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Role of CRRT (Continuous Renal Replacement Therapy) as Management of Cardiac Arrest Patient with Postpartum Acute Lung Edema and Cardiomyopathy in the ICU

Army Zaka Anwary[1*] and Indriasari[2]

[1] *Fellowship of Intensive Care, Department of Anesthesia and Intensive Care, Hasan Sadikin General Hospital Bandung*

[2] *Department of Anesthesia and Intensive Care, Hasan Sadikin General Hospital Bandung*

***Corresponding Author**: Army Zaka Anwary, E-mail: armyzaka@gmail.com

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ABSTRACT

Introduction: Continuous renal replacement therapy (CRRT) is a type of renal replacement therapy commonly used in the ICU for hemodynamically unstable patients. CRRT can comprehensively achieve various goals such as reducing inflammatory mediators, alleviating fluid overload, and correcting acidosis. Therefore, whether CRRT is necessary and when to initiate CRRT are basic questions that intensivists must consider when treating critical patients in the ICU to achieve favorable outcomes.

Method: This retrospective case report analyzed patient clinical data. The patient provided written informed consent to publish their case details and accompanying images.

Results: We present the case of a 23-year-old woman with acute pulmonary edema and peripartum cardiomyopathy who experienced cardiac arrest prior to cesarean section. Postoperatively, the patient was admitted to the ICU, and CRRT was initiated to correct various complications. During treatment, the patient experienced clinical improvement and improvement in respiratory and renal function parameters until she was discharged from the ICU without neurological deficits.

Conclusion: Early assessment, the decision to use CRRT, and comprehensive therapy are crucial for the successful treatment of post-cardiac arrest patients with pregnancy-related acute pulmonary edema and peripartum cardiomyopathy in the ICU.

Keywords

Acute pulmonary edema, Cardiac arrest, Peripartum cardiomyopathy, Continuous renal replacement therapy

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INTRODUCTION

Managing patients after cardiac arrest in the ICU requires comprehensive therapies to achieve a positive outcome. When a patient's circulation has been successfully restored following cardiac arrest, various inflammatory mediators are released, leading to hypoperfusion of vital organs and often accompanied by shock, resulting in hemodynamic instability. Continuous renal replacement therapy (CRRT) is a treatment that can be considered for various purposes, such as reducing inflammatory mediators, reducing fluid overload, and correcting acidosis, among other things. CRRT is often used in the ICU, especially when a patient's condition does not allow regular hemodialysis. Therefore, it is essential for intensivists to understand the different cases that can potentially benefit from CRRT management.

RESULT

A 23-year-old woman (gravida 2, para 1) with a twin pregnancy at 34-35 weeks' gestation came to the hospital with complaints of shortness of breath that had worsened and did not improve with rest. The patient had a history of hypertension in pregnancy and severe pre-eclampsia (blood pressure 162/112 mmHg, proteinuria +4). Due to respiratory failure that did not improve with oxygen administration (respiratory rate 34x/minute,

SpO_2 75% with a non-rebreather mask), immediate intubation was performed. After intubation, the patient experienced cardiac arrest and received 3 cycles of cardiopulmonary resuscitation and 1 defibrillation. After the patient's pulse returned, an emergency cesarean section was performed. The patient could not be extubated immediately postoperatively due to increased oxygen requirements and remained intubated until transferred to the ICU for further treatment.

In the ICU, achieving a mean arterial pressure (MAP) of 65 mmHg required hemodynamic support with the use of norepinephrine at 0.2 mcg/kg/minute and dobutamine at 10 mcg/kg/minute. Postoperative laboratory results showed metabolic acidosis (pH 7.13; HCO_3 16; pCO_2 47.5; pO_2 92; BE -12; SaO_2 94). Echocardiography was performed to assess the patient's hemodynamic status immediately, and the results supported cardiogenic shock (low cardiac output, high SVR, fluid responder; CO: 3.6 L/minute, SVR: 1667 dynes/sec/cm^{-5}, collapsibility index 13%). The results of the ultrasound examination of the lungs showed a B profile. The patient remained severely hypoxemic and required mechanical ventilation with an FiO_2 up to 100%. X-ray examination showed diffuse bilateral opacity, accompanied by bilateral crackles on auscultation (Figure 1). Our patient was assessed as having acute pulmonary edema and peripartum cardiomyopathy. We administered sedation and muscle relaxants to reduce O_2 consumption. As urine production was low (0.15 cc/kg/hour), we administered the loop diuretic furosemide at a continuous rate of 10 mg/hour.

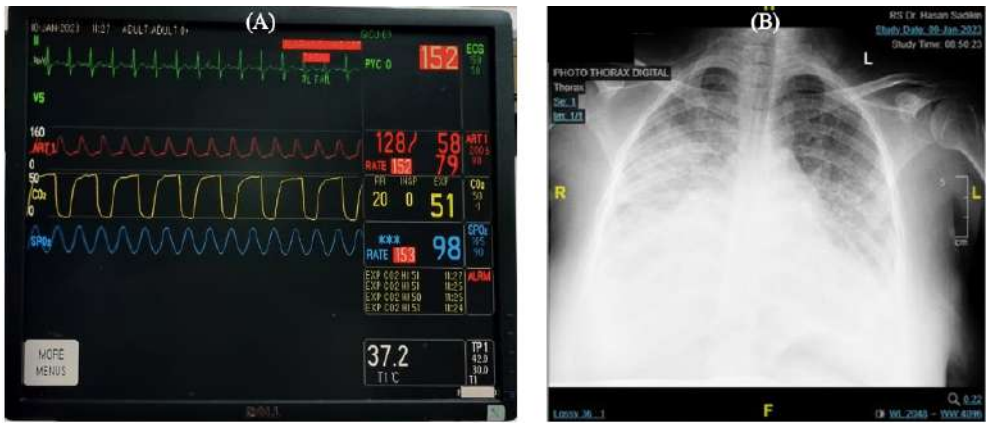

Figure 1.(A) Patient's hemodynamic status when first came to the ICU. (B) Patient's thorax X-Ray

Due to decreasing urine production to the point of almost anuria and an increase in serum creatinine (sCr) to 4.42 mg/dL (baseline sCr at admission was 1.09 mg/dL), we diagnosed the patient with acute kidney injury (AKI). We initiated the use of CRRT on the second day of treatment, utilizing the CVVHDF (Continuous Veno-Venous Haemodiafiltration) mode with a blood flow of 80-100 mL/minute titrated, pre-filtration replacement fluid of 700 mL/hour, post-filtration replacement fluid of 700 mL/hour, dialysate fluid 700 mL/hour, and fluid removal of 50 mL/hour (refer to figure 2). Heparin was administered at a dose of 700 U/hour, and we monitored the results of coagulation factor tests, blood gas analysis, and electrolytes as needed. The patient is still on mechanical ventilation with Pressure Control mode at 100% FiO2 and 10 cmH_2O PEEP. In the next 24 hours, there was an improvement in oxygenation and pO_2 levels, with blood gas analysis results showing a PaO_2/FiO_2 ratio of 280. This allowed for a reduction in the fraction of O2 in the ventilator and PEEP.

During the use of CRRT, the patient's urine output increased to 0.8-1 cc/kg/hour. Additionally, the patient's serum creatinine level fell to 3.19 mg/dL. The dose of hemodynamic support (norepinephrine and dobutamine) also decreased until it could be stopped. The use of CRRT lasted for three days until it was finally stopped because of a suspected disturbance characterized by increased transmembrane pressure (TMP) that did not respond to interventions. On day 4 of ICU stay, the patient underwent a spontaneous awakening trial

and was found to be calm and cooperative (Richmond Agitation Sedation Scale: 0, Critical Pain Observation Tool: 2).

Figure 2. Running CRRT

Follow-up chest X-ray results showed resolution of bilateral opacity (figure 3). Weaning from mechanical ventilation was continued, and the patient was extubated on day 6 of treatment. After 24 hours post-extubation, the patient was transferred from the ICU.

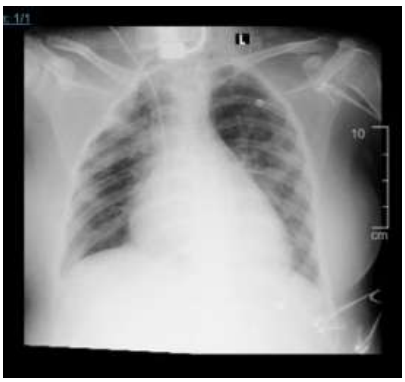

Figure 3. Thorax X-Ray after CRRT showed improvement of lung edema.

DISCUSSION

Acute kidney injury (AKI) is a common condition found after cardiac arrest, with an incidence reaching up to 50% during treatment. One-third of patients require kidney replacement therapy.(1) AKI is defined as a daily urine output <0.5 mL/kg/hour and/or an increase in serum creatinine (sCr) levels of at least 0.3 mg/dL or 1.5 times the increase from the baseline value. The criterion for AKI is an increase of ≥0.3 mg/dL from baseline within 48 hours, and the absence of chronic kidney disease or baseline sCr.(2) A meta-analysis showed that the median AKI occurs on the first and second day after cardiac arrest.(3) Various factors that occur during cardiac arrest contribute to the emergence of AKI, such as the duration of cardiac arrest, accumulated doses of epinephrine, and whether there is post-resuscitation shock. (3,4)

In the first 72 hours after cardiac arrest, a condition called post-arrest syndrome occurs, where various inflammatory mediators are released, resulting in hypoperfusion and vascular hyperpermeability often referred to as "sepsis-like syndrome." This leads to organ dysfunction, including the kidneys. Another mechanism that causes AKI after cardiac arrest is renal hypoperfusion due to post-resuscitation shock.(1,3) The incidence of AKI after cardiac arrest is associated with increased mortality and shows a relationship with neurological recovery outcomes.(1)

CRRT management is one of the modalities used to assist kidney function in critically ill patients with AKI, especially patients who are hemodynamically unstable.(5) The use of CRRT has been shown to be

effective in reducing circulating cytokines and resulting in a decrease in the proinflammatory state.(6) Various modes of techniques have been developed to use CRRT, depending on the mechanism for clearing the solute. The continuous veno-venous haemofiltration (CVVH) mode uses the convection principle and can remove medium molecular weight substances through an ultrafiltration process. The continuous veno-venous haemodialysis (CVVHD) mode, through the principle of diffusion facilitated by dialysate fluid, is effective for removing small molecular weight substances. The continuous veno-venous hemodiafiltration (CVVHDF) mode is a hybrid mode that combines dialysate flow from CVVHD with ultrafiltration from CVVH. The CVVHDF mode can remove blood urea nitrogen (BUN), creatinine, and inflammatory mediators during therapy of AKI.(5)

The question of whether to initiate renal replacement therapy (RRT) and when to do so are two fundamental questions that intensivists encounter in cases of severe AKI. The optimal timing for initiating RRT itself is not clearly defined. The indications for initiating continuous renal replacement therapy (CRRT) are generally the same as those for starting RRT, including volume overload, severe metabolic acidosis, electrolyte disturbances, and to relieve uremic symptoms. Although this indication depends on the intensivist's interpretation and is used as a semi-objective sign, in many patients, RRT is initiated in a state of persistent or progressive AKI even though the criteria have not been met.(2,5) In our case, we initiated CRRT because the patient had severe AKI, pulmonary edema, and metabolic acidosis. We chose CRRT over hemodialysis to prevent potential hypotensive effects (when we initiated the patient's CRRT using dobutamine 10 mcg/kg/minute and norepinephrine 0.2 mcg/kg/minute).

In our case report, the patient is pregnant, and the physiology of pregnant patients differs from that of normal individuals. In pregnancy, increased renal blood flow results in glomerular hyperfiltration accompanied by a decrease in serum creatinine levels that can last up to 12 weeks postpartum. Values that should be normal in non-pregnant patients may become abnormal in pregnancy (7). Normal plasma creatinine drops to 44 umol/L (0.49 mg/dL), and values above 70.72 umol/L (0.79 mg/dL) must be considered abnormal to avoid being late in suspecting AKI. The lack of consensus regarding the definition of AKI in pregnancy makes the diagnosis of AKI during pregnancy difficult. Hypertensive disease during pregnancy is one of the causes of AKI during pregnancy through the mechanism of endothelial injury, followed by secondary effects of loss of intravascular volume, vasoconstriction, and activation of inflammatory mediators and coagulation cascades. Hypertensive disease during pregnancy is also a risk factor for peripartum cardiomyopathy (7,8). Peripartum cardiomyopathy can lead to complications of acute pulmonary edema due to the heart's inability to maintain its pumping function.

There are no specific criteria for stopping CRRT. The initial manifestation of the recovery of kidney function is an increase in urine output. The Beginning and Ending Supportive Therapy for the Kidney (BEST Kidney) study states that a urine output >400 mL/day without additional diuretic therapy is a predictor of successful stopping of CRRT.(5) The most common complication during CRRT is clotting of the extracorporeal circuit. Strategies for minimizing the risk of clotting of the extracorporeal circuit include using higher blood flows, minimizing the filtration fraction (ultrafiltration ratio to plasma flow), and optimal use of vascular access catheters. In our patient, increased transmembrane pressure may indicate clotting of the filter, clogging of the membrane, or malfunction of the catheter. After interventions (ensuring vascular access, increasing blood flow) showed no improvement, we decided to stop CRRT. Urine production when we stopped CRRT had reached 1 cc/kg/hour without diuretic therapy, and the patient's oxygenation level had also improved, so CRRT did not need to be re-initiated.

Neurological outcome is certainly one of the things that need to be considered in the management of patients after cardiac arrest. The incidence of AKI after cardiac arrest is associated with increased mortality and shows a relationship with neurological recovery. (9) The severity of the post-resuscitation syndrome is the main factor that determines neurological outcome. Various comprehensive therapies are combined to improve neurological outcome. Studies show that only less than 10% of CPR procedures created an outcome without neurological damage.(10) Cardiovascular instability that occurs can contribute to secondary brain injury.

Existing case reports have also reported that CRRT has good outcomes in patients experiencing cardiac arrest through the following mechanisms: eliminating inflammatory products such as cytokines, removing toxic products, and the ability to manipulate temperature.(10) In experimental studies of renal injury models, AKI contributes to inflammatory injury to the hippocampus, changes in the permeability of the blood-brain barrier, and potentiation of oxidative stress in cerebral tissues. However, other studies suggest that kidney damage does not have a clear correlation with neurological outcomes.(11-13) In patients with cardiac arrest, mediators that cause secondary brain injury, such as glutamine and glutamate, also appear. However, studies that tried to eliminate plasma levels of glutamine and glutamate using CRRT did not show a significant reduction in plasma levels.(14) In our case report, the patient was successfully discharged from the ICU without any neurological deficits.

CONCLUSION

We present a case report in which a woman with post-cardiac arrest with peripartum acute pulmonary edema and cardiomyopathy required mechanical ventilation. This condition can be treated successfully with multimodal therapy in combination with CRRT. The use of CRRT is a useful modality that can reduce the time to mechanical ventilation and improve lung function. Given the evidence from studies that CRRT can reduce proinflammatory states, further investigation is needed to assess the wider use of CRRT as an intervention in pregnant patients with acute pulmonary edema and cardiac arrest in the future. Moreover, it takes a multidisciplinary team such as anesthesiologists, intensivists, cardiologists, obstetricians, and others to get the best outcome

DECLARATIONS

Ethics approval and consent to participate. Permission for this study was obtained from the Ethics Committee of Universitas Padjadjaran (UNPAD) / Center General Hospital Dr. Hasan Sadikin.

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

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COMPETING INTERESTS

None.

AUTHORS' CONTRIBUTIONS

All authors significantly contribute to the work reported, whether in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas. Contribute to drafting, revising, or critically reviewing the article. Approved the final version to be published, agreed on the journal to be submitted, and agreed to be accountable for all aspects of the work.

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