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BEYOND THE USUAL SUSPECTS: A RARE CASE OF DKA COMPLICATED BY EMPHYSEMATOUS PYELONEPHRITIS AND GASTRIC MUCORMYCOSIS

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Abstract
A 55-year-old woman with poorly controlled type 2 diabetes mellitus presented with dyspnea, fever, abdominal pain, and was diagnosed with diabetic ketoacidosis (DKA) and acute kidney injury (AKI). Imaging revealed emphysematous pyelonephritis (EPN) with gas-forming destruction of the left kidney, while upper gastrointestinal endoscopy and biopsy confirmed Helicobacter pylori-associated chronic gastritis complicated by invasive gastric mucormycosis. Urine culture grew *Candida albicans* and endotracheal aspirate yielded *Acinetobacter*, highlighting polymicrobial infection. This constellation of EPN, DKA, and gastric mucormycosis is exceedingly rare and underscores the need for high clinical suspicion in diabetic patients with multiorgan symptoms. The patient was managed with insulin infusion, DJ stenting, broad-spectrum antibiotics, and liposomal amphotericin B, but was discharged against medical advice. This case emphasizes the diagnostic and therapeutic challenges posed by simultaneous renal and gastric fungal infections in an immunocompromised host and contributes unique insight to the limited literature on such presentations

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Introduction

Diabetes mellitus, especially when poorly controlled, predisposes patients to severe and often life-threatening infections. Among these, **emphysematous pyelonephritis (EPN)** is a necrotizing infection of the renal parenchyma characterized by gas formation, and it carries a high mortality risk if not promptly diagnosed and managed [1,2]. The pathophysiology involves gas-producing organisms in an immunocompromised renal environment, often facilitated by hyperglycaemia, impaired perfusion, and urinary stasis [1]. The coexistence of **diabetic ketoacidosis (DKA)** further compounds the clinical severity, as the acidotic and hyperglycaemic milieu augments fungal or bacterial virulence and impairs host immunity [3]. Cases have been documented in which EPN precipitated DKA, underscoring a bidirectional and synergistic worsening of clinical states [4].

Compounding the complexity, invasive **gastric fungal infections** such as mucormycosis or candidiasis remain rare yet aggressively destructive complications in immunocompromised hosts.5,6 Gastric mucormycosis, in particular, is associated with high mortality rates and often requires a combination of antifungal therapy and surgical debridement for optimal outcomes [5]. The gastrointestinal form accounts for only a small fraction of invasive mucormycosis cases and typically presents with nonspecific abdominal symptoms, making early recognition challenging [6]. Meanwhile, gastric candidiasis or fungal colonization has been reported in diabetic and immunosuppressed individuals, sometimes masquerading as ulcerative or mass lesions [7].

The present case involves a 55-year-old woman with longstanding type 2 diabetes and additional comorbidities, who presented with dyspnea, abdominal pain, fever, and multiorgan dysfunction. Imaging and microbiologic evaluations revealed features of emphysematous pyelonephritis and invasive gastric fungal disease, complicated further by acute kidney injury, profound anemia, and

metabolic derangements. This combination of pathologies in a single patient is exceedingly rare and poses significant diagnostic and therapeutic challenges. Through this report, we aim to highlight the intersecting pathogenic mechanisms, diagnostic dilemmas, and management strategies in such a multifaceted clinical scenario.

Case Presentation

A 55-year-old woman with a known history of type 2 diabetes mellitus for six years, obstructive airway disease, and bipolar disorder presented with complaints of shortness of breath, diffuse abdominal pain, and fever, all persisting for five days. The dyspnea had progressed from Modified Medical Research Council (MMRC) Grade II to Grade IV. Abdominal pain was described as diffuse, dull-aching, intermittent, and not related to food intake. She also reported dysuria but denied any vomiting or alteration in bowel habits.

She was initially admitted to another hospital, where she was diagnosed with acute kidney injury (AKI), prompting her referral to our center for further management. On arrival, she was found to be in hemodynamic shock with a blood pressure of 70/40 mmHg, heart rate of 120 bpm, and respiratory rate of 30/min. Clinical examination revealed pallor without icterus or lymphadenopathy. Chest auscultation revealed bilateral inspiratory crepitations. Cardiovascular and abdominal examinations were unremarkable. Given her instability, she was intubated and started on vasopressor support with noradrenaline.

Investigations revealed normocytic normochromic anemia (hemoglobin 5.8 g/dL), leukocytosis (total count 21,240/mm³, 82% neutrophils), elevated urea (118 mg/dL), creatinine (4.8 mg/dL), hypernatremia (153 mmol/L), hyperkalemia (5.8 mmol/L), and metabolic acidosis (bicarbonate 12 mmol/L). Serum ketones were positive, and HbA1c was markedly elevated at 12%, confirming diabetic ketoacidosis (DKA). Chest radiograph showed bilateral lower zone opacities with increased bronchovascular markings suggestive of pulmonary edema. CT thorax revealed right lower lobe consolidation with edema.

CT-KUB demonstrated an enlarged left kidney nearly replaced by ill-defined collections and confluent gas pockets, consistent with emphysematous pyelonephritis. A DJ stent was placed, and cultures from urine and endotracheal aspirate grew *Candida albicans* and *Acinetobacter* spp., respectively. Antifungal treatment with fluconazole and antibiotic therapy with minocycline were initiated as per sensitivity.

Upper GI endoscopy revealed gastric ulcers, and biopsy confirmed *Helicobacter pylori*-associated chronic gastritis with evidence of invasive fungal infection morphologically consistent with mucormycosis. PET-CT showed FDG uptake in the gastric wall without mass lesion. Liposomal amphotericin B was initiated. Although surgical consultation was obtained, conservative management was advised. A follow-up endoscopy was planned, but the patient was discharged against medical advice due to personal reasons.

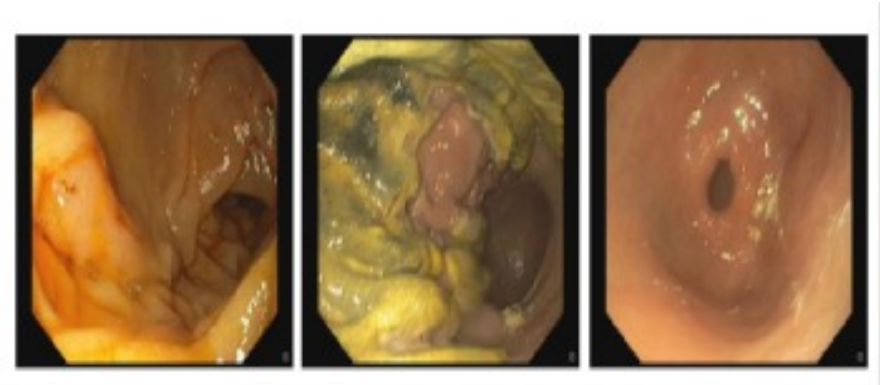

Fig 1: UGI scopy

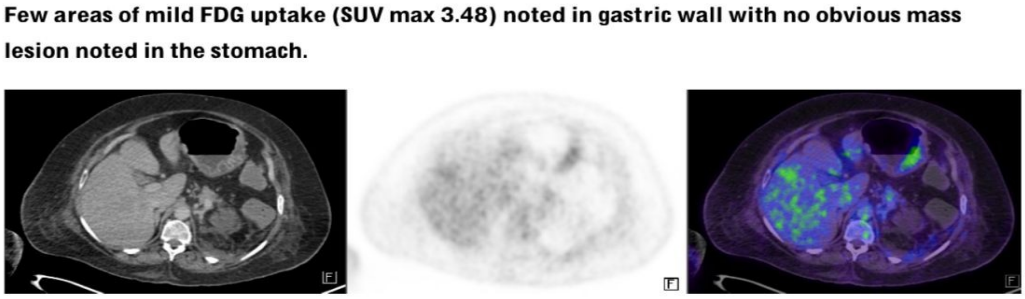

Fig 2: PET Sscan

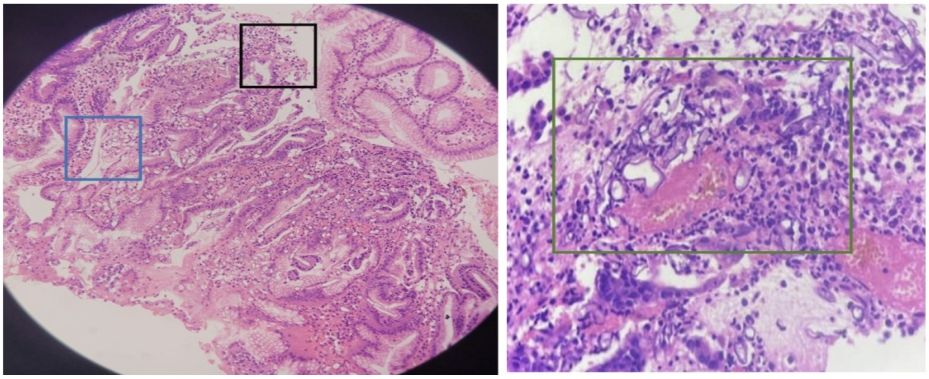

Fig 3: H &E staining shows gastric epithelium with subepithelium showing dense mixed inflammation with areas of ulceration (black box) and necrosis (Blue box) along with broad aseptate fungal hyphae with obtuse-angled branching with angioinvasion (Green box) consistent with mucormycosis

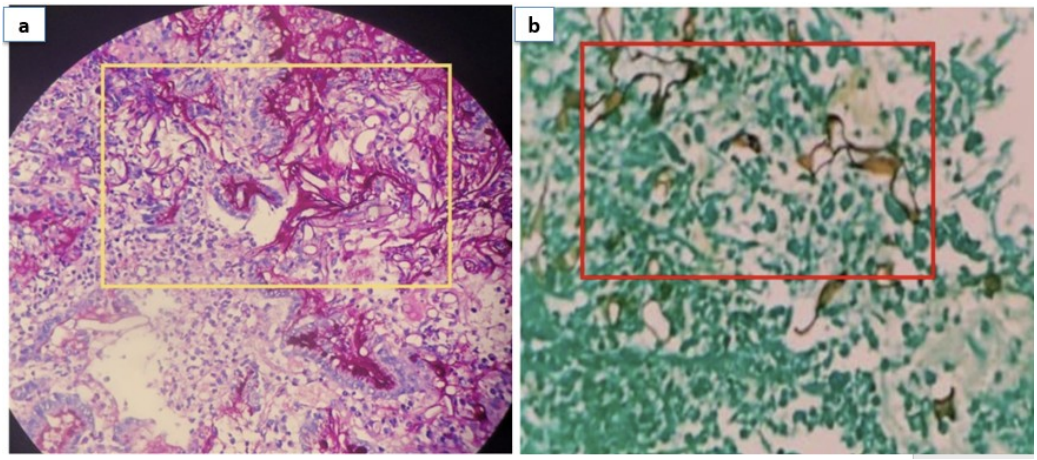

Fig 4: Histopathology a) Periodic Acid Schiff staining showing broad ribbon like aseptate magenta stained hyphae consistent with mucormycosis.b) Grocott's methenamine silver (GMS) staining showing fungal hyphae (Red box) appearing brown on a counterstained green background.

Discussion

This patient illustrates a striking convergence of rare and life-threatening pathologies in a single host: emphysematous pyelonephritis (EPN), diabetic ketoacidosis (DKA), and invasive gastric fungal infection. Each of these entities is serious in isolation; their simultaneous occurrence poses major diagnostic and therapeutic challenges. Here, we review key pathophysiologic considerations,

diagnostic strategies, therapeutic dilemmas, prognostic issues, and highlight the uniqueness of our case in the light of published literature.

Diabetes mellitus impairs multiple arms of host immunity, including neutrophil chemotaxis, phagocytosis, intracellular killing, and complement activation, thereby increasing susceptibility to infections of unusual severity and by opportunistic organisms [8]. In diabetic patients, uncommon necrotizing infections like emphysematous pyelonephritis, emphysematous cholecystitis, or mucormycosis are disproportionately represented [9]. EPN in particular has been repeatedly documented as one of the classical "diabetic only" infections [10]. In DKA, the acidic, hyperglycemic, and ketone-rich milieu further hampers immune function, encourages fungal growth, and may worsen microvascular perfusion. Thus, DKA sometimes acts not only as a complication but as a permissive environment for opportunistic fungal infections. Some reports describe mucormycosis presenting or exacerbated during DKA episodes.

EPN is an acute, necrotizing infection of the renal parenchyma (with possible extension to perinephric tissues), characterized by gas formation by bacterial metabolism [10]. Typical risk factors include poorly controlled diabetes, urinary tract obstruction, and immunosuppression [11]. The classical causative organisms are **gas-forming bacteria** such as *Escherichia coli* and *Klebsiella pneumoniae*, which dominate reported series (49–67% for *E. coli*, 20–24% for *Klebsiella*) [10]. Other pathogens, seldom, include anaerobes, *Proteus*, *Clostridium*, and rarely fungal species or *Candida* in immunocompromised patients [10]. Mortality in EPN remains substantial, historically between 40% and 90% in older series, particularly when diagnosis or intervention is delayed [10]. More recent series adopting CT-guided drainage and conservative approaches report lower mortality. Misgar et al. documented a 10-year experience with more modern management and improved survival [11]. A critical decision in EPN management is whether to adopt conservative measures (intravenous antibiotics plus percutaneous drainage) or more aggressive intervention (nephrectomy). Abdul-Halim et al. reviewed cases where early nephrectomy was correlated with better outcomes in severe disease, while drainage alone sometimes led to residual infection or recurrence [12]. The choice hinges on hemodynamic stability, extent of infection (gas spread, parenchymal destruction), renal salvage potential, and surgical risk.

Although bacterial EPN is well known, EPN caused by fungal pathogens is exceedingly uncommon. A recent case describes **EPN due to mucormycosis** — a necrotizing fungal infection inflicting gas production in renal tissue — in a non-diabetic patient [13]. However, in most of these, the fungal involvement is limited to the kidney and often discovered histologically, not concomitantly in a distant organ (like stomach). The concurrence of EPN and invasive gastric fungus in a diabetic under DKA is, to our knowledge, unheard of in PubMed literature.

Gastrointestinal mucormycosis, particularly gastric mucormycosis, is rare but well-described in immunocompromised patients, especially those with diabetes or DKA. Several case reports note its high mortality and often silent presentation [14]. In one case, emphysematous gastritis was associated with invasive mucormycosis—a rare presentation where gas enters the gastric wall by fungal invasion. That report shows how both entities (gas in gastric wall + fungal invasion) are independently life-threatening, and their co-occurrence is extremely rare [14]. Our patient's biopsy evidence of fungal invasion in gastric ulcers (consistent with mucormycosis) alongside imaging/functional evidence of EPN represents a dual-site severe fungal/bacterial interplay in a diabetic host. No mass lesion was seen on PET-CT, underscoring that gastric fungal disease may elude radiologic detection initially.

Several features make our case particularly unique: Concomitant severe infection in the kidney (gas-forming EPN) and stomach (invasive fungal gastritis) in one patient is exceptional. Most literature reports focus on single organ involvement. In our case, DKA and AKI coexisted with EPN and fungal disease. This triad not only complicated management but also likely served as a vicious cycle—DKA weakens host defenses; infection worsens metabolic derangement. Urine culture grew *Candida albicans* (an unusual EPN pathogen) and respiratory culture grew *Acinetobacter*. The microbial diversity indicates polymicrobial/invasive potential rather than a typical monomicrobial EPN. Gastric

fungal infection was confirmed histologically from biopsy, while imaging and PET-CT lacked an obvious mass lesion. This shows the insidious nature of gastric mucormycosis and the need for high suspicion, especially in diabetic patients with abdominal symptoms. The need to balance aggressive surgical interventions (nephrectomy, gastric resection) versus conservative management under hemodynamic instability posed a major dilemma. The patient's eventual discharge against medical advice prevented full outcome assessment, but the management decisions stand as instructive. There is, to our knowledge, no prior case where emphysematous pyelonephritis (with gas formation), DKA, and simultaneous gastric fungal invasion are documented in a single diabetic patient.

In diabetic patients presenting with abdominal pain, fever, AKI, and metabolic derangement, one must consider EPN early and suspect additional infection foci beyond urinary tract. CT scanning is the gold standard in diagnosing EPN, providing high sensitivity to detect gas in renal parenchyma or perinephric tissue [10]. In our patient, CT KUB delineated confluent gas pockets replacing the kidney. In contrast, gastric fungal disease may not manifest as distinct mass lesions or typical radiologic hallmarks; hence biopsy remains critical. Empirical broad-spectrum antimicrobials are necessary, but culture-guided therapy is crucial. Fungal biopsies in gastric ulcers helped clinch the diagnosis; relying on imaging/PET alone would have risked underdiagnosis. The optimal management for such combined catastrophic infections is not established, but general principles may guide therapeutic decisions:

In any case of EPN or invasive fungal disease, prompt hemodynamic stabilization (fluids, vasopressors if needed), correction of metabolic derangements (insulin therapy in DKA), and support of organ perfusion are fundamental. Broad-spectrum antibiotics that cover usual EPN pathogens are required. In our case, after culture results, therapy was tailored (e.g., minocycline for *Acinetobacter*, fluconazole for *Candida*). For suspected or proven mucormycosis, liposomal amphotericin B remains the mainstay. Early initiation is associated with improved outcomes in invasive fungal disease. Given her hemodynamic compromise, a purely surgical approach (nephrectomy) would carry high risk. Thus, percutaneous drainage or DJ stenting plus supportive therapy may be preferable in critically ill patients. Several reports support percutaneous drainage plus antibiotics as effective in select EPN cases [15]. Abdul-Halim et al. also indicated that early nephrectomy may benefit those who can tolerate surgery [12]. Coordination between nephrology, urology, infectious diseases, gastroenterology, and critical care is vital, especially when weighing risks and benefits of surgical versus conservative strategies in unstable patients.

In gastric mucormycosis, surgery (resection or debridement) in addition to antifungal therapy is often advocated, given poor penetration of antifungals in necrotic tissue. However, surgical risk in a patient with multi-organ failure must be balanced. In our patient, surgical opinion favored conservative management given the unstable status. A plan for repeat endoscopy to assess response is prudent, but patient factors (e.g., discharge against advice, logistics) may preclude that in real-world settings, as in our case.

Outcomes in EPN are strongly influenced by prompt diagnosis, rapid initiation of therapy, and appropriate intervention selection. Delay in diagnosis or drainage/nephrectomy increases mortality risk [10]. In invasive gastric mucormycosis, prognosis is dismal if diagnosis is delayed. Many published cases report mortality despite aggressive therapy, especially when fungal invasion is deep or widespread [14]. The coexistence of DKA and multi-organ bacterial/fungal infection further worsens prognosis. Pitfalls include misattribution of symptoms to one organ (e.g. attributing abdominal pain to only renal origin), neglecting to biopsy gastric lesions, or over-reliance on imaging when lesions are subtle. The absence of a defined mass lesion on PET-CT in our patient reinforces that imaging cannot replace tissue diagnosis in fungal disease. Because our patient left hospital against medical advice, the final outcome is unknown—a limitation. However, the case stands as a potent reminder of the need for vigilance when diabetic patients present with overlapping systemic and abdominal features.

From this case, the following lessons emerge:

1. In diabetics with systemic infection and abdominal symptoms, think beyond single organ processes —coexisting pathologies may be present.
2. Always consider invasive fungal disease in diabetic ketoacidosis, particularly with GI symptoms or failure to respond to antibacterial therapy.
3. Gastric lesions in high-risk patients should be biopsied early; a negative imaging study does not exclude invasive fungal involvement.
4. In EPN complicated by hemodynamic instability, a conservative approach (drainage, stenting, tailored antimicrobial therapy) may be safer initially than radical surgery, but the decision must be individualized.
5. Multidisciplinary planning is essential; decisions on surgery must balance risk versus potential therapeutic gain in an unstable host.

Our case represents an unprecedented combination of EPN with gas formation, DKA, and concurrent invasive gastric fungal disease in a diabetic patient. Its rarity highlights the need for clinician awareness of overlapping severe infections in immunocompromised hosts. Early imaging, culture/histopathology, and judicious therapeutic choices are key.

Conclusion

This case highlights the complex interplay between uncontrolled diabetes mellitus, diabetic ketoacidosis, and severe opportunistic infections involving multiple organ systems. The simultaneous occurrence of emphysematous pyelonephritis and invasive gastric fungal infection in the setting of DKA is exceptionally rare and has not been widely documented in the literature. Early recognition, prompt imaging, tissue diagnosis, and timely antimicrobial and antifungal therapy are pivotal to survival. Our patient's clinical course exemplifies how overlapping pathologies in immunocompromised hosts can obscure diagnosis and challenge conventional management strategies. Clinicians must maintain a high index of suspicion for polymicrobial and multisystem involvement in diabetic patients presenting with systemic symptoms and gastrointestinal complaints. This case also highlights the value of multidisciplinary collaboration and the critical role of biopsy in identifying uncommon etiologies such as gastric mucormycosis, even in the absence of a radiological mass. Ultimately, this report adds to the limited evidence on the co-occurrence of renal and gastrointestinal fungal infections in diabetics and reinforces the importance of holistic, aggressive, and individualized patient care in such high-risk scenarios.

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