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ORIGINAL ARTICLE

Phenotypic and genotypic analysis of hypermucoviscous *Klebsiella pneumoniae* (hmkp) strain

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ABSTRACT

Background and Objectives: Hypermucoviscous *Klebsiella pneumoniae* exhibits distinct phenotypic and genetic characteristics that distinguish it from the classic *K. pneumoniae* pathogen. The aim of current study was to investigate some phenotypic and genetic markers used for hmKp identification.
Materials and Methods: Seventy-one *K. pneumoniae* isolates were obtained from the respiratory care unit in Al-Diwanyiah Teaching Hospital \Diwanyah, Iraq, from the first of November 2024 to the first of March 2025. The bacteria were identified, and antibiotic sensitivity testing was performed using VITEK 2 ID-GN and AST cards. Hypermucoviscosity was assessed using the string test, and an investigation into several adherence and virulence genes was conducted for all isolates. Then, multi-locus sequence typing was performed for hypermucoviscous *K. pneumoniae* isolates.
Results: 3 (4.22%) of 71 isolates were hypermucoviscous. The virulence and adherence genes were present in 100% of the isolates, whereas *rmpA* was only found in hypermucoviscous isolates. The results showed that the hmKp isolates were members of clonal group 147 (CG147) and were assigned to sequence type (ST) 293.
Conclusion: The string test is the primary phenotypical diagnosis for hmKp, while the genetically encoded *rmpA* gene is the most reliable genetic marker for hmKp identification. However, MLST is not beneficial for identification. The central positioning of ST392 within the MST highlights its potential role as an emerging high-risk clone.

Keywords: *Klebsiella pneumonia*; Hypermucoviscosity; Bacterial capsules; *rmpA* gene; Virulence

INTRODUCTION

Klebsiella pneumoniae is the most commonly isolated microorganism from both community and hospital-acquired infectious diseases (1). It's a member of the Enterobacteriaceae that causes bloodstream infection, pneumonia, and urinary tract infection (2). More recently, however, a new hypermucoviscous *K. pneumoniae* (hmKp) strain has been primarily found in Southeast Asia (3). hmKp strains are characterized by a gelatinous morphology on solid media, and this can be identified as a positive 'string test' semi-quantitatively (4). Such strains might cause numerous serious infections, including liver abscess, endophthalmitis, meningitis, and metastasize to infect multiple organs (5). Infection with hmKp may result in lethal community-acquired infections in immunocompetent individuals (6). However, recent research has revealed renal insufficiency and hospital-acquired infection as predisposing factors for the infection (7). Furthermore, nosocomial transmission and healthcare-associated infections (HAI) outbreaks have increased recently (8). It has been well-established for a long time that hypermucoviscosity is strongly

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correlated with hypervirulence in hmKp strains (9).

Hypermucoviscous (hmKp) and hypervirulent have thus typically been synonymous (10). However, recent studies have highlighted that there are many fundamental differences between hypermucoviscosity and hypervirulence (11). *K. pneumoniae* hypervirulence is a product of multiple factors. Hypervirulence cannot be conferred by a single phenotypic attribute or genetic variable (12). It is insufficient to declare one strain hypervirulent solely based on hypermucoviscosity ascertained by the string test (13). Additionally, earlier studies have shown that several virulence variables, besides the hypermucoviscous phenotype (14), such as the type III fimbrial adhesin gene (*mrkD*), the regulator mucoid phenotype A (*rmpA*), and the carbapenem gene (bla_{KPC}), play a crucial role in antibiotic resistance, especially in association with the *mrkD* gene (15). The primary objective of the current study was to identify several phenotypic and genetic markers used for hmKp identification.

MATERIALS AND METHODS

Samples. Seventy-one *K. pneumoniae* isolates were obtained from patients admitted to the Respiratory Care Unit (RCU) at Al-Diwanyah Teaching Hospital. Isolates were identified using VITEK 2 ID-GN cards (BioMérieux, France). Susceptibility to antibiotics was determined using VITEK 2 AST-GN cards, performed according to the standard CLSI methodology (Institute, 2024).

The string test was used to identify hmKp phenotypically; positive strains are defined as those with a sticky string formed by bacterial colonies after being stretched on an agar plate (16).

Testing the ability to form biofilm. A microtiter plate method was used to assess bacterial biofilm formation. First, a Luria broth culture of bacteria was added to each well, along with 200 µL of trypticase soy broth (TSB), then the mixture was incubated for 18 hours at 37°C. The wells were then filled with 10 µL of a 24-hour microbial suspension adjusted to 0.5 McFarland turbidity standard. After that, the plates were incubated for 24 hours at 37°C. Subsequently, every well was drained and given three saline washes. Each well was then filled with 200 µL of a 10% crystal violet solution, which was then allowed to sit at room temperature for 20 minutes. After that, the wells were cleaned and dried three times using normal saline. Following the addition of 200 µL of dimethyl sulfoxide (DMSO) to each well, the plate's optical density (OD) was measured at 630 nm using an ELISA reader. To quantify biofilm formation, the test OD was compared to a control OD: strong biofilm: OD >0.65, moderate biofilm formation: 0.3<OD≤0.5, weak biofilm: OD≤0.3, while OD≤0.15 indicated no biofilm formation (17).

Identification of metallo-β-lactamase-producing isolates phenotypically. Imipenem alone and imipenem mixed with boronic acid were used to detect class A beta-lactamase (metallo-β-lactamase-producing) isolates such as KPC-positive bacteria, using the combination disk test (CDT). Boronic acid (10 µL) was applied to an imipenem disk. The diffusion method was used, with bacteria streaked on Mueller-Hinton agar. The plates were incubated for 24 hours at 37°C. A carbapenemase-positive isolate was identified by an inhibitory zone of at least 5 mm around the imipenem-β-lactamase inhibitor disc, which was also larger than the zone around the imipenem disc alone (18).

Molecular methods. The PCR detection kits used in this study included the Presto™ Mini gDNA Bacteria Kit, manufactured by Geneaid (Taiwan), and the GoTaq® Green PCR Master Mix, manufactured by Promega (USA).

Virulence and adhesion genes detection. PCR was used to detect carbapenem resistance genes. A particular primer, PCR products, and PCR conditions for detecting carbapenem-resistant genes (*rmpA, mrkD*, bla_{KPC}, and bla_{VIM}) are provided in Table 1. The thermocycler conditions were (95°C, 30 sec, 58°C, 30 sec, 72°C, 60 sec for 30 cycles). The products were analyzed on a 1% agarose gel stained with 1X Tris-Acetate-EDTA buffer. Safe Stain was used to stain the gels, and UV light was used to view the PCR products.

Multi-locus sequence typing of hypermucoviscus isolates. The characterization of *K. pneumoniae* clinical isolates was performed, and their clonal group (CG) was identified using the Multi-locus Sequence Typing (MLST) technique. The Protocol used followed the original conditions described by

(Diancourt et al., 2005) (19). MLST is considered a suitable technique for describing the genetic relationships between bacterial isolates because it offers precise and portable data that makes multiuser multinational databases possible. The seven housekeeping genes used in this technique are described in Table 2.

PCR amplification conditions. The initial denaturation was at 94°C for 2 minutes. Denaturation during the cycles was at 94°C for 20 seconds. The annealing temperature was 50°C for all genes, except for *gapA*, which was 60°C, and *tonB*, which was 45°C. The extension temperature was 72°C for 30 seconds. The final extension was at 72°C for 5 minutes. PCR involved 35 cycles of amplification. For *tonB*, $MgCl_2$ must be used at a concentration of 50 mM, while for all other genes, 25 mM 1-$MgCl_2$ is required

RESULTS

This descriptive study analyzed respiratory samples from patients infected with *Klebsiella pneumoniae* who were admitted to the hospital's respiratory care unit. Bacterial isolates were identified, and their antibiotic susceptibility was assessed using the VITEK 2 system with ID-GN and AST cards. Based on the string test, 3 out of 71 isolates (4.22%) exhibited a hypermucoid phenotype.

The antimicrobial sensitivity results showed that all 71 isolates (100%) of *K. pneumoniae* were multidrug-resistant (MDR). As described in Fig. 1, resistance rates varied significantly across different antibiotic categories. A Chi-square test was conducted to evaluate the response of bacteria to antibiotics, with results categorized by the type of antimicrobial agent (sensitive, intermediate, or resistant).

A statistically significant correlation was found by the test ($\chi^2 = 354.6$, df = 48, $P < 0.001$), suggesting that resistance patterns varied depending on the antibiotic class. The prevalence of hypermucoviscous isolates was 4.22% (3 out of 71; 95% CI: 0.9%-11.7%). The majority of isolates (55/71, 77%) demonstrated the ability to form a strong biofilm, with an average optical density (OD) of 6.3 ± 0.42, while 13 isolates (18.3%) exhibited weak biofilm formation (OD = 0.3 ± 0.11). The three hmKp isolates demonstrated moderate biofilm-forming ability with an average OD = 0.49 ± 0.06.t.

MICs for the three hmKp isolates are described in Table 3.

The results of biofilm formation showed that 55 (77%) of 71 isolates formed a strong biofilm (OD = 6.3), and 13 (18.30%) of 71 isolates formed a weak biofilm (OD = 0.3). The three hmKp isolates had an OD of 0.49, defined as moderate biofilm formers (Fig. 2).

To complete the phenotypical identification of metallo-β-lactamase-producing isolates (CDT), as mentioned above, the results indicated that the combination of boronic acid and imipenem increased

Table 1. The PCR oligonucleotides used used for molecular detection

Oligo.	Primers Sequence (5'-3')	NCBI Code reference
rmpA	F: ACTGGGCTACCTCTGCTCA	KJ469368.1
	R: TCAGTAGGCATTGCAGCCT	
mrkD	F: GGAACCCACATCGACATTCA	MZ380316.1
	R: CAACGGTGATGCTGGACATG	
bla_{KPC}	F: CTGTAAGTTACCGCGCTGAG	NC_014312.1
	R: CAGGTTCCGGTTTTGTCTCC	
bla_{VIM}	F: AAGTCCGTTAGCCCATTCCG	CP034084.1
	R: ACTCATGAACGGCACAACCA	

Table 2. Housekeeping genes used in MLST of *K. pneumoniae*

Gene	Forward Primer	Reverse Primer
rpoB	GGCGAAATGGCGAGAACA	GAGTCTTCGAAGTTGTAACC
gapA	TGAAATATGACTCCACTCACGG	CTTCAGAAGCGGCTTTGATGGCTT
Mdh	CCCAATCTCGCTTCAGGTCAG	CCGTTTTTCCCCACAGCAG
Pgi	GAGAAAAACCTGCCTGTCTGCTGGC	CGCGCCACGCTTTATAGCGTTAAT
phoE	ACCTACCGCAACACCGACTTCTTCGG	TGATCAGAACTGGTAGTGAT
infB	CTCGCTGCTGGCTATATTCG	CGCTTTCAGCTCAAGACTTC
tonB	CTTTATACCTCGGTAATCAGGTT	ATTCGCCGGCTGRGCRGGAG

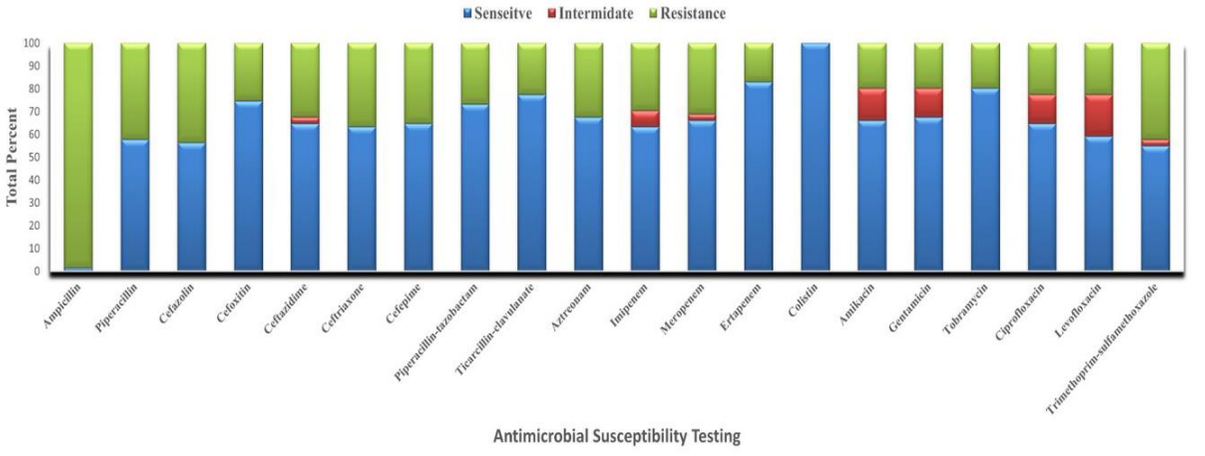

Fig. 1. Antibiotic resistance pattern in *Klebsiella pneumoniae* isolates

Table 3. Minimal inhibitory concentrations (MIC a; mg/L) of three hmKp isolates

Antibiotic	*K. pneumoniae* clinical isolates		
	1	2	3
Aminoglycosides			
Amikacen	S < 2.0	S < 1.0	S < 2.0
Gentamicin	S < 1.0	S < 1.0	S < 1.0
Tobramycin	S < 2.0	S < 1.0	S < 1.0
β-lactamases			
Ticarcillin/Clavulanic Acid	S 16.0	R 32.0	R 32.0
Cefepim	R > 64.0	R 8.0	R 64.0
Cefotaxime	R 8.0	R 64.0	R 64.0
Ceftazidim	I 8.0	R > 64.0	R 64.0
Cefazolin	R 64.0	R 64.0	R 8.0
Ertapenim	S < 0.5	R 8.0	R 8.0
Imipenem	R 8.0	R 16.0	R 8.0
Meropenem	R 8.0	R 8.0	R 16.0
Piperacilln/ Tazobactam	S < 4.0	R >128.0	R >32.0
Fluoroquinolons			
Ciprofloxacin	R > 4.0	R > 4.0	R > 4.0
Levofloxacin	R > 4.0	R > 4.0	S < 0.25
Lipopeptides			
Colistin	S 0.5	S 1.0	S 0.5
Folate Pathway Antagonists			
Trimthoprim/ Sulfamethoxazol	R >320.0	R >320.0	R >320.0

a: Level of resistance according to (Institute, 2024) guidelines: R, resistant; I, intermediate; S, sensitive

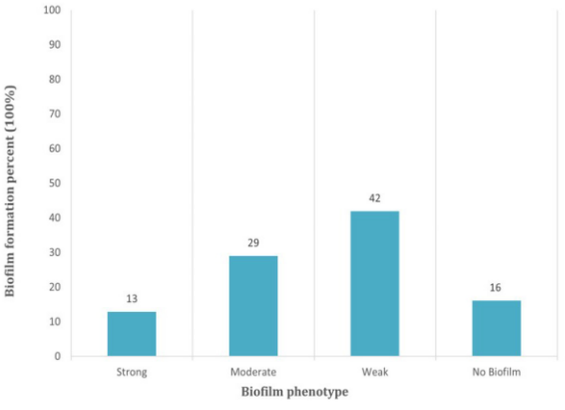

Fig. 2. The results of the ability to form biofilm among different *K. pneumoniae* isolates

the inhibition zone only in hmKp isolates, while this combination did not affect non-hypermucoviscous *K. pneumoniae* isolates.

The study also included an investigation of virulence and adhesion genes. The results showed that all investigated genes, except the *rmpA* gene, were present at 100%. In contrast, the *rmpA* gene was isolated from the hmKp isolates only.

A genetically based MLST test was conducted for the genotypic analysis of 3 isolates in the field of study, and the results showed that all the isolates belonged to Clonal Group 147 (CG147) and Sequence Type 392 (ST392). (https://bigsdb.pasteur.fr/). The MST diagrams generated from MLST data, categorized by country, source, year, and ST groupings, revealed that ST392, isolated from Iraq in 2025 from a sputum sample, is first recorded in Iraq, and it occupies a central position within a diverse clonal network.

ST392 showed close phylogenetic proximity to high risk clones such as ST15, ST147, and ST307, which are widely recognized for their association with multidrug resistance (MDR) and virulence (Fig. 3).

DISCUSSION

K. pneumoniae is characterized by having many virulence factors, including hypermucoviscosity, one of which is related to capsular formation. Several studies have attributed capsular K1\K2 serotypes and the genes associated with capsule formation. In clinical settings, the development of carbapenem resistance (CR) and hypervirulence (hv) as separate evolutionary pathways for *K. pneumoniae* poses a serious risk. Nonetheless, there has been an increasing recognition in recent years of several strains of *K. pneumoniae* that combine the two characteristics, leading to serious clinical consequences. Carbapenem-resistant hypervirulent *K. pneumoniae* (CRhvKP) usually arises when carbapenem-resistant or hypervirulent *K. pneumoniae* acquire plasmids containing either virulence or CR-encoded genes. A thorough definition of hvKp includes the hypermucoviscous phenotype, the genotype of the associated virulence gene, and the clinical signs of metastatic infection (20). However, the string test is still the optimal phenotypic test for detecting the hypermucoviscous property o*f K. pneumoniae*; it can't be considered the gold standard test for hmKp identification, as it is often described as lacking accuracy and rigidity (21). Therefore, it's important to investigate some genes associated with hypermucoviscous strains. Regarding the antibiotic resistance results, all isolates including the hmKp isolates were MDR. This contradicts the previous belief that highly viscous isolates are often susceptible to antibiotics or do not possess the trait of multi-resistance (22). This is consistent with the possibility that these isolates may become invasive and hypervirulent strains. Analysis of the clinical isolates revealed that *K. pneumoniae* was highly resistant to beta-lactamase enzymes in different patients. These isolates have greater than 99.9% identity. Differences were observed between the strains regarding resistance behavior to levofloxacin and ticarcillin/clavulanic acid (Table 3). Gene sequences corresponding to virulence factors, such as the *mrkD* and *rmpA* villus type 3 genes, were found in hmKp isolates. Similarly, resistance genes, such as bla_{KPC-3}, but not bla_{VIM} were found as well. Biofilm

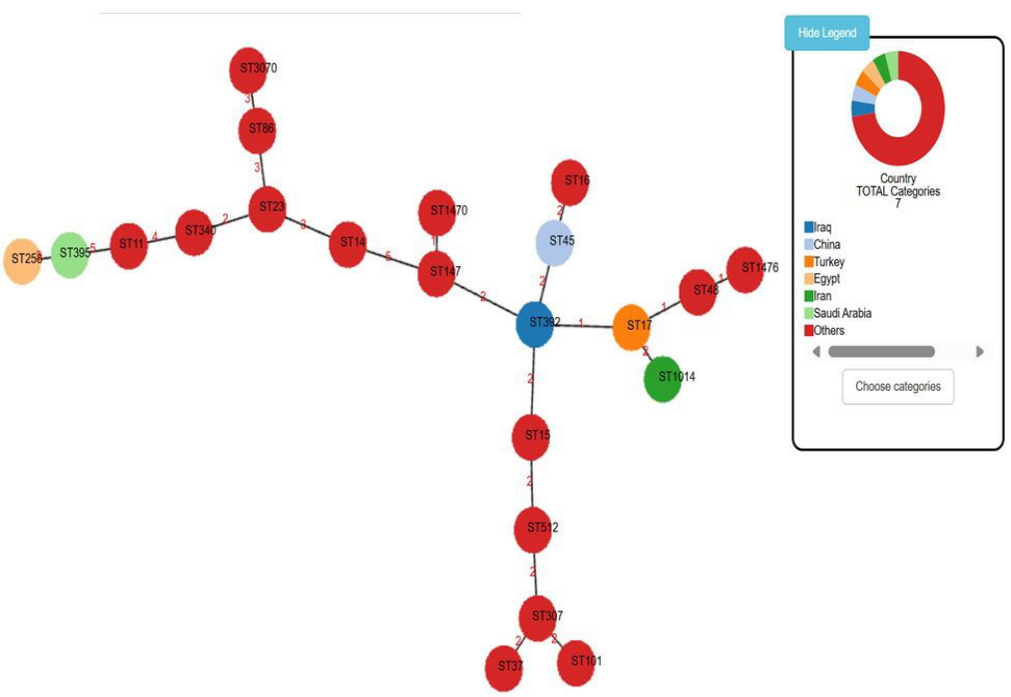

Fig. 3. Minimum Spanning Tree (MST) colored by Country.
The phylogenetic relationships of ST392 and related STs based on geographical origin

production was quantified, and it was observed that all three isolates were moderate biofilm producers. Our results consistently agree with several current studies that had investigated the ability of classical *K. pneumoniae* to be higher than that of hmKp (23). Physically, studies indicate that the formation of the highly viscous capsule hinders contact between cells and biofilm formation. However, hmKp strains often exhibit downregulated expression of type III fimbriae, which is essential for biofilm formation (24). Transcription levels of genes associated with antibiotic resistance (bla_{KPC} and bla_{VIM}), high viscosity, biofilm formation, and adherence (*mrkA* and *pgaA*) were evaluated as described in previous studies (22). The results revealed a significant increase in transcript levels of the *mrkD*, *rmpA*, and bla_{KPC} genes, indicating that the associated proteins' expression may contribute to enhanced antibiotic resistance. However, the bla_{VIM} genes tested appeared to be absent in hmKp isolates. We recently identified three clinical isolates of *K. pneumoniae* ST392 that produce the KPC-3 protein, which is associated with respiratory tract infections found in ST392 clinical isolates. In all visualizations: ST392 (Iraq) clustered near ST15 (blood, China), ST147 (CSF, Saudi Arabia), and ST307 (wound, Turkey). The allelic differences between ST392 and neighboring STs ranged from 1 to 2 loci, indicating strong evolutionary relatedness. The diversity of countries and sources underscores the global nature of high-risk clones, suggesting possible international transmission routes or shared evolutionary pressures. Clonal group CG147, which includes ST147 and ST392, has been identified as a major contributor to hospital outbreaks due to its carriage of critical resistance genes, such as bla_{NDM-5}, $bla_{OXA-181}$, and occasionally bla_{KPC} (16). The presence of capsular locus KL64 in both ST147 and ST392 further supports the hypothesis of a common evolutionary lineage (25). The close relationship between ST392 and ST15 is particularly concerning, as ST15 is a globally disseminated MDR clone frequently linked to resistance determinants such as CTX-M-15 and carbapenemases. Importantly, the ST392 isolate analyzed in this study harbors key resistance and virulence genes, including bla_{KPC}, *rmpA*, and *mrkD*. bla_{KPC} Encodes *K. pneumoniae* carbapenemase, conferring resistance to carbapenems, a last-resort antibiotic. *rmpA*: Regulates hypermucoviscosity, enhancing virulence by promoting capsule production and resistance to phagocytosis. *mrkD*: Encodes adhesins involved in biofilm formation, facilitating persistence on medical devices and host tissues. This genetic profile mirrors patterns observed in other high-risk clones, suggesting convergent evolution driven by selective pressures in healthcare settings. The phylogenetic clustering observed in this study aligns with previous genomic analyses, reinforcing the role of horizontal gene transfer, mobile genetic elements, and antibiotic pressure in shaping the evolution of these clones.

CONCLUSION

The string test is the primary phenotypical diagnosis for hmKp, while the *rmpA* gene is the most significant genetic marker for hmKp identification. However, the central positioning of ST392 within the MST highlights its potential role as an emerging high-risk clone. Continuous genomic surveillance and detailed characterization of ST392 isolates are crucial for monitoring the spread of resistance and virulence factors. Integrating MLST-based phylogenetics with metadata (e.g., geographical origin, source, year, and genetic markers) provides a valuable understanding of the epidemiology of *K. pneumoniae* and supports proactive infection control measures.

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