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Review

Hypervirulent *Klebsiella pneumoniae* ST23: Hypervirulence Meets Antimicrobial Resistance

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Abstract: Hypervirulent *Klebsiella pneumoniae* can cause community-acquired invasive infections such as pyogenic liver abscess and endophthalmitis in healthy individuals compared to the classical *K. pneumoniae*, causing opportunistic healthcare-associated infections. Globally, *K. pneumoniae* clonal group 23, including sequence type (ST) 23, is the dominant clone causing hypervirulent invasive infections. *K. pneumoniae* ST23 clones harbor capsular serotype K1, together with hypervirulence-associated genes such as *ybt*, *iuc*, *iro*, *rmpA*, and *rmpA2* encoded on hypervirulence plasmids, which can define the hypervirulence of *K. pneumoniae* ST23. Historically, antimicrobial resistance (AMR) has not been associated with *K. pneumoniae* ST23. However, the emergence of carbapenem-resistant *K. pneumoniae* ST23 has been reported from various countries, and regional clustering was reported, suggesting the spread of these strains globally. Genomic surveillance by whole-genome sequencing method should be implemented on a global scale to highlight the global and local trends in antimicrobial resistance and hypervirulence in *K. pneumoniae* ST23 clones.

Keywords: *Klebsiella pneumoniae*; hypervirulent; carbapenem-resistant; ST23

1. Introduction

On 17 March 2021, the European Center for Disease Prevention and Control (ECDC) issued its first Rapid Risk Assessment for the emergence of hypervirulent *Klebsiella pneumoniae* (hvKp) ST23 carrying carbapenemase genes in European Union/European Economic Area (EU/EEA) countries [1]. The risk assessment was performed based on the isolation of hvKp ST23 from clinical samples isolated in Ireland. The analysis revealed that several of the isolates carried carbapenemase genes, most frequently $bla_{\text{OXA-48}}$.

Furthermore, on 14 February 2024, the ECDC issued the update for the 2021 Rapid Risk Assessment [2], since the number of EU/EEA countries reporting cases of hvKp ST23 had increased from four to ten countries. The investigation found that strains of hvKp ST23-K1 lineage carrying carbapenemase genes were spreading between healthcare facilities in several European countries, and the risk assessment emphasized the importance of detecting hvKp early to prevent further dissemination in healthcare settings of hvKp ST23 carrying carbapenemase genes as a healthcare-associated pathogen.

In early 2024, the Global Antimicrobial Resistance and Surveillance System on Emerging Antimicrobial Reporting (GLASS-EAR) issued a request for information to assess the current global situation of the increased identification of isolates of hvKp ST23 carrying carbapenemase genes. Among the 43 countries providing the response, 16 countries reported the presence of hvKp, and 12 countries reported the presence of hvKp ST23-K1 strain. The presence of hvKp ST23 was reported in at least one country in all six World Health Organization (WHO)

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regions. Based on these results, the WHO issued a report on the global situation of hvKp ST23 and recommended that member states increase their laboratory diagnostic capacity to ensure early and reliable identification of hvKp, as well as strengthen molecular testing capabilities for detecting relevant virulence genes alongside resistance genes [3].

These warnings from public health organizations underscore the importance of public surveillance to deal with the emerging threats posed by the spread of hvKp ST23 carrying carbapenemase genes. Every physician who cares for patients with bacterial diseases should be aware of the emerging threat of antimicrobial-resistant pathogens [4]. Most notably, hospital-onset carbapenem-resistant Enterobacterales (CRE) were among the three pathogenic microorganisms posing the urgent antimicrobial resistance threats in the United States [5]. However, among various *K. pneumoniae* clones, why should we focus on hvKp ST23? What makes the combination of carbapenem resistance and hypervirulence in *K. pneumoniae* ST23 so much concerning?

2. Evolution of Hypervirulent *K. pneumoniae*

In human hosts, *K. pneumoniae* can play the role of either commensal, opportunistic pathogens, or true pathogens. The rate of gut commensal colonization by *K. pneumoniae* in community individuals varies among different geographic regions. A previous survey on healthy Chinese carriers of *K. pneumoniae* across different Asian countries or regions revealed a notable prevalence of *K. pneumoniae* colonization in these populations. The isolation rate was highest in Malaysia (87.7%), followed by Taiwan (75%), Singapore (61.1%), Hong Kong (58.8%), Chinese mainland (57.9%), Thailand (52.9%), Vietnam (41.3%), and Japan (18.8%) [6].

The majority of *K. pneumoniae* infections globally are opportunistic healthcare-associated infections (HAIs). The most common manifestations are pneumonia, urinary tract infections, and wound infections, any of which can progress to bacteremia [7]. The intestinal carriage of *K. pneumoniae* is a key risk factor for HAIs, and genomic comparisons indicate that gut-colonizing strains are the most common source of *K. pneumoniae* infections in these settings [8].

On the other hand, in the community setting, *K. pneumoniae* can act as true pathogens and can cause severe community-acquired infections (CAIs) in otherwise healthy individuals. Common manifestations of such CAIs include pyogenic liver abscess, endophthalmitis, necrotizing fasciitis, and meningitis, which are often accompanied by bacteremia and/or metastatic spread [9]. The ability to cause invasive CAIs in healthy individuals is the characteristic of hypervirulent *K. pneumoniae* (hvKp) compared to classical *K. pneumoniae* (cKp), which is related to opportunistic HAIs. Infection by hvKp was initially reported from Taiwan as the cause of pyogenic liver abscess and endophthalmitis [10]. Even though the disease burden of hvKp infections has been concentrated in East Asian and Southeast Asian countries, hvKp strains are increasingly detected outside of Asia and have been steadily reported globally [11].

3. Population Genomics of Hypervirulent *K. pneumoniae*

K. pneumoniae population comprises hundreds of lineages that differ from each other by ~0.5% nucleotide divergence [12]. These lineages correspond closely to clonal groups (CGs) defined by core-genome multilocus sequence typing (cgMLST), and the resulting groups are referred to as clones and are identified and labeled based on the seven-gene multilocus sequence types (STs) [13]. *K. pneumoniae* infections can be caused by diverse clones that are widely geographically distributed. However, a subset of these clones is disproportionately contributing to global disease burden, which can be referred to as global problem clones [14].

In terms of hvKp, infections are dominantly related to the same subset of global problem clones. The most common clonal group is CG23, including ST23, ST57, ST26, and ST163 [15]. ST23 and its one- (ST1005 and ST218) and three-locus (ST65) variants comprised 80% of isolates from patients with liver abscess in Taiwan [16]. Furthermore, ST23 was identified among the most common clones associated with *K. pneumoniae* bloodstream infections in the Asia-Pacific region, accounting for 8.5% of isolates from blood cultures [17]. These results showed that *K. pneumoniae* ST23 clone is associated with invasive infections, such as pyogenic liver abscess and bacteremia, making the clone a hypervirulent clone.

4. Genetic Background of Hypervirulence in *K. pneumoniae*

Numerous genetic factors contribute to the ability of *K. pneumoniae* to cause diseases in humans, and these disease-related genes are called pathogenic genes. *K. pneumoniae* strains universally harbor a subset of chromosomally encoded core pathogenic genes to establish opportunistic infections in humans. These genes include *ent* encoding the core siderophore enterobactin, *fim* encoding type 1 fimbriae, *mrk* encoding type 3 fimbriae, as well as variable capsular polysaccharide (K antigen) and lipopolysaccharide (O antigen) [14].

Among 79 capsular serotypes of *K. pneumoniae*, capsular types K1 and K2 are associated with invasive infections in humans as well as in mouse models [7,18]. Notably, the capsular serotype of most of ST23 strains studied so far was K1, suggesting that capsular serotype K1 is one of the major components of hypervirulence in *K. pneumoniae* ST23 clones [12,19]. However, it has been shown that non-K1/K2 hvKp strains could cause invasive infections and lethal diseases, suggesting that the capsular serotype alone cannot define the hypervirulence of hvKp ST23 [9].

In addition to the core pathogenic genes mentioned above, numerous accessory gene-encoded virulence factors are shown to enhance the severity of *K. pneumoniae* infections and the propensity to cause disease. Accessory siderophores such as yersiniabactin, aerobactin, and salmochelin (encoded by *ybt*, *iuc*, and *iro*, respectively) are associated with invasive CAIs in humans compared with classical HAIs or asymptomatic carriage [12]. Colibactin, encoded by *clb*, is primarily associated with the liver abscess clones CG23 and CG66 [14], and in CG23, it is restricted to the globally disseminated CG23-I sublineage that accounts for most CG 23 liver abscess cases [20]. Hypermucoviscosity has been regarded as one of the most important virulence factors for *K. pneumoniae*. This phenotype originates from capsular overproduction due to the presence of one or both of the accessory regulator genes *rmpA* or *rmpA2* [11]. Even though these genes are preferentially present in hypervirulent clones such as CG23 [13], capsular overproduction can also be observed in cKp strains, suggesting that the capsular overproduction cannot define the hypervirulence of hvKp ST23 by itself [9].

Virulence-associated genes, including *iuc*, *iro*, *rmpA* and *rmpA2*, are located on the virulence plasmid, and the presence of the virulence plasmid in the isolated strain is recognized as an important predictor for clinical hypervirulence [21]. In addition, a recent study revealed that hvKp strains carrying all of these virulence genes, namely *iucA*, *iroB*, *rmpA*, *rmpA2*, together with *peg-344*, can increase the severity of disease and mortality compared to cKp strains without some or all of these genes [22]. These results suggest that the presence of these virulence genes could define the hypervirulence of *K. pneumoniae*.

5. Antimicrobial Resistance in Hypervirulent *K. pneumoniae*

Historically, antimicrobial resistance and hypervirulence have been associated with non-overlapping populations of *K. pneumoniae* [14]. In CAI-associated hvKp clones such as ST23 antimicrobial resistance (AMR) was only occasionally reported, whereas AMR was prevalent in HAI-associated cKp clones. However, the convergence of AMR and hypervirulence in the same strains has been eroding the boundary between AMR and hypervirulent clones, escalating the public health threat posed by *K. pneumoniae*.

The convergence of carbapenem resistance and hypervirulence is based on three distinct mechanisms. The first one is the acquisition of a carbapenem-resistance plasmid by hypervirulent *K. pneumoniae* strains, resulting in carbapenem-resistant hypervirulent *K. pneumoniae* (CR-hvKp) [23]. The second one is the acquisition of virulence plasmid by classical *K. pneumoniae* strains, leading to the emergence of hypervirulent carbapenem-resistant *K. pneumoniae* (hv-CRKp), such as hv-CRKp ST11 clones spreading in China [24]. The third one is the emergence of a hybrid plasmid by the recombination of carbapenemase genes into a virulence plasmid [25].

CR-hvKp ST23 strains have been reported from various geographic regions. KPC-2-producing hvKp ST23 strains have been identified in Argentina [23], China [26], Poland [27], and the United States [28]. KPC-3-producing hvKp ST23 strain was isolated in China [29]. HvKp ST23 strain carrying a highly transmissible plasmid encoding KPC-2 and VIM-1 was isolated in Chile [30]. Furthermore, among the 104 carbapenem-resistant *K. pneumoniae* isolates from various healthcare institutions in Japan, eighty-three isolates harbored $bla_{\text{IMP-6}}$ and 21 isolates harbored $bla_{\text{IMP-1}}$, among which 4 *K. pneumoniae* ST23 strains with virulence genes could be identified [31]. It is of particular concern that carbapenem-resistant hvKp ST23 strains have been reported from different countries across the globe, suggesting that these strains may have been spreading globally. However, most of these studies have reported on the isolation of a single strain, and the genetic relationship between isolated strains has not been shown. If the strains did not spread within a specific location, we would have a chance to contain these strains before they spread more widely.

However, a recent report from Ireland revealed an alarming situation in the country [19]. A total of 1769 *K. pneumoniae* isolates were submitted to the Galway Reference Laboratory Services (GRLS) [32] between 2019 and 2023, and 1095 (62%) isolates carried a carbapenemase-encoding gene. The GRLS has received and sequenced all first instances of carbapenemase-producing Enterobacterales (CPE) isolates detected in patients in Ireland since 2018. This service is linked to the national program of testing rectal-colonizing *K. pneumoniae* isolates with CPE upon hospital admissions, together with testing *K. pneumoniae* isolates suspected on clinical grounds as hvKp from the outbreak-associated hospitals, therefore making the collection of isolates biased towards carbapenemase

producers. Carbapenemase-non-producing hvKp associated with rectal colonization would not be detected and may represent a much higher proportion of hvKp than is apparent from this study.

A total of 96 ST23 *K. pneumoniae* genomes were identified in the dataset, and 90 genomes comprising 88 genomes belonging to the capsular type 1 clade (ST23-KL1) and 2 genomes belonging to the capsular type 57 clade (ST23-KL57) were used in downstream analysis. Among these 90 isolates, 76 isolates (84%) carried carbapenemase genes, including $bla_{\text{OXA-48}}$ (73/76), $bla_{\text{OXA-181}}$ (1/76), and $bla_{\text{NDM-1}}$ (2/76). The isolates were collected from patients in 18 healthcare facilities across Ireland, consisting of isolates from screening swabs (n = 59), invasive infections (n = 19), non-invasive infections (n = 11), and the hospital environment (n = 1). The ST23-KL1 genomes were compared with all ST23-KL1 genome sequences in the Pathogenwatch database (n = 424), and the phylogenetic comparison by using TreeCluster identified 88 clusters. The Irish isolates fell into two distinct clusters. The largest cluster consisted of 72 isolates, of which 68 isolates were collected from 8 hospitals located in the South and Southeast Ireland. The genomic cluster resembled most closely with the genomes from India, Vietnam, and Canada. The second cluster consisted of 9 isolates and was collected from 3 hospitals located in the East of Ireland. This genomic cluster resembled most closely with the genomes from India, Japan, and the United States.

These results warn us that, once a carbapenem-resistant hvKp ST23 clones are introduced into a geographic legion, either within locally or abroad, these strains could have the potential to spread in the community and cause CAIs in colonized patients with these strains. Due to the multidrug resistance of these strains, antimicrobial treatment of these patients could be challenging.

6. Genomic Surveillance of Carbapenem-Resistant Hypervirulent *K. pneumoniae*

Genomic surveillance has been implemented in elucidating the patterns of clonal spread of carbapenem-resistant *K. pneumoniae* in Europe [33]. Given the genetic diversity and complexity of *K. pneumoniae* clones, whole-genome sequencing (WGS) is becoming a crucial tool for epidemiology and surveillance of this pathogen. It is particularly useful to monitor global emergence and distribution of problem clones, track the convergence of hypervirulence and antimicrobial resistance, and explore links between asymptomatic reservoirs and clinical infections.

Regarding the spread of carbapenem-resistant hvKp ST23 on a global scale, the importance of surveillance cannot be overstated. Considering the nature of infections by hvKp ST23, *K. pneumoniae* isolates from patients with community-acquired invasive infections, especially with unusual metastatic infections, should be the primary target of surveillance. In addition, to elucidate the incidence of gastrointestinal carriage of hvKp ST23 in the community, utilization of surveillance cultures of *K. pneumoniae* in certain settings (e.g., admission in intensive care units) or of genomic data from national or sub-national population study, such as performed in Norway [34], should be considered.

Genomic analysis of these isolates by using WGS should be an ideal method to monitor and explore the epidemiology of this pathogen, since WGS will allow the simultaneous detection of species, lineage, K-loci, O-loci, core and accessory virulence genes, and antimicrobial resistance genes. Furthermore, WGS is particularly useful to determine strain relatedness with which to assess evidence of clonal transmission and dissemination. Several genome analysis tools are publicly available and utilized to analyze publicly available *K.* genomes to highlight the global trend in antimicrobial resistance and hypervirulence [35].

7. Conclusions

The global emergence and spread of carbapenem-resistant hvKp ST23 warrants the surveillance effort to monitor both antimicrobial resistance and hypervirulence in *K. pneumoniae* strains isolated from both healthy individuals and clinical patients. Whole-genome sequencing is an ideal tool for this surveillance, and the genomic information of *K. pneumoniae* should be collected from as many countries and regions as possible to track the global spread of this clone and analyzed by genome analysis tools to highlight the global and local trend in antimicrobial resistance and hypervirulence in *K. pneumoniae* ST23 clones. The surveillance efforts by ECDC and WHO should be supported and implemented by the participating parties and institutions. Through these efforts, we would be able to reveal the real nature of this formidable pathogen.

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A.T.: conceptualization, writing—original draft preparation and editing; L.K.S.: writing—reviewing and editing. Both authors have read and agreed to the published version of the manuscript.

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Conflicts of Interest

The authors declare no conflict of interest.

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