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Breaching the Diaphragm: A Thoracic Emergency from a Pyogenic Liver Abscess

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Abstract

Aim & Background: Klebsiella pneumoniae is a virulent Gram-negative bacillus. It poses significant toxicological problems in immunocompromised hosts, particularly those with uncontrolled diabetes mellitus.

Case Description: We report a case of a 44-year-old male admitted to Ananthapuri Hospital, Kerala, in July 2025. The patient presented with fever, abdominal swelling, and neck pain. It progressed to a pyogenic liver abscess (PLA) with complicated by pleural empyema(hepatopleural fistula) due to K. pneumoniae. Initial findings included hyperglycemia (random blood sugar 375 mg/dL), thrombocytopenia (68,000/cu.mm), and elevated C-reactive protein (150 mg/L). Imaging revealed a 5 × 4 cm liver abscess in segment VII and right pleural effusion. Pleural fluid and urine cultures confirmed K. pneumoniae ($\geq 10^7$ CFU/mL). Treatment involved ceftazidime-avibactam, insulin infusion, intercostal drainage, and right thoracotomy with decortication. Antibiotic de-escalation to piperacillin-tazobactam and oral cefuroxime, alongside glycemic control, led to resolution by discharge.

Conclusion: This case highlights the toxicological role of K. pneumoniae's capsular polysaccharides and endotoxins in exacerbating infections in diabetic patients

Clinical significance: This emphasizes the importance of early diagnosis, surgical intervention, and antibiotic stewardship to mitigate resistance and toxicity risks in the Indian context.

Keywords: Klebsiella pneumoniae, pyogenic liver abscess, empyema, diabetes mellitus

Introduction

In India Klebsiella pneumoniae has emerged as a dominant cause of community-acquired pyogenic liver abscesses, particularly among individuals with diabetes mellitus[1]. *Klebsiella pneumoniae*'s hypervirulent strains (hvKP) produce capsular polysaccharides (K1/K2 serotypes) and lipopolysaccharides (LPS). This acts as endotoxins which evade phagocytosis and trigger systemic

inflammation through cytokine release (IL-1β, TNF-α)[2]. Thus contributing to high morbidity among infected individuals. In uncontrolled diabetes, the hyperglycemic state impairs the neutrophil function and it promotes advanced glycation end-products (AGEs). This can amplify bacterial virulence and tissue necrosis. The recent research on this has indicated that lactate which is elevated in diabetic states, enhances *K. pneumoniae* capsule biosynthesis via the phosphotransferase system (PTS)-CRP axis which increases invasiveness [3].

Pyogenic liver abscesses can rupture into the pleural cavity, causing empyema that compromises lung function and escalates the sepsis risk. The mortality rate ranges from 1520% in complicated cases. In India, where diabetes prevalence is high the *K. pneumoniae* accounts for 40-60% of pyogenic liver abscesses. The delayed diagnosis and antimicrobial resistance exacerbate the adverse outcomes[4]. The toxicological burden includes *K.*

pneumoniae's ability to form biofilms and resist antibiotics, necessitating stewardship to prevent carbapenem-resistant strains (CRKP)[4].

This case report describes a rare presentation of *K. pneumoniae* pyogenic liver abscess with empyema in a newly diagnosed diabetic patient in Kerala, India, highlighting the toxicological interplay of bacterial virulence and host factors. It underscores the need for toxicological research into *K. pneumoniae* virulence factors to guide novel therapeutic strategies in resource-limited settings, where early intervention and antibiotic stewardship are critical.

Case Description

On July 31, 2025, a 44-year-old male, presented to Ananthapuri Hospital, Trivandrum, with a 2week history of intermittent high-grade fever (up to 39°C), chills, rigors, and neck pain radiating to the shoulders. The person also had a 3-week history of a swelling on the right lateral abdomen. He has no recent travel, trauma, alcohol use, or animal exposure, and had no known comorbidities. Examination revealed fever (38.5°C), tachycardia (110 bpm), and hypertension (140/90 mmHg). The abdominal swelling 5 × 5 cm soft-to-firm was tender but non-mobile, with no signs of peritonism.

Laboratory investigations uncovered undiagnosed type 2 diabetes mellitus, with random blood glucose of 375 mg/dL and HbA1c of 11.2%. Hematological parameters showed leukocytosis (14,500/cu.mm, 80% neutrophils, 10% bands), thrombocytopenia (68,000/cu.mm), and anemia (hemoglobin 10.2 g/dL). Inflammatory markers were elevated: C-reactive protein (CRP) at 150 mg/L and procalcitonin at 2.5 ng/mL. Liver function tests indicated mild transaminitis (AST 120 IU/L, ALT 95 IU/L) and hypoalbuminemia (2.8 g/dL). Serology for typhoid, dengue, and malaria were negative. Urine analysis revealed pyuria, prompting culture.

A chest X-ray showed mild right pleural effusion, and abdominal ultrasound suggested a hypoechoic hepatic lesion. Contrast-enhanced CT abdomen confirmed a 5 × 4 cm multiloculated hypoechoic lesion in liver segment VII, consistent with a pyogenic abscess, with mild splenomegaly (13 cm) and no biliary obstruction (Figure 1,2). On August 2, 2025, the patient developed severe pleuritic chest pain, tachypnea (28 breaths/min), tachycardia (130 bpm), and hypotension (90/60 mmHg), indicating sepsis. Arterial blood gas revealed type 1 respiratory failure (PaO2 60 mmHg, PaCO2 32 mmHg). Xray showed loculated right pleural effusion with compressive atelectasis(Figure 3). Thoracentesis aspirated 200 mL of purulent pleural fluid (pH 7.1, LDH 1200 IU/L, glucose 20 mg/dL), with Gram-negative rods on staining. Quantitative culture grew *K. pneumoniae* at $\geq 10^7$ CFU/mL, sensitive to ceftazidime, piperacillintazobactam, and meropenem. Urine culture isolated the same strain, while blood cultures were negative. The diagnosis was *K. pneumoniae* PLA with transdiaphragmatic rupture causing empyema, precipitated by uncontrolled diabetes.

Empirical ceftazidime-avibactam (2.5 g IV every 8 hours) was initiated for potential multidrugresistant *K. pneumoniae*, alongside insulin infusion targeting blood glucose of 140-180 mg/dL. A 28F intercostal drain (ICD) was placed in the right 5th intercostal space, draining 1.5 L of pus initially. Repeat CT on August 7 showed persistent pleural loculations, prompting right posterolateral thoracotomy with decortication on August 8. Intraoperatively, a thick fibrinopurulent peel was removed, allowing full expansion of all three right lung lobes. The liver abscess was not drained, as imaging suggested pleural decompression. Histopathology confirmed suppurative inflammation. In the ICU, the patient received non-invasive ventilation (NIV), high-flow

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nasal cannula (HFNC), and analgesia (paracetamol, fentanyl). By postoperative day 3, CRP fell to 45 mg/L and procalcitonin to 0.8 ng/mL(10).

By August 10, the patient was ambulating and tolerating oral intake. Antibiotics were deescalated to piperacillin-tazobactam (4.5 g IV every 6 hours) for 7 days, then oral cefuroxime (500 mg twice daily) for 10 days, per stewardship protocols(8). The ICD was removed on August 11 after output <100 mL/day and chest X-ray confirmed lung re-expansion (Figure 4). Glycemia was managed with subcutaneous insulin and metformin. The patient was discharged on August 13, 2025, with normalized platelets (250,000/cu.mm) and HbA1c of 7.8% at 1-month follow-up on September 13, 2025, with resolved abscess and effusion on ultrasound.

Discussion

This case shows the toxicological burden of *K. pneumoniae* in India's diabetic population. The pathogen's virulence factors like capsular polysaccharides and LPS exploits the host immune system to cause invasive infections[2]. The K1/K2 capsule inhibits phagocytosis by mimicking host glycans. This mechanism is amplified due to glycated immunoglobulins and reduced complement activity in diabetes[3]. LPS activates TLR4-NF-κB pathways, inducing a cytokine storm that drives systemic inflammation. This is evidenced by the patient's elevated CRP and procalcitonin[5]. In India, hvKP accounts for 50-70% of PLAs. Also diabetes increases the susceptibility, with odds ratio about 8-12 for abscess formation in patients with HbA1c more than 9[6].

Hyperglycemia (375 mg/dL) would have likely increased the *K. pneumoniae* virulence through the lactate-mediated upregulation of capsule biosynthesis. This is shown in recent studies that PTS-CRP axis enhances cps gene expression in high-glucose environments[7]. This promotes bacterial invasiveness which explains the rapid progression to empyema, seen in 10-15% of PLAs in India[6]. Transdiaphragmatic rupture occurred due to abscess expansion, with pus tracking into the pleural space which caused loculated empyema and respiratory compromise.

Therapeutically, the case highlights the challenge of balancing antimicrobial efficacy against resistance risks, critical in India, where CRKP prevalence is 20-40% in tertiary centers. Ceftazidime-avibactam was chosen for its activity against ESBL and carbapenemase-producing *K. pneumoniae*, minimizing endotoxin release. De-escalation to piperacillin-tazobactam and cefuroxime adhered to India's National Treatment Guidelines for Antimicrobial Use, reducing selective pressure for multidrug-resistant organisms[4]. Surgical decortication was critical, as percutaneous drainage is contraindicated in ruptured PLAs with empyema due to endotoxin dissemination risks[8]. Glycemic control via insulin infusion was vital, as euglycemia (140-180 mg/dL) restores neutrophil function and attenuates AGE-RAGE-mediated inflammation, reducing mortality by up to 50% in diabetic PLA patients[9].

Specific challenges include delayed presentation due to low health literacy and limited access to imaging in rural areas. Negative initial serologies underscore the need for culture-driven diagnosis, as empirical antibiotics may fail against hvKP[10]. Toxicologically, anti-toxin therapies (e.g., anti-capsular antibodies or TLR4 inhibitors) could reduce antibiotic reliance, addressing resistance concerns[10]. Limitations include the lack of molecular typing for hvKP (e.g., rmpA gene) and no hepatic drainage, justified by decompression. Future research in India should explore metabolomics to elucidate lactate-toxin interactions and promote community-based diabetes screening.

Conclusion

This case of *K. pneumoniae* PLA with empyema in a diabetic patient highlights the toxicological synergy between bacterial endotoxins and hyperglycemia, driving invasive infections in India's high-diabetes-burden population. Multidisciplinary management including targeted antibiotics, surgical intervention, and glycemic control are critical for a favorable outcome.

Clinical significance

The case advocates for toxicological research into *K. pneumoniae* virulence factors and stewardship to curb antimicrobial resistance, a growing concern in resource-constrained settings. Early diabetes screening and accessible diagnostics could mitigate such complications, reducing morbidity in vulnerable communities.

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Conflicts of Interest: The authors declare no conflicts of interest.

Patient Consent: Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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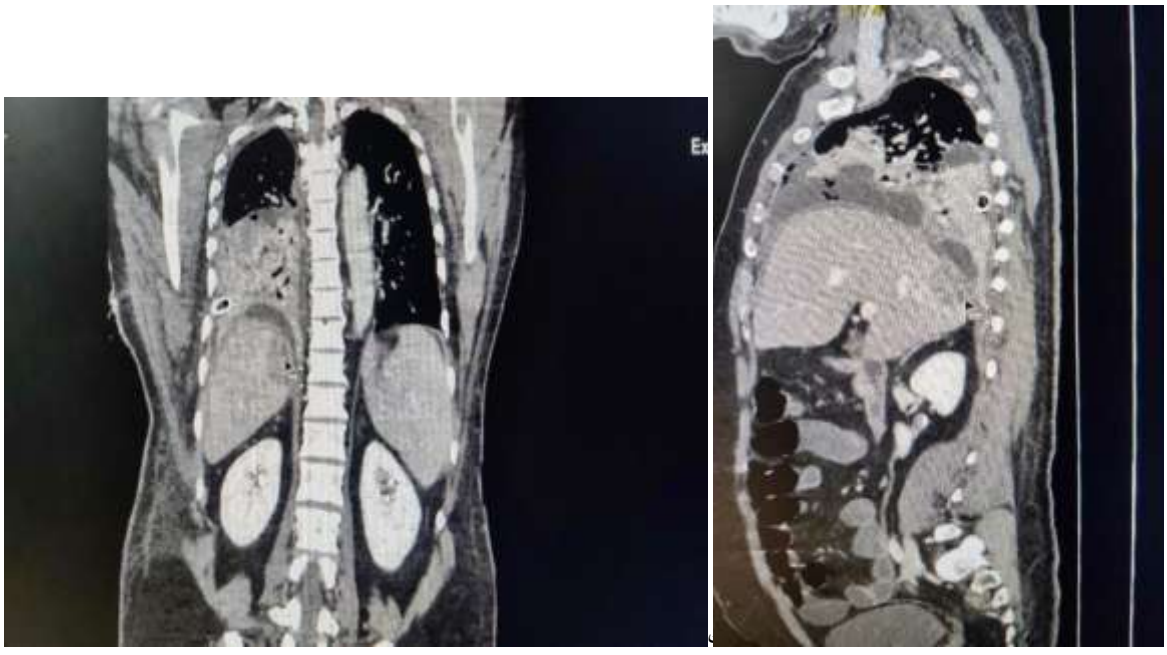
[Figure 1, 2: Contrast-enhanced CT abdomen demonstrating a 5 × 4 cm hypoechoic lesion in liver segment VII (arrow), consistent with pyogenic abscess, with mild splenomegaly.]



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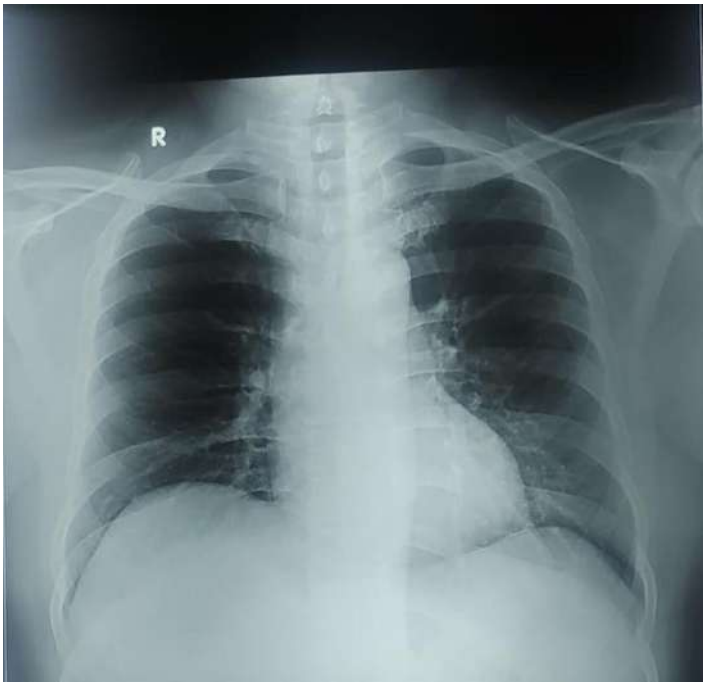
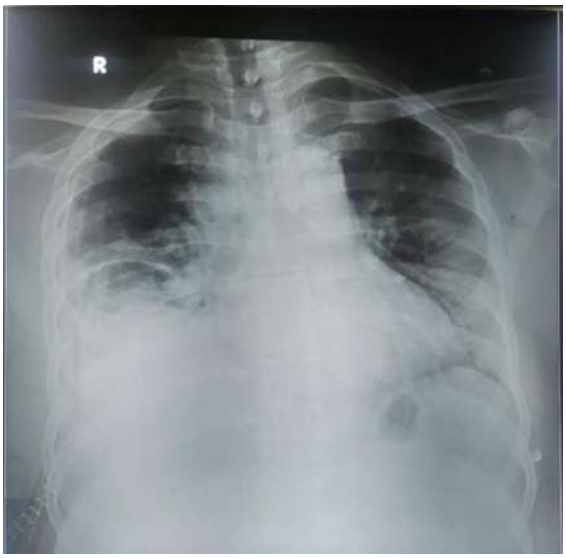
[Figure 3: Pre-operative CT chest revealing loculated right pleural effusion with compressive atelectasis.]



[Figure 4: Post-operative chest X-ray showing lung re-expansion and ICD in situ



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