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Case Report

Others Section

Septic Pulmonary Embolism with Hepatic and Renal Abscesses in a Diabetic Patient Mimicking Tuberculosis or Metastasis: A Case Report

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ABSTRACT

Septic Pulmonary Embolism (SPE) is a rare but life-threatening pulmonary condition characterised by the embolic spread of infected thrombi from extrapulmonary sources into the lungs. It most commonly occurs as a complication of systemic infections and presents with cavitating lung lesions, respiratory compromise, and multi-organ involvement. Immunocompromised individuals, particularly those with uncontrolled diabetes mellitus, are especially vulnerable to such infections. We present a case of a 41-year-old man with poorly controlled diabetes and a prior clinical diagnosis of pulmonary tuberculosis, who presented with a progressive cough, dyspnoea, and abdominal pain. Imaging revealed multiple cavitary lesions in the lungs, hepatic abscesses, and renal abscesses with partial renal vein thrombosis. Microbiological investigations confirmed infection with Klebsiella pneumoniae and Pseudomonas aeruginosa. A differential diagnosis of SPE, pulmonary tuberculosis, and cystic metastases was considered. The patient initially responded well to culture-sensitive antibiotics and improved glycaemic control. However, three months later, he was readmitted with worsening respiratory symptoms and hyperglycaemia, and despite appropriate interventions, he developed respiratory failure and succumbed to septic shock. This case highlights the diagnostic complexity of SPE, its overlap with other pulmonary pathologies, and the crucial role of metabolic control and vigilant follow-up in diabetic individuals with systemic infections.

Keywords: Cavitary lung lesions, Diabetes mellitus, Glycaemic control, Hepatic abscess, Klebsiella pneumoniae, Renal vein thrombosis

CASE REPORT

A 41-year-old male, a farmer, presented with a progressive cough, mucoid whitish expectoration, and dyspnoea on exertion {Modified Medical Research Council (mMRC) Grade I-II} for three months. He had a known history of type 2 diabetes mellitus for five years and was on Glycomet GP2 BD. He also experienced oral ulcers for one month, dull abdominal pain for approximately 20 days that was insidious in onset, non-radiating, and gradually progressive in nature. The abdominal pain was continuous and not associated with meals or bowel movements, with no specific aggravating or relieving factors. The patient reported a significant unintentional weight loss of approximately 6 kg over the preceding two months, along with decreased appetite and heartburn. There was no history of seasonal or diurnal variation, paroxysmal nocturnal dyspnoea, orthopnoea, vomiting, or nausea. The patient had a prior hospitalisation two months before presentation for fulminant hepatitis and had been clinically diagnosed with pulmonary tuberculosis based on symptoms and chest imaging, for which he was empirically started on Anti-Koch's Treatment (AKT). However, the absence of microbiological confirmation and subsequent findings led to reconsideration of this diagnosis.

Physical examination revealed generalised weakness but was otherwise unremarkable. Initial laboratory investigations showed an elevated C-Reactive Protein (CRP) of 122.81 mg/L, while liver and kidney function tests were within normal limits. Blood culture demonstrated growth of Klebsiella pneumonia, and bronchoalveolar lavage revealed Pseudomonas aeruginosa. Sputum for Acid-Fast Bacilli (AFB) and Truenat for Mycobacterium Tuberculosis (MTB) were negative.

On initial evaluation, the complete blood count revealed a markedly elevated CRP level of 122.81 mg/L, suggestive of ongoing systemic inflammation. Liver and kidney function tests were within normal limits [Table/Fig-1a-d], ruling out hepatic or renal impairment at baseline. Blood culture showed growth of Klebsiella pneumoniae, while bronchoalveolar lavage samples revealed P. aeruginosa, both of which indicated active systemic and pulmonary infections, respectively [Table/Fig-1d]. Sputum testing for AFB and the Truenat assay for MTB were negative, helping exclude active tuberculosis. Chest X-ray demonstrated multiple cavitary lesions in the right lung, correlating with the patient's respiratory symptoms [Table/Fig-2]. High-Resolution Computed Tomography (HRCT) of the thorax showed multiple cavitating nodules with surrounding ground-glass opacities [Table/Fig-3]. Further imaging with Contrast-Enhanced Computed Tomography (CECT) of the abdomen and pelvis showed multiple hepatic and renal abscesses, along with mild free fluid in the abdomen [Table/Fig-4,5], supporting a systemic septic process consistent with septic emboli.

Laboratory variable	Result	Reference range
Haemoglobin (g/dL)	8.8	12-16
Total red blood cells (million cells/mm^3)	4.48	0-1.070
White blood cells (cells/µL)	4300	3,500-9,000
Platelet count (x10^5)	1.8	1.5-4.5
Haematocrit (%)	32.3	42-53
Granulocyte (10^9)	7.5	1.5-8.5
Lymphocyte (%)	20	25-45
Monocyte (%)	04	3-7
Eosinophils (%)	01	1-6
Basophils (%)	00	0-0.75

[Table/Fig-1a]: Complete blood count.

Laboratory variable	Result	Reference range
Urea (mg/dL)	25	9-45
Creatinine (mg/dL)	0.5	0.5-1.5
Serum sodium (mEq/L)	139	135-145
Serum potassium (mEq/L)	3.6	3.5-4.5

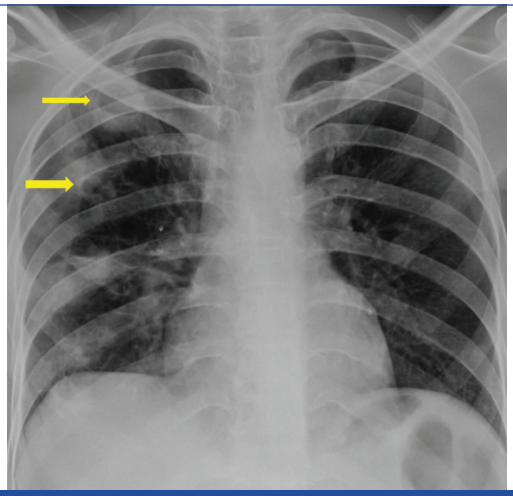
[Table/Fig-1b]: Kidney function test.

Laboratory variable	Result	Reference range
HbA1c (%)	9.5	5.4-6.4
D-dimer (ng/mL)	1892	<500
C-reactive protein (mg/L)	122.81	<3
HbsAg	Non-reactive	
HIV	Non reactivemi9	
HCV	Non-reactive	
Blood culture	Klebsiella pneumoniae	
BAL culture	Pseudomonas aeruginosa	

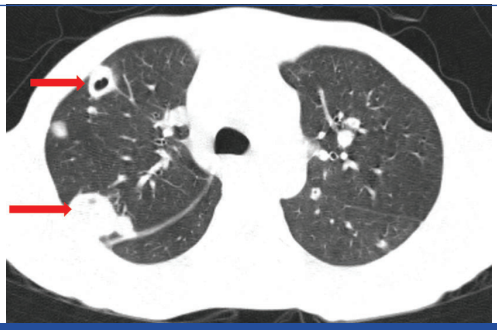
[Table/Fig-1c]: Liver function test.
HbA1c: Glycosylated haemoglobin; HbsAg: Heaptitis B surface antigen; HIV: Human immunodeficiency virus; HCV: Hepatitis C virus; BAL: Bronchoalveolar lavage

Laboratory variable	Result	Reference range
Alkaline phosphatase (U/L)	213	53-128
Serum glutamic-pyruvic transaminase (U/L)	24	7-56
Serum glutamic-oxaloacetic transaminase (U/L)	41	8-33
Total protein (g/dL)	6.6	6.0-8.3
Albumin (g/dL)	2.2	3.4-5.4
Globulin (g/dL)	4.4	2.0-3.5
Total bilirubin (mg/dL)	1.5	0.1-1.0
Conjugated bilirubin (mg/dL)	0.4	0-0.3
Unconjugated bilirubin (mg/dL)	1.1	0.2-0.8

[Table/Fig-1d]: Other relevant test.



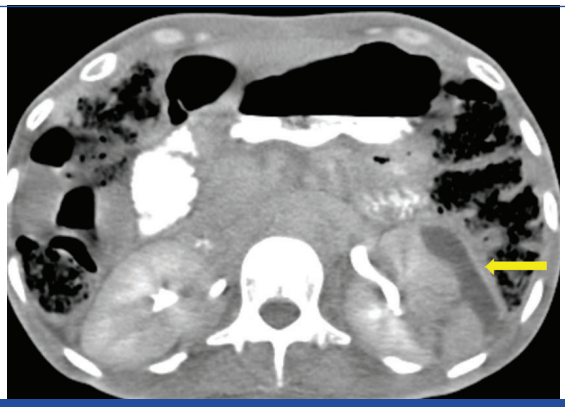
[Table/Fig-2]: Chest X-ray postero-anterior view showing ring-like shadow forming the appearance of a cavity seen in the upper and middle zone (yellow arrow) of the right hemithorax.



[Table/Fig-3]: Axial HRCT thorax showing multiple cavitatory lesions (red arrow) in the right hemithorax.



[Table/Fig-4]: Axial section of CECT abdomen and pelvis showing multiple areas of low density seen scattered throughout the liver in segments V, VI, and VII with rim enhancement giving double target sign, some of these appear coalescent, forming cluster sign (yellow arrow).



[Table/Fig-5]: Axial section of CECT abdomen and pelvis showing a hypodense area seen in the left kidney with surrounding perinephric collection (yellow arrow).

INVESTIGATIONS

Differential Diagnosis

The differential diagnoses considered included septic emboli, pulmonary tuberculosis, and cystic metastasis. Pulmonary tuberculosis was initially suspected due to the chronic respiratory symptoms and cavitary lung lesions; however, it was ruled out based on negative sputum AFB staining and Truenat assay for MTB, as well as lack of microbiological confirmation despite empirical anti-Koch's therapy. Cystic metastasis was considered due to the presence of cavitary pulmonary lesions and hepatic involvement, but tumour markers (CEA and CA-19-9) were within normal limits and no primary neoplastic source was identified on imaging, making metastatic disease unlikely.

The final diagnosis was septic pulmonary embolism with hepatic and renal abscesses, secondary to gram-negative bacterial sepsis in the setting of uncontrolled diabetes mellitus. This conclusion was supported by positive blood cultures for K. pneumoniae, isolation of P. aeruginosa from bronchoalveolar lavage, and radiological findings consistent with septic embolic dissemination to the lungs, liver, and kidneys.

Treatment

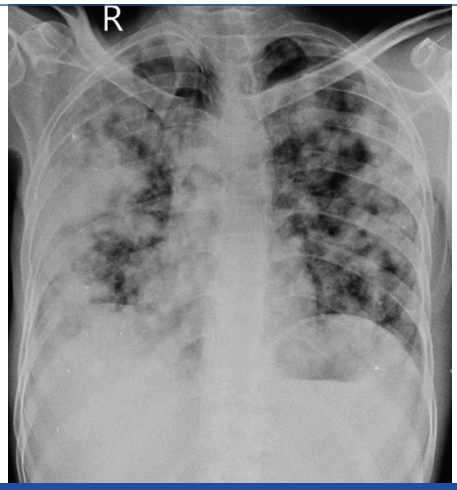
The patient was managed with culture-specific antibiotics targeting K. pneumoniae and P. aeruginosa. Initial in-hospital management included tablet AKT-4 (combination tablet of four anti-tubercular drugs – isoniazid 75 mg, rifampicin 150 mg, pyrazinamide 400 mg, and ethambutol 275 mg)- once daily, injection meropenem 1 g intravenous three times daily for 14 days, injection doxycycline 100 mg twice daily for 14 days, injection Mixtard 12 units at 8:00 AM and 12 units at 8:00 PM, injection low molecular weight heparin (enoxaparin sodium) 0.4 mL subcutaneously twice daily for 7 days, injection Orofer-S in 100 mL normal saline for 3 days followed by tablet Orofer XT once daily, tablet Shelcal 500 mg once daily, tablet Mucinac 600 mg three times daily, tablet Limcee 500 mg once

daily, tablet Udiliv (ursodeoxycholic acid) 300 mg twice daily, topical TESS (triamcinolone acetonide) oral paste, and chlorhexidine mouth gargles three times daily.

Blood glucose levels were closely monitored during hospitalisation. Once glycaemic control was achieved, the patient was transitioned to oral metformin 500 mg and glimepiride 1 mg twice daily.

Outcome and Follow-Up

The patient showed significant improvement clinically and radiologically following 14 days of treatment, with reduced cough and resolution of abdominal pain. No further respiratory complaints were reported at the time of discharge. However, over the next three months, he developed worsening symptoms, including breathlessness (mMRC Grade III) and increased mucoid expectoration. Upon readmission, random blood sugar was 367 mg/dL, and a follow-up chest X-ray revealed bilateral heterogeneous opacities, indicating disease progression [Table/Fig-6]. Follow-up Chest X-ray showing bilateral heterogeneous opacities. He was admitted to the critical care unit, where glycaemic control was re-established and treatment was resumed with injection meropenem 1 g intravenously three times daily and injection doxycycline 100 mg intravenously twice daily, based on prior culture-sensitive reports.



[Table/Fig-6]: Follow-up chest x-ray showing multiple cavitary lesions in both lung fields.

Despite aggressive management, the patient developed respiratory failure and septic shock and ultimately succumbed to his illness on the fifth day following readmission. This case highlights the critical role of glycaemic control in managing systemic infections in diabetic patients. Insulin resistance and uncontrolled hyperglycaemia likely contributed to the flare-up of sepsis and worsened clinical outcomes. Routine clinical and radiological follow-up is essential for long-term management.

DISCUSSION

This case underscores the diagnostic complexity involved in differentiating between SPE, pulmonary tuberculosis, and cystic metastasis in a diabetic patient presenting with multi-organ involvement. The patient exhibited hepatic and renal abscesses alongside cavitating lung lesions and renal vein thrombosis, ultimately confirmed to be caused by K. pneumoniae and P. aeruginosa. These findings, supported by radiology and microbiology, pointed toward a diagnosis of SPE with an extrapulmonary septic source, emphasising the role of comprehensive imaging and early culture-guided therapy.

The pathophysiology of SPE is rooted in the embolic spread of infected thrombi from remote sites such as soft tissue, bone, or intra-abdominal organs into the pulmonary circulation, producing parenchymal inflammation and infarction. In our case, the renal and hepatic abscesses likely served as the origin of embolisation. Radiological findings of peripheral cavitating nodules with bilateral distribution were consistent with SPE. Yildirim S et al., similarly reported SPE in a diabetic patient secondary to a temporal boil, presenting with bilateral cavitating lung lesions. However, unlike the cutaneous source in their case, ours involved deeper visceral organs and vascular thrombosis, suggesting a more extensive and aggressive septic dissemination [1].

The immunocompromised status associated with poorly controlled diabetes is a well-established risk factor for severe infections, including SPE. Hyperglycaemia impairs neutrophil chemotaxis and phagocytosis, increases bacterial virulence, and fosters a hypercoagulable state, all of which contribute to increased susceptibility and rapid progression of infections [2,3]. Katsumata M et al., documented a case of K. pneumoniae pyelonephritis in a diabetic individual that evolved into SPE, though without hepatic involvement or vascular thrombosis [2]. In contrast, our patient had simultaneous renal and hepatic abscesses with thrombosis, highlighting a more disseminated and life-threatening clinical picture.

Epidemiological shifts in SPE have been reported in multiple studies. Historically associated with right-sided endocarditis, SPE is now increasingly linked to extrapulmonary sources. In a retrospective analysis of 41 cases, Goswami et al., reported that skin, soft tissue, and deep organ infections were more common origins than cardiac sources [3]. Similarly, Ye R et al., in their systematic review of 168 cases, identified K. pneumoniae in only 11 cases, with hepatic abscess being a predominant source [4]. Additional support comes from Cheng DL et al., who presented three cases of K. pneumoniae SPE in diabetic patients, all with hepatic abscess and poor glycaemic control [5]. Our case adds to this cluster of Klebsiella-associated SPE in diabetic hosts - uncommon, but increasingly recognised.

Our patient's deterioration despite early antibiotic initiation contrasts with other case reports where favourable outcomes followed timely treatment. For instance, Cheng DL et al., described K. pneumoniae liver abscess syndrome with metastatic spread, including pulmonary septic emboli and endophthalmitis, particularly in diabetics [5]. Additionally, Chou DW et al., reported a series of patients with septic pulmonary embolism secondary to K. pneumoniae liver abscesses, where the majority were diabetic and demonstrated characteristic cavitary pulmonary nodules with variable outcomes, including fatal cases, highlighting the poor prognosis in those with inadequate glycaemic control [6]. Unlike the patient in Yildirim S et al.,'s report, who improved with antibiotics alone [1], our patient succumbed to respiratory failure and septic shock, reinforcing the importance of vigilant follow-up and aggressive glycaemic management in systemic infections.

In conclusion, this case expands the spectrum of SPE presentations in diabetic individuals and supports the necessity of integrating radiological, microbiological, and metabolic data for accurate diagnosis and treatment. It also highlights the need for clinicians to consider SPE in the differential diagnosis when evaluating diabetic patients with respiratory symptoms and multiple organ abscesses - even in the absence of traditional cardiac sources or identifiable primary malignancy.

CONCLUSION(S)

This case highlights the diagnostic and management complexities of SPE in an immunocompromised diabetic patient. It underscores the need for clinicians to maintain a high index of suspicion when multiple cavitary lesions and extrapulmonary abscesses are present. Differentiating between tuberculosis, septic emboli, and metastatic disease is essential to prevent misdiagnosis, particularly in high Tuberculosis (TB)-burden settings. Early and accurate microbiological diagnosis is critical to guide targeted antibiotic therapy. Uncontrolled diabetes serves as both a predisposing and aggravating factor in the development and progression of systemic infections. Vigilant follow-up with clinical, radiological, and metabolic monitoring is imperative to improve outcomes in similar cases.

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