ORIGINAL ARTICLE

Epidemiology, Clinical Characteristics, and Outcomes of Carbapenem-Resistant Enterobacterales in a Specialist Hospital in Sabah, Malaysia: A Retrospective Cohort Study.

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Abstract
Introduction: Carbapenem-resistant Enterobacterales (CRE) represent a growing global health threat associated with high mortality, limited treatment options, and substantial healthcare burden. In Malaysia, increasing carbapenem resistance among Enterobacterales has raised concerns, yet data from East Malaysia remain scarce. This study aimed to describe the epidemiology, clinical characteristics, microbiological profiles, and outcomes of patients with CRE in a major referral hospital in Sabah, Malaysia, and to identify factors associated with CRE infection. **Methods:** We conducted a single-centre retrospective cohort study at Hospital Queen Elizabeth II involving adult patients with at least one CRE-positive culture between January 2020 and December 2021. CRE was defined according to CDC criteria. Patients were classified as having CRE infection or colonisation based on clinical and laboratory findings. Demographic data, healthcare exposures, antimicrobial use, microbiological characteristics, and outcomes were extracted from medical records. The primary outcome was 30-day all-cause in-hospital mortality. **Results:** A total of 101 patients were included, of whom 40 (39.6%) had CRE infection and 61 (60.4%) had CRE colonisation. The median time to CRE detection was approximately two weeks of hospitalization. *Klebsiella pneumoniae* was the predominant organism (68.3%), with New Delhi metallo-β-lactamase (NDM) identified in 79.2% of isolates. Prior exposure to broad-spectrum antibiotics, invasive devices, ICU admission, and recent surgery were common. Thirty-day in-hospital mortality was significantly higher among patients with CRE infection compared with colonisation (52.5% vs 21.3%). Higher Charlson comorbidity index was associated with mortality, while delays in CRE identification were frequently observed. **Conclusion:** CRE colonisation and infection impose a substantial clinical burden in this tertiary hospital, with high mortality among infected patients and limited therapeutic options. Strengthened infection prevention measures, targeted surveillance, optimized antimicrobial stewardship, and improved access to effective anti-CRE therapies are urgently needed to mitigate CRE transmission and improve patient outcomes.
Keywords: *Antimicrobial stewardship, carbapenem-resistant enterobacterales, CRE colonisation, CRE infection, infection prevention and control.*

Introduction

Carbapenems belong to the β-lactam class of antibiotics and possess broad-spectrum bactericidal properties, making them important agents for treating severe infections due to multidrug-resistant Gram-negative bacteria [1]. Nevertheless, the global emergence of carbapenem-resistant Enterobacterales (CRE) threatens to compromise the effectiveness of carbapenems. In Malaysia, surveillance data have demonstrated a concerning rise in carbapenem resistance among *Klebsiella pneumoniae*, with meropenem and imipenem resistance rates increasing approximately two-fold over a five-year period between 2017 to 2021, from 2.9% and 2.7% to 5.6% and 4.9%, respectively [2].

The emergence and spread of CRE represent a significant public health challenge, as they often cause infections with higher mortality, prolonged hospitalisation, and poorer clinical outcomes compared with carbapenem-susceptible infections [3,4]. CRE has been designated an urgent antimicrobial resistance concern by the United States Centers for Disease Control and Prevention due to its substantial resistance profile, scarcity of effective treatments, and the expense and toxicity linked to newer antimicrobial therapies [5,6]. Reported risk factors for CRE infection include exposure to broad-spectrum antibiotics, presence of indwelling medical devices, prolonged hospitalisation, and admission to intensive care units [7–9]. Moreover, CRE colonisation—especially among critically ill patients—has been shown to significantly increase the risk of subsequent invasive infection [10,11].

Hospital Queen Elizabeth II (HQE II), a major specialist and referral hospital for Sabah's west coast, offers extensive tertiary and subspecialty services. Local surveillance has identified a 2.4-fold increase in CRE-positive cultures between 2020 and 2021, raising concerns regarding the burden and transmission of CRE within the institution. Nevertheless, data describing the epidemiology, clinical characteristics, and risk factors associated with CRE in Malaysia—particularly in the Borneo region—remain limited. Therefore, this study aims to describe the epidemiology and clinical characteristics of patients with CRE-positive cultures in HQE II and to identify factors associated with true CRE infection. Additionally, we seek to characterise the microbiological profile and antimicrobial susceptibility patterns of CRE isolates encountered in our setting. Findings from this study may enhance clinicians' understanding of CRE colonisation and infection, help stratify patient risk, and inform targeted surveillance and infection prevention strategies to curb the spread of CRE.

Methods

Study design and population

This single-centre retrospective cohort study was conducted at Hospital Queen Elizabeth II (HQE II), a 300-bed specialist referral hospital in Sabah, Malaysia. Adult patients (≥18 years) with at least one positive culture for carbapenem-resistant Enterobacterales (CRE) from any clinical or surveillance specimen during hospitalisation between 1 January 2020 and 31 December 2021 were included. CRE was defined per CDC criteria as Enterobacterales resistant to at least one carbapenem (meropenem, imipenem-cilastatin, ertapenem, or doripenem) and/or carbapenemase-producing [12].

Patients were identified from infection control and microbiology records and followed retrospectively from the date of first CRE isolation. Based on clinical assessment and documentation, patients were categorised into two cohorts: CRE infection and CRE colonisation. CRE infection was defined as CRE isolation from a clinical specimen in the presence of clinical evidence of active infection, consistent with CDC surveillance principles. Evidence included signs of infection (e.g., fever >38.0°C, chills, hypotension, or localising symptoms), laboratory markers of systemic inflammation (e.g., elevated CRP or WBC), radiological evidence of infection, and/or physician documentation noting infection requiring

antimicrobial therapy. CRE colonisation was defined as CRE isolation without clinical signs or symptoms and without antimicrobial therapy for CRE infection. Only the first CRE episode per patient was included.

Data Collection

Patient data were extracted from medical records using a structured form. Variables included demographics (age, gender, race, ward, infective diagnosis, time from admission to CRE positivity, length of stay), comorbidities, healthcare exposures, microbiological characteristics, and outcomes. Healthcare exposures were defined as: prior antibiotic exposure within 6 months, ICU admission within 1-month, prior hospitalisation within 3 months, device implantation or surgical intervention within 1 year, hospitalisation lasting more than 1month, prior contact with CRE cases, and presence of invasive devices (urinary catheters or central venous lines) at the time of CRE detection. Microbiological characteristics included specimen type, CRE species, carbapenemase genes detected, and susceptibility to polymyxins. The primary outcome was all-cause 30-day in-hospital mortality, measured from the date of first CRE isolation. Patients with incomplete medical records for key variables were excluded from analysis.

Microbiological Methods

Clinical specimens included blood, tracheal aspirate, pus, sputum, urine, wound, tissue, and rectal swabs for CRE screening. Samples were cultured on agar and incubated for 24 hours. Bacterial identification was performed using MALDI-TOF (Vitek MS) or the Vitek 2 automated system with the Vitek GN card (bioMérieux, Durham, NC, USA), with *E. coli* ATCC 25922 as the reference strain for quality control.

Enterobacterales isolates underwent antimicrobial susceptibility testing using Vitek 2 AST according to CLSI guidelines [13,14]. Isolates resistant to at least one carbapenem (ertapenem, meropenem, or imipenem) were classified as CRE per CDC criteria [12]. Confirmed CRE isolates were sent to the Institute for Medical Research (IMR) for PCR-based detection of carbapenemase genes, including NDM-1, IMP, OXA-48, OXA-181, and other genes. All PCR assays at IMR were performed using validated standard protocols with positive and negative controls.

Data Analysis

Data were analysed using SPSS version 24 (15). Baseline characteristics of patients with CRE-positive cultures and risk factors for CRE infection versus colonisation were summarised using descriptive statistics. Categorical variables were presented as frequencies and percentages, and continuous variables as means (SD) or medians (IQR), depending on distribution normality. Associations between patient demographics and CRE infection versus colonisation were assessed using the Chi-square test or Fisher's exact test for categorical variables, and the Independent t-test or Mann–Whitney U test for continuous variables. Among patients with confirmed CRE infection, associations between clinical characteristics and 30-day all-cause in-hospital mortality were further examined by univariate logistic regression. If the p-value was <0.25, the corresponding characteristics were analysed using multivariate logistic regression. For all analyses, a p-value < 0.05 was considered statistically significant.

Ethical Approval

The study was conducted in accordance with the Declaration of Helsinki and Malaysian Good Clinical Practice Guidelines and was approved by the Medical Research and Ethics Committee (NMRR ID-22-00948-DCT [IIR]).

Results

Patient cohort and baseline characteristics

A total of 101 patients with CRE-positive cultures were included. Among these, 40 (39.6%) had CRE infection, and 61 (60.4%) had CRE

colonisation. The cohort was predominantly male (72.3%) with a mean age of 53.5 ± 15.3 years and a mean Charlson comorbidity index (CCI) of 2.46 ± 1.78. The median hospital stay was 30 days (IQR 17.5–49.5).

Respiratory tract infections were the most frequent primary infection site (28.7%), followed by skin and soft tissue (20.8%) and intra-abdominal infections (15.8%). The most common healthcare exposures were the presence of indwelling devices (88.1%), prior ICU admission (70.3%), and prior surgical intervention (56.4%). Regarding prior antibiotic exposure, 52.5% of patients had received piperacillin-tazobactam, and 48.5% had received carbapenem.

When comparing infection and colonisation groups, patients with CRE infection had slightly higher CCI scores (2.55 ± 1.82 vs 2.39 ± 1.76) and skin and soft tissue infections were more frequent among them. Thirty-day in-hospital mortality was higher in the CRE infection group (52.5%) than in colonised patients (21.3%). Full baseline characteristics and risk factors are detailed in Table 1, and ward distribution is illustrated in Figure 1.

CRE Isolate Characteristics

Klebsiella pneumoniae was the most common CRE species (68.3%), followed by *Enterobacter cloacae* (12.9%). Among the isolates, the NDM genotype was predominant (79.2%), while IMP genes were identified in 4% of cases. No OXA-48 or OXA-181 carbapenemase genes were detected. Approximately 10% of the isolates did not demonstrate carbapenemase genes, and seven isolates were not subjected to further genotyping.

Specimen sources differed by patient category. For CRE infection, blood samples accounted for the majority of isolates (55%), whereas colonisation was primarily detected via rectal swabs (77.1%). Table 2 provides a full summary of isolate characteristics, including species, genotypes, and specimen sources.

Clinical Factors and 30-Day In-Hospital Mortality

Among the 40 patients with CRE infection, 19 survived while 21 died within 30 days of CRE isolation. In the univariate analysis, older age and higher Charlson comorbidity index were significantly associated with 30-day in-hospital mortality ($p<0.05$), as shown in Table 3. However, these associations did not remain statistically significant in the multivariate analysis. Patients who died had a significantly shorter median length of hospital stay compared with survivors [22 days (IQR: 10 – 30.5) vs. 47 days (IQR: 20 - 55), $p=0.005$].

Discussion

This study describes an overview of the epidemiology, clinical characteristics, microbiological profiles, and outcomes of carbapenem-resistant Enterobacterales (CRE) in a major specialist hospital in Sabah, Malaysia. Importantly, this is among the few studies from East Malaysia to distinguish CRE colonisation from CRE infection. This important distinction provides clinically relevant insights into potential opportunities for early intervention in a resource-limited referral setting where prevention strategies play important roles.

In our cohort, CRE was detected after a median hospital stay of approximately two weeks. This suggests that prolonged inpatient exposure contributes greatly to both colonisation and its subsequent infection. Similar patterns have been reported elsewhere, although the timing of CRE detection varies by setting; a study from China observed earlier colonisation at 11 days, with infection developing later at 17 days of hospitalization [16]. These differences likely reflect the variation in screening intensity, patient case-mix, and infection control practices. The healthcare exposures most frequently observed among CRE-positive patients—including the presence of invasive devices [17,18], prior ICU admission [7], and recent surgical procedures [16]—are consistent with earlier studies. This is

consistent with the recognized progression of colonisation in critically ill patients followed by infection when host defences are breached. These findings highlight the importance of targeted CRE surveillance and prevention efforts, especially in high-risk populations, such as in critical care and post-operative settings.

Antibiotic exposure was common in our cohort, with nearly half of the patients receiving carbapenems or beta-lactam/beta-lactamase inhibitor (BLBLI) combinations. Although causality cannot be inferred from a retrospective standpoint, this may indicate substantial antimicrobial selection pressure within the institution. Similar associations between BLBLI use and CRE emergence have been reported elsewhere in Malaysia, Singapore, and Europe, with a study from Greece identifying anti-pseudomonal penicillin use as an independent risk factor for carbapenem-resistant *Klebsiella pneumoniae* [17,19,20]. Higher ceftazidime exposure was observed among colonised patients, which may reflect prior healthcare exposure and repeated hospital contact rather than direct causation, potentially indicating antimicrobial selection pressure in patients undergoing ongoing surveillance or recurrent hospitalisation. Locally, our institution recorded high consumption of piperacillin–tazobactam and meropenem in 2021 (66.7 and 40.2 DDD/1,000 patient-days, respectively). This further highlights the considerable antimicrobial exposure burden and the need for sustained stewardship efforts. Notably, antimicrobial stewardship activities in our centre were significantly challenged during the COVID-19 pandemic. A survey conducted in the United Kingdom reported a 64% reduction in routine antimicrobial stewardship activities, including multidisciplinary ward rounds and audits, during the pandemic [21]. Although stewardship activity was not formally evaluated in our study, similar disruptions may have occurred in our setting and could potentially have contributed to increased or prolonged broad-spectrum antibiotic use. However, this association remains speculative and cannot be directly concluded from the present data. Nevertheless, these observations highlight the potential vulnerability of antimicrobial resistance containment efforts during major healthcare system disruptions.

Interestingly, CRE colonisation was more frequently detected than CRE infection in our study, contrasting with reports from several international cohorts [16,22]. This finding likely reflects the institution's infection control policy of active CRE screening during ward transfers and systematic contact tracing following CRE detection. Consistent with this practice, documented CRE contact was substantially more common among colonised patients (65.6%) than infected patients (27.5%) in our cohort, suggesting that enhanced surveillance and contact tracing preferentially identified asymptomatic carriers before progression to invasive infection. This approach aligns with international recommendations advocating early identification of both symptomatic and asymptomatic CRE carriers [23,24]. The predominance of colonisation suggests a critical window for intervention, during which infection prevention measures—such as isolation, device review, and antimicrobial optimization—may reduce progression to invasive disease. A meta-analysis reported a 16.5% risk of developing CRE infection following colonisation, emphasizing that a substantial proportion of colonised patients progress to infection [25]. Therefore, proactive surveillance remains important in limiting both individual patient risk and nosocomial transmission.

Consistent with the national surveillance data, *Klebsiella pneumoniae* was the predominant CRE organism in our cohort, with New Delhi metallo-β-lactamase (NDM) being the most frequently identified carbapenemase gene [26]. Interestingly, all IMP-producing isolates were observed exclusively in the infection group, which may suggest a higher virulence or pathogenic potential, although this observation is limited by small numbers. The high prevalence of carbapenemase-producing CRE is very

concerning, as these organisms are able to transmit resistance via mobile genetic elements and are associated with a higher risk of progression from colonisation to infection [27,28]. Studies have shown that patients colonized with CP-CRE have a markedly higher likelihood of developing infection during the same hospitalization compared to non-CP-CRE carriers (36% vs. 3%) [29]. These findings reflect the predominance of NDM-producing CRE in both our cohort and the region's dominance and thus reinforce the need for robust infection control and access to molecular surveillance.

Treatment of CRE infection in our setting remains challenging, particularly due to the predominance of NDM-producing isolates and limited access to newer anti-CRE therapies. Although interpretation of polymyxin susceptibility is constrained by substantial missing data, reduced susceptibility was observed among some tested isolates, raising concerns regarding the effectiveness of currently available treatment options. Despite international guidelines recommendation of newer agents such as ceftazidime–avibactam plus aztreonam or cefiderocol for NDM-producing CRE [6]; access to these therapies remains limited in many low- and middle-income countries, including Malaysia due to cost and availability. These findings underscore the importance of prioritizing resource allocation for both diagnostic capacity and access to effective anti-CRE agents at tertiary referral centres managing patients with severe infections.

The overall 30-day all-cause in-hospital mortality in this cohort was high, especially among CRE-infected patients, where mortality was more than double that observed in colonised patients. Notably, the mortality rate in our cohort was higher than that reported in neighbouring Singapore (35.5%) [30], although such comparisons should be interpreted cautiously due to potential differences in patient case-mix, infection severity, screening practices, and healthcare settings. Several factors may have contributed to these outcomes. Patient-related factors, including a higher comorbidity burden, as reflected by the Charlson comorbidity index, were significantly associated with mortality. This is consistent with a systematic review by Hu et al., which identified comorbidities as a strong predictor of adverse outcomes in CRE infection [31]. Furthermore, microbiological factors including the predominance of CP-CRE may have limited effective treatment options. Additionally, system-level factors, such as delays in CRE identification and the limited availability of newer anti-CRE therapies such as ceftazidime-avibactam and cefiderocol may have played a role. In our institution, targeted agents such as polymyxin were generally reserved for a confirmed CRE infections rather than for empirical treatment, reflecting a need to balance timely treatment against antimicrobial toxicity and stewardship considerations. Interestingly, the longer length of stay observed among survivors (47 vs 22 days) likely reflects survivorship bias, where patients who died early had shorter hospitalisation durations and therefore a reduced opportunity to accrue prolonged hospital stays. Collectively, these observations emphasize the importance of early risk stratification, vigilant clinical monitoring, and timely initiation of appropriate therapy in managing patients at high risk of CRE.

Overall, our findings emphasize that CRE colonisation and infection represent interconnected but distinct clinical states, each with important implications for infection prevention, antimicrobial stewardship, and patient outcomes. By identifying high-risk populations and characterizing local CRE epidemiology, this study provides a foundation for targeted interventions aimed at reducing CRE transmission and improving clinical outcomes in similar healthcare settings.

Limitations

This study has several limitations. Its retrospective design may be subject to incomplete or missing data, and causal relationships cannot be established. As a single-centre study with a

relatively small sample size—particularly among patients with CRE infection—the generalizability of findings may be limited. The small number of patients and mortality events within the CRE infection group also limited the statistical power of the multivariate analysis, which may explain why variables significant in the univariate analysis did not remain significant after adjustment. A high proportion of missing polymyxin susceptibility data limits interpretation of resistance patterns and treatment implications. Additionally, a detailed assessment of the timing, appropriateness, and adequacy of antimicrobial therapy was not feasible, nor could progression from colonisation to infection be evaluated longitudinally. Future prospective multicentre studies are warranted to address these gaps.

Conclusion

Our study provides important insights into the epidemiology, clinical characteristics, and outcomes of CRE in a major referral hospital in Sabah, Malaysia. These findings can aid healthcare providers in identifying and risk-stratifying patients at risk of CRE colonisation or infection, enabling more targeted surveillance and early interventions. Despite a substantial mortality burden, treatment options remain limited in our setting, especially in the context of emerging resistance to commonly used agents such as polymyxins. Notably, the predominance of NDM-producing CRE further restricts therapeutic options and has important implications for antimicrobial policy, laboratory capacity, and access to newer anti-CRE agents. Together, these results underscore the urgent need to strengthen infection prevention and control measures, optimize antimicrobial stewardship practices, and improve access to effective anti-CRE therapies to mitigate transmission and improve patient outcomes.

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Conflict Of Interest

The authors declared no conflict of interest may arise from this study.

Author's Contribution

ABA and GQL conceptualized and designed the study, conducted the literature review and coordinated data collection. ABA drafted the initial manuscript. GQL performed the statistical analysis and contributed to data interpretation. AJI contributed to manuscript drafting and critical revision for important intellectual content. NHZ and NSF MJ assisted with microbiological data collection and laboratory-related data interpretation. GSR provided overall supervision, guided study execution, and critically reviewed the manuscript. All authors read and approved the final manuscript.

Table 1. Patients' baseline clinical characteristics and risk factors for CRE infection and CRE colonisation

Parameters	Total (n=101)	Infection (n=40)	Colonisation (n=61)	p- value*
Demographics				
Age in years, mean (SD)	53.5(15.3)	53.4(14.9)	53.5 (15.7)	0.95[a]
Male sex (%)	73 (72.3)	27 (67.5)	46 (75.4)	0.39[b]
Race				
Native Sabahan (%)	78 (77.2)	31 (77.5)	47 (77.0)	0.96 [b]
Chinese (%)	15 (14.9)	5 (12.5)	10 (16.4)	0.59 [b]
Others (%)	8 (7.9)	4 (10)	4 (6.5)	0.71[c]
Duration from admission to index culture, median days (IQR)	16 (8-29)	15 (7.3-28.5)	16 (8.5-29.5)	0.58[d]
Comorbidities				
Charlson comorbidity index, mean (SD)	2.4 (1.8)	2.55 (1.8)	2.39 (1.8)	0.67[a]
Diabetes Mellitus (%)	45 (44.6)	19 (47.5)	26 (42.6)	0.63[b]
Chronic Kidney Disease (%)	19 (18.8)	8 (20)	11 (18.0)	0.81 [b]
Malignancies (%)	11 (10.9)	6 (15)	5 (8.2)	0.28 [b]
Primary site of infection				
Respiratory (%)	29 (28.7)	9 (22.5)	20 (32.8)	0.26 [b]
Skin & soft tissue (%)	21 (20.8)	10 (25)	11 (18.0)	0.40 [b]
Intra-abdominal (%)	16 (15.8)	6 (15)	10 (16.4)	0.85 [b]
Bacteraemia (%)	10 (9.9)	7 (17.5)	3 (4.9)	0.04[c]
Others (%)	25 (24.8)	8 (20)	17 (27.9)	0.47 [b]
Healthcare exposures				
Presence of device (%)	89 (88.1)	37 (92.5)	52 (85.2)	0.27 [b]
History of ICU admission (≤1 month) (%)	71 (70.3)	32 (80)	40 (65.6)	0.12 [b]
History of surgical intervention (≤1 month) (%)	57 (56.4)	28 (70)	29 (47.5)	0.03 [b]
CRE contact (%)	51 (50.5)	11 (27.5)	40 (65.6)	<0.001 [b]
Previous hospital encounter (≤3 months) (%)	37 (36.6)	17 (42.5)	20 (32.8)	0.32 [b]
Prolonged hospital stays (≥1 month) (%)	29 (28.7)	11 (27.5)	18 (29.5)	0.83 [b]
History of implantation (≤1 year) (%)	12 (11.9)	5 (12.5)	7 (11.5)	0.88 [b]
Antibiotic exposure (≤6 months)				
Piperacillin-tazobactam (%)	53 (52.5)	27 (67.5)	26 (42.6)	0.01 [b]
Carbapenem (%)	49 (48.5)	24 (60)	25 (41.0)	0.06 [b]
Amoxicillin-clavulanic acid (%)	33 (32.7)	11 (27.5)	22 (36.1)	0.37 [b]
Ceftazidime (%)	22 (21.8)	3 (7.5)	19 (31.1)	0.01[c]
Cefuroxime (%)	21 (20.8)	12 (30)	9 (14.8)	0.07 [b]
Ceftriaxone (%)	17 (16.8)	10 (25)	7 (11.5)	0.08 [b]
Ampicillin-Sulbactam (%)	17 (16.8)	7 (17.5)	10 (16.4)	0.88 [b]
Vancomycin (%)	16 (15.8)	9 (22.5)	7 (11.5)	0.14 [b]
Cefepime (%)	14 (13.9)	5 (12.5)	9 (14.8)	0.68 [b]
Clindamycin (%)	10 (9.9)	5 (12.5)	5 (8.2)	0.48 [b]
Polymyxin E (Colistin) (%)	2 (1.9)	1 (2.5)	1 (1.6)	1[c]
Outcomes				
Length of stay, median (IQR)	30 (17.5-49.5)	28.5 (15.8 – 47)	35 (17.5 – 51.5)	0.27[d]
30-day in-hospital mortality (%)	34 (33.7)	21 (52.5)	13 (21.3)	0.001[b]

***** *statistically significant at p<0.05;* [a] *Independent t-test;* [b] *Chi-square;* [c] *Fisher exact test;* [d] *Mann- Whitney U test*

Table 2. Characteristics of CRE isolates

Parameters	Total (n=101)	Infection (n=40)	Colonization (n=61)
Culture Source			
Rectal Swab (%)	47 (46.5)	0	47 (77.1)
Blood (%)	28 (27.7)	22 (55)	6 (9.8)
Tracheal/Sputum (%)	8 (8.0)	3 (7.5)	5 (8.2)
Tissue (%)	7 (6.9)	7(17.5)	0
Others (%)	11 (10.9)	8 (20)	3 (4.9)
Species			
Klebsiella pneumonia (%)	69 (68.3)	27 (67.5)	42 (68.9)
Enterobacter cloacae (%)	13 (12.9)	6 (15)	7 (11.5)
Citrobacter freundii (%)	5 (4.9)	1 (2.5)	4 (6.6)
Escherichia coli (%)	5 (4.9)	0	5 (8.2)
Serratia marcescens (%)	4 (4.0)	3 (7.5)	1 (1.6)
Others (%)	5 (4.9)	3 (7.5)	2 (3.2)
Genotypes			
New Delhi Metallo-beta-lactamase (NDM) (%)	80 (79.2)	30 (75)	50 (82)
Imipenemase (IMP) (%)	4 (4.0)	4 (10)	0
No detection (%)	10 (9.9)	4 (10)	6 (9.8)
Not available (%)	7 (6.9)	2 (5)	5 (8.2)
Susceptibility to Polymyxin			
Intermediate (%)	25 (24.8)	19 (47.5)	6 (9.8)
Resistant (%)	5 (4.9)	4 (10)	1 (1.6)
Not available (%)	71 (70.3)	17 (42.5)	54 (88.5)

Table 3. Univariate and multivariate logistic regression analyses of factors associated with 30-day in-hospital mortality among patients with CRE infection (n = 40)

Variable	Univariate logistic			Multivariate logistic		
	COR	**95% CI**	**P value**	**AOR**	**95% CI**	**P value**
Gender						
Female	1					
Male	1.46	0.36 – 5.51	0.58			
Age	1.05	1.00-1.10	0.027*	1.02	0.94-1.11	0.62
Comorbidities						
Charlson comorbidity index	1.55	1.02-2.34	0.024*	1.41	0.60-3.29	0.43
Diabetes Mellitus						
No	1					
Yes	1.01	0.29-3.50	0.99			
Chronic Kidney Disease						
No	1					
Yes	1.68	0.34-8.18	0.52			

Healthcare exposures						
Presence of device						
No	1					
Yes	0.53	0.04-6.34	0.61			
History of ICU admission (≤1 month)						
No	1					
Yes	1.13	0.24-5.34	0.87			
History of surgical intervention (≤1 month)						
No	1					
Yes	0.71	0.18-2.80	0.63			
CRE contact						
No	1					
Yes	0.677	0.17-2.73	0.58			
Previous hospital encounter (≤3 months)						
No	1					
Yes	1.03	0.29-3.62	0.96			
Prolonged hospital stays (≥1 month)						
No	1					
Yes	0.23	0.05-1.05	0.05	0.17	0.02-1.68	0.13
History of implantation (≤1 year)						
No	1					
Yes	0.56	0.08-3.79	0.55			
Antibiotic exposure (≤6 months)						
Carbapenem						
No	1					
Yes	1.8	0.50-6.46	0.37			
Piperacillin-tazobactam						
No	1					
Yes	2.33	0.60-9.02	0.22	4.42	0.35-55.3	0.25
Cefepime						
No	1					
Yes	0.56	0.08-3.79	0.55			
Ceftazidime						
No	1					
Yes	1.89	0.16-22.75	0.61			
Ceftriaxone						
No	1					
Yes	1.5	0.35-6.42	0.58			
Ampicillin-Sulbactam						
No	1					
Yes	0.29	0.05-1.75	0.16	0.86	0.08-9.61	0.90
Amoxicillin-Clavulanate						
No	1					
Yes	1.12	0.28-4.51	0.87			

AOR–adjusted odd ratio; CI–confidence interval; COR–crude odd ratio
Only variables with p<0.25 in univariate analysis were entered into the multivariate model.
* p-value <0.05 denotes statistical significance.

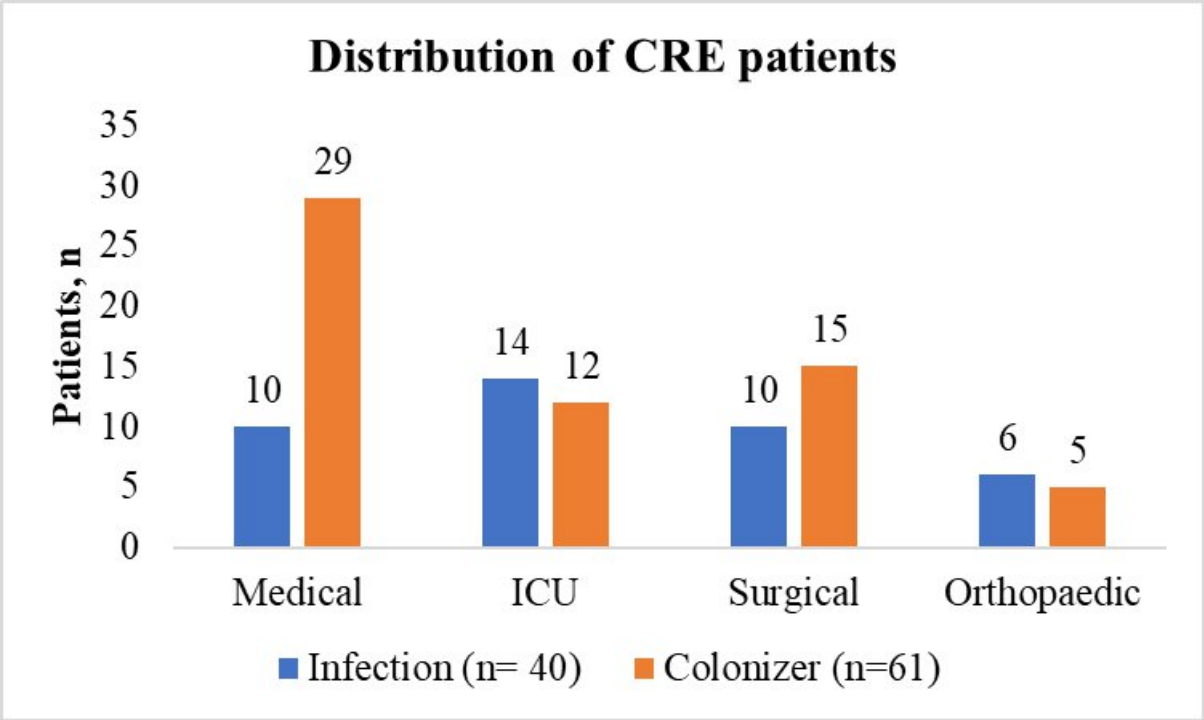

Figure 1. Distribution of CRE patients

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