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Detection of the blaKPC Carbapenemase Gene in Multidrug-Resistant Enterobacterales Isolated from Infected Surgical Wounds at a Tertiary Care Hospital in Syria

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ABSTRACT: Klebsiella pneumoniae carbapenemase (KPC) is a class A serine carbapenemase that hydrolyses almost the entire β-lactam range, including the carbapenems, and its gene is usually carried on conjugative plasmids. KPC dominates the carbapenemase population in parts of East Asia and the Americas but is reported only sporadically in the Eastern Mediterranean, where OXA-48 and NDM prevail, and no data exist for Syria. This study set out to measure the prevalence of blaKPC, and to establish whether it lies on the chromosome or on a plasmid, in multidrug-resistant Enterobacterales recovered from infected surgical wounds. Swabs were taken from the infected wound sites of 50 patients admitted to the General Surgery Department of a tertiary care hospital in Damascus between May and July 2026. Isolates were identified biochemically and by real-time PCR with species-specific assays, susceptibility to 15 antibiotics was determined by disk diffusion according to CLSI M100 (33rd edition), and blaKPC was sought in genomic and plasmid DNA by real-time PCR with SYBR Green. Sixty-seven Gram-negative isolates were recovered, 55 of which (82.09%) were Enterobacterales: Klebsiella pneumoniae 23, Escherichia coli 19 and Enterobacter spp. 13. Every isolate was multidrug-resistant, 13 were extensively drug-resistant and three were pandrug-resistant. blaKPC was detected in eight isolates (14.55%): in chromosomal DNA in six (10.91%) and in plasmid DNA in five (9.09%), three isolates carrying it at both sites. Carriage was concentrated in K. pneumoniae (6 of 23, 26.09%), with one E. coli and one Enterobacter isolate positive, both on a plasmid only. Carriers were resistant to meropenem, gentamicin, ciprofloxacin and cotrimoxazole in 100% of cases and to imipenem in 87.50%, against 72.34% and 46.81% respectively among non-carriers, and half of them met the criteria for extensive or pandrug resistance. In one specimen a K. pneumoniae and an E. coli isolate were both blaKPC-positive with plasmid carriage in each. The detection of blaKPC in 14.55% of Enterobacterales is appreciably higher than the figures reported from neighbouring countries, and its plasmid location, together with its recovery from two species in a single wound, indicates a potential for horizontal transfer that warrants molecular surveillance.

1. INTRODUCTION

Infection of a surgical wound is among the most frequent healthcare-associated infections, and the organisms responsible are predominantly Gram-negative. *Enterobacterales* in particular combine ready access to a surgical site with an unusual capacity to acquire resistance determinants, and the loss of the carbapenems as a therapeutic option in these organisms is one of the most serious developments in clinical microbiology of the past two decades.

Carbapenem resistance in *Enterobacterales* is conferred chiefly by three carbapenemase families: the Ambler class A enzyme KPC, the class B metallo-β-lactamase NDM, and the class D oxacillinase OXA-48. KPC was first described in *Klebsiella pneumoniae* and hydrolyses penicillins, cephalosporins, aztreonam and the carbapenems. The crystal structure of KPC-2 shows an active site remodelled relative to non-carbapenemase class A enzymes: the catalytic serine is displaced outwards and the pocket is shallower, which admits the bulky carbapenem nucleus and explains the breadth of the hydrolytic spectrum [1].

Which carbapenemase predominates is strongly geographical. KPC is the leading enzyme in parts of East Asia and the Americas; in a survey of 935 carbapenem-resistant *Enterobacterales* from 36 Chinese hospitals, $bla_{\text{KPC-2}}$ was present in 51.6% of isolates and was the single most common determinant [2]. In the Eastern Mediterranean the picture is reversed. A two-year series of 131 carbapenem-resistant *Enterobacterales* from a tertiary hospital in Riyadh found $bla_{\text{OXA-48}}$ in 42.7% and bla_{NDM} in 28.2% but bla_{KPC} in only four isolates, about 3% [3], and a three-year rectal colonisation survey in Istanbul found bla_{KPC} in 6.1% of carbapenemase-positive samples against 84.0% for $bla_{\text{OXA-48}}$ [4]. A genomic survey of multidrug-resistant *Enterobacterales* from Jordan likewise identified bla_{NDM} and $bla_{\text{OXA-48}}$ as the carbapenemases of consequence [5].

What makes bla_{KPC} a problem of infection control rather than of individual therapy is its location. The gene is typically carried on conjugative plasmids, often embedded in the transposon *Tn4401*, and plasmids of this kind have been shown to transfer between *Citrobacter freundii*, *Klebsiella oxytoca* and *Escherichia coli* within a single intensive care unit and then between hospitals [6]. Plasmids carrying bla_{KPC} frequently carry additional determinants: one recently described replicon carried $bla_{\text{KPC-2}}$ together with $bla_{\text{CTX-M-65}}$, $bla_{\text{TEM-1}}$ and the 16S rRNA methylase rmtB, so that a single conjugation event transferred resistance to three drug classes at once [7]. The clinical stakes are raised further by the fact that KPC variants arising under treatment confer resistance to ceftazidime-avibactam, one of the few agents retaining activity against KPC producers [8].

No data on bla_{KPC} in Syria have been published, and the organisms infecting surgical wounds in Syrian hospitals have been characterised only by phenotype. In a previous study of the same patient cohort we examined the AcrAB efflux pump, an intrinsically encoded mechanism of multidrug resistance, and recommended that the acquired β-lactamase and carbapenemase determinants be investigated next [9]. The present work addresses bla_{KPC}.

2. AIM OF THE STUDY

The study aims to determine the prevalence of the bla_{KPC} carbapenemase gene in multidrug-resistant *Enterobacterales* isolated from infected surgical wounds in a general surgery department of a tertiary care hospital in Syria, to establish whether the gene is carried on the chromosome or on a plasmid in each species, and to relate its presence to the antibiotic resistance phenotype of the isolates.

3. MATERIALS AND METHODS

Study design: A cross-sectional study.

Samples: Fifty patients were enrolled, all of them admitted to the General Surgery Department of a tertiary care hospital in Damascus and all of them having undergone a surgical procedure of one kind or another during the enrolment window, which ran from the start of May 2026 to the end of July 2026. The group comprised 37 men (74%) and 13 women (26%), and the sampling site in every case was the infected surgical wound.

Patients were sampled once they showed signs or symptoms of wound infection, a sterile cotton swab being passed over the affected site. Each swab was transferred without delay into a sterile liquid preservation medium composed of xanthan gum, L-cysteine, sodium thioglycolate and peptone [10], in which it was held until it reached the laboratory for culture and subsequent testing.

The isolates examined here were recovered from the same prospective collection described in our earlier report on the AcrAB efflux pump in this patient group [9], so the isolation and susceptibility data are shared between the two reports and are presented here as context for the genetic findings, which are new. Gram-negative bacilli that were not *Enterobacterales*, namely *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, were recorded during isolation but excluded from the analyses reported below, because the literature on bla_{KPC} is concentrated in the *Enterobacterales* and comparison is only meaningful within that group.

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1. ISOLATION AND IDENTIFICATION OF THE BACTERIA

Three lines of evidence were used to establish that a specimen contained Gram-negative bacilli: a stained smear prepared by the Gram method, growth on media selective for this group, and the biochemical profile of the resulting colonies.

Culture was carried out on EMB agar and on cetrimide agar (Himedia Laboratories, India), both prepared as the manufacturer directs. Every specimen was streaked on each of the two media and the plates were held at 37 °C for 24 hours, a period sufficient for discrete colonies to develop.

Colonies were tested for oxidase activity, a positive reaction pointing to *Pseudomonas aeruginosa*. Species assignment then rested on a panel of biochemical reactions — indole, methyl red, Voges-Proskauer and citrate — read alongside the pattern of growth on Kligler iron agar.

Identification was confirmed at the molecular level. Total bacterial DNA was recovered with the GeneAll® Exgene™ Cell SV mini kit (South Korea) following the supplier's protocol, after which species were assigned by real-time polymerase chain reaction using species-specific kits (Genetic PCR Solutions™, Genetic Analysis Strategies, Spain). Reactions were assembled in nuclease-free tubes to a 20 µl final volume, the composition of which is given in Table 1; the cycling conditions supplied with these kits are reproduced in Table 2.

Table 1. Composition of the reaction mixture for isolate typing (20 µl final volume)

Reaction component	Volume (µl)
Nuclease-free water	9
Reaction mix	5
Primer and probe mix	1
Extracted DNA	5

Table 2. Thermal amplification programme for isolate typing

Cycles	Step	Temperature	Time
1	Initial denaturation	95 °C	2 min
40	Denaturation	95 °C	5 s
40	Annealing and extension	60 °C	20 s

2. ANTIBIOTIC SUSCEPTIBILITY TESTING

Each isolate was tested against antibiotics drawn from several classes by disk diffusion on Mueller Hinton agar, the procedure and the interpretive criteria following document M100 of the Clinical and Laboratory Standards Institute, 33rd edition [11]. Commercial disks (Biomaxima, Poland) carrying defined contents were used, as follows: amikacin (30 µg), aztreonam (30 µg), ampicillin/sulbactam (20 µg), cefepime (30 µg), cefoperazone (75 µg), ceftazidime (30 µg), chloramphenicol (30 µg), ciprofloxacin (30 µg), colistin (10 µg), cotrimoxazole (25 µg), doxycycline (30 µg), gentamicin (10 µg), imipenem (10 µg), meropenem (10 µg) and tigecycline (15 µg).

Colonies of each strain were suspended until the turbidity matched a 0.5 McFarland standard. A 50 µl volume of this suspension was spread so as to cover the whole surface of the Mueller Hinton agar, the disks were laid on the seeded plate, and incubation followed at 37 °C for 24 hours. Interpretation rested on the diameter of the zone of inhibition surrounding each disk, measured once incubation was complete and referred to the standard tables.

Isolates were classified as multidrug-resistant (MDR) when they were non-susceptible to at least one agent in three or more antimicrobial classes, extensively drug-resistant (XDR) when they remained susceptible to agents in no more than two classes, and pandrug-resistant (PDR) when they were non-susceptible to all agents tested.

3. Detection of the *bla*$_{KPC}$ gene

Every isolate shown to be multidrug-resistant on the testing described above was examined for *bla*$_{KPC}$, the search covering chromosomal and plasmid DNA separately. The two fractions were recovered with different kits from the same supplier, the GeneAll® Exgene™ Cell SV mini kit for genomic DNA and the GeneAll® Exprep™ Plasmid SV mini kit for plasmid DNA (South Korea), each used exactly as its protocol directs.

Detection of *bla*$_{KPC}$ in the genomic and plasmid DNA was carried out by real-time polymerase chain reaction using the specific primers shown in Table 3 [12], supplied by GenScript (Singapore). GeneAll® RealAmp™ 2X SYBR qPCR Master Mix was used to prepare the reaction mixture to a final volume of 20 µl as shown in Table 4, and thermal amplification was carried out as shown in Table 5.

Table 3. Primers used for the detection of the *bla*$_{KPC}$ gene

Gene	Forward primer (5'→3')	Reverse primer (5'→3')
bla$_{KPC}$	CGTCTAGTTCTGCTGTCTTG	CTTGTCATCCTTGTTAGGCG

Table 4. Composition of the reaction mixture for gene detection (20 µl final volume)

Component	Concentration	Volume
2X SYBR qPCR Master Mix	2X	10 µl
Template DNA	(10 pg–10 ng)/µl	—
Forward primer	100 µM	0.1 µl
Reverse primer	100 µM	0.1 µl
Nuclease-free water	—	to 20 µl

Table 5. Thermal amplification programme for detection of the *bla*$_{KPC}$ gene

Step	Temperature	Time	Cycles
Enzyme activation	95 °C	15 min	1
Denaturation	95 °C	20 s	40
Annealing	59 °C	40 s	40
Extension	72 °C	30 s	40

4. RESULTS

1. ISOLATION AND IDENTIFICATION OF THE BACTERIA

Sixty-seven Gram-negative isolates grew from the specimens of the 50 patients, and real-time PCR assigned each of them to species. The *Enterobacterales* accounted for 55 of the 67 (82.09%) and are the subject of everything that follows; the other 12 (17.91%) were non-fermenting Gram-negative bacilli — nine *Pseudomonas aeruginosa* and three *Acinetobacter baumannii* — and took no further part in the analysis.

Two species of Gram-negative bacilli were recovered from 17 of the specimens (34%), the remaining 33 (66%) yielding a single species each. *Klebsiella pneumoniae* was the commonest of the *Enterobacterales* at 23 isolates (41.82%), ahead of *Escherichia coli* at 19 (34.55%) and *Enterobacter* spp. at 13 (23.64%).

2. ANTIBIOTIC SUSCEPTIBILITY

Disk diffusion testing showed that every *Enterobacterales* strain isolated from the specimens was resistant to at least one agent in three or more antimicrobial classes; all 55 isolates were therefore multidrug-resistant. Thirteen of them (23.64%) met the criteria for extensive drug resistance and three (5.45%) were resistant to every agent tested and were classified as pandrug-resistant.

Resistance was highest among the β-lactams. Every isolate was resistant to aztreonam (100%), and resistance reached 96.36% for ceftazidime, 87.27% for both cefepime and cefoperazone and 83.64% for ampicillin/sulbactam. Among the carbapenems, 76.36% of isolates were resistant to meropenem and 52.73% to imipenem. Outside the β-lactams, 83.64% were resistant to ciprofloxacin and to cotrimoxazole, 67.27% to chloramphenicol, 61.82% to amikacin and 58.18% to gentamicin. The two agents that retained the greatest activity were tigecycline, to which 23.64% of isolates were resistant, and colistin, to which 34.55% were resistant. Table 6 presents the full susceptibility and resistance profile of each species against the 15 antibiotics tested.

Table 6. Antimicrobial susceptibility phenotype of the *Enterobacterales* isolates

Antibiotic	Klebsiella (n = 23)			*E. coli* (n = 19)			*Enterobacter* (n = 13)		
	S%	I%	R%	S%	I%	R%	S%	I%	R%
Amikacin	13.00	8.70	78.30	31.60	26.30	42.10	15.40	23.10	61.50
Ampicillin/sulbactam	4.30	4.30	91.30	26.30	5.30	68.40	0.00	7.70	92.30
Aztreonam	0.00	0.00	100.00	0.00	0.00	100.00	0.00	0.00	100.00
Cefepime	8.70	0.00	91.30	0.00	5.30	94.70	0.00	30.80	69.20
Cefoperazone	4.30	4.30	91.30	5.30	0.00	94.70	15.40	15.40	69.20
Ceftazidime	4.30	0.00	95.70	5.30	0.00	94.70	0.00	0.00	100.00
Chloramphenicol	8.70	17.40	73.90	21.10	10.50	68.40	15.40	30.80	53.80
Ciprofloxacin	4.30	4.30	91.30	15.80	5.30	78.90	15.40	7.70	76.90
Colistin	65.20	0.00	34.80	68.40	0.00	31.60	61.50	0.00	38.50
Cotrimoxazole	4.30	0.00	95.70	26.30	5.30	68.40	15.40	0.00	84.60
Doxycycline	21.70	21.70	56.50	52.60	0.00	47.40	30.80	30.80	38.50
Gentamicin	26.10	8.70	65.20	36.80	15.80	47.40	30.80	7.70	61.50
Imipenem	21.70	4.30	73.90	68.40	0.00	31.60	38.50	15.40	46.20

Antibiotic	Klebsiella (n = 23)			*E. coli* (n = 19)			*Enterobacter* (n = 13)		
	S%	I%	R%	S%	I%	R%	S%	I%	R%
Meropenem	8.70	8.70	82.60	10.50	15.80	73.70	30.80	0.00	69.20
Tigecycline	34.80	21.70	43.50	68.40	21.10	10.50	76.90	15.40	7.70

S: susceptible; I: intermediate; R: resistant. Values are the percentage of isolates of each species. Disk contents are given in the Materials and Methods.

3. Detection of the *bla*$_{KPC}$ gene

Thermal amplification by real-time PCR with SYBR Green fluorescent dye demonstrated the *bla*$_{KPC}$ gene in eight of the 55 *Enterobacterales* isolates (14.55%). The gene was present in the chromosomal DNA of six isolates (10.91%) and in the plasmid DNA of five (9.09%); three isolates carried it at both sites, three carried it on the chromosome only and two on a plasmid only.

Carriage was unevenly distributed between species. In *Klebsiella pneumoniae* the gene was detected in six of 23 isolates (26.09%), present in the chromosomal DNA of all six and additionally in the plasmid DNA of three. In *Escherichia coli* it was detected in a single isolate of 19 (5.26%) and in *Enterobacter* spp. in a single isolate of 13 (7.69%); in both of these the gene was found on a plasmid only and in no chromosome. The distribution is set out in Table 7 and shown in Figure 1.

Table 7. Prevalence of the *bla*$_{KPC}$ gene in the chromosomal and plasmid DNA of the *Enterobacterales* isolates

Species	Chromosomal DNA	Plasmid DNA	Either site
Klebsiella pneumoniae (n = 23)	6 (26.09%)	3 (13.04%)	6 (26.09%)
Escherichia coli (n = 19)	0 (0.00%)	1 (5.26%)	1 (5.26%)
Enterobacter spp. (n = 13)	0 (0.00%)	1 (7.69%)	1 (7.69%)
Total (n = 55)	6 (10.91%)	5 (9.09%)	8 (14.55%)

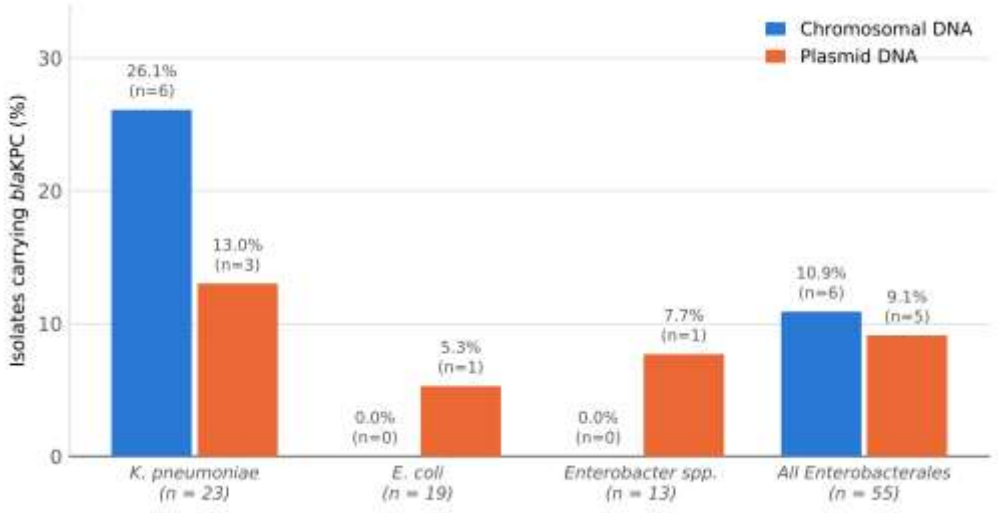


Figure 1. Carriage of the *bla*$_{KPC}$ gene in the chromosomal and plasmid DNA of each *Enterobacterales* species

The presence of the gene was reflected in the resistance phenotype. All eight carriers were resistant to ampicillin/sulbactam, aztreonam, cefoperazone, ciprofloxacin, cotrimoxazole, gentamicin and meropenem, and seven of eight (87.50%) to amikacin, cefepime, ceftazidime and imipenem. The contrast with the 47 isolates in which the gene was not detected was marked for several agents: imipenem resistance was 87.50% among carriers against 46.81% among non-carriers, meropenem 100% against

72.34%, gentamicin 100% against 51.06% and tigecycline 62.50% against 17.02%. Colistin was the only agent to which carriers were less often resistant than non-carriers, at 25.00% against 36.17%. Table 8 gives the full comparison.

The severity of the resistance phenotype followed the same pattern. Of the eight carriers, four were classified as multidrug-resistant, three as extensively drug-resistant and one as pandrug-resistant, so that half of them fell into the two most severe categories, against 16 of 55 isolates (29.09%) in the collection as a whole.

In one specimen, taken from a single patient, both a *Klebsiella pneumoniae* and an *Escherichia coli* isolate were recovered and both were *bla*$_{KPC}$-positive, the gene being present in the plasmid DNA of each and additionally in the chromosome of the *K. pneumoniae* isolate.

Figure 2 shows the amplification curves indicating the presence of the *bla*$_{KPC}$ gene in the isolates studied.

Table 8. Resistance to each antibiotic among the *Enterobacterales* isolates carrying the *bla*$_{KPC}$ gene and among those in which it was not detected

Antibiotic	Carriers (n = 8)	Non-carriers (n = 47)
Amikacin	7 (87.50%)	57.45%
Ampicillin/sulbactam	8 (100.00%)	80.85%
Aztreonam	8 (100.00%)	100.00%
Cefepime	7 (87.50%)	87.23%
Cefoperazone	8 (100.00%)	85.11%
Ceftazidime	7 (87.50%)	97.87%
Chloramphenicol	5 (62.50%)	68.09%
Ciprofloxacin	8 (100.00%)	80.85%
Colistin	2 (25.00%)	36.17%
Cotrimoxazole	8 (100.00%)	80.85%
Doxycycline	5 (62.50%)	46.81%
Gentamicin	8 (100.00%)	51.06%
Imipenem	7 (87.50%)	46.81%
Meropenem	8 (100.00%)	72.34%
Tigecycline	5 (62.50%)	17.02%

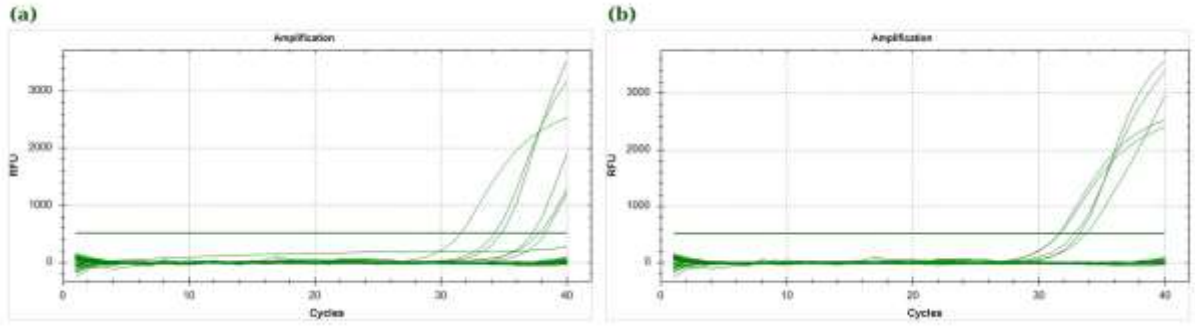


Figure 2. Amplification curves obtained by real-time PCR with SYBR Green showing the presence of the *bla*$_{KPC}$ gene in the isolates studied: (a) in the chromosomal DNA, (b) in the plasmid DNA. The horizontal line marks the fluorescence threshold.

5. DISCUSSION

bla$_{KPC}$ was detected in 14.55% of the multidrug-resistant *Enterobacterales* recovered from these infected surgical wounds. This figure is appreciably higher than those reported from neighbouring countries. In the two-year Riyadh series of 131 carbapenem-resistant *Enterobacterales*, *bla*$_{KPC}$ was found in only four isolates, approximately 3% [3], and in the Istanbul colonisation survey it accounted for 6.1% of carbapenemase-positive samples against 84.0% for *bla*$_{OXA-48}$ [4]. In the Jordanian genomic survey, *bla*$_{NDM}$ and *bla*$_{OXA-48}$ were the carbapenemases identified and *bla*$_{KPC}$ was not among the determinants reported [5]. Against the Chinese picture, where *bla*$_{KPC-2}$ reaches 51.6% of carbapenem-resistant *Enterobacterales* and is the dominant enzyme [2], the present figure

is of course low; but the comparison that matters for this setting is the regional one, and against that the detection rate is unexpected.

Two readings are possible and the data do not separate them. The first is that KPC-producing lineages have become established locally, which would be consistent with the sustained disruption of infection control and antibiotic stewardship in Syrian hospitals. The second is a difference in case mix: the comparison series were drawn either from isolates already shown to be carbapenem-resistant or from colonisation screening, whereas every multidrug-resistant *Enterobacterales* isolate was tested here regardless of its carbapenem phenotype, which widens the denominator in a way that is not directly comparable. Distinguishing between these readings would require sequencing to confirm the variant and strain typing to establish clonality, neither of which was within the scope of this study.

Carriage was concentrated in *Klebsiella pneumoniae*, in which the gene was found in 26.09% of isolates, against 5.26% of *Escherichia coli* and 7.69% of *Enterobacter* spp. This matches the organism in which the enzyme was first described and in which it remains most frequently reported, and it is consistent with the recognised role of *K. pneumoniae* as the principal accumulator of resistance determinants among the *Enterobacterales*. The pattern of location differed between species in a way that is worth noting: in *K. pneumoniae* the gene was present on the chromosome in every positive isolate and additionally on a plasmid in half of them, whereas in the single *E. coli* and the single *Enterobacter* isolate it was found on a plasmid only. A plasmid-borne determinant in a species in which it is otherwise absent is the expected signature of recent acquisition rather than of clonal descent.

The most consequential observation is that in one wound two different species, a *K. pneumoniae* and an *E. coli*, were both *bla*$_{KPC}$-positive with the gene on a plasmid in each. Horizontal transfer of a *bla*$_{KPC}$-carrying plasmid between genera has been documented directly in the clinical setting: an IncN plasmid carrying *bla*$_{KPC-2}$ within transposon *Tn4401g* spread between *Citrobacter freundii*, *Klebsiella oxytoca* and *Escherichia coli* in seven critically ill patients and subsequently between hospitals [6]. The present finding does not prove transfer, which would require plasmid typing and conjugation experiments, but it is exactly the configuration in which transfer has been shown to occur, and a surgical ward in which a third of infected wounds yield two organisms supplies both the donors and the recipients.

The association between carriage and phenotype was consistent. All carriers were resistant to meropenem and seven of eight to imipenem, against 72.34% and 46.81% among non-carriers, and half of the carriers met the criteria for extensive or pandrug resistance against 29.09% of the collection overall. The gradient extended beyond the β-lactams, to gentamicin (100% against 51.06%) and tigecycline (62.50% against 17.02%), which the gene itself does not explain and which points to the co-location of *bla*$_{KPC}$ with determinants for other classes on the same mobile element. That arrangement has been documented directly on a plasmid carrying *bla*$_{KPC-2}$ together with *bla*$_{CTX-M-65}$, *bla*$_{TEM-1}$ and the aminoglycoside methylase rmtB within overlapping transposon structures [7].

The therapeutic implication is immediate. KPC is inhibited by avibactam, and ceftazidime-avibactam is among the few agents that retain activity against KPC producers; but resistance to it emerges under treatment through variants arising in the Ω loop of the enzyme, and such variants have been recovered in 4% of patients treated with the combination [8]. Where *bla*$_{KPC}$ is present alongside a metallo-β-lactamase, which avibactam does not inhibit, the combination offers no benefit at all. Colistin and tigecycline were the agents to which carriers were least often resistant in this series, but at 25.00% and 62.50% respectively neither can be relied upon empirically without susceptibility testing.

Limitations. The study was conducted in a single department of a single hospital over three months, and eight positive isolates is a small denominator, so the confidence interval around the 14.55% figure is wide. Detection was by real-time PCR with SYBR Green and specific primers, which establishes the presence of the target sequence but does not identify the KPC variant, confirm that the gene is expressed, or characterise the plasmid carrying it; sequencing, replicon typing and conjugation experiments would be needed for that. Phenotypic confirmation of carbapenemase production, for instance by an immunochromatographic assay of the kind validated for KPC and the other principal carbapenemases [13], was not performed, so genotype could not be reconciled with phenotype isolate by isolate. The isolation and susceptibility results are shared with our earlier report on the same cohort [9] and should not be counted twice in any pooled analysis.

6. CONCLUSION

bla$_{KPC}$ was present in 14.55% of multidrug-resistant *Enterobacterales* isolated from infected surgical wounds in this general surgery department, a prevalence higher than that reported from neighbouring countries, where OXA-48 and NDM are the carbapenemases of consequence and KPC is uncommon. Carriage was concentrated in *Klebsiella pneumoniae* and was accompanied by near-total carbapenem resistance and by extensive or pandrug resistance in half of the carriers.

The gene was found on plasmids in five of the eight carriers, and in the two species other than *K. pneumoniae* it was found on a plasmid only. In one wound, two species were positive simultaneously with plasmid carriage in each. These observations indicate a determinant that is mobile rather than confined to a single lineage, and one that routine susceptibility testing will not detect. Sequencing to confirm the variant, plasmid characterisation and strain typing are the necessary next steps, and molecular surveillance of carbapenemase genes should accompany routine testing in surgical units in this setting.

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AUTHOR CONTRIBUTIONS

Razan AL Debs: conceptualization, methodology, investigation, formal analysis, writing of the original draft, review and editing. Nader Ghanayem: methodology, validation, molecular analysis, data curation.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ETHICS STATEMENT

The study was approved by the Research Ethics Committee of the Faculty of Pharmacy, Damascus University in 2025 (approval number SRC-1810). Informed consent was obtained from all participants, and the specimens were handled anonymously.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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