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Case Report

Successful Continuous Renal Replacement Therapy (CRRT) for Acute Kidney Injury (AKI) with Septic Shock Underwent Long Coronary Artery Bypass Graft Procedure (CABG)

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ABSTRACT

Background: Acute kidney injury (AKI) occurs in significant numbers of patients undergoing cardiopulmonary bypass surgery for coronary artery disease. AKI patients requiring renal replacement therapy (RRT) after cardiac surgery were at higher risk of postoperative mortality. One of the modes of RRT is Continuous renal replacement therapy (CRRT). CRRT can keep the hemodynamics of the patient stable, provide excellent control of azotemia, support beneficial immunomodulation, increase the clearance of inflammatory mediators, and potentially lessen ongoing or repeated renal ischemia. As a better alternative to traditional intermittent hemodialysis, CRRT has now emerged as the leading form of RRT for patients with AKI post-CABG because CRRT can keep hemodynamics more stable.
Case: We report a case report of a patient who had a long aorta cross-clamp in the CABG procedure. He got AKI with a shock condition during hospitalization in the intensive care unit. The patient had been done with CRRT and had a good response after the procedure. The incidence of AKI after cardiac surgery in this patient may increase with several risk factors, such as surgical bleeding, diabetes mellitus, pre-operative renal dysfunction, low left ventricle ejection fraction (LVEF), the use of cardiopulmonary bypass (CPB) machine and infection. An imbalance between renal oxygen supply and oxygen demand will induce AKI. This patient had done CRRT and showed good clinical and laboratory condition after the procedure.
Conclusion: CRRT is a good choice for AKI patients post-CABG procedure with shock conditions.

Keywords: coronary artery bypass graft, continuous renal replacement therapy, acute kidney injury, coronary artery disease, septic shock, sepsis

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INTRODUCTION

With a 30% frequency, AKI is a frequent complication following cardiac surgery. 2% to 5% of AKI patients need CRRT, and the mortality rate was between 50% and 80%.[1,2,3] Reoperative renal impairment, diabetes mellitus, low left ventricle ejection fraction, emergency surgical procedure, prolonged cross-clamp time (>60 minutes), blood transfusion, infection, and use of cardiopulmonary bypass machine are risk factors for acute kidney injury (AKI) after cardiac surgery.[3,4] An imbalance between the oxygen supply and demand in the kidneys causes acute renal failure. If mean arterial pressure is lower than the renal optimal autoregulation value, renal blood flow will decrease. Reduced renal blood flow will decrease the glomerular filtration rate (GFR), impacting tubular oxygen reabsorption and reducing renal oxygen consumption. Reduction of renal blood flow is a significant cause of renal failure in cardiac surgery.[2] CPB-induced systemic inflammatory response syndrome (SIRS) frequently results in AKI and acute lung injury that requires positive pressure mechanical ventilation. Continuous renal replacement therapy (CRRT) is an effective treatment for AKI following cardiac surgery since the patient typically has an unstable hemodynamic after CABG. [5,6]

Anuria, volume overload, which is unresponsive to diuretic therapy, uremic symptoms, electrolyte and acid-base imbalance that is not responsive to medication, particularly metabolic acidosis and severe hyperkalemia, are indications for initiating RRT in AKI patients.4 RRT consists of continuous low-efficiency dialysis (SLED), Intermittent hemodialysis (IHD) and CRRT. Using a traditional dialysis machine, SLED patients receive treatment for around eight hours, which allows for a slower removal of fluid and toxins. CRRT is one of the modes of RRT that offers slower fluid removal than IHD. The benefits of CRRT included avoidance and early control of fluid accumulation and overload, uremia-related problems, acid-base and electrolyte or metabolic imbalances, and unnecessary or excessive diuretic exposure. CRRT also promotes advantageous immunomodulation and increases the clearance of inflammatory mediators. As a result, CRRT is essential for preserving fluid, electrolyte, and acid-base homeostasis, as well as for treating and preventing potentially life-threatening AKI and organ damage. When there is fluid overload, catabolism, hemodynamic instability or shock condition, or a septic infection with AKI that makes intermittent hemodialysis challenging to administer, CRRT becomes crucial.[4] CRRT is one of the modes of RRT that is a good choice for CABG patients other than IHD and SLED because patients after CABG tend to have low ejection fraction, unstable hemodynamics, shock conditions, and use vasopressors (2). Because of all of the causes, in this report, we will present a case about a patient with AKI and shock condition after the CABG procedure who had an excellent clinical and laboratory condition after CRRT.

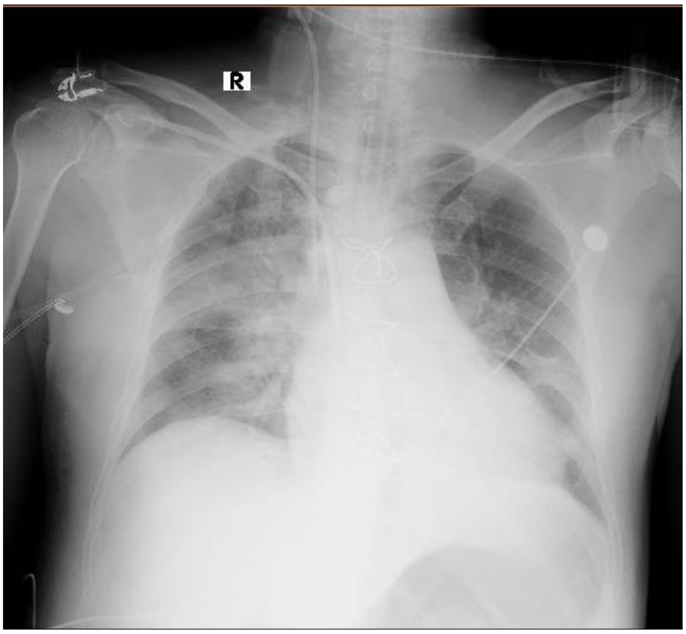

Figure 1. Chest X-ray indicated pneumonia

CASE

A 53-year-old male weighing 55 kilograms was referred to our hospital for elective coronary artery bypass grafting (CABG) surgery. He had previously been diagnosed with coronary artery disease (CAD) and exhibited three-vessel disease. He complained of chest pain a year before being hospitalized, especially when he had heavy activity. The patient had hypertension and diabetes mellitus with routine treatment. On admission, his blood pressure and blood test were normal. Echocardiography showed that the dimensions of the heart chamber (right atrium, right ventricle, left atrium, and left ventricle) were normal. The ejection fraction was 51%. He had diastolic dysfunction and hypokinetic in mid apical anterior, mid anteroseptal, and mid anterolateral. There were also mild mitral regurgitation and tricuspid regurgitation with an intermediate probability of pulmonary hypertension. The diagnostic coronary angiography showed 70-99% stenosis in the proximal mid-left anterior descending artery, 70-90% in the proximal–distal of the right coronary artery, and 90% in the obtuse marginal one artery. The preoperative laboratory showed that the serum urea level was 48 mmol/L, and the serum creatinine level was 1,46 mmol/L. Blood gas analysis is normal as pH 7.4; PaO_2 93.8; $PaCO_2$ 31.6; base excess -5.4. We assessed this patient with American Society of Anesthesiologists (ASA) 3, heart failure, hypertension, diabetes mellitus, coronary artery disease, acute kidney injury stage 1, and intermediate probability of pulmonary hypertension.

He had done the CABG procedure with a cross-clamp time of 90 minutes. The patient was extubated in good clinical condition. On day three of the operation, the patient's condition decreased. He felt shortness of breath and desaturation; we intubated this patient again, and the saturation was only 94% with 100% oxygen. There were bilateral rhonchi in the lung, and the urine production had decreased to 0.25 cc/kg/h for 12 hours, supported by norepinephrine 0.1 µg/kg/minutes, dobutamine 15µg/kg/minutes, epinephrine 0.6 µg/kg/minutes and the blood pressure was 115/44 mmHg (mean arterial pressure 68). The laboratory showed blood gas analysis was metabolic acidosis with respiratory alkalosis, pH 7.24, PaO_2 135.8 mmHg, base excess, -12.8 mmol/L, and PaCO2 32.1 mmHg. Other laboratory examinations indicated thrombocytopenia 107.000, inflammatory reaction (white blood cell count 10,660; and C-reactive protein [CRP] level 15.68 mg/L, procalcitonin 46.49) level, 5,91 mmol/L, serum urea level, 218 mM/L). We assessed this patient who underwent respiratory failure, pneumonia (**Figure 1**), uremic lung, lung edema, AKI stage III, and septic shock with unstable hemodynamics. This condition indicates that he should be done with CRRT.

On the 4th day after the operation, he was scheduled for CRRT. The mode of CRRT was continuous venovenous hemodiafiltration (CVVHDF) with ultrafiltration 20-30 mL/kg/h, blood flow 120-180 mL/h, and heparin dose was 250-500 IU/h. The patient was done with CRRT for three days and showed good clinical and laboratory conditions. He was extubated from the ventilator; his saturation was 100% in 8lpm of the simple mask. We found minimal rhonchi in the lung. The urine production was 1.87 cc/kg/h, and the serum creatinine level had decreased to 2.44 mM/L. Besides RRT, the patient also got meropenem as an antibiotic for pneumonia. Before CRRT, the patient had a septic shock with a high dose of vasopressor; after CRRT, the vasopressor could be stopped, and he had a stable hemodynamic. The patient was discharged 11 days after the surgery (**Table 1**).

DISCUSSION

Diagnostic criteria of AKI include anuria (negligible urine output for six h), severe metabolic acidosis, and clinical complications of uremia, encephalopathy, pericarditis, and neuropathy.[1] The risk factors of AKI after cardiac surgery are preoperative renal dysfunction, diabetes mellitus, low left ventricle ejection fraction, emergency surgical procedure, long cross-clamp period, blood transfusion, infection, and the use of a cardiopulmonary bypass machine.[3,4] In this patient, the risk factors of AKI, such as surgical bleeding, diabetes mellitus, pre-operative renal dysfunction, low LVEF, and the use of a CPB machine for more than 60 minutes happened. Surgery-related factors, including the administration of anticoagulants, the consumption and dilution of clotting factors, systemic inflammatory reactions, and fibrinolysis linked to the utilization of CPB, may impact intraoperative bleeding. Among them, 15% to 20% of patients experience significant bleeding during cardiac surgery, and these patients consume 80% of the blood products utilized in these procedures.[1] Hemodynamic instability induced by substantial blood loss stands as a primary contributor to acute kidney injury (AKI), a factor that markedly elevates patient mortality rates following surgery.[6,7] Massive intraoperative hemorrhage was an additional risk factor for mortality in AKI patients. During cardiac surgery, bleeding is a factor in severe haemodilution, and it is commonly known that AKI is associated with the lowest hematocrit.

Table 1. Patient Clinical and Laboratory Values after CRRT

Data	Day 4	Day 5	Day 6	Day 7
BP (mmHg)	73/39 (NE 0.8μg/kg/minute, dobutamine 15μg/kg/minutes, epinephrine 0.8 μg/kg/minutes)	149/40 (dobutamine 15μg/kg/minutes, epinephrine 0.2 μg/kg/minutes)	138/62 (dobutamine 12μg/kg/minutes)	146/61 (dobutamine 2 μg/kg/minutes)
HR (times/minutes)	120	89	92	94
Saturation	97 (FiO_2 100%)	100% (FiO_2 50%)	99% (FiO_2 40%)	100% (FiO_2 40%)
Urine Output	0.17cc/kg/h	0.3cc/kg/h	1.4cc/kg/h	1.87cc/kg/h)
Potassium (mE1/L)	4.22	3.3	3.13	3.72
Ureum (mmol/L)	218	161.8	99.3	88.6
Creatinin (mmol/L)	8.15	4.89	3.02	2.44
pH	7.38	7.35	7.28	7.3
PaO_2	245	111.9	102,6	157.2
$PaCO_2$	32	26.8	32,5	39.6
Base excess	-6	-10.7	-11.4	-7.1
Platelets ($x10^9$/L)	128	125	119	126
APTT	44.3	51.7	25.3	24.9

BP: Blood pressure; HR: Hearth rate; APTT: activated partial thromboplastin time

The main explanation for this correlation is that increased renal tubular transport raises the energy requirements of the kidneys. Second, hemodilution's reduced oxygen supply might worsen the dysfunction caused by renal ischemia, especially in the oxygen-sensitive renal medulla.

It is still debatable if blood transfusions following cardiac surgery have an impact on AKI, in contrast to intraoperative bleeding. According to specific research, receiving blood transfusions worsens the systemic inflammatory response syndrome that CPB started by triggering a second inflammatory reaction, which increases the risk of AKI.[8] CPB-induced systemic inflammatory response syndrome leads to AKI and acute lung injury that requires positive pressure mechanical ventilation.

Kidney and lung organ dysfunction are closely related and can be roughly classified as hemodynamic and neurohormonal. In addition, Fuhrman et al. reported that AKI was associated with cardiac procedures.[1,3] Consequently, patients with AKI and significant cardiac dysfunction are excellent candidates for CRRT. AKI has typically been linked to either an inflammatory reaction or cardiogenic dysfunction in individuals undergoing cardiac surgery. In this clinical context, CRRT may reduce the left ventricular end-diastolic pressure by maximizing the Frank-Starling relationship and enhancing cardiac function by removing excessive fluid and myocardial depressants.[5,11]

The development of AKI occurs in around 40% of patients who are admitted to the intensive care unit (ICU), with sepsis being the most common cause. Dialysis is necessary for these patients if their renal parameters deteriorate. Patients who have AKI require RRT. Between 50% and 70% of AKI patients who needed hemodialysis and were admitted to the intensive care unit (ICU) are thought to have a higher mortality rate.[19] Anuria, uremic symptoms, volume overload not responsive to diuretic therapy, and electrolyte and acid-base imbalance not responding to medical therapy—in particular, severe hyperkalemia and metabolic acidosis—are indications for starting RRT in AKI patients.[4] The Kidney Disease Improving Global Outcome (KDIGO) guideline suggests intermittent or continuous renal replacement therapy (CRRT) for hemodynamically stable patients. CRRT is the recommended mode of renal replacement therapy (RRT) for patients with hemodynamic instability.[8] Our patient had been performing CRRT because of AKI stage III, uremic symptoms, and shock condition. The goal of CRRT was to help patients who were hemodynamically unstable and resistant to anti-diuretics regain better homeostasis. This treatment modality has been proposed to offer superior hemodynamic and cardiovascular stability owing to hypothermia, which increased venous return and blood pressure when compared with intermittent hemodialysis.[9,10]

It is widely accepted that mortality and morbidity are affected by the dosage of peritoneal dialysis in patients who require chronic and peritoneal dialysis. As a result, it was also assumed that the RRT dosage would have an impact on the outcome of AKI patients. One of the most significant changes in patient care in the intensive care unit was the introduction and implementation of CRRT.

CRRT is more than just metabolic management compared to other RRT modes. Better fluid balance is made possible by CRRT, which also gives rise to more aggressive nutritional support and may positively impact immune function and overall health outcomes.[6,8,9,10] CRRT provides continuous and consistent fluid removal and uremic toxin clearance. It is further classified into venovenous and arteriovenous categories. It is easy to titrate their intensity to prevent or treat volume overload quickly. Hemofiltration, for example, has been shown in numerous studies to enhance lung and heart function in patients with AKI and cardiogenic shock following cardiac surgery. This may lessen the requirement for inotropic support, which helps ensure the survival of patients.[12] Several ways to administer CRRT have been developed. Slow continuous ultrafiltration is the therapy employed exclusively for volume control. The more widely used types are continuous venovenous hemodialysis (CVVHD), continuous venovenous hemofiltration (CVVH), or continuous venovenous hemodiafiltration (CVVHDF). Volume removal and solute clearance are both provided by CRRT, with the differences between these modalities related to the mechanisms for solute clearance.

In CVVH, a hydrostatic gradient produces a high ultrafiltration rate across the semi-permeable hemofilter membrane, and convection occurs by solute transport.[18] Dialysate is perfused over the dialysis membrane's external surface in CVVHD, and solutes leave the blood to enter dialysate via diffusing along their concentration gradient. Pump-driven continuous veno-venous hemodiafiltration (CVVHDF) is a further CRRT variation that offers higher ultrafiltration rates. This allows for the achievement of higher dosages when combined with standard CRRT.[7,8,18] In this patient, we choose CVVHDF mode because it can improve renal function and increase the clearance of inflammatory mediators. It was proven in this patient we diagnosed AKI and shock condition; he had been performed CVVHDF, ultrafiltration 20-30 mL/kg/h, blood flow 120-180 mL/h, and heparin dose was 250-500 IU/h. On the first day after the operation, the vasopressor dose could be decreased, and the next day, it could be stopped. The patient had excellent clinical and laboratory conditions three days after he got CRRT.

The 2017 study by Mishra et al. demonstrated that CRRT is better than SLED in fluid balance maintenance. The amount of fluid removed was lower in CRRT[17], but there was no discernible difference between the other modalities. The recovery of renal function and intensive care unit care duration were considered significant indicators of clinical outcome and survival rate. Our findings indicated a trend toward improved renal recovery in patients receiving a higher dose of CVVHDF, in contrast to earlier research. Among the survivors of the high and low-dose groups, the rates of chronic dialysis were 19% and 50%, respectively.[12,14,15]

CONCLUSION

AKI in post-CABG patients is usually correlated with unstable hemodynamics. When hemodynamic instability, fluid overload, catabolism, and sepsis with AKI are present to the point that intermittent hemodialysis is challenging to administer, CRRT becomes crucial. Moreover, CRRT can promote beneficial immunomodulation and improve the removal of inflammatory mediators. In light of this, CRRT can be a good and safe choice for AKI patients after cardiac surgery with shock conditions.

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CONFLICT OF INTEREST

The author declares there is no conflict of interest.

REFERENCES

1. Lagny MG, Jouret F, Koch JN, et al. Incidence and outcomes of acute kidney injury after cardiac surgery using either criteria of the RIFLE classification. *BMC Nephrol*. 2015;16:76.
2. Kowalik MM, Lango R, Klajbor K, et al. Incidence- and mortality-related risk factors of acute kidney injury requiring hemofiltration treatment in patients undergoing cardiac surgery: a single-center 6-year experience. *J Cardiothorac Vasc Anesth*. 2011;25(4):619–624.
3. Hein OV, Birnbaum J, Wernecke KD, et al. Three-year survival after four major post-cardiac operative complications. *Crit Care Med*. 2006;34(11):2729–2737.
4. Liu C, Zhang HT, Yue LJ, et al. Risk factors for mortality in patients undergoing continuous renal replacement therapy after cardiac surgery. *BMC Cardiovasc Disord*. 2021 Oct 21;21(1):509.
5. Koyner JL, Murray PT. Mechanical ventilation and the kidney. *Blood Purif*. 2010;29(1):52–68.
6. an den Akker JP, Egal M, Groeneveld AB. Invasive mechanical ventilation as a risk factor for acute kidney injury in the critically ill: a systematic review and meta-analysis. *Crit Care*. 2013;17(3):R98.
7. Joe J, Samsu Z, Retno A, Zahara R, Prakoso R, Ardiyan A. Risk of Acute Renal Failure Requiring Renal Replacement Therapy after Cardiac Surgery. *Indonesian Journal of Cardiology*. 2014; 34(3): 147-53.
8. Fuhrman DY, Nguyen LG, Sanchez-de-Toledo J, et al. Postoperative acute kidney injury in young adults with congenital heart disease. *Ann Thorac Surg.* 2019;107(5):1416–20
9. Oh TK, Song IA. Postoperative acute kidney injury requiring continuous renal replacement therapy and outcomes after coronary artery bypass grafting: a nationwide cohort study. *J Cardiothorac Surg*. 2021; 16: 315
10. Lugones F, Chiotti G, Carrier M, et al. Continuous renal replacement therapy after cardiac surgery. Review of 85 cases. *Blood Purif*. 2004;22(3):249-55
11. Lewington AJ, Cerda J, Mehta RL. Raising awareness of acute kidney injury: a global perspective of a silent killer. *Kidney Int.* 2013;84(3):457–67.
12. Rosner MH, Okusa MD. Acute kidney injury associated with cardiac surgery. *Clin J Am Soc Nephrol*. 2006;1(1):19–32.
13. Vives M, Hernandez A, Parramon F, et al. Acute kidney injury after cardiac surgery: prevalence, impact and management challenges. *Int J Nephrol Renovasc Dis*. 2019;12:153–66.

14. Priyanka P, Zarbock A, Izawa J, Gleason TG, Renfurm RW, Kellum JA. The impact of acute kidney injury by serum creatinine or urine output criteria on major adverse kidney events in cardiac surgery patients. *J Thorac Cardiovasc Surg*. 2021;162(1):143-151.e7.
15. Baldwin I, Bellomo R, Naka T, Koch B, Fealy N. A pilot randomized controlled comparison of extended daily dialysis with filtration and continuous veno-venous hemofiltration: Fluid removal and hemodynamics. *Int J Artif Organs.* 2007;30:1083-9.
16. Kielstein JT, Schiffer M, Hafer C. Back to the future: Extended dialysis for treatment of acute kidney injury in the Intensive Care Unit. *J Nephrol*. 2010;23:494-501.
17. Mishra SB, Singh RK, Baronia AK, Poddar B, Azim A, Gurjar M. Sustained low-efficiency dialysis in septic shock: Hemodynamic tolerability and efficacy. *Indian J Crit Care Med*.2016;20:701-7.
18. Lobo VA. Renal Replacement Therapy in Acute Kidney Injury: Which Mode and When?. *Indian J Crit Care Med*. 2020 Apr;24(Suppl 3):S102-S106.
19. Fatoni, AZ, Kestriani ND. Acute Kidney Injury pada Pasien Kritis. *Majalah Anestesia dan Critical Care*. 2018. 36;2 :64-76.