Clinical and microbiological characteristics of hypervirulent *Klebsiella pneumoniae* (hvKp) in a hospital from North China

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Research article

Keywords: Hypervirulent *Klebsiella pneumoniae*, Drug-resistance, Risk factor, Capsular serotype

Posted Date: November 8th, 2019

DOI: <https://doi.org/10.21203/rs.2.17050/v1>

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Version of Record: A version of this preprint was published at The Journal of Infection in Developing Countries on June 30th, 2020. See the published version at <https://doi.org/10.3855/jidc.12288>.

Abstract

Background: The clinical and molecular characteristics of hypervirulent *Klebsiella pneumoniae* (hvKp) in various provinces of China have been reported, however, there have been few reports in Hebei Province, North China. **Methods:** The hvKp was identified by PCR amplification of hypervirulence-related genes, the hypermucoviscous phenotype was determined by the "string test", the drug susceptibility analysis was performed using the VITEK® 2 Compact Bacterial Identification and Monitoring System. The molecular epidemiological characteristics of the strains were analyzed by pulsed-field gel electrophoresis (PFGE), and the capsular serotype of hvKp strain was detected by PCR. **Results:** A total of 59 hvKp were detected, and the hypermucoviscous positive rate of hvKp was significantly higher than that of classical *Klebsiella pneumoniae* (cKp). Univariate analysis showed that the age [odds ratio (OR) = 1.025], a pulmonary disease (OR = 5.886), or a polymorphonuclear (PMN) cell count (OR = 1.045) were statistically significant risk factors for hvKp infection. HvKp are more susceptible to antibacterial drugs than cKp ($P < 0.05$), and one ESBLs-producing hvKp strain was detected. The main capsular serotype of hvKp were K2, K57 and K1. PFGE indicated that the 59 strains of hvKp could be classified into 51 PFGE band types, forming 6 PFGE clusters (A, B, C, D, E, and F clusters, at least three isolates with $> 75\%$ similarity for each cluster). **Conclusions:** The hvKp rate in our study was significantly higher than that in previous reports. Pulmonary disease (OR = 5.373) was an independent risk factors associated with hvKp infection. And K2, K57 and K1 serotype were the main serotype of hvKp in our study.

Background

Klebsiella pneumoniae is a well-known opportunistic pathogen that often causes infection such as pneumonia, sepsis, urinary tract infections and soft tissue infections in immunocompromised people. However, in 1986, Liu reported seven cases of pyogenic liver abscess associated with septic endophthalmitis caused by community-acquired *K. pneumoniae* infections, and some patients were accompanied by other infection syndromes such as pulmonary embolization, purulent meningitis or suspicious prostatic abscess [1]. Subsequently, Nassif reported seven K1, K2 serotypes of *K. pneumoniae* showed high virulence ($LD_{50} < 10^3$ CFU) in the mouse model, and found that four selected strains harbored a large plasmid with size of ~ 180 kb which was absent in the low virulence ($LD_{50} > 10^6$ CFU) strains, further studies revealed that the plasmid contains gene cluster encoding aerobactin and its receptor [2]. In 2004, Fang [3] reported a liver abscess associated with meningitis and endophthalmitis caused by *K. pneumoniae*. The clinical features of the strains reported above differed from classical *K. pneumoniae* such as causing liver abscesses and severe metastatic infections (endophthalmitis, meningitis etc.) even in immunocompromised people. These strains are more virulent than classical *K. pneumoniae* (cKp), so they are designated as hypervirulent *Klebsiella pneumoniae* (hvKp). Initially, the infections caused by hvKp were primarily reported in Asian countries, like China, Singapore, Korea and Vietnam, now, more and more cases are being reported in other parts of the world, and a few hvKp strains even acquired resistance genes [4]. The clinical and molecular characteristics of hvKp in various provinces of China have been reported, however, there have been few reports in Hebei Province. In this

study, hvKp strains were screened from isolates collected in 2015 from a medical center in Hebei Province by PCR amplification of virulence related genes: plasmid-borne *rmpA* (*p-rmpA*, *p-rmpA2*), aerobactin synthase gene (*iucA*), salmochelin siderophore biosynthesis (*iroB*), putative transporter (*peg-344*) and putative carboxymuconolactone decarboxylase family (*peg-589*) together. Risk factors of hvKp infection were also studied.

Methods

Collection and identification of *K. pneumoniae* clinical isolates

A total of 113 non-repetitive *K. pneumoniae* in 2015 were collected from The First Affiliated Hospital of Hebei North University, a Grade-III Class-A Hospitals in Hebei Province from north China, and stored in the Chinese Academy of Medical Sciences Collection Center of Pathogen Microorganisms (CAMS-CCPM-AP). *K. pneumoniae* isolates were identified by detecting *K. pneumoniae*-specific hemolysin gene (*khe*) [5]. Primers, product sizes, and PCR conditions are listed in Table S1 in the supplemental material.

String test

The hypermucoviscosity phenotype of *K. pneumoniae* was determined by string test described previously [4]. The string test is positive when a sterile inoculating loop being able to generate a viscous string of $\geq$ 5 mm in length by stretching a single colony of a strain on a 5% Columbia blood agar plate.

Identification of hvKp

According to the latest research results, hvKp strains were defined by PCR amplification of virulence related genes: plasmid-borne *rmpA* (*p-rmpA*, *p-rmpA2*), aerobactin synthase gene (*iucA*), salmochelin siderophore biosynthesis (*iroB*), putative transporter (*peg-344*) and putative carboxymuconolactone decarboxylase family (*peg-589*) together [6]. Specific primers, product sizes, and PCR conditions are listed in Table S1 in the supplemental material.

Patient information

To investigate the clinical characteristics of hvKp and cKp, the following data were collected by clinical laboratory: gender and age, *K. pneumoniae* infection acquisition, underlying disease (diabetes mellitus, hypertension, cardiovascular disease, neurologic disorder, pulmonary disease, cancer and digestive disease), infection type (pneumonia, liver abscess and urinary infection), catheter (drainage tube, stomach tube, urinary catheter and central intravenous catheter), host responsibility including white blood cell (WBC) count, polymorphonuclear (PMN) cell count. Additionally, the data of empirical antimicrobial therapy was also used to analyze risk factors of hvKp.

Antimicrobial susceptibility test

The susceptibility to antimicrobial agents and the identification of the ESBLs-producing *K. pneumoniae* clinical isolates was performed by VITEK[®] 2 Compact Bacterial Identification and Monitoring System in accordance with the manufactory's instructions. The MICs of the ESBLs-producing strain were further determined using broth microdilution method, and the results were interpreted by 2018 Clinical and Laboratory Standards Institute (CLSI) guidelines. *Escherichia coli* ATCC[®] 25922[™] was used as the quality control strain.

Capsular serotype gene detection by PCR

The K1, K2, K5, K16, K20, K54, K57 capsule serotype genes were detected by PCR as described previously [7,8], using primers listed in Table S1 in the supplemental material, the PCR products were visualized by 1% agarose gel electrophoresis.

Pulsed-field gel electrophoresis (PFGE)

PFGE was performed on 59 hvKp isolates as follows: the genome DNAs of each strain were digested with restriction enzyme *Xba*I for overnight at 37 °C and electrophoresed for 18.5h at 14 °C, 120 degree angle, with switch times of 5 and 25 s at 6V/cm, and *Salmonella* serotype Braenderup strain (H9812) was used as the DNA size marker. The PFGE patterns were analyzed by BioNumerics software using the Dice Similarity coefficient. Strains with similarity coefficient of 100% were considered as the same PFGE type, while more than 75% of similarity was used as the threshold of a cluster.

Statistical analysis

Statistical analysis was performed using SPSS statistical software. The chi-square test was used to analyze categorical variables, and Student's *t* test was used to analyze continuous variables. Difference with $P < 0.05$ was considered to be statistically significant. Logistic regression was used to identify risk factors for hvKP infection. All variables with $P < 0.1$ were included in the multivariate Logistic regression.

Results

HvKp and hmKp identification

Among 113 isolates, 59 isolates were identified to be hvKp by hypervirulent-associated gene detection, the remaining 54 isolates were considered to be cKp. String test was used to determine the hypermucoviscosity phenotype of *K. pneumoniae* (Figure 1), and the proportion of hypermucoviscous *K. pneumoniae* (hmKp) isolates among hvKp (40.68%, 24/59) was significantly higher than that among cKp (14.81%, 8/54), with *p* value lower than 0.01.

Patient characteristics and risk factors for hvKp infection

The patient characteristics of hvKp and cKp are shown in Table 1. The mean age of hvKp group was older than the cKp group (65.64 ± 1.805 vs 56.59 ± 3.359 years, $P = 0.0167$). Pulmonary disease was

highly associated with the hvKp group as their underlying disease. Compared with cKp group, a higher number of patients with the hvKp presented with liver abscess (8.47% vs 0.00%, $P = 0.0287$). Urinary catheter was more frequently to be the source of hvKp than cKp (37.29% vs 20.37%, $P = 0.0482$). We also found that patients with hvKp had a higher PMN cell count than cKp infected patients (16.47 ± 4.623 vs 6.859 ± 0.6932 , $P = 0.0514$). Univariate analysis showed that the age [odds ratio (OR) = 1.025], a pulmonary disease (OR = 5.886), or a PMN cell count (OR = 1.045) were statistically significant risk factors for hvKp infection. Multivariate analysis showed that pulmonary disease (OR = 5.373) was an independent risk factors associated with hvKp infection (table 2).

Table1 Clinical characteristics of *K. pneumoniae*

Characteristics	hvKp (n=59)	cKp (n=54)	P value
Age, years, mean $\pm$ SD	65.64 $\pm$ 1.805	56.59 $\pm$ 3.359	0.0167 ^a
Male sex	43 (72.88%)	36 (66.67)	0.4718
Community-acquired	39 (66.10%)	30 (55.56%)	0.2508
Underlying conditions			
Diabetes mellitus	14 (23.73%)	9 (16.67%)	0.3571
Hypertension	20 (33.90%)	20 (37.04%)	0.7275
Cardiovascular disease	6 (10.17%)	10 (18.52%)	0.2035
Neurologic disorder	18 (30.51%)	17 (31.48%)	0.9110
Pulmonary disease	37 (62.71%)	12 (22.22%)	< 0.0001 ^a
Cancer	11 (18.64%)	6 (11.11%)	0.2632
Digestive disease	4 (6.78%)	5 (9.26%)	0.6268
Infection type			
Pneumonia	17 (28.81%)	16 (29.63%)	0.9241
Liver abscess	5 (8.47%)	0 (0.00%)	0.0287 ^a
Urinary infection	2 (3.39%)	2 (3.64%)	0.9430
Catheter			
Drainage tube	14 (23.73%)	9 (16.67%)	0.3517
Stomach tube	13 (22.3%)	6 (11.11%)	0.1210
Urinary catheter	22 (37.29%)	11 (20.37%)	0.0482 ^a
Central intravenous catheter	13 (22.03%)	10 (18.52%)	0.8290
Surgery	15 (25.42%)	12 (22.22%)	0.6429
Host responsibility			
WBC (10^9 /L)	15.98 $\pm$ 4.588	9.411 $\pm$ 0.234	0.1768
PMN cell count	16.47 $\pm$ 4.623	6.859 $\pm$ 0.6932	0.0514 ^a
Empirical therapy			
β -Lactam/ β -lactamase inhibitor combination	29 (49.15%)	29 (53.70%)	0.6287
Aminoglycosides	1 (1.69%)	2 (3.70%)	0.5070
Carbapenem	6 (10.17%)	6 (11.11%)	0.8711
Glycopeptides	0 (0.00%)	1 (1.85%)	0.2938
Fluoroquinolone	7 (11.86%)	7 (12.96%)	0.8595
Cephalosporin	2 (3.39%)	2 (3.70%)	0.9281
Penicillin	1 (1.69%)	0 (0.00%)	0.3366

^a A *P* value of < 0.05 was considered to be statistically significant. *WBC* white blood cell, *PMN*, polymorphonuclear.

Table 2. Regression analysis of variables associated with hvKp infections

variables	Univariate analysis		Multivariate analysis	
	OR 95% CI	P value	OR 95% CI	P value
Age	1.025 (1.003-1.047)	0.024	1.019 (0.995-1.044)	0.119
Pulmonary disease	5.886 (2.565-13.508)	0.000	5.373 (2.275-12.688)	0.000
PMN cell count	1.045 (0.993-1.100)	0.091	1.042 (0.986-1.101)	0.146
Urinary catheter	1.875 (0.828-4.245)	0.132		

OR odds ratio, CI confidence interval, PMN, polymorphonuclear

Antimicrobial resistance among hvKp and cKp isolates.

K. pneumoniae is naturally resistant to ampicillin, in consistent with this, all the *K. pneumoniae* demonstrated resistance to ampicillin (Table 3). Generally, hvKp isolates are more susceptible to antimicrobial agents compared to cKp (Table 3), examples are ampicillin/sulbactam, cefazolin, ceftriaxone, gentamicin, tobramycin, ciprofloxacin, levofloxacin and trimethoprim/sulfamethoxazole ($P < 0.05$). The ESBL positive rate in cKp isolates was significantly higher than that in hvKp isolates ($P < 0.01$). only one ESBL-producing hvKp was detected (Kp 15-87).

Table 3. The antimicrobial resistance profiling of hvKp and cKp isolates

Compounds	Resistant bacteria n (%)			χ^2	P value
	Total (n=113)	hvKp (n=59)	cKp (n=54)		
Ampicillin	113 (100)	59 (100.00)	54 (100.00)	NA	NA
Ampicillin/Sulbactam	11 (9.73)	1 (1.69)	10 (18.52)	9.082	0.0026 ^a
Cefazolin	13 (11.50)	1 (1.69)	12 (22.22)	11.67	0.0006 ^a
Cefuroxime	14 (12.39)	4 (6.78)	10 (18.52)	3.579	0.0585
Ceftazidime	5 (4.42)	1 (1.69)	4 (7.41)	2.176	0.1402
Ceftriaxone	11 (9.73)	1 (1.69)	10 (18.52)	9.082	0.0026 ^a
Cefepime	4 (3.54)	1 (1.69)	3 (5.56)	1.231	0.2673
Aztreonam	6 (5.31)	1 (1.69)	5 (9.26)	3.209	0.0732
Gentamicin	6 (5.31)	0 (0.00)	6 (11.11)	6.923	0.009 ^a
Tobramycin	2 (1.77)	0 (0.00)	2 (3.70)	5.409	0.0200 ^a
Ciprofloxacin	7 (6.19)	0 (0.00)	7 (12.96)	8.153	0.0043 ^a
Levofloxacin	5 (4.42)	0 (0.00)	5 (9.26)	5.716	0.0168 ^a
Nitrofurantoin	43 (38.05)	19 (32.20)	24 (44.44)	1.792	0.1807
Trimethoprim/Sulfamethoxazole	6 (5.31)	0 (0.00)	6 (11.11)	6.923	0.009 ^a
ESBLs (+)	10 (8.85)	1 (1.69)	10 (18.52)	9.082	0.0026 ^a

^a A P value of < 0.05 was considered to be statistically significant, NA not applicable

ESBLs-producing hvKp

The antibiotic resistance phenotype of an ESBLs-producing hvKp named *Kp* 15-87 was further analysed by MIC determination using broth microdilution method with antibiotics from different groups. *Kp* 15-87 was resistant to nine antibiotics, including penicillin (ampicillin [AMP], MIC > 512 µg/ml; piperacillin [PIP], MIC > 512 µg/ml), cephalosporin I (cefazolin [CFZ], MIC > 512 µg/ml), cephalosporin II (cefuroxime [CXM], MIC > 512 µg/ml), cephalosporin III (ceftazidime [CAZ], MIC = 16 µg/ml; ceftriaxone [CRO], MIC = 512 µg/ml; cefotaxime [CTX], MIC = 256 µg/ml), cephalosporin IV (cefepime [FEP], MIC = 64 µg/ml), and monobactam (Aztreonam [ATM], MIC = 64 µg/ml).

Molecular characteristics of hvKp isolates

The distribution of capsular serotypes of the 59 hvKp was as follows: K2 (17, 28.81%), K57 (15, 25.42%), K1 (14, 23.73%), K20 (2, 3.39%), K5 (1, 1.69%), K16 (1, 1.69%), K54 (1, 1.69%), and 8 strains did not belong to any of the tested serotypes. PFGE results showed that the 59 hvKp could be classified into 51 band types, forming 6 PFGE clusters (A, B, C, D, E, and F clusters, at least three isolates with > 75% similarity for each cluster). PFGE cluster A included 13 K1 strains, cluster B included 5 K2 strains, cluster C included 6

K57 strains and 1 K-non-typable strain, cluster D included 5 K57 strains, cluster E included 9 K2 strains and cluster F included five K-non-typable strains (Figure 2).

Discussion

Although hvKp has been described for more than 30 years since its first report, the gold standard for hvKp laboratory detection has not yet been determined. The putative hvKp infection is primarily determined by clinical features and/or a positive string test (using a sterile inoculating loop being able to generate a viscous string of > 5 mm in length by stretching a single colony of a strain on a blood agar plate). However, the two criteria are becoming increasingly problematic. The clinical definition of hvKp infection is the development of tissue-invasive, community-acquired infections in a healthy host, which excludes hvKp infections in patients with comorbidities, immunocompromization or in a health care environment [9]. Positive string test is used widely to identify hvKp strains currently, but data suggested that string test is not accurate enough to detect hvKp, because hypermucoviscous phenotype was also observed in cKp [6].

The hypervirulence of the hvKp strains is partly mediated by genes on large virulence plasmids (220-kb pK2044, 219-kb pLVPK, 230-kb pRJA166b etc.) [10-12] or chromosomal islands. Curing of hypervirulence-associated plasmids can significantly reduce the virulence of the strain [13]. And a recent study showed that cKp can exhibit a hypervirulent phenotype by acquiring a roughly 170-kb pLVPK-like plasmid and lead to a fatal outbreak of ventilator-associated pneumonia in a Chinese hospital [14]. The genetic structures of these virulence plasmids were characterized by whole genome sequencing (WGS), and the results suggested that these plasmids carry hypervirulent genes, such as siderophore-mediated iron-acquisition system genes, *iucABCDiutA* and *iroBCDN*; mucus phenotype-regulated gene, *p-rmpA/A2*; putative metabolite transporter-encoding gene, *peg-344*; putative carboxymuconate decarboxylase family-encoding gene, *peg-589*. Another way to mediate the hypervirulent phenotype of hvKp is the integrative and conjugative element (ICE), for instance, 76-kb ICE (*ICEKp1*) in the hvKp strain NTUH-K2044 and *ICEKp10* in sublineage CG23-I [15,16]. However, the virulence genes carried on ICEs are not unique to hvKp, the cKp also contains genes encoding yersiniabactin and colibactin. Therefore, a recent study suggested that the genes on virulence plasmids, i.e. *iroB*, *iucA*, *p-rmpA*, *p-rmpA2*, *peg-344* and *peg-589* can differentiate the hvKp-rich and cKp-rich strain cohorts with high accuracy, and these genes can also accurately predict mortality in a murine sepsis model [6].

In this study, we identified hvKp by PCR checking of these virulence genes, and found that 59 among 113 (52.21%) *K. pneumoniae* strains in the studied hospital were hvKp. The detection rate was generally higher than other reports in China. The detection rate of hvKp defined by positive string test was 31.4% (22/70) from 2008 till 2012 in Beijing Chao-Yang Hospital [17]. Another study reported that 45.7% (92/202) *K. pneumoniae* isolates were hvKp defined by aerobactin detection at Beijing Tsinghua Changgung Hospital and Chinese PLA General Hospital from June 2008 to July 2017 [18]. In a multicenter study, *K. pneumoniae* isolates were collected from 10 cities in China during February to July 2013, and hvKp were designated as aerobactin-positive strains. The study found that 37.8% (87/230)

isolates were hvKp, and the prevalence of hvKp varied among different cities, with the highest rate in Wuhan (73.9%) and the lowest in Zhejiang (8.3%) [19]. Meanwhile, the hvKp proportion of our study was significantly higher than those of foreign reports. Based on being positive for either *iucA*, or *iroB* gene, a total of 65 *K. pneumoniae* isolated from 65 patients with hospital-acquired infections in the ICUs of Mansoura University Hospital in Egypt were subjected to hvKp identification, of which only four strains were identified as probable hvKp [20]. With *magA* and *rmpA* genes being amplified by end point PCR, only three (7.9 %) out of 38 *K. pneumoniae* isolates carried one or both hvKp-associated genes in Houston, TX, USA [21]. Of 31 blood cultured *K. pneumoniae* isolates, 4 isolates were identified as hvKp by hypermucoviscous phenotype and *magA/rmpA* genotypes in South Australian hospitals [22].

In the previous risk factor studies, patients with community-acquired infections and underlying disease like diabetes mellitus, cancer and hypertension are more likely to encounter hvKp infection, and males might be slightly more likely to be infected than females [23]. However, in our study, multivariate regression analysis show that pulmonary disease (OR = 5.373) appeared to be independent risk factors associated with hvKp infection. The ratios of patients with older ages or a higher PMN cell count among hvKp infection were higher than those among cKp infection, but only showed a statistical trend in univariate analysis.

Capsule polysaccharide (CPS) is necessary for *K. pneumoniae* virulence, and *K. pneumoniae* has over 70 serotypes (K1 to K78) classified by CPS antigens (K antigens). In contrast to previous studies that the serotype in hvKp is mainly K1 and K2 [19,24], we found that the main capsular serotype of hvKp in this hospital is K2 (17, 28.81%), K57 (15, 25.42%) and K1 (14, 23.73%), and the rate of K57 type was even slightly higher than that of the K1 type. K57 has also been reported in other reports with different detection rates, such as 9.6% (8/84) [25], 13.6% (3/22) [17], 10.4% (10/96) [26] and 18.9% (7/37) [27], suggesting that the serotype distribution of hvKp varied in different regions. The 59 strains of hvKp were also subjected to PFGE molecular typing analysis, and the results suggested that the same serotype strains can almost be classified into the same cluster, but the same cluster can be divided into different band types according to different genetic backgrounds, indicating that PFGE molecular typing technology can further differentiate various strains based on genetic characters.

In consistent with previous reports that hvKp was highly susceptible to commonly used antimicrobial agents compare to cKp, we found that hvKp strains were significantly less resistant than cKp to ampicillin/sulbactam, cefazolin, ceftriaxone, gentamicin, tobramycin, ciprofloxacin, levofloxacin and trimethoprim/sulfamethoxazole. Antimicrobial resistance can be mediated by chromosomal mutations, or by mobile elements such as plasmids, insertion sequences, and transposons. The higher drug susceptibility of hvKp may be due to hvKp hyper-expression of capsule, which may provide a physical barrier against transformation and conjugation and also CRISPR/Cas systems, which defend against foreign DNA that does manage to penetrate or maintain in the cell [16]. However, in recent years, many multidrug-resistant hvKps have been reported, such as ESBLs-producing, carbapenem resistant, or colistin resistant hvKp [28,12,29-31]. In our study, we also identified an ESBL-producing hvKp (*Kp* 15-87) with resistance to a broad spectrum of antibiotics. At present, there is still a lack of controlled trials to evaluate

the efficacy of drugs for the treatment of hvKp infection. Considering the hypervirulence and low-resistance of hvKp, we have hoped for anti-virulence drugs targeting virulence factors as a new type of therapeutic drugs [32]. Examples are siderophores and the corresponding siderophore-membrane receptors or transporters which play critical role in the synthesis, secretion and reuptake of iron chelators, and have a great contribution to growth and virulence of hvKp [33-35]. K1-serotype capsules are expressed by the majority of hvKp strains, phage and phage-encoded proteins and novel therapeutic antibodies (Abs) targeting the capsules polysaccharide (CPS) of these hvKp strains are also potential tool to treat and prevent infection with hvKp [36,37].

Conclusions

The hvKp rate was significantly higher than that in previous reports. Pulmonary disease (OR = 5.373) was an independent risk factors associated with hvKp infection. And K2, K57 and K1 serotype were the main serotype of hvKp in our study.

Abbreviations

hvKp: hypervirulent *Klebsiella pneumoniae*; PFGE: pulsed-field gel electrophoresis; cKp: classical *Klebsiella pneumoniae*; OR: odds ratio; PMN: polymorphonuclear; WBC: white blood cell; CI, confidence interval; CLSI: Clinical and Laboratory Standards Institute; hmKp: hypermucoviscous *Klebsiella pneumoniae*; AMP: ampicillin; PIP: piperacillin; CFZ: cefazolin; CXM: cefuroxime; CAZ: ceftazidime; CRO: ceftriaxone; CTX: cefotaxime; FEP: cefepime; ATM: Aztreonam; ICE: conjugative element; WGS: whole genome sequencing; ICU: intensive care units; CPS: Capsule polysaccharide; Abs: antibodies; SD: standard deviation

Declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the Institutional Review Board Ethics Committee of the First Affiliated Hospital of Hebei North University and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. The need for informed consent to participate was waived by the Institutional Review Board Ethics Committee of the First Affiliated Hospital of Hebei North University because we used deidentified retrospective data.

Consent for publication

Not applicable.

Availability of data and materials

All data generated or analysed during this study are included in this published article and its supplementary information files.

Competing interests

The authors declare that they have no conflict of interest.

Funding

This study was supported by the CAMS Initiative for Innovative Medicine (grant number 2016-I2M-3-014), National Mega-project for Innovative Drugs (grant number 2019ZX09721-001) and the National Natural Science Foundation of China (grant number 81621064). The study funders had no role in the study design, data collection, analysis, interpretation, manuscript preparation or the decision to publish.

Authors' contributions

YY, CL and XueY designed the study. YY and JL performed data analysis and drafted the manuscript. JL, WZ, XH and TN participated in the collection of strains and clinical information. YY performed the phenotypic and genotypic analysis of the clinical isolates. XinY, XW, CL and XueY reviewed the paper and provided recommendations. All authors read and approved the final manuscript.

Acknowledgements

We thank the entire staff at Beijing Key Laboratory of Antimicrobial Agents, Institute of Medicinal Biotechnology, Chinese Academy of Medical Sciences & Peking Union Medical College for their daily contributions to this study.

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Additional File Legend

Additional file 1: **Table S1.** Primers, product sizes, and PCR conditions used for this study.

Figures

Figure 1

Hypermucoviscous phenotype of *K. pneumoniae*

Figure 2

PFGE analysis of the hvKp strains. NT: non-typable serotype

Supplementary Files

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