

From Chronic Cough in Primary Care to Invasive *Klebsiella Pneumoniae* Liver Abscess Syndrome Complicated by Endogenous Enophthalmitis and Pulmonary Embolism: A Case Report

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Abstract

Case Report

Invasive *Klebsiella pneumoniae* liver abscess syndrome (IKLAS) is an increasingly recognised clinical entity characterised by pyogenic liver abscess, bacteraemia and metastatic infection, particularly in patients with diabetes mellitus. Early manifestations are frequently non-specific and may mimic common primary care presentations. We report the case of a 58-year-old man with poorly controlled type 2 diabetes mellitus and hypertension who initially presented to primary care with nocturnal positional dry cough. Symptoms were managed conservatively as probable gastroesophageal reflux disease or upper airway cough syndrome. Over the following weeks, he developed progressive fatigue, anorexia, significant weight loss, right upper quadrant pain, fever and jaundice. Urgent referral to secondary care revealed severe sepsis with marked leukocytosis, cholestatic liver dysfunction, acute kidney injury and multiple hepatic abscesses. Blood cultures and liver abscess cultures grew *Klebsiella pneumoniae*. The clinical course was complicated by endogenous endophthalmitis causing visual impairment and right-sided pulmonary embolism requiring anticoagulation with apixaban. Management included broad spectrum intravenous antibiotics, image-guided drainage of liver abscesses, intravitreal antimicrobial therapy and multidisciplinary specialist care. The patient was discharged on a prolonged antimicrobial regimen consisting of two weeks of intravenous antibiotics followed by four weeks of oral therapy. This case highlights the importance of recognising evolving red flag symptoms in primary care and demonstrates the potentially devastating metastatic complications of invasive *Klebsiella pneumoniae* infection.

Keywords: *Klebsiella pneumoniae*, pyogenic liver Abscess, primary care, chronic cough, endogenous endophthalmitis, pulmonary embolism, diabetes mellitus, sepsis.

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INTRODUCTION

Pyogenic liver abscess (PLA) is an uncommon but potentially life-threatening infection associated with significant morbidity and mortality. Over recent decades, *Klebsiella pneumoniae* has emerged as the predominant pathogen responsible for community acquired liver abscesses in several regions worldwide, particularly among patients with diabetes mellitus (Siu *et al.*, 2012). Hypervirulent strains have been associated with a distinctive invasive syndrome characterised by bacteraemia and metastatic spread to distant organs including the eye, lung and central nervous system (Shon *et al.*, 2013).

Diagnosis can be challenging because early symptoms are often non-specific and overlap with common conditions managed in General Practice. Constitutional symptoms, chronic cough, weight loss

and abdominal discomfort are frequently encountered in primary care and are often attributable to benign pathology. However, progressive symptoms require careful reassessment and robust safety-netting.

This report describes a patient whose illness evolved from an apparently benign chronic cough presentation in primary care to severe disseminated *Klebsiella pneumoniae* infection complicated by liver abscesses, and endogenous endophthalmitis and pulmonary embolism.

CASE REPORT

A 58-year-old male with poorly controlled type 2 diabetes mellitus (HbA1C >10%), hypertension and dyslipidaemia initially attended primary care in February 2026 with an intermittent dry cough occurring exclusively when lying supine. He denied fever, wheeze,

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chest pain, rhinorrhoea or exertional symptoms. Clinical examination was unremarkable apart from mild sinus tachycardia. Oxygen saturations were normal.

A provisional diagnosis of gastroesophageal reflux disease, upper airways cough syndrome or airway hypersensitivity was considered. Symptomatic treatment with dextromethorphan was prescribed alongside lifestyle advice, including head elevation during sleep and avoidance of airway irritants.

Over the subsequent weeks, the patient developed progressive fatigue, anorexia and unintentional weight loss of approximately 10-12kgs. Two weeks before admission he developed intermittent right upper quadrant abdominal pain associated with fever and chills. He was initially treated elsewhere for presumed gastritis without improvement.

Three days before emergency presentation he developed worsening abdominal pain, jaundice, dark urine, vomiting and profound weakness. Reassessment identified pallor, diaphoresis, jaundice and clinical evidence of systemic illness. He was urgently referred to hospital with suspected sepsis.

Initial investigations demonstrated marked leucocytosis (WBC 44.8-50 x 10⁹/L), neutrophilia, CRP 253 mg/L, procalcitonin 91.5 ng/mL, elevated bilirubin, cholestatic liver function abnormalities and acute kidney injury. Blood cultures subsequently grew *Klebsiella Pneumoniae*.

Abdominal ultrasonography demonstrated hepatomegaly and a heterogeneous lesion within the left hepatic lobe with associated intrahepatic biliary dilatation. Contras-enhanced CT imaging confirmed multiple multiloculated hepatic abscesses predominantly affecting the left lobe, with the largest collection measuring approximately 9 x 5 x 4 cm. Additional findings included a left lower lobe pulmonary infiltrate and rectosigmoid wall thickening.

The patient was treated with aggressive intravenous fluid resuscitation and intravenous ceftriaxone. Ultrasound guided drainage of the liver abscess yielded approximately 100 ml of purulent material. Culture results confirmed *Klebsiella pneumoniae* bacteraemia and pyogenic liver abscess syndrome.

During admission, he developed progressive visual impairment affecting the right eye. He described floaters, black spots and a curtain-like visual defect. Ophthalmological examination demonstrated conjunctival injection, hypopyon, and vitritis. A diagnosis of endogenous endophthalmitis was established. He underwent urgent ophthalmological intervention including anterior chamber tap, vitreous

sampling, pars plana vitrectomy and intravitreal administration of ceftazidime and vancomycin.

Further inpatient investigation identified the right-sided pulmonary embolism. Anticoagulation was commenced with apixaban, which was continued following discharge.

Transthoracic echocardiography demonstrated preserved left ventricular systolic function with no evidence of infective endocarditis. Tumour markers include AFP, CEA and CA-19-9 were normal. MRI/MRCP and colonoscopic assessment were planned following resolution of acute infection because of significant weight loss, anaemia and bowel wall thickening.

The patient demonstrated gradual clinical improvement following source control and antimicrobial therapy. Renal function normalised, inflammatory markers improved, and bilirubin levels decreased. He was discharged on a prolonged antimicrobial regimen consisting of two weeks of intravenous antibiotics followed by four weeks of oral antibiotic therapy under specialist follow-up.

DISCUSSION

This case illustrates the diagnostic challenges posed by invasive *Klebsiella pneumoniae* liver abscess syndrome in primary care. The patient's initial presentation with isolated positional cough was clinically consistent with common benign diagnosis such as reflux disease or upper airway cough syndrome. At that stage, there were no features mandating extensive investigation.

However, the subsequent development of constitutional symptoms, progressive weight loss and abdominal pain represented an evolving clinical picture requiring diagnostic reconsideration. Continuity of care and repeated clinical review are fundamental strengths of general practice and were critical in recognising deterioration.

Diabetes mellitus is one of the strongest recognised risk factors for invasive *Klebsiella* infection. Hyperglycaemia impairs neutrophil chemotaxis and phagocytosis, increasing susceptibility to severe infection and metastatic dissemination (Wang *et al.*, 1998). The patient's markedly elevated HbA1C likely contributed significantly to disease severity.

Endogenous endophthalmitis remains one of the most devastating complications of invasive *Klebsiella* syndrome. Visual symptoms may initially appear minor, yet delayed diagnosis can result in permanent blindness. The presence of floaters, blurred vision or ocular discomfort in patients with *Klebsiella* bacteraemia

should trigger urgent ophthalmological assessment (Sheu *et al.*,2019).

An additional notable feature of this case was the development of pulmonary embolism. Whilst venous thromboembolic disease is increasingly recognised in severe systemic infection, inflammatory activation, immobility and endothelial dysfunction likely contributed to thrombogenesis. Management required anticoagulation with apixaban in addition to ongoing infection control.

Current management principles for pyogenic liver abscess include prompt antimicrobial therapy, source control through image-guided drainage and prolonged antibiotic treatment. Treatment courses commonly extend to four to six weeks depending on the radiological response and microbial findings.

This case emphasises the need for vigilance and managing evolving symptoms in primary care and highlights the importance of maintaining broad differentials in patients with diabetes presenting with constitutional symptoms.

CONCLUSION

Invasive *Klebsiella pneumoniae* liver abscess syndrome should be considered in patients with diabetes who present with progressive in constitutional symptoms, abdominal pain and systemic inflammation. This case demonstrates how a seemingly benign primary care presentation evolved into severe disseminated infection, complicated by endogenous endophthalmitis and pulmonary embolism. Early recognition, prompt referral, source control and multidisciplinary management are essential to improve outcomes. Safety-netting and longitudinal reassessment remain crucial components of effective primary care practice.

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