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ORIGINAL RESEARCH

Prevalence and Molecular Characteristics of *Klebsiella pneumoniae* Harboring the *Pks* Island from Cancer Patients in China

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Purpose: *Klebsiella pneumoniae* harbors a gene cluster, polyketide synthase island (PKS), which is responsible for colibactin synthesis which induces double-stranded DNA breaks and associated with increased pathogenicity and cancer development. However, there is limited information on *pks*-positive *K. pneumoniae* in cancer patients in China. This study aimed to investigate the prevalence and molecular characteristics of *K. pneumoniae* harboring the *pks* island in patients with cancer in China and to explore its potential pathogenicity and clinical significance.

Methods: Among 279 nonrepetitive *K. pneumoniae* isolated from all cancer patients in China, the presence of *pks* genes were determined by PCR and the molecular characteristics were detected by whole-genome sequencing. Clinical characteristics and antimicrobial susceptibility were also investigated.

Results: The *pks* gene cluster was detected in 35 (12.54%) of the 279 isolates. All isolates were less resistant to most antimicrobial agents, and there were no significant differences in the rates of susceptibility between *pks*-positive and *pks*-negative isolates to most antibiotics, except for sulfonamides. Among *pks*-positive isolates, ST23 (19, 54.29%) and K1 (17, 48.57%) were the dominant sequence types and serotypes, respectively, and the majority harbored multiple virulence genes, including aerobactin, enterobactin, salmochelin, and yersiniabactin.

Conclusion: The distribution of *pks*-positive *K. pneumoniae* in different types of cancer combined with its hypervirulent determinants highlighted the potential pathogenicity of genotoxins, which requires close clinical attention and epidemic tracking.

Keywords: *PKS* island, colibactin, *Klebsiella pneumoniae*, cancer, hypervirulent *Klebsiella pneumoniae*

Introduction

Klebsiella pneumoniae is not only a common pathogen causing nosocomial infections but also an important cause of community-acquired infections, that colonizes human mucosal surfaces such as the nasopharynx and the gastrointestinal tract.[1] In recent years, with the prevalence of multidrug-resistant and hypervirulent *K. pneumoniae* in the world, the incidence rate of *K. pneumoniae* infections has risen dramatically, such as urinary tract infection, pneumonia, liver abscess, and so on.[1] Compared with the classical *K. pneumoniae* (cKp), hypervirulent *K. pneumoniae* (hvKp) possesses higher toxicity, which can cause severe infection in immunocompromised people, with high pathogenicity and mortality.[2] Although many factors contribute to the high virulence of the hvKp, virulence factors, including capsule, siderophores, lipopolysaccharide, and fimbriae, play an essential role in the pathogenesis of several diseases.[3–6] Numerous reports have

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shown that K1 and K2 serotypes are strongly associated with hvKp among 79 serotypes of *K. pneumoniae*.[7,8] Additionally, some genes, rmpA, iutC, and ybtA, which are responsible for the production of high viscosity, iron-acquiring factors, aerobactin and yersinia actin, respectively, have been associated with the hypervirulence of *K. pneumoniae*.[5,9] Recently, the *pks* (polyketide synthase) gene cluster, as a new virulence factor, has aroused great public concern.[10]

The p*ks* gene cluster is a genetic locus that was first described in some *Escherichia coli* strains from the B2 phylogroup by Nougayrede in 2006.[11] It contains 19 genes (clbA to clbS) with 54 kb and encodes a multi-enzyme complex capable of producing a genotoxin called colibactin. Previous studies have shown that colibactin can cleave host DNA double strands, resulting in cell cycle arrest, DNA damage, and mutations.[12,13] Moreover, it increases the likelihood of serious complications of bacterial infections. For instance, production of colibactin by *pks*$^{+}$ *E. coli* exacerbates lymphopenia associated with septicemia and increases the morbidity and mortality of urosepsis and meningitis in immunocompromised mice.[14,15] Additionally, *pks*-positive *E. coli* has been associated with mutations in colorectal cancer.[13,16,17] Subsequently, the *pks* island has also been found in several other members of the *Enterobacteriaceae* family, such as *Citrobacter koseri*, *K. pneumoniae*, and *Enterobacter aerogenes*, but was found to be relatively infrequent.[18–20] A study in Europe showed that the prevalence of the *pks* gene cluster was 34% in *E. coli* strains of phylogenic lineage B2, but only 3.5% in *K. pneumoniae* clinical isolates.[18] While the predominance of *pks* genes in bloodstream-sourced *K. pneumoniae* is approximately 25.6% and 26.8% in Taiwan and Changsha, respectively,[21,22] little is known about its epidemiology in clinical isolates from cancer patients in China.

Given the potential role of the *pks* gene cluster in cancer and its association with hypervirulence, it is crucial to investigate the prevalence and molecular characteristics of *pks*-positive *K. pneumoniae* in patients with cancer. This study aimed to address this gap by examining the presence of the *pks* gene cluster and analyzing the clinical and molecular features of *pks*-positive *K. pneumoniae* isolates from patients with cancer in China. Understanding the distribution and characteristics of these isolates will provide valuable insights into their pathogenic potential, and inform clinical practice and epidemic surveillance.

Materials and Methods

Bacterial Isolates Collection

A total of 279 non-repetitive clinical *K. pneumoniae* isolates were obtained from all cancer patients in China at Cancer Hospital Chinese Academy of Medical Sciences, Shenzhen Center between January 2022 and June 2024. All cases were diagnosed according to the International Classification of Diseases, 10th Revision (ICD-10) and presented with clinical evidence of infection (including clinical symptoms, laboratory indicators, and microbiological evidence). These strains were isolated from diverse specimens, including sputum, blood, urine, drainage fluid, bile, catheter, gastric juice, vaginal secretion, and wound secretion. The collection, isolation, and culture of all clinical specimens must be performed under aseptic conditions and comply with the standards of CLSI (Clinical and Laboratory Standards Institute) guidelines and WHO Laboratory Biosafety Manual. After being isolated and purified, these strains were preserved at −80 °C in a tube containing 20% glycerol for a long time. The full 10 μL loop of colonies after balancing to room temperature were spread onto the Columbia blood agar (Oxoid, Brno, Czech Republic) and incubated at 37 °C for 24 h in 5% CO^{2} atmosphere. At the same time, the information of these patients was also collected. This study was approved by the hospital ethics committee (Approval No: JS2024-18-1).

Identification and Antimicrobial Susceptibility Testing

Isolates were identified by by matrix-assisted laser desorption-ionization time-of-flight mass spectrometry (MALDI-TOF MS; bioMerieux SA, Lyons, France) according to the manufacturer's protocol. Antimicrobial susceptibility testing was performed using automatic microbial identification and the antibiotic sensitivity analysis system, Vitek 2 Compact (bioMerieux SA, Lyons, France). The results of the antibiotic sensitivity test were determined based on the breakpoints recommended in the guidelines of the 2023 Clinical and Laboratory Standards Institute (CLSI).

Identification of the Pks Gene Cluster in Clinical K. pneumoniae Isolates

Genomic DNA was extracted from 279 clinical isolates using a bacterial DNA extraction kit (Tiangen Biochemical Technology, Beijing, China) and quantified using Qubit 4.0 according to the manufacturer's instructions. PCR was used to detect *pks* genes (clbA, clbB, clbN, and clbQ). The primers and amplification conditions used in the present study for *pks* detection are listed in Table 1.[11] The PCR products were visualized using 2% agarose gel electrophoresis.

The positive of *pks* gene clusters were verified by blasting whole genomic coding ORFs against *E. coli clb* reference genes (GenBank accession: AM229678.1)[11] with both identity and coverage threshold greater than 80%.

Whole-Genome Sequencing and Analysis

A total amount of 0.2 μg of DNA per sample was used as input material for DNA library preparations using the Rapid Plus DNA Lib Prep Kit (RK20208) (Beijing Baiao Innovation Technology, China). Subsequently, the library quality was assessed on the Agilent 5400 system (AATI) and quantified by real-time PCR (1.5 nM). The qualified libraries were pooled and sequenced on Illumina platforms (Illumina, San Diego, CA, USA). Sequencing reads were assembled using Shovill (1.1.0) (https://github.com/tseemann/shovill), and the contamination and completeness of the assembled genome were assessed using CheckM (v1.2.2).[23] Whole-genome annotation was performed using the Prokka software (1.14.6).[24]

SNP distance and phylogenetic tree construction were performed for pks-positive strains. Phylogenetic analysis was conducted using IQ-TREE software (version 2.3.5) and visualized with the ggtree package in R (version 4.4.2). The K159 strain was used as the reference genome, and core genomic SNPs (cgSNPs) were identified using Snippy (v4.6.0) (https://github.com/tseemann/snippy).

Sequence types (ST) and serotypes were determined from whole-genome data using Kleborate (2.2.0)[25] against pubMLST database[26] and Kaptive database.[27]

Virulence genes and antibiotic resistance genes were identified using the ABRicate (1.0.1)[28] and AMRFinderPlus (3.11.14)[29] from genome assembly, respectively.

Statistical Analysis

All analyses were performed with the Statistical Package for the Social Sciences version 28.0 (SPSS, Chicago, IL, USA). Significance of differences in frequencies and proportions was tested by the χ^2 test or Fisher's exact test. A P-value <0.05 was considered statistically significant.

Results

Clinical Characteristics of Pks-Positive K. pneumoniae

Among 279 *K. pneumoniae* isolates, 35 (12.54%) *pks* gene cluster positive representatives were identified, which were mainly isolated from the sputum (20, 57.14%). The clinical characteristics of the patients who isolated *K. pneumoniae* isolates are presented in Tables S1 and S2. The average age of patients with *pks*-positive *K. pneumoniae* was 59, and most of them were male (27, 77.14%). And the diagnosis of lung cancer (15, 42.86%) was predominant in patients harbouring *pks*-positive isolates, followed by gastric cancer (3, 8.57%). But comparing with patients infected by *pks*-negative *K. pneumoniae*, there

Table 1 Primers Used for Amplification of the Tested *Pks* Genes

Target Gene	Primers	Nucleotide Sequence (5'-3')	Temperature (°C)	PCR product size (bp)
clbA	clbAF	CTAGATTATCCGTGGCGATTC	48	1311
	clbAR	CAGATACACAGATACCATTCA		
clbB	clbBF	GATTTGGATACTGGCGATAACCG	52	579
	clbBR	CCATTTCCCGTTTGAGCACAC		
clbN	clbNF	GTTTTGCTCGCCAGATAGTCATTC	54	733
	clbNR	CAGTTCGGGTATGTGTGGAAGG		
clbQ	clbQF	CTTGTATAGTTACACAACTATTTC	48	821
	clbQR	TTATCCTGTTAGCTTTCGTTC		

was no significant difference in age, specimen source, infections position, and sexes in patients harbouring *pks*-positive isolates ($P > 0.05$) (Table 2).

Antimicrobial Susceptibility of Pks-Positive Isolates

There was no significant difference in rates of susceptibility between the *pks*-positive and *pks*-negative *K. pneumoniae* isolates to most antibiotics, including β-lactam/β-lactamase inhibitors, fluoroquinolones, cephamycin, aminoglycosides, and carbapenems, except for sulfonamides (Tables S3 and S4). For example, the susceptibility rates of cefoperazone sulbactam, piperacillin tazobactam, cefuroxime, ceftazidime, ceftriaxone, cefepime, amikacin were 100%, 85.71%, 74.29%, 91.43%, 85.71%, 88.57%, and 100% in the *pks*-positive *K. pneumoniae*, and compared with the *pks*-negative *K. pneumoniae*, where the respective rates for these antibiotics were 95.90%, 88.52%, 72.95%, 84.02%, 77.05%, 84.02%, and 98.36% (Table 3). Although there was a tendency that the pks^+ *K. pneumoniae* isolates were less resistant to carbapenem agents tested versus *pks*-isolates (100% vs 98.36%), the difference was insignificant. Sulfamethoxazole was the only agent to which *pks*-positive isolates were significantly more susceptible than *pks*-negative isolates (100% vs 75.82%, $P<0.001$) (Table 3).

Molecular Characteristics of Pks-Positive K. pneumoniae

In this study, whole-genome sequencing of 35 pks^+ *K. pneumoniae* isolates was performed, and the detailed quality assessment results are shown in Table S5. The average genome size of 35 pks^+ *K. pneumoniae* isolates was 6.02 Mbp,

Table 2 Clinical Data of Patients Infected with *Pks*-Positive and *Pks*-Negative *K. pneumoniae*

Characteristics	N (%) of pks-Positive	N (%) of pks-Negative	P value
Total (279)	35	244	
Age (median, IQR)	63 (53–69)	63 (51–69.25)	0.875
Male (*n*, %)	27 (77.14%)	149 (61.06%)	0.065
Specimen source			
Respiratory	20 (57.14%)	112 (45.90%)	0.213
Urine	4 (11.43%)	42 (17.21%)	0.388
Gastric juice	1 (2.86%)	6 (2.46%)	>0.999
Vaginal secretion	1 (2.86%)	3 (1.23%)	>0.999
Wound secretion	2 (5.71%)	23 (9.42%)	0.687
Drainage	4 (11.43%)	25 (10.25%)	>0.999
Blood	3 (8.57%)	25 (10.25%)	0.994
Infections position			
Lung infection	21 (60%)	108 (44.26%)	0.081
Urinary tract	4 (11.43%)	43 (17.62%)	0.500
Bloodstream infection	3 (8.57%)	25 (10.25%)	0.994
Hepatophyma liver abscess	2 (5.71%)	2 (0.82%)	0.129
Abdominal infection	1 (2.86%)	9 (3.69%)	>0.999
Diagnosis			
Lung cancer	15 (42.86%)	57 (23.36%)	0.014
Gastric cancer	3 (8.57%)	13 (5.33%)	0.702
Liver cancer	2 (5.71%)	10 (4.10%)	>0.999
Nasopharynx cancer	2 (5.71%)	7 (2.87%)	0.704
Ovarian cancer	2 (5.71%)	7 (2.87%)	0.704
Breast cancer	1 (2.86%)	13 (5.33%)	0.832
Colorectal cancer	1 (2.86%)	16 (6.56%)	0.633
Esophagus cancer	1 (2.86%)	18 (7.38%)	0.526
Pancreatic cancer	1 (2.86%)	14 (5.74%)	0.760

Table 3 Susceptibility of *Pks*-Positive and *Pks*-Negative *K. pneumoniae* to Antimicrobials

Antibiotics	N (%) of *pks*-Positive (n=35)	N (%) of *pks*-Negative (n=244)	P value
Sulfamethoxazole	35 (100%)	185 (75.82%)	<0.001
Levofloxacin	30 (85.71%)	178 (72.95%)	0.105
Piperacillin tazobactam	30 (85.71%)	216 (88.52%)	0.630
Cefoperazone sulbactam	35 (100%)	234 (95.90%)	0.619
Cefuroxime	26 (74.29%)	178 (72.95%)	0.868
Ceftazidime	32 (91.43%)	205 (84.02%)	0.371
Ceftriaxone	30 (85.71%)	188 (77.05%)	0.246
Cefepime	31 (88.57%)	205 (84.02%)	0.654
Imipenem	35 (100%)	240 (98.36%)	>0.999
Ertapenem	35 (100%)	240 (98.36%)	>0.999
Meropenem	35 (100%)	240 (98.36%)	>0.999
Amikacin	35 (100%)	240 (98.36%)	>0.999

and the average GC content was 57.38%. The average largest were 0.72 Mbp, and N50 scaffolds were 0.29 Mbp in length, indicating the high assembling quality. The result of genome sequencing showed that virulence associated serotype K1 (17, 48.57%) was the predominant serotype, and K2 accounted for 25.71% in *pks*-positive *K. pneumoniae* (Figure 1). Six other K serotypes (K116 (3), K113 (2), K20 (1), K25 (1), K57 (1), and K62 (1)) accounted for 25.72% of isolates.

Among the 35 *pks*-positive *K. pneumoniae*, the multilocus sequence typing showed that the predominant sequence types were ST23 (19, 54.29%) and ST65 (8, 22.86%), while another six STs each had no more than 3 strains, ST133 (3, 8.57%), ST268 (1, 2.86%), ST348 (1, 2.86%), ST380 (1, 2.86%), ST592 (1, 2.86%), and ST792 (1, 2.86%) (Figure 1). The whole genomic phylogeny and SNP distance were inferred, and we found that there is no direct and recent transmission (cgSNP differences less than 20) among ST23 and ST65 isolates (Figure 1).

Virulence genes were prevalent in *pks*-positive isolates, particularly the siderophore systems (aerobactin, enterobactin, salmochelin, and yersiniabactin) which played different roles in infection within the host. In 35 *pks*-positive isolates,

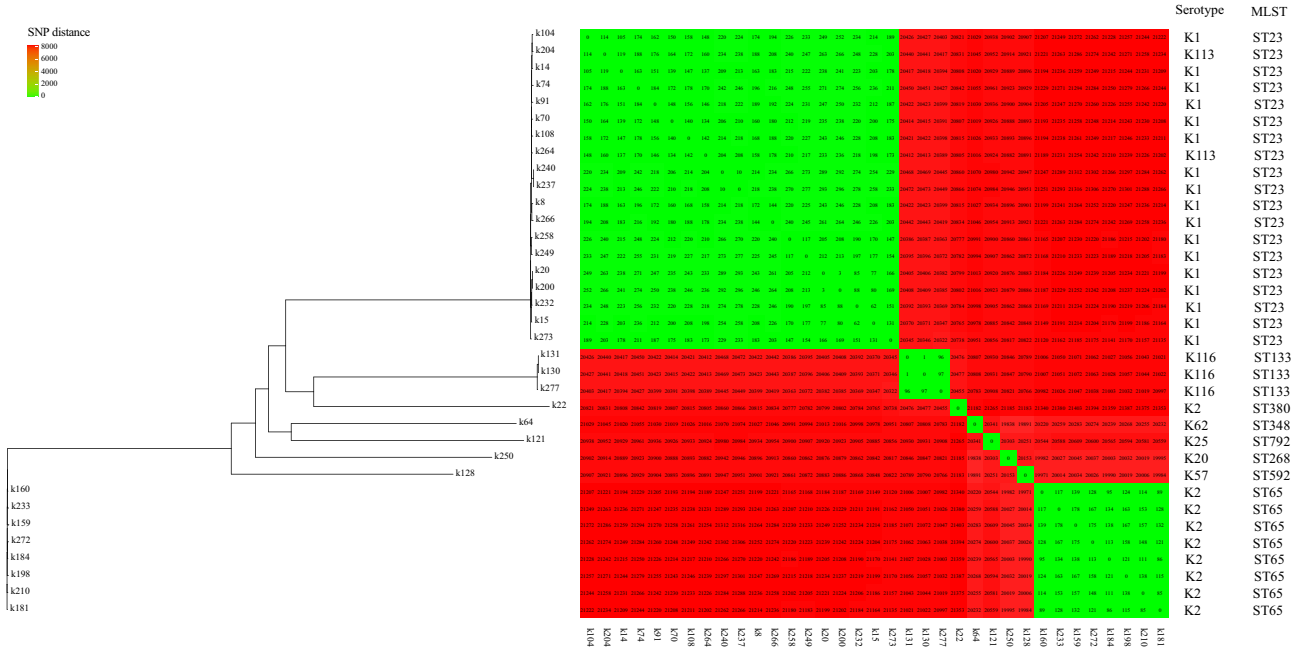

Figure 1 Phylogenetic tree based on SNP sites in core genes of 35 *pks*-positive strains.

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Enterobactin synthase genes (entAB, fepC) and yersiniabactin siderophore system genes (ybtA/E/P/Q/S/T/U/X, irp1, irp2) were at least 97.14%, meanwhile the aerobactin siderophore synthesis system genes (iucA/B/C/, iutA) and salmochelin genes (iroB/C/D/N) were at least 85.71% (Table 4). Furthermore, rmpA genes, which were the positive regulator of the mucoid phenotype, and peg-344, which could encode an intracellular transporter protein, were, respectively, found in 62.86% and 54.29% of *pks*-positive isolates (Table 4).

As for antibiotic resistance genes, *pks*-positive isolates harbored some β-lactamase genes, including blaCTX-M, blaTEM, and blaSHV. Only four isolates proved positive for CTX-M-1 group, and two isolates proved positive for CTX-M-9 group. Additionally, the screen of SHV β-lactamase genes showed that the frequencies of SHV-11, SHV-75, SHV-26, and SHV-207 were 30 (85.71%), 3 (8.57%), 1 (2.86%), and 1 (2.86%), respectively. And only two isolates were blaTEM-1 positive. However, no *pks*-positive isolates proved positive for the genes that confer resistance towards carbapenems.

Table 4 Virulence Genes and Drug Resistance Genes of *Pks*-Positive *K. pneumoniae*

Characteristics	N of *pks*-Positive Isolates	% of *pks*-Positive Isolates
Virulence genes		
RmpA	22	62.86%
RmpA2	4	11.43%
peg-344	19	54.29%
entA	35	100%
entB	35	100%
fepC	35	100%
ybtA	35	100%
ybtE	35	100%
ybtP	35	100%
ybtQ	35	100%
ybtS	34	97.14%
ybtT	35	100%
ybtU	35	100%
ybtX	35	100%
irp1	35	100%
irp2	34	97.14%
iroB	30	85.71%
iroC	30	85.71%
iroD	30	85.71%
iroN	30	85.71%
iucA	30	85.71%
iucB	30	85.71%
iucC	30	85.71%
iutA	31	88.57%
Drug resistance genes (β-lactamase genes)		
CTX-M-3	2	5.71%
CTX-M-14	2	5.71%
CTX-M-15	2	5.71%
SHV-11	30	85.71%
SHV-75	3	8.57%
SHV-26	1	2.86%
SHV-207	1	2.86%
TEM-1	2	5.71%

Discussion

The *pks* gene island, encoding the genotoxin colibactin, has garnered significant attention due to its ability to induce DNA double-strand breaks and transient G2-M cell cycle arrest in host cells.[12] This genotoxic activity suggests that colibactin may contribute to various disease entities, including newborn meningitis, urinary tract infections, bloodstream infections, and potentially cancer development.[15,22,30] In addition, some studies reported that the *pks*-positive *E. coli* was more highly represented in CRC patients and could promote human CRC development.[17,31] Our study is the first to investigate the prevalence and molecular characteristics of *K. pneumoniae* harboring the *pks* island in Chinese cancer patients, providing valuable insights into its epidemiology and clinical significance in this specific population.

Up to now, there have been few epidemic reports on emerging *pks*-positive *K. pneumoniae*. In Europe and Iraq, the occurrence of *pks*-positive *K. pneumoniae* was 3.5%[18] and 7.14%,[20] respectively. In this study, the prevalence of the *pks* gene cluster among *K. pneumoniae* isolates was 12.54%, which was higher than those reported in the literature. But in two previous studies conducted in Taiwan and Changsha, the positive rates of *pks*-positive *K. pneumoniae* isolated from blood was 16.8%[32] and 26.8%,[22] respectively. And some studies revealed that the prevalence of *pks* gene in *E. coli* was high, ranging from 29.2% to 72.7%.[31,33,34] Therefore, we found that the epidemiological distribution of *pks*-positive strains exhibits regional and interspecies differences, which may be associated with environmental, host, and pathogen factors.

Colibactin encoded by the *pks* gene cluster has been shown to induce host DNA damage, thus may contribute to higher mutation rates that drive the occurrence of tumors. By analyzing 3668 Dutch samples of different cancer types, a study found that the colibactin was present in a variety of tumors.[35] Our findings backed up the above results, which documented *pks*-positive *K. pneumoniae* had been isolated from different types of cancer patients. Jens Puschhof et al proved that the *pks* gene cluster was present at a higher frequency in colorectal cancer compared to other types of cancer.[35] And the presence of *pks*-positive *K. pneumoniae* has been found in 4–27% colon cancer patients.[18,21,32,36] However, our findings revealed that *pks*-positive *K. pneumoniae* isolates were predominantly associated with lung cancer patients (42.86%), followed by gastric cancer, which was different from the above researches that reported higher prevalence in colorectal cancer patients. This may be due to the specific patient population and sampling bias, as only parenteral specimens were collected. However, this highlights the potential role of *pks*-positive *K. pneumoniae* in various types of cancer, not limited to colorectal cancer. Further studies are needed to elucidate the specific mechanisms by which *pks*-positive *K. pneumoniae* contributes to cancer development and progression.

There are many similarities between *pks*-positive *K. pneumoniae* and hvKp. Firstly, previous studies have revealed that hvKp were almost exclusively of serotype K1 or K2, and ST23 and ST65 were predominant sequence types.[5,7] On the other hand, the hvKp K1 strains were strongly associated with ST23, while the hvKp K2 strains belong to different STs (ST65, ST86, and others).[5,8] In our study, the great majority (74.28%) of *pks*-positive isolates belonged to K1 or K2 serotype. And all K1 strains belong to ST23, whereas K2 strains were divided into two major clades, ST65 and ST380. To investigate whether there is transmission or possible outbreaks among single ST isolates, whole-genomic phylogeny and SNP distance were inferred, and we found that there is no direct and recent transmission (cgSNP differences less than 20) among ST23 and ST65 isolates, suggesting the patients get these infections from different sources. Two ST133 isolates, k130 and k131, showed almost no cgSNP differences (Figure 1), suggesting direct transmission among their host patients. However, the mechanism of transmission still needs further study. Secondly, another study suggested that hvKp were positive for several virulence factors, such as iucA, iroB, peg-344, rmpA, and so on.[5,7] Our study found that *pks*-positive isolates generally carried several virulence genes. Additionally, the high prevalence of rmpA and peg-344 genes indicates that these isolates may exhibit a mucoid phenotype, which is associated with increased resistance to phagocytosis and host immune responses.[5] Therefore we assumed that the emerging *pks* genotoxic trait is associated with the virulence genes of hvKp. We also found that the *pks*-positive strains in this study showed high sensitivity to most antibiotics, which is likely due to the fact that most of these isolates belong to K1 and K2 serotype to protect bacteria from phagocytosis and inhibit the host immune response. And compared with *pks*-negative strains, *pks*-positive strains showed higher sensitivity to sulfamethoxazole ($P<0.05$), which provided an important reference for antibiotic treatment. Although the rate of MDR in *pks*-positive isolates is low at present, the presence of β-lactamase genes, such as blaCTX-M, blaTEM, and blaSHV, indicates that these isolates have the potential to develop multidrug resistance. Therefore,

continued surveillance of antimicrobial resistance patterns in *pks*-positive *K. pneumoniae* is essential to guide appropriate treatment strategies and prevent the emergence of multidrug-resistant strains.

While our study provides important insights into the prevalence and molecular characteristics of *pks*-positive *K. pneumoniae* in cancer patients, several limitations should be acknowledged. The sample size was relatively small, and only parenteral specimens were included, which may limit the generalizability of our findings. Additionally, the study was conducted in a single center, and further multicenter studies with larger sample sizes are needed to confirm our results.

Recently, it was described that the exposure to *pks*-positive *E. coli* is responsible for mutational signature in colorectal cancer, so it seems that *pks*-positive bacteria can induce mutation of CRC driver genes and, therefore, *pks* may become a marker of CRC carcinogenesis and therapy.[31] Future research should focus on elucidating the specific mechanisms by which *pks*-positive *K. pneumoniae* contributes to cancer development and progression. Additionally, longitudinal studies are needed to monitor the evolution of antimicrobial resistance in these isolates and to develop targeted therapeutic strategies.

Conclusion

Our study highlights the potential pathogenicity of *pks*-positive *K. pneumoniae* in cancer patients in China, emphasizing the need for close clinical attention and epidemic tracking. The findings underscore the importance of continued surveillance and research to better understand the role of this genotoxic pathogen in cancer-associated infections.

Ethics Statement

This study was approved by the ethics committee of Cancer Hospital Chinese Academy of Medical Sciences, Shenzhen Center (Approval No. JS2024-18-1). This study was retrospective and associated with bacterial drug susceptibility and the genetic information of the specimens, hence our ethical petition for exemption from informed consent was accepted. All patients have been informed that their samples will be used for research and have signed informed consent for sample collection. The data of all patients in this study were collected anonymously and ensured the confidentiality of their information. This study was conducted in accordance with the guidelines set out in the Declaration of Helsinki.

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Disclosure

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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