JIDC | THE JOURNAL OF INFECTION IN DEVELOPING COUNTRIES

Original Article

Emergence of NDM and OXA-48-like producing Enterobacterales in Northern Morocco

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Abstract

Introduction: Carbapenemase-producing Enterobacterales (CPE) have widely emerged as a global health threat due to the spread of resistance genes such as New Delhi metallo-β-lactamase (*bla*$_{NDM}$) and oxacillinase-48-like β-lactamases (*bla*$_{OXA-48-like}$). Data on the molecular epidemiology of CPE in northern Morocco remain limited. Hence, this study aimed to report the epidemiological profile of carbapenem-resistant Enterobacterales and their molecular analysis.

Methodology: A prospective study was conducted from July 2024 to July 2025 at Mohammed VI University Hospital, Tangier. Ninety-two non-duplicate Enterobacterales isolates were collected across different departments. Species were identified via the matrix-assisted laser desorption/ionization - time-of-flight (MALDI-TOF MS) technique and antimicrobial susceptibility testing was performed according to the guidelines of the European Committee on Antimicrobial Susceptibility Testing (EUCAST). Carbapenemase production was screened using GeneXpert® Carba-R (Cepheid, Sunnyvale, USA). Associations between clinical, microbiological, and molecular data were assessed using Chi square or Fisher's exact tests.

Results: *Klebsiella pneumoniae* dominated across all isolates (75/92; 81.5%), followed by *Escherichia. coli* (8/92; 8.7%). *Bla*$_{NDM}$ was found in 65/92 (70.6%), *bla*$_{OXA-48-like}$ in 17/92 (18.5%), and both in 10/92 (10.9%). Most cases occurred in neonatal (38%) and intensive care units (31.5%). Resistance exceeded 70% for several major antibiotics; 29.3% were colistin-resistant. Mortality was 46.7%. Diabetes ($p < 0.01$) and severe burns ($p < 0.01$) were significantly associated with carbapenemase carriage.

Conclusions: CPE, especially NDM-producing *Klebsiella pneumoniae*, represent a major threat in northern Morocco, notably in neonatal and critical care units. Their high resistance and mortality highlight the urgent need for reinforced infection control, molecular surveillance, and rapid diagnostics.

Key words: antimicrobial resistance; carbapenemase; Enterobacteriaceae; NDM; OXA 48; Morocco.

J Infect Dev Ctries 2026; 20(8):1179-1186. doi:10.3855/jidc.22277

(Received 25 August 2025 – Accepted 23 February 2026)

Introduction

Carbapenem-resistant Enterobacterales (CRE) have emerged as a major global health concern due to their ability to evade nearly all available antibiotic treatments, including carbapenems, which are often considered the last line of defense against multidrug-resistant Gram-negative bacteria [1]. Poor infection control practices, such as inadequate screening for asymptomatic carriers and postponed contact precaution implementation, are frequently linked to the persistence of CRE [2]. Thus, preventing and containing the spread of these pathogens effectively is essential. Recognizing the severity of this threat, the World Health Organization has designated CRE as critical priority pathogens, underscoring the urgent need for novel antimicrobial agents and robust infection control measures [3].

Carbapenem resistance in Enterobacterales is largely mediated by the production of carbapenems enzymes, which is commonly associated with other mechanisms, including the decreased permeability of the outer membrane and/or the overexpression of efflux pumps [4]. According to the molecular categories of the Ambler classification, carbapenemases can be classified as Class A carbapenemases (e.g., *Klebsiella pneumoniae* carbapenemase (KPC)), Class B metallo-β-lactamases (e.g., New Delhi metallo-β-lactamase (NDM), Verona integron-encoded metallo-β-lactamase (VIM), imipenemase (IMP)), and Class D carbapenemases, including the increasingly more common class D carbapenemases encoded by *bla*$_{OXA-48-like}$ [5].

The New Delhi metallo-β-lactamase (NDM) and OXA-48-like carbapenemases are two of the most concerning carbapenemases as they are distributed worldwide and are often isolated from both hospital and

community sources [4]. NDM was first described in 2008 in a *Klebsiella pneumoniae* strain isolated from an Indian patient and is currently endemic in the Indian subcontinent, Middle East, and North Africa [6]. NDM-producing strains are of particular concern, as they frequently co-harbor many resistance determinants that are both plasmid- and chromosomal-encoded, thus resulting in a high level of multidrug resistance, with very limited therapeutic options [7,8].

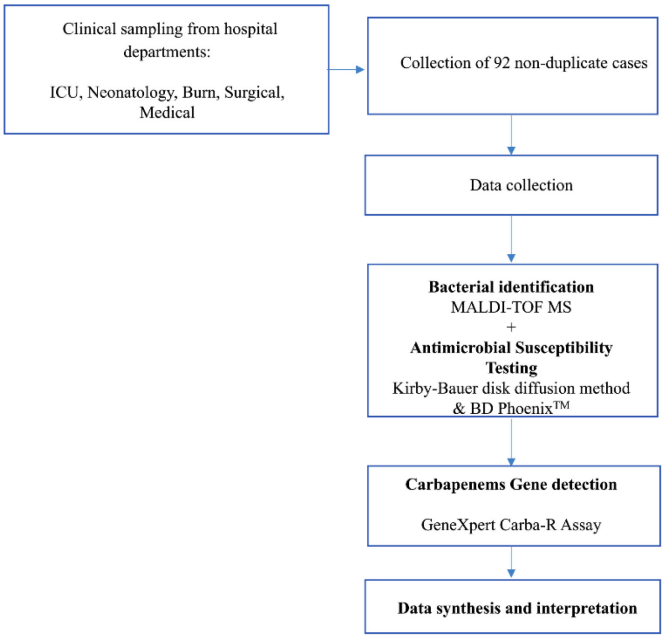
On the other hand, OXA-48-like carbapenemases were originally reported in Turkey, and since then, widely spread in the Mediterranean and Europe [9]. Their dissemination is promoted by efficient plasmid dissemination, and the often-limited spectrum of resistance they confer can escape attention in case of early exposure to classical susceptibility testing [10].

The high mobilization of these resistance determinants is mainly due to the active movement of mobile genetic elements such as plasmids and transposons. These contribute to both intra- and interspecies gene transfer and frequently contain multiple resistance genes. Consequently, they often provide cross-resistance to other important antibiotic categories, including aminoglycosides, fluoroquinolones, and polymyxins; hence leading to the development of extensively drug-resistant (XDR) and even pan-drug-resistant (PDR) bacterial strains [11,12].

The molecular epidemiology of CRE has been insufficiently studied in Morocco, especially in the northern regions. Knowledge of regional patterns of carbapenem resistance is important for therapy and infection control.

This study was performed in the University Hospital of Tangier to analyze the epidemiological trends, resistance patterns, and clinical aspects of carbapenemase-producing Enterobacteriaceae. The study was performed with the GeneXpert® Carba-R which is reported to have excellent diagnostic accuracy for carbapenemase-producing organisms [13,14]. The aim was to detect the major carbapenemase-encoding genes' NDM and OXA-48-like variants to contribute to the assessment of the extent of CRE circulation within the North African region, and improve the fine-tuning of CRE-targeted infection control measures as recommended by the CRE guidelines.

Figure 1. Study methodology.



Methodology

Patients and study design

This was a one-year prospective observational study carried out from July 2024 to July 2025 at the Mohammed VI University Hospital (CHU) in Tangier, Morocco, a regional tertiary care center. A series of 92 non-duplicate clinical Enterobacterales isolates were obtained from different hospital wards: intensive care, neonatology, burn unit, surgical, and medical.

The study was conducted according to the ethical rules of the Declaration of Helsinki, and anonymity and rights of the participants were secured [15].

Data collection

Data on demographics and clinical features were collected from the patients' charts and hospital electronic health records. The information collected included patient age, gender, hospital ward, type of specimen, site of infection, previous use of antibiotics, and clinical course. These factors were chosen to investigate possible etiology and epidemiological characteristics related to CPE infections.

Bacterial identification

Isolates of positive clinical strains were identified by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI TOF; Bruker Daltonics, Bremen, Germany) [16,17]. This technique enables rapid and accurate species-level identification by analyzing unique proteomic fingerprints [18].

Antimicrobial susceptibility testing

Antimicrobial susceptibility testing was performed by the Kirby-Bauer disk diffusion method and verified by BD Phoenix™ (Becton Dickinson, Sparkes, Maryland, USA) minimum inhibitory concentration (MIC) panels according to the European society of

Clinical Microbiology and Infectious Diseases guidelines (EUCAST) [19]. The resistance breakpoints used were as follows: inhibition zones of ≤ 22 mm for ertapenem, ≤ 19 mm for imipenem or meropenem, MICs of ≥ 0.5 mg/L for ertapenem, and ≥ 2 mg/L for imipenem and meropenem. Multidrug resistant (MDR), corresponding to non-susceptibility to at least one agent in three or more antimicrobial categories; extensively drug-resistant (XDR), corresponding to non-susceptibility to at least one agent in all but two or fewer antimicrobial categories; and pan-drug-resistant (PDR), corresponding to non-susceptibility to all agents in all antimicrobial categories tested; were classified according to international standards [20].

Detection of carbapenemase genes

The GeneXpert Carba-R assay (Cepheid, Sunnyvale, California, USA) was used for molecular detection of carbapenemase genes, targeting the five major carbapenemase genes (bla_{KPC}, bla_{NDM}, bla_{VIM}, bla_{IMP}, and $bla_{OXA-48-like}$). This system combines sample preparation, DNA extraction, amplification, and target sequence detection in disposable prefilled assay cartridges. Xpert® Carba-R cartridges contain polymerase chain reaction (PCR) buffer, DNA polymerase, and two internal quality controls. This real-time PCR system enables high-speed detection (approximately 1 hour) of carbapenemase-producing organisms directly from bacterial isolates, allowing for decisions to be made in a timely manner for therapy and infection control. It has been shown to be highly sensitive and specific in several studies [21].

Statistical analysis

The collected data were analyzed with IBM SPSS Statistics for Windows version 21.0. (IBM Corp, Armonk, NY, USA). Descriptive statistics were used to summarize the data, including patient demographics, clinical characteristics, types of clinical samples, and department distribution.

Quantitative variables, such as age, MALDI TOF score, and length of hospital stay, were presented as means and standard deviations; while qualitative variables were expressed as frequencies and percentages. The Chi-square test or Fisher's exact test was employed to assess associations between categorical variables. A p value of less than 0.05 was considered statistically significant.

A flow chart summarizing the study methodology is presented in Figure 1.

Results

A total of 92 patients with suspected CPE infections were included in the study. Among them, 65.2% (n = 60) were male and 34.8% (n = 32) were female. The mortality rate within the sample was 46.7%.

Patients were hospitalized in various units: 38.0% (n = 35) in the neonatal unit, 31.5% (n = 29) in intensive care units (ICUs), 16.3% (n = 15) in medical wards, 8.7% (n = 8) in surgical wards, and 5.5% (n = 5) in the burn unit. The mean duration of hospitalization was 22 days (range: 1–100 days), with 57.6% (n = 53) of patients hospitalized for more than 14 days.

Regarding age distribution, 47.8% (n = 44) were under 1 year of age, 5.4% (n = 5) were aged 1–18 years, 27.2% (n = 25) were between 19 and 64 years, and 19.6% (n = 18) were aged 65 years or older.

Prior colonization with multidrug-resistant organisms (MDROs) was observed in 21.7% (n = 20), recent antibiotic use in 47.8% (n = 44), mechanical ventilation in 41.3% (n = 38), recent surgical intervention in 38.0% (n = 35), and invasive medical devices were present in 92.4% (n = 85) of patients.

Comorbidities were common; 25.0% (n = 23) had cardiovascular disease, 13.0% (n = 12) had diabetes, 9.8% (n = 9) had chronic lung disease, and 7.6% (n = 7) had chronic kidney disease. Less frequent conditions included hepatopathies (n = 4), neuropathies (n = 4), malignancies (n = 3), hematologic disorders (n = 2), and extensive burns (n = 5).

Clinical isolates were most commonly recovered from urine samples (34.8%, n = 32), followed by blood cultures (31.5%, n = 29), respiratory samples (12.0%, n = 11), catheters (10.9%, n = 10), pus (7.6%, n = 7), cerebrospinal fluid (2.2%, n = 2), and joint fluid (1%, n = 1).

Microbiological identification revealed *Klebsiella pneumoniae* as the predominant pathogen, accounting for 81.5% (n = 75) of isolates, followed by *Escherichia coli* (8.7%, n = 8), *Enterobacter cloacae* (5.4%, n = 5), *Serratia marcescens* (3.3%, n = 3), and *Citrobacter freundii* (1.1%, n = 1). Proteomic fingerprinting via MALDI-TOF showed a mean score of 2.27 (SD ± 0.15, range 1.79–2.51), ensuring reliable species identification across all isolates.

All isolates were resistant to ertapenem (100%; n = 92), and 83.7% were resistant to both meropenem and imipenem (n = 77); these isolates were therefore classified as suspected CPE.

Molecular testing via the GeneXpert® Carba-R assay identified carbapenemase genes in all isolates: 70.6% (n = 65) of isolates harbored the bla_{NDM} gene, 18.5% (n = 17) $bla_{OXA-48-like}$, and 10.9% (n = 10) carried

the *bla*$_{NDM}$ and *bla*$_{OXA-48-like}$ genes simultaneously (Figure 2). Additional characteristics and microbiological data are summarized in Table 1 and Table 2.

Antimicrobial susceptibility testing showed high resistance rates: 89.1% to gentamicin, 73.9% to amikacin, 87.0% to ciprofloxacin, and 93.5% to trimethoprim-sulfamethoxazole. Resistance to colistin was observed in 29.3% of isolates (Figure 3). Antibiotic susceptibility varied among isolates. *Klebsiella pneumoniae* and *Escherichia coli* showed the highest susceptibility to amikacin, while resistance to gentamicin and Bactrim was widespread across most isolates (Table 3).

Several parameters showed statistically significant associations with the presence of carbapenemase genes. Clinically, diabetes mellitus ($p = 0.002$) and severe burn injuries ($p = 0.008$) were significantly linked to gene carriage. From a microbiological perspective, bacterial species distribution was also significant ($p = 0.023$), with *Klebsiella pneumoniae* predominating among

Table 1. Demographic and clinical characteristics of the patients.

Variables	N (%)
Age	
0 to 1 years	44 (47.8)
1 to 18 years	5 (5.4)
19 to 64 years	25 (27.2)
> 65 years	18 (19.6)
Gender	
Female	32 (34.8)
Male	60 (65.2)
Department	
ICU	29 (31.5)
Neonatology	35 (38)
Medical ward	15 (16.3)
Surgical ward	8 (8.7)
Burn unit	5 (5.5)
Comorbidities	
Diabetes	12 (13)
Cardiopathy	23 (25)
Nephropathy	7 (7.6)
Hepatopathy	4 (4.3)
Hemopathy	2 (2.8)
Neuropathy	4 (4.3)
Pneumopathy	9 (9.8)
Burns	5 (5.4)
Cancer	3 (3.3)
Immunosuppressive therapy	8 (8,7)
History of multi-drug-resistant bacteria	20 (21.7)
Recent antibiotic use	44 (47.8)
ICU stay	70 (76.1)
Extended hospital stay (> 14 days)	53 (57.6)
Mechanical ventilation	38 (41.3)
Recent surgery	35 (38)
Medical devices	85 (92.4)
Mortality	43 (46.7)
Resistance to other antibiotics	
Gentamicin	82 (89.1)
Amikacin	68 (73.9)
Bactrim	86 (93.5)
Ciprofloxacin	80 (87)
Colistin	27 (29.3)

Figure 2. Distribution of Carbapenemase genes Among isolates (NDM (New Delhi Metallo-β-lactamase), OXA-48-like (Oxacillinase-48-like β-lactamases) and Co-producers).

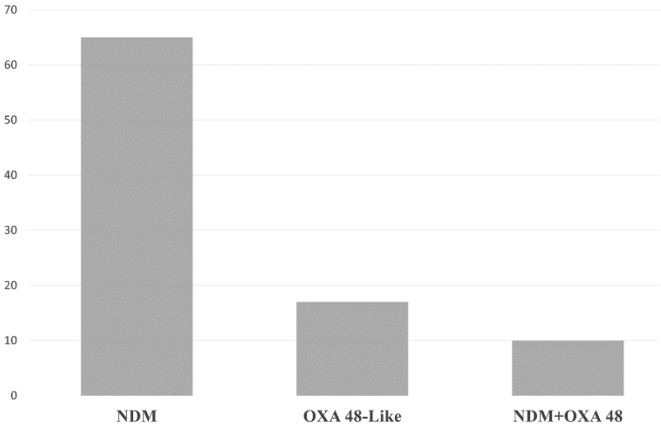


Figure 3. Antibiotic Resistance Rates Among Clinical Isolates. NDM, New Delhi Metallo-β-lactamase; OXA-48-like, Oxacillinase-48-like β-lactamases.

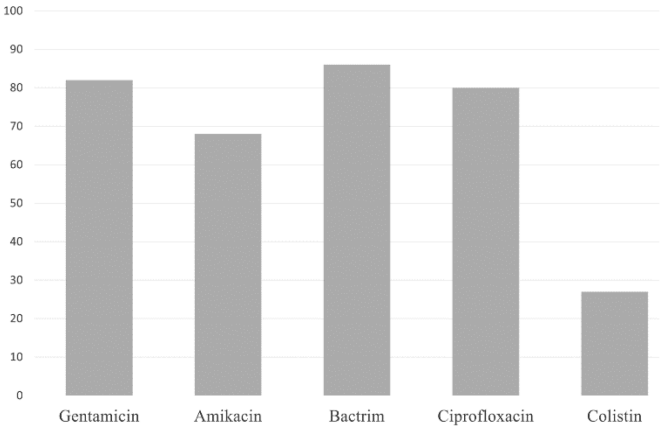


Table 2. Summary of sample characteristics and GeneXpert detection.

Sample type	N (%)
Urine	32 (34.8)
Blood culture	29 (31.5)
Protected distal sampling	11 (12)
PUS	7 (7.6)
Cerebrospinal fluid	2 (2.2)
Catheter	10 (10.9)
Synovial fluid	1 (1)
Species identified	
Klebsiella pneumoniae	75 (81.5)
Escherichia coli	8 (8.7)
Citrobacter freundi	1 (1.1)
Serratia marcescens	3 (3.3)
Enterobacter cloacae	5 (5.4)
GeneXpert results	
NDM	65 (70.7)
OXA 48-Like	17 (18.5)
NDM OXA 48-Like	10 (10.9)

NDM: New Delhi metallo-β-lactamase; OXA 48-like: oxacillinase-48-like β-lactamase; ICU: intensive care unit; MDR: multidrug-resistant.

Table 3. Antibiotic susceptibility of the isolates.

Antibiotics	Species					Total
	Klebsiella pneumoniae	*Escherichia coli*	*Citrobacter freundii*	*Enterobacter cloacae*	*Serratia marcescens*	
Gentamicin	2	6	0	0	2	10
Amikacin	13	6	1	2	2	24
Bactrim	0	4	0	0	2	6
Ciprofloxacin	6	4	0	0	2	12
Colistin	51	8	1	5	0	92

isolates carrying *bla*$_{NDM}$ and multiple resistance genes (Supplementary Table 1).

Antibiotic resistance profiles revealed strong associations with carbapenemase genotypes, particularly resistance to amikacin ($p < 0.001$), ciprofloxacin ($p < 0.001$), gentamicin ($p < 0.001$), trimethoprim-sulfamethoxazole ($p = 0.021$), and colistin ($p = 0.004$). Lastly, hospital ward distribution was another significant factor ($p = 0.013$), with ICUs and neonatal wards showing the highest prevalence of *bla*$_{NDM}$ multi-gene positive isolates.

Several parameters showed statistically significant associations with the presence of carbapenemase genes. Clinically, diabetes mellitus ($p = 0.002$) and severe burn injuries ($p = 0.008$) were significantly associated with carbapenemase gene carriage. Bacterial species distribution was also significant ($p = 0.023$), with *Klebsiella pneumoniae* being the predominant species among isolates carrying *bla*$_{NDM}$ and multiple resistance genes.

Antibiotic resistance profiles demonstrated strong associations with specific carbapenemase genotypes, particularly resistance to amikacin ($p < 0.001$), ciprofloxacin ($p < 0.001$), gentamicin ($p < 0.001$), trimethoprim-sulfamethoxazole ($p = 0.021$), and colistin ($p = 0.004$).

Finally, hospital ward distribution was another associated factor ($p = 0.013$), with ICUs and neonatal wards showing the highest prevalence of *bla*$_{NDM}$ multi-gene positive isolates (Table 3).

Discussion

The results of this study describe the epidemiological characteristics of CPE from northern Morocco. The *bla*$_{NDM}$ gene was found in 70.6% of isolates. Both *bla*$_{OXA-48-like}$ and *bla*$_{NDM}$ were found in only 10.9%, and *bla*$_{OXA-48-like}$ in only 18.5%. This distribution is different from previous national data, including the 2018 study conducted in Marrakech, that described *bla*$_{OXA-48-like}$ in 30.5% and *bla*$_{NDM}$ in 20.6% of the isolates, with a much higher co-expression rate of the two genes (29%), suggesting potential inter-regional geographical and/or temporal differences in circulating resistance mechanisms [22,23].

The lower carriage rate seen in this study may be due to changes in clonality or regional variation in plasmid spread. It is worth noting that the findings are similar to a previous report from burn units in Tunisia, where *bla*$_{NDM}$ was found to be the most prevalent carbapenemase gene among the carbapenemases that were detected, with an overall rate of 59%; followed by *bla*$_{OXA-48-like}$, detected by 33% in monospecies and 7% in coproduction [24].

Interestingly, in the present study, burn was found to be significantly correlated to the carriage of carbapenemase genes ($p = 0.008$); in line with the observation reported in Tunisia, where burn patients were a major issue for the emergence and spread of MDR organisms because of long-term hospitalization, large antibiotic regimen, and immunosuppressed situation [24,25].

The distribution of genotypes across hospital wards was also notable; neonatal units (38%) and ICUs (31.5%) had the majority of NDM and dual-gene isolates, reflecting global reports identifying these high-dependency settings as epicenters of CPE transmission, driven by invasive care, broad-spectrum antimicrobial use, and immature or compromised host defenses [26–29]. Moreover, diabetes mellitus was significantly associated with carbapenemase carriage ($p = 0.002$), consistent with other findings that hyperglycemia-related immune dysfunction and repeated healthcare exposures predispose diabetic patients to CPE colonization and infection [26,30].

As for the isolates' species, *Klebsiella pneumoniae* was the most frequently isolated, accounting for 81.5% of cases. Its presence showed a significant association with NDM and co-harboring isolates. This is consistent with worldwide epidemiology and is a major driver of the OXA-48-like enzyme spread in Europe [31], as well as the main source of dissemination of NDM in Asia and North Africa [32–34].

The resistance profile of these isolates was particularly alarming. The resistance rates were high for gentamicin (89.1%), amikacin (73.9%), ciprofloxacin (87%), trimethoprim-sulfamethoxazole (93.5%), and particularly for colistin (29.3%); all significantly associated with carbapenemase production. [35,36].

These findings align with regional and international reports, including outbreaks in Tunisia involving

colistin-resistant *Klebsiella pneumoniae* ST101 strains co-producing NDM and OXA-48 [37], as well as strains from India and China where NDM frequently coexists with resistance determinants such as 16S rRNA methyltransferases (*armA*, *rmtB*) and *mcr-1*, further compounding resistance to aminoglycosides and colistin [38,39].

NDM represents nearly 30% of CPE cases in Western Europe, mainly associated with imported infections or local outbreaks [40]. Importantly, the co-production of NDM and $bla_{\text{OXA-48-like}}$ enzymes is increasingly being identified (up to 5–10% of cases in some settings, particularly in the United Kingdom) [41,42]. In South and Southeast Asia, NDM is still the most common carbapenemase, causing more than 50% of CPE cases, while OXA-48-like enzymes, particularly the OXA-181 and the OXA-232, rank second in prevalence. The prevalence of dual NDM + OXA-48 producers is also increasing in countries such as Thailand and India [43]. In East Asia, however, where IMP and VIM have been most prevalent, recent evidence suggests that NDM-1 and NDM-5 have emerged as the predominant variants among CPE [32].

It is also interesting to note that similar rates of NDM and OXA-48-like enzymes were identified from African CPE isolates in a previously published systematic review, with each constituting 43% of reported cases [44]. Nevertheless, large disparities exist across both regions and within countries. In other regions, such as Batna, Algeria, $bla_{\text{OXA-48-like}}$ was highly prevalent (82.1%), while NDM was the least prevalent (5.6%) [45]. Conversely, one multicenter survey from Saudi Arabia including 13 hospitals and 519 isolates revealed $bla_{\text{OXA-48-like}}$ in 71.2% of isolates, bla_{NDM} in 20.7%, and co-carriage of the genes in 8% of isolates [46].

However, the distribution of these genes in the city of Jazan (2020–2021) was slightly different ($bla_{\text{OXA-48-like}}$ 65.1% and bla_{NDM} 9.3%) [47]. Recent studies in Riyadh ICUs had also described ongoing outbreaks of *Klebsiella pneumoniae* strains with NDM-1 and OXA-48, some of which were colistin-resistant [48]. Clinically, these epidemiological patterns have important implications. The overall mortality rate in the present study was 46.7%, aligning with global reports that document mortality rates ranging from 30% to 60% in CPE infections [49]. Although no statistically significant differences in mortality were observed between genotypes in this study, previous research indicates that NDM-associated infections may be linked to poorer clinical outcomes, largely due to XDR and limited treatment options [50].

Strengths and limitations

The prospective design and inclusion of isolates from multiple hospital wards strengthen the representativeness of the data. On the other hand, the study was limited by its single-center design, affecting generalizability; and by the relatively small sample size.

Conclusions

The findings of this study reveal a significant outbreak of CPE in northern Morocco, predominantly involving *Klebsiella pneumoniae* strains carrying bla_{NDM} and $bla_{\text{OXA-48-like}}$ genes. These resistant strains were primarily isolated from neonatal units and ICUs, affecting vulnerable populations such as those with burns or diabetes. A high rate of MDR was observed and this underscores the urgent need for reinforced infection control measures, ongoing molecular surveillance, and rapid diagnostic strategies.

Acknowledgements

The authors would like to thank the staff of the participating laboratory and hospital for their technical assistance and support during sample collection and analysis.

Authors' contributions

ME-H, KR: study conception and design; KR: supervision, critical revision of the manuscript; AH, EMEG, HEM, KEH, NC: data collection, sample processing, laboratory analyses, manuscript writing; RAS: data interpretation; YA, ME-H: initial draft of manuscript, data analysis, results interpretation.

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Conflict of interest

No conflict of interest is declared.

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Annex – Supplementary Items

Supplementary Table 1. Distribution of carbapenemase genes (*NDM*, *OXA-48-like*, and co-producing isolates) according to patients' characteristics.

Variables	*NDM* (n = 65)	*OXA 48-like* (n = 17)	*NDM- OXA-48 like* (n = 10)	*p* value
Age				
0 to 1 years	35	4	5	
1 to 18 years	4	1	0	0.111
19 to 64 years	18	5	2	
> 65 years	8	7	3	
Gender				
Male	43	9	8	0.342
Female	22	8	2	
Department				
Neonatology	28	2	5	
ICU	19	7	3	
Medical ward	9	4	2	0.013
Surgical ward	8	0	0	
Burn unit	1	4	0	
Sample type				
Urine	18	9	5	
Blood culture	24	3	2	
Protected distal sampling	6	3	2	
Pus	6	1	0	0.214
Cerebrospinal fluid	2	0	0	
Material	9	1	0	
Synovial fluid	0	0	1	
Comorbidity				
Diabetes				
Yes	4	7	1	0.002
No	61	10	9	
Cardiopathy				
Yes	14	6	3	0.442
No	51	11	7	
Nephropathy				
Yes	6	1	0	1.000
No	59	16	10	
Hepatopathy				
Yes	3	0	1	0.492
No	62	17	9	
Hemopathy				
Yes	1	1	0	0.508
No	63	16	10	
Neuropathy				
Yes	4	0	0	0.734
No	61	17	10	
Pneumopathy				
Yes	8	0	1	0.266
No	57	17	9	
Burns				
Yes	1	4	0	0.008
No	64	13	10	
Cancer				
Yes	3	0	0	1.000
No	62	17	10	
Immunosuppressive therapy				
Yes	7	1	0	0.849
No	58	16	10	
History of BMR				
Yes	15	4	1	0.786
No	50	13	9	
Recent antibiotic use				
Yes	33	8	3	0.508
No	32	9	7	
ICU Stay				
Yes	48	13	9	0.630
No	17	4	1	
Extended hospital stay (> 14 days)				
Yes	38	10	5	0.892
No	27	7	5	

Variables	*NDM* (n = 65)	*OXA 48-like* (n = 17)	*NDM- OXA-48 like* (n = 10)	*p* value
Mechanical ventilation				
Yes	28	7	3	0.796
No	37	10	7	
Recent surgery				
Yes	25	7	3	0.888
No	40	10	7	
Invasive medical devices				
Yes	60	16	9	1.000
No	5	1	1	
Mortality				
Recovery	31	11	7	0.272
Death	34	6	3	
Resistance to other antibiotics				
Gentamicin	63	10	9	0.000
Amikacin	58	3	7	0.000
Trimethoprim-sulfamethoxazole (TMP-SMX)	63	13	10	0.021
Ciprofloxacin	63	9	8	0.000
Colistin	23	0	4	0.004
Species				
Klebsiella pneumoniae	56	10	9	0.023
Escherichia coli	2	5	1	
Citrobacter freundi	0	1	0	
Serratia marcescens	3	0	0	
Enterobacter cloacae	4	1	0	

NDM: New Delhi Metallo-β-lactamase; OXA 48-like: oxacillinase-48-like β-lactamase; ICU: intensive care unit; MDR: multidrug-resistant.