CASE REPORT

Management of Continuous Renal Replacement Therapy Following Coronary Artery Bypass Grafting in the Intensive Care Unit

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ABSTRACT

Background: Acute kidney injury (AKI) after cardiac surgery is a serious complication with a high occurrence, leading to increased morbidity and mortality. Continuous renal replacement therapy (CRRT) is the preferred method for replacing kidney function in patients with hemodynamic instability, especially during the critical postoperative period in the intensive care unit (ICU).

Case: A 76-year-old man with a history of ischemic heart disease and chronic heart failure underwent off-pump coronary artery bypass grafting (CABG). The patient had comorbidities including obesity, obstructive sleep apnea (OSA), chronic obstructive pulmonary disease (COPD), pneumonia, and chronic kidney disease (CKD). During intensive postoperative care, the patient experienced a progressive decrease in urine output, rising urea and creatinine levels, and significant fluid overload. CRRT with continuous venovenous hemodiafiltration (CVVHDF) mode was started on the first day of ICU admission, and clinical improvement was observed after four days of therapy.

Discussion: Patients with multiple comorbidities often experience a deterioration in kidney function after surgery, requiring prompt intervention. The critical role of CRRT in stabilizing fluid and metabolic balance, while simultaneously maintaining hemodynamic stability, cannot be overstated. Careful monitoring of volume status, hemodynamics, and laboratory results is essential to determine the duration of therapy and evaluate its effectiveness.

Conclusion: CRRT is an effective treatment for patients after CABG with AKI and hemodynamic instability. A team-based approach and proper monitoring are crucial for the success of therapy and patient recovery.

Keywords: acute kidney injury; continuous renal replacement therapy; coronary artery bypass grafting; hemodynamics; intensive care unit

INTRODUCTION

Acute kidney injury (AKI) affects approximately 30% of patients undergoing cardiac surgery and vascular procedures.[1] Cardiac surgery carries a high risk of mortality and morbidity, primarily attributed to the use of cardiopulmonary bypass (CPB), the substantial fluid shifts between the intracellular and extracellular compartments, and the elevated likelihood of hemodynamic alterations. AKI necessitating dialysis occurs in 2-5% of patients after cardiac surgery and contributes to 50-80% of mortality rates.[2,3] Consequently, preoperative renal dysfunction elevates the risk of severe postoperative complications and mortality.

Coronary artery bypass grafting (CABG) is a common procedure to treat diseased heart arteries. However, complications like postoperative AKI occur in 20–30% of cardiac surgery patients.[1] Severe AKI requires replacement kidneys, like continuous renal replacement therapy (CRRT), which can develop morbidity and mortality.

Post-CABG AKI is caused by a combination of factors, including hypoperfusion during surgery, nephrotoxic agents, and CPB-related contributors, including prolonged CPB[4], longer aortic cross-clamp times[5], hemodilution and anemia cause low renal oxygen delivery[6], and systemic inflammation, which can cause significant acute kidney damage.

CRRT is indicated in post-CABG patients with AKI and persistent oliguria, excessive fluid accumulation, electrolyte imbalances, metabolic acidosis, encephalopathy, uremic syndrome, and multiorgan failure (MODS).[7,8] It's preferred over intermittent therapy due to unstable hemodynamics in post-CABG patients, especially in the initial 48-72 hours.[9] CRRT reduces hypotension, improves volume control, and can be used simultaneously with inotropic or vasopressors, effectively managing excess fluid.

CASE

A 76-year-old patient weighing 100 kilograms and measuring 160 centimeters underwent an off-pump CABG procedure for chest pain that had persisted for two and a half years. Congestive heart failure secondary to ischemic heart disease was diagnosed, and routine treatment included furosemide, rosuvastatin, valsartan, xarelto, clopidogrel, and aspirin. One month before hospitalization, the patient experienced shortness of breath during physical activity, angina, and chest tightness.

A coronary angiography was performed, revealing a 30% distal stenosis in the left main artery, 70-80% proximal ostial stenosis of the left diagonal artery, and 40-60% mid stenosis, along with 30-40% high diagonal ostial stenosis. The patient exhibited left coronary dominance. An echocardiographic assessment indicated an ejection fraction of 60%, an increase from the initial ejection fraction of 25%.

The patient had a history of chronic obstructive pulmonary disease (COPD) and received routine treatment, including the use of a combivent nebulizer and pulmicort every eight hours. The patient's American Society of Anesthesiologists (ASA) physical status was 3, and they were diagnosed with grade 2 obesity (height 160 cm, weight 100 kg, body mass index 39.1 kg/m^2), obstructive sleep apnea (OSA), pneumonia, COPD, chronic kidney disease (CKD), and coronary artery disease (CAD) 1VD.

The surgical procedure lasted for three hours and thirty minutes, resulting in a cumulative fluid balance of +1925 cc and a urine output of 0.87 cc/kgBW/hour.

The patient was admitted to the ICU for seven days and received therapy, including the replacement of a CRRT from day one to day four. CRRT was initiated due to a deterioration in urine output, which had previously been 1.1 cc/kgBW/hour, to 0.18 cc/kgBW/hour, along with an increase in blood urea nitrogen (BUN) to 49 mg/dL and creatinine to 3.74 mg/dL, and a positive fluid balance of 2100 cc.

DISCUSSION

The patient's preoperative glomerular filtration rate (GFR) was 15.5 mL/min/1.73m². GFR is the most comprehensive indicator of glomerular function, representing the rate of substance filtration through the glomeruli. A normal GFR for adult men ranges from 90 to 120 mL per minute. A GFR below 15 mL per minute indicates end-stage renal failure, requiring dialysis. Based on the CKD stage, our patient is Stage 4 (Table 1).

Patients with heart disease and kidney disease often have cardiorenal syndrome (CRS). CRS is a clinical condition characterized by simultaneous heart and kidney dysfunction, where dysfunction in one organ leads to acute or chronic dysfunction in the other.[11] Combined heart disease and kidney failure worsen quality of life, prognosis, and healthcare system burden.[12,13] Various types of CRS (Table 2).

CRS's pathophysiology involves neurohumoral dysfunction, endothelial activation, and proinflammatory cytokine release. These mechanisms lead to cardiac, renal, and organ dysfunction. In type I and II CRS, cardiac dysfunction causes venous congestion or diminished cardiac output, reducing GFR and triggering the renin angiotensin aldosterone system (RAAS) and neuroendocrine hormones like arginine-vasopressin and endothelin, which contribute to kidney injury.[14] Clinical studies show elevated plasma catecholamines in renal dysfunction, suggesting sympathetic hyperactivity in type III and IV CRS.[14] Affected kidneys enhance sympathetic activation, leading to hypertension, cardiac injury, and kidney deterioration. RAAS activation increases angiotensin II levels, causing aldosterone secretion, sodium, and water retention. Angiotensin II also affects cardiomyocytes and renal tubular cells, causing cellular hypertrophy, apoptosis, and fibrosis.

Volume overload, caused by cardiac or renal dysfunction, stretches endothelial cells. Biomechanical stress activates the endothelium, producing prooxidant, proinflammatory, and vasoconstrictive substances. Elevated pro-inflammatory cytokines, such as TNF and IL-6, affect myocardial and renal function, accelerating heart failure.[15]

Cardiac and renal dysfunction can lead to intestinal hypoperfusion and congestion, resulting in the translocation of bacterial endotoxin (lipopolysaccharide [LPS]) into the systemic circulation.[16] This activation of circulating immune cells, coupled with the release of cytokines such as TNF Alpha, Interleukin-1 (IL-1), and IL-6, can exacerbate myocyte and renal dysfunction.[15]

Cardiac failure alone doesn't cause kidney dysfunction, but Mullens et al. found that in patients with decompensated heart failure and low cardiac output (as shown by elevated

central venous pressure upon admission), inadequate reduction in CVP during hospitalization worsens kidney function.[17] CVP and GFR are inversely related in chronic heart failure. High CVP increases renal venous pressure, which increases renal interstitial hydrostatic pressure. If this exceeds tubular hydrostatic pressure, tubular collapse and reduced ultrafiltration pressure can lead to kidney dysfunction.

Our patient's ejection fraction declined at the onset of the attack. Two and a half years prior, it was 25%, accompanied by symptoms of congestion like shortness of breath and generalized swelling. Based on these observations, we conclude our patient has CRS type 2. The pathophysiology of CRS (Figure 1).

Patients undergoing cardiac surgery are at risk for perioperative AKI. Perioperative AKI following cardiac surgery is associated with risk factors such as age over 56 years, male gender, active congestive heart failure, ascites, diabetes, and hypertension. The surgical procedure itself increases AKI (intraperitoneal surgery). The risk of perioperative AKI remains a major concern in vascular and cardiac surgery. Sepsis and blood transfusions also increase the risk of AKI. General management principles include identifying and reducing renal injury causes, minimizing exposure to nephrotoxic agents (NSAIDs, radiographic contrast), and maintaining renal blood flow. This can be achieved by rapid correction of intravascular volume depletion and maintenance of adequate systemic arterial pressure.[3]

Several perioperative factors also affect renal blood flow directly through hemodynamic effects or indirectly through stimulation of the sympathetic nervous system or arginine vasopressin (AVP). Regardless of the cause, decreased renal blood flow tends to reduce GFR by reducing renal cortical blood flow. Likewise, decreased renal blood flow increases the risk of renal medullary ischemia. The effects of these changes are sodium and water conservation and, consequently, decreased urine output.

Many perioperative factors affect renal blood flow through changes in cardiac output or systemic arterial pressure. Anesthetic agents generally have significant direct effects on hemodynamics, either by reducing systemic vascular resistance, suppressing myocardial function, or reducing preload. Likewise, perioperative hypovolemia (from preoperative fasting, fluid shifts, acute hemorrhage, or a combination of these factors) will decrease cardiac output and systemic arterial pressure using similar direct effects on renal blood flow.

Because the kidneys have a rich autonomic innervation, renal blood flow is also highly sensitive to sympathetic nervous system activity.[18] Sympathetic stimulation increases renal vascular resistance, which has two significant effects. First, perfusion is diverted from the kidneys to other organs, such as the brain and liver, to maintain perfusion of these critical organs.[18] Second, renal arteriolar constriction decreases glomerular capillary pressure and reduces GFR. Whether the cause is pain, surgical stimuli, or exogenous catecholamines, excessive sympathetic stimulation can decrease glomerular blood flow and decrease urine output.[18]

Painful stimuli release AVP, increasing water reabsorption from collecting tubules and concentrating urine.[19] Acute hemorrhage also increases sympathetic tone, reducing renal blood flow.

Studies show that certain anesthetics during surgery and surgical stress can affect kidney function. However, some anesthetics induce anti-inflammatory, anti-necrotic, and anti-apoptotic effects that protect against AKI. Propofol, for example, potentiates its protective effect against ischemia and reperfusion due to its cytoprotective effects.[20,21]

Surgical complications, such as anemia, abnormal calcium/phosphate homeostasis, and inflammation, also exacerbate the risk of perioperative AKI. Lower eGFR is also a consequence and cause of decreased left ventricular systolic function and heart failure.[22] Furthermore, kidney function is a sensitive indicator of vascular health and hemodynamic stability, both of which are important perioperative determinants. A study by Noyez et al. demonstrated a correlation between preoperative kidney function and postoperative kidney failure, along with estimated GFR compared with serum creatinine.[23] A creatinine clearance level <50 ml/min was a strong predictor of postoperative kidney failure and the need for postoperative dialysis.[23]

Cardiac surgery and vascular surgery represent high risks. Most patients are exposed to CPB machines, large amounts of fluid administration, and the risk of hemodynamic instability.[24] The use of a CPB machine triggers various pathophysiological processes that can lead to complications. The pathophysiological causes of CPB machine use include disruption of pulsatile flow, blood flow rate patterns, exposure of blood to non-physiological surfaces and shear stress, hemodilution, the presence of systemic stress and inflammatory response, and hypothermia and hyperthermia.

The use of a CPB machine will expose red blood cells to non-physiological surfaces that will result in mechanical damage to red blood cells in the form of impaired function and integrity of red blood cells, red blood cells become stiffer and easily destroyed, thus disrupting blood flow, especially in the microcirculation. Red blood cells that are destroyed and experience hemolysis will produce red blood cells without hemoglobin, which is believed to be an independent predictor of acute kidney failure after the use of a CPB machine.[25]

A systemic inflammatory response occurs due to trauma from cardiac surgery, including major surgery, which is exacerbated by the use of the CPB. The systemic inflammatory response is the activation of several innate immune system components that results in a wide range of body-wide responses ranging from moderate inflammation to hemodynamic decline, organ dysfunction, and death.[25]

Research conducted by Domoto et al. demonstrated a relationship between preoperative renal dysfunction and a high incidence of morbidity and mortality after CABG surgery, showing an increase in serum creatinine and a decrease in eGFR calculated by the Cockcroft-Gault formula.[26] The study showed that lower preoperative eGFR was an independent predictor of all-cause mortality (HR = 0.983, p = 0.026) and cardiac mortality (HR = 0.963, p = 0.006) after CABG, indicating that impaired renal function before surgery markedly worsens long-term outcomes.[26] The relationship between CKD and poor outcomes after CABG in patients who did not undergo dialysis has been analyzed in several studies, including: (1) high comorbid factors in patients along with CKD, such as old

age, low preoperative hemoglobin, history of old myocard infarction (OMI), and Stroke. Patients with CKD often experience a disease progression process from the mentioned comorbid factors, even though these factors are determinants that contribute directly to poor outcomes in post-cardiac surgery; (2) CKD is a strong independent risk factor for cardiovascular disease. This may reflect increased inflammation and oxidative stress associated with kidney function. Renal dysfunction can be associated with many physical changes, such as high homocysteine levels, hyperuricemia, hypercalcemia, and uremia, all of which have adverse cardiovascular effects; (3) It has been reported that patients with CKD have a greater frequency of coronary artery occlusions that accompany three coronary vessels and involvement of the left main coronary artery (LCA), compared along with patients without CKD. This suggests that patients, along with CKD, may have more extensive coronary disease before surgery than patients without CKD.[26]

The devastating consequences of CKD may lead to a global decrease in oxygen supply to the myocardium, due to severe damage to the epicardial coronary vessels and decreased coronary reserve due to microvascular disease.[26]

Perioperative AKI associated with cardiac surgery is classified as modifiable and non-modifiable. These include renal insufficiency, diabetes mellitus, peripheral vascular disease, COPD, congestive heart failure, LCA disease, and left ventricular efficiency (EF) <30%. Modifiable procedure-related risk factors include surgical urgency, duration of CPB, off-pump surgery, non-pulsatile CPB flow, hemolysis, hemodilution, and rewarming, including hypothermic arrest.[27]

AKI is associated with various physiological changes, such as high homocysteine levels, hyperuricemia, hypercalcemia, and uremia, all of which can lead to adverse cardiovascular effects. Patients with kidney failure also have a higher frequency of CAD involving three coronary vessels or left main CAD, compared to patients without kidney disease. The ultimate consequence of kidney disease is a global reduction in myocardial oxygen delivery due to epicardial vascular damage.[26]

The consequences of AKI on organ function include the heart, lungs, brain, liver, immunology, and other organ systems. In the liver, AKI causes intestinal barrier damage and increased intestinal translocation and delivery of endotoxins and microorganisms to the portal system. This leads to liver inflammation and liver cell apoptosis, along with the overproduction and release of systemic proinflammatory cytokines.[28] AKI is also associated with cerebral dysfunction, including uremic encephalopathy.[29] Activation of the neuroinflammatory cascade leads to increased vascular permeability and blood-brain barrier breakdown.[29] In the heart, AKI is associated with CRS, a condition characterized by simultaneous heart and kidney failure. The mechanisms by which AKI triggers cardiac dysfunction include fluid overload and decreased myocardial contractility caused by uremia. In the lung, the effects of AKI are due to activation of the inflammatory cascade, which leads to increased pulmonary vascular permeability and pulmonary neutrophil infiltration. This leads to fluid accumulation within the lung tissue, leading to pulmonary edema. In the immunological system, AKI has a significant impact on humoral and

cellular immunity and overall immunocompetence. This is due to a combination of increased oxidative stress, impaired reticuloendothelial clearance, and decreased clearance of circulating cytokines, leading to a high risk of infection.[30] Consequences of AKI on various organ functions (Figure 2).

Patient has been on maintenance for 8 days in the surgical ICU, along with the use of CRRT on the day of maintenance from 1 to 4. Optimal time for starting CRRT in patients post cardiac surgery still become topic controversial topic, and special studies discussing specifically post-operation CABG are still limited. Many studies define time CRRT initiation in different ways, starting from biochemical parameters like the improvement level of creatinine and urea, to physiological parameters like decreased urine output or excess liquid. Variations in definition become an obstacle in formulating guidelines for global consensus. The lack of consistency also opens room for an individual approach according to the clinical condition of every patient.

Patients who experience Cardiac Surgery-Associated Acute Kidney Injury (CSA-AKI) are predisposed to get more CRRT compared to patients with AKI that is not associated with cardiac surgery. This is due to the high risk of excess fluid, heart dysfunction post-surgery, and hemodynamic instability.[31] In conditions with existing complications like severe hyperkalemia, metabolic acidosis, or pulmonary edema, CRRT should be started to prevent worsening conditions in non-surgical AKI patients, but in CSA-AKI, it can be initiated moreover formerly along without waiting for the emergence of signs.[32]

In patients, CRRT was carried out because there was a decline in urine output, which was previously 1.1 cc/ kgBB /hour, became 0.18 cc/ kgBB /hour, along with increased BUN 49 and creatinine 3.74, and along with fluid balance positive 2100 cc. Based on our patients' preoperative staging, they have entered the stage 4 category. A retrospective study by Li et al.[31] shows that patients, along with CSA-AKI, CRRT can be initiated at a stage earlier (kidney disease: improving global outcomes [KDIGO stage 1–2]) compared to non-perioperative AKI patients. This happens to patients who undergo cardiac surgery, who tend to experience an excess of fluid postoperatively. Risk increased in patients along with ventricular dysfunction ventricles left ventricular dysfunction, and right ventricular dysfunction, along with pulmonary hypertension, before surgery.[33] Excess volume >10% is associated with improved mortality, and significant early initiation of CRRT reduces risk this is in patients, along with CSA-AKI.[34] Condition: excess fluid post close CABG surgery relation along with use of the machine shortcut heart pulmonary (CPB).

Several studies have yielded mixed results regarding the optimal time to initiate CRRT in post-cardiac surgery. Campos et al.[35] concluded that early CRRT initiation (<48 hours after ICU admission) does not significantly impact patient survival rates post-cardiac surgery with AKIN 2-3. In contrast, the study revealed that the No group maintained a consistent baseline, while the group with a delayed CRRT initiation exhibited a significantly higher urine output. However, the study of Tripathi et al.[36] recommends CRRT initiation when the bicarbonate level is <16.83 mg/dL, and Ji et al.[37] Initiation of CVVHD <12 hours after urine output <0.5 ml/kg/hour (KDIGO 1) is associated with better clinical outcomes.

Two meta-analyses from Cui et al.[38] and Liu et al.[39] The comparison of "early" CRRT compared to "late" CRRT involves studies that are partly big in the incidence of AKI in KDIGO stages 1 and 2, even several studies involving more stages beginning again (for example, an increase of 10% in serum creatinine postoperation). Studies conclude that although not all "early" CRRT shows an impact on survival rate and length of stay, early CRRT initiation tends to be beneficial in controlling balance fluids and electrolytes, as well as preventing complications more continued. Liu et al report that early CRRT lowers 28-day mortality and length of ICU stay in patients post cardiac surgery.[39]

From various findings, it can be concluded that the timing of CRRT initiation in patients post-CABG is highly dependent on their clinical and physiological parameters. Although there is still no universal consensus, there is an increasing body of evidence suggesting that initiating CRRT earlier in patients with mild AKI (KDIGO 1–2) is more beneficial, particularly when accompanied by impaired kidney perfusion or excessive fluid retention. This early intervention appears to result in improved clinical outcomes.[39]

Determining CRRT needs in patients post-cardiac surgery, including CABG, is still a complex clinical challenge. In recent years, the approach based on scoring predictive and machine learning models has been developed to increase the accuracy of predictions and help make more correct decisions. Cleveland clinical score, SOFA kidney score, KDIGO AKI Staging, Renal Anginal Index, and Furosemide stress test can be used. Become a guide in evaluating the need for a therapy replacement kidney.

The Cleveland clinical score can predict acute renal failure in patients undergoing cardiac surgery (Table 3).

With interpretation Score 0–2: Risk low (<1%), Score 3–5: Risk moderate (2–10%), Score ≥6: Risk high (>10%). Consider CRRT if the patient has a score > 6 and is accompanied by unstable hemodynamics or fluid overload. In patients with a score of 11, an accompanying overload of fluid. Kidney sofa score (Table 4) can also be used together with other scoring to predict CRRT needs, along with SOFA scoring > 3, CRRT is highly considered.

CRRT is indicated in patients' post-operative CABG along with AKI stage 3 criteria: creatinine increases more than 3x from baseline, or more than 4 mg/dL, or urine output <0.3 ml/kg/hour for >24 hours, or anuria >12 hours. Accompanied by along with indications of absolute CRRT in the form of fluid overload, diuretic resistance, hyperkalemia refractory, severe metabolic acidosis (pH <7.2), encephalopathy, uremic /pericarditis, and persistent oliguria/anuria. Patient, with a kidney SOFA value of 3, resistance to diuretic, and potassium value of 5.3 mEq/L, and oliguria (UOP 0.18 cc/kgBB/hour), was strongly indicated for CRRT.

Besides scoring conventional, study Tichy et al.[41] successfully identified proenkephalin A 119–159 (PENK) as a promising new biomarker in predicting successful discontinuation of CRRT in patients post cardiac surgery. PENK levels of 290 ± 175 pmol/L indicate good predictive ability along with an area under the curve (AUC) of 0.798 and sensitivity/specificity of 64% and 89%, respectively, at the threshold value of 126.7 pmol/L. This biomarker can play

an important role in evaluating the right time for a free patient from CRRT therapy safely and efficiently. Further development of technology intelligence artificial has generated predictive models based on machine learning, as shown in previous studies.[42]

Studies that compare seven machine learning algorithms, including AdaBoost, LightGBM, Gaussian Naïve Bayes (GNB), Complement Naïve Bayes (CNB), Multi-layer Perceptron (MLP), K-Nearest Neighbors (KNN), and Support Vector Machine (SVM). The NAM model shows performance best, along with an accuracy prediction CRRT requirement reached 86.2%, AUC training set 0.856, and AUC validation set 0.817. A total of 17 clinical parameters were significantly correlated with CRRT needs, including variables like cTnT, CKMB, albumin, NYHA class, serum creatinine, BUN, age, QRS, atrial fibrillation, and LVDd. Findings. This signifies potential strength from the integration of multi-parametric clinical data and machine learning algorithms in designing a system to support clinical decision-making for predicting early CRRT needs in patients post-CABG.

Patient CRRT was carried out in CVVHDF mode along with a planned dose of 25 cc/kgBW /hour. With replacement 1250 cc/hour and dialysis 1250 cc/hour, along with blood flow 130 milliliters per minute. With a target fluid removal of 94 cc/hour. Guidelines from KDIGO. The general recommended use of CRRT dose of 20–25 ml/kg/hour as standard therapy in patients with AKI.[43] However, until now, there is no specialized subpopulation that guides patients who specifically discuss optimal dosages after surgery, particularly cardiac surgery, such as CABG surgery. Another study reported significant variability, indicating that the safe and effective dose can range from 35 to 40 ml/kg/hour, depending on the patient's individual characteristics and therapeutic objectives.[44]

Study by Bent et al.[45], in studies retrospectively on patients along with acute renal failure post operation heart show that along with rate flow blood 200 to 250 mL/m and ultrafiltration of 2 L/hour (equivalent to along with 35 ml/kg/hour) along with aggressive and early CVVH associated along with number more deaths low compared to the predicted 40% in 65 post-operative AKI in cardiac surgery patient. This indicates that therapy can be aggressive and give profit in survival, as long as given within the framework at the right time. Li et al.[46], compare the patient post-cardiac surgery along with CVVH dose low (18.1 ± 3.6 ml/kg/hour) and dose high (45.2 ± 7.9 ml/kg/hour). The results show that the group, along with dose tall, experienced a decline in mortality, a significant improvement in 30 days, and during home care illness (mortality 73.3% vs 45.4%; p=0.002). In contrast, another study [22] shows that there is no significant difference in mortality between low dose (25 ml/kg/hour) and high dose (40 ml/kg/hour). However, the incidence of hypophosphatemia was recorded as higher in the high-dose group, indicating an increased risk of metabolic side effects from aggressive therapy. In studies, most recently, Li et al.[31] find that in patients along with CSA-AKI, even if CRRT is given more initially (KDIGO 1–2), the dose used is still equivalent to that of non-surgical AKI patients, indicating that the clinical benefit originates more lots originate from time initiation, not from improvement or decline in dose.

Based on the above study, it can be concluded that the dose standard 20–25 ml/kg/hour can still be used on patients post-cardiac surgery, including post-CABG, provided that given at the right and appropriate clinical indication. Use of high dose (>35 ml/kg/hour) can be considered in severe cases or worsening in a fast way, but needs to be customized along with tolerance to the hemodynamics of patients and metabolic risks. Thus, the optimal strategy in the administration of CRRT to patients' post-operation cardiac surgery not only lies in the dose only, but also in individualized therapy, proper time of initiation, and monitoring effect side effects in a strict manner.

In patients, after settings are made on the CRRT Prismaflex machine, along with CVVHDF mode, along with planning Dose 25 cc/kgBW/hour. With replacement 1250 cc/hour and dialysis 1250 cc/hour, along with blood flow 130 milliliters per minute, the obtained fraction filtration is > 20%. Several CRRT modes are available, among others: (1) slow continuous ultrafiltration (SCUF). Requires a blood pump and an effluent pump. The effluent pump is needed to create hydrostatic pressure. No dialysis fluid or replacement fluid is required. The primary purpose of SCUF is to remove excess fluid from the body; (2) continuous venovenous hemofiltration (CVVH). Requires a blood pump, effluent pump, and replacement pump. Dialysate fluid is not used. Plasma and solute fluids are used. Removed through convection and ultrafiltration. CVVH is very effective in removing solutes and is indicated for cases of uremia, severe metabolic acidosis, or lack of electrolyte balance, along with or without excess fluid; (3) continuous venovenous hemodialysis (CVVHD). Requires a blood pump, effluent pump, and dialysis pump. A replacement fluid pump is not required. Plasma and solute fluids are removed through diffusion and ultrafiltration. CVVHD is very similar to intermittent hemodialysis, effective for removing small to medium molecular weight molecules. Solutes are removed through diffusion and ultrafiltration; (4) continuous venovenous hemodiafiltration (CVVHDF). Requires a blood pump, effluent pump, dialysis pump, and replacement pump. Plasma fluid is removed through diffusion, convection, and ultrafiltration. CVVHDF mode selection for removing inflammatory mediators (patient along with results pneumonia x-ray and results number leukocytes 16,100) and small molecule in the form of potassium (patient potassium 5.4 mEq /L), BUN 49mEq/L, creatinine patients (3.74), as well as excess liquid.

Small molecules (<300 daltons) = urea, creatinine, kalium are more effective by diffusion. Medium molecules (500-5000 daltons)=B12, more effective by convection. Large molecules (5000-50000 daltons) = globulin, cytokines, and myoglobin, are more effective by convection.[47] The weight molecule (Figure 3). Changes in infection markers and molecules dissolved in our patient (Table 5).

From day 1 to day 4, patients underwent CRRT installation with CVVHDF mode to remove inflammatory mediators and small-molecule substances. Turn to Recovery inflammation on the 4th day of CRRT installation is visible in improved urine output, indicating hemodynamic repair, a decline in vasopressor dose, and improved lactate levels.

Nevertheless, I have not yet observed the kidney repair function because the BUN and creatinine levels remain unchanged. The effective CRRT table appears to show improvement in potassium, blood sugar, and throwaway molecule levels. However, it does not yet demonstrate significant effectiveness in reducing BUN and creatinine levels. This discrepancy may be attributed to the fact that BUN and creatinine production are significantly greater than the amount discharged by the CRRT engine. So that CRRT operators should be able to arrange a more optimal effluent dose.

The effluent dose used in patients is a dose of 20 cc/kg/hour. This can be improved up to 40 cc/kg/hour. When done, hemodynamic CRRT placement is adjusted so that dobutamine and vasopressors are increased, along with a dobutamine dose of 7.5 mcg/ kgBW/min, and norepinephrine 0.15 mcg/kgBW/minute is added to compensate for the change in hemodynamics during CRRT use.

In patients, after settings are made on the CRRT machine, along with CVVHDF mode, planning dose is 25 cc/kgBW/hour. With replacement 1250 cc/hour and dialysis 1250 cc/hour, along with blood flow 100 milliliters per minute, the obtained fraction filtration was>25 %. Fraction filtration obtained from the calculation of effluent blood flow divided by pump blood flow, mainly the result times, along with the subtraction of 1 minus hematocrit (Figure 4). Fraction filtration of more than 20 percent will increase clots in the CRRT filter and reduce filter performance. Done adjustment dose to 20 cc/ kgBW /hour along with replacement of 1000 cc/hour and dialysate of 1000 cc/hour, along with Blood Flow of 130 ml/ minute obtained faction 20% filtration is a necessary important fraction filtration settings in maintaining circuit integrity and maintaining filter performance.

Patient uses fluid dialysis and replacement Prismacol BGK 0/2.5 along with contents in Table 6. Prismasol BGK 0/2.5 has a potassium content of 0 mEq/L, which is initially appropriate for the patient, but can cause hypokalemia due to its risk. In patients, the initial potassium content before CRRT usage is 5.3 mEq /L, but on the 2nd day of CRRT use, patients experience hypokalemia and require potassium replacement therapy. Content Prismasol BGK 0/2.5 also has content dextrose 100 mOsm /L patient, along with dextrose content 239 mOsm /L before use to 98-160 mOsm /L during usage. Selection-type fluids and contents contribute to osmolarity and the content of substances in plasma.

The Integrity CRRT circuit is affected by the fraction filtration, predilution fluid, and anticoagulants. In patients, after settings are made on the CRRT machine, along with CVVHDF mode, along with planning dose of 25 cc/kgBW/hour is used. With replacement 1250 cc/hour and dialysate 1250 cc/hour, along with a blood flow of 130 milliliters per minute. Fluid replacement is provided with the pre-filter to prevent filter clots.

Administer anticoagulants to patients post-cardiac surgery to prevent bleeding complications, as the risk of bleeding after surgery is elevated. CRRT necessitates the use of an anticoagulant to maintain the circulation of blood within a filter, ensuring its smooth functioning and preventing freezing. Some choices of anticoagulants in CRRT

circuit: (1) without anticoagulants, used in conditions risk of bleeding is high, such as <24 hours post-operation. However, the filter life tends to be short; (2) systemic heparin, the dose is low, but it increases the risk of systemic bleeding. Must be monitored strictly along with periodic aPTT examination; (3) regional heparin is administered intravenously and neutralized before its return to the patient's circulation using protamine. Rarely used; (4) regional citrate anticoagulation (RCA), the mainstay in modern ICU.[47] Although systemic bleeding is not a cause, monitoring of calcium levels and heart function is necessary. This patient is being administered systemic heparin along with a dose of 5 IU/kgBW /hour because there is not available of anticoagulant citrate, which is an option in cases with moderate risk bleeding. APTT monitoring is recommended to be done every 6-8 hours, along with a target of APTT 1.5-2 x of control. In this patient, the activated partial thromboplastin time (APTT) is not achieved to facilitate clotting on the filter during the use of CRRT on the third day. Next, this guide discusses anticoagulant use on CRRT machines (Figure 5). Selection of dose, rate, speed flow, use of fluid predilution, as well as the use of anticoagulants, play an important role in extending the integrity of circuits and filters.

On the 3rd day of treatment, the addition of antibiotic moxifloxacin 400 mg, based on the algorithm management of Community Acquired Pneumonia (CAP) patients. Options CAP Gram-negative antibiotic therapy along with double antipseudomonal activity: Non-Beta-lactamase based agent in the ICU is fluoroquinolones, including ciprofloxacin 400 mg/ 8 hours or Levofloxacin 750 mg/ 24 hours. Given the patient's condition and reduced kidney function, the decision was made to prescribe moxifloxacin 400 mg/24 hours. Combined with Meropenem 1 g/ 8 hours. Patient shows enhancement in clinical and improvement in urine output.

Based on the RSBI assessment after previously Spontaneous Awakening Trial and the Spontaneous Breathing Trial patient was extubated on day 5; however very much sputum and saturation down to 92%. N-acetylcysteine nebulizer therapy and HFNC installation have been completed. Oxygenation will be titrated to a non-rebreathing mask at 10 lpm. The patient can be transferred to inpatient care on the 7th day.

Table 1. CKD stages[10]

Stage 1	Kidney damage with normal or increased GFR, GFR $\geq$ 90 mL/min /1.73 m2
Stage 2	Kidney damage with mild GFR reduction, GFR 60-89 mL/min/1.73 m2
Stage 3	Moderate decrease in GFR, GFR 30-59 mL/min /1.73 m2
Stage 4	Severe decrease in GFR, GFR 15-29 mL/min /1.73 m2
Stage 5	failure, GFR < 15 (or dialysis)

Table 2. CRS Classification[12]

Type	Type	Description	Example
1.	Acute cardiorenal	Heart Failure (HF) causes AKI	Acute coronary syndrome causes acute HF and AKI
2.	Chronic cardiorenal	Chronic HF leading to CKD	Chronic HF
3.	Acute renocardial infarction	AKI causes Acute HF	Uremic cardiopathy associated with AKI
4.	Chronic renocardiac	CKD Causes HF	Left ventricular hypertrophy and diastolic HF due to CKD
5.	Secondary	Systemic diseases cause HF and CKD	Sepsis, vasculitis Diabetes mellitus

Description: heart failure (HF), chronic kidney disease (CKD), acute kidney injury (AKI)

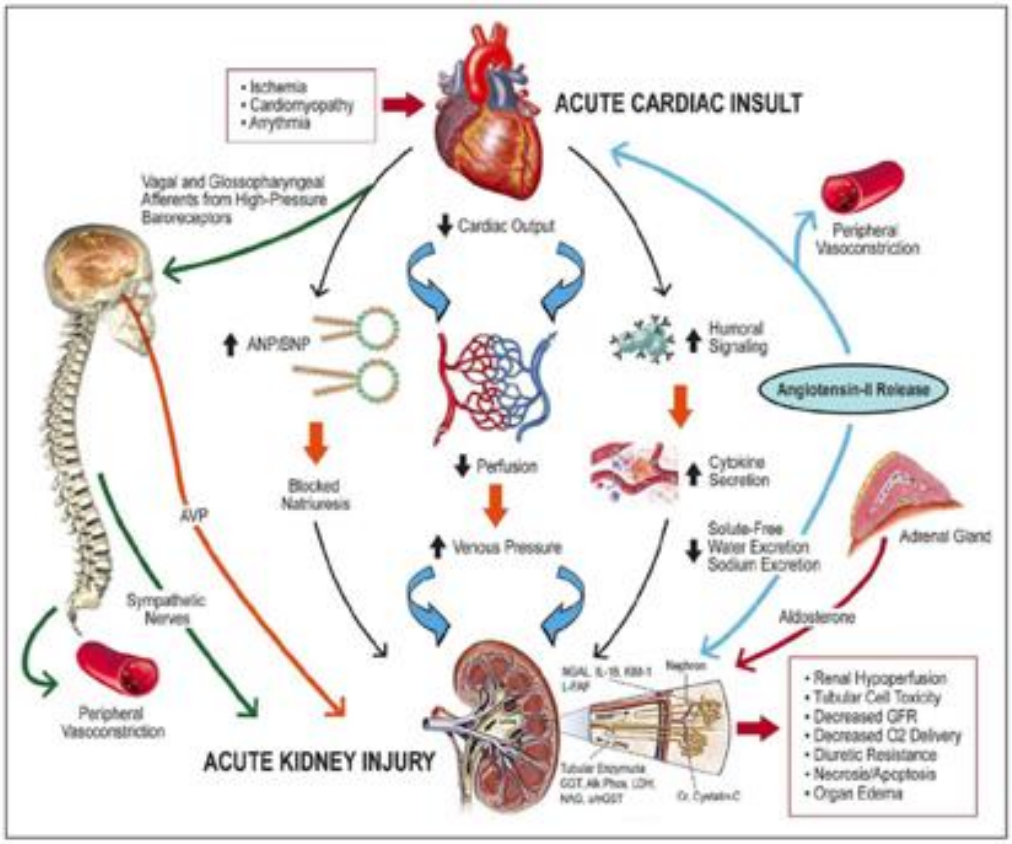


Figure 1. Pathophysiology of CRS[12]

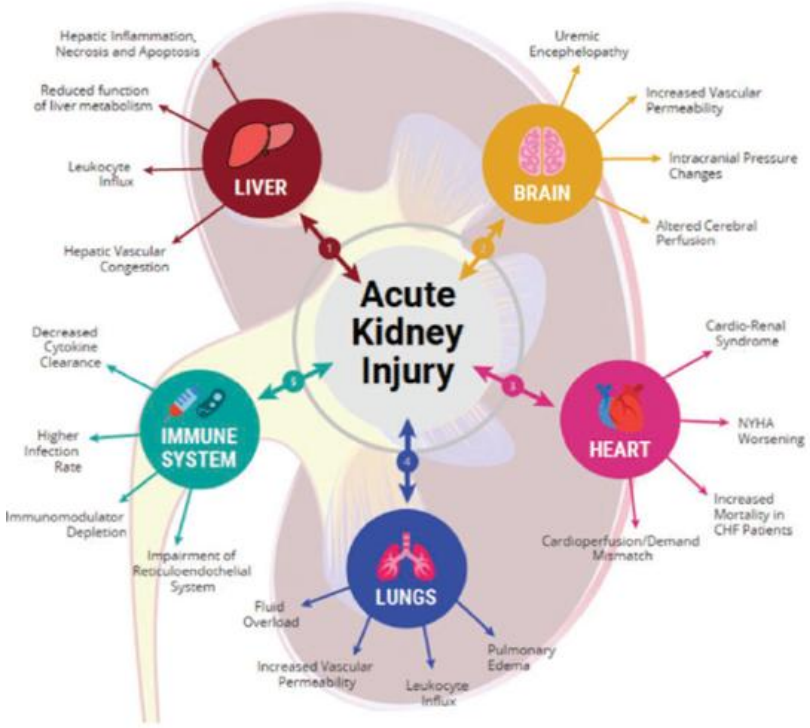


Figure 2. Consequences of AKI on various organ functions

Table 3. Cleveland clinical score[40]

Age ≥ 65 years	2 points
Type sex man	1 point
Fail heart congestive heart failure (CHF)	2 points
Faction ejection ≤ 35%	2 points
Diabetes mellitus	1 point
Disease lungs obstructive chronic (COPD)	1 point
Hypertension	1 point
History of stroke	1 point
Serum creatinine > 1.2 mg/dL	2 points
Operation emergency	2 points
Operation test heart	2 points
Thoracic aortic surgery	3 points

Table 4. Kidney SOFA score

Kidney SOFA Value	Creatinine (mg/dL)	Production Urine (mL/ day)
0	< 1.2	> 500
1	1.2 – 1.9	-
2	2.0 – 3.4	< 500 (for 24 hours)
3	3.5 – 4.9	< 200 (for 24 hours)
4	≥ 5.0	< 100 (for 24 hours) or anuria

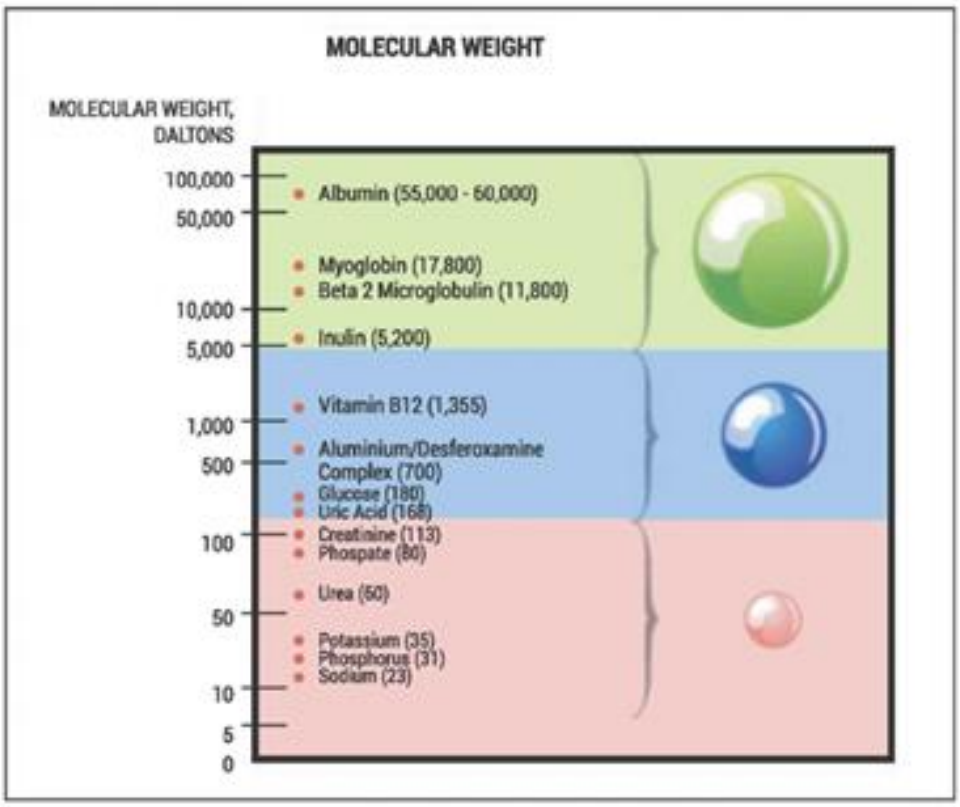


Figure 3. Molecular weight of Substance[47]

Table 5. Changes in infection markers and molecules dissolve

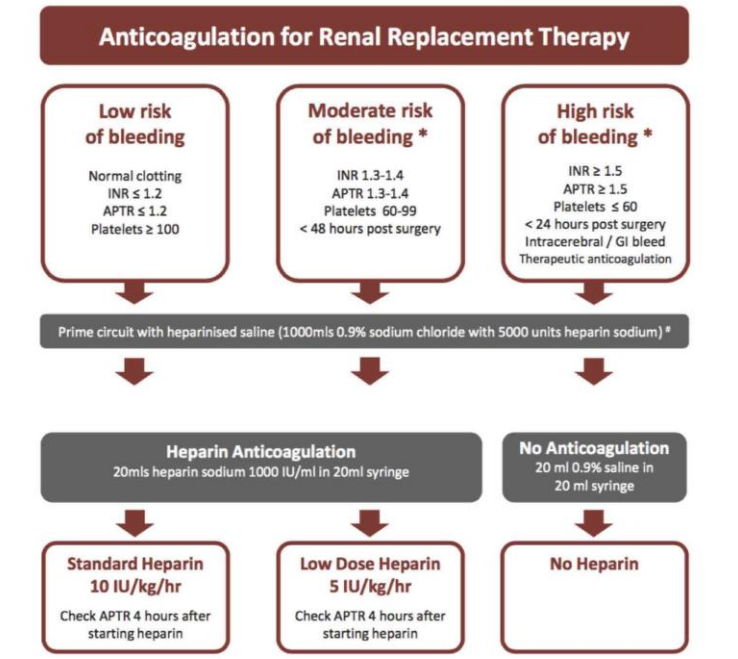
	BUN	Create	UOP cc/Kg/hour	HCO3-	GDS	Potassium	AL	Procalcitonin	Dobutamin mcg/kg/ min	Vascon mcg/kg/ min	Lactate
Day 0	33	1.8	1.1	19.7	239	4.3	13.5	0.067	5	0.15	1.7
day 1	37	2.4	0.18	24.6	233	5.3	16.1		7.5	0.15	2.3
day 2	41	3.51	0	21.8	167	4.9	24.1	0.82	7.5	0.08	2.3
day 3	26	2.4	0.32	21	98	2.47	22.9	0.669	7.5	0.1	2
day 4	49	3.74	2.1	21.7	135	3.74	16	1.24	7.5	0.15	1.5
day 5	60	2.95	1.4	25.9	140	2.98	17.11	0.794	3	0	1.2
day 6	71	2.36	1.05	28	166	4	14.5	0.308	0	0	1.5
day 7	77	2.08	1.5	29.8	150	4	14.1	0.38	0	0	1.1

$$FF = Qef / Qb (1 - Hct)$$

Figure 4. Calculation filtration faction[47]

Table 6. Contents fluid dialysate

	Plasma	Prismasol BGK 4/2.5	Prismasol BGK 2/3.5	Prismasol BGK 0/2.5	Prismasol BGK 4/0/1.2	Prismasol BGK 2/0	Prismasol BB22GK 4/0	Prismasol BK 0/0/1.2
Potassium K (mEq/L)	3.5-5	4	2	0	4	2	4	0
Calcium Ca (mEq/L)	2.3-2.5	2.5	3.5	2.5	0	0	0	0
Magnesium Mg (mEq/L)	1.4-2	1.5	1	1.5	1.2	1	1.5	1.2
Sodium Na (mEq/L)	135-145	140	140	140	140	140	140	140
Chloride Cl (mEq/L)	100-108	113	111.5	109	110.2	108	120.5	106.2
Bicarbonate HCO3 (mEq/L)	22-26	32	32	32	32	32	22	32
Lactate (mEq/L)	0.5-2.2	3	3	3	3	3	3	3
Dextrose (mg/dl)	70-110	100	100	100	100	100	100	0
Osmolarity (mOsm/L)	280-296	300	296	292	295	291	296	282



APTR	Bleeding$	Action		Monitoring
		Standard Heparin	Low Dose Heparin	
1.4 or less	None	Continue 10 IU/kg/hr	Continue 5 IU/kg/hr	Check APTR 12 hourly unless otherwise indicated
1.5 or more	None	Reduce to 5 IU/kg/hr	Stop heparin	Recheck APTR 4 hrs after heparin dose change
ANY	Yes	Reduce to 5 IU/kg/hr or run heparin free depending on significance of bleeding	Stop heparin	Check APTR immediately on identifying new bleeding Recheck APTR 4 hrs after reducing or stopping heparin

Figure 5. Management of anticoagulants in patients on CRRT[47]

CONCLUSION

CRS is an important term to describe the clinical condition of simultaneous heart and kidney dysfunction; dysfunction of one organ will cause acute or chronic dysfunction of the other organs. Perioperative AKI following cardiac surgery is associated with risk factors such as age over 56 years, male gender, active congestive heart failure, ascites, diabetes, and hypertension. Several perioperative factors also affect renal blood flow directly by hemodynamic effects or indirectly through stimulation of the sympathetic nervous system or AVP. Cardiac surgery and vascular surgery represent high risks. Most patients are exposed to CPB machines, large amounts of fluid administration, and the risk of hemodynamic instability. Timing of CRRT initiation in patients post-CABG is very dependent on the clinical and physiological parameters of patients. Although not yet, there is a universal consensus, increasingly, lots of proof shows that initiation earlier in mild AKI (KDIGO 1–2), especially when accompanied by disturbed kidney perfusion or excess fluid, is associated with results in more clinical benefits. Approach-based scoring predictive and machine learning models have been developed to increase the accuracy of prediction and help make more correct decisions. Cleveland Clinical Score, SOFA kidney score, KDIGO AKI Staging, Renal Anginal Index, and Furosemide stress test can be used. Become a guide in evaluating the need for a therapy replacement kidney. Standard dose 20–25 ml/kg/hour can still be used on patients' postcardiac surgery, including post-CABG, provided that given at the right and appropriate clinical indication. Use of high dose (>35 ml/kg/hour) can be considered in severe cases or worsening in a way fast but needs to be customized along with tolerance to hemodynamics, patients, and metabolic risks. The selection of fluids and their contents contributes to the osmolarity and substance content of plasma. The selection of dose, rate of speed, flow, fluid predilution, and the utilization of anticoagulants significantly contribute to the preservation of circuit and filter integrity.

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