AKADÉMIAI KIADÓ

Acta Microbiologica et Immunologica Hungarica

70 (2023) 1, 22–28

DOI:
10.1556/030.2023.01921

RESEARCH ARTICLE

Detection of multidrug-resistant *Acinetobacter baumannii* from burn patients and healthcare workers in Iran

FARZANEH FIROOZEH[1,2], FATEMEH BAKHSHI[1], MASOUD DADASHI[1], FARZAD BADMASTI[3]*, MOHAMMAD ZIBAEI[2,4] and NARGES OMIDINIA[1]

[1] Department of Microbiology, School of Medicine, Alborz University of Medical Sciences, Karaj, Iran

[2] Evidence-based Phytotherapy and Complementary Medicine Research Center, Alborz University of Medical Sciences, Karaj, Iran

[3] Department of Bacteriology, Pasteur Institute of Iran, Tehran, Iran

[4] Department of Parasitology and Mycology, School of Medicine, Alborz University of Medical Sciences, Karaj, Iran

Received: November 5, 2022 • Accepted: January 5, 2023
Published online: January 13, 2023

ABSTRACT

Multidrug-resistant (MDR) *Acinetobacter baumannii* is a serious global health threat. Burn patients are at high risk to acquire *A. baumannii* infections from endogenous sources. This study evaluated carbapenem resistance and clonal relatedness of *A. baumannii* isolated from burn patients and healthcare workers (HCWs).

The study was performed in 100 non-duplicated *A. baumannii* isolates from nasal and hand samples of hospitalized burn patients and HCWs in two hospitals of Iran from June 2020 to August 2021. Antimicrobial susceptibility testing was performed and carbapenemase genes were detected by PCR. Clonal relatedness of *A. baumannii* isolates was determined by two single-locus sequence-based typing of $bla_{\text{OXA-51-like}}$ and *ampC* and by multilocus sequence typing (MLST).

All *A. baumannii* isolates were found to be MDR while susceptible to colistin. The *intI1,* conserved segments of class 1 integron (*intI1* CS), bla_{IMP}, bla_{VIM}, $bla_{\text{OXA-51-like,}}$ and $bla_{\text{OXA-23-like}}$, genes were detected in 32.5%, 29.1%, 36%, 95.3%, 100%, 100%; and 14.3%, 14.3%, 21.4%, 92.9%, 100%, and 85.7% of isolates from patients and from healthcare workers, respectively. The $bla_{\text{OXA-58}}$, and $bla_{\text{OXA-143}}$ were not detected among the isolates. Using dual-locus $bla_{\text{OXA-51-like}}$ and *ampC* sequence-based typing (SBT), the isolates obtained from nasal samples of burn patients were grouped into 3 clusters including $bla_{\text{OXA-317,}}$ $bla_{\text{ADC-88}}$ (72.1%); $bla_{\text{OXA-64,}}$ *ampC-25* (18.6%); and $bla_{\text{OXA-69,}}$ *ampC-1* (9.3%). While only allele type $bla_{\text{OXA-317,}}$ $bla_{\text{ADC-88}}$ was determined among isolates from HCWs. MLST results showed *A. baumannii* ST136, ST25, and ST1 from burn patients. However, *A. baumannii* strains from HCWs belonged to ST136. Our findings indicate high prevalence of globally spreading of MDR *A. baumannii* ST136 carrying $bla_{\text{OXA-23-like}}$ from nasal and hand samples of burn patients and HCWs.

KEYWORDS

Acinetobacter baumannii, multidrug-resistant, burn patients, healthcare workers

INTRODUCTION

Acinetobacter baumannii is considered as one of the most challenging bacteria causing hospital infections [1]. This organism usually targets the most vulnerable hospitalized patients, especially those with impaired skin integrity and respiratory disorders [2]. The threat caused by this bacterium is especially due to its ability to persist for a long time in the direct environment of the patient [3]. It can also survive on healthcare workers' (HCWs) hands, increasing the risks of

*Corresponding author.
E-mail: fbadmasti2008@gmail.com

cross-transmission between patients [4]. Interestingly, this bacterium has been at the forefront of scientific attention as a result of its enormous ability to acquire antibiotic resistance determinants [2]. The synergism between antibiotic resistance and the eccentric potential of *Acinetobacter* to survive for prolonged periods in the hospital environment enhances its ability for nosocomial spread [5]. Although antibiotic resistance may not be a virulence factor, it is considered the most important determining factor in the clinical outcome of *Acinetobacter* infections [6]. According to the available reports, currently *A. baumannii* strains are resistant to all known antibiotics [2]. A problem of critical importance is the increase of carbapenem resistance among *A. baumannii* strains [7]. *Acinetobacter* isolates have demonstrated a complex interaction of diverse mechanisms of resistance to carbapenems, of which the production of oxacillinases (OXA) and the absence of PBP-2 are the most common mechanisms [8]. The predominant oxacillinases including OXA-23, OXA-24 or OXA-40, OXA-51, OXA-58, and OXA-143, are responsible for most of the carbapenem resistance cases in many regions of the world [6, 7]. Also, the global increase in the number of metallo-β-lactamases (MBLs) including IMP, VIM, SIM, and NDM in *A. baumannii* is an unfortunate event in this pathogen [9]. Carbapenemases have a high potential for widespread expansion because their encoding genes are usually accompanied by transposable genetic elements, including integrons [10, 11]. The increase in hospital outbreaks caused by carbapenem-resistant *A. baumannii* strains all over the world makes it necessary to monitor the epidemic evolution of multidrug-resistant (MDR) strains by grouping them in clonal lineages [12]. Among the methods most commonly used to trace the clonal spread of *A. baumannii* worldwide, multilocus sequence typing (MLST) is considered as the gold standard [12]. The potential of dual-locus-based typing methods using $bla_{\text{OXA-51-like}}$ and *ampC* genes to accurately assign *A. baumannii* isolates to international clones has been evaluated in some studies [13].

In the current study, MLST and simple, low-cost, and rapid sequence-based typing (SBT) were used to determine the clonal relatedness of *A. baumannii* strains isolated from nasal and hands samples of burn patients and healthcare workers.

MATERIALS AND METHODS

Identification of *A. baumannii*

This cross-sectional study was performed in two University Hospitals (Shahid Motahari Burn Hospital in Tehran and Shahid Madani Hospital in Karaj) during June 2020 to August 2021. A total of 100 non-duplicate *A. baumannii* isolates were recovered from nasal and hands of hospitalized burn patients and HCWs. Hand and nasal samples were obtained with standardized procedures using sterile cotton-tipped swabs [14]. Identification of isolates as *A. baumannii* were performed by standard microbiological methods as described previously [1]. Briefly, the collected nasopharyngeal and hand swabs (a single swab for both hands) were inoculated and incubated in 2 mL of brain heart infusion broth (Merck, Germany) for 18 h at 37 °C. The volume of 10 µL of the broth were subcultured on to MacConkey agar (Merck, Germany) and blood agar (Merck, Germany) for 48 h at 37 °C. *A. baumannii* isolates were identified by biochemical tests and confirmed using polymerase chain reaction (PCR) of the *rpoB* and $bla_{\text{OXA-51}}$ genes and sequencing [1].

Antimicrobial susceptibility testing

Disk diffusion assays were performed using antibiotic disks containing ciprofloxacin, ceftazidime, gentamicin, doxycycline, minocycline, trimethoprim/sulphamethoxazole, and ampicillin-sulbactam. In addition, the minimum inhibitory concentration (MICs) of imipenem and colistin were evaluated by the broth microdilution method. All procedures and results interpretation were done according to the Clinical and Laboratory Standards Institute (CLSI) guidelines and European Committee on Antimicrobial Susceptibility Testing (EUCAST) [https://www.eucast.org]. MDR *A. baumannii* strains were determined according to antibiotic resistance profiles.

Characterization of class I integrons and detection of carbapenemase genes

Genomic DNA extraction was performed by boiling method and the amplification of integrase and carbapenemase genes including *intI1*, *intI1* CS (conserved segments of class 1 integron), bla_{IMP}, bla_{VIM}, $bla_{\text{OXA-23-}}$like, $bla_{\text{OXA-58}}$, $bla_{\text{OXA-51-like}}$, and $bla_{\text{OXA-143}}$ was conducted by PCR assays for all *A. baumannii* isolates. The thermal cycling conditions and the PCR primers used have been described in our previous studies [1, 7].

Dual-locus sequence typing

All *A. baumannii* isolates were genotyped by dual-locus sequence-based typing of $bla_{\text{OXA-51-like}}$ and *ampC* genes. For this propose PCR amplification and subsequently, sequencing of $bla_{\text{OXA-51-like}}$ and *ampC* genes were performed, as previously described [15].

Multilocus sequence typing (MLST)

Sequence types (STs) were identified by MLST analysis. PCR amplification and sequencing of seven housekeeping genes (*pyrG, gltA, fusA, recA, cpn60, rpoB* and *rplB*) were performed using primers and PCR conditions described at http://pubmlst.org/abaumannii/. Identification of STs of strains were done after comparing with the known sequences in the *A. baumannii* PubMLST database and clonal complexes (CCs) were determined by eBURST software program [https://eburst.mlst.net].

RESULTS

Bacterial collection of *A. baumannii*

During the study period 100 non-duplicate *A. baumannii* isolates were isolated from 86 hospitalized burn patients

[29 (33.7%) female and 57 (66.3%) male] and 14 HCWs [6 (42.9%) female and 8 (57.1%) male]. The studied *A. baumannii* isolates were recovered from nasal and hands of hospitalized burn patients [42/86 (48.8%) nasal and 44/86 (51.2%) hands] and HCWs [7/14 (50%) nasal and 7/14 (50%) hands] in two university hospitals in Tehran and Karaj. The characteristics of the strains isolated from 2 hospitals are summarized in (Table 1).

Profiles of antibiotic resistance

The results of antibiotic susceptibility testing showed that out of 86 *A. baumannii* strains isolated from hospitalized burn patients were 86 (100%), 86 (100%), 86 (100%), 82 (95.3%), and 81 (94.2%) resistant to imipenem, ceftazidime, trimethoprim/sulphamethoxazole, ciprofloxacin, and gentamicin, respectively. The study of the resistance pattern of the 14 *A. baumannii* strains isolated from the HCWs, indicated the presence of resistance of 13 (92.9%), 14 (100%), 14 (100%), 13 (92.9%), and 11 (78.6%) to imipenem, ceftazidime, trimethoprim/sulphamethoxazole, ciprofloxacin, and gentamicin respectively. The rate of sensitivity to colistin, minocycline, doxycycline and ampicillin-sulbactam was high so that 100%, 98.8%, 97.7%, and 94.2%, of *A. baumannii* isolates from hospitalized patients were sensitive to the mentioned antibiotics, respectively. In addition, all HCWs isolates were sensitive to colistin, minocycline, doxycycline and only 7.1% of them were resistant to ampicillin-sulbactam. The antibiotic resistance profiles revealed that all the studied *A. baumannii* strains were MDR, while pan-drug resistant (PDR) phenotype was not identified in any of the isolates.

Detection of integrons and carbapenemases genes

Amplification of integron genes showed *intI1* and *intI1* CS genes were carried in 30% and 27% of total *A. baumannii* isolates, respectively. Separately the *intI1* and *intl CS* genes were detected in 28/86 (32.5%) and 25/86 (29.1%) of isolates from patients, and in 2/14 (14.3%) and 2/14 (14.3%) of isolates from HCWs, respectively. Out of 30 *intI1* positive *A. baumannii* isolates, 3 isolates recovered from patients, lacked gene cassettes and were recognized as empty integrons.

The bla_{IMP}, bla_{VIM}, $bla_{\text{OXA-51-like,}}$ and $bla_{\text{OXA-23-like}}$ genes were detected in 31/86 (36%), 82/86 (95.3%), 86/86 (100%), 86/86 (100%), and 3/14 (21.4%), 13/14 (92.9%), 14/14 (100%), and 12/14 (85.7%), of isolates from patients and HCWs, respectively. Furthermore, the $bla_{\text{OXA-58}}$, and $bla_{\text{OXA-143}}$ genes were not found among any of the clinical and HCWs isolates.

SBT and MLST

Locus SBT of $bla_{\text{OXA-51}}$-like, revealed three different OXA-51-like alleles including $bla_{\text{OXA-317}}$, $bla_{\text{OXA-64,}}$ and $bla_{\text{OXA-69}}$,

Table 1. The characteristics of *A. baumannii* strains isolated from nasal and hands of burn patients and health workers in two hospitals

Characteristic	Source	Shahid Motahari Hospital $N = 74$ N (%)	Shahid Madani Hospital $N = 26$ N (%)	All $N = 100$ N (%)
Sex				
Male		51 (68.9)	14 (53.8)	65 (65)
Female		23 (31.1)	12 (46.2)	35 (35)
Age (years)				
≤10		6 (8.1)	4 (15.4)	10 (10.0)
10–25		12 (16.2)	4 (15.4)	16 (16.0)
26–50		44 (59.5)	14 (53.8)	58 (58.0)
51–76		12 (16.2)	4 (15.4)	16 (16.0)
Mean ± SD				26.5 ± 11.9
Isolates				
Hospitalized patients	Nasal	32 (43.2)	10 (38.5)	42 (42.0)
	Hands	32 (43.2)	12 (46.1)	44 (44.0)
Healthcare workers	Nasal	5 (6.8)	2 (7.7)	7 (7.0)
	Hands	5 (6.8)	2 (7.7)	7 (7.0)
Resistance to antibiotic				
IMP		74 (100.0)	25 (96.2)	99 (99.0)
CAZ		74 (100.0)	26 (100.0)	100 (100.0)
SXT		74 (100.0)	26 (100.0)	100 (100.0)
CIP		71 (95.9)	24 (92.3)	95 (95.0)
GM		69 (93.2)	23 (88.5)	92 (92.0)
CL		0 (0.0)	0 (0.0)	0 (0.0)
DXT		2 (2.7)	0 (0.0)	2 (2.0)
MN		1 (1.4)	0 (0.0)	1 (1.0)
SAM		5 (6.8)	1 (3.8)	6 (6.0)
MDR		74 (100.0)	26 (100.0)	100 (100.0)

CAZ, ceftazidime; CIP, ciprofloxacin; CL, colistin; DXT, doxycycline; GM, gentamicin; IMP, imipenem; MN, minocycline; SAM, ampicillin-sulbactam; SXT, trimethoprim/sulphamethoxazole; MDR, multidrug-resistant.

which were identified in 62 (72.1%), 16 (18.6%) and 8 (9.3%) of isolates from patients respectively, whereas only allele type $bla_{\text{OXA-317}}$ was determined among isolates from HCWs. The SBT results of the *ampC* demonstrated three allele types including *ampC-1* and *ampC-25* belonged to CC1, and CC25 respectively and $bla_{\text{ADC-88}}$ belonged to singleton clones (Table 2). According to MLST genotyping results, three ST types including ST136, ST25, ST1, were identified in 62 (72.1%), 16 (18.6%) and 8 (9.3%) of *A. baumannii* strains isolated from patients respectively, whilst ST136 was detected in all isolates from HCWs. See Tables 2 and 3.

DISCUSSION

Acinetobacter species are often transmitted to patients through the hospital environment and the colonization of the hands of health workers, however, the spread of bacteria from infected or colonized patients is known to be one of the main routes of nosocomial spread of this bacterium [16]. Comparing the antibiotic resistance patterns of *A. baumannii* strains showed that all studied isolates were MDR, although the resistance rates were higher among clinical strains. In addition, all the isolates except one strain isolated from the healthcare worker, were imipenem resistant *A. baumannii*. High resistance to carbapenems is commonly reported in clinical strains of *A. baumannii* [17, 18]. Isolation of MDR/carbapenem-resistant *A. baumannii* strains from the hands of healthcare personnel is worrisome and can lead to rapid spread and hospital outbreaks of resistant clones. These findings also indicate the need to change procedures in the management of outbreaks due to MDR bacteria and to improve some practices and techniques in the studied hospitals. Studies have shown that compared to MBLs, OXA-type carbapenemases have a higher prevalence among *A. baumannii* strains, although the hydrolytic activities of MBLs toward carbapenems are 100 to 1000-fold more potent than that of OXA-type enzymes [2]. In the present study, consistent with other studies, $bla_{\text{OXA-23-like}}$ had the highest prevalence among carbapenemase genes and was found in all *A. baumannii* strains except two isolates from nasal and hand of HCWs [17, 19, 20]. We also found a high prevalence of the bla_{VIM} gene among *A. baumannii* strains, which could be the main mechanism of carbapenem-resistance in the studied *A. baumannii* isolates in association with the $bla_{\text{OXA-23-like}}$ gene. A large number of studied bla_{VIM}-positive *A. baumannii* isolates carried class 1 integrons. The association between *intI1* and bla_{VIM} genes are frequently documented in MDR *A. baumannii* isolates, which could be the reason for the wide spread of VIM metallo-β-lactamase enzyme in the present study. Interestingly, all bla_{VIM}-negative *A. baumannii* isolates lacked class 1 integrons, although bla_{VIM} (+), *intI1* (−) strains were also identified, whose wide distribution could be related to other mobile genetic elements. The simultaneous presence of OXA- and MBL type enzymes were detected in majority of our *A. baumannii* isolates. The presence of both OXA- and MBL carbapenemases in the same strains have been reported from different countries [21, 22].

MLST is a sequence-based typing method with high discriminatory power, however is expensive and laborious [23]. In this research, the typing and comparison of *A. baumannii* isolates from patients and HCWs was also investigated by a combination of simpler and more affordable typing methods including sequence-based typing of $bla_{\text{OXA-51-like}}$ and *ampC*. According to PCR-sequencing of $bla_{\text{OXA-51-like}}$ we identified a high frequency 76/100 (76%) of $bla_{\text{OXA-317}}$, ST136 allele type in the *A. baumannii* isolates from patients and HCWs, which is equivalent to $bla_{\text{ADC-88}}$ *ampC* allele [24, 25]. The mentioned allele type was the only ST isolated from the HCWs of our studied hospitals. ST136 has been previously identified in *A. baumannii* isolates from burn wound infection by our group [1]. This ST has also been reported from Taiwan and Kurdistan Region, Iraq [23, 25]. The results of previous studies have shown, ST136 belongs to CC92, the largest and the most widely disseminated clonal complex worldwide, which includes isolates with the ability to acquire resistance genes and the potential to persist in the hospital environment [26]. Accordingly, identification of patients and HCWs colonized with *A. baumannii* ST136 type in this study is worth considering. Another finding was that ST25 and ST1 were the other allele types that were only detected in *A. baumannii* isolates from patients. According to the available evidence, the clonal complexes 1 (CC1) and 25 (CC25) (Pasteur scheme) are emerging clones with a global spread and are responsible for several outbreaks of *A. baumannii* in the world [27]. In addition, the majority of hospital infections caused by *A. baumannii* belong to international clone IC1 and more recently to IC7, which are equivalent to clonal complexes CC1, and CC25 and ST types ST1, and ST25 [28, 29].

CONCLUSION

Our findings indicate the high prevalence and wide distribution of globally-spread *A. baumannii* ST136 carrying $bla_{\text{OXA-23-like}}$ within the clinical settings. Nevertheless, ST1, and ST25 could be considered as emerging lineages.

DECLARATIONS

Conflicts of interest: The authors declare that there are no conflicts of interest.

Funding: No funding sources.

Competing interests: None declared.

Ethical statement: All methods were carried out in accordance with the relevant guidelines and regulations of Ethics Clearance Committee of the Alborz University of Medical Sciences (ethic code: IR. ABZUMS.REC.1398.201).

Table 2. Presence of carbapenemase and integrons genes, $bla_{\text{OXA-51-like}}$ and *ampC* allele types, MLST sequence types, and clonal lineages of *A. baumannii* isolates from nasal and hands samples of burn patients

Isolate (n)	Hospital	Source	Carbapenemases genes				Integrons (*intI1*)	Cassettes (*intl CS*)	$bla_{\text{OXA-51-like}}$ allele	*ampC* (bla_{ADC}) allele number	Pasteur's MLST sequence type	Clonal lineages
			bla_{VIM}	bla_{IMP}	$bla_{\text{OXA-51-like}}$	$bla_{\text{OXA-23-like}}$						
6	SMT	Nasal	+	–	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
5			+	+	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
3			+	+	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
10			+	–	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
2			+	–	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
1			+	–	+	+	+	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			–	–	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
1			+	+	+	+	+	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			–	+	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
1			+	–	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
1			+	+	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
5		Hands	+	+	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
5			+	–	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
12			+	–	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
2			+	+	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
2			+	–	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
1			+	+	+	+	+	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			–	+	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
1		Hands	+	+	+	+	+	+	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
1			+	+	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	–	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
1			–	–	+	+	–	–	$bla_{\text{OXA-69}}$	*ampC-1*	ST1	CC1
4	SM	Nasal	+	+	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
5			+	–	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	–	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
5		Hands	+	–	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
3			+	+	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25
1			+	+	+	+	–	–	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	–	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	+	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	–	+	+	–	–	$bla_{\text{OXA-64}}$	*ampC-25*	ST25	CC25

MLST: multilocus sequence typing, ST: sequence type, CC: clonal complex; SMT: Shahid Motahari; SM: Shahid Madani.

Table 3. Presence of carbapenemase and class I integron genes, $bla_{\text{OXA-51-like}}$ and *ampC* allele types, MLST sequence types, and clonal lineages of *A. baumannii* isolates from nasal and hands samples of healthcare workers

Isolate (n)	Hospital	Source	Carbapenemases genes				Integrons (*intI1*)	Cassettes (*intI CS*)	$bla_{\text{OXA-51-like}}$ allele	*ampC* (bla_{ADC}) allele number	Pasteur's MLST sequence type	Clonal lineages
			bla_{VIM}	bla_{IMP}	$bla_{\text{OXA-51-like}}$	$bla_{\text{OXA-23-like}}$						
4	SMT	Nasal	+	-	+	+	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	+	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
4		Hands	+	-	+	+	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	+	+	+	+	+	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1	SM	Nasal	+	+	+	+	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			+	-	+	-	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1		Hands	+	-	+	-	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton
1			-	-	+	+	-	-	$bla_{\text{OXA-317}}$	$bla_{\text{ADC-88}}$	ST136	Singleton

MLST, multilocus sequence typing; ST, sequence type; CC, clonal complex; SMT: Shahid Motahari; SM: Shahid Madani.

ACKNOWLEDGMENT

The technical assistances of the Microbiology Laboratory of the Department of Microbiology, Alborz University of Medical Sciences and the Bacteriology Laboratory of the Pasteur Institute of Iran are appreciated.

REFERENCES

1. Nikibakhsh M, Firoozeh F, Badmasti F, Kabir K, Zibaei M. Molecular study of metallo-β-lactamases and integrons in *Acinetobacter baumannii* isolates from burn patients. BMC Infect Dis 2021; 21(1): 782. https://doi.org/10.1186/s12879-021-06513-w.
2. Peleg AY, Seifert H, Paterson DL. *Acinetobacter baumannii*: emergence of a successful pathogen. Clin Microbiol Rev 2008; 21(3): 538–82. http://doi.org/10.1128/CMR.00058-07.
3. Risser C, Pottecher J, Launoy A, Ursenbach A, Belotti L, Boyer P, et al. Management of a major carbapenem-resistant *Acinetobacter baumannii* outbreak in a French intensive care unit while maintaining its capacity unaltered. Microorganisms 2022; 10(4): 720. http://doi.org/10.3390/microorganisms10040720.
4. Barnes SL, Morgan DJ, Harris AD, Carling PC, Thom KA. Preventing the transmission of multidrug-resistant organisms: modeling the relative importance of hand hygiene and environmental cleaning interventions. Infect Control Hosp Epidemiol 2014; 35(9): 1156–62. https://doi.org/10.1086/677632.
5. Falah F, Shokoohizadeh L, Adabi M. Molecular identification and genotyping of *Acinetobacter baumannii* isolated from burn patients by PCR and ERIC-PCR. Scars Burn Heal 2019; 5: 2059513119831369. https://doi.org/10.1177/2059513119831369.
6. Wong D, Nielsen TB, Bonomo RA, Pantapalangkoor P, Luna B, Spellberg B. Clinical and pathophysiological overview of *Acinetobacter* infections: a century of challenges. Clin Microbiol Rev 2017; 30(1): 409–47. https://doi.org/10.1128/CMR.00058-16.
7. Bakhshi F, Firoozeh F, Badmasti F, Dadashi M, Zibaei M, Khaledi A. Molecular detection of OXA-type carbapenemases among *Acinetobacter baumannii* isolated from burn patients and hospital environments. Open Microbiol J 2022; 16: 1–6.
8. Fernández-Cuenca F, Martínez-Martínez L, Conejo MC, Ayala JA, Perea EJ, Pascual A. Relationship between beta-lactamase production, outer membrane protein and penicillin-binding protein profiles on the activity of carbapenems against clinical isolates of *Acinetobacter baumannii*. J Antimicrob Chemother 2003; 51(3): 565–74.
9. Dortet L, Poirel L, Errera C, Nordmann P. CarbAcineto NP test for rapid detection of carbapenemase-producing *Acinetobacter* spp. J Clin Microbiol 2014; 52(7): 2359–64. https://doi.org/10.1128/JCM.00594-14.
10. Firoozeh F, Aghaseyed-Hosseini M, Zibaei M, Piroozmand A. Detection of bla_{KPC} and bla_{GES} carbapenemase genes in *Klebsiella pneumoniae* isolated from hospitalized patients in Kashan, Iran. Recent Pat Antiinfect Drug Discov 2016; 11(2): 183–8. https://doi.org/10.2174/1574891X11666160813192556.
11. Amiri A, Firoozeh F, Moniri R, Zibaei M. Prevalence of CTX-M-type and PER extended-spectrum β-lactamases among *Klebsiella* spp. isolated from clinical specimens in the teaching hospital of

Kashan, Iran. Iran Red Crescent Med J 2016; 18(3): e22260. https://doi.org/10.5812/ircmj.22260.

12. Pournaras S, Gogou V, Giannouli M, Dimitroulia E, Dafopoulou K, Tsakris A, et al. Single-locus-sequence-based typing of $bla_{\text{OXA-51-like}}$ genes for rapid assignment of *Acinetobacter baumannii* clinical isolates to international clonal lineages. J Clin Microbiol 2014; 52(5): 1653–7. https://doi.org/10.1128/JCM.03565-13.
13. Hamouda A, Evans BA, Towner KJ, Amyes SG. Characterization of epidemiologically unrelated *Acinetobacter baumannii* isolates from four continents by use of multilocus sequence typing, pulsed-field gel electrophoresis, and sequence-based typing of $bla_{\text{OXA-51-like}}$ genes. J Clin Microbiol. 2010; 48(7): 2476–83. https://doi.org/10.1128/JCM.02431-09.
14. Morgan DJ, Liang SY, Smith CL, Johnson JK, Harris AD, Furuno JP, et al. Frequent multidrug-resistant *Acinetobacter baumannii* contamination of gloves, gowns, and hands of healthcare workers. Infect Control Hosp Epidemiol 2010; 31(7): 716–21. https://doi.org/10.1086/653201.
15. Nazari M, Azizi O, Solgi H, Fereshteh S, Shokouhi S, Badmasti F. Emergence of carbapenem resistant *Acinetobacter baumannii* clonal complexes CC2 and CC10 among fecal carriages in an educational hospital. Int J Environ Health Res 2021: 1–11. https://doi.org/10.1080/09603123.2021.1892036.
16. Spellberg B, Bonomo RA. "Airborne assault": a new dimension in *Acinetobacter baumannii* transmission. Crit Care Med 2013; 41(8): 2042–4. https://doi.org/10.1097/CCM.0b013e31829136c3.
17. Shamsizadeh Z, Nikaeen M, Nasr Esfahani B, et al. Detection of antibiotic resistant *Acinetobacter baumannii* in various hospital environments: potential sources for transmission of *Acinetobacter* infections. Environ Health Prev Med 2017; 22: 44. https://doi.org/10.1186/s12199-017-0653-4.
18. Custovic A, Smajlovic J, Tihic N, Hadzic S, Ahmetagic S, Hadzagic H. Epidemiological monitoring of nosocomial infections caused by *Acinetobacter baumannii*. Med Arch 2014; 68(6): 402–6. https://doi.org/10.5455/medarh.2014.68.402-406.
19. Pajand O, Rezaee MA, Nahaei MR, Mahdian R, Aghazadeh M, Soroush MH, et al. Study of the carbapenem resistance mechanisms in clinical isolates of *Acinetobacter baumannii*: comparison of burn and non-burn strains. Burns 2013; 39(7): 1414–9. https://doi.org/10.1016/j.burns.2013.03.024.
20. Gao J, Zhao X, Bao Y, Ma R, Zhou Y, Li X, et al. Antibiotic resistance and OXA-type carbapenemases-encoding genes in airborne *Acinetobacter baumannii* isolated from burn wards. Burns 2014; 40(2): 295–9. https://doi.org/10.1016/j.burns.2013.06.003.
21. Peleg AY, Franklin C, Walters LJ, Bell JM, Spelman DW. OXA-58 and IMP-4 carbapenem-hydrolyzing beta-lactamases in an *Acinetobacter junii* blood culture isolate from Australia. Antimicrob Agents Chemother 2006; 50(1): 399–400. https://doi.org/10.1128/AAC.50.1.399-400.2006.
22. Seyyedi Abhari S, Badmasti F, Modiri L, Aslani MM, Asmar M. Circulation of imipenem-resistant *Acinetobacter baumannii* ST10, ST2 and ST3 in a university teaching hospital from Tehran, Iran. Journal of Medical Microbiology 2019; 68(6): 860–5. Available from: https://doi.org/10.1099/jmm.0.000987.
23. Piran A, Shahcheraghi F, Solgi H, Rohani M, Badmasti F. A reliable combination method to identification and typing of epidemic and endemic clones among clinical isolates of *Acinetobacter baumannii*. Infect Genet Evol 2017; 54: 501–7. https://doi.org/10.1016/j.meegid.2017.08.018.
24. Johnson JK, Robinson GL, Zhao L, Harris AD, Stine OC, Thom KA. Comparison of molecular typing methods for the analyses of *Acinetobacter baumannii* from ICU patients. Diagn Microbiol Infect Dis 2016; 86(4): 345–50. https://doi.org/10.1016/j.diagmicrobio.2016.08.024.
25. Gaiarsa S, Batisti Biffignandi G, Esposito EP, Castelli M, Jolley KA, Brisse S, et al. Comparative analysis of the two *Acinetobacter baumannii* multilocus sequence typing (MLST) schemes. Front Microbiol. 2019; 10: 930. https://doi.org/10.3389/fmicb.2019.00930.
26. Alcántar-Curiel MD, Rosales-Reyes R, Jarillo-Quijada MD, Gayosso-Vázquez C, Fernández-Vázquez JL, Toledano-Tableros JE, et al. Carbapenem-resistant *Acinetobacter baumannii* in three tertiary care hospitals in Mexico: virulence profiles, innate immune response and clonal dissemination. Front Microbiol 2019; 10: 2116. https://doi.org/10.3389/fmicb.2019.02116.
27. Bonnin RA, Potron A, Poirel L, Lecuyer H, Neri R, Nordmann P. PER-7, an extended-spectrum beta-lactamase with increased activity toward broad-spectrum cephalosporins in *Acinetobacter baumannii*. Antimicrob Agents Chemother 2011; 55(5): 2424–7. https://doi.org/10.1128/AAC.01795-10.
28. Adams-Haduch JM, Onuoha EO, Bogdanovich T, Tian GB, Marschall J, Urban CM, et al. Molecular epidemiology of carbapenem-nonsusceptible *Acinetobacter baumannii* in the United States. J Clin Microbiol 2011; 49(11): 3849–54. https://doi.org/10.1128/JCM.00619-11.
29. Sahl JW, Del Franco M, Pournaras S, Colman RE, Karah N, Dijkshoorn L, et al. Phylogenetic and genomic diversity in isolates from the globally distributed Acinetobacter baumannii ST25 lineage. Sci Rep 2015; 5: 15188. https://doi.org/10.1038/srep15188.