CASE REPORT

Acute severe pancreatitis following live donor kidney transplant

V. Ravindran[1], N. Niranjan[1], T. Tharmalingam[1], B. Thangarajah[2], S. Matheivaanan[3], V. Satchithanantham[1]

[1]Department of Surgery, Faculty of Medicine, University of Jaffna, Sri Lanka
[2] Department of Medicine, Faculty of Medicine, University of Jaffna, Sri Lanka
[3] Teaching Hospital, Jaffna, Sri Lanka

Keywords: Acute pancreatitis, Kidney transplantation, Immunosuppressive therapy, Corticosteroids, Drug-induced pancreatitis

Introduction

Acute severe pancreatitis is a relatively rare but serious complication in the early postoperative period of kidney transplantation. [1] It presents diagnostic and therapeutic challenges due to the altered physiology of transplant patients and the use of high-dose immunosuppressive therapy. [1]

Case presentation

A 54-year-old male with a history of diabetic nephropathy developed chronic kidney disease, which progressed to end-stage renal disease (ESRD). He was maintained on hemodialysis for six months prior to undergoing a living donor renal transplantation. The patient was initiated on a standard triple immunosuppressive regimen for renal transplantation. Induction therapy was administered with basiliximab 20mg on Day0 and Day 4, tacrolimus 3.5mg bd, mycophenolate mofetil 500mg tds, and methyl prednisolone 500mg during induction of anesthesia. Immediate graft function observed postoperatively, as evidenced by prompt normalization of serum creatinine levels and satisfactory urine output.

On postoperative day 2, the patient developed reduced urine output and abdominal distension, without associated abdominal pain, refractory vomiting, or any clinically identifiable cause. Initial investigations were as follows, (Table 1)

NCCT abdomen was suggestive of acute pancreatitis (Fig.1), and a timely serum amylase test was requested, which returned elevated at 1696 U/L, thereby fulfilling the Atlanta criteria and confirming the diagnosis of acute severe pancreatitis.

At the time of diagnosis, the 2nd dose of methylprednisolone was already given, and it was decided to complete the induction with the second dose of basiliximab 20mg as per original induction regime.

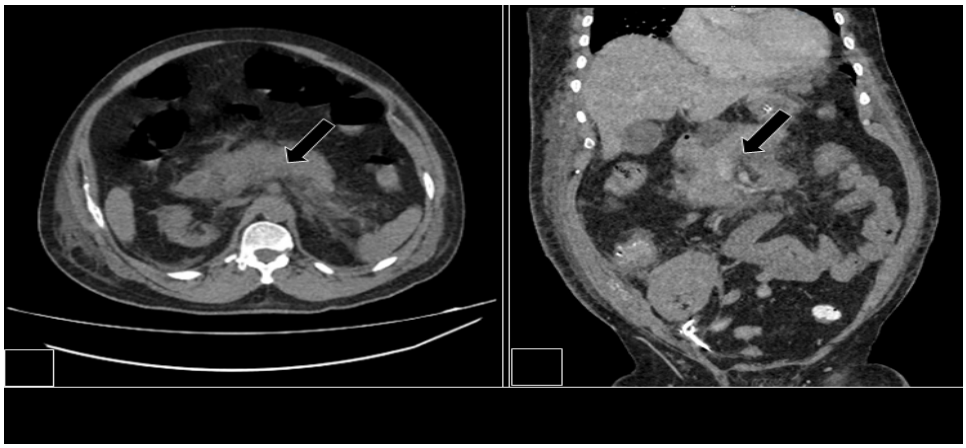

Figure 1: Non contrast CT demonstrating peripancreatic inflammation

Correspondence: V. Satchithanantham
E-mail:vinojan@univ.jfn.ac.lk
https://orcid.org/0000-0002-7172-997X
Received: 14-01-2026 Accepted: 29-03-2026
DOI: https://doi.org/10.4038/sljs.v44i1.9323

Table 1: Investigations

Investigations	Results
White blood cells	25.26×10^9/L
C-Reactive protein	143.6mg/dl
Serum amylase	1696U/l
Aspartate Aminotransferase	26 U/l
Alanine Aminotransferase	20 U/l
Serum creatinine	644 µmol/l
Blood urea	16.3 mmol/l
Serum Calcium	2.27 mmol/l
Ultrasound scan of the abdomen	Gaseous distention of abdomen, Midline structures obscured, Bowel loops not distended Minimal free fluid in the hepatorenal pouch noted Gallbladder distended and no calculus observed
Non- Contrast Computed Tomography- Abdomen & Pelvis (Figure 1)	Fat stranding around the pancreas, bilateral perinephric spaces, and the root of the mesentery. Minimal ascites and mild right-sided pleural effusion were noted. The transplanted kidney was in the right iliac fossa with two stents in situ and mild perinephric fluid collection.
High-Resolution Computed Tomography- Chest	Bilateral lower lobe consolidation with small pleural effusion
Upper Gastrointestinal Endoscopy	Evidence of recent bleeding and multiple gastric erosions has been seen.

The course was further complicated by probable acute tubular necrosis necessitating regular hemodialysis for 10 days till the improvement of graft function. Further, the patient developed hematemesis and upper gastrointestinal endoscopy revealed multiple gastric erosions, which were managed with proton pump inhibitors. Unfortunately, his condition deteriorated further, and the High resolution CT chest revealed bilateral lower lobe consolidation with associated pleural effusion. Sputum cultures grew *Acinetobacter* and *Klebsiella* species, confirming a diagnosis of hospital-acquired pneumonia (HAP) complicated with acute respiratory distress syndrome (ARDS).

The patient required endotracheal intubation and mechanical ventilation for four days. He was treated with a combination of Polymyxin, Teicoplanin, and Levofloxacin, alongside chest physiotherapy and postural drainage to support respiratory function and promote secretion clearance. Due to the severity of illness and complications, the patient had a prolonged Intensive Care Unit (ICU) stay, a total of 21 days. During the ICU stay low dose tacrolimus was continued with maintenance IV hydrocortisone of 50 mg 6 hourly and other immunosuppressants were withheld.

He recovered gradually over the course of 21 days from acute severe pancreatitis and its complications. Immuno suppressive therapy was reintroduced to a modified steroid free regime following discharge from ICU. At 45 days post-transplant, the patient's graft function recovered normally without further complications.

Discussion

The first reported case of acute pancreatitis post-renal transplantation dates to 1964. [2] The incidence among renal transplant recipients ranges from 2% to 7%, with a high mortality rate of 50% to 100%. [1] Given the multifactorial etiology of post-transplant pancreatitis, it is often difficult to pinpoint a single cause. Immunosuppressive agents, however, are a significant contributing factor. [1]

Drug-induced pancreatitis represents up to 5% of all acute pancreatitis cases. Diagnosing drug-induced pancreatitis (DIP) lacks definitive clinical or laboratory criteria. Instead, the diagnosis is often based on a combination of clinical observations: resolution of symptoms and normalization of pancreatic enzymes after stopping the suspected drug, exclusion of more common causes, and recurrence of symptoms if the drug is reintroduced. [3] However, re-introducing the drug is rarely feasible in critically ill patients, even though it can help narrow down the list of possible offending agents. Determining the true cause becomes especially challenging in patients on multiple medications, as excluding all other potential causes or drugs is difficult. [4]

The Badalov classification system categorizes drug-induced acute pancreatitis into five classes: Ia, Ib, II, III, and IV. This classification is based on several factors, including the number of published case reports, whether re-challenge with the drug led to recurrence of pancreatitis, and the latency period between drug exposure and symptom onset. In the Badalov classification system steroids fall into Ib category & Tacrolimus into category IV. [4]

Our patient had generalized abdominal distension from day 2 with features of paralytic ileus. He did not have any abdominal pain. Initially we proceeded with a Non-Contrast CT scan of the abdomen as there was a risk of contrast induced nephropathy. This picked up some fat stranding around the pancreas. According to the revised Atlanta classification we arrived at the diagnosis with radiological and biochemical components.

Due to the immunosuppressed state and altered physiology of transplant recipients, clinical symptoms of acute pancreatitis

may be subtle or atypical, as observed in this case. Therefore, early recognition through biochemical markers and imaging is essential. [1] Prompt diagnosis, appropriate supportive care, and careful adjustment of immunosuppressive therapy are key to improving patient and graft survival. [5]

Even after the resolution of acute pancreatitis, immuno suppressive agents remain a potential risk factor for recurrence. Minimizing corticosteroid exposure while maintaining adequate immunosuppression is crucial to prevent both pancreatitis and allograft rejection. [5]

Conclusion

Patients presenting with abdominal symptoms after renal transplantation should be evaluated for acute pancreatitis, even in the absence of typical symptoms. Early diagnosis and prompt management can improve both patient outcomes and graft survival.

References

1.Tabakovic M, Salkic NN, Bosnjic J, Alibegovic E. Acute pancreatitis after kidney transplantation. Case Reports in Transplantation. 2012;2012(1):768193.

2.Starzl TE. Experience in renal transplantation. WB Saunders company; 1964.

3.Mallory A, Kern Jr F. Drug-induced pancreatitis: a critical review. Gastroenterology. 1980 Apr 1;78(4):813-20.

4.Chadalavada P, Simons-Linares CR, Chahal P. Drug-induced acute pancreatitis: prevalence, causative agents, and outcomes. Pancreatology. 2020 Oct 1;20(7):1281-6.

5.Renning JA, Warden GD, Stevens LE, Reemtsma K. Pancreatitis after renal transplantation. The American Journal of Surgery. 1972 Mar 1;123(3):293-6.

Learning Points:

- Clinical symptoms of acute pancreatitis may be subtle or atypical due to the immunocompromised state in transplant patients.
- Diagnosing drug-induced pancreatitis is often based on a combination of clinical observations.
- Steroid-induced pancreatitis can happen even after a single high-dose exposure in transplant induction.