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Frequency of Carbapenemase Genes in *Acinetobacter baumannii* complex Isolates from Burn Wounds in Motahari Hospital, Tehran, Iran

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ABSTRACT

Background: *Acinetobacter baumannii* complex (Acb complex) are opportunistic Gram-negative bacteria responsible for a diverse array of nosocomial infections. In recent years, carbapenem-resistant Acb complex has become a global concern. Carbapenemases are one of the most important mechanisms of resistance to carbapenems. This study aimed to measure the prevalence of carbapenemase genes in Acb complex isolates from burn wounds in a burn center in Iran.
Materials & Methods: During six months, 50 Acb complex isolates were collected from the wounds of burn patients admitted to Motahari hospital in Tehran (2020-2021). Antimicrobial susceptibility testing was performed for the isolates using Kirby-Bauer disc diffusion method based on the Clinical and Laboratory Standards Institute 2020 guidelines. DNA extraction was done by boiling method. The existence of bla_{OXA-51}, bla_{OXA-23}, bla_{IMP}, bla_{NDM-1}, and bla_{KPC} genes was evaluated by PCR and gel electrophoresis.
Findings: All isolated bacteria were confirmed as Acb complex based on positive PCR results for the presence of the bla_{OXA-51} gene. According to the antibiotic susceptibility testing results, the isolates showed 100% resistance to ceftazidime, 98% to ciprofloxacin, amikacin, and imipenem, and 94% to gentamicin and piperacillin-tazobactam. The most prevalent carbapenemase genes among the isolates were bla_{OXA-51} and bla_{OXA-23} (100%), followed by bla_{IMP} (26%), bla_{NDM-1} (14%), and bla_{KPC} (4%).
Conclusion: Carbapenem resistance and the prevalence of carbapenemase genes among Acb complex isolates has reached an alarming rate. Collaborative global efforts are crucial to safeguard antibiotic effectiveness and enhance patient care amidst escalating antimicrobial resistance challenges.

Keywords: *Acinetobacter baumannii*, Drug resistance, Carbapenems, Carbapenemase

CITATION LINKS

[1] Kyriakidis I, Vasileiou E, Pana ZD, Tragiannidis A. Acinetobacter baumannii Antibiotic... [2] Hassan RM. Molecular characterization of... [3] Azimi L, et al. Survey of various carbapenem-resistant... [4] Wasfi R. Co-Existence of Carbapenemase-Encoding... [5] Nasiri MJ, et al. Prevalence and Mechanisms of Carbapenem... [6] Nguyen M, Joshi SG. Carbapenem resistance in... [7] Beigverdi R. Status of carbapenem-resistant Acinetobacter baumannii... [8] Nordmann P, Poirel L. Epidemiology and Diagnostics of... [9] Vijayakumar S, et al. Insights into the complete genomes of... [10] CLSI. Performance standards for Antimicrobial... [11] Gholami M, Efflux Pump Inhibitor Phenylalanine... [12] Hou C, Yang F. Drug-resistant gene of blaOXA-23... [13] Poirel L, Walsh TR, Cuvillier V, Nordmann P. Multiplex PCR for... [14] Khosravi AD, Mihani F. Detection of metallo... [15] Woodford N, et al. Multiplex PCR for genes encoding prevalent... [16] Magiorakos AP, et al. Multidrug-resistant, extensively drug-resistant... [17] Pournajaf A, et al. Molecular characterization of carbapenem-resistant... [18] Salehi B. Emergence and characterization of... [19] Lukovic B, et al. The first nationwide multicenter... [20] El-Badawy MF. Characterization of phenotypic... [21] Khodaei H, Eftekhar F. Detection of kpc-type... [22] Khalid F, Saleem S, Ahmad I. High prevalence of... [23] AlAmri AM. Molecular Surveillance of Multidrug-Resistant... [24] Gupta N. Molecular Characterization of... [25] Farshadzadeh Z, et al. Wide distribution of carbapenem resistant... [26] Azimi L. Characterization of Carbapenemases in... [27] Elshamy AA, Aboshanab KM. A review on bacterial... [28] Hakemi vala M, et al. Detection of ambler class A, B and D β-Lactamases... [29] Hsu LY. Carbapenem-Resistant Acinetobacter... [30] Aruhomukama D, et al. bla(VIM)- and bla(OXA)-mediated... [31] Zhao Y, et al. Outbreak of carbapenem-resistant...

Introduction

Acinetobacter baumannii complex (Acb complex) are opportunistic Gram-negative bacteria responsible for a diverse array of nosocomial infections, including pneumonia, soft tissue infections, urinary tract infections, catheter-associated infections, and burn infections [1] .

These resilient pathogens exhibit tolerance to wide variations in temperature, pH, and humidity and could persist on dry surfaces for up to a month. In recent years, multidrug-resistant (MDR) Acb complex has become a global concern, imposing a substantial burden on healthcare systems and leading to elevated morbidity and mortality rates [2].

Last-resort antibiotics deployed against MDR Acb complex include carbapenems, colistin, and tigecycline. Carbapenems, recognized for their superior efficacy and fewer side effects, stand as the primary line of defense [3]. However, widespread resistance to carbapenems has elevated carbapenem-resistant Acb complex (CRAB) to the forefront of critical pathogens on the World Health Organization's priority list for research and development of new antibiotics [4]. The predominant mechanism of resistance to carbapenems in Acb complex involves the production of carbapenemase enzymes, a subset of beta-lactamases classified into A, B, C, and D groups based on Ambler's molecular classification [5].

Class A enzymes, known as serine-beta-lactamases, are plasmid encoded and thus highly spreadable, taking the third place among carbapenemases based on frequency. Class A enzymes, notably *Klebsiella pneumoniae* carbapenemases (KPC), have gained much attention due to their continuous global reporting and increasing prevalence [4].

Class B enzymes, known as metallo-beta-lactamases (MBLs), rank as the second most prevalent beta-lactamases after Class D enzymes. MBLs are remarkably more potent than other beta-lactamases (100 to 1000 times) and predominantly found in integrons [6].

Noteworthy examples include New Delhi metallo-beta-lactamase-1 (NDM-1) and imipenemase (IMP) enzymes encoded by *blaNDM-1* and *blaIMP* genes, respectively, playing a significant role in the resistance mechanism [7].

Class C enzymes are not considered carbapenemases as their main role is to hydrolyze cephalosporins [8].

Class D enzymes, classified as serine-beta-lactamases, are referred to as oxacillinases. Among them, the OXA-23 enzyme encoded by the *blaOXA-23* gene stands out as the most common acquired resistance mechanism globally [9]. It is often located in plasmid elements belonging to the IS4 family, facilitating its rapid mutation and expansion of its range of activities [5].

Objectives: Given the emergence of CRAB, this study aimed to quantify the frequency of *blaOXA-23*, *blaNDM-1, blaIMP,* and *blaKPC* genes in Acb complex isolates collected from patients with burn infections in Motahari Burn Center in Tehran, Iran.

Materials and Methods

Bacterial isolates: In this descriptive cross-sectional study, a total of 50 Acb complex isolates were collected from wound specimens of burn patients admitted to the sole specialized burn center in Tehran over a six-month period spanning from December 2020 to June 2021.

All procedures related to the isolation and identification of the isolates were performed following the established standard methods. Phenotypic methods such as oxidase, catalase, motility, culture on MacConkey agar (Merck Co., Germany), triple sugar iron agar (TSI) (Merck Co., Germany), and citrate tests were used to identify Acb complex, and then

PCR (polymerase chain reaction) detection of the *blaOXA-51* gene was used to confirm the isolates as Acb complex [9]. To ensure proper preservation of the isolates for subsequent evaluations, all isolates were stored in 10% glycerol tryptic soy broth (TSB) (Merck Co., Germany) at -70 °C.

Antimicrobial susceptibility testing (AST): The antimicrobial susceptibility of the isolates was assessed using Kirby-Bauer disc diffusion method in accordance with the guidelines outlined by the Clinical and Laboratory Standards Institute (CLSI, 2020) [10]. All antibiotic disks, including ceftazidime (30 µg), ciprofloxacin (5 µg), imipenem (10 µg), amikacin (30 µg), gentamicin (10 µg), and piperacillin-tazobactam (100/10 µg), were procured from Mast (UK) and Rosco (Denmark). *Escherichia coli* ATCC 25922 and *Pseudomonas aeruginosa* (*P. aeruginosa*) ATCC 27853 were used as control strains.

DNA extraction and PCR method

DNA extraction: About 1000 µL of bacterial suspension containing 107 bacteria/mL was transferred to microtubes. The microtubes were incubated at 100 °C in a boiling water bath for 5 minutes. Following this period, the DNA-containing suspension underwent vigorous homogenization by vortexing for 10 seconds, and the tube was promptly frozen on ice. The resulting DNA sample was then stored at -20 °C for subsequent analyses. The quality of the extracted DNA samples was assessed by measuring the absorbance at 260/280 nm using a spectrophotometer (DeNovix, USA).

Confirmation of bacterial identity: Confirmation of the identity of the bacterial isolates as Acb complex was established through PCR detection of the *blaOXA-51* gene.

Evaluation of resistant genes: The PCR reaction mixture comprised 12.5 µL of Taq 2X Master Mix Red containing 1.5 mM MgCl2 (Ampliqon, Denmark), 7.5 µL of sterile distilled water, 1 µL of each diluted forward and reverse primers (10 pmol/µL) (Bioneer, Korea), and 3 µL of sample DNA. The primer sequences are listed in Table 1.

Thermal cycling conditions were as follows: initial denaturation at 94 °C for 5 min; 36 cycles of amplification consisting of denaturation at 94 °C for 1 min, annealing at 55 °C for 1 min, and extension at 72 °C for 1 min; and a final extension at 72 °C for 5 min. Gel electrophoresis was performed using 1% agarose gel at 100 V for 45 min in 1X TBE buffer (Tris-borate-EDTA). The results were visualized under ultraviolet illumination using a gel documentation system. *P. aerugino-*

Table 1) Genes and primer oligonucleotide sequences used in PCR

Gene	Primer	Primer Sequence	Band Size	Reference
bla_{OXA-51}	F	TAATGCTTTGATCGGCCTTG	353 bp	[12]
bla_{OXA-51}	R	TGGATTGCACTTCATCTTGG		
bla_{NDM-1}	F	GGTTTGGCGATCTGGTTTTC	621 bp	[13]
bla_{NDM-1}	R	CGGAATGGCTCATCACGATC		
bla_{IMP}	F	GAAGGCGTTTATGTTCATAC	587 bp	[14]
bla_{IMP}	R	GTATGTTTCAAGAGTGATGC		
bla_{KPC}	F	CGTCTAGTTCTGCTGTCTTG	798 bp	[13]
bla_{KPC}	R	CTTGTCATCCTTGTTAGGCG		
bla_{OXA-23}	F	GATCGGATTGGAGAACCAGA	501 bp	[15]
bla_{OXA-23}	R	ATTTCTGACCGCATTTCCAT		

F: forward R: reverse

sa ATCC 780165 and a clinical *A. baumannii* isolate from our previous study were used as positive controls and a tube of Master Mix with sterile distilled water was used as negative control in PCR evaluation [11].

Statistical analysis: The recorded results underwent analysis using SPSS software Version 19 (SPSS Inc., Chicago, IL, United States). AST results were calculated based on the proportion of non-susceptible isolates to all isolates. Non-susceptible isolates to at least one antimicrobial agent in three or more different antimicrobial groups were considered as MDR [16].

Findings

Study patients and bacterial confirmation: Out of a total of 50 burn wound patients, 78% (n=39) were male, and 22% (n=11) were female. The mean age of the studied patients at admission was 36.52 years, ranging from 6 to 82 years. The average body surface burn area was 41.78%, with a minimum and maximum of 3 and 96%, respectively. The most frequent cause of burns among the studied patients was liquefied natural gas tank explosion (28%) followed by boiling water (22%) and gasoline fire (12%). The mortality rate was 24% as the outcome of the applied treatments.

Identification of all bacterial isolates as Acb complex was confirmed by standard bacteriological assays, including oxidase negativity and catalase positivity, no hydrogen sulfide production on TSI agar (K/K H2S negative), non-motility, citrate utilization, and growth on MacConkey agar. Furthermore, PCR detection of the *blaOXA-51* gene confirmed the isolates as Acb complex.

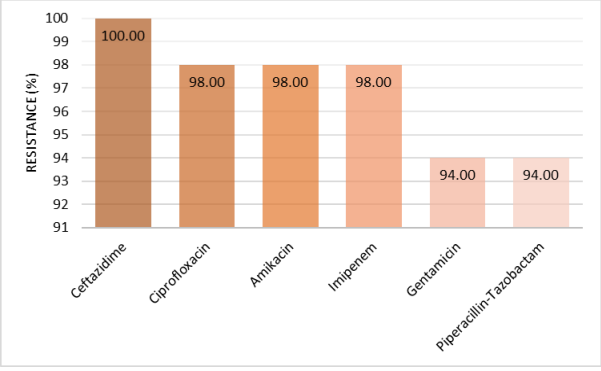

Figure 1) Frequency of resistance to antibiotics among isolates

AST: The AST analysis results revealed a striking pattern among Acb complex isolates in this study. All 50 isolates demonstrated complete resistance to ceftazidime, corresponding to a 100% resistance rate. Additionally, 49 isolates exhibited resistance to ciprofloxacin, amikacin, and imipenem. Moreover, a significant proportion of the isolates, specifically 47 out of 50, were resistant to gentamicin and piperacillin-tazobactam, resulting in a 94% resistance rate for these antibiotics (Figure 1). All 50 Acb complex isolates showed MDR pattern.

PCR: The *blaOXA-23* gene was universally present in all examined Acb complex isolates in this study. The distribution of carbapenemase genes among these isolates revealed distinctive prevalence rates.

The *blaOXA-23* gene was detected in all isolates. Additionally, 13 isolates carried the *blaIMP* gene, while 7 isolates harbored the *blaNDM-1* gene. Furthermore, 2 isolates were identified as carrying the *blaKPC* gene.

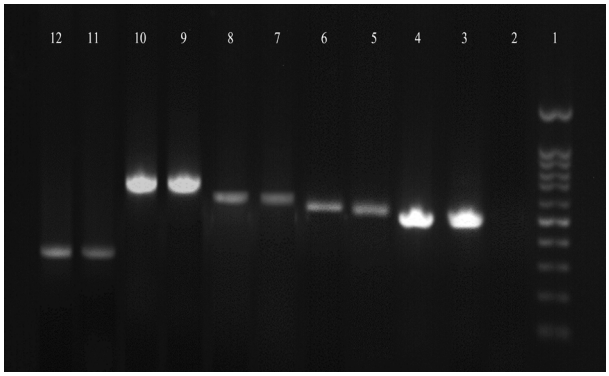

Figure 2) Gel electrophoresis of PCR products of carbapenemase genes. Lane 1: ladder (100 bp marker); lane 2: negative control; lanes 4-3: positive samples for *blaOXA-23* (501 bp); lanes 6-5: positive samples for *blaIMP* (587 bp); lanes 8-7: positive samples for *blaNDM-1* (621 bp); lanes 10-9: positive samples for *blaKPC* (798 bp); lanes 12-11: positive samples for *blaOXA-51* (353 bp)

Gel electrophoresis of PCR products and the distribution of carbapenemase genes are illustrated in Figures 2 and 3. The phenotypic and genotypic resistance patterns observed in this study are presented in Table 2.

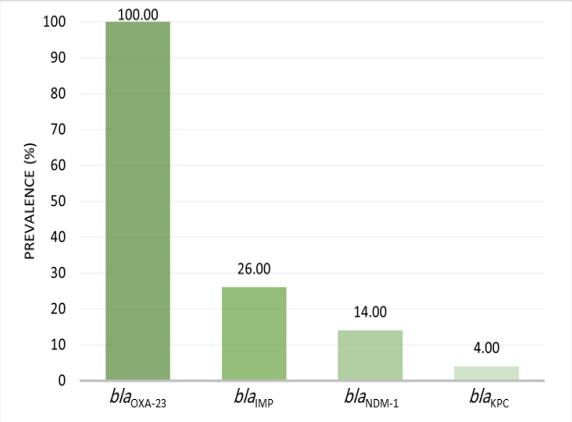

Figure 3) Prevalence of carbapenemase-encoding genes among the isolates

Discussion

In this study, all Acb complex isolates collected from wounds of burn patients in Motahari Burn Center in Tehran showed MDR profile. All 50 isolates exhibited resolute resistance to ceftazidime, representing a 100% resistance rate. Additionally, 49 isolates demonstrated concurrent resistance profiles against ciprofloxacin, amikacin, and imipenem, indicating a notable 98% resistance rate. Furthermore, a substantial proportion of the isolates, specifically 47 out of 50 isolates, displayed resistance to gentamicin and piperacillin-tazobactam, resulting in a 94% resistance rate to these antibiotics. Carbapenemases as a major mechanism of resistance to carbapenems were present in all isolates. The most prevalent gene among the isolates was *blaOXA-23* (100%). Also, *blaIMP* and *blaNDM-1* were present in 26 and 14%

Table 2) The phenotypic and genotypic resistance patterns among isolates

Carbapenemase Genes	Resistance to Antibiotics	Number of Isolates
bla_{OXA-23}	CAZ, CIP, IPM, AK, GEN, PTZ	25
bla_{OXA-23}, bla_{IMP}	CAZ, CIP, IPM, AK, GEN, PTZ	9
bla_{OXA-23}, bla_{NDM-1}	CAZ, CIP, IPM, AK, GEN, PTZ	4
bla_{OXA-23}, bla_{KPC}	CAZ, CIP, IPM, AK, GEN, PTZ	1
bla_{OXA-23}, bla_{IMP}, bla_{NDM-1}	CAZ, CIP, IPM, AK, GEN, PTZ	2
bla_{OXA-23}, bla_{IMP}, bla_{NDM-1}, bla_{KPC}	CAZ, CIP, IPM, AK, GEN, PTZ	1
bla_{OXA-23}, bla_{IMP}	CAZ, CIP, IPM, GEN, PTZ	1
bla_{OXA-23}	CAZ, IPM, AK, GEN, PTZ	1
bla_{OXA-23}	CAZ, CIP, AK, GEN, PTZ	1
bla_{OXA-23}	CAZ, CIP, IPM, AK, PTZ	2
bla_{OXA-23}	CAZ, CIP, IPM, AK, GEN	2
bla_{OXA-23}	CAZ, CIP, IPM, AK	1

CAZ: ceftazidime, CIP: ciprofloxacin, IPM: imipenem, AK: amikacin, GEN: gentamicin, PTZ: piperacillin-tazobactam

of the isolates, respectively. The *blaKPC* gene was detected in two (4%) isolates.

Previous studies conducted in Motahari hospital have also reported alarming resistance rates[17]. In a study by Salehi et al. (2018), 100 and 40% of *A. baumannii* isolates collected from clinical specimens in a hospital in Tehran were MDR and extensively drug-resistant (XDR), respectively [18]. These findings align with global trends observed in other countries like Saudi Arabia (MDR=100%) and Serbia (MDR=85.6%, XDR=40%, pandrug-resistant (PDR)=4.3%), demonstrating a widespread escalation of antibiotic resistance in *A. baumannii* isolates [19, 20]. The resistance profiles of Acb complex isolates in this study were substantial. These findings are in line with the results of several studies conducted in Iran. Pournajaf et al. (2018) reported similar resistance rates in Motahari burn hospital, with 97.3% resistance to ceftazidime, 100% to ciprofloxacin, and 94.5% to imipenem [17]. Furthermore, Khodaei and Eftekhar (2017) detected 100% resistance to imipenem and meropenem in a hospital in Tehran from 2014 to 2015 [21]. Systematic reviews conducted in Iran in 2019 and 2020 demonstrated 81.1% resistance to imipenem and 85.1% resistance to carbapenem [5, 7]. Comparative studies conducted in Saudi Arabia and Pakistan have exhibited similar distressing resistance patterns. In a study conducted in Saudi Arabia from 2017 to 2018, resistance to ceftazidime, ciprofloxacin, and piperacillin-tazobactam was 100%, and resistance to imipenem was 98.1%, whereas in another study in Pakistan, all 681 isolates were resistant to ceftazidime and piperacillin-tazobactam, 85.5% were resistant to imipenem, and 98.5% were resistant to gentamicin [22, 23]. A nationwide study conducted in Serbia in 2018 revealed that 93.7% of the isolates were resistant to either meropenem or imipenem, with 92.4% displaying resistance to both antibiotics [19]. These findings underscore the urgency of implementing stringent protocols to prevent nosocomial CRAB infections as a primary measure and the necessity of developing novel treatment strategies as a secondary priority. To combat the rising threat of antimicrobial resistance, there is an imperative need for better understanding and real-time monitoring of common resistance mechanisms.

One of the most important resistance mechanisms in CRAB is the production of various beta-lactamase enzymes, specifically categorized as carbapenemases in A, B, and D Ambler classes [6]. Class D carbapenemase-encoding genes, particularly the OXA-type variants, have been consistently identified in Acb complex isolates. Among them, the *blaOXA-23* gene is notably one of the most frequently-reported genes, ranking as the second most prevalent gene in Acb complex strains only after the intrinsic *blaOXA-51* gene. This prevalence underscores the importance of *blaOXA-23* among Acb complex isolates as highlighted in previous studies [24]. In this study, the *blaOXA-23* gene was identified in all 50 (100%) Acb complex isolates. Notably, a previous study in the same hospital indicated that 75.4% of CRAB isolates collected from 2016 to 2017 harbored *blaOXA-23*-like genes [17]. Other investigations within the same hospital have reported prevalence rates of 77% for *blaOXA-23*-like genes and 83% for the *blaOXA-23* gene among CRAB isolates [25, 26]. Azimi et al. (2020) reported the presence of the *blaOXA-23* gene in 76.5% of carbapenem-resistant *A. baumannii* isolates collected from nine provinces of Iran during 2016 to 2017 [3]. Two systematic reviews conduced in Iran in 2019 reported prevalence rates of 73.7 and 55.3% for *blaOXA-23*-carrying CRAB strains [5, 7]. In a study in Saudi Arabia, a prevalence rate of 68.9% was reported for the *blaOXA-23* gene among isolates collected from 2017 to 2018 [23]. In another study

in Egypt, 55.9% of CRAB isolates collected from 2017 to 2018 and 77.7% of isolates collected from 2018 to 2019 carried *blaOXA-23*-like genes [2, 4], while the prevalence rate of the *blaOXA-23* gene among *A. baumannii* isolates in Serbia in 2018 was 34.5% [19].

The *blaIMP* gene, an important member of MBLs, gained attention as the first known plasmid-encoded MBL in 1991 in Japan, contributing to a better understanding of this class of enzymes [5, 27]. In the current study, the *blaIMP* gene was identified in 13 (26%) Acb complex strains using PCR. Similarly, previous investigations conducted in the same hospital during 2016-2017 reported a 30.4% prevalence of the *blaIMP* gene among CRAB isolates, while no *blaIMP* gene was detected during 2012-2013 [17, 26, 28]. A multicenter study conducted in Iran during 2016 to 2017 revealed a 0.3% prevalence of the *blaIMP* gene among CRAB isolates [3]. Systematic reviews in Iran in 2019 reported varying prevalence rates, one of which indicated a 16.7% prevalence of the *blaIMP* gene among CRAB isolates [7]. Studies conducted in other countries highlight variations in *blaIMP* gene detection rates, underscoring diverse dynamics in different healthcare settings. A study conducted on *A. baumannii* isolates from cancer patients in Egypt during 2017-2018 reported a prevalence of 5.8% for the *blaIMP* gene [4].

The *blaNDM-1* gene, first reported in New Delhi (India) and later in nearby countries, was identified in seven (14%) Acb complex isolates in this study [29]. The prevalence rate of the *blaNDM-1* gene in Motahari hospital during 2016-2017 was 4.3%, whereas no *blaNDM-1* gene was detected in the isolates of the same hospital during 2012-2013 [17, 25, 26, 28]. Azimi et al. (2020) detected the *blaNDM-1* gene in 9.4% of CRAB isolates collected from nine provinces of Iran during 2016-2017, while a systematic review in 2019 estimated a 2.7% prevalence of the *blaNDM-1* gene in Iran [3, 7]. In Egypt, diverse prevalence rates have been reported. The National Cancer Institute reported a 67.7% prevalence of the *blaNDM-1* gene during 2017-2018, and another study indicated an 11.7% prevalence during 2018-2019 in various wards of a hospital [2, 4]. In another study in Serbia, 3.2% of isolates collected from different sources in 2018 carried the *blaNDM-1* gene [19].

Moreover, the detection of the *blaKPC* gene in two (4%) Acb complex isolates in this study is a noteworthy finding. In Motahari hospital, the prevalence of the *blaKPC* gene among CRAB isolates during 2016-2017 was 5.8%, contrary to most other Iranian studies reporting no *blaKPC* gene detection rate [17]. In Azimi et al.'s study, the *blaKPC* gene was not detected in CRAB isolates collected from nine provinces of Iran during 2016-2017 [3]. A systematic review in Iran in 2019 indicated a 0% prevalence of all Class A carbapenemase-encoding genes (including *blaKPC*) in CRAB isolates [3, 7]. In Uganda, no *blaKPC* gene was found in carbapenem-resistant *A. baumannii* isolates collected from 2015 to 2017 [30]. Likewise, in China and Saudi Arabia, the *blaKPC* gene was not detected in isolates collected during 2013 to 2015 and 2017, respectively [20, 31]. A study in Egypt reported a 10.7% prevalence for the *blaKPC* gene among CRAB isolates collected from 2018 to 2019 [2].

In this study, an alarming antibiotic resistance profile and a significant prevalence of carbapenemase genes were observed in Acb complex isolates collected from a burn center in Iran. Differences in the findings of different studies might be due to multifaceted factors, potentially encompassing variations in local antibiotic prescription practices or nuances in the specific primer sets employed in these studies. Such divergences emphasize the complexities involved in comprehensively understanding and accu-

rately identifying the prevalence of specific resistance genes within different clinical and geographical contexts. In this study, complementary investigations could include detection of insertion sequences, genotyping of isolates, and thorough epidemiologic studies. Furthermore, due to certain limitations, we were unable to utilize the minimum inhibitory concentration (MIC) test to identify CRAB. Although most reports show the ascending trend of resistance patterns and mechanisms among CRAB isolates, there are experiences of successful eradication of CRAB outbreaks in the world, which could be a model for future actions. For example, in the intensive care unit (ICU) of a hospital in China, the prevalence of CRAB increased from 25% in 2010 to 92% in 2014, but the implementation of necessary, though simple, preventive measures reduced its prevalence to 11.4% in 2017 [31].

Conclusion

In summary, this study sheds light on the prevalence of carbapenemase genes in Acb complex isolates, revealing an alarming prevalence of isolates carrying *blaOXA-23*, *blaIMP*, *blaNDM-1*, and *blaKPC* genes. The increased prevalence of carbapenemase genes in this study shows the rapid spread of CRAB, especially in developing countries facing challenges like antibiotic misuse and limited healthcare resources. The widespread dissemination of carbapenemase genes is a significant global health threat. Therefore, urgent measures need to be implemented, including enhanced surveillance, stringent infection control practices, prudent antibiotic use, and collaborative international efforts to address these challenges and combat the growing menace of carbapenemase producing Acb complex.

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Authors' contributions: S. Motamedi contributed to conceptualization, investigation, data curation, formal analysis, visualization, and writing (original draft preparation). A. Najafikhah contributed to methodology, investigation, data curation, writing (original draft preparation). M. Hakemi-Vala contributed to conceptualization, methodology, funding acquisition, project administration, resources, supervision, validation, and writing (review & editing).

Conflicts of interests: None declared by authors.

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Consent to participate: Patients were satisfied to participate in the study.

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