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A CLINICAL CASE OF RECURRENT RENAL TRANSPLANT PYELONEPHRITIS IN AN IMMUNOSUPPRESSED PATIENT

N.R. Akramov[1], A.N. Gainullov[2], K.V. Sitkina[2], M.E. Nagiev[2], M.I. Aliev[1]

[1]Kazan State Medical Academy (KSMA) – branch of the Federal State Budgetary Educational Institution of the Russian Ministry of Health
[2]State Autonomous Health Care Institution "Republican Clinical Hospital of the Ministry of Health of the Republic of Tatarstan", Kazan, Russian Federation

Summary. The article presents a clinical case of stage treatment of a 60-year-old patient with acute serous pyelonephritis of a kidney transplant detected 1 year and 5 months after the operation. The first stage in the patient's management tactics, after the clinical diagnosis was established, was empirical antibiotic therapy using broad-spectrum drugs. De-escalation of antibiotic therapy was performed based on the data of microbiological studies until complete elimination of the pathogen from the urine. Drug therapy also included hematopoiesis stimulators and proton pump inhibitors, which was determined by the presence of concomitant pathologies in the blood system and gastrointestinal tract of the patient.

***Keywords**: kidney transplantation, bacterial infection immunosuppression, infectious complications, urinary tract infection.*

Introduction. Urinary tract infections (UTIs) are among the most common problems in modern transplant surgery. According to several studies, these complications occur in 50–70% of patients [1, 2]. Notably, in 80% of cases, infectious complications develop within the first year post-transplantation, with the peak incidence occurring during the first month. Predisposing factors for UTI development include prior infections; recipient age and gender; metabolic disorders (specifically diabetes mellitus); a history of native kidney cysts, chronic pyelonephritis, or genitourinary tract anomalies; prolonged hemodialysis; long-term bladder catheterization; bladder atony; post-transplant urological complications (such as urinary fistulas due to anastomotic leakage, stenosis, or bleeding from the anastomosis site causing bladder tamponade; urinary fistulas resulting from ureteral necrosis; obstruction—including ureteral stricture, compression by a lymphocele, hematoma, or spermatic cord, ureteral kinking, or obstruction by blood clots or calculi); and graft dysfunction [1, 3, 4, 8–11].

In addition to the factors listed above, key determinants of the severity of clinical manifestations of infectious complications, as well as the duration and outcomes of hospitalization, include immunosuppressive therapy and infection of the recipient with immunomodulatory viruses–specifically, viruses of the genus *Cytomegalovirus*–and enterobacteria such as *Escherichia coli* and *Klebsiella pneumoniae* [5–7].

In recent years, the median age of patients undergoing kidney transplantation has reached 55 years. Due to the age-related decline in immunological reactivity, acute graft rejection occurs less frequently, whereas infectious complications are more common. These complications play a leading role among the causes of graft rejection [12, 13].

Thus, the primary focus of clinical practice aimed at reducing the risk of graft loss is adequate antimicrobial therapy, the management of urological complications, and the elimination of hyperimmunosuppression (upon its clinical and laboratory identification).

Clinical case. Patient A., 60 years old, was admitted to the kidney transplantation department of the Republican Clinical Hospital of the Ministry of Health of the Republic of Tatarstan complaining of general weakness, evening fevers reaching 38 °C, and a change in urine color to red. His medical history indicates that he has been ill since 2017, when lower-extremity edema developed; subsequent evaluation revealed chronic glomerulonephritis. He was followed by nephrologists at his place of residence. In 2020, his condition deteriorated; chronic kidney disease (CKD) was diagnosed, and maintenance hemodialysis was initiated three times a week. Dialysis tolerance was satisfactory, and there were no complications. In 2023, a kidney allotransplantation was performed, with the graft placed in the right iliac fossa (Fig. 1). Graft function was preserved, and the patient received immunosuppressive therapy with methylprednisolone, tacrolimus, and mycophenolic acid. There is no evidence of graft rejection episodes.

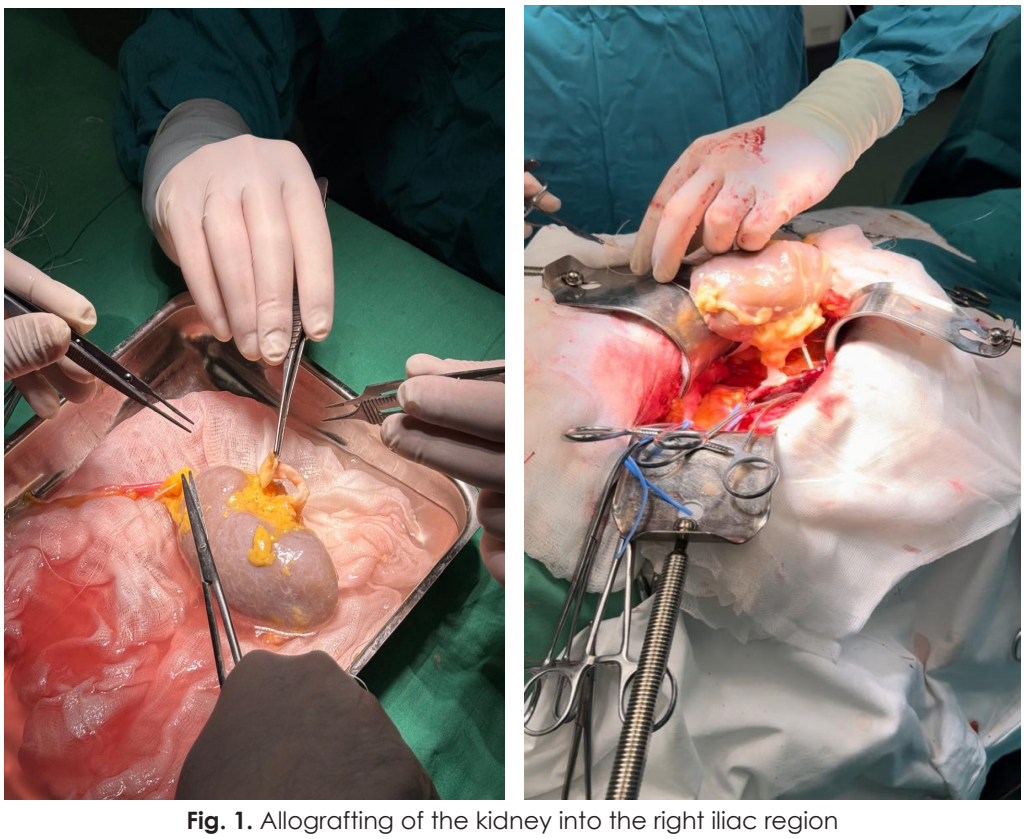


Fig. 1. Allografting of the kidney into the right iliac region

Computed tomography (CT) of the abdominal cavity and retroperitoneal space revealed renal abnormalities, specifically reduced kidney size, irregular contours, and parenchymal thinning. The pelvicalyceal systems of both kidneys were not dilated, and no calculi were detected. A cystic lesion measuring 45 × 50 mm was observed in the right kidney. The perirenal adipose tissue appeared streaky. A transplanted kidney was identified in the right iliac region; its renal pelvis was dilated to 13.0 mm, and the surrounding adipose tissue showed signs of infiltration and contained fluid layers (Fig. 2).

Renal ultrasound supplemented the clinical picture by revealing signs of focal infiltration within the kidney transplant, manifesting as three hypoechoic lesions–measuring 20×12 mm, 12 mm in diameter, and 11 mm in diameter–with an absence of blood flow in the affected areas. Chest CT scanning revealed signs of a destructive lesion in the upper lobe of the right lung and pericardial effusion. Echocardiography demonstrated left ventricular myocardial hypertrophy, left atrial enlargement, and grade I mitral and tricuspid regurgitation. Flow velocity in the left ventricular outflow tract was slightly elevated; other valvular flow velocities were within normal limits. Pericardial fluid was present.

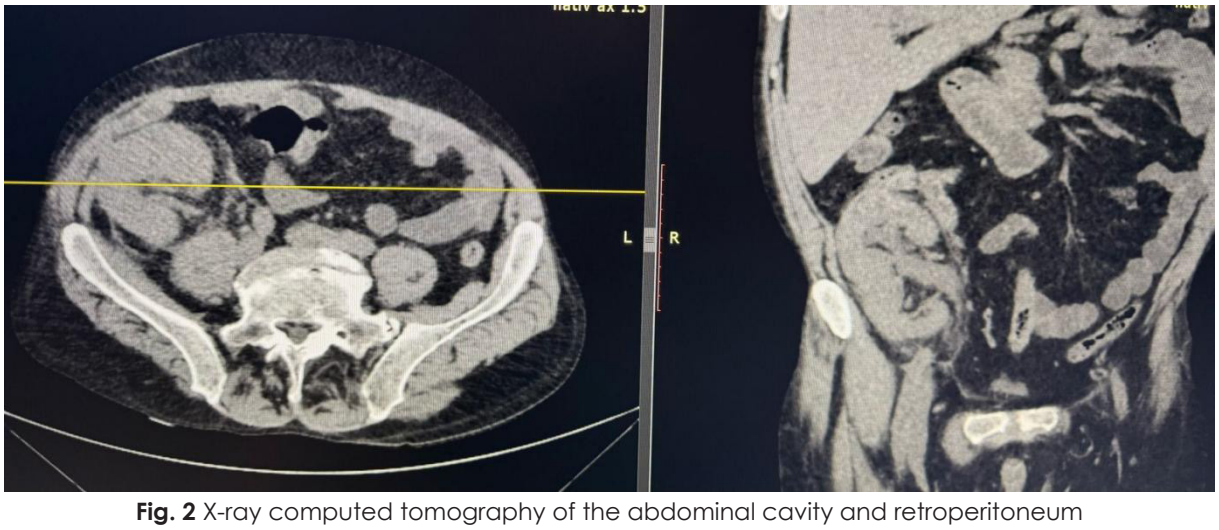


Fig. 2 X-ray computed tomography of the abdominal cavity and retroperitoneum

Changes in laboratory parameters were observed in the complete blood count, characterized by mild anemia and marked leukopenia, presumably of combined (bacterial and immunosuppressive) origin. A rapid COVID-19 test yielded a negative result. Molecular testing of blood for Epstein-Barr virus, herpes simplex virus types 1 and 2, and cytomegalovirus; testing for HIV-1/2 antibodies (IgM, IgG) and p24 antigen; testing for *Treponema pallidum* antibodies; testing for hepatitis B and C viral antigens; and blood culture for sterility (including antibiotic susceptibility testing) all yielded negative results. Urinalysis revealed decreased specific gravity, turbidity, a high number of leukocytes per high-power field, and a small number of bacteria per high-power field.

Laboratory and instrumental examination findings:

Microbiological data: 09–13.01.25. Analyzed specimen: Urine (single sample). Urine culture: growth detected from enrichment broth (CFU/mL). Organisms isolated: 1. *Enterococcus faecium*. Antibiotics/*Enterococcus faecium* isolates: Ampicillin R / 16 mcg/mL; Ciprofloxacin R / >4 mcg/mL; Gentamicin (high-level) R / >128 mcg/mL; Imipenem R / >256 mcg/mL; Vancomycin S / <4 mcg/mL

Recommendations: Antibiotic therapy adjustment: Pharmacotherapy: . Treatment regimen: Ceftriaxone/sulbactam 1.5 g IV bolus twice daily (15 vials) + Linezolid 600 mg IV bolus twice daily (15 vials); Bacterial cultures: blood culture at peak fever; Monitoring treatment efficacy:: after 48–72 hours: CBC with differential, creatinine, CRP, INR, urinalysis.

Laboratory and instrumental test results (as of Jan 21, 2025) show positive dynamics: CRP 107.7 → 2.8 mg/L, creatinine 195 → <... μmol/L. BUT Hb is 79! K is 5.6!!!

Microbiological data: 19(20)–22.01.25. Analyzed specimen: Spot urine sample. Urine culture: No microflora detected (CFU/mL).

Recommendations: Correction of anemia; continue antibiotic therapy. Pharmacotherapy: Ceftriaxone 1.5 g IV bolus twice daily (12 vials) + Linezolid 600 mg IV bolus twice daily (12 vials) – continue. Bacterial cultures: repeat. Monitoring treatment efficacy: Every 48–72 hours – CBC with differential, creatinine, CRP, INR, urinalysis.

Thus, based on the examination and a medical consultation, the patient was assigned the following clinical diagnosis: "Chronic kidney disease, stage 5; status post-kidney allotransplantation; chronic glomerulonephritis; exacerbation of chronic transplant pyelonephritis." The pharmacological treatment regimen included hematopoietic stimulants, oxazolidinones, proton pump inhibitors (prescribed due to concomitant erosive gastritis), and combination cephalosporin therapy (Table 1).

Upon discharge, the patient is advised to follow a specific diet and undergo monthly follow-ups with a general practitioner regarding acute kidney injury for the first three months, followed by annual check-ups. Scheduled laboratory tests are required. Medications: Metipred 8 mg orally once daily; Tacrolimus 2 mg orally once daily. Mycophenolic acid is to be discontinued.

Discussion. This clinical case highlights the need to develop personalized management algorithms for transplant recipients that account for both pathogen resistance profiles and the patient's individual metabolic characteristics. Managing urinary tract infections in kidney transplant recipients is a key issue in modern transplant medicine, given the high risk of graft loss associated with such infections. Active research is currently underway to establish principles for creating individualized treatment regimens and long-term monitoring strategies, with the aim of reducing the risk of graft loss and improving patient quality of life. Equally important is the preventive aspect: educating patients on hygiene and the early recognition of infection symptoms, as well as implementing regular screening for asymptomatic bacteriuria—a condition that frequently serves as a predictor of complications

Table 1. Patient's medication regimen

Name of the drug	Pharmacological group	Dosage used
Ferinject	Parenteral iron preparation (hematopoiesis stimulator)	2 ml once daily, single intravenous injection
Darbestim	Hematopoiesis stimulant	0.3 mL once daily, subcutaneously as a single dose.
Linezolid-Advanced	Oxazolidinones	600 ml twice daily, IV, daily until discontinuation
Esomeprazole	Proton-pump inhibitors	40 mg once daily, IV, until discontinuation.
Ceftriaxone + Sulbactam + Sodium chloride	Cephalosporin combinations	Twice daily, intravenously, slowly, for 7 days.

in this patient population. Integrating these approaches into clinical practice has the potential to significantly reduce the incidence of graft loss and improve long-term patient outcomes.

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