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CASE REPORT

Invasive Klebsiella syndrome in a case in Bolivia

Síndrome invasivo por Klebsiella a propósito de un caso en Bolivia

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ABSTRACT

Klebsiella pneumoniae is a Gram-negative bacterium that requires oxygen and is capable of fermenting lactose. It can cause serious infections such as pneumonia, bacteremia, and liver abscesses. There are two main subtypes: the classic strain (cKP) and the hypervirulent strain (hvKP), the latter being identified by its highly viscous mucus phenotype and its remarkable ability to invade. The most common hvKP lineage is ST23-K1, although K2 lineages (ST65, ST86, ST66) have also been recognized in different parts of the world, including South America. hvKP can affect healthy individuals and those with additional diseases, with diabetes mellitus being a relevant risk factor due to its association with immune dysfunction and hyperglycemia, conditions that favor bacterial growth and spread through the circulatory system. The case presented concerns a 45-year-old diabetic woman who was admitted with severe diabetic ketoacidosis and pneumonia caused by *Klebsiella pneumoniae*. Despite intensive treatment that included antibiotics and respiratory support, the patient developed multiple lung abscesses and a possible septic embolism, dying after 18 days in intensive care unit.

Keywords: Diabetic Ketoacidosis; Hemangioma; *Klebsiella Pneumoniae*; Lung Abscess; Pneumonia.

RESUMEN

La *Klebsiella pneumoniae* es una bacteria de tipo gramnegativa, que necesita oxígeno y es capaz de fermentar lactosa, pudiendo ocasionar infecciones graves como neumonía, bacteriemia y abscesos en el hígado. Hay dos principales subtipos: la cepa clásica (cKP) y la hipervirulenta (hvKP), siendo esta última identificada por su fenotipo de alta viscosidad mucosa y su notable capacidad para invadir. El linaje más común de la hvKP es el ST23-K1, aunque también se han reconocido linajes K2 (ST65, ST86, ST66) en distintas partes del mundo, incluyendo América del Sur. La hvKP puede afectar a individuos sanos y a aquellos con enfermedades adicionales, siendo la diabetes mellitus un factor de riesgo relevante debido a su conexión con la disfunción inmunológica y la hiperglucemia, condiciones que favorecen el crecimiento bacteriano y la propagación a través del sistema circulatorio. El caso clínico presentado se refiere a una mujer diabética de 45 años que fue admitida con una severa cetoacidosis diabética y neumonía provocada por *Klebsiella pneumoniae*. A pesar del tratamiento intensivo que incluía antibióticos y asistencia respiratoria, la paciente desarrolló múltiples abscesos pulmonares y un posible embolismo séptico, falleciendo luego de 18 días en la unidad de terapia intensiva.

Palabras clave: *Klebsiella Pneumoniae*; Cetoacidosis Diabética; Neumonía; Absceso Pulmonar; Hemangioma.

INTRODUCTION

Klebsiella pneumoniae is an aerobic, gram-negative, lactose-fermenting, rod-shaped bacterium that was first isolated and identified in the late 19th century and has been a common human pathogen ever since.[1] It is a global pathogen with remarkable genetic, phenotypic, and pathogenic diversity, capable of causing serious and potentially fatal infections such as pneumonia, bacteremia, and complicated urinary tract infections. It can acquire new genetic material, which has led to its differentiation into two circulating subtypes: classical (cKP) and hypervirulent (hvKP). These, in turn, have clinical, geographical, and molecular differences and can cause nosocomial infections in immunocompromised patients or those with underlying diseases.[2]

Strains belonging to different lineage groups cause classic infections that are sensitive to drugs in community settings, while hypervirulent infections often have high rates of multidrug resistance. Sequence type (ST) 23 strains are the dominant lineage and are associated with capsule type K1. Other common hypervirulent lineages include ST65, ST86, and ST66, which are associated with capsule K2.[3]

Early descriptions of hypervirulent strains were primarily concentrated in South and Southeast Asia; however, the number of reports of infections attributed to hvPK isolation has recently increased worldwide. In European countries, most reports consist of sporadic cases, with the most frequent combination of ST and K types being ST23-K1, detected in Germany, Italy, Switzerland, and Poland, and most cases are associated with a high virulence score. Recent surveillance studies have shown that in North America, the most frequently detected hvKp was ST23-K1, and greater genetic diversity was detected, including K2 lineages combined with different STs, such as ST66, ST390, and ST375. Sporadic cases have also been reported in South America, in various countries such as Argentina, Brazil, Chile, and Mexico, which also belong to the same lineage described worldwide, ST23-L1 and ST86-K2.[2]

A common feature of the hypervirulent strain is a hypermucoviscous phenotype that develops through the production of different virulence determinants. The mucoid phenotype regulator A (RmpA) is a crucial regulator of capsule production, leading to hypermucoviscosity and increased virulence, and is associated with specific capsular polysaccharide serotypes. Interestingly, serotypes K1 and K2 are most frequently associated with hypervirulent diseases. Notably, a higher percentage of invasive infections, including pyogenic liver abscesses, was associated with serotype K2. Serotypes K1 and K2 show greater resistance to phagocytosis and intracellular death by macrophages compared to other serotypes.[4]

During infections caused by hypervirulent *K. pneumoniae*, multiple foci of infection and metastatic spread are detected. These interesting and specific characteristics are unique to this strain and are generally not observed in infections caused by classical *K. pneumoniae*. The mechanism of multiple foci of infection can be explained by the local invasion of hypervirulent K. *pneumoniae*, its penetration into different tissues, or by a primary infection in the human body that serves as a source of subsequent bacteremia and distant infections.[4] Infection with hypervirulent *Klebsiella pneumoniae* typically occurs in multiple sites and subsequently spreads, making treatment and control challenging.[5] In the last decade, *Klebsiella pneumoniae* has also evolved to become resistant to multiple drugs. In particular, strains have emerged that are resistant to last-line antibiotics, such as carbapenems.[6]

One study suggested that diabetes was directly related to the invasiveness of *Klebsiella pneumoniae*, with the mechanism likely related to high glucose levels stimulating the biosynthesis of capsular polysaccharides and the gene expression of hvKp, which increases resistance to phagocytosis and contributes to the development of an invasive syndrome.[1] In diabetic patients, elevated blood glucose levels lead to vascular disease and a reduction in blood oxygen content in tissues. These factors promote bacterial growth and proliferation; therefore, patients infected with *Klebsiella pneumoniae* are prone to hematogenous dissemination, which can cause multiple abscesses in areas such as the intracranial region, lungs, and liver.[7]

In our case, we describe a patient with severe diabetic ketoacidosis with a focus of infection in the lungs, with a tracheal secretion culture on admission that isolated *Klebsiella pneumoniae* and, as incidental findings, the presence of multiple hepatic hemangiomas.

CASE REPORT

A 45-year-old female patient with a history of type 2 diabetes mellitus, poor adherence to treatment, recurrent urinary tract infections, and uterine myomatosis. She was admitted through the emergency department on 05/19/2025, presenting with a clinical picture of approximately 4 days' evolution characterized by general malaise, asthenia, adynamia, spasmodic abdominal pain in the mesogastrium and hypogastrium, pollakiuria, dysuria, and foul-smelling urine. On physical examination, she was in fair general condition, drowsy, in pain, and restless. Laboratory tests were requested, revealing leukocytosis with a left shift, severe metabolic acidosis with a pH of 6,76, HCO3 of 1,3 mmol/L, EB of -33,5 mmol/L, and blood glucose of 344 mg/dL. A general urine test also reported the presence of leukocytes, bacteria, and ketones.

Office studies are complemented by a non-contrast abdominal CT scan, which identifies a liver lesion in segment VI/VII of indeterminate appearance (figure 1).

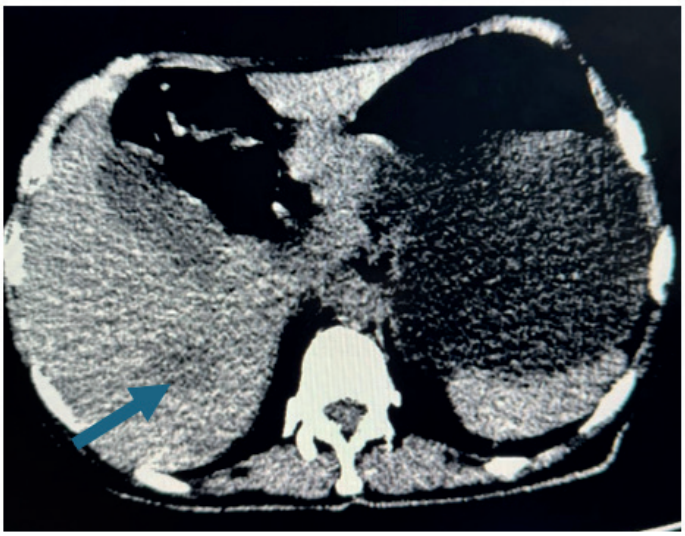

Figure 1. Non-contrast abdominal CT scan showing a liver lesion in segment VI/VII of indeterminate appearance (blue arrow).

In the emergency department, fluid resuscitation is performed and supported with supplemental O2 at 2 L/min. However, her general condition deteriorated further, with profuse diaphoresis, tachycardia, and tachypnea, so the intensive care unit was called to assess her. The patient was found to be in fair to poor general condition, slightly drowsy, hemodynamically stable, tachycardic, and with Kussmaul breathing. Therefore, in the context of severe diabetic ketoacidosis, she is admitted to the intensive care unit, where continuous infusion insulin therapy is initiated, fluid resuscitation with crystalloids, broad-spectrum antibiotic treatment with Imipenem and Vancomycin to cover multi-resistant gram-positive and gram-negative bacteria due to a history of repeated infections, in addition to requesting laboratory tests, sputum culture, urine culture, blood culture, and viral respiratory panel.

He presented an unfavorable course with persistent fever, drowsiness, and hemodynamic hypotension, for which vasopressor therapy (norepinephrine) was initiated. Tachycardia, increased FiO2 requirements, signs of respiratory failure, and poor ventilatory mechanics. Therefore, the airway is protected by orotracheal intubation and connection to a mechanical ventilator. An ultrasound scan is performed at the patient's bedside, showing the inferior vena cava with a distensibility index of 28 % and slight distension of the suprahepatic veins compatible with veXus 1, suggestive of mild or initial congestion.

Over the following days, with her condition remaining stable, she remained under analgesic sedation, orotracheally intubated, and connected to a volume-controlled mechanical ventilator with FiO2 of 100 % and PEEP: 10. She was evaluated by the endocrinology and pulmonology departments, who agreed with the established management. On 05-22-2025, the results of a tracheal secretion culture identified *Klebsiella pneumoniae* with sensitivity to aminoglycosides, cephalosporins, quinolones, and carbapenems. The viral panel for influenza, COVID-19, and respiratory syncytial virus was negative. Blood and urine culture results showed no isolation of germs.

Antibiotic treatment is continued, and the patient shows slow but favorable progress, even with high ventilatory parameters and vasopressor support. Therefore, a new tracheal secretion culture and a contrast-enhanced chest, abdomen, and pelvis CT scan are requested.

On 05/29/2025, a contrast-enhanced chest and abdominal CT scan was performed, revealing multiple abscessed cavities in both lung fields associated with probable septic pulmonary embolism, atelectasis of the posterior and lateral segments of the right lower lobe caused by fluid filling of the segmental bronchi. Hepatic lesion in segment VI/VII of indeterminate appearance, which should be considered to have the exact etiology as the pulmonary lesions, and an image suggestive of hepatic hemangioma in segment VIII (figure 2).

During follow-up by the pulmonology service, a fibrobronchoscopy is planned as soon as the patient's condition allows. The result of the tracheal secretion culture on 06-04-2025 again identifies *Klebsiella pneumoniae,* which is sensitive to cephalosporins, aminoglycosides, quinolones, and carbapenems. Additionally, Acinetobacter baumannii is isolated, which is sensitive to colistin; therefore, it is added to the antibiotic regimen. Over the following days, her condition deteriorates, with episodes of hypotension and an increased need for vasopressor support. No progress is made, and she remains on high ventilatory settings. A neuromuscular relaxant is added to improve ventilation, but there is no improvement in ventilation. Due to the patient's condition and state, it was not possible to perform fiberoptic bronchoscopy. The patient died on 06-06-2025 after 18 days of hospitalization in the intensive care unit.

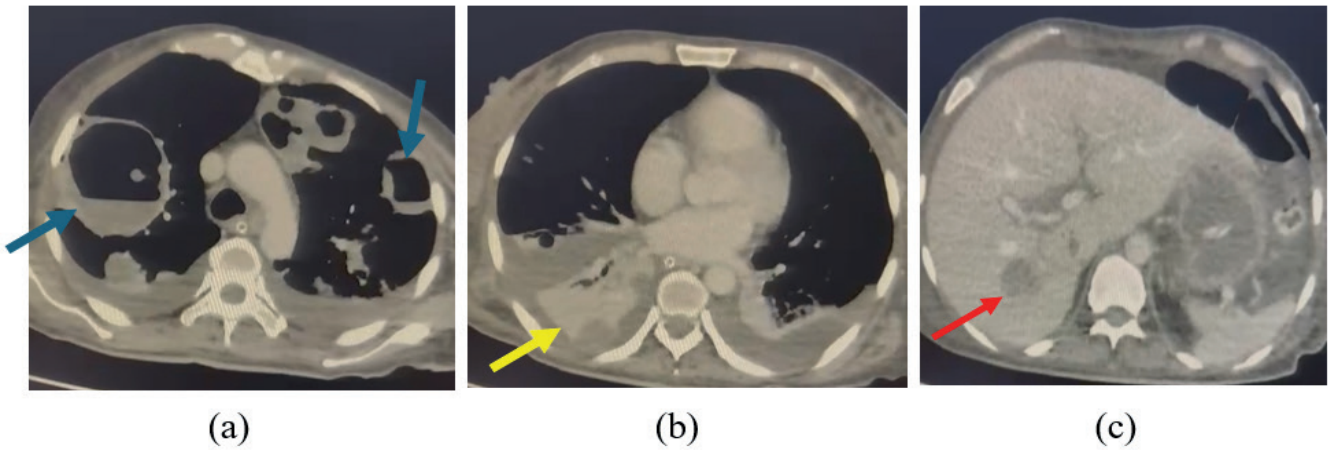

Figure 2. Chest CT scan with contrast showing abscess cavities in both lung fields (blue arrows) associated with probable septic pulmonary embolism (a), atelectasis of the right lower posterior lateral segment (yellow arrow) caused by fluid filling of the segmental bronchi (b). Contrast-enhanced abdominal CT scan showing a liver lesion in segment VI/VII (red arrow) of indeterminate appearance (c).

DISCUSSION

The hvKP strain has become a virulent pathogen capable of causing invasive community-acquired infections even in healthy individuals, characterized by metastatic spread and the formation of pyogenic tissue abscesses. Initially a cause of pyogenic liver abscesses, it can also cause infections in other organs and systems, such as pneumonia, necrotizing fasciitis, endophthalmitis, meningitis, and non-hepatic abscesses.[2]

The interactions of hypervirulent *Klebsiella pneumoniae* with tissue macrophages were explored using an infection model and a porcine organ perfusion model. The evidence suggested that, unlike non-hypervirulent strains, which are effectively eliminated from tissues, hypervirulent strains replicate in hepatic macrophages and resist neutrophil-mediated elimination, leading to abscess formation, as in our case.[8]

The first infections caused by a hypervirulent *Klebsiella pneumonia* virus were recorded between 1980 and 1990 in Southeast Asia. These early reports described a specific clinical picture in which community-acquired pyogenic liver abscesses predominated and were associated with serious complications such as meningitis and endophthalmitis. Subsequently, several reports in Asia described the role of this strain in various infections.[4]

HvKP isolates are usually susceptible to antibiotics, but they are exceptionally virulent, which has been attributed to two types of factors: mucoid regulators and siderophores. These capsules enable the bacteria to evade the host's innate immune response by inhibiting phagocytosis, opsonization, and complement-mediated killing. They also limit the movement of DNA into and out of the bacterial cell, thereby restricting recombination and horizontal gene transfer.[9]

Community-acquired invasive primary liver abscess syndrome caused by *Klebsiella pneumoniae* was first reported in Taiwan in 1986. These patients generally had no history of hepatobiliary disease, as was the case with our patient. Subsequently, in 2012, invasive *Klebsiella pneumoniae* syndrome was defined as a liver abscess and its distant infections caused by *Klebsiella pneumoniae* infection. The lungs, central nervous system, and eyes are the most common metastatic sites, although only one-third of them are diagnosed at admission, as seen in our patient.[1]

Invasive *Klebsiella* pneumonia syndrome is a rare condition characterized as a disseminated, malignant infection, typically diagnosed as meningitis or endophthalmitis secondary to a liver abscess or *Klebsiella* pneumonia, with a reported incidence of 2 % or less, even in Taiwan, which has the highest reported burden.[10]

Diabetes is a significant risk factor for acquiring hvKp infection compared to cKp infection and for developing metastatic complications in patients with liver abscess. Not all studies have consistently found this association, suggesting that the connection between diabetes and hvKp may vary by region or clinical presentation. This is a clinical situation compatible with our patient; however, due to the limitation of being able to identify these strains in our setting.[11]

Nevertheless, considering our patient, diabetes mellitus is the most significant risk factor for the development of liver abscess caused by *Klebsiella pneumoniae*, likely due to neutrophil dysfunction in diabetic patients with poor glycemic control, which reduces the phagocytosis capacity of capsular varieties K1 and K2.[12] Defining hypervirulence based solely on clinical characteristics can be complicated, and our institution does not have access to the test, which has been approved by the FDA for use in microbiology laboratories. However, it cannot distinguish between hvKP strains and cKP strains.[13]

HvkP is associated with significant morbidity and mortality, with patients with invasive syndrome having a

mortality rate ranging from 3 % to 31 %. Treatment of infections requires adequate control of the source of infection and active antibiotic therapy, with a typical treatment duration of two to six weeks, depending on the location and extent of the infection.[11]

Over the years, *Klebsiella pneumoniae*, regardless of cKP or hvKP, has become increasingly resistant to drugs, and resistance to carbapenems is of great concern. This resistance, evident in our patient, is primarily conferred by the carbapenemase gene and the oxacillinase gene, which are predominantly carried by mobile genetic elements. It has now become a significant threat to public health worldwide, as it causes high mortality and medical burden.[14] There have been increasing reports of multidrug resistance associated with the hvKP strain, with virulence genes and lethal hospital outbreaks. Modification of the lipopolysaccharide leads to a reduction in its negative charge, which in turn reduces its affinity for colistin, resulting in drug resistance.[15]

Hypervirulent *Klebsiella pneumoniae* (*hvKP*) is an emerging pathogen with a high potential to cause severe and disseminated infections, especially in patients with predisposing factors such as diabetes mellitus. Its ability to resist phagocytosis and acquire antimicrobial resistance genes makes it a growing clinical and epidemiological challenge.

The case presented illustrates the aggressiveness of this bacterium and the challenges of managing it, even with appropriate antibiotic therapies. It is essential to strengthen microbiological surveillance, promote metabolic control in diabetic patients, and establish protocols for the early detection and comprehensive treatment of hypervirulent *Klebsiella pneumoniae* infections in order to reduce their high morbidity and mortality rates.

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CONFLICT OF INTEREST

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