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Emphysematous Pyelonephritis Masquerading as Diabetic Ketoacidosis: A Case Report

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ABSTRACT

Emphysematous pyelonephritis (EPN) is a rare, fulminant, gas-forming necrotising infection of the renal parenchyma and surrounding tissue that occurs almost exclusively in patients with diabetes mellitus and carries substantial morbidity and mortality if not recognised promptly. Diabetic ketoacidosis (DKA) is itself a recognised precipitant and consequence of severe infection in such patients, and when the two coexist the metabolic emergency can dominate the clinical picture and delay diagnosis of the underlying urological source. We report the case of a 49-year-old man with poorly controlled type 2 diabetes mellitus (glycated haemoglobin 12.8%), hypertension and heart failure with reduced ejection fraction who presented with a two-day history of left flank pain, vomiting, fever and breathlessness, and was found to be in septic shock requiring vasopressor support. Initial evaluation revealed severe leukocytosis, diabetic ketoacidosis (pH 7.27, bicarbonate 14 mmol/L, blood glucose 434 mg/dL with positive ketones), acute kidney injury and electrolyte derangement. Cross-sectional imaging confirmed left-sided emphysematous pyelonephritis with obstructive uropathy, and urine culture grew Enterobacter species. The patient was managed with intensive care support, intravenous fluid and insulin therapy, broad-spectrum antibiotics, vasopressors, and urological source control with double-J (DJ) ureteric stenting, avoiding the need for nephrectomy. He improved steadily and was discharged on basal-bolus insulin therapy with a plan for elective stent removal. This case underscores the importance of maintaining a high index of suspicion for emphysematous pyelonephritis in any diabetic patient presenting with diabetic ketoacidosis accompanied by flank pain or unexplained sepsis, and illustrates that early imaging combined with a multidisciplinary medical-urological approach can achieve renal preservation even in critically ill patients.

Keywords: Emphysematous pyelonephritis; diabetic ketoacidosis; urosepsis; septic shock; acute kidney injury; double-J stenting; type 2 diabetes mellitus

INTRODUCTION

Emphysematous pyelonephritis (EPN) is an uncommon but life-threatening necrotising infection of the renal parenchyma and perinephric tissue characterised by the presence of gas within the collecting system, parenchyma, or perinephric space. It occurs predominantly in patients with poorly controlled diabetes mellitus and is thought to result from a combination of hyperglycaemia-driven impaired tissue perfusion, gas-forming facultative anaerobic organisms, and local tissue necrosis. Escherichia coli is the most frequently isolated organism, followed by Klebsiella pneumoniae, with Enterobacter species and other Enterobacteriaceae implicated less commonly.

Diabetic ketoacidosis (DKA) and EPN can present concurrently, as severe infection is a well-recognised precipitant of DKA, while the resulting profound dehydration, acidosis and catabolic stress can in turn aggravate renal hypoperfusion and infection severity, creating a self-perpetuating cycle. When a patient with diabetes presents in DKA with septic shock, the metabolic derangement frequently dominates initial clinical attention, and a concealed urological source such as EPN may be overlooked unless specifically sought with cross-sectional imaging. Delayed diagnosis is strongly associated with increased mortality, historically reported in excess of 40 percent in case series predating widespread use of minimally invasive drainage techniques, though contemporary outcomes have improved considerably with early recognition, percutaneous or ureteric drainage, and aggressive medical management. We describe a case in which DKA and septic shock were the presenting features of underlying left-sided emphysematous pyelonephritis, successfully managed with combined intensive medical therapy and urological source control.

CASE PRESENTATION

A 49-year-old man with a known history of poorly controlled type 2 diabetes mellitus (previously managed with oral hypoglycaemic agents and intermittent insulin, with a glycated haemoglobin of 12.8%), hypertension, and heart failure with reduced ejection fraction was referred from an outside hospital with a two-day history of severe abdominal pain, vomiting, fever and breathlessness. At the referring centre he had been haemodynamically unstable, with a systolic blood pressure of approximately 80 mmHg requiring a noradrenaline infusion at 5 mL/hour.

On arrival at our institute he was conscious, alert and following commands. Abdominal examination revealed marked tenderness over the left lumbar and hypochondriac region, raising suspicion of an underlying renal pathology. There was no family history of note, no known drug allergies, and no significant surgical history.

Investigations

Initial laboratory evaluation revealed severe infection with leukocytosis (total leukocyte count 24,300 /µL; absolute neutrophil count 21.36 $\times 10^3$/µL), with haemoglobin 10.5 g/dL and a platelet count of 1.5 lakh/µL. Arterial blood gas analysis demonstrated a high anion-gap metabolic acidosis consistent with diabetic ketoacidosis: pH 7.27, pCO_2 25 mmHg, bicarbonate 14 mmol/L, base deficit 15.4, blood glucose 434 mg/dL, with positive serum/urine ketones. Notable electrolyte abnormalities included hyponatraemia (sodium 123

mmol/L) and hyperkalaemia (potassium 5.6 mmol/L). Oxygenation was modestly impaired (PO_2 72 mmHg, SpO_2 approximately 92% on supplemental oxygen) with a lactate of 1.4 mmol/L and haematocrit of 34%.

The constellation of unexplained sepsis, left flank tenderness and metabolic derangement prompted cross-sectional imaging, which supported a diagnosis of left emphysematous pyelonephritis with associated obstructive uropathy. Urine culture subsequently grew Enterobacter species, confirming the causative organism.

Total leukocyte count	24,300 /µL
Absolute neutrophil count	21.36 ×10³/µL
Haemoglobin	10.5 g/dL
Platelet count	1.5 lakh/µL
Arterial pH	7.27
pCO_2	25 mmHg
Bicarbonate (HCO_3^-)	14 mmol/L
Base excess	−15.4
Blood glucose	434 mg/dL (ketones positive)
Serum sodium	123 mmol/L (hyponatraemia)
Serum potassium	5.6 mmol/L (hyperkalaemia)
Lactate	1.4 mmol/L
PO_2 / SpO_2	72 mmHg / ~92% on oxygen
Haematocrit	34%
Urine culture	Enterobacter species
Imaging diagnosis	Left emphysematous pyelonephritis with obstructive uropathy

Diagnosis

Based on the clinical presentation, laboratory findings and imaging, the patient was diagnosed with:

- Left emphysematous pyelonephritis (Enterobacter species)
- Urosepsis with septic shock
- Diabetic ketoacidosis
- Acute kidney injury
- Decompensated glycaemic control on a background of uncontrolled type 2 diabetes mellitus
- Hypertension
- Heart failure with reduced ejection fraction (HFrEF)

Management

The patient was managed in a critical care setting with a coordinated medical and urological approach. Initial resuscitation comprised aggressive intravenous fluid therapy, continuous intravenous insulin infusion for correction of diabetic ketoacidosis, and close electrolyte monitoring with correction of the hyponatraemia and hyperkalaemia. Broad-spectrum intravenous antibiotic therapy with meropenem was instituted empirically for severe urosepsis, alongside vasopressor support with noradrenaline for septic shock. Supportive measures included antiemetic therapy with ondansetron, proton pump inhibition with pantoprazole, supplemental oxygen, and close haemodynamic monitoring.

Given the imaging evidence of obstructive uropathy complicating the emphysematous pyelonephritis, the urology team performed source control with double-J (DJ) ureteric stenting, allowing decompression and drainage in lieu of more radical surgical intervention such as nephrectomy.

Clinical Course and Outcome

Following source control and continued intensive care management, the patient demonstrated gradual but consistent clinical improvement. Haemodynamic status stabilised, permitting tapering and eventual discontinuation of vasopressor support. Urine output improved progressively, indicating recovery of the acute kidney injury, while the metabolic abnormalities associated with diabetic ketoacidosis resolved with continued insulin therapy and fluid management. The patient also achieved defervescence, with resolution of fever reflecting a favourable response to antibiotic therapy and the drainage procedure.

At the time of discharge, the patient was clinically stable with stable haemodynamics, resolving infection, improving urine output, and renal parameters under continued monitoring. He was discharged on a basal-bolus insulin regimen (insulin degludec/insulin aspart combination, 100 IU/mL, administered subcutaneously twice daily before meals) with advice for elective DJ stent removal after four weeks. Antiplatelet therapy with clopidogrel 75 mg was initiated following specialist cardiology consultation, in the context of his underlying heart failure with reduced ejection fraction.

At outpatient follow-up approximately three weeks after the index presentation, the patient was alert and haemodynamically stable (temperature 96.3°F, blood pressure 122/85 mmHg, heart rate 110 beats per minute, SpO_2 99% on room air, weight 56.2 kg, body mass index 21.68 kg/m², Modified Early Warning Score 2). He continued to be followed for diabetic care, including dietary counselling, self-monitoring of blood glucose, hypoglycaemia awareness education (capillary blood glucose below 70 mg/dL), foot care, retinal screening, and periodic laboratory review encompassing fasting and postprandial glucose, glycated haemoglobin, lipid profile, serum creatinine, urine routine examination and urine albumin-to-creatinine ratio.

DISCUSSION

Emphysematous pyelonephritis is a rare but severe necrotising infection that predominantly affects patients with diabetes mellitus, in whom it accounts for the overwhelming majority of reported cases. The pathogenesis is thought to relate to fermentation of high tissue glucose concentrations by gas-forming uropathogens in a setting of impaired local immune response and tissue perfusion, with obstruction of the urinary tract as seen in this

patient further predisposing to disease progression by promoting stasis and impairing clearance of infected, gas-producing material. Left-sided involvement, as observed here, has been reported to be somewhat more common than right-sided disease in several case series, though the reasons for this laterality preference remain incompletely understood.

A distinguishing feature of this case is the manner of presentation: the patient's clinical picture was dominated at the outset by profound metabolic derangement diabetic ketoacidosis with severe acidaemia, hyperglycaemia and electrolyte disturbance together with septic shock requiring vasopressor support, rather than by overt urological symptoms. While flank tenderness was present on examination and appropriately prompted further evaluation, the coexistence of DKA can readily overshadow an underlying renal source of sepsis, particularly in a patient already labelled as having poorly controlled diabetes. This case illustrates the reciprocal relationship between EPN and DKA: severe infection is a recognised precipitant of ketoacidosis, while the resulting acidosis, dehydration and catabolic state may in turn exacerbate renal hypoperfusion and infection severity. Clinicians should therefore maintain a high index of suspicion for an occult renal or urological source of sepsis in any diabetic patient presenting with DKA accompanied by flank pain, unexplained leukocytosis disproportionate to other apparent sources, or sepsis refractory to initial resuscitation, and should pursue early cross-sectional imaging typically non-contrast or contrast-enhanced computed tomography to identify or exclude emphysematous pyelonephritis.

Management of EPN has evolved considerably over recent decades. Historically, emergency nephrectomy was considered standard treatment, particularly for more extensive (higher-grade) disease, but outcomes have improved with a shift toward a kidney-preserving strategy combining aggressive medical management — broad-spectrum antibiotics, glycaemic and metabolic correction, and haemodynamic support — with minimally invasive source control such as percutaneous catheter drainage or ureteric stenting, reserving nephrectomy for patients who fail to respond to conservative drainage or who have extensive non-viable renal parenchyma. In the present case, the presence of obstructive uropathy made ureteric decompression with a double-J stent an appropriate and effective source-control measure, performed in conjunction with intensive medical therapy, and the patient avoided nephrectomy with good clinical recovery.

This case also highlights the importance of multidisciplinary coordination between critical care, endocrinology and urology in the management of critically ill diabetic patients with urosepsis. Tight glycaemic control following recovery, structured diabetic education, and scheduled follow-up including planned removal of the ureteric stent are essential components of comprehensive post-discharge care to reduce the risk of recurrence and further renal injury.

CONCLUSION

Emphysematous pyelonephritis should be considered in any patient with diabetes mellitus presenting with diabetic ketoacidosis, septic shock and flank pain, even when the metabolic emergency appears to dominate the clinical picture. Prompt cross-sectional imaging, early broad-spectrum antimicrobial therapy, aggressive correction of metabolic derangement, and timely urological source control favouring minimally invasive

drainage over nephrectomy where feasible can achieve favourable outcomes and renal preservation, as demonstrated in this case.

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