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"Hypervirulent *Klebsiella Pneumonia:* From Community-Acquired Infections to Multidrug Resistant Hospital Threats"

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KEYWORDS

Hypervirulent *Klebsiella pneumoniae;* Virulence factors; Antimicrobial resistance; Liver abscess; metastatic infection; Molecular diagnosis

ABSTRACT:

Hypervirulent *Klebsiella Pneumoniae (*hvKp) has emerged as important and rapidly expanding public health concern due to its capacity to cause severe, invasive infections in both immunocompromised and otherwise healthy individuals, unlike classical *K pneumoniae* strains that are commonly linked to hospital acquired infections, hvKp is associated with community-acquired disease and is cauterized by its ability to disseminate from primary infection sites to multiple distinct organs, leadings to complication such as liver abscess, blood stream infection, meningitis, endophthalmitis, and pneumoniae. Enhanced photogenic potential of hvKp is largely attributed to key virulence determinants including a thick capsular polysaccharides layer, hypermucoviscosity phenotype, efficient iron acquisition system such as aerobactin, and virulence- associated plasmids-encoded genes. Over recent decades, hvKp has demonstrated a significant shift in epidemiological patterns, with increasing reports from hospital settings and high-risk patient populations such as individuals with increasing with diabetes mellitus and prolonged healthcare exposure. Particular concern is the growing convergence of hypervirulence, antimicrobial resistance, including carbapenem resistance, which complicates therapeutic management, contribute to adverse clinical outcomes. Accurate identification of hvKp requires integration of clinical presentation with phenotypic and molecular diagnostic approaches targeting specific virulence markers. Effective management relies on timely diagnosis, Appropriate antimicrobial therapy, adequate source control, wild infection prevention depends on surveillance, antimicrobial stewardship, strict hospital hygiene practices. This review summarizes current understanding of hvKp epidemiology, structural and functional characteristics, pathogenic mechanisms, post susceptibility, clinical manifestations, diagnostic strategies and treatment challenges highlighting urgent need for improved detection methods and novel therapeutic inventions to limit its global impact.

1. Introduction

Hypervirulent *Klebsiella Pneumonia* **(hvKp)** is high aggressive form of *Klebsiella pneumonia* that has recently become a serious global health concern [1,4]. Classical *K. pneumoniae* mainly induce hospital acquired infection in immunocompromised or elderly patients, whereas hvKp consist ability to induce undesirable infection in healthy persons living community [1,3]. The hypervirulent form was first recognised in Asia, particularly in Taiwan, where it was identified significant major role to induce of liver abscesses in patients without underling diseases [3,7]. Considering that hvKp have

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reported all across globally, is known to induce a worldwide range of infections involving pneumonia bloodstreams infections meningitis as well as eye infections [1,4,5]. A distinguishing trait of hvKp infection their cability to spread from primary side to distinct organs leading to complications and increased morality [3,7].

The strong disease-causing ability of hvKp is mainly due to its unique virulence characteristics. These contains production of thick capsules of polysaccharide efficient iron-acquisition system and the presence of specific virulence often located on plasmids. [1,2,6] These features help the bacterium escape from immune response of host and endure inside human body [6].

In laboratory settings hvKp strains often shows a sticky and shiny appearance due to excessive capsule production, although this feature alone is not sufficient for accurate identification [3]. Therefore, genetic and molecular methods are increasingly used to distinguish hypervirulent stains from classical ones [3,6]. Initially the most hvKp's trains were susceptible to commonly used antibiotics. However recent studies have reported coming into view of hypervirulent strains that are opposed to multiple antibiotics [2,4,7]. The convergence of high wireless and antibiotic resistance had made hvKp a significant public health threat and complicated treatment options [2,4]. A clear understanding of the epidemiology virulence mechanisms and resistance Pattern of hypervirulent *Klebsiella Pneumoniae* is essential for early diagnosis effective treatment and the development preventive strategies [1,5,6]

2. History and evolution

2.1. Early recognition and historical background: -

Klebsiella pneumoniae has been known for many years as a bacterial pathogen main associated with hospital acquired infections. Earlier study described that pneumonia urinary tract infections bloodstream infect infections, wound infections especially in elderly individuals those who with weakened immune systems. These infections where usually caused by classical *klebsiella pneumoniae* strains and showed limited ability to evade distinct organs [1,2]. Before the discovery of hypervirulent strains *klebsiella pneumoniae*. Severe invasive infections in health where individuals were rare, and cases acquired cases were uncommon. This remained unchanged until unusual clinical cases began to appear in Asia [3,4].

2.2. Emergence The hypervirulent Pathotype

In the late 1980s And early 1990s Clinicals in Taiwan Reported a Form of *Klebsiella Pneumoniae* Infection. Previously healthy individuals developed liver abscesses without any prior liver diseases. Many patients showed spread of infection to the brain and lungs leading to serious ramifications like endophthalmitis and meningitis [3,15]. Further investigations showed that these infections were caused by a distinct group of strains with much higher virulence. These trains were named hypervirulent *klebsiella pneumoniae* (hvkp). Unlike classical strains hvKp caused severe community acquired infections and had a strong ability to spread within the body [2,4].

2.3. Global spread:

Initially hvKp Infections wear reported mainly East and Southeast Asia. Over time, similar cases Where documented In Europe, North America, South America and regions. These reports confirmed that hvKp was Limited To a specific geographic area [4,7,9]. Studies from different countries showed that hvKp retained its invasive nature across diverse populations. The increasing number of global reports highlighted hvKp as an emerging worldwide public health threat rather than a regional pathogen [8,12].

2.4. Current understanding of hvKp evolution and chang epidemiology

Current evidence indicates that hvKp evolved through the acquisition of virulence associated genes many of which carried on large plasmids. These genes enhance capsule production and iron acquisition, allowing hvKp to survive and multiply inside the human host. This genetic profile clearly distinguishes hvKp from classical *klebsiella pneumoniae* strains [2,6,10]. The epidemiology of hvKp has also changed over time. While hvKp Was initially associated mainly with liver abscesses in community settings, recent studies reported wide range of infections including pneumonia and bloodstream infection hvKp is now increasingly detected in hospital environments, including intensive care units indicating a shift in its epidemiological behaviours [11,16][Fig1]

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Figure 1: History and evolution of hypervirulent *Klebsiella Pneumoniae*

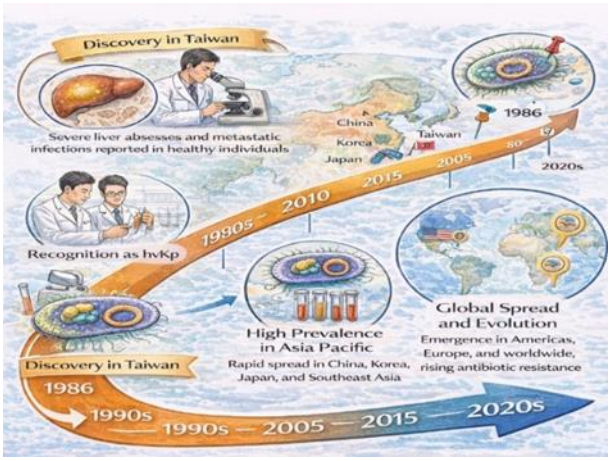

Figure 1 History and evolution of hypervirulent Klebsiella Pneumoniae Highlighting Its Recognition in Taiwan, association With Liver Abscess Syndrome and subsequent global dissemination [1- 4]

2.5. Evolution Towards Antiwar Resistance

Early hvKp strains were generally sensitive to most antibiotics which allowed successful treatment despite sever diseases. This antibiotic susceptibility was a key feature that differentiated hvKp from classical hospital-associated multidrug-resistance stains [2,4]. In recent years hvKp has acquired anti biotics resistance genes through plasmids and horizontal gene transfer. Resistance hvKp strains, including carbapenem resistant variants, have now been reported worldwide. This strain unites dominant virulence on account of finite treatment options pretending serious loot from challenge [12,13,16].

2.6. Current understanding of hvKp evolution

Current research shows that hvKp evolution is an active and ongoing process. The acquisition of plasmids carrying such as rmpA, rmpA2 and aerobactin has played central role in shaping hvKp virulence. These genes increase Capsule thickness and improve iron uptake Which are critical for survival during infection [6,10,14]. Recent studies also indicate increasing inter reaction between hvKp and classical *K. Pneumoniae* strains in hospital settings this hyper virulence with antibiotic ajit resistance this evolutionary convergence represents a threat 2 health and highlight the need for continuous surveillance [11,12,16].

3. Epidemiology of hypervirulent *Klebsiella pneumonia*

3.1 Geographical distribution

Hypervalent Klebsiella Pneumoniae was first identified mainly in East Asia, particularly in Taiwan. Early epidemiological studies consistently reported a high number of hvKp associated liver abscesses cases in this region. These findings established Asia primary geographic origin of hvKp infections [1-3]. Subsequent studies showed that hvKp is no longer confined to Asia cases have been reported from Europe, North America, South America, Australia and Africa. Systematic reviews and global surveillance studies confirm that hvKp has achieved worldwide distribution although the highest burden remains in Asian countries [4,5,20]

3.2 Population affected

Early epidemiological reports showed that hvKp mainly affected healthy individuals without major underlying diseases. Many patients wear middle-aged adults Who developed severe infection despite having no history of immunosuppression. This feature clearly distinguished hvKp from classical K. pneumonia which premier league affects hospitalised or immunocompromised patients [2,6,8]. More recent studies indicate a shift in affected population hvKp is now increasingly reported in elderly patients, individuals with diabetics, liver diseases or cancer and those admitted to intensive care units. This change suggests that hvKp has expanded its epidemiological niche [11,16,19]

3.3 Community-acquired and hospital associated infections

HvKp was initially recognised as community-acquired pathogen most early cases occurred patients who without know recent hospital exposure and community acquired liver abscesses was the most common clinical presentation [2,15]. However, recent epidemiological data show a growing number hospital-associated hvKp infections. hvKp strains have been isolated from hospital outbreaks and ICU settings. This trend reflects adaptation of hvKp to healthcare environments and increased opportunities for transmission within hospitals [11,16,12]

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3.4 Disease pattern and clinical presentation

Epidemiological studies consistently report liver abscesses as the most common infection caused by hvkp. Other frequent clinical manifestations include bloodstream infection, pneumonia, urinary tract infections meningitis and eye infection a defining epidemiological feature of hvKp is its ability to cause met static spread up distinct organs. [3,15,18]. Recent reports indicate diversification of disease pattern. hvKp is now increasingly detected in respiratory and bloodstream infections particularly in hospitalised patients. This expansion of clinical presentation reflects changing epidemiological behaviours [11,22]

3.5 Epidemiological trends and temporal changes

Long term studies and bibliometric analysis show a steady increase in Hvkp-related publications and reported cases Over the past two decades. This trend reflects both improved detection and a true rise in hvKp infections worldwide [20]. One more important epidemiological observation is the rising prevalence that overlap between hvKp and antimicrobial resistance. Surveillance studies document the growing number of hvKp strains carrying resistance genes, including carbapenems the convergence significantly influence the current epidemiological of hvkp [19,21] [fig2]

4 Public health importance:

The epidemiology of hvKp highlights its importance as a global public health concern. Its ability to infect healthy individuals, spread internationally, adapt to hospital settings, and acquire antibiotic resistance make hvKp a high-risk pathogen. Epidemiological studies emphasise the need for continuous surveillance and early detection to limit its spread [4,5,16]

Fig 2: Epidemiology of hypervirulent *Klebsiella Pneumonia.*

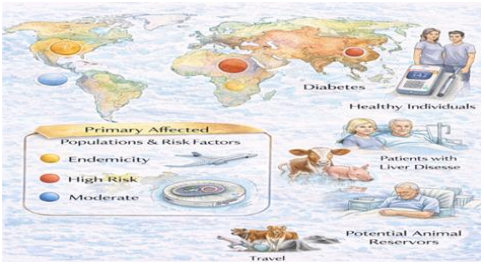

Figure 2. Epidemiology of hypervirulent *Klebsiella pneumonia* showing its origins in east Asia, global dissemination, affected population, and major risk factors (5,6,7,8,9].

4. Structure and function of hypervirulent *Klebsiella pneumonia*

4.1. Cell envelop structure

Hypervirulent *Klebsiella pneumonia* is Gram negative bacterium with complex cell envelope. Its structure includes an inner membrane, a thin peptidoglycan layer, and an outer membrane. The outer membrane contains lipopolysaccharide, which plays a role in immune recognition and protection against host defence [1,6]. Compared to a classical *K. pneumoniae, hvKp* shows structural adaptations that enhance in the host. These include changes in surface components that reduces recognition by immune cells and increase resistance to serum killing [2,18].

4.2. Capsular polysaccharide and its function

The most important structural feature of hvKp is its thick capsular polysaccharide layer. These capsular surrounds the bacterial cell and gives hvKp its characteristics hypermucoviscous appearance. The capsular acts as a anatomic wall helps to depends bacteria through engulfment of cells and complement- mediated killing [3,24]. Production of capsule in hvKp is tightly supervised through specific genes, including *rmpA* and *rmpA2*. These genes increase capsule synthesis, resulting in enhanced virulence. Studies Show that capsule is essential for invasive disease and metastatic spread to distant organs [3,14,24]. [Fig3]

Figure 3: The structure of hyper virulent *klebsiella pneumoniae.*

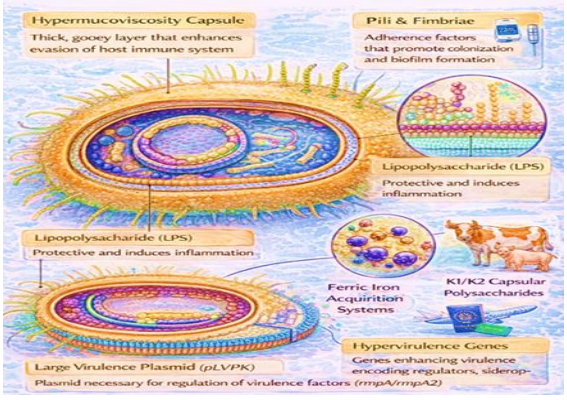

Figure 3. The structure of hyper virulent *klebsiella pneumoniae.* The schematic highlights the, Outer

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membrane lipopolysaccharide, Fimbriae, Iron acquisition System and Viral Plasmid Encoding Key regulators such as *rmpA/ rmpA2*[1-6].

4.3. Hypermucoviscosity phenotype

HvKp often displays a hypermucoviscous Phenotype, which is identified by the formation of viscous strings during laboratory testing. This phenotype is Linked to increase capsule and contributes to bacterial persistence in host tissues [6,18]. However, recent studies show that hypermucoviscosity and hypervirulence are not always identical. Some hvKp strains may lack the classical string test positive but still retain high virulence due to underlying genetic factors [2,6].

4.4. Iron acquisition system and function role

Iron acquisition is a critical functional trait of hvKp. The bacterium possesses multiple Siderophore system that allow it to capture iron from the host environment. Among these, aerobactin play the most important role in hvKp virulence [10,19]. Functional stud demonstrates that aerobactin significantly enhances bacterial growth during infection Contributes more to virulence that other iron uptake systems. This efficient iron acquisition Supports rapid multiplication and severe disease progression [14,22].

4.5. Outer membrane proteins and adhesion

HvKp expresses several outer membrane proteins that support adhesion host tissues. These proteins allow the bacteria to attach to epithelial cells and establish infection. Adhesion is a functional key step to colonisation and disease development [1,6] Some structural proteins also contribute to biofilm formation, which enhances persistence on medical devices and within host tissue. Biofilm formation supports survival in hospital environments and contributes to chronic infections [11,25].

4.6. Interaction between structure and virulence function:

The structural components of hvKp work together to enhance its virulence. The capsule prevents immune clearance, iron acquisition system support growth, and surface proteins aid in attachment and invasion. This coordinate structure function relationship explains the high pathogenic potential of hvKp [2,10]. Recent research emphasis the change in structural regulation directly affects virulence function. Genetic alterations that increase capsule synthesis or iron uptake led to more severe disease's outcomes, highlighting the close link between structure and function in hvKp biology [19,24].

5. Pathogenesis of hyper virulent *Klebsiella pneumoniae*

5.1. Colonisation and entry into the host

Hypervirulent *Klebsiella pneumonia* commonly colonises the human gastrointestinal tract and upper respiratory tract. Several studies indicate that intestinal carriage acts as the main reservoir for HVKP. From this site, the bacteria can either the bloodstream, especially when host Barriers are weakened [1,2,27]. Colonisation is supported by surface structure such as fimbriae and outer membrane proteins, which allow hvKp to attach firmly to epithelial cells. This attachment is critical step in the development of infections [6,10].

5.2. Roll up capsule in immune evasion

The most important pathogenic factor of hvKp is its thick capsular polysaccharide. This capsule surrounds the bacterial cell and protects it from host immune response. The capsule avoids engulfment of cells through neutrophils and microphages and reduce eliminating through additional system [3,24]. HvKp strains often produce large amount of capsule compared to classical strains. This increased capsule production allows hvKp to survive even in healthy individuals with strong immune defence [2,18].

5.3. Hypermucoviscosity and survival advantages

Many hvKp strains shows a hypermucoviscous phenotype, which is linked to increased capsule synthesis. This phenotype helps the bacteria persist in host tissue and resist clearance by immune cells [6,18]. However, recent molecular studies shows that hypermucoviscosity alone does not always indicate hypervirulence. Some hvKp strains may lack strong mucoviscosity but still induce severe disease due to the presence of key virulence genes [27,30]

5.4. Iron acquisition and bacterial growth

For the development and growth of bacteria the requirement Iron is important, that's limited inside human body. hvKp has evolved highly efficient iron acquisition system to overcome this limitation. Among

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these systems aerobactin plays the most important role in hvKp pathogenicity 10,14]. Studies show that aerobactin allows hvKp to grow rapidly during infections, leading to high bacterial loads. This rapid growth contributes directly to tissue damage and disease severity [14,19]. [Fig 4]

Figure 4: Pathogenesis of hypervirulent *Klebsiella pneumoniae*

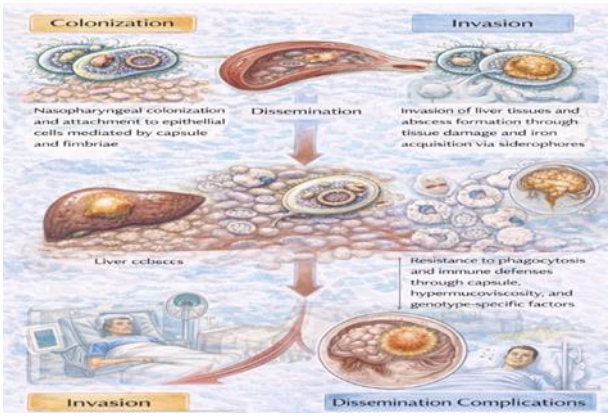

Figure 4 Pathogenesis of hypervirulent *Klebsiella pneumoniae.* Colonisation, immune evasion through capsule and hypermucoviscosity siderophore-mediated iron acquisition, bloodstream invasion and metastatic dissemination to liver, eye, and central nervous system [1,2,7]

5.5. Invasion bacteremia and dissemination

After local infection is established, hvKp can invade surrounding tissue and enter the blood stream. This leads to bacteremia, which is key step in hvkp pathogenesis. Bloodstream infection induced through hvKp reports are increased from both community and hospital settings [15,26]. Once in the bloodstream, hvKp can spread to distant organs. The liver is the most common target, resulting in liver abscess formation. HvKp can also spread to the eyes, brain, lungs and other organs, causing metastatic such as endophthalmitis and meningitis [3,15,29]

5.6. Host damage and inflammatory response

HvKP causes disease through both direct bacterial effects and host inflammatory responses. High bacterial numbers trigger strong inflammation, which leads to tissue destruction and abscess formation. This process explains the sever clinical manifestation seen in hvKp infection [1,22]. The ability of hvKp to persist for long periods within tissue further increase host damage and complicates treatment outcomes [6,18].

5.7. Impact of antibiotics resistance on pathogenesis

Recent studies highlight that antibiotics resistance has become an important factor in hvKp pathogenesis. HvKp strains carrying resistance gene can survive antibiotics therapy allowing prolonged infection and further spread with in the host [19,21,28]. Carbapenem-resistance and extensively drug-resistance hvKp strains have been associated with serve bloodstream infections and poor clinical outcomes. These strains combine strong virulence with limited treatment options, significantly increasing diseases disease severity [21,26,28]. Overall, hvKp pathogenesis involves intestinal colonization, immune evasion through capsule production efficient iron acquisition, bloodstream invasion and dissemination to multiple organs. The interaction of this mechanism explains why hvKp causes severe and invasive disease compared to classical *Klebsiella pneumoniae* strains [2,10,27].

6. Host susceptibility and risk factors

6.1. Susceptibility in healthy individuals

Unlike classical *Klebsiella pneumoniae*, hypervirulent *Klebsiella pneumoniae* can infect people who have no major underlying disease. Several studies reported hvKp infections in young and middle-aged adults who were previously healthy. This shows that hvKp has strong diseases-causing ability even when the host immune system is normal [1,2,27]. The high virulence if hvKp, especially its thick capsule and strong iron-acquisition system, allows it to overcome normal host defences. Because of this host susceptibility is not limited to immunocompromised individuals [3,22].

6.2. Role of diabetes mellitus

Diabetes mellitus is the most consistently reported risk factors for hvKp infections. Many Clinical and epidemiological studies show a high proportion of hvKp infection in diabetic particularly prone to severe manifestation such as liver abscesses and metastatic infections. Case-based studies demonstrate that hvKp can easily spread to multiple organs in diabetic individuals [15,29].

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Figure 5: Host susceptibility and risk factor for hypervirulent *Klebsiella Pneumoniae*

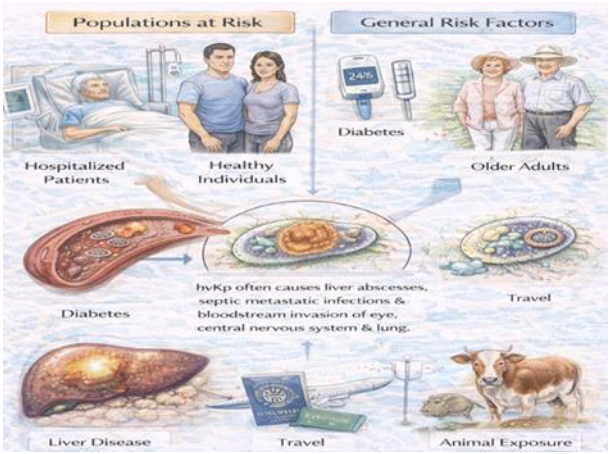

Figure5. Host susceptibility and risk factor for hypervirulent *Klebsiella Pneumoniae*

HvKp affects both healthy individuals and high-risk populations, with diabetic mellitus being the most important predisposing factor. Hospitalization, antibiotics exposure, advance age and gastrointestinal colonization further increase susceptibility and disease [1,7,17]

6.3. Gastrointestinal colonization as risk factor.

Colonization of the gastrointestinal tract is considered a major risk faor hvkp for hvKp infection studies suggest that hvKp first colonizes the intestine and later enters the blood stream. Individuals carrying hvKp in their gut have higher risk of developing invasive disease [2,27]. Disruption of gut barriers, due to illness or antibiotic use, may increase the chance of bacterial translocation into bloodstream, leading to systematic infection [22].

6.4. Hospitalization and Health-care exposure

Recent reports show an increasing number of hvKp infections occurring in hospital settings prolonged hospitalization, intensive care unit admission, and invasive procedure increase the risk of hvKp infection, especially with resistance strains [16,26,31]. Health-care exposure also increases the risk of acquiring carbapenem-resistance and extensively drug-resistance hvKp strains, which are associated with worse outcomes [21,28]

6.5. Antibiotics exposure and selection pressure

Prior exposure to broad-spectrum antibiotics is an important risk factor for hvKp infections. Antibiotics use can disrupt normal microbiota and promote colonization by hvKp. It also creates selection pressure that favour resistance hvKp strains [16,28]

Studies shoes that resistance hvKp strains are increasingly detect in patients with history of antibiotics treatment, particularly in hospital environments [16,19].

6.6. Age and other host factors

Older age has been identified as an additional risk factor in several epidemiological studies. Elderly individuals may have reduced immune functions and multiple comorbidities, increasing their susceptibility to hvKp infections [16,26] Other contributes factors include chronic liver diseases, malignancy and invasive medical devices, although hvKp infections are not limited to these groups [1,31]. In summary, hvKp can infect both healthy and vulnerable individuals. Diabetes mellitus, gastrointestinal colonization, hospitalization, antibiotic exposure and older age are the most important risk factors. Capacity of hvKp to induce disease even in healthy swarms highlight its high virulence and public health importance [2,3,22] [Fig5]

7. Different diseases caused by hypervirulent *klebsiella pneumoniae*

7.1. Liver Abscess

Liver abscess is usual and well recognized disease induced through hvKp. Unlike classical Klebsiella Pneumoniae, hvKp can induce liver abscess in the people without previous liver diseases. These abscesses are often large and many may occur without an obvious source of infection [1,3,15]. HvKp liver abscess are clinically important because they frequently lead to spread of infection to other organs. Many studies describe liver abscess as the primary site from which hvKp disseminates through the bloodstream [15,22].

7.2. Bloodstream infection and sepsis

HvKp is a major of bloodstream infections. Once the bacteria enter the blood, they can induce sepsis, which is a life-threatening condition. bloodstream infection induced through hvKp has proclamed in both community and hospital settings [26,31]. Several studies show that

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hvKp bacteremia is associated with sever disease and high-risk complications, especially when the strain is antibiotic-resistance [19,21,26].

7.3. Metastatic infections

A unique feature by hvKp infection is to aptitude that induce metastatic spread. HvKp cam move from the primary infection site to distant organs through the blood stream. This leads to secondary infection in organ such as the eyes, brain, lungs and bones [3,15]. Metastatic infections are commonly seen in patients with liver abscess or bacteremia and are associated with poor clinical outcomes [15,29].

7.4. Endophthalmitis

Endophthalmitis is a severe eye infection caused by hvKp dissemination to the eye. If often develops suddenly and can lead to permanent vision loss. HvKp-related endophthalmitis has been string linked to liver and blood stream infection [3,15]. Many cases reports describe endophthalmitis occurring even before the primary infection is diagnosed, highlighting the aggressive nature of hvKp [29].

7.5. Central nerve system infection

HvKp can invade the central nervous system and causes meningitis or brain abscess. These infections are rare but sever. They usually occur due to hematogenous spread from the blood stream [3,22]. Central nerve system involvement is associated with high morbidity and often requires prolonged treatment [22,29].

7.6. Pneumonia

HvKp can induce pneumoniae, particularly sever community-acquired pneumonia. Compared to classical strains, hvKp pneumoniae is more invasive and may rapidly progress to bacteremia [2,18]. Hospital-acquires pneumonia caused by hvkp has also been reported, especially in intensive care units and among patients with antibiotic exposure [16,31]

7.7. Urinary tract infections

Although less common, hvKp can induce urinary tract infections. There infections may act as a source for subsequent bloodstream infection, especially in hospitalized patients [31,32]. Urinary tract infection caused by hvKp are increasingly reported in settings where resistant strains are circulating [19,28].

Figure 6: Different diseases caused by hypervirulent *Klebsiella pneumoniae*

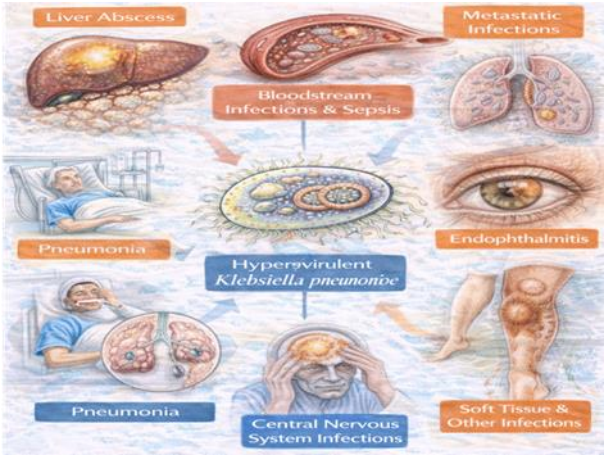

Figure 6 Different diseases caused by hypervirulent *Klebsiella* pneumoniae, include liver abscess, blood stream infection and sepsis, metastatic infections, endophthalmitis, central nervous system infections, pneumonia, urinary tract infections and soft tissue infections [1-7].

7.8. Soft tissue and other infections

HvKp has stated to induce soft tissue infections, including abscesses within skin and muscles. Bone and joint infections have also been described, usually following blood stream spread [22,29]. These infections further demonstrated the ability of hvKp to disseminate and survive in multiple tissue environments [1,22]. Hypervirulent *Klebsiella Pneumoniae* caused a vast scale of disease, as well as liver abscesses, bloodstream infection, sepsis, metastatic infection, endophthalmitis, meningitis, pneumoniae, urinary tract infections, and soft tissue infections. The ability for spreading through blood stream and infect multiple organs distinguished hvKp from classical *Klebsiella pneumoniae* [2,3,5]. [Fig 6]

8. Diagnosis of hypervirulent *Klebsiella* pneumoniae

8.1. Clinical suspicion and initial assessment

Diagnosis of Klebsiella pneumoniae begins with clinical suspicion. HvKp should be suspected in sufferers with severe non hospital acquired infection. such as liver abscess, bloodstream infection, or metastatic infection,

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especially when the patient has diabetes or no obvious immune deficiency [1,3,15,36]. Unlike classical strains, hvKp often causes invasive diseases in otherwise healthy individuals. This unusual clinical pattern helps clinicians consider hvkp during early diagnosis hypervirulent [2,22].

8.2. Conventional microbiological identification

Routine laboratory diagnosis starts with isolation of *Klebsiella pneumoniae* from clinical samples such as blood, pus, urine, or urine or respiratory specimens. Standard cultures methods and biochemical tests identify the organism at the specific level but cannot distinguish hvKp from classical strains [12,17] Therefore, conventional identification is necessary but not sufficient foe conforming hypervirulence [3,27].

8.3. Hypermucoviscosity and string test

The string test is a simple laboratory method used as a screening tool for hvKp. In this test a colony forming unit (CFU) is extended with an bacteriological loop. Origination of carious string test longer than 5 mm is determined as positive strain of hvKp. Many hvKp strains show that they are string test negative and some of classical strains are string test positive. This shows that the string test alone cannot authentic diagnose hvKp [8,30,33].

8.4. Capsular serotyping

Capsular serotyping is an important diagnostic approach. HvKp is commonly associated with capsular types K1 and K2, although others serotypes have also been reported. Detection of these capsular types supports the diagnosis of hvKp [3,27,29]. However capsule types alone do not conform hypervirulence, as not all K1 or K2 strains are hypervirulent [39].

8.5. Detection of virulence gene

Molecular detection of virulence gene is considered one of the most reliable methods for diagnosis hvKp. Key gene include *rmpA, rmpA2, iucA* (aerobactin) and *iroB* (salmochelin). Presence of these genes of these strongly corelates with hypervirulent behaviours [10,13,14]. Among these, aerobactin-related genes are considering the most sensitive markers for hvkp. Studies consistently showed that aerobactin exist in moreover hvKp strains rather than in classical strains [10,38].

8.6. Virulence Plasmid detection

Many hvKp strains carry large virulence plasmids that encode capsule regulators and siderophore system. Detection of these plasmids using molecular methods support hvKp identification. Recent studies showed that virulence plasmids can also carry antibiotics resistance genes, complicating diagnosis and treatment [37,40]. The global spread of conjugative virulence plasmids has increased the importance of plasmids analysis in hvKp diagnosis [37].

8.7. Genome profiling and molecular epidemiology

Genome profiling provides most comprehensive diagnostic information. It allows identification of virulence genes, resistance genes, capsule types, and sequence types in a single analysis. This method is especially useful for outbreak investigation and epidemiological surveillance [9,31,40]. Although highly accurate, whole genome sequencing in expensive and not routinely available in all diagnostics laboratories [31].

8.8. Differentiation from classical and mdr strains

Diagnosis of hvKp requires careful differentiation from classical multidrug-resistant *Klebsiella pneumoniae*. Some strains both hypervirulence and multidrug resistance, while others Carry genetic markers of both but show low actual virulence. This highlights the need for combined phenotypic and genotypic approaches [33,39]. Therefore, diagnosis should rely on multiple criteria rather than a single test [33]. In summary, diagnosis of hvKp demands a mix of microbiological identification, phenotypic screening, molecular testing. Detection of key virulence genes especially aerobactin, along with clinical presentation, provides the most reliable diagnosis. Advanced molecular methods improve accuracy but should not complement, not replace, clinical judgement [10,27,38]

9. Treatment of hypervirulent *Klebsiella pneumonia*

9.1. General principles of treatment

Treatment of hypervirulent calcium pneumonia requires early recognition and prompt initiation of appropriate therapy. Hvkp infections often progress rapidly and can involve multiple organs. Therefore, delay in treatment is associated with poor outcomes [1,3,36]. Management usually requires a combination of effective antimicrobial

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therapy and source control Such drainage of abscesses when present [15,22].

9.2 Antimicrobial therapy for susceptible hvkp strains

Traditionally hvKp strains were susceptible to many commonly used antibiotics. Third generation cephalosporins, such as ceftriaxone, I have been widely used and shown to be effective in treating hvkp infections, specific liver absorptions and bloodstream infection s[3,15]. Other antibiotics include fluroquinolones and aminoglycosides, have also been used successfully in susceptible hvKp infections. Antibiotic selection should always be guided by antimicrobial susceptibility testing [12,17].

9.3 Treatment of liver abscesses and invasive infection

For Hvkp associated liver abscesses antibiotic therapy alone may not be sufficient. Many studies emphasise the importance of Percutaneous or surgical drainage in addition to antibiotics. Drainage reduces the bacterial load and improves treatment outcomes [15,29]. Investment infections with metastatic spread, Such as endophthalmitis or meningitis, Require prolonged antibiotic therapy close monitoring. Early intervention is critical to prevent permanent organ damage [15,22].

9.4 Management of multi drug resistance hvkp

Recent studies show an increase in number of Hvkp strains that are multidrug-resistance or carbapenem resistance. These infections are difficult to treat and require use of last resort antibiotics [19,21,24]. Carbapenems have be used for severe infection caused by extended spectrum beta lactamase is producing hvKp. However, carbapenems resistance hvKp strains require alternative treatments based on susceptibility test[21,28,40].

9.5 Treatment challenges with resistance virulence convergence.

Several recent studies highlighted the emergence of Hvkp strains That carry both hyper virulence and resistance gene on plasmids. These trains often show limited response to standard therapy and are associated with mortality [21,33,37]. Interestingly, some streams with genetic markers at both resistance and hypervirulence show actual virulence, but they still require aggressive antimicrobial treatment because of resistance [33].

9.6 Adjunctive And emerging therapeutic approaches

New takeaway strategies are being explored to overcome limitations of antibiotic therapy. Targeting Virulence factors, rather than bacterial survival, is one emerging approach. Aerobactin synthesis protein have been proposed as antivirulence targets, which may reduce bacterial pathogenicity without increasing resistance pressure [38]. Phage-based therapies and the uses of mucolytic agents to disrupt the hypermucoviscous capsule are also being investigated as adjacent treatment particularly for difficult to treat hvKp infections [21].

9.7 Supportive care and clinical monitoring

Patient with severe HVKP infection often require intensive support care, including management sepsis, organ dysfunction and complications. Close clinical monitoring is essential due to risk of rapid deterioration [16,36]. Follow up imaging and microbial testing are recommended to ensure resolution of infections and prevent relapse.[15] Over all, the treatment of hvKp involves early diagnosis, prompt initiation of effective antibiotics, and appropriate source control. While many hvKp please trains remain susceptible to formal antibiotics, the rise of multi drug resistance hvKp has complicated treatment. Emerging anti-virulence and adjunctive therapies offer potential features options but are not yet standard clinical practise [22,37,38]

10. Infection control and prevention of hypervirulent *Klebsiella pneumoniae*

10.1. Importance of infection control

Hypervirulent *klebsiella pneumonia* causes severe and rapidly spreading infections. Many studies report high mortality and frequent invasive disease. Because hvKp can spread in hospitals and the community, strong infection control measures are essential to prevent outbreak and reduce transmission [3,16,31]. The problem is more serious when hvKp also show multidrug resistance, which limits treatment option and increase the risk of hospital spread [19,21].

10.2. Hospital infection control measures

Strict hospital infections control practises are required to prevent hvKp transmission. Studies from icus add

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teaching hospital shows that hvKp can spread through contact with contaminated surfaces, medicinal equipment, and the hands of health care workers [16,31]. Standard precautions such as hand hygiene, use up gloves and gowns and proper cleaning of hospital environments are strongly recommended. Isolation of infected or colonised patients is important, especially in intensive care units [16,26]

10.3 Screening and early detection

Early detection of hvKp please an important role in infection prevention. laboratory screening of hypermucomviscosity virulence Je Jeans and resistance markers Helps identifying high risk stains early [2,13] . Several studies emphasise a routine survey violence in hospitals, especially for bloodstream infection and carbapenem resistant strains, to prevent unnoticed spread [26,30].

10.4 Control of multi-drug resistance hvkp

The emergence of hvKp strain scare in both virulence and resistance genes has increased infection control challenges. These strains often spread through plasmids that can move between bacteria, increasing the risk of outbreaks [21,37]. In such cases, enhanced infection control measures, including cohorting of patients and strict antimicrobial stewardship are required to limit spread [19,37].

10.5 Antimicrobial stewardship

In appropriate antibiotics use contribute to development and spread of resistance hvKp strains. Many uploaded studies highlight the importance of antimicrobial stewardship programs to reduce unnecessary antibiotics exposure [16,19]. Rational antibiotics use helps slow the emergence of resistance and reduces selective pressure that favours multidrug-resistance hvKp [19].

10.6 Community- level prevention

Although hvkp was initially considered a community-acquired pathogen, recent reports show increasing hospital-associated infections, especially in high-risk individuals such as patients with diabetes [15,29]. Public awareness and timely medical care can reduce complication and limit transmission [15].

10.7 Prevention of Invasive and Metastatic Infection

Early treatment of primary infections, such as liver abscess or bloodstream infection, helps prevent metastatic spread to the eye, brain or lungs. Follow-up monitoring and imaging are recommended to ensure complete resolution [15,36]. Prompt source control also reduces bacterial spread within the host and the healthcare settings [15].

10.8 Ongoing studies

New preventive approaches are being explored. These includes targeting virulence factors such as capsules productions and siderophore systems, which may reduce the ability of hvKp to causes severe diseases [20,38]. Research into vaccines antivirulence therapies is ongoing, but these strategies are not yet available for routine uses [22,38]. Infection control and prevention of hvKp rely on early detection, strict hospital hygiene, antimicrobial stewardship, and surveillance for resistance strains. The growing convergence of hypervirulence and drug resistance makes prevention as important as treatment [16,21,37].

Limitations

This review limited by the heterogeneity of available studies, which includes a high proportion of retrospective analyses, case reports, and single-center observations. Much of the epidemiological and clinical data originates from East and Southeast Asia, potentially limiting global generalizability. These is no universally accepted definition or diagnostic standard for hypervirulent *Klebsiella pneumoniae,* and reliance on variable phenotypic markers and virulence genes prevention strategies remains limited, as most recommendations are based on observational data rather than randomized clinical trials. Finally, the rapid evolving convergence of hypervirulence and antimicrobial resistance means that conclusion drawn from current literature may requore ongoing revision as new data emerge.

Conclusion

Hypervirulent Klebsiella Pneumoniae had become a serious and evolving threat to public health because of its ability to cause severe, fast-progressing infections in a wide range of patients. Unlike classical strains, it can infect healthy individuals and often leads to invasive diseases such as liver abscess, blood stream infections,

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meningitis, eye infections, and other form of metastatic spread. This aggressive behaviour is mainly driven by its thick protective capsules, Increased mucoviscosity, And highly efficient iron uptake systems like aerobactin, which allow the bacterium to survive host immune defence and multiply rapidly.

Although hvKp was first recognised as community-acquired infection in Asia, It has now spread globally and is increasingly detected in hospital environments. A major concern is the growing overlap between hyper virulence and antibiotics resistance, including resistance to last line drugs, which makes treatment more difficult and increase the risk of poor outcomes. Reliable diagnosis therefore depends on combining clinical findings with molecular detection of a key virulence marker.

Timely diagnosis, appropriate antibiotic therapy based on susceptibility testing, and effective source control are essential for successful management. Alongside treatment, continuous surveillance, Strong infection control practises, and responsible antibiotic use as critical to limit the spread and impact of hypervirulent *Klebsiella pneumonia*

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