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Management of Acute Pulmonary Edema in Kidney Transplant Patients in the ICU

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ABSTRACT

Introduction: Kidney transplantation is the standard treatment for end-stage renal disease (ESRD), significantly improving survival rates and quality of life. However, pulmonary complications are a leading cause of morbidity and mortality post-transplant.
Case Report: This case report presents a 50-year-old male with chronic kidney disease (CKD), diagnosed five months prior and undergoing regular hemodialysis. He had a history of hypertension and diabetes mellitus. The patient underwent a 9-hour kidney transplant surgery without complications. Post-operatively, urine output was minimal (5 mL), prompting vasopressor support to elevate the mean arterial pressure above 150 mmHg, which improved renal function. On days one and two post-surgery, the patient developed respiratory distress, with a chest X-ray revealing pulmonary edema. Continuous furosemide infusion was initiated to manage fluid overload, leading to improvement in the patient's respiratory status. By day four, the patient was stable and transferred from the ICU to a regular room. Pulmonary complications, including pulmonary edema, affect up to 80% of kidney transplant recipients in the first year post-transplant and contribute to high morbidity and mortality.
Conclusion: This case emphasizes the importance of early recognition and management of pulmonary edema through fluid management and vasopressors. Timely intervention, including diuretic therapy, is crucial for stabilizing kidney transplant recipients and improving patient outcomes. The report highlights the need for further research to establish evidence-based guidelines for fluid management in kidney transplant patients. Effective management is essential for enhancing post-operative recovery and quality of life in transplant recipients.

Keywords Chronic Kidney Disease, Kidney Transplantation, Hypertension, Pulmonary Edema

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INTRODUCTION

Chronic Kidney Disease (CKD) is a prevalent medical condition that has a significant negative impact on the healthcare system. In 2019, the federal government signed an Executive Order titled "Advancing American Kidney Health", which, among its objectives, aimed to facilitate and promote kidney transplantation as the optimal modality for kidney replacement therapy [1-3]. Kidney transplantation is considered the best treatment option for patients with end-stage kidney disease (ESKD) because it improves the quality of life compared to dialysis and extends patient survival. For kidney transplant recipients, the survival rate one year post-transplant can exceed 95% [3].

Pulmonary complications are common after kidney transplantation, and a variety of infectious and non-infectious complications can be observed. Infectious complications significantly increase morbidity and mortality rates in these patients. Several risk factors contribute to the development of pulmonary

complications, including advanced age, smoking status, comorbidities, pre-transplant peritoneal dialysis, cadaveric kidney transplantation, heavy immunosuppression, and rejection treatment [1].

Intraoperative fluid management significantly affects the post-transplant outcomes. However, the amount and type of fluids administered, as well as the monitoring techniques, vary considerably across institutions, with limited prospective randomized trials and meta-analyses to guide fluid management in kidney transplant recipients [2].

Pulmonary complications may be exacerbated in patients with comorbidities such as hypertension and diabetes mellitus and those receiving immunosuppressive therapy (e.g. mycophenolate, azathioprine, or tacrolimus). Recognising these risk factors allows the identification of high-risk patient groups, enabling close monitoring to prevent post-transplant pulmonary diseases. This proactive approach can reduce the overall morbidity and mortality in kidney transplant recipients [4].

This case report presents the case of a kidney transplant patient who developed acute pulmonary oedema due to fluid overload and required treatment in the General ICU at Hasan Sadikin Hospital, Bandung. The patient was admitted to the ICU for four days, and on the second day, he experienced severe respiratory distress. Lung ultrasonography revealed a B-profile in both lung fields, which was confirmed by chest radiography and indicated pulmonary oedema. This was attributed to inadequate urine output. Continuous administration of furosemide was effective but required careful blood pressure management to maintain renal perfusion, particularly given the patient's preexisting chronic hypertension.

CASE REPORT

A 42-year-old man with a history of CKD managed with regular haemodialysis (HD) for 5 months presented to the emergency department (ED) with complaints of severe shortness of breath. The patient was diagnosed with diabetes mellitus (DM) and chronic hypertension, which was intermittently managed. He had a history of maximum blood glucose level of 350 mg/dL and hypertension (200/120 mmHg). The patient was referred for kidney transplantation with his wife as the donor. Pre-transplant evaluation and counselling were completed, and both the recipient and donor consented to the procedure.

Table 1. Chronological Status of the Patient in ICU

Day	GCS	Blood Pressure (mmHg)	Heart Rate (bpm)	Respiratory Rate (bpm)	SpO2 (%)	Oxygen Therapy	Fluid Balance (mL)
1	E4V5M6	194/78 (no support)	99–101	16–18	100	3L/min nasal cannula	-510
2	E4V5M6	160/66 (nicardipine 0.5 mcg/kg/min)	105	25–30	95	3L/min nasal cannula	+164
3	E4V5M6	164/71 (nicardipine 0.25 mcg/kg/min)	107	19–23	100	3L/min nasal cannula	-2195
4	E4V5M6	170/84 (nicardipine 0.5 mcg/kg/min)	111	16–18	99	3L/min nasal cannula	+500

Upon presentation to the ED, the patient was alert and oriented, with the following vital signs: blood pressure, 150/90 mmHg; heart rate, 98 beats/min; respiratory rate, 27 breaths/min; and oxygen saturation, 96% on room air. His body temperature was 37.6°C. Physical examination did not reveal conjunctival pallor or scleral jaundice in the patient. Chest auscultation revealed bilateral vesicular breath sounds without any additional sounds. Cardiovascular examination revealed regular heart sounds with no additional murmurs, and the abdominal examination was unremarkable, with normal bowel sounds.

Laboratory tests showed haemoglobin 8.9 g/dL, haematocrit 24.8%, leukocyte count 14,490/mm3, platelet count 304,000/mm3, urea 46.2 mg/dL, creatinine 2.81 mg/dL, sodium 135 mEq/L, potassium 3.4 mEq/L, chloride 99 mEq/L, and blood glucose 158 mg. Arterial blood gases (ABG) showed a pH of 7.453, pCO2 of 27.3 mmHg, pO2 of 107.1 mmHg, and HCO3 of 19.3 mEq/L. Chest radiography revealed cardiomegaly and aortic atherosclerosis. aorta. Electrocardiography (ECG) showed sinus rhythm at 100 beats

per minute, and echocardiography revealed a dilated left ventricle (LV) with concentric LV hypertrophy (LVH), borderline left ventricular systolic function (LVEF 53%), and mild mitral regurgitation (MR). No evidence of pulmonary hypertension (PH) or right ventricular dysfunction was observed.

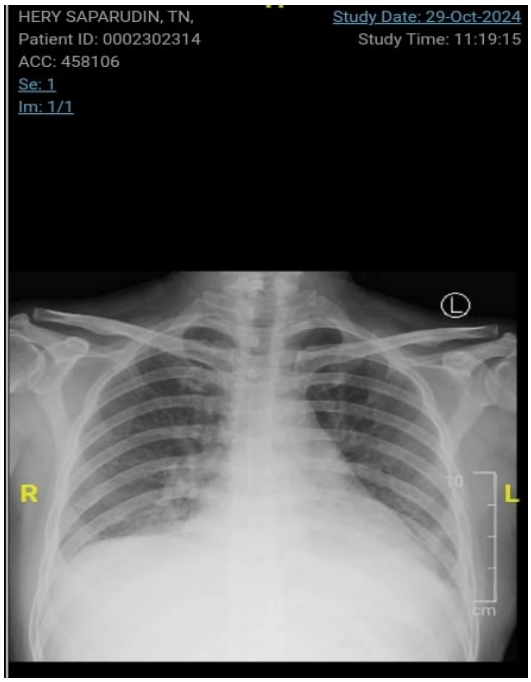

Figure 1. Chest X-ray of the patient before surgery

The patient underwent kidney transplantation, with his wife donating her organ. The surgery, which lasted 5.5 hours, was performed under general anaesthesia with appropriate intraoperative monitoring. Preoperative anaesthesia assessment classified the patient as ASA III owing to comorbidities such as CKD, hypertension, and diabetes. Intraoperative monitoring included noninvasive blood pressure (NIBP), electrocardiography (ECG), oxygen saturation (SpO2), temperature, central venous pressure (CVP), end-tidal CO2 (EtCO2), arterial line, and bispectral index (BIS). Anaesthesia was induced using fentanyl, propofol, and rocuronium, and endotracheal intubation was performed. A central venous catheter (CVC) and arterial line were placed, and a quadratus lumborum (QL) block was administered for regional anaesthesia.

Intraoperative fluid management included 1,000 mL of crystalloid resuscitation, and 400 mL of blood loss was replaced intraoperatively. The patient's urine output was minimal during surgery, and renal Doppler ultrasonography revealed inadequate renal perfusion. Subsequently, norepinephrine and dobutamine were administered as vasopressors to elevate the systolic blood pressure above 150 mmHg, resulting in improved renal perfusion and improved urine output. The patient was extubated postoperatively and transferred to the ICU for close monitoring of the haemodynamic parameters.

The patient was admitted to the ICU post-transplantation on 3 November 2024 with stable haemodynamics but requiring vasopressor support (dobutamine 5 µg/kg/min and norepinephrine 0.1 mcg/kg/min) to maintain a mean arterial pressure (MAP) of 110 mmHg. His heart rate was 101 beats per minute, respiratory rate was 22–24 breaths per minute on a simple mask at 6 L/min, and oxygen saturation was 96–98%. On the second postoperative day, the patient developed respiratory distress, and lung ultrasonography revealed a B-profile in both the lung fields. Chest radiography confirmed the diagnosis of pulmonary oedema, and the patient was treated with continuous furosemide infusion.

As part of the management strategy, the fluid balance of the patient was closely monitored and maintained. Diuresis was targeted at 1 mL/kg/h, and further fluid resuscitation was guided by urine output and haemodynamics. A significant improvement in respiratory status was noted, and the patient’s blood pressure was managed with nicardipine to maintain a target systolic pressure of 160 mmHg.

Pulmonary complications, such as pulmonary oedema, are common in kidney transplant recipients and can be exacerbated by fluid overload, pre-existing comorbidities such as hypertension and diabetes mellitus, and intraoperative management. In this case, early identification and management of fluid overload through furosemide administration and careful haemodynamic monitoring were crucial for stabilising the patient. The importance of maintaining an optimal fluid balance, especially in high-risk patients, is crucial.

Upon admission to the ICU, patient management followed the FAST HUG BID protocol, which included feeding, analgesia, sedation, thromboprophylaxis, head-up position, ulcer prophylaxis, and glucose control. Initially, the patient underwent gastric lavage, followed by test feeding. For pain management, tramadol was administered via a syringe pump, with additional doses of paracetamol and continuous bupivacaine for the nerve block. To manage his blood glucose, insulin was infused at a rate of 6 U/h with hourly glucose monitoring, aiming for a target range of 140-180 mg/dL. Fluid balance and diuresis were carefully monitored, with a urine output of 1 cc/kg/h maintained.

The patient was also prescribed cefazolin for infection prophylaxis, and continuous haemodynamic support with dobutamine and norepinephrine was administered. Fluid therapy was adjusted to maintain a negative fluid balance, and regular assessments, including echocardiography and kidney ultrasonography, were performed to monitor the potential complications.

Table 2. Laboratory Results and Antibiotics Administered

Day	Hemoglobin (g/dL)	Leukocytes (mm3)	Urea (mg/dL)	Creatinine (mg/dL)	Glucose (mg/dL)	Troponin I (ng/mL)	BNP (pg/mL)	Meropenem (g)
H0	8.9	14,490	46.2	2.81	158	0.56	8171	1
H1	7.3	18,730	107.3	4.3	142	0.98	-	1
H2	7.6	20,410	101.5	3.57	115	-	-	1
H3	7.1	17,000	123.9	2.73	174	-	-	1
H4	7.3	13,830	137	2.28	189	-	-	1

Progress and Complications

On the first night in the ICU, the patient's blood pressure increased despite the discontinuation of vasopressors. Nicardipine was introduced to achieve a target mean arterial pressure of 150-160 mmHg. On the second day, chest radiography revealed cardiomegaly and pulmonary oedema, prompting further investigation. Lung ultrasonography revealed bilateral B-profile changes, which indicated pulmonary oedema. Furosemide was administered, and diuresis improved. The patient's fluid balance improved, and adjustments were made to the patient’s medication regimen.

By day two, the patient's blood pressure was managed with nicardipine, and the urine output improved to approximately 100 mL/h. The insulin infusion rate was adjusted to maintain blood glucose levels between 140-180 mg/dL. Despite these improvements, the patient's pulmonary condition remained a concern, and a repeat chest radiograph confirmed the presence of pulmonary oedema. Echohemodynamic studies revealed a cardiac output (CO) of 4.7 L/min and a cardiac index (CI) of 3.06 L/min/m², suggesting that his haemodynamic status remained suboptimal.

Day 3 and 4 Management

By day three, the patient's blood pressure was further stabilised with nicardipine infusion, and his diuresis was more effectively managed with furosemide. His fluid balance remained positive but slowly approached the target of a neutral balance. His renal function continued to improve, with a urine output of approximately 180 mL/h. Echocardiography on the third day revealed further improvements, with CO increasing to 6.7 L/min and CI to 4.33 L/min/m². The patient's condition continued to improve, and the blood pressure was maintained at the target systolic pressure of 160 mmHg.

By day four, the patient showed further progress, with blood pressure stabilising at 160/78 mmHg and a continued reduction in the need for vasopressor support. The urine output remained within the target range of 1-1.3 mL/kg/hour. The patient’s renal and cardiac functions continued to stabilise, and he was transitioned

from a clear liquid to a soft diet. The plan for the day included further monitoring of blood glucose levels, with insulin adjusted accordingly, and continued management of the patient's fluid balance and renal function.

This case highlights the complex nature of managing post-kidney transplant patients with multiple comorbidities, including diabetes mellitus, hypertension, and ESRD. Postoperative complications, particularly pulmonary oedema, require careful monitoring and intervention, including diuretics and vasopressors, to stabilise haemodynamics. This case underscores the importance of a multidisciplinary approach with continuous monitoring and adjustments to the therapeutic plan, including fluid management, glucose control, and renal function monitoring. Although the patient experienced significant challenges during the postoperative period, timely intervention and careful management resulted in stabilisation and recovery of the patient. This case emphasises the importance of personalised ICU care to ensure that patients receive appropriate interventions to optimise outcomes following kidney transplantation.

Table 3. Summary of Key Parameters

Day	Blood Pressure (mmHg)	Heart Rate (bpm)	Urine Output (mL/hour)	Fluid Balance (mL)	Medication Adjustments	Key Interventions
1	140/81	101	5	0	Dobutamine, Norepinephrine, Insulin infusion	Gastric lavage, test feeding, ultrasound
2	161/68	101	100-50-100	+164	Nicardipine, Furosemide	Chest X-ray, Lung Ultrasound, Fluid adjustment
3	164/71	101	170-180-180	-2195	Furosemide, Meropenem	Echohemodynamics, Fluid titration
4	160/78	101	135-90-120	-908	Furosemide, Prograf, Myfortic	Transition to soft diet, Echo assessment

DISCUSSION

Kidney transplantation remains the most common solid organ transplantation, providing a viable solution for patients with end-stage renal disease (ESRD). This procedure significantly enhances the patients' quality of life and life expectancy; however, the postoperative period is often complicated by multiple factors, such as hypertension, fluid imbalance, and pulmonary complications. In this case, a patient with pre-existing hypertension and diabetes mellitus type 2, undergoing kidney transplantation demonstrated various complications typical of high-risk transplant recipients.

Intraoperative fluid management plays a critical role in kidney transplantation outcomes. Maintaining an optimal mean arterial pressure (MAP) is essential for ensuring adequate renal graft perfusion. MAP should be maintained between 80-110 mmHg during the perioperative period to avoid delayed graft function (DGF) [1-2]. In the present case, the patient experienced a significant drop in blood pressure during surgery, which was managed by administering crystalloids and vasopressors. Following this intervention, blood pressure stabilised, and graft perfusion improved as confirmed by postoperative ultrasound. This intervention highlights the importance of haemodynamic optimisation in preventing early graft dysfunction and improving the outcomes of transplantation.

Hypertension is a common and significant complication of kidney transplantation that affects long-term graft survival and increases the risk of cardiovascular diseases. The pathophysiology of post-transplant hypertension is multifactorial, including pre-existing hypertension, medication side effects, and changes in kidney function post-transplant [2-5]. The patient in this case was classified as having persistent hypertension because of a history of poorly controlled blood pressure before transplantation. Nicardipine, a calcium channel blocker, was selected for its positive effects on graft function and compatibility with immunosuppressive therapies. Managing blood pressure post-transplant is crucial for preserving graft function and preventing cardiovascular complications, and strict control of blood pressure, particularly in patients with a history of hypertension, is essential for optimal outcomes [6-8].

Pulmonary complications, such as acute respiratory failure (ARF) and pulmonary oedema, are common after kidney transplantation and are often exacerbated by factors such as immunosuppressive therapy and pre-existing comorbidities. The patient developed acute respiratory failure on day two post-surgery, marked by an increase in respiratory rate and decreased oxygen saturation. Chest radiography and ultrasound revealed signs of pulmonary oedema, which was managed with continuous diuretic therapy (furosemide) to improve urine output and reduce fluid overload. Pulmonary complications, particularly in high-risk patients like this one, necessitate careful monitoring and early intervention to prevent long-term morbidity [9-10].

Acute kidney injury (AKI) remains a major concern in kidney transplant recipients and often manifests as delayed graft function (DGF) or early graft failure. In the present case, the patient experienced a temporary reduction in urine output, which was attributed to poor renal perfusion following a drop in blood pressure during surgery. Timely interventions, including fluid resuscitation and vasopressor administration, restored adequate perfusion and urine output to acceptable levels. This case underscores the importance of vigilant monitoring of graft function and early recognition of AKI to prevent long-term graft failure and improve post-transplant survival [11-13].

Patients undergoing kidney transplantation are at a high risk of infection owing to the need for immunosuppressive therapy to prevent graft rejection. In this case, the patient developed an increase in the white blood cell count, which prompted the initiation of broad-spectrum antibiotics. Effective management of infections is crucial for preventing complications, especially because transplant recipients are highly susceptible to bacterial, viral, and fungal infections. Appropriate antibiotic stewardship, guided by clinical signs and laboratory findings, is essential for improving outcomes in the post-transplant period [14-15].

CONCLUSION

Kidney transplantation is the most prevalent solid organ transplantation, offering significant improvements in the quality of life of patients with end-stage renal disease (ESRD). However, careful management of comorbidities, such as hypertension and diabetes mellitus, is essential to prevent post-transplant complications. In this case, the patient experienced acute kidney injury (AKI) due to impaired graft perfusion, which was likely triggered by a drop in blood pressure during the surgery. This resulted in reduced urine output and suboptimal graft function, as highlighted by postoperative ultrasound findings. Additionally, fluid overload leads to pulmonary oedema and heart failure, necessitating continuous diuretic therapy and antihypertensive management to restore the baseline blood pressure and support graft function. Postoperative immunosuppressive therapy also requires careful monitoring for infection, leading to the initiation of broad-spectrum antibiotics due to significant leukocytosis. This case underscores the importance of optimal perioperative management, including haemodynamic stability, infection prevention, and graft function monitoring, to ensure successful long-term outcomes in kidney transplant recipients.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors contributed to the work, including data analysis, drafting, and reviewing the article. They approved the final version and were accountable for all aspects.

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