates, while $bla_{\text{OXA-51}}$was detected in30 isolate (88.2 %) and $bla_{\text{OXA-23}}$ was detected in 28 isolate (82.4 %). These results are matching with multiple studies conducted in Egypt where only $bla_{\text{OXA-51 and}}$ $bla_{\text{OXA-23}}$ were detected[24] where both genes were detected in 100 % in all isolated species in the study. Also study of Al-Agamy et al. [25] prevalence for both genes was 85 and 80 % respectively and study of Abdulzahra et al. [32] with prevalence 100 & 80 % for both genes respectively.

The $bla_{\text{OXA-23}}$ was the only detected OXA carbapenemase gene in 100 % of isolated *A. baumannii* species in three studies one in Egypt by Ahmed et al. [27], another in Qatar by Rolain et al. [33] and in Korea by Song et al. [9].

Results of Kock et al. [34] are largely in agreement with our results as the most frequently detected OXA carbapenemase genes were $bla_{\text{OXA-51}}$(83 %) and$bla_{\text{OXA-23}}$ (59 %) and less frequently $bla_{\text{OXA-56}}$ (3 %). Also our results agreed with Saranathan et al.[35] in Southern India where also the most frequently detected OXA carbapenemase genes were $bla_{\text{OXA-51}}$(100%) and$bla_{\text{OXA-23}}$(56%) and less frequently $bla_{\text{OXA-24}}$(14 %).

Against our results, Khorsi et al. [36] reported that in Algeria the only isolated OXA carbapenemase genes were $bla_{\text{OXA-23}}$ (67.02 %) and$bla_{\text{OXA-24}}$ (20.21 %). They didn't detect any $bla_{\text{OXA-51}}$ gene which is the most frequently detected gene in most of studies.

CONCLUSION

The high drug resistance of isolated *A. baumannii* in our study and coexistence of $bla_{\text{OXA-51}}$ and $bla_{\text{OXA-23}}$, suggested acquisition of several genetic elements as plasmids, insertion sequences and resistance islands. To control spread of infection, infection control practices and guidelines along with care bundles must be implemented.

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