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# Successful Conservative Treatment of Extensive Emphysematous Pyelonephritis Due to Extended-Spectrum B-Lactamase-Producing *Klebsiella Pneumoniae* in a Renal Allograft Patient

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## Abstract

Emphysematous pyelonephritis is a rare, life-threatening complication that can occur in poorly controlled diabetic patients after renal transplantation, and may require nephrectomy. We report a case of extensive emphysematous pyelonephritis due to Extended-Spectrum β-Lactamase (ESBL)-producing Klebsiella pneumoniae in a renal allograft patient, successfully managed by antibiotics and percutaneous drainage. Early detection of emphysematous pyelonephritis in renal transplant patients with diabetes presenting acute pyelonephritis is crucial. Antibiotics against ESBL enterobacteriaceae could be interesting in first line in these pyelonephritis.

**Keywords: Emphysematous pyelonephritis; Extended-spectrum β-lactamase-producing *klebsiella pneumoniae*; Renal allograft**

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## Introduction

Emphysematous Pyelonephritis (EPN) is an acute severe bacterial or fungal necrotizing infection of the renal parenchyma and perirenal tissue, which results in the presence of gas within the renal parenchyma, collecting system, or perinephric tissue [1], 96% of cases occur in poorly controlled diabetic patients [2]. Urinary tract obstruction and immunosuppression are other predisposing factors. EPN is caused by gas-forming uropathogens, most frequently *Escherichia coli* (*E. coli*) and *Klebsiella pneumoniae* (*K.pneumoniae*) [1]. Prognosis is poor and emergency nephrectomy may be required [1]. Some authors describe a mortality rate of more than 80% [1]. We describe here a case of a renal transplant patient who presented several poor prognostic factors of EPN (renal allograft, diabetes, EPN caused by extended-spectrum β-lactamase-producing klebsiella pneumoniae, an uric acute renal failure requiring dialysis, drowsiness) and whose progression was favorable without recourse to nephrectomy.

## Case Presentation

A 72-year-old man with diabetes received a renal allograft from a deceased donor in Mars 2015 for diabetic nephropathy. Comorbidities included high blood pressure and severe arterial disease. He was on triple immunosuppression with tacrolimus, prednisone and mycophenolate mofetil. He had been hospitalized 6 months earlier for Enterobacter cloacae osteitis treated by anti biotherapy and a trans-metatarsal amputation.

On admission, he presented an acute fever of 40°C with confusion, abdominal pain and vomiting. His vital signs were normal with tachycardia of 110 bpm. Acute kidney injury was detected with a serum creatinine of 2.44 mg/dl. His white blood cell count was12400/mm$^3$, albumin 17 g/L, CRP 169 ng/L. His HbA1C was 10.5%.

A Computer Tomography (CT) scan (Figure 1) of the abdomen revealed a hydroaeric bladder with parietal pneumatosis and numerous intraparenchyma air pockets in the renal graft, corresponding to stage 3 in the Al-Geizawi classification (Table1) [1]. There was no pyelocaliceal dilatation.

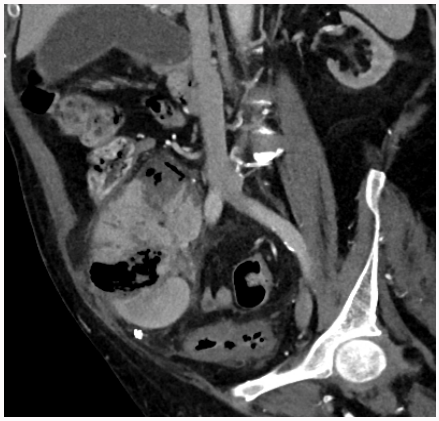

**Figure 1**:

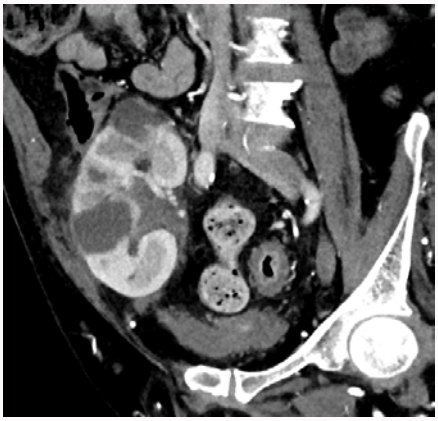

**Figure 2**:

An antibiotic treatment with ceftriaxone was administered. Urine and blood culture grew *K. pneumoniae* with extended-spectrum β-lactamase activity within 48h. The organism was gentamicin sensitive and ceftriaxone resistant. Therefore, it was decided to switch the antibiotherapy to imipenem and to administer one gentamicin shot. The immunosuppression treatment, tacrolimus and mycophenolate mofetil, were discontinued because of sepsis and septic encephalopathy.

Three days after admission, the patient became anuric with a serum creatinine of 4.40 mg/dl. Emergency hemodialysis was started. After three dialysis sessions, diuresis was observed but the patient remained in a feverish state despite the improvement of renal function. Seven days after admission we increased the dose of imipenem with no effect. It was decided to perform a percutaneous catheter drainage (9 days after admission). The insertion of perinephric and intranephric drains together with the antibiotherapy led to a clinical improvement of the patient. A CT scan performed 4 days after percutaneous drainage showed less emphysema in the graft with no collection of gas. Tacrolimus and mycophenolate mofetil were reintroduced on day 15 after admission. Imipenem was continued for a total of 7 weeks. The drain was removed 19 days after insertion.

Another CT scan (Figure 2) performed 9 weeks after admission, showed that the triangular opacity and aeric pockets had disappeared and that the medio-nephric collection had decreased. The patient had fully recovered his renal function 60 days after admission.

## Discussion

We describe the successful medical management of an elderly diabetic patient with a renal allograft who developed extensive EPN due to ESBL-producing *K. pneumoniae*. This case report illustrates the feasibility of first-line antibiotherapy for this life-threatening infection [1].

We have previously published a study of patients who developed Acute Pyelonephritis (APN) after renal transplantation [3]. Out of 172 consecutive renal allograft patients followed for 5 years, 25 (18.7 %) patients had 41 episodes of APN, none with EPN. Multivariate analysis identified that independent risk factors associated with APN were: female recipient (16 women/25: 64%); episodes of acute rejection; and the number of urinary tract infections [3]. Diabetes did not emerge as an independent risk factor. E. coli was the main uropathogen, being responsible for about half of the cases of APN with *K. pneumoniae* observed in only about 3 % of the cases [3].

**Table 1:** EPN of renal allograft according to Al-Geizawi et al. [1].

| | |
|---|---|
| Stage 1 | Gas in the collecting system |
| Stage 2 | Gas replacing <50% of renal parenchyma, with minimum spread to the surrounding tissues<br>Sepsis rapidly controlled |
| Stage 3 | Gas replacing >50% of renal parenchyma, or extensive spread of infection in the perinephric area, or patient with evidence of multiple organ failure, uncontrolled sepsis, or shock not responding to medical management |

Whereas APN is a common post-transplantation complication, EPN is a rare occurrence in renal allografts: only 24 cases (including the current report) of EPN have been published in the English language literature [4]. Diabetes emerges as a major risk factor of EPN involving 20 of the 24 published cases, and particularly poorly controlled diabetes [4]. The time from renal transplantation to onset of EPN ranges from 2 weeks to 12 years with a male predominance (61%). The main uropathogens are E. Coli (45.8%) and *K. pneumoniae* (25%) in the 24 published reports. The presence of necrotic tissue with a high glucose concentration provides the ideal environment for such bacteria to ferment glucose to lactose, carbone dioxide and hydrogen. The lack of Gerota's fascia around the allograft may also facilitate spread, leading to higher grades of sepsis.

EPN was first described in native kidneys [5]. Following a study of 48 patients with native EPN, Huang and Tseng proposed a clinicoradiological classification of four classes corresponding to prognostic severity [5]. Class 4 includes bilateral EPN or EPN in a solitary kidney which means that EPN in a renal allograft is included in this most severe class. Al Geizawi went on to publish a specific classification for allograft recipients in 2010 (Table 1) which recommends allograft nephrectomy for stage 3 disease [1].

Among the 24 published cases of allograft EPN, four were Al Geizawi stage 1-2 and eight were stage 3. Overall, 12 were treat by allograft nephrectomy, eight underwent percutaneous drainage and four were treated by antibiotics only (all four were Al Geizawi stage 1-2). All the patients with stage 3 were treated by allograft nephrectomy except ours. We decided to postpone nephrectomy and perform percutaneous drainage as the patient's septic status and renal function showed signs of improvement with antibiotic treatment and drainage.

Another aggravating factor for the prognosis of EPN is infection by ESBL-producing *K. pneumoniae*. Espinar et al. demonstrated that diabetes mellitus is an independent risk factor which increases by 6

the risk of acquiring infections by extended-spectrum β-lactamase-enterobacteriaceae in renal transplant patients [6]. The other identified risk factors are delayed graft function, previous antibiotic exposure, antibiotic prophylaxis and relapsing urinary tract infections [6].

Our patient was diabetic, had received antibiotic prophylaxis and undergone antibiotherapy 6 months previously. Our first-line treatment option, ceftriaxone, also proved to be ineffective delaying administration of an effective antibiotherapy for ESBL-producing *K. pneumoniae* infection for 48 hours. All these negative prognostic factors would argue for first-line nephrectomy according to the Al Geizawi staging. The fact that he was successfully managed by an effective ESBL enterobacteria antibiotic treatment, incites us to question nephrectomy as first-line treatment in a renal allograft patient who presents EPN.

In conclusion, post-renal transplantation APN can be complicated by a rare but life-threatening entity – EPN – particularly in poorly controlled diabetic patients. In these patients, a prompt CT scan should be performed at the first signs of increased severity. Once diagnosed, EPN can be managed conservatively in a hemodynamic stable patient even in severe cases of allograft EPN. First-line treatment should consist of an effective ESBL enterobacteria antibiotic before recourse to nephrectomy.

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