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FULMINANT EMPHYSEMATOUS PYELONEPHRITIS IN A TRANSPLANT KIDNEY (CLINICAL OBSERVATION AND LITERATURE REVIEW)

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Emphysematous pyelonephritis (EPN) is a necrotizing infection of the renal parenchyma and its surrounding areas that causes gas accumulation around the renal parenchyma, collecting system and surrounding tissues in the process of vital activity of several microorganisms. EPN occurs nearly exclusively in people with diabetes. Treatment strategies for EPN have evolved over the past 20 years, with minimally invasive procedures replacing nephrectomy, which has resulted in lower mortality rates (12.5–13%). EPN is rare in kidney transplant (KT) recipients and is characterized by a severe, often fulminant course with a high rate of adverse outcomes, which is determined primarily by background immunosuppressive therapy. There is no universally accepted consensus on the radiographic classification of EPN in KT recipients and its management. We present the first description of EPN in transplanted kidney in a 45-year-old woman with post-transplant diabetes, obesity and recurrent urinary tract infections. Massive antibiotic therapy (ABT), percutaneous nephrostomy, transplantectomy, renal replacement therapy, selective cytokine adsorption, and ventilatory support were all administered on the patient after she was admitted to the hospital with increasing clinical symptoms of sepsis and multiple organ failure. Death occurred on the fourth day after disease onset. The article examines 38 clinical cases from the English-language segment of the medical literature from the late 1970s to the present. EPN in KT recipients is characterized by the predominance of male gender, including among the deceased, rapid development of sepsis and acute kidney injury. There was no statistically significant difference in the frequency of emergency transplantectomies among surviving and deceased patients. Mortality was 28%. The issue of EPN in transplanted kidney requires more research and the development of optimal therapeutic plans, including surgical strategies.

Keywords: emphysematous pyelonephritis, renal graft, diabetes mellitus, transplantectomy, clinical case.

INTRODUCTION

Emphysematous pyelonephritis (EPN) is a rare, severe infection of the kidney that causes gas to accumulate in the tissues. It's characterized by a necrotizing inflammation of the renal parenchyma, collecting ducts, and surrounding tissues [1, 2]. Gas formation results from the metabolic activity of certain bacteria, including *Escherichia coli*, *Klebsiella pneumoniae* and some others, which primarily generate gas through glucose fermentation. Consequently, EPN predominantly occurs in patients with diabetes mellitus (DM). The current mortality rates for EPN in patients with native kidneys range from 12.5% to 13% [3, 4].

EPN in kidney transplant (KT) recipients is extremely rare, with only a few dozen cases reported worldwide to date. When it does occur, EPN in KT recipients is typically severe and fast-developing, leading to a high rate of unfavorable outcomes, including graft loss. Due to the limited number of reported cases, there is no established consensus on its diagnosis and management in KT recipients. We present the first documented case of EPN in a KT recipient in the Russian Federation.

Objective of the study: the aim of this study is to explore the clinical features and progression of EPN in KT recipients based on our case report and existing literature. We also seek to compare the radiologic classification approaches for EPN in native kidneys versus KT recipients and discuss modern strategies for patient management, including surgical interventions.

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MATERIALS AND METHODS

This study presents a clinical case of EPN in a KT recipient with post-transplant diabetes mellitus (PTDM), obesity, and recurrent urinary tract infections (UTIs). The analysis includes initial clinical and laboratory data, the course of the disease, radiologic diagnostics, conservative and surgical treatments, and pathomorphological findings about the KT. Thirty-eight cases of EPN in KT recipients, reported from the late 1970s to the present, were reviewed and summarized from English-language medical literature.

CLINICAL CASE

A 46-year-old female patient was evaluated at a consultative and diagnostic nephrology center starting in 2017, following allogeneic kidney transplantation from a deceased donor. Her medical history includes end-stage renal failure due to chronic glomerulonephritis in 2016, which required treatment with hemodialysis. KT function was immediate, and she was placed on a triple-drug immunosuppressive therapy (IST) regimen (tacrolimus, mycophenolic acid, and methylprednisolone).

In the post-transplant period, the patient developed insulin-dependent diabetes mellitus with difficult-to-control hyperglycemia, as well as recurrent UTIs. The patient was hospitalized on three separate occasions and received multiple courses of antimicrobial therapy. Her baseline serum creatinine level remained stable, not exceeding 130 μmol/L. However, a sudden deterioration occurred on February 9, 2024, marked by a fever of up to 38.5 °C, pain localized to the KT area, worsening general weakness, nausea, repeated vomiting, and the onset of anuria.

She was initially admitted to a local hospital and, on February 10, 2024, was transferred to our clinic with a preliminary diagnosis of renal graft dysfunction. Upon admission, the above symptoms persisted, though her mental status remained unchanged.

Physical examination: height 155 cm, weight: 100 kg, body mass index (BMI): 41.6 kg/m^2, and stable hemodynamics.

Local findings: Marked palpation tenderness in the left iliac region (transplant zone), with edema of the surrounding soft tissues of the anterior abdominal wall.

Laboratory screening in the emergency unit revealed metabolic acidosis and hyperlactatemia. Additional findings included leukocytosis (14.86 × 10^9/L), hemoglobin 128 g/L, C-reactive protein (CRP) 37.5 mg/L, elevated serum creatinine at 256.4 μmol/L, plasma glucose 10.35 mmol/L, and glycated hemoglobin (HbA1c) at 9.9%. In light of acute kidney injury (AKI) and associated metabolic disorders, the patient was admitted to the intensive care unit (ICU). A contrast-enhanced multislice computed tomography (MSCT) scan was subsequently performed (Fig. 1).

Contrast-enhanced abdominal and pelvic MSCT (Fig. 1) revealed edema of the peritransplant and periureteral soft tissues. Gas bubbles were identified within the lumen of the renal pelvis and calyces of the allograft

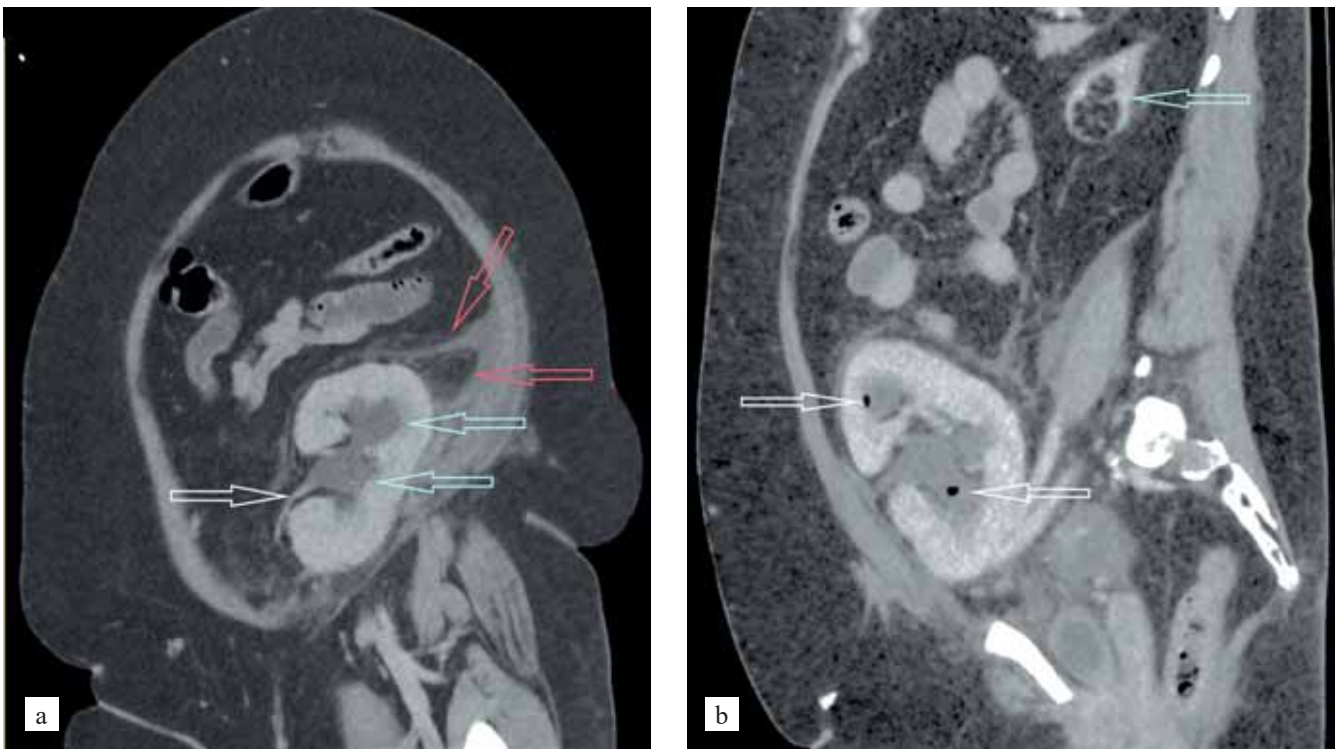

Fig. 1. Contrast-enhanced MSCT of abdominal and pelvic organs, venous phase: a, sagittal plane: gas bubbles in the kidney transplant calyxes (arrow), shriveled kidney (blue arrow); b, oblique plane: narrowing in the area of the ureteropelvic junction obstruction in kidney allograft (arrow), dilated calyces (blue arrow), perigraft tissue edema (red arrow)

(Fig. 1, a). A pronounced narrowing of the pelviureteric junction was observed, with high-density material within its lumen (Fig. 1, b), accompanied by dilatation of the pelvicalyceal system and a non-dilated ureter. The transplant parenchyma showed homogeneous contrast enhancement, with no signs of structural destruction. No excretion of contrast agent was observed during examination.

An emergency percutaneous nephrostomy (PCN) was performed, yielding urine mixed with mucous-purulent material. Given the high initial risk factors for multidrug-resistant flora – including decompensated diabetes mellitus, obesity, ongoing IST, prior antimicrobial treatment, recurrent UTIs, and recent hospitalization – empirical antibiotic therapy with piperacillin/tazobactam was initiated.

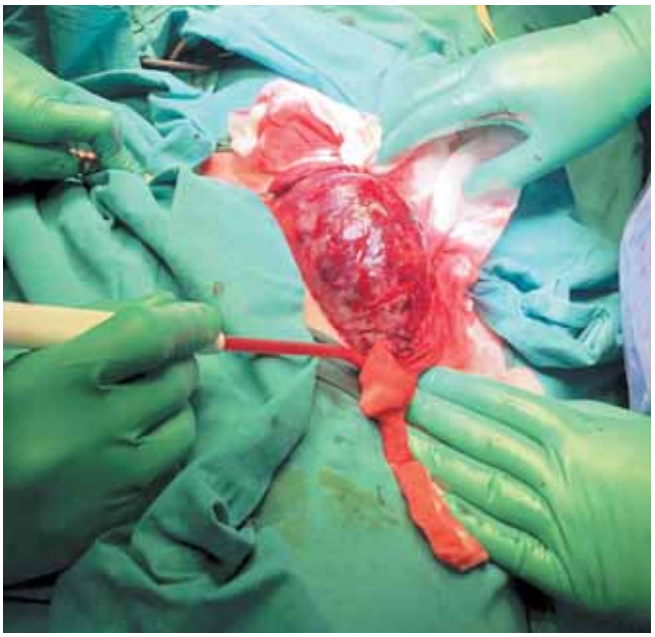

Fig. 2. Renal allograft during transplantectomy. Purulent debris areas are visible through the graft capsule

Renal replacement therapy was started in the form of prolonged venovenous hemodiafiltration. However, within the first 24 hours of observation, the patient experienced a rapid and profound deterioration of vital functions, progressing to distributive shock and multiple organ failure. This necessitated mechanical ventilation and vasopressor therapy.

Laboratory findings revealed a dramatic escalation in systemic inflammatory markers: leukocytosis (47.8 × 10^9/L), C-reactive protein (CRP) 447.6 mg/L, interleukin-6 (IL-6) >1000 pg/mL (reference: 0.00–6.40), interleukin-2 (IL-2) 5054 GE/mL (reference: 158–623), procalcitonin (PCT) >13 ng/mL, total protein 33.4 g/L, albumin 17 g/L, and platelets 34 × 10^9/L.

Considering the fulminant course of EPN in the KT recipient, complicated by systemic inflammatory response syndrome (SIRS) and multiple organ failure, a multidisciplinary team concluded that emergency transplantectomy was indicated for life-saving purposes. Surgical intervention was performed on February 12, 2024 (Fig. 2).

Following the isolation of Escherichia coli producing extended-spectrum beta-lactamases from both urine and blood cultures, antimicrobial therapy was escalated to meropenem in combination with amikacin. Intensive care measures included a multimodal extracorporeal detoxification strategy, comprising selective hemoperfusion, cytokine adsorption, and therapeutic plasma exchange.

Despite comprehensive treatment, the patient's condition continued to deteriorate rapidly. One day after the transplantectomy and on day 4 from disease onset, she died due to progressive multiple organ failure and refractory distributive shock. Findings from pathomorphological examination of the explanted kidney transplant are presented in Fig. 3.

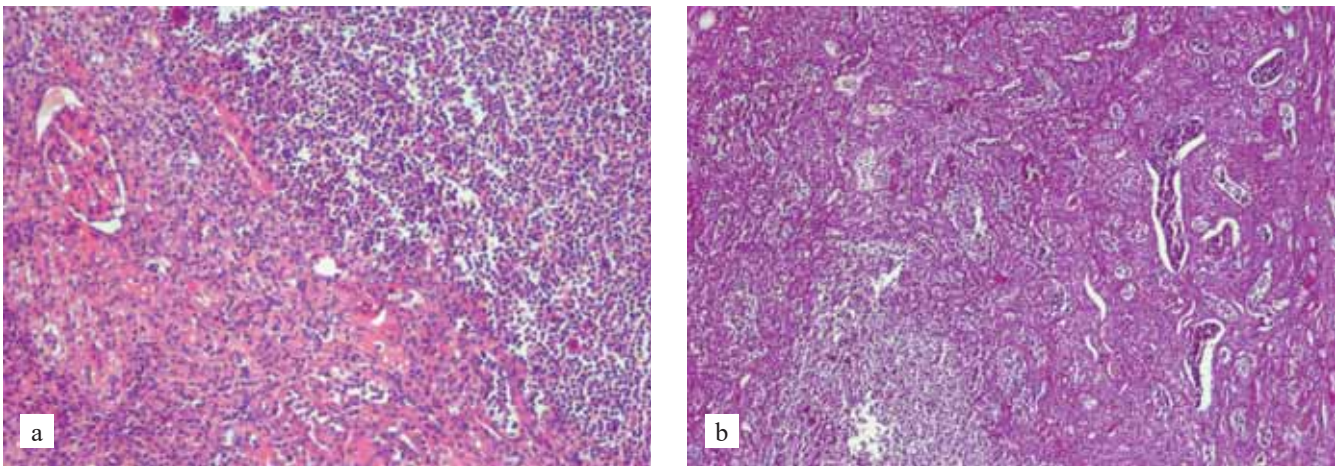

Fig. 3. Cortical layer of the kidney with diffuse, predominantly neutrophilic infiltration of the interstitium and abscess formation. Leukocytic cylinders are detected in the tubule lumen. Interstitial fibrosis and tubular atrophy. The glomerulus appears ischemic: a, H&E stain, magnification 40×; b, PAS, magnification 40×

DISCUSSION

By the end of the 19th century, several reports had described the presence of gas within the kidneys and urinary tract. In 1898, Kelly and MacCallum presented their clinical observations and summarized the data available at that time. They identified three primary causes of "pneumaturia": gas formation within the urinary tract due to invasive interventions or trauma (e.g., urological procedures or masturbation); the presence of fistulous connections between the bladder and bowel (congenital, acquired, or iatrogenic); and UTIs caused by gas-forming microorganisms [5].

The term emphysematous pyelonephritis (EPN) was introduced later, following the 1962 publication by Schultz and Klorfein, who analyzed 13 cases of renal and upper urinary tract gas accumulation due to infection [6]. In true EPN, the presence of gas is directly attributable to microbial activity, primarily from gas-producing pathogens.

It is important to recognize some historical terminological ambiguity: while "emphysematous pyelonephritis" technically describes infections involving both the kidney parenchyma and upper urinary tract, the term is sometimes used in the literature to include isolated "emphysematous pyelitis," which is limited to the collecting system.

Features of pathogens in emphysematous pyelonephritis

In approximately 70% of EPN cases, *Escherichia coli* is identified as the primary causative agent. Other members of the *Enterobacterales* order, most notably *Klebsiella pneumoniae* and *Proteus spp.*, as well as non-fermenting Gram-negative bacteria such as *Pseudomonas aeruginosa*, serve as less common etiologic agents [3, 7]. Up to 33% of the isolated pathogens are producers of extended-spectrum beta-lactamases [1, 2]. These bacteria are characterized by a high degree of structural heterogeneity and a frequent association with multidrug resistance [2, 8, 9].

Considerable attention has been directed toward identifying bacterial virulence factors that contribute to the onset and fulminant progression of EPN. In a comparative study, Tseng and Wu evaluated a broad spectrum of pathogenicity determinants expressed by E. coli strains isolated from EPN cases and contrasted them with strains obtained from patients with acute kidney infections not associated with gas formation.

The virulence genes of E. coli strains isolated from both groups were remarkably similar. However, a notable distinction was the significantly higher prevalence of the urovirulence-specific protein (usp) gene in EPN-associated strains – detected in 94% of patients with EPN versus only 67% in those with non-EPN. Additionally, there was a trend toward a lower frequency of the papG allele II gene among EPN pathogens [10]. Interestingly, epidemiological studies in both adult and pediatric populations have consistently demonstrated a predominant presence of the papG gene in strains responsible for acute pyelonephritis and recurrent UTIs in women [11].

EPN pathogens exhibit high biochemical activity, with the ability to shift to mixed acid and alcohol fermentation of glucose – processes that result in the production of hydrogen and carbon dioxide. In DM patients, elevated glucose levels in renal tissues create an ideal environment for the proliferation of gas-forming bacteria and promote high metabolic rates that lead to massive gas accumulation. In addition, uropathogenic strains of *E. coli* are known to produce a cytotoxic necrotizing factor, which induces tissue necrosis. The breakdown of necrotic tissue further contributes to additional release of methane and ammonia through the catabolism of amino acids [12].

Clinical presentation and risk factors for adverse outcomes in emphysematous pyelonephritis

The clinical presentation of EPN largely mirrors that of acute purulent pyelonephritis [1, 2]. Patients typically present with fever and chills, flank pain (often in the lumbar or subcostal regions), nausea, and vomiting. In cases of a fulminant course, there may be extensive manifestations of distributive shock and SIRS. Mental status disturbances, ranging from mild confusion to coma, are possible. The underlying causes of altered mental status in EPN patients should be evaluated individually, considering factors such as systemic intoxication, uncontrolled hypotension, DM decompensation (e.g., hyperglycemia or ketoacidosis), and, air embolism affecting the cerebral venous system. Altered mental status is a critical symptom that influences diagnostic and therapeutic tactics. There are isolated reports of the so-called "gas embolism" phenomenon in EPN cases, with gas being observed in the pulmonary artery, pelvic vessels, and even the upper sagittal and cavernous sinuses in patients with EPN of native kidneys [13, 14].

For a time, it was believed that the development of EPN required the presence of three conditions: DM (particularly poorly controlled diabetes), urinary tract obstruction, and an infectious agent capable of producing gas. However, as more data became available, it became evident that not all these factors need to be present for EPN to develop [15]. Huang et al. identified four key factors that play a determining role in the pathogenesis of EPN: presence of gas-producing bacteria, high tissue glucose levels, impaired tissue perfusion, and an altered immune response [12]. According to a meta-analysis by Desai et al. (2022), more than 80% of patients with EPN have DM, 16% have urolithiasis, and 20.5% suffer from obstructive uropathy [3]. In contrast, in the 1980s, it was believed that urinary tract obstruction accompanied EPN in at least 40% of cases, particularly in bilateral lesions or in cases where the only kidney was affected by EPN [7].

EPN involving native kidneys is more common in women, with a prevalence 1.8 to 6 times higher according to different studies. This is thought to be due to the anatomical characteristics of the female urogenital system. The left kidney is considered to be more vulnerable [5, 7, 12]. Interestingly, despite the higher incidence of EPN in women, men tend to experience a more unfavorable outcome (as highlighted in a meta-analysis by Ngo et al., 2022) [4]. Other risk factors for an unfavorable outcome in EPN include: signs of developing distributive shock (such as hemodynamic instability on admission, confusion, and the need for pressor therapy), confusion despite stable hemodynamic indices, laboratory parameters indicating the intensity of systemic inflammatory response, and secondary disorders of hemostasis and acid-base balance (initial thrombocytopenia, hypoalbuminemia, hyponatremia, hyperlactatemia, metabolic acidosis), AKI, and the extent of gas expansion as seen on CT imaging [1, 4, 12, 16, 17].

Approaches to diagnosis

In the 1930s and 1940s, early reports suggested the possibility of visualizing renal gas through radiography [15]. Currently, the primary method for diagnosing EPN is native computed tomography (CT). This method not only helps to identify the presence of gas but also allows for the assessment of its extent. The radiological classification proposed by Huang and Tseng in 2000 [12] is widely recognized as the best method for classifying EPN. According to this classification, EPN is divided into four classes based on the presence of gas in the collecting system, renal parenchyma, peri- and paranephric spaces, and whether one or both kidneys are involved (Table 1).

It is important to note that the renal fascia, with its anterior leaflet known as Gerota's fascia, encircles the kidney along with the surrounding fatty tissue, dividing the retroperitoneal space into two areas: the perinephric space (located within the renal fascia) and the perinephric space (located outside it).

Ultrasound (US) imaging has limited sensitivity for visualizing renal gas in patients with EPN. The primary ultrasound indicator of gas within the renal parenchyma and collecting system is the presence of linear hyperechogenic foci of varying sizes, often accompanied by distal reverberations. The characteristic "dirty shadow", which is a type of distal acoustic shadow, helps differentiate gas accumulation from a renal nodule. In some cases, the movement of these hyperechogenic gas foci within the collecting system, as the patient changes body position, can assist in distinguishing them from nodules [18].

An important indirect sign of gas presence in the perirenal space is the disappearance of renal visualization, which is particularly noticeable during KT ultrasound. However, it is crucial to note that ultrasound has a low sensitivity for diagnosing EPN, meaning that the absence of ultrasound signs does not exclude the diagnosis of EPN.

Table 1

Radiological classification of EPN (Huang-Tseng, 2000 [12])

Class	Gas detection zone
Class 1	Gas in the collecting system only
Class 2	Gas in the renal parenchyma with no extension beyond the organ
Class 3A	Extension of gas or abscess to perirenal space
Class 3B	Extension of gas or abscess to pararenal space
Class 4	Bilateral EPN or solitary kidney with EPN

Treatment approaches for emphysematous pyelonephritis

Over the past two decades, treatment approaches for EPN have evolved significantly. Nephrectomy as the first-choice strategy has given way to minimally invasive interventions, such as percutaneous nephrostomy (PCN), ureteral stenting, and abscess drainage, all in conjunction with aggressive antibiotic therapy (ABT) [1].

This shift toward less invasive tactics is largely driven by the high mortality rates associated with emergency nephrectomy. According to a meta-analysis by Desai et al. (2022), which included data from 1146 patients (1980–2020), the cumulative mortality rate for EPN was 12.5%. However, the mortality rate specifically for those undergoing emergency nephrectomy was significantly higher at 27% [3].

The choice of empirical ABT for EPN is a complex process that requires careful consideration of several factors. These include the risk of infection with multidrug-resistant bacteria, the patient's specific prognosis, and changes in drug pharmacokinetics, especially in cases of hypoalbuminemia or critical conditions. Previously recommended third- and fourth-generation cephalosporins, as well as fluoroquinolones, are no longer as effective due to the widespread resistance of Enterobacterales bacteria producing extended-spectrum beta-lactamases, which are common pathogens in UTIs, including EPN [8, 9]. Therefore, carbapenems from Group 2 and "new" inhibitor-protected cephalosporins remain the most appropriate choices for initiating therapy [19–21].

An attempt to algorithmize the management of EPN patients was made by Huang and Tseng in 2000. They analyzed the course and outcomes of EPN in 48 patients and identified thrombocytopenia, AKI, shock, and impaired consciousness as key risk factors. According to their algorithm, patients in grade 1 or 2 should receive ABT and PCN. For grade 3 or 4 patients with one risk factor, ABT and PCN are still indicated, but if two or more risk factors are present, nephrectomy should be considered [12]. This algorithm has been widely adopted in clinical practice; however, in light of modern resuscitation strategies that have evolved over the last 25 years, we believe that the approach, especially regarding risk

factors, may need to be updated. To this day, determining the optimal therapeutic strategy for EPN remains a subject of ongoing debate.

Emphysematous pyelonephritis involving the renal graft

We identified 38 cases of EPN in KT recipients published in the English-language medical literature from the late 1970s to the present day [22–58]. The characteristics and course of EPN in KT recipients, based on this literature analysis and our current observation, are summarized in Table 2.

An analysis of the data in the table shows a cumulative mortality rate of 28%. Among KT recipients, males were predominant (59%), and the age range was from 12 to 76 years, with a mean age of 51 ± 14 years. In the fatal cases, there was a clear male predominance (n = 9, 82%), compared to male representation (n = 14, 50%) in the surviving group. The mean age of surviving versus dead patients was not significantly different, at 49 ± 15 *vs.* 56 ± 12 years (p = 0.17). DM was present in 82% of cases, and PTDM developed in 9 patients (23%).

Failure to achieve glycemic and glycated hemoglobin targets was a common finding among KT recipients who developed EPN. A notable anamnestic risk factor was the presence of recurrent UTIs, observed in 35% of cases. Unlike the general population with EPN in native kidneys, obstructive uropathy was rarely reported among KT recipients.

Several isolated reports have linked the onset of EPN in KT recipients with urologic or angiographic procedures performed shortly before the disease debut, particularly among DM-free patients. For example, Althaf et al. [47] described a case of EPN following transurethral resection of the prostate. Boltan et al. [38] attributed the development of EPN to bladder catheterization, identifying it as an iatrogenic trigger. Salehipour et al. [41] reported the rapid onset of EPN and graft loss in a patient who underwent renal artery stenting while febrile. A notable case was also presented by Spanish researchers, who diagnosed EPN three weeks after renal artery embolization in a non-functioning KT [35].

The most common presenting symptoms were fever (76%) and abdominal pain (58%), typically localized to the graft area, though in some cases the pain was diffuse or associated with palpable tension over the transplant site. Confusion was reported in 30% of patients, while oliguria or anuria occurred in 28%. Gastrointestinal symptoms such as nausea or vomiting were present in 20%, whereas diarrhea or constipation were documented only sporadically.

A rare but notable case involved the simultaneous occurrence of EPN in the kidney transplant and both native kidneys [58]. Additionally, EPNs affecting non-functioning grafts have been reported in three observations [35, 51, 53], highlighting the diagnostic challenge of distinguishing between non-functioning kidney graft intolerance syndrome and infectious complications, as both may present with similar clinical features [59]. Although comprehensive laboratory data were often lacking, leukocytosis with a neutrophilic shift was commonly observed, suggesting a significant systemic inflammatory response in many cases.

Data on the causative pathogen were available for 33 out of 39 patients. *E. coli* was the most frequently detected organism, isolated in 20 cases (60.6%) from urine and/or blood cultures. In two of these cases, *E. coli* was found in combination with *Klebsiella pneumoniae* and *Staphylococcus epidermidis*. *K. pneumoniae* alone was detected in 7 patients (21.2%). Less commonly reported pathogens included *Bacteroides* species (2 cases), *Enterobacter* (1), *Salmonella* in combination with *Enterobacter* (1), *Proteus* species (1), and *Candida glabrata* (1).

IST is a clear predisposing factor for EPN in KT recipients. The time interval from transplantation to onset of EPN – effectively the duration of IST – ranged widely from 2 weeks to 11 years. However, the influence of specific IST regimens or the duration of immunosuppression on the risk of developing EPN remains uncertain. Interestingly, a recent case reported the development of severe fungal EPN necessitating transplantectomy just one week after initiation of empagliflozin, likely triggered by drug-induced glycosuria [55].

AKI was reported in 26 patients (67%), including the present case. The development of oligo/anuria due to AKI at the onset of EPN is characteristic in KT recipients, as the infection typically involves the only functioning kidney. In contrast, AKI in EPN affecting native kidneys is less common and usually occurs in cases of bilateral involvement or in patients with a solitary native kidney.

Due to the lack of consistent reporting in the reviewed cases, it is not possible to reliably assess the impact of body mass index (BMI) on the clinical course and prognosis of EPN in KT recipients. However, our patient was morbidly obese, which likely contributed to challenges in maintaining adequate personal hygiene.

Instrumental diagnosis of EPN in KT recipients requires specific consideration. It should be emphasized that in seven cases, KT ultrasound either revealed or suggested the presence of gas in the parenchyma or collecting system. Despite this, ultrasound remains the primary method for diagnosing EPN in KT recipients.

As the number of documented cases of EPN in KT recipients increased, it became clear that the radiological classification system proposed by Huang and Tseng had limitations. First, this classification automatically categorizes EPN in KT as grade 4, since the infection typically affects only a single kidney. Second, the classification's division of the disease into peri- and paranephric spaces is only applicable in native kidneys, where Gerota's fascia is present.

Table 2

Publications on clinical cases of EPN in renal allografts (1977–2024)

Author, publication year	Age, sex	DM	Uncontrolled DM	RUTIs	Clinical presentation on admission	Causative agent	Gas distribution on admission	Treatment	Outcome
Parameswaran et Feest 1977 [22]	53, f	Yes (PTDM)	N/A	No	Low-grade fever, pain around the KT, AKI, confusion	*Proteus spp.*	KT	TE + ABT	Alive
Brenbridge et al. 1979 [23]	33, m	Yes (PTDM)	N/A	No	Low-grade fever, pain around the KT, AKI	*E. coli*	KT + perirenal space	TE + ABT	Alive
Balsara et al. 1985 [18]	32, m	No	-	No	Fever, confusion	*E. coli*	KT + RCS	PD + ABT	Alive
Potter et al. 1985 [24]	31, f	Yes	N/A	Yes	Fever, pain around the KT, AKI	*E. coli*	KT + perirenal space	TE + ABT	Alive
O'Donnell et al. 1985 [25]	27, m	Yes	N/A	N/A	Fever, tension in the KT area	*Enterobacter spp*	KT + perirenal space	ABT	Alive
Glen et al. 1989 [26]	66, f	Yes	N/A	N/A	Fever, confusion	*E. coli*	N/A	PD + ABT	Alive
Kalra et al. 1993 [27]	35, m	No	-	N/A	Painful urination	*K. pneumoniae*	N/A	TE + ABT	Dead
Akalin et al. 1996 [28]	62, m	Yes	N/A	N/A	Painful urination, confusion	*K. pneumoniae*	RCS	ABT	Alive
Cheng et al. 2001 [29]	55, m	Yes (PTDM)	No	No	Fever, pain around the KT	*E. coli*	KT	PD + ABT	Alive
Iqubal et al. 2004 [30]	39, f	Yes (PTDM)	No	Yes	Fever, abdominal pain, AKI, confusion	*E. coli*	KT + perirenal space	PD + ABT	Alive
Ishigami et al. 2004 [31]	67, f	Yes (PTDM)	No	No	Low-grade fever, pain around the KT	Not detected	RCS	TE + ABT	Alive
Al-Makadma et Al-Akash 2005 [32]	12, m	No	-	Yes	Fever, vomiting, abdominal pain, tension in the KT area, AKI	*E. coli*	RCS	ABT	Alive
Fujita et al. 2005 [33]	49, f	Yes	Yes	No	Fever, pain around the KT, blood in urine, AKI, confusion	*Salmonela spp.* + *Enterobacter spp.*	KT + perirenal space	TE + ABT	Alive
Arai et al. 2006 [34]	61, m	Yes	N/A	N/A	Abdominal pain, AKI, confusion	*E. coli*	KT + perirenal space	TE + ABT	Dead
Ortiz et al. 2007 [35]	40, m	No	-	No	Fever, abdominal pain	*Bacteroides capillosus*	KT + RCS	TE + ABT	Alive
Chuang et al. 2007 [36]	51, m	Yes (PTDM)	Yes	No	Fever, abdominal pain	*E. coli*	RCS	PD + ABT	Alive
Baliga et al. 2007 [37]	52, f	Yes	No	Yes	Fever, pain around the KT, vomiting, AKI, confusion	*E. coli*	KT	ABT	Alive
Boltan et al. 2008 [38]	76, m	Yes	Yes	No	Fever, AKI	*K. pneumoniae*	KT + perirenal space	PD + TE + ABT	Alive
Debnath et al. 2009 [39]	52, f	Yes	N/A	Yes	Fever, abdominal pain, AKI	N/A	KT	ABT	Alive
Schmidt et al. 2009 [40]	55, m	Yes	N/A	No	Fever, abdominal pain, AKI	*E. coli*	KT + perirenal space	TE + ABT	Alive

End of table 2

Author, publication year	Age, sex	DM	Uncontrolled DM	RUTIs	Clinical presentation on admission	Causative agent	Gas distribution on admission	Treatment	Outcome
Salehipour et al. 2010 [41]	23, f	No	-	No	Fever, nausea, vomiting, blood in urine, pain around the KT, AKI	N/A	KT + perirenal space	TE + ABT	Alive
Al-Geizawi et al. 2010 [42]	58, f	Yes	Yes	No	Fever, nausea, vomiting, AKI, confusion	*K. pneumoniae*	KT	PD + ABT	Alive
Alexander et al. 2012 [43]	51, f	Yes (PTDM)	No	Yes	Fever, abdominal pain, vomiting, AKI, confusion	*K. pneumoniae*	KT + perirenal space	PD + ABT	Alive
Tsai et al. 2012 [44]	46, m	Yes	N/A	No	Fever, pain on palpation of KT	*E. coli*	KT	ABT	Dead
Agreda Castaneda et al. 2014 [45]	74, f	Yes	Yes	No	Fever, AKI	*E. coli*	KT	TE + ABT	Alive
Tienza et al. 2014 [46]	53, m	Yes	Yes	Yes	Low-grade fever, weakness, AKI	*S. epidermidis* + *E. coli*	KT + RCS	PD + ABT	Alive
Althaf et al. 2014 [47]	71, m	No	-	Yes	Fever, abdominal pain, vomiting, AKI, confusion	*E. coli*	KT + perirenal space	ABT	Dead
Narcisse et al. 2016 [48]	62, f	Yes (PTDM)	No	No	Fever, abdominal pain, diarrhea, AKI	*K. pneumoniae*	KT	TE + ABT	Alive
Alhajjaj et Pasha 2016 [49]	71, m	Yes	N/A	N/A	Shortness of breath, constipation, vomiting, tension in the KT area, AKI	N/A	KT + perirenal space	ABT	Dead
Oliveira et al. 2016 [50]	58, m	Yes	N/A	Yes	Fever, weakness	*E. coli* + *K. pneumoniae*	KT + perirenal space	PD + TE + ABT	Dead
Bansal et al. 2016 [51]	60, m	Yes	Yes	No	Fever, abdominal pain	*Bacteroides*	KT	TE + ABT	Dead
Crouter et al. 2017 [52]	61, m	Yes	Yes	No	Fever, shortness of breath, AKI, confusion	N/A	KT	ABT	Alive
Rajaian et al. 2019 [53]	44, m	Yes	Yes	Yes	Fever, tension in the KT area	*E. coli*	2 KT + perirenal space	TE + ABT	Alive
Ambinder et al. 2021 [54]	51, m	No	-	No	Fever, weakness, AKI	N/A	KT	PD + TE + ABT	Dead
Cases-Corona et al. 2022 [55]	53, m	Yes	No	Yes	N/a	*Candida glabata*	N/A	TE + ABT	Alive
Abu Jawdeh et al. 2022 [56]	49, f	Yes	Yes	Yes	Normothermia, abdominal pain, AKI, confusion	*E. coli*	KT + perirenal space	TE + ABT	Alive
Hassanein et al. 2022 [57]	51, f	Yes	N/A	No	Worn out	*K. pneumoniae*	KT	TE + ABT	Dead
Chippa et al. 2022 [58]	71, m	Yes	Yes	Yes	On a ventilator from another facility	*E. coli*	KT + perirenal space	ABT	Dead
Trushkin et al. 2024	46, f	Yes (PTDM)	Yes	Yes	Fever, pain around the KT, AKI	*E. coli*	RCS	PD + TE + ABT	Dead

Note: DM, diabetes mellitus; PTDM, post-transplant diabetes mellitus; RUTIs, recurrent urinary tract infections; AKI, acute kidney injury; KT, kidney transplant; RCS, renal collecting system; PD, percutaneous drain; ABT, antibiotic therapy; TE, transplantectomy.

Table 3

EPN stages in renal allografts (Al-Geizawi, 2010 [42])

Stage 1	Gas in the collecting system
Stage 2	Gas in <50% of the renal parenchyma, with minimum extension to perirenal space, quickly controlled sepsis
Stage 3	Gas in >50% of the renal parenchyma or extensive spread to perirenal space or evidence of organ failure, or uncontrolled sepsis, or refractory shock

The absence of Gerota's fascia at the transplant site results in a more rapid spread of the purulent destructive process within the abdominal cavity in KT recipients.

In 2010, Al-Geizawi et al. proposed a revised classification system that accounts for the unique characteristics of KT, including an assessment of gas distribution in the allograft based on CT findings, as well as some clinical features of patients [42] (Table 3).

According to the authors, CT imaging plays a crucial role in the early detection and ongoing monitoring of EPN. Stages 1 and 2, as identified on CT, justify the use of minimally invasive, nephron-preserving surgical interventions, which serve as an alternative to emergency nephrectomy. Stage 3, however, necessitates more aggressive surgical management. Schmidt et al. recommend using the "pulmonary window" mode on CT to better visualize the true distribution of gas within the allograft parenchyma and surrounding tissues, [40].

However, an analysis of EPN outcomes in KT recipients, including both published cases and our own clinical experience, suggests that the patient's classification stage according to the Al-Geizawi et al. system does not necessarily correlate with the disease outcome. In our case, radiological findings indicated stage 1, which is typically associated with a favorable prognosis. Additionally, several studies have highlighted the presence of severe comorbidities in patients who experienced fatal outcomes. These included acute myocardial infarction in a patient with severe mitral valve disease [51], sudden cardiac death [49], fulminant hepatitis [34], and EPN in a patient with COVID-19 complicated by cryptococcal infection [58]. The exacerbation of underlying comorbidities in the context of EPN in KT recipients likely plays a key role in the onset of a fatal outcome, regardless of the radiological stage of the disease.

CONCLUSION

The analysis of literature data and our own clinical experience leads to several practical considerations for the structured management of patients with EPN in KT recipients.

1. An optimal management algorithm for patients with EPN in KT recipients has yet to be developed due to the limited number of published cases and data.
2. The presence of initial DM and ongoing IST significantly increases the likelihood of infection dissemination, leading to SIRS, multiple organ failure, and distributive shock.
3. The presence of gas in the allograft, regardless of its spread according to the Al-Geizawi et al. classification, represents a poor prognostic indicator. It negatively affects both graft survival and the overall clinical course of the disease.
4. Patients with EPN in KT recipients should be promptly transferred to the ICU, regardless of baseline hemodynamic status, renal function, or acid-base balance, to initiate comprehensive intensive therapy, considering the patient's comorbid background.
5. The decision regarding the volume and sequence of surgical interventions (PCN and transplantectomy) should be carefully individualized, guided by a multidisciplinary team approach.

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