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Molecular Epidemiology of Carbapenem-resistant *Acinetobacter baumannii* Isolates in a Senegalese University Teaching Hospital

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: The emergence and spread of carbapenem-resistant *Acinetobacter baumannii* are critical in hospitals, particularly in intensive care units (ICUs), which represents a public health concern worldwide. In this study, we investigated the molecular epidemiology of multi-drug resistant *A. baumannii* (MDR-AB) in Dakar, Senegal.
Methods. The *A. baumannii* was isolated from Eosin Methylene Blue Agar culture and identified using API 20NE strip test and MALDI-TOF. The antimicrobial susceptibility testing was performed using the disk diffusion method. Simplex and multiplex-polymerase chain reactions with appropriate primers were used to detect and sequence the following β-lactamase genes: Two class D carbapenem hydrolyzing oxacillinases ($bla_{\text{OXA-51}}$ and $bla_{\text{OXA-23}}$), three class B metallo-β-lactamase genes (bla_{IMP}, bla_{VIM} and bla_{NDM}), and five class A β-lactamase genes (bla_{PER}, bla_{SHV}, bla_{VEB}, bla_{TEM}, *and* bla_{GES}).

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Results: A total of 29 strains of MDR-AB were isolated from patients hospitalized at Aristide Le Dantec University teaching hospital in Dakar, Senegal. Among the 29 MDR-AB strains isolated, 11 (37.9%) were isolated from ICUs, 5 (17.2%) from pediatric surgery, and 13 (44.8%) from other departments. The MDR strains were isolated from urine and pus samples with 12 (41.4%) and 9 (31.0%), respectively. All isolates were positive for the *A. baumannii* specific gene bla_{OXA-51}. The bla_{OXA-51} and bla_{OXA-23} genes coexisted in 26 (89.65%) of the strains. The bla_{IMP} and bla_{VIM} genes were not detected among the selected strains. Moreover1 (3.4%) strain elicited the gene coding for metallo-β-lactamase NDM-1. 2 (6.9%) isolates turned out to produce the penicillinase TEM-2.
Conclusions: Carbapenem resistance in Senegalese strains of *A. baumannii* is predominantly due to the worldwide disseminated gene bla_{OXA-23}, with a subset of strains due to NDM-1 and TEM-2. Systemic molecular surveillance network should be established for further efficient monitoring of MDR strains in Senegal.

Keywords: Acinetobacter baumannii; Carbapenemase; molecular epidemiology; multiplex PCR; NDM-1; TEM-2.

1. INTRODUCTION

Acinetobacter is a gram-negative aerobic bacilli or coccobacilli that belong to the *Moraxellaceae* family [1]. They are ubiquitous and can survive on dry surfaces for up to a month, are frequently carried on the skin of healthcare workers, and increase the likelihood of both colonizing patients and contaminating medical equipment. There are several species of *Acinetobacter* which can cause disease in humans, and *A. baumannii* accounts for almost 80% of infections [2]. *A. baumannii* infections usually occur in patients hospitalized in intensive care units (ICU) [3] and may cause important nosocomial infections [4]. City-acquired infections, especially pneumonia, are more common in tropical climates settings [5]. *A. baumannii* infection is associated with 19 to 54% mortality [6].

Nosocomial *Acinetobacter* pneumonia is frequently multi-lobar and complicated. *A. baumannii* can also cause wound and suppurative infections (e.g. abscesses) in any organ, including the lungs, urinary tract, skin, and soft tissue. Bacteremia and septic shock can occur from *A. baumannii* infection, and both are associated with a poor prognosis. *Acinetobacter* organisms can cause meningitis (mainly after a neurosurgical procedure), cellulitis, or phlebitis on indwelling venous catheter, eye infections, endocarditis on native valve or prosthesis, osteomyelitis, septic arthritis, or abscesses of the pancreas and liver. The significance of the isolation of *Acinetobacter* in clinical specimens, such as respiratory secretions in case of intubation or in specimens from open wounds, is difficult to interpret as it is often only responsible for colonization [2,7].

A. baumannii has long exhibited inherent resistance to many antimicrobials. Multidrug resistant (MDR) *A. baumannii* strains are defined as strains active in at least 3 classes of antimicrobials; some MDR isolates are resistant to all antimicrobials. Severe infections with *A. baumannii* are often treated by combining antibiotics: carbapenems (imipenem or meropenem) or ampicillin/sulbactam plus an aminoglycoside; the colistin-minocycline combination is the last treatment choice used in cases of extreme resistance [8]. For over 20 years, cases of acquired resistance of *A. baumannii* to carbapenems have been increasingly reported in all continents [9]. Until the discovery in 2011 of the genes encoding resistance to beta-lactams, tetracyclines, and glycopeptides, the excessive consumption of antibiotics were suspected as the main cause of the emergence of resistance genes [10]. With the increase in MDR bacteria, imipenem has become the drug of last choice for the treatment of nosocomial infections caused by *A. baumannii*. However, their efficacy is being increasingly compromised by the emergence of carbapenem-hydrolyzing beta-lactamases of molecular Ambler class B (VIM, IMP) and class D (OXA-23, OXA-58) [11].

In Africa, high rates of MDR *A. baumannii* infection have been demonstrated in several countries. Ugochukwu et al., in 2014, Lowe et al., in 2018, and Ben Cheikh et al., in 2018 evaluated the epidemiology of MDR of *A. baumannii* in Nigeria, South Africa, and Tunisia, respectively, which showed that the rate of MDR of *A. baumannii* ranged from 13 to 89% [12-14]. The main mechanism of resistance to carbapenems in Africa is represented by the production of OXA_{-23} [15].

The recognition of MDR isolates is a major laboratory challenge, and their inappropriate or delayed detection may have negative impacts on patient management and on the implementation of infection control measures [9]. To the best of our knowledge, very few studies were carried out on molecular characterization of carbapenem-resistant *A. baumannii* clinical isolates in Senegal. The aim of this study was to evaluate molecular epidemiology of MDR *A. baumannii* (MDR-AB) isolated in Aristide Le Dantec hospital, the major University Teaching Hospital in Dakar, Senegal.

2. MATERIALS AND METHODS

2.1 Isolation of *A. baumannii*

A total of 33 non-duplicate suspected MDR-AB strains were isolated from patients hospitalized in intensive care units (ICUs), pediatric surgery, and other departments of Aristide Le Dantec University hospital in Dakar, Senegal. Clinical specimens were cultured on Eosin Methylene Blue (EMB) agar (Merck, Germany), and incubated at 37°C for 24 h. Clinical isolates were identified using an API 20NE strip test (bioMerieux, Durham, USA). Species confirmation was performed using Matrix-Assisted Laser Desorption/Ionization Time of Flight (MALDI-TOF) system (Bruker Daltonik, GmbH, Bremen, Germany).

2.2 Antibiotic Susceptibility Testing

Antimicrobial susceptibility testing was performed using the disk diffusion method (Bio-Rad, France), as recommended by the Antibiogram Committee of the French Society for Microbiology (CA-SFM , 2015) [16]. Briefly, bacterial suspensions at 10^7 CFU/ml, adjusted with a McFarland densitometer, were inoculated on Mueller-Hinton agar and incubated for 24 hours at 37°C. The following antibiotics were tested: ticarcillin, (TIC, 75 µg), clavulanic acid/ticarcillin (TCC, 10/75 µg), clavulanic acid/amoxicillin (AMC, 10/20 µg), piperacillin (PIP, 100 µg), tazobactam/piperacillin (TZP, 10/100 µg), cefoxitin (CTX, 30 µg), ceftazidime (CAZ, 30 µg), cefepime (30 µg), aztreonam (ATM, 30 µg), imipenem (IMP, 10 µg), meropenem (MEM, 10 µg), gentamicin (GM,10µg), and tobramycin (TB, 30 µg), amikacin (AN, 30 µg), ciprofloxacin (CIP, 5 µg), and colistin (CST, 30 µg).

Pseudomonas aeruginosa ATCC 278539 were used for quality control.

Table 1. Oligonucleotide primers sequence used for PCR and sequencing of *Acinetobacter baumannii*

Targets	Primer names	Primer sequences	Position from ATG
bla$_{OXA-23}$	MOXA-23-F1	GAT-CGG-ATT-GGA-GAA-CCA-GA	261-280
	MOXA-23-R1	ATT-TCT-GAC-CGC-ATT-TCC-AT	742-761
bla$_{OXA-51}$	MOXA-51-F1	TAA-TGC-TTT-GAT-CGG-CCT-TG	252-273
	MOXA-51-R1	TGG-ATT-GCA-CTT-CAT-CTT-GG	588-607
bla$_{VIM}$ group 1	MuVIM-gr1-F1	GCW*-AGT-CCG-TTA-GCC-CAT-T	58-77
	MuVIM1-gr1-R1	R*R*C-GAC-TGA-GCG-ATT-TK*T-GT	776-796
bla$_{VIM}$ group 2	SeqVIM2-Fw1	TGT-TCA-AAC-TTT-TGA-GTA-AGT-TAT-TG	2-28
	SeqVIM2-Rv2	ACT-GAG-CGA-TTT-GTG-TGC	774-792
bla$_{NDM}$	MuNDM-F1	GGG-ATT-GCG-ACT-TAT-GCC-AA	406-426
	MuNDM-R1	TGG-CTC-ATC-ACG-ATC-ATG-CT	730-750
bla$_{IMP}$	IMP1-2-F	GTT-TAT-GTT-CAT-ACW*-TCG-TT	99-119
bla$_{PER}$	MuPER-F2	TGC-ATC-AGG-TB*G-ATC-AGG-G	245-26
	MuPER-R2	GY*G-CTT-CAT-TTG-CK*A-CCA-C	499-518
bla$_{SHV}$	MuSHV-F1	CGC-CAT-TAC-CAT-GAG-CGA-TAA	363-38
	MuSHV-R1	CCC-GCA-GAT-AAA-TCA-CCA-CAA	761-782
bla$_{VEB}$	MuVEB-F1	CCC-GAT-GCA-AAG-CGT-TAT-GA	192-21
	MuVEB-R1	CCG-GAA-GTC-CCT-GTT-TTA-TGA-G	704-726
bla$_{TEM}$	MuTEM-F1	TCT-CAA-CAG-CGG-TAA-GAT-CCT	144-165
	MuTEM-R1	TCA-GTG-AGG-CAC-CTA-TCT-CAG	827-848
bla$_{GES}$	GES-1A	ATG-CGC-TTC-ATT-CAC-GCA-C	1-19
	GES-1B	CTA-TTT-GTC-CGT-GCT-CAG-G	844-863

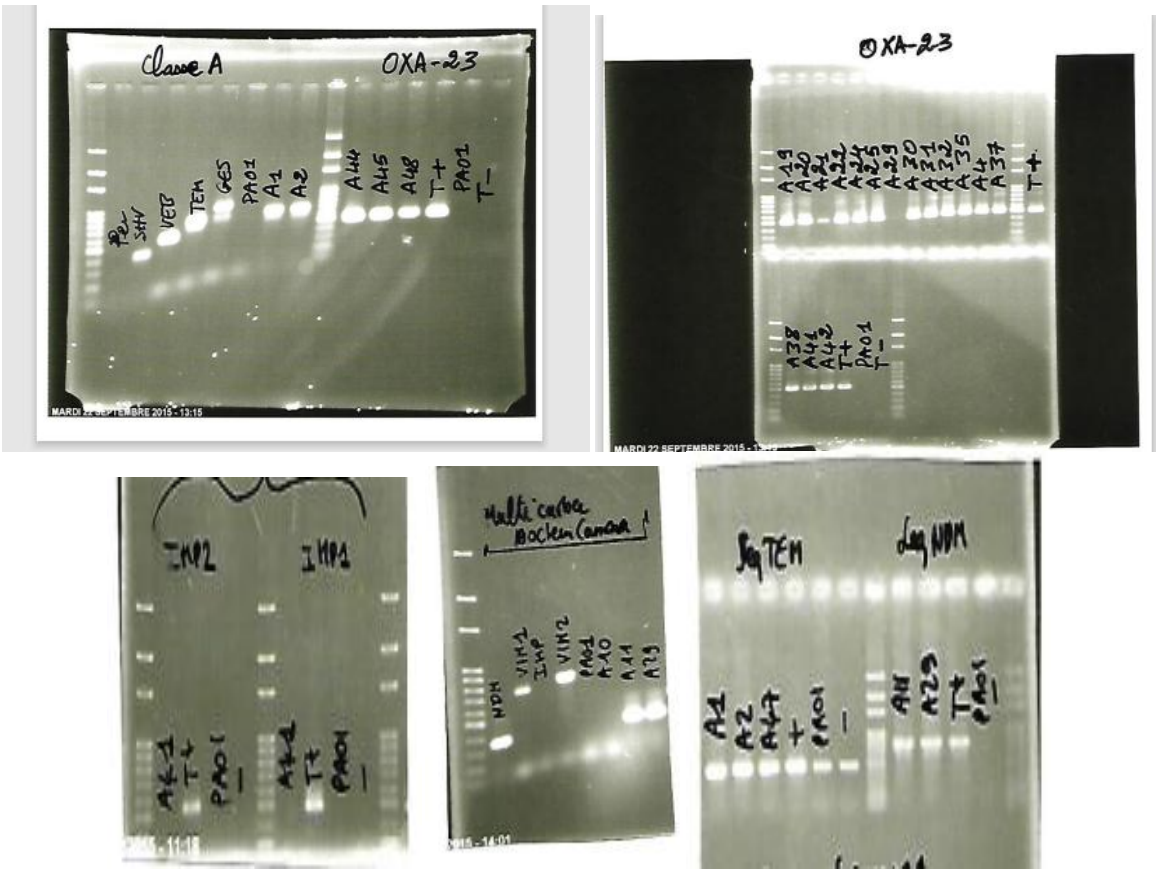

Fig. 1. Picture of gel

2.3 PCR and Sequencing of β-lactamase Genes

Total DNA was extracted from the bacterial isolates using the MagAttract HMW DNA Kit (Qiagen, Hilden, Germany), according to the manufacturer's instruction. Genes coding for OXA carbapenemase genes grouped into two class D carbapenem hydrolyzing oxacillinases (*bla*$_{OXA-51}$, and *bla*$_{OXA-23}$), three class B metallo-β-lactamase genes (*bla*$_{IMP}$, *bla*$_{VIM}$ and *bla*$_{NDM}$), and five class A β-lactamase genes (*bla*$_{PER}$, *bla*$_{SHV}$, *bla*$_{VEB}$, *bla*$_{TEM}$, and *bla*$_{GES}$) were sought by Simplex- and multiplex-polymerase chain reaction. The total information of the PCR primers utilized in our study is shown in Table 1. Each PCR reaction mix comprised 100 ng genomic DNA, 50 µL Nuclease-free water, 0.5 U Taq DNA Polymerase (5U/µL), 10 µL 5x MyTaq^{TM} Red Reaction Buffer, and 1 µL of each primer, in a total volume of 50 µL. The PCR reactions were initially denatured at 95°C for 1 minute, followed by 30 cycles of denaturation at 95°C for 15 seconds, and annealing at 60°C for 15 seconds, with a final extension at 72°C for 15 seconds. The PCR products were separated by 1.5% agarose gel electrophoresis and visualized using ethidium bromide staining and UV light.

2.4 Statistical Analysis

Differences in categorical variables between groups were analyzed using Fisher's exact tests. The level of significance for all statistical tests was set at $p < 0.05$. Statistical analyses were performed with Epi Info (version 7, CDC, USA) software.

3. RESULTS AND DISCUSSION

3.1 Distribution of Carbapenem-resistant *A. baumannii*

Of the 33 isolates initially isolated and identified with API NE, 29 isolates were confirmed as *A. baumannii* by MALDI-TOF, and the presence of *bla*$_{OXA-51}$-like gene. All 29 confirmed *A. baumannii* species were MDR pattern. Of these, 11(37.9%), 9(31.0%), and 5(17.2%) were collected from patients hospitalized in intensive care units (ICUs), general surgery and pediatric surgery, respectively (Table 2). Most of the MDR-

AB strains 12 (41.4%) were found in urine samples. Only 2 (6.9%) were isolated from bronchial secretions as described in Table 2.

3.2 Susceptibility Profiles of *A. baumannii* Isolates to Antimicrobial Agents

The results of *A. baumannii* susceptibility profile was depicted in table 3. All isolates were resistant to clavulanic acid/amoxicillin and clavulanic acid/ticarcillin combinations, aztreonam, cefoxitin, ciprofloxacin, pefloxacin, piperacillin, and ticarcillin. There was no statistically significant difference between the ICU and paediatric department. Among the aminoglycosides tested, gentamicin, tobramycin, and amikacin remained active in 4 (13.8%), 9 (31%), and 22 (75.9%) of cases, respectively, all these MDR isolates were susceptible to colistin.

3.3 Distribution of Carbapenemase-encoding Genes

Table 4 shows the distribution of carbapenem resistance genotypes. The genes encoding bla_{VIM}, bla_{IMP}, bla_{VIM}, $bla_{PER,}$ bla_{SHV}, bla_{VEB}, and bla_{GES} were not detected in our study. All 29 isolates (100%) were positive for the *A. baumannii* specific gene bla_{OXA-51}. The bla_{OXA-51} and bla_{OXA-23} genes coexisted in 25 (86.9%) isolates. Among the 25 bla_{OXA-23} positive strains, 1 (3.4%) expressed the gene coding $bla_{TEM.}$ Interestingly, among the 29 MDR-AB, 1 (3.4%) were positive for bla_{NDM-1} and $bla_{TEM-2,}$ but not $bla_{OXA-23.}$

Table 2. Distribution of carbapenem-resistant *Acinetobacter baumannii* according to services and pathological products

	Parameters	
Departments/Types of samples	**n, (%)**	**95% CI**
Hospital unit		
ICUs	11 (37.9)	20.7 - 57.7
Pediatric surgery	5 (17.2)	5.8 - 35.8
General surgery	9 (31.0)	15.3 - 50.8
Other	4 (13.8)	3.9 - 31.7
Specimen type		
Blood	5 (17.2)	5.8 - 35.5
Puncture liquid	1 (3.4)	0.1 - 17.8
Pus	9 (31.0)	15.3 - 50.8
Bronchial secretions	2 (6.9)	0.8 - 22.8
Urines	12 (41.4)	23.5 - 61.1

Table 3. Antimicrobial susceptibility profile of *A. baumannii*

Antibiotics	**Resistant**		**Susceptible**	
	n (%)	**95% CI**	**n (%)**	**95% CI**
Amoxicillin/Clavulanic acid	29 (100)	100 - 100	0 (0)	0 - 0
Ticarcillin/Clavulanic acid	29 (100)	100 - 100	0 (0)	0 - 0
Amikacin	7 (24.1)	10.3 - 43.5	22 (75.9)	56.5 - 49.7
Aztreonam	29 (100)	100 - 100	0 (0)	0 - 0
Cefepime	26 (89.7)	72.6 - 97.8	3 (10.3)	2.2 - 7.4
Cefoxitin	29 (100)	100 - 100	0 (0)	0 - 0
Ceftazidime	24 (82.8)	64.2 - 94.2	5 (17.2)	5.8 - 35.8
Ciprofloxacin	29 (100)	100 - 100	0 (0)	0 - 0
Colistin	0 (0)	0 - 0	29 (100)	100 - 100
Gentamicin	25 (86.2)	68.3 - 96.1	4 (13.8)	3.9 - 31.7
Piperacillin	29 (100)	100 - 100	0 (0)	0 - 0
Imipenem	29 (100)	100 - 100	0 (0)	100 - 100
Meropenem	29 (100)	100 - 100	0 (0)	0 - 0
Piperacillin/Tazobactam	28 (96.6)	82.2 - 99.9	1 (3.4)	0.1 - 17.8
Ticarcillin	29 (100)	100 - 100	0 (0)	0 - 0
Tobramycin	20 (69)	49.2 - 84.7	9 (31)	15.3 - 50.8

Table 4. Distribution of carbapenemase-encoding genes in *A. baumannii* strains

Carbapenemases-encoding genes	Number (%)	95% CI
bla$_{OXA-51}$ only	29 (100)	-
bla$_{OXA-51}$ and *bla*$_{OXA-23}$	25 (86.2)	68.3 - 96.1
bla$_{OXA-51}$ *bla*$_{OXA-23}$ and *bla*$_{TEM-2}$	1 (3.4)	0.1 - 17.8
bla$_{OXA-51}$ and *bla*$_{NDM-1}$	1 (3.4)	0.1 - 17.8
bla$_{OXA-51}$ and bla$_{TEM-2}$	2 (6.9)	0.8 - 22.8

3.4 Antibiotic Susceptibility Profiles of Carbapenemase-Encoding genes of *A. baumannii*

The *bla*$_{OXA-23}$ positive strain was more resistant to antibiotics than strains that harbor both *bla*$_{OXA-23}$ and *bla*$_{TEM-2}$ (Table 5). We also found that the *bla*-$_{TEM}$ positive strain was susceptible to tobramycin and amikacin. In contrast, the strains positive for both the blaNDM-1, and $_{bla}$TEM-2 genes were all resistant to gentamicin and tobramycin as shown in Table 5.

In this study, we evaluated the molecular epidemiology of *A. baumannii* resistance to carbapenems, particularly imipenem, in patients at Aristide Le Dantec university teaching hospital. A total of 29 strains collected from patient's specimen in different hospital departments were identified using API NE galleria, MALDI-TOF mass spectrometry, and the presence of *bla*$_{OXA-51}$ gene detected by PCR. Our confirmation of *A. baumannii* by *bla*$_{OXA-51}$ was higher than what was previously carried out in Senegal [17].

Our results showed that the majority of *A. baumannii* strains have been isolated from patients hospitalized in intensive care units (ICU), as previously reported in Africa, China, Europa, and America [17-21].

In this present study, these *A. baumannii* strains were mainly isolated from urine (41.4%), pus (31.0%), and blood (17.2%) samples. A study done by Nadia Jaidane et al., in 2016 from the University Hospital of Sahloul (Tunisia) reported 38.2% of *A. baumannii* isolates from blood [19]. Hilina Maitbonor et al., in 2018 from Ethiopia reported lower rates of 5% and 4.1% of *A baumannii* isolates respectively from blood and urine cultures [22].

We found high resistance rates to beta-lactam and aminoglycosides antibiotic classes. Resistance to beta-lactams was associated with other antibiotics families, such as aminoglycosides including gentamicin, tobramycin and amikacin and fluoroquinolones (ciprofloxacin: 100%). Recently, a study conducted by Anane et al., [18] in South Africa showed that more than 81% of *A. baumannii* clinical isolates had shown that resistance to beta-lactams were associated with those of other families of antibiotics such as aminoglycosides and fluoroquinolones with resistance rates ranging from 50 to 87% CRAB has become a real health problem. In our studies all strains of *A. baumannii* were resistant to carbapenems. In Ethiopia, Germany Italy and China, the proportion of *A. baumannii* isolates resistant to carbapenem increased from 13.5% to 98.7% [21-24]. Carbapenems are generally considered to be the most effective antibacterial agents for treatment of multidrug-resistant bacterial infections, including Gram-negative bacilli. However, with widespread use of these antibiotics, the prevalence of bacterial resistance to carbapenems has increased rapidly. This increase in *Acinetobacter* species could be explained, in large part, to the association of resistance mechanisms: over expression of low-spectrum beta-lactamases extended with efflux pumps, impermeability, or expression of carbapenem-hydrolyzing beta-lactamases, known as carbapenemases [25]. In this study, carbapenemase-encoding genes were investigated to decipher the mechanism of carbapenem resistance.

Molecular detection of carbapénèmase-encoding genes has shown that the production of OXA-type carbapenemase is predominant in *A. baumannii* [26]; the *bla*$_{OXA-51-like}$ gene was detected in all 29 strains of *A. baumannii*, *bla*$_{OXA-23}$ is frequently found in health facilities, and known as a worldwide disseminated gene [27]. Our results showed that *bla*$_{OXA-23}$ was the second predominant mechanism of resistance to carbapenems, with 89.7% of prevalence rate. This rate was higher than previously reported in Senegal, showing a prevalence of 15.38% [17]. High prevalence of MDR *A. baumannii* harboring *bla*$_{OXA-23-like}$ have been reported in some African countries, namely South Africa (70%), Tunisia (93.9%) and Libya (70%) [18,19,28]. Studies carried out in South America (Argentina,

Table 5. Distribution of carbapenemase-encoding genes *A. baumannii* and its antibiotic susceptibility profiles

Antibiotic	Resistant n (%)				Susceptible n (%)				p
	***bla*$_{OXA-23}$**	***bla*$_{NDM-1}$**	***bla*$_{TEM-2}$**	***bla*$_{OXA-23}$ and *bla*$_{TEM-2}$**	***bla*$_{OXA-23}$**	***bla*$_{NDM-1}$**	***bla*$_{TEM-2}$**	***bla*$_{OXA-23}$ and *bla*$_{TEM-2}$**	
Amoxicillin/Clavulanic acid	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	
Ticarcillin/Clavulanic acid	25 (86.2%)	1 (100%)	2 (100%)	1 (100%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Amikacin	7 (28%)	0 (0%)	0 (0%)	0 (0%)	18 (72%)	1 (100%)	2 (100%)	1 (100%)	.687
Aztreonam	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Cefepime	25 (100%)	1 (100%)	0(0%)	0 (0%)	0 (0%)	0 (0%)	2 (100%)	1 (100%)	**< .001**
Cefoxitin	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Ceftazidim	23 (92%)	1 (100%°	0 (0%)	0 (0%)	2 (8%)	0 (0%)	2 (100%)	1 (100%)	**< .001**
Ciprofloxacin	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Colistin	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Gentamicin	21 (84%)	1 (100%)	2 (100%)	1 (100%)	4 (16%)	0 (0%)	0 (0%)	0 (0%)	.863
Piperacillin	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Imipenem	24 (96%)	0 (0%)	0 (0%)	0 (0%	1 (4%)	1 (100%)	2 (100%)	1 100%)	**<.001**
Meropenem	24 (96%)	0 (0%)	0 (0%)	0 (0%	1 (4%)	1 (100%)	2 (100%)	1 100%)	**<.001**
Piperacillin/Tazobactam	25 (100%)	1 (100%)	2 (100%)	0 (0%)	0(100%)	0 (0%)	0 (0%)	1 (100%)	**< .001**
Ticarcillin	25 (100%)	1 (100%)	2 (100%)	1 (3.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	-
Tobramycin	19 (95%)	1 (100%)	0 (0%)	0 (0%)	6 (5%)	0 (0%)	2 (100%)	1 (100%)	**< .001**

Ecuador, Chile, Bolivia, Uruguay, and Paraguay) showed that the *bla*$_{OXA-23}$ gene was recovered in all participating medical centers and in all isolates from seven of nine centers with an overall prevalence of 76.9% [29]. In Asia, particularly in Saudi Arabia, *bla*$_{OXA-23}$ was detected among isolated *A. baumannii* strains in Riyadh and the Eastern Province with 53% and 75.5% of prevalence rates, respectively [30]. In Germany, Gamal Wareth et al. showed that the *bla*$_{OXA-23}$ gene was found in 14% of isolated *A. baumannii* strains of human origin [31], which is lower than prevalence reported in our study. Regarding other β-lactamases classes, our study showed that 6.9% of the isolates had *bla*$_{TEM-2}$ gene. Bacteria synthetize this gene are generally from the *Enterobacteriaceae* family, and this gene was described from *Escherichia coli* and *Klebsiella* clinical isolates in India, and subsequently reported in *A. baumannii strains* [26].

However 1 strain harbored the gene coding for metallo-β-lactamase NDM-1. *Bla*$_{NDM-1}$ were carried along with *bla*$_{OXA-\ 51}$. The prevalence of *bla*$_{NDM-1}$ was 3.4% which is lower than recently reported in Africa namely Nigeria (38.1%) [32], Tunisia (3.6%) [19].

4. CONCLUSION

Carbapenem resistance in Senegalese strains of *A. baumannii* is predominantly due to the worldwide disseminated gene *bla*$_{OXA-23}$, with a subset of strains due to NDM-1. These AB-MDR strains were mainly isolated in ICUs with limited treatment options. Nevertheless, the use of colistin, a polypeptide antibiotic, as an alternative for infections caused by carbapenem-resistant Gram-negative bacilli should be considered in Senegal. Systemic molecular surveillance in the “One Health” network should be established for further efficient monitoring of MDR strains.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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