Case Report

Invasive *Klebsiella pneumoniae* liver abscess syndrome presenting with septic pulmonary emboli, hepatic vein thrombosis, ascites, and endogenous endophthalmitis: A case report

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ABSTRACT

Invasive liver abscess syndrome (ILAS) caused by hypervirulent *Klebsiella pneumoniae* is characterized by a pyogenic liver abscess with hematogenous metastatic spread. Although commonly reported in East Asia, cases from India remain relatively uncommon. Diabetes mellitus is the most important predisposing factor. We report a case of a 44-year-old diabetic male who presented with community-acquired pneumonia complicated by ascites and septic shock. Persistent fever and worsening pulmonary infiltrates prompted further evaluation, which revealed a segment VI liver abscess, right hepatic vein thrombosis, and multiple septic pulmonary emboli. The patient also developed right-sided endogenous endophthalmitis due to metastatic spread. Blood and liver pus cultures grew *Klebsiella pneumoniae*. Ascitic fluid had high SAAG and high protein, secondary to hepatic vein thrombosis. Infective endocarditis and central nervous system involvement were excluded. The patient was managed with culture-directed meropenem therapy, pigtail drainage of the liver abscess, and intravitreal antibiotics, resulting in resolution of sepsis, although visual recovery remained limited. This case highlights that ILAS, which initially presented as community-acquired pneumonia, was subsequently identified as metastatic infection originating from a primary liver abscess. Invasive *Klebsiella pneumoniae* liver abscess is an emerging global threat that is being increasingly reported from India.

Key words: Diabetes mellitus, Endogenous endophthalmitis, Hepatic abscess, Hypervirulent *Klebsiella pneumoniae*, Invasive liver abscess syndrome, Septic pulmonary emboli

Klebsiella pneumoniae is a Gram-negative, encapsulated bacillus belonging to the family Enterobacteriaceae. Although *K. pneumoniae* has been traditionally associated with pneumonia, urinary tract infections, and nosocomial bacteremia in immunocompromised hosts, hypervirulent *K. pneumoniae* (hvKP) strains have emerged over the past three decades as the predominant pathogen responsible for community-acquired pyogenic liver abscesses, particularly in East and Southeast Asia [1]. The invasive liver abscess syndrome (ILAS) is commonly described as a monomicrobial *K. pneumoniae* liver abscess occurring without predisposing hepatobiliary abnormalities or other intra-abdominal infections [1]. The risk of infection is significantly increased in individuals with impaired host defenses, such as diabetes mellitus, alcoholism, malignancy, chronic liver disease, chronic obstructive pulmonary disease, and immunosuppressive therapy, including glucocorticoid use. Diabetes mellitus is consistently recognized as the most significant predisposing condition due to impaired neutrophil chemotaxis and phagocytic activity, facilitating bacterial invasion and dissemination [2]. hvKP strains possess unique virulence determinants such as aerobactin, rmp1, and rmp2 which confer resistance to phagocytosis, serum killing, and hematogenous dissemination [3]. hvKP ILAS is characterized by primary liver abscess formation accompanied by bacteremia and metastatic spread to distant organs. Metastatic complications occur in approximately 10–16% of affected patients and represent the hallmark of invasive disease [2]. The most frequently involved sites include the eyes, lungs, central nervous system (CNS), soft tissues, prostate, and bones [2]. Early recognition is clinically important because delayed diagnosis may result in rapid progression to septic shock, permanent visual loss, neurological deficits, or death. Management typically consists of prolonged intravenous antibiotic therapy and prompt drainage of the liver abscess and multidisciplinary evaluation for metastatic involvement.

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We report a case of a 44-year-old male with *Klebsiella* ILAS with septic pulmonary embolism and endogenous endophthalmitis presenting as community-acquired pneumonia. This case highlights the aggressive metastatic potential of hvKP infection and emphasizes the importance of early recognition, comprehensive systemic evaluation, and timely multidisciplinary management to reduce morbidity and improve clinical outcomes.

CASE PRESENTATION

A 44-year-old man from Dolibari, Kamrup, Assam, Northeast India, presented with complaints of low-grade fever and non-productive cough for 7 days. He had a history of vomiting, occurring 3–4 times daily for a similar duration. The patient was a known diabetic, non-compliant, on therapy. There was no history of diarrhea, abdominal pain, dysuria, or altered sensorium. There was no history of dyspnea, chest pain, palpitations, syncope, presyncope, or hemoptysis.

On examination, the patient had pallor and cold peripheries. He was hypotensive and tachycardic. Systemic examination of the respiratory system revealed coarse crepitations over the right lower lobe. His abdomen was soft, distended, and non-tender with positive shifting dullness. The remainder of the examination was unremarkable.

The patient on admission was hypotensive and hyperglycemic. Chest radiograph revealed a heterogeneous opacity in the left lower lobe. Initial investigations revealed leukocytosis, deranged renal function, and elevated lactate (Table 1). Blood cultures were sent, and the patient was started on broad-spectrum antibiotics (injection piperacillin-tazobactam and tablet azithromycin), intravenous fluids, and other supportive measures. The patient was admitted to the intensive care unit and provisionally managed as community-acquired pneumonia in sepsis with septic shock. In view of persistent fever and new-onset crepitations in bilateral chest fields, a repeat chest X-ray was done on the 3rd day of admission, which revealed multiple heterogeneous lesions throughout both lung fields. The patient further deteriorated, requiring vasopressors, and empirical Gram-positive antimicrobial coverage was added (injection vancomycin).

Abdominal ultrasonography revealed a hepatic abscess in segments VI and VII of the liver with moderate ascites (Table 2). With hepatic abscess and worsening of chest radiograph, the possibility of a hepatic abscess with secondary septic pulmonary emboli was considered.

Table 1: Hematological and biochemical parameters of the patient

Parameter	14/04/2025	19/04/2025	24/04/2025	05/05	Reference values
Hb g/dL	8.6	8.2	8.4	8.3	12–15
Total WBC count ($\times10^9$/L)	15.9	15.6	13.5	7.48	4–10
Platelet ($\times10^9$/L)	120	243	206	230	150–410
Serum Sodium mmol/L	143	144	136	137	137–145
Serum potassium mmol/L	4.0	3.5	3.9	4.2	3.5–5.1
Procalcitonin ng/mL	60.7	15.76	3.35	0.739	<0.02
CRP mg/dL	273	81.6	67	22	<10
Urea mg/dL	110	82	34	32	15–36
Creatinine mg/dL	1.4	1.0	0.8	0.8	0.5–1.04
AST U/L	231	352	55	28	14–36
ALT U/L	357	152	50	12	<35
ALP U/L	221	179	214	154	38–126
GGT U/L	300	58	60	38	12–43
Albumin g/dL	2.9	2.4	2.6	3.0	3.5–5
D dimer mg/L FEU	-	7.38	-	-	<0.5
ESR mm/hr	-	82	-	-	<20
Ferritin ng/mL	1000	-	-	-	3–300
Transferrin saturation %	11	-	-	-	20–40
Vitamin B12 pg/mL	>1000	-	-	-	239–950
Folate ng/mL	17.5	-	-	-	4.74–20.2
Urine ACR	504	-	-	-	30–300
HbA1c	-	12.2	-	-	<5.7
Ascitic fluid total WBC count/µL	160 cells	-	-	-	<250
Ascitic fluid protein g/dL	2.6	-	-	-	<2.5
Ascitic fluid Albumin g/dL	0.9	-	-	-	-
Ascitic fluid ADA U/L	7.48	-	-	-	<40
Ascitic fluid cytology	Negative for malignancy	-	-	-	-

Hb: Hemoglobin, WBC: White blood cell, CRP: C-reactive protein, AST: Aspartate transaminase, ALT: Alanine transaminase, ALP: Alkaline phosphatase, GGT: Gamma glutamyl transferase, ESR: Erythrocyte sedimentation rate, ACR: Albumin creatinine ratio, HbA1c: Glycosylated hemoglobin, ADA: Adenosine deaminase

Table 2: Radiological parameters of the patient

Modality	Report
2D ECHO	Normal chamber dimensions, Normal valve function, No Regional Wall Motion Abnormality, left ventricular ejection fraction 60%, Grade I left ventricular diastolic dysfunction, no clot, vegetations, or pericardial effusion, pulmonary artery normal in caliber, intra-atrial septum drop out +
Ultrasound abdomen	Ill-defined heterogeneous lesion measuring 6 cm×5.5 cm×4.7 cm (~80 mL) with multiple air foci is noted in segment VI and VII of right lobe of liver - ?liver abscess with post-interventional changes; moderate ascites, bilateral renal parenchymal disease.
CECT thorax and abdomen	• Multiple peripherally enhancing hypodense nodular lesions with some of them having internal air foci noted in bilateral upper and lower lobes - suggestive of septic pulmonary emboli (likely hematogenous spread from liver abscess). • Small bilateral loculated pleural effusions noted with adjacent subsegmental atelectatic changes. • A hepatic abscess with multiple air pockets and an air-fluid level is seen in segment 6 of the right lobe of the liver with diffuse adjacent parenchymal edema. Hypodense, non-enhancing thrombosis of a tributary of the right hepatic vein is noted. • Diffuse atrophy of the entire pancreas is noted with dilatation of the main pancreatic duct (measuring about 6.5 mm) – suggestive of chronic pancreatitis. • Few non-obstructing calculi noted in both kidneys. • Moderate ascites noted.
Review ultrasound–post pigtail insertion	Residual liver abscess measuring 3.2 cm×2.9 cm×2.5 cm is noted in segment VI and VII of the right lobe with post-interventional changes, moderate ascites.
MRI brain	No significant abnormality detected.

2D ECHO: 2 Dimensional echocardiography, CECT: Contrast-enhanced computed tomography, MRI: Magnetic resonance imaging

Contrast-enhanced computed tomography (CECT) of the thorax was done, which confirmed the septic pulmonary emboli with hematogenous spread from liver abscess and thrombosis of the right hepatic vein (Fig. 1 and Table 2).

During the hospital stay, the patient had new-onset diminution of vision in his right eye. Ophthalmological examination revealed 3+ vitreous flare, yellowish white fluffy exudates, pale disc, and hazy view due to vitreous exudates, suggestive of endogenous endophthalmitis in the right eye. Vitreous humor was aspirated and sent for culture, and the patient was started on intravitreal antibiotics.

Initial blood cultures grew *Klebsiella pneumoniae*, and antibiotic therapy was escalated as per sensitivity (injection meropenem) while Gram-positive coverage was stopped (Table 3). Upon upgrading antibiotics, the patient became afebrile, vasopressors were weaned off, and inflammatory markers gradually normalized. The pus from the liver abscess was aspirated and sent for culture and sensitivity, which also grew *K. pneumoniae*, confirming the liver abscess to be the primary source of emboli. Ascitic fluid analysis revealed high SAAG and high protein ascites, and after excluding a cardiac etiology, it was presumed to be secondary to the right hepatic vein thrombosis.

Transthoracic echocardiography was done to rule out infectious endocarditis as a possible source for the septic emboli. Magnetic resonance imaging (MRI) of the brain was done to rule out a brain abscess (Table 2). Pigtail catheter drainage of the abscess was done, and the patient received a 28-day course of injection meropenem and three doses of intravitreal antibiotics along with intravitreal dexamethasone.

The patient improved symptomatically; however, right eye visual acuity remained at counting fingers at 3 m. He was discharged on oral fluoroquinolones for 2 more weeks and further followed up in the Medicine and Ophthalmology outpatient department.

DISCUSSION

hvKP causing ILAS has been recognized in East Asia for more than four decades and is being increasingly reported from India, although reports from northeastern India remain scarce. Moreover, ILAS presenting with septic pulmonary embolism and hepatic vascular thrombosis has been rarely described in the Indian literature (Table 4) [4,5].

The hypervirulent K1/K2 capsular serotypes possess thick, hypermucoviscous capsules that resist phagocytosis and complement-mediated killing. They also have virulence genes such as *rmpA* and *rmpA2* upregulate capsule production, while magA, siderophores, and fimbrial adhesins facilitate tissue invasion, allowing the organism to breach the gut barrier, which is presumed to be the portal of entry, followed by seeding of the liver via the portal circulation, leading to suppurative necrosis with destruction of hepatic parenchyma and dense neutrophilic infiltration and liquefaction necrosis [6]. Subsequent intravascular invasion occurs, causing hepatic venous or portal venous thrombophlebitis. The inflamed and thrombosed vessels serve as a nidus for septic embolization, carrying bacterial aggregates to distant organs.

Metastatic infection is the hallmark of invasive *Klebsiella* liver abscess syndrome, usually in the form of endophthalmitis, meningitis, and brain abscess [4]. Other manifestations include lumbar or cervical spondylitis and discitis, septic pulmonary emboli, lung abscess, psoas abscess, splenic abscess, necrotizing fasciitis, neck abscess, and osteomyelitis [4,7]. Endophthalmitis is the most feared complication, occurring in up to 10–15% of cases. Patients

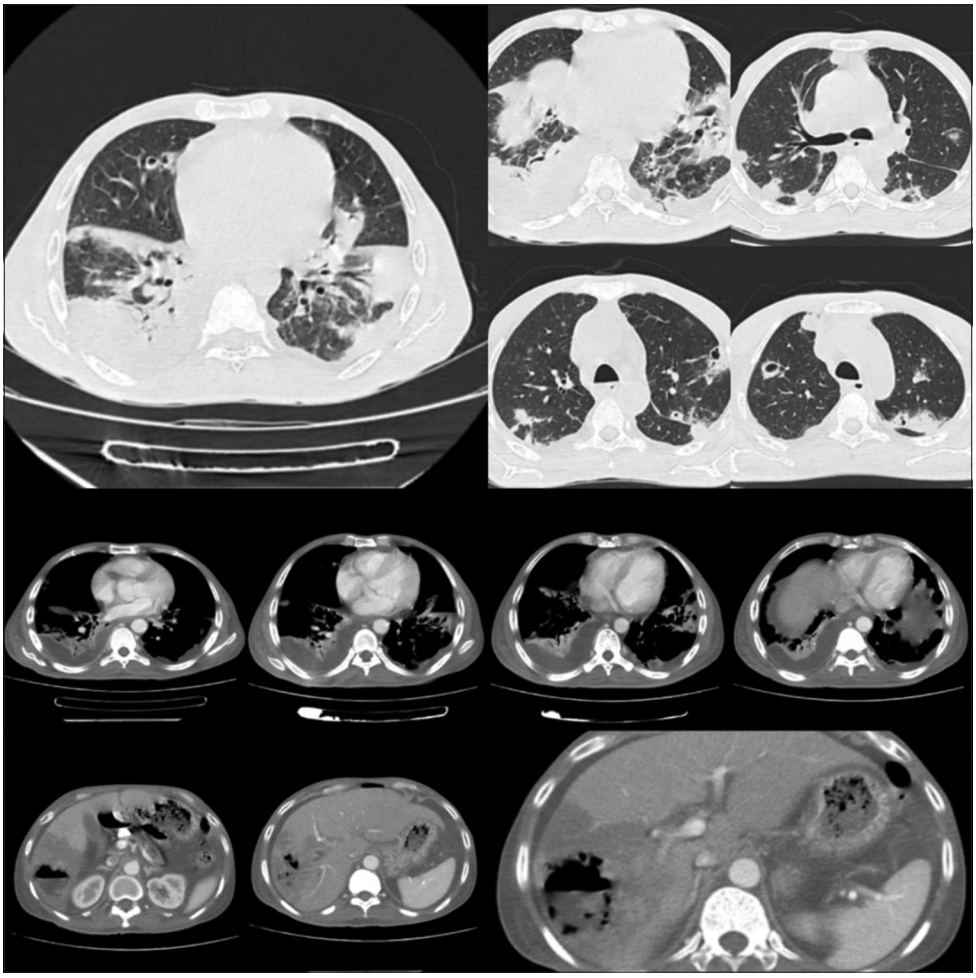

Figure 1: Contrast-enhanced computed tomography of the thorax and abdomen demonstrating multiple air foci in bilateral lungs with an abscess in the right lobe of liver

Table 3: Microbiological parameters of the patient

Test	Result
HbsAg	Non-reactive
Anti HCV	Non-reactive
HIV	Non-reactive
Blood culture April 13, 2025	*Klebsiella pneumoniae* grown after aerobic incubation (both bottles)
Pus culture April 20, 2025	*K. pneumoniae* grown after 48 h of incubation
Vitreous humor culture May 03, 2025	No growth after 48 h of incubation
Blood culture May 04, 2025	Sterile after 5 days of incubation (2 bottles)
Scrub typhus IgM/ Leptospirosis IgM/Typhidot IgM/Dengue NS1	Negative
Ascitic fluid microbiology	No growth in culture No acid–fast bacilli seen, MTB not detected

HbsAg: Hepatitis B surface antigen, HCV: Hepatitis C virus, HIV: Human immunodeficiency virus

typically present with acute diminution of vision, ocular pain, conjunctival injection, hypopyon, and vitritis. Despite prompt systemic antibiotics, intravitreal antimicrobial therapy, and vitrectomy when indicated, visual outcomes remain poor, with many patients progressing to severe permanent visual impairment or blindness because of rapid intraocular tissue destruction [7,8]. Because many of these complications develop early and may initially progress silently, a high index of suspicion and targeted evaluation, especially ophthalmologic assessment, CNS imaging when indicated, and careful respiratory evaluation, are essential in all suspected cases of hypervirulent *Klebsiella* infection [9].

The patient was initially admitted with suspicion of left lower lobar community-acquired pneumonia in septic shock. He was initially managed for septic shock with IV fluids and empiric antibiotics, but his fever and inflammatory markers remained elevated persistently despite aggressive management. The turning point in this case was the worsening radiographic pattern with multiple heterogeneous pulmonary lesions, which was initially attributed to bronchopneumonia. The next pivotal shift happened when evaluation for ascites incidentally revealed a right lobe hepatic abscess.

Follow-up CECT thorax confirmed septic pulmonary emboli, with thrombosis of the right hepatic vein. The hepatic vein thrombosis explained the high-SAAG, high-protein ascites of the patient, consistent with partial Budd-Chiari physiology. The development of blurred vision during hospitalization prompted evaluation for metastatic spread beyond the lungs, and ophthalmologic evaluation confirmed endogenous endophthalmitis. Blood and pus aspirate cultures grew *K. pneumoniae*,

Table 4: Comparison of characteristics of cases of invasive liver abscess syndrome reported from India

Author, Year	Presentation	Major findings	Management
Nayak *et al*., 2022 [5]	Middle-aged diabetic male with septic shock and emphysematous *K. pneumoniae* liver abscess	Bacteremia, brain abscess, meningitis	Drainage+prolonged IV antibiotics
Abi *et al*., 2018 [11]	Adult male with fever and cryptogenic *K. pneumoniae* liver abscess	No metastatic complications	Percutaneous drainage+IV antibiotics
Hamide *et al.,* 2022 [12]	Middle-aged diabetic male with fever, abdominal pain and *K. pneumoniae* liver abscess	Native-valve infective endocarditis	Percutaneous drainage+IV antibiotics
Roy *et al*., 2025 [13]	53-year-old female with fever, right upper quadrant pain, emphysematous *K. pneumoniae* liver abscess with gas under the diaphragm	Mimicked hollow viscus perforation	Pigtail drainage+IV antibiotics
Nagendra *et al.,* 2025 [14]	63-year-old female with shoulder pain with *K. pneumoniae* liver abscess	NSTEMI, Takotsubo cardiomyopathy	Percutaneous drainage+meropenem
Present case	44-year-old diabetic male with community-acquired pneumonia with septic shock and *K. pneumoniae* liver abscess	Septic pulmonary emboli, right hepatic vein thrombosis, endogenous endophthalmitis, ascites	Meropenem, pigtail drainage, intravitreal antibiotics

corroborating the final diagnosis and identifying the liver as the primary focus. Additional investigations to evaluate metastatic spread, including 2-dimensional echocardiography and MRI brain, were negative.

The differential diagnoses included melioidosis, staphylococcal septic emboli secondary to infective endocarditis, pulmonary tuberculosis, disseminated fungal infection, and pyogenic liver abscess caused by organisms other than *K. pneumoniae*. The identification of a liver abscess, septic pulmonary emboli, hepatic vein thrombosis, endogenous endophthalmitis, and concordant blood and pus cultures growing *K. pneumoniae* established the diagnosis of invasive *Klebsiella* liver abscess syndrome.

Although *Klebsiella* causing ILAS is usually sensitive to cephalosporins, the causative organism here was resistant to cephalosporins and broad-spectrum penicillins, requiring carbapenems [10]. Management required timely escalation to carbapenem therapy, targeted intravitreal antibiotics, vasopressor support, and percutaneous abscess drainage. With aggressive supportive care and source control, the patient showed clinical and biochemical recovery.

A review of previously reported Indian cases confirms that diabetes mellitus continues to be the most consistently identified predisposing factor for ILAS. The patients presented with fever and symptoms suggestive of hepatic abscess. The metastatic complications have been heterogeneous, encompassing CNS involvement, infective endocarditis, and stress cardiomyopathy (Table 4) [5,11-14]. In contrast, our patient initially presented with community-acquired pneumonia and septic shock, which delayed recognition of the primary hepatic source. Equally noteworthy is the constellation of metastatic complications observed in this patient, which has been infrequently described in the Indian literature and further expands the recognized clinical spectrum of ILAS.

CONCLUSION

This case highlights the emerging recognition of invasive hypervirulent *Klebsiella* liver abscess syndrome in India, especially among diabetics. What initially appeared to be community-acquired pneumonia was subsequently recognized as a multisystem disseminated infection with septic pulmonary emboli, hepatic vein thrombosis with ascites, and endogenous endophthalmitis, reflecting the aggressive metastatic nature of ILAS. Early imaging, culture correlation, and recognition of atypical progression were crucial in establishing the diagnosis. Timely carbapenem therapy, source control with drainage, and intravitreal antibiotics enabled successful clinical recovery. Physicians must maintain a high index of suspicion for hypervirulent *Klebsiella* in septic patients with unusual pulmonary or ocular findings to prevent irreversible morbidity.

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