

# A History of *Klebsiella pneumoniae* Acute Respiratory Distress Syndrome: A Case Report and Review of Literature

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## Abstract

**Background:** *Klebsiella pneumoniae* is a well-known human pathogen, and recently, that is distinctly associated with a unique invasive syndrome. This syndrome is characterized by primary liver abscess and extrahepatic complications resulting from bacteremic dissemination to the lung. We report the infrequent adult case of primary liver abscess caused by *Klebsiella pneumoniae*, with acute respiratory distress syndrome in China.

**Case presentation:** A 23-year-old man with persistent hyperpyrexia and a rapidly progressing of shock. On admission, he had loss of consciousness and became more hypoxemic and dyspneic despite mask oxygen, then he was rapidly placed a tracheal intubation ventilator assists breathing. Laboratory data showed severe inflammation, liver dysfunction, thrombocytopenia, an increased serum creatinine level, and coagulopathy. Ultrasound showed a low density area in the right lobe of the liver. Percutaneous drainage of the liver abscess was performed. The blood and fester cultures showed *Klebsiella pneumoniae* infection. Final diagnosis was septic shock and ARDS caused by *Klebsiella pneumoniae*. About one month of antibiotic therapy the patient's clinical condition improved and discharged.

**Conclusions:** Although th0065 primary liver abscess caused by *K. pneumoniae* and there was no bacterial phenotype or a hypermucoviscous experiment, this patient was be like to invasive liver abscess syndrome and physicians should recognize this syndrome, and complete inspection, appropriate diagnosis and targeted therapy is essential for patients.

## Keywords

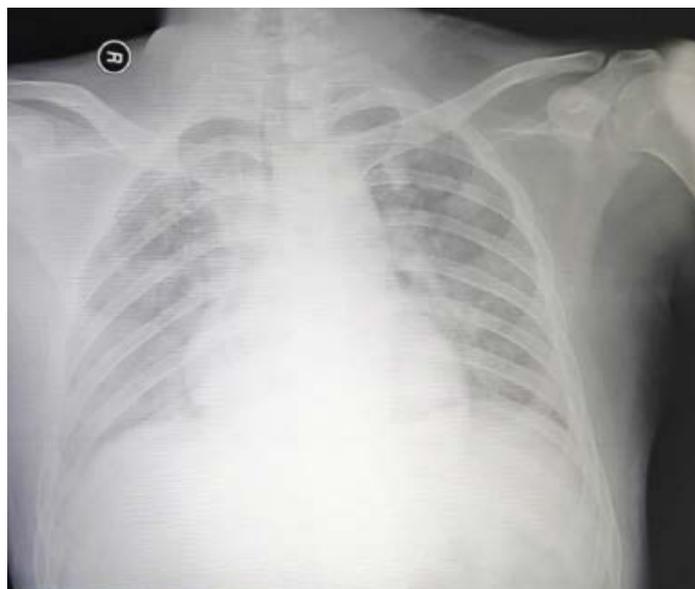
*Klebsiella pneumoniae*, Acute respiratory distress syndrome, Liver abscess

## Introduction

*Klebsiella pneumoniae* (KP) is an increasingly recognized cause of hospital-acquired infections (HAIs) and a unique invasive syndrome consisting of pyogenic liver abscess including liver abscess, pneumonia, meningitis and endophthalmitis [1,2]. Intrathoracic complications of invasive *Klebsiella pneumoniae* liver abscess syndrome (IKPLAS) have been characterized sporadically but have not been the subject of an all-encompassing investigation. Until now, there is few reports of the acute respiratory distress syndrome (ARDS) as a consequence of IKPLAS. Herein we report the newly appeared a case of acute respiratory distress syndrome in China. We present a case of a 23-year-old man in our hospital, and he presented fever on admission, microbiological investigations were performed and empiric antibiotic therapy with cefotaxime was started. Blood and fester cultures were positive for KP. Abdominal ultrasonography showed presence of liver abscess that was located on the right lobe of the liver. After a few days of antibiotic therapy, the patient's clinical condition improved. Correct microbiology identification of this kind of strain was essential for appropriate clinical management.



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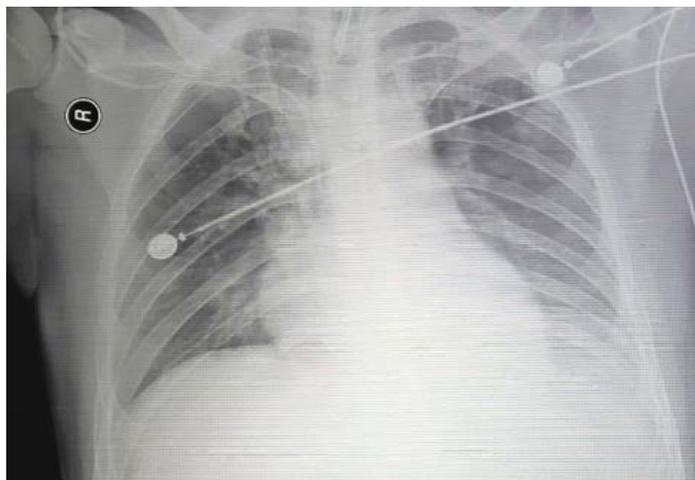


**Figure 1:** Chest radiograph showing bilateral pneumonia (23 October 2015).

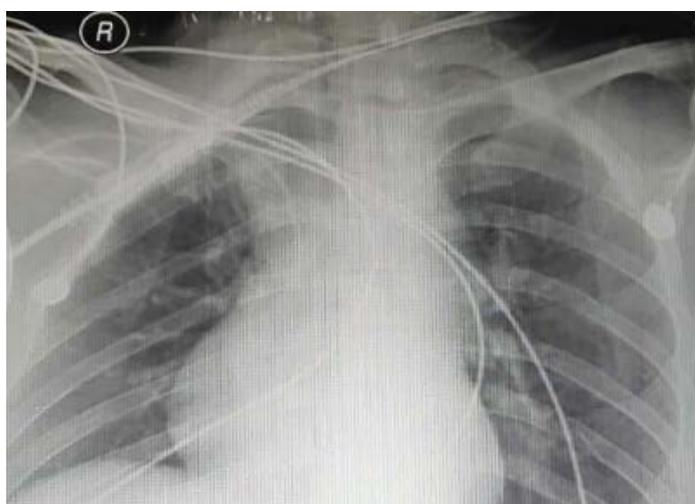
## Case Report

About 5 days ago, a 23-year-old man appeared dizziness and fever, and the maximum body temperature was 39 °C. The anti-inflammation treatment in the local hospital is not effective, and he had persistent hyperpyrexia and a rapidly progressing of shock. For further treatment, he was transferred to emergency intensive care unit in our hospital. He had a history of hepatitis B for 10 years and developed mouth and weight loss for one month. On the day of admission, the blood tests revealed a white blood cell count of  $22 \times 10^9/L$  (94.33% neutrophils), newly emerged severe thrombocytopenia (platelet of  $4 \times 10^9/L$ ), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) values of 1166 and 709 U/L respectively, gamma-glutamyl transpeptidase (GGT) of 42 U/L, alkaline phosphatase (ALP) level of 208 U/L and total bilirubin of 12.8  $\mu\text{mol/L}$ , moreover he presented a concentration of ultrasensitive C-reactive protein of 264.7 mg/L, while the anti-HCV was negative. He became more hypoxemic and dyspneic despite mask oxygen and was placed a tracheal intubation ventilator assists breathing. His initial vital signs were as follows: Blood pressure 121/74 mmHg, pulse 125 beats/min, respiratory rate 30 breaths/min, oxygen saturation 100% and a temperature of 36.5 °C (drug sedation). Physical examination revealed a tracheal intubation ventilator assists breathing, normal cardiac auscultation, the lower left lung is scattered in the moist rales and a soft, non-tender abdomen. Initial laboratory evaluation revealed a blood pH of 6.7 with a serum bicarbonate of 4.6 mmol/L (reference range 22-29). The serum glucose level was 11.65 mmol/L, and the urinalysis was positive for ketones (3+) and glucose (1+). Two consecutive days of Blood cultures were collected. Chest radiograph showed bilateral pneumonia ([Figure 1](#)). Abdominal ultrasonography performed revealed a complex avascular hepatic mass-like lesion with both cystic and solid components measuring 6.9 cm and behind it existed gas reflection. A diagnosis of liver abscess, septic shock and acute respiratory distress syndrome, and type 2 diabetes mellitus. In the Emergency Department (ED), the patient received 6 liters of intravenous crystalloid, and an insulin infusion was started. Empirical treatment with triple antibiotics were conducted, including biapenem, teicoplanin and ornidazole. On day three of admission, his temperature rose to as high as 39.5 °C. Sputum culture was collected. KP was isolated from the blood and only resistant against ampicillin. At this time, chest radiograph showed lower right lung infection and left pleural effusion ([Figure 2](#) and [Figure 3](#)). On day four of admission, percutaneous puncture drainage guided by B ultrasound was carried out for bedside placement of a drainage catheter and abscess specimens was sent for culture. On day six of admission, pan-resistance *acinetobacter baumannii* was detected in sputum culture. The fester cultures were identified as only ampicillin





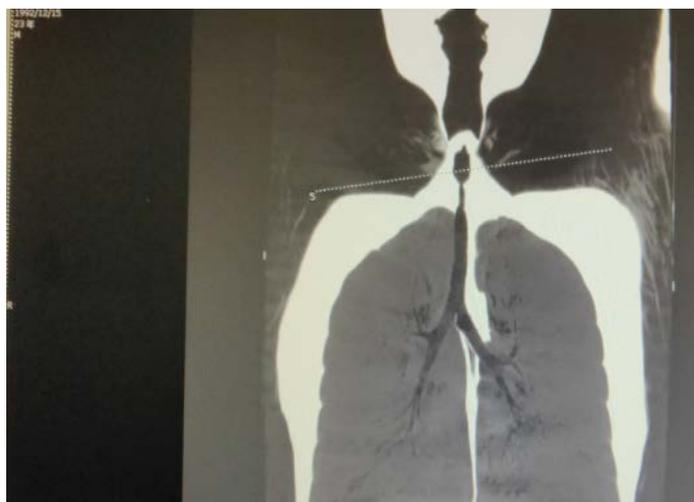
**Figure 2:** Chest radiograph showing lower right lung infection and left pleural effusion at the time of *Klebsiella pneumoniae* (KP) bacteremia (26 October 2015).



**Figure 3:** Chest radiograph showing lower right lung infection and left pleural effusion (5 November 2015).

resistant KP. In the ICU, the patient was performed five times CRRT for bedside and multiple inputs concentrate red blood cells, plasma and platelets. On day nine of admission, the ventilator went off successfully because of his good cough response, sputamentum reduction,  $SPO_2$  100%, breathe smoothly and PH 7.45. The 11<sup>th</sup> day of admission, KP only sensitive to trimethoprim/sulfamethoxazole and tigecycline was detected. On day twelve of admission, antibiotics changed to tigecycline and ornidazole. In hospital for 14<sup>th</sup> day, the percutaneous puncture and drainage was repeated application. Then fever disappeared and blood test analysis showed an outstanding decrease of inflammation markers, specifically we found a white blood cell count  $8.38 \times 10^9/L$  (80.8% neutrophils, PLT  $176 \times 10^9/L$ ), AST and ALT values of 17 and 21 U/L respectively, GGT of 158 U/L, ALP level of 115 U/L and a concentration of ultrasensitive C-reactive protein of 7.9 mg/L, total bilirubin was within the normal range. On 15<sup>th</sup> day, the pulmonary infection was completely absorbed on the chest X-ray and the patient was transferred in hepatic surgery wards. Continue antibiotic treatment with cefoperidol sodium sulbactam sodium and left ornidazole about 3 weeks. Blood and drainage cultures were negative. Review of blood routine and blood biochemical tests were in the normal range. The drainage fluid significantly decreased, then the puncture drainage catheter was pulled out and the patient was discharged with a better health condition. About two months later, the patient developed difficulty breathing again and admitted to liver surgery wards. Abdominal ultrasonography showed complete absorption of hepatic abscess and no lung infection was found on the chest CT. Lung function tests indicate obstructive ventilation and spiral CT 3D





**Figure 4:** CT chest scan and 3D reconstruction showing the 7<sup>th</sup> cervical horizontal trachea is slightly narrower (18 January 2016).



**Figure 5:** Chest radiograph showing tracheostomy and enhanced lung markings (22 January 2016).

reconstruction of the trachea revealed tracheal stenosis (Figure 4). Then, the patient was transferred to the thoracic surgery department for tracheal stenosis resection and reconstruction (Figure 5). The patient was completely cured and discharged after at 10 days after surgery.

## Discussion

In China, pyogenic liver abscesses are predominantly caused by KP (77.1% of all cases) and 45.2% of KP clinical isolates associated with PLA [3,4]. A distinctive, invasive community-acquired KP syndrome with systemic abscess formation, and high fatality has been increasingly reported in the recent decades. Majority of these patients present with a clinical syndrome of a pyogenic liver abscess, with fever, leukocytosis, and upper quadrant abdominal pain.

Patients with diabetes mellitus seem to be particularly vulnerable to cryptogenic invasive KP liver abscess syndrome [5]. Diabetes mellitus, particularly with poor glycemic control, has been identified as one of the strongest risk factors for KP strains causing liver abscess. KP is frequently associated with infections in patients with impaired host defenses like diabetes mellitus, as well as our patient had the same risk.

To the best of our knowledge, there are few reports known to us have heretofore reported IKPLAS to the development of ARDS. Gupta A had reported that Invasive *Klebsiella pneumoniae* liver abscess syndrome as a cause of acute respiratory distress syndrome [6]. IKPLAS is increasingly being recognized across the globe as a



potentially morbid infection that carries a mortality rate of 4-11%, might be capable of seeding the lungs and resulting in ARDS, but one that can be effectively treated with prompt drainage and appropriate antibiotics [6,7]. The extent of pulmonary involvement can be sufficient to cause respiratory failure in otherwise normal hosts. Correct microbiology identification of invasive *Klebsiella pneumoniae* was essential for appropriate clinical management [8]. The Chen's report pointed out that only the results of liver abscess pus culture were taken into account if both blood and pus cultures were positive [9]. IKPLAS is defined by *Klebsiella pneumoniae*, which is isolated from the abscess aspirate or blood of a patient with imaging findings consistent with a liver abscess in the absence of underlying hepatobiliary disease [10]. Some risk factors have been indicated severe complications of KP liver abscess, including thrombocytopenia ( $< 100,000/\text{mm}^3$ ), alkaline phosphatase  $> 300$  U/L, gas formation in the abscess, the initial platelet count in our patient was  $4000/\text{mm}^3$  with alkaline phosphatase of 208 U/L. Hypervirulent *Klebsiella pneumoniae* (hvKp) strains are often susceptible to most of the antibiotics including ciprofloxacin, more than three generation cephalosporins and carbapenems. Infections cause of hvKp are increasingly recognized worldwide and the proportion of PLA caused by hvKp showed a varied geographic distribution. It can lead to organ and life-threatening disease in healthy individuals from the community merits concern, which has been magnified by increasing descriptions of multiply drug-resistant (MDR) and extensively drug-resistant (XDR) strains. The limitation for this report is that has not done microbiological experiment in serum types and virulence genes of *Klebsiella pneumoniae*. We did not test the serotype of *K. pneumoniae* because of the lack of adequate equipment. In future work, physicians should detection of bacterial typing and virulence against KP and recognize invasive *Klebsiella pneumoniae* liver abscess syndrome, furthermore, appropriate diagnosis and targeted therapy is essential for saving patients' lives and preserving organ function.

## Acknowledgments

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## Conflicts of Interest

The authors declare that they have no competing interest.

## Ethical Approval

Ethical approval for the current study was obtained by the local institutional review board (Ethics Committee of the Wuhan of Hubei, China).

## References

1. Kuo SH, Lee YT, Li CR, et al. Mortality in emergency department sepsis score as a prognostic indicator in patients with pyogenic liver abscess. *Am J Emerg Med* 2013;31:916-21.
2. Magill SS, Edwards JR, Bamberg W, et al. Multistate point-prevalence survey of health care-associated infections. *N Engl J Med* 2014;370:1198-208.
3. Wang J, Yan Y, Xue X, Wang K, Shen D. Comparison of pyogenic liver abscesses caused by hypermucoviscous *Klebsiella pneumoniae* and non-*Klebsiella pneumoniae* pathogens in Beijing: a retrospective analysis. *J Int Med Res* 2013;41:1088-97.
4. Guo Y, Wang S, Zhan L, et al. Microbiological and Clinical Characteristics of Hypermucoviscous *Klebsiella pneumoniae* Isolates Associated with Invasive Infections in China. *Front Cell Infect Microbiol* 2017;7:24.
5. Siu LK, Yeh KM, Lin JC, Fung CP, Chang FY. *Klebsiella pneumoniae* liver abscess: a new invasive syndrome. *Lancet Infect Dis* 2012;12:881-7.
6. Fazili T, Sharngoe C, Endy T, Kiska D, Javaid W, Polhemus M. *Klebsiella pneumoniae* liver abscess: An emerging disease. *Am J Med Sci* 2016;351:297-304.
7. Gupta A, Bhatti S, Leytin A, Epelbaum O. Novel complication of an emerging disease: Invasive *Klebsiella pneumoniae* liver abscess syndrome as a cause of acute respiratory distress syndrome. *Clin Pract* 2017;8:1021.



8. Scapaticci M, Biscaro M, Burelli F, Cadamuro L, Biscaro R, Bartolini A. A case of invasive infection caused by a highly virulent strain of *Klebsiella pneumoniae* displaying hypermucoviscosity in a patient with hepatic involvement without liver abscess. *Infez Med* 2017;125:362-5.
9. Chen CH, Wu SS, Chang HC, Chang YJ. Initial presentations and final outcomes of primary pyogenic liver abscess: a cross-sectional study. *BMC Gastroenterol* 2014;14:133.
10. Shin SU, Park CM, Lee Y, Kim EC, Kim SJ, Goo JM. Clinical and radiological features of invasive *Klebsiella pneumoniae* liver abscess syndrome. *Acta Radiol* 2013;54:557-63.

