Scientia et Cura: Illuminating the Dark Side of CRRT for Optimal Patient Benefits

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Introduction. Continuous Renal Replacement Therapy (CRRT) is commonly used in patients with hemodynamic instability but is associated with potential complications. Understanding these complications can improve CRRT efficiency and patient outcomes.
Methods. This cross-sectional study investigated CRRT complications in patients > 18 years old who underwent CRRT at a tertiary medical center from 2017 to 2022. Data were collected from patient records and the hospital's laboratory system.
Results. A total of 178 patients underwent CRRT for fluid overload (38%) and non-septic acute kidney injury (AKI) (35%). The most common CRRT modalities were hemofiltration (54%) and hemodiafiltration (31%). Among patients, 76% experienced a reduction in platelet count (mean decrease of 40% ± 24). Hemoglobin declined by ≥ 1 g/dL in 58% of patients. Phosphorus decreased in 64.6% of patients (mean reduction of 33%) and potassium decreased in 50% (mean reduction of 18%), but these reductions were not statistically significant (*P*-values: 0.73 and 0.88). Vasopressors were stopped in 27% of patients, and the dose was reduced in 50.4%. No significant hypothermia, allergic reactions, pneumothorax, hemothorax, or air embolism were reported. The survival rate at hospital discharge was 64% (123 out of 178).
Conclusion. CRRT is a safe and efficient treatment for AKI, with notable reductions in platelet count and vasopressor dependency. However, reductions in phosphorus and potassium were not significant, indicating manageable complications.

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INTRODUCTION

Continuous renal replacement therapy (CRRT) is a dialysis modality to support kidney function among patients with severe acute kidney injury (AKI).[1] CRRT provides continued and sustained support for patients who are unable to filter waste products and excess fluids adequately. Over the last 30 years, this procedure has been progressively utilized due to its effectiveness and safety in managing AKI in specific clinical situations.[2] Today,

CRRT is a widely used treatment modality in the field of nephrology and critical care medicine. As our understanding of its role in providing renal support in AKI is improved, it is included in many guidelines and recommendations.[3] The range of technology and machines to offer greater flexibility in tailoring treatments has increased, and clinical applications other than AKI, such as sepsis, multiple organ failure, and fluid overload has expanded.[4-6] There is growing clinical expertise due to the increasing availability of specialized training and education programs. Nevertheless, the applicability of CRRT can vary across different healthcare settings and different regions of the world. In some areas or hospitals with limited resources, other forms of renal replacement therapy, such as intermittent hemodialysis, prolonged intermitted renal replacement therapy, daily hemodialysis, or peritoneal dialysis, may be used.[5]

While CRRT is generally considered a safe and effective treatment modality, like any other procedure, it does carry the risk of potential adverse events or complications, such as hemodynamic instability (e.g., hypotension at the time of connection and during the procedure), bleeding, clotting, and vascular access complications, infections, electrolyte imbalances, acid-base disorders, hypothermia, and air embolism.[7,8] Being aware of the common complications of CRRT can assist in optimizing the management of critically ill patients who require this treatment modality.[8]

It is essential to recognize that the overall risks associated with CRRT can vary depending on factors such as the underlying conditions, individual response to therapy, and the expertise of the healthcare team. To our knowledge, there is no report of potential adverse events related to CRRT from Iran. Therefore, the present study investigates possible common complications of CRRT, including electrolyte disturbances (calcium, phosphorus, potassium, magnesium, and sodium) blood factors disorders (platelets, hemoglobin, white blood cells), complications related to venous catheter insertion, bleeding, hypothermia, the effect of CRRT on blood pressure and acid-base status.

MATERIAL AND METHODS

Study Setting and Participants

This investigation is a cross-sectional study conducted on all patients > 18 years old who underwent CRRT in the ICU in a tertiary medical center in Tehran from 2017 to 2022. Patients who died during the first CRRT session, those who did not tolerate CRRT due to being in a state of shock, and cases with significant missing data were excluded from the study (Figure 1).

Data Collection

All demographic, laboratory, clinical, and outcome data (before, during, and after CRRT) were extracted from the medical records. All information was registered in pre-designed questionnaires. The time of the CRRT-related procedures and the date of the tests were also obtained from the patient's medical records.

Implementation and Measurements

This study was conducted in Imam Hossein Hospital, a tertiary medical center in Tehran, Iran. and reviewed and approved by the ethics committee in Imam Hossein Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran IR.SBMU.MSP.REC1399.752). To assess the influence of CRRT, we evaluated the trends of the examined variables in comparison to the baseline level.

We stratified patients based on their baseline platelet count, i.e., patients who had platelets ≥ 150,000 per microliter and those with baseline platelet count of < 150,000 per microliter. We then categorized thrombocytopenic patients (platelets < 150,000 per microliter) into three subgroups based on their platelet counts, i.e., 100,000-150,000, 50,000-100,0000, and < 50,000 per microliter. The normal range of hemoglobin and white blood cells was considered to be 12-16 g/dL and 4.3-10.8 10^9/L, respectively.[3]

Any acute and significant bleeding episode during CRRT, need for emergency blood transfusion, or hemoglobin level decline by ≥ 1 g/dL was considered a bleeding complication. Hypothermia was defined as a temperature < 35 degrees Celsius.[8] The normal ranges of potassium, total calcium corrected by albumin level, magnesium, phosphorus, and sodium were considered 3.5-5 mmol/L, 8.5-10-5 mg/dL, 1.7-2.4 mg/dL, 2.4-5.5 mg/dL, and 135-145 meq/L, respectively.[3] Mean arterial pressure (MAP) and changes in vasopressor doses were used to investigate the effect of CRRT on blood pressure. The MAP of < 60 mmHg, or the need to start or increase the vasopressor doses during CRRT, was considered a

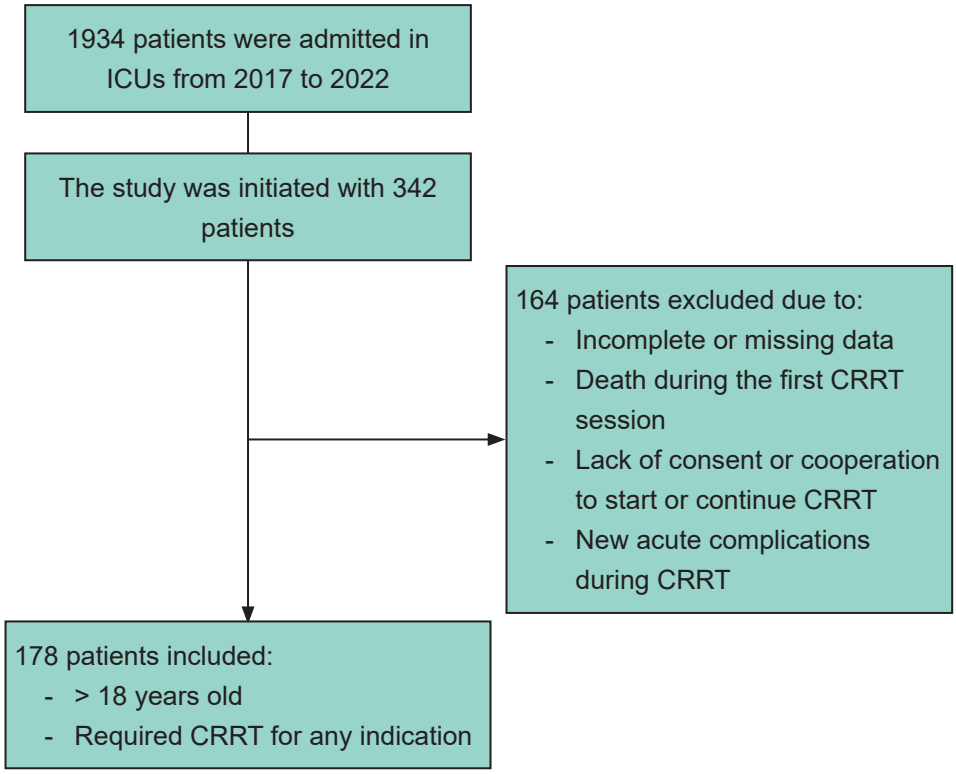

Figure 1. Flowchart of Study Design and Participant Enrollment.

hypotensive episode.[8] According to the information in the patient's medical records, complications related to the catheter, allergic reactions, and hypersensitivity reactions were also recorded.

Statistical Analysis

Mean values and standard deviations were used for continuous variables and frequency and percentages were used for categorical variables. The data were measured with the Smirnov-Kolmogorov test to determine the type of data distribution. The paired t-test was used to compare the before and after data. The results were analyzed by using SPSS version 22 software, IBM Corp., Armonk, NY. A *P*-value level of < .05 was considered statistically significant for all tests.

RESULTS

Out of 342 patients 164 patients were excluded from the study due to incomplete or missing data, death during the first CRRT session, or cooperation to start or continue CRRT, and new-onset acute complications during CRRT. Therefore, 178 patients were included in the final analyses. Table 1 shows a brief description of the demographic findings of the patients.

One hundred twenty-three patients (64.1%) survived and were discharged from the hospital, and 55 patients (39.9%) died during hospitalization. All patients had one or more underlying diseases. The frequency of underlying diseases is given in Table 1.

Table 1. Demographic characteristics and underlying diseases* of patients

Variable	Number (%)
Sex	
male	102 (57.30)
female	76 (42.70)
Age (years)	52.8 ± 14.3
Duration of CRRT (hours)	43.78 ± 82
Cardiovascular Diseases	78 (44)
Diabetes Mellitus	73 (41)
Hypertension	83 (47)
Chronic kidney disease (CKD)	66 (37)
Cancer	16 (9)
Lung Disease	15 (8.5)
Cerebrovascular Diseases	12 (6.7)
Connective Tissue Diseases	9 (5)
Liver Disease	11 (6.1)

*A patient can have one or more underlying diseases

Complications Related to Catheter

Out of 178 patients who were included in the study, three cases had catheter dysfunction, which required catheter exchange. None of the patients had catheter thrombosis, as confirmed by the color Doppler ultrasound. In 18 patients (10.9%), a small hematoma was observed at the catheter insertion site (right-sided internal jugular vein), which did not require any interventions. Also, there was no report of pneumothorax, hemothorax, or air embolism.

Hypothermia and Allergy

There was no evidence of allergic reactions or hypothermia (temperature < 35°C) during CRRT in the studied patients.

Acidemia and Alkalemia

One hundred and forty-seven patients (81%) had acidemia (pH < 7.35) before the CRRT initiation, which resolved following CRRT initiation in 114 patients. Eleven patients had alkalemia (pH > 7.45), and they all recovered to their normal pH levels after CRRT. Twenty patients had normal pH before and during the CRRT treatment.

Changes in Hemoglobin

One hundred and three patients (58%) had ≥ 1 g/dL decrease in hemoglobin level during CRRT. Out of 178 patients, fifty-nine (33%) received one or more units of packed red blood cells during treatment. Sixty-six patients (90%) had hemoglobin levels lower than normal before and during CRRT.

Platelet changes

An apparent decrease in platelet count after CRRT was noted ($P \leq .05$; Table 2).

Changes in white blood cells

In 107 patients (60.1%), white blood cells decreased by an average of 54%, while in 71 patients (39.88%), they increased by an average of 37%. Among 178 patients, 17 exhibited leukopenia (WBC < 4,500) at baseline, which persisted in five cases.

Potassium Changes

Table 3 shows changes in potassium levels. Out of 178 patients, potassium decreased by 18% in 50 patients (28%) and increased by an average of 19% in 21 patients (11.8%). The reduction in potassium level was mild and not statistically significant. Nineteen out of 178 patients (26%) had hyperkalemia before starting CRRT, which remained elevated in five patients even after CRRT initiation.

Sodium Changes

Sodium decreased by an average of 3% in 44

Table 2. Platelets changes

primary platelets range (103 per microliter)	Number (Percentage) of Patients	Decline in platelets from baseline		Increase in platelet from baseline		*P*
		Number (%)	The average percentage of platelets decrease mean ± SD	Number (%)	Average percentage of platelet increase mean ± SD	
150 <	79 (44.38)	62 (78.48)	28.01 ± 19.06	17 (21.15)	12.01 ± 8	.0011
150 >						
100-150	51 (28.65)	45 (88.2)	38.85 ± 20.67	3 (5.8)	-	
		Three patients (5%) had no change in platelet count before and after CRRT				.0748
50-100	43 (24.15)	28 (65.11)	52 ± 31.4	15 (34.8)	35 ± 23.91	
50 >	5 (2.80)	-	-	5	-	

Table 3. Changes in Electrolytes and Minerals

Electrolytes and Minerals	Number of Patients	decrease from the initial amount		Increase from the initial amount		*P*
		Number (Percentage)	Average Percent (Decrease) Mean ± SD	Number (Percentage)	Average Percent (Increase) Mean ± SD	
Potassium	178	50 (28)	18 ± 10.64	21 (11.8)	19 ± 12.74	.7344
Sodium	178	44 (24.7)	2.93 ± 1.92	41 (23)	3.9 ± 2.61	.0532
Phosphorus	178	115 (0.64)	33.67 ± 18.04	44 (24.7)	34.1 ± 15.2	.8887
Calcium	178	85 (47.4)	6.89 ± 5.17	74 (41.5)	9.10 ± 7.99	.0377
Magnesium	178	68 (38.2)	17.06 ± 10.90	43 (24)	18.39 ± 15.96	.6029

patients (24.7%) and increased by an average of 4% in 41 (23%). Sixty-three patients (35.4%) had hyponatremia before starting CRRT, and 31 of them had hyponatremia after CRRT. Also, seven patients (3.9%) had hypernatremia before starting CRRT, and one of them had persistent hypernatremia after CRRT initiation to end.

Phosphorus Changes

Table 3 shows changes in phosphorus levels. Phosphorus decreased by an average of 33% in 115 patients (64%) and increased by an average of 34% in 44 patients (24.7%). The reduction in phosphorus level was not statistically significant. Twenty-seven patients (15.1%) had hypophosphatemia before starting CRRT, and 52% of these cases remained lower and outside the normal range after CRRT.

Magnesium Changes

In our cohort, 111 patients had data related to magnesium levels, out of which, magnesium decreased by an average of 17% in 68 patients (38.2%) and increased by 18% in 43 patients (24%). Fifty patients (45.0%) had magnesium levels lower than normal before starting CRRT. After CRRT, 37 out of the 50 patients (74 %) still had magnesium levels lower than normal. The level of magnesium in 12 patients was higher than normal before starting the treatment, and two of them had a higher-than-normal level of magnesium after the end of CRRT.

Calcium Changes

From 178 patients, calcium in 85 patients (47.7%) decreased by an average of 7%, and in 74 patients (41.5%) increased by an average of 9%. The reduction of calcium was statistically significant ($P < .005$). Fifty-three patients (29.7%) had subnormal calcium before starting CRRT, and 50% of these cases were still at a lower level than the normal range after CRRT.

Changes in Blood Pressure

Fifty-seven patients (32%) did not receive vasopressors before and during CRRT. One hundred twenty-one patients (68%) received vasopressors before CRRT, out of which 33 (27.2%) patients did not require vasopressors following completion of CRRT. Twenty-seven (22%) patients who required vasopressors before CRRT initiation required higher vasopressor doses by the end of CRRT treatment, and 61 (50%) patients required less doses. Seven (4%) patients had a decline of MAP to < 60 mmHg after CRRT initiation. No significant correlation was found between MAP and vasopressor doses before and after CRRT.

Duration (length) of the treatment

In this study, the average duration of the treatment was 46.5 hrs for the Continuous Venovenous Hemofiltration and 41.65 hrs for the Continuous Venovenous Hemodialysis. The duration of CRRT for patients who used a combination of CVVH and CVVHD was 42.65 hrs.

DISCUSSION

This cross-sectional study aimed to investigate the potential complications associated with continuous renal replacement therapy (CRRT). The types of CRRT methods used in this study were CVVH, CVVHD, and CVVHD + CVVH, with frequencies of 97(54%), 26(15%), and 55(31%), respectively. The results indicated a significant decrease in platelet count, phosphorus, and potassium levels following CRRT.

CRRT is a widely used treatment modality in nephrology and critical care medicine. It is particularly beneficial for patients with AKI who are critically ill, hemodynamically unstable, who are unable to tolerate rapid changes in fluid and electrolytes. CRRT may also be used in other scenarios, such as when the kidneys fail to provide adequate support for increased demand, such as sepsis, multi-organ failure, fluid overload, and patients who cannot tolerate intermittent hemodialysis.[1,2] While considered a safe and effective modality, it does carry some potential risks.[8,22] In the present study, complications related to vascular access and catheter placement were not evident. However, Akhoundi *et al.* reported that the most common complications related to catheters were bleeding (15%) and hematoma (1.3%).[8] Graham *et al.* reported pneumothorax in 2.4% of patients and hemothorax in 0.4% of them.[11]

We did not observe any complications such as catheter dislodgement, kinking, bleeding, severe hematoma at insertion site, malfunction resulting in insufficient blood flow, or decreased therapeutic efficacy. Only three out of 178 patients needed a catheter exchange. The reason could be attributed

to placing the catheter under ultrasound guidance, experienced surgical team, and closed monitoring of the vascular access site by the healthcare team. There was no evidence of allergy or hypersensitivity reaction occurring at baseline or during CRRT. Also, none of the studied patients had clinically significant hypothermia during CRRT. In the study of Akhoundi *et al.*, clinically significant hypothermia was reported in 44 percent of patients.[8] The absence of hypothermia in our investigation could be attributed to the use of blood warmers, proper monitoring of ambient temperature, use of solutions with appropriate temperature, and use of warming devices. The health care team closely monitored and managed the patient's body temperature to prevent hypothermia.

Results showed a statistically significant decrease in platelet count after CRRT. Platelets play a crucial role in clotting and maintaining hemostasis. The review of other studies also showed a high prevalence of thrombocytopenia. The frequency of thrombocytopenia in patients treated with CRRT was reported to be 18% in the study of Vinsonneau *et al.*[4] In another study, Jason and his colleagues demonstrated an average platelet reduction of 48%.[11]

During CRRT, there can be interactions between the CRRT and platelet function due to several factors, such as platelet activation and aggregation leading to platelet consumption, dilutional effects, platelet sequestration within extracorporeal circuits, anticoagulants used such as heparin, the modality of CRRT, and patients' underlying conditions.[2] Several factors can predispose the patient to thrombocytopenia, and further research is necessary to determine if CRRT alone triggers thrombocytopenia or if the combination of all factors and conditions contributes to the reduction of platelets. Vanderschueren *et al.* reported more than one cause for the development of thrombocytopenia.[16] The suggestion that CRRT alone may induce thrombocytopenia or that a set of factors and conditions also play a role in causing thrombocytopenia in patients undergoing CRRT has yet to be precisely determined. However, being aware of the critical issue that patients may be prone to thrombocytopenia after CRRT can increase our readiness to predict, prevent, and treat possible complications.[24-25]

A decrease in WBC count was observed in 60% of the patients. Leukopenia can be caused by factors such as sequestration of WBCs within the circuit, dilutional factors, or related drug effects. One hundred three patients experienced a decrease in hemoglobin of ≥ 1 g/dL, and 59 received ≥ 1 unit of packed red blood cells during CRRT. Many studies have shown hemoglobin drop during CRRT. Pschowski *et al.* reported that among patients with anemia prior to initiating CRRT, 83% experienced a 5% decrease in hemoglobin levels. In another study, new anemia occurred during CRRT in 31% of patients, and 33.7% needed packed red blood cell transfusion.[8] New acute bleeding, requiring emergency packed cell transfusion, was not reported.

Many factors, such as blood loss, heparin use, and the potential for induced bleeding, can influence hemoglobin levels. Frequent blood draws for laboratory tests, bone marrow suppression in critical illness, and low nutrients like iron are other causes of anemia. We cannot accurately determine whether the drop in hemoglobin observed in the participants was related to CRRT or i is the result of other factors, including the underlying disease and the acute and critical conditions of the patients.

CRRT aims to restore and maintain the bodily metabolic milieu within normal range. However, complications can still occur. Rapid removal or imbalances in fluid replacement can lead to electrolyte disturbances such as dysnatremia, dyskalemia, and calcium or phosphate imbalances. In some studies, hypokalemia was observed in 5-25% of patients who underwent renal replacement therapy.[18,19] Dyskalemia during CRRT can be caused by a variety of conditions, such as the underlying medical conditions (metabolic disorders, rhabdomyolysis, tumor lysis syndrome), medications (Angiotensin-Converting Enzyme inhibitors (ACEi), Angiotensin Receptor Blockers (ARB), Mineralocorticoid Receptor Antagonists (MRA), Non-Steroidal Anti-Inflammatory Drugs (NSAIDs), Heparins), CRRT-related factors (wrong/inappropriate use of dialysate or replacement fluids, inadequate or excessive potassium removal or clearance, insufficient CRRT dose, inappropriately selected filter, aggressive fluid removal or ultrafiltration rate), excessive potassium intake (from IV or blood products or parenteral solutions), rapid shifts in fluid balance (during CRRT initiation or discontinuation), gastrointestinal losses (diarrhea,

vomiting, GI bleeding) and concurrent therapies such as Extracorporeal Membrane Oxygenation (ECMO) or Therapeutic Plasma Exchange (TPE).[2] So proper monitoring of serum potassium levels, adjusting the CRRT prescription, managing underlying conditions, and appropriate potassium replacement or removal is crucial to prevent and manage dyskalemia in patients undergoing CRRT. Hypokalemia was not significant in this study (50 out of 178 patients). This was a mild negative change. On the other hand, 21 patients experienced hyperkalemia. Therefore, in this study, CRRT did not cause additional remarkable onset of hypo- or hyperkalemia.

CRRT can help correct sodium imbalances by removing excess sodium or providing replacement as needed. Sodium levels can fluctuate during CRRT. Hyponatremia can occur due to dilution from fluid replacement or removal. Conversely, hypernatremia can occur if the replacement fluids used in CRRT have a higher sodium concentration or insufficient sodium removal. Close monitoring of sodium levels is important, and adjustments in replacement fluid composition or dialysate sodium concentration can be made in order to maintain appropriate sodium balance. In our study, sodium decreased by an average of 3% in 44 patients and increased by 4% in 41 patients. None of the studied patients developed a new onset of hypo- or hypernatremia as a complication.

CRRT can lead to phosphate imbalance, typically hypophosphatemia, which warrants accurate consideration due to its critical impact on patients' outcomes (muscle weakness, respiratory complication, hemolysis, impaired cardiac function, and neurological symptoms). Phosphate is distributed in intracellular and extracellular compartments, with only a small fraction in the bloodstream. During CRRT, the removal rate depends on the modality used (CVVH, CVVHD, CVVHDF) and the dialysis prescription (blood flow, membrane permeability). Indeed, because continuous filtration and dialysis remove phosphate from the blood, often faster than it can be replaced, and the fact that solutions frequently do not contain or are low in phosphate, hypophosphatemia can be an essential issue. On the other hand, the underlying conditions that necessitate CRRT, such as sepsis, trauma, or other critical illnesses, can also contribute to phosphate imbalance. Many patients on CRRT are unable to consume adequate nutrition orally or enterally, so regular monitoring of phosphate levels is crucial for patients undergoing CRRT. In this study, we regularly checked the serum phosphorus, calcium, and magnesium levels (every six hours) to closely monitor the serum level of phosphate and other critical electrolytes (especially in the initial stages of CRRT). In some studies, the incidence of hypophosphatemia during kidney replacement therapy varies from 11-65%.[18,19] In the study done by Hendrix *et al.*, it was shown that hypophosphatemia occurred in 63% of patients undergoing renal replacement therapy.[20] In another study, hypophosphatemia occurred in more than 50% of patients.[19] In the study of Broman, hypophosphatemia was observed in 80% of patients who underwent CRRT.[21] In our study, CRRT did not cause new hyper- or hypophosphatemia as a defined complication.

One of the potential complications associated with CRRT is magnesium imbalance. Standard CRRT solutions may contain low magnesium concentrations. The continuous nature of CRRT means ongoing loss of magnesium, especially if the treatment is prolonged or intensive. On the other hand, excessive administration of magnesium (IV or oral) in the setting of reduced renal function can lead to hypermagnesemia. The exact prevalence rate of hypomagnesemia remains unknown. Hypomagnesemia is common among critically ill patients undergoing renal replacement therapy.[22] Macedo *et al.* also emphasized that hypomagnesemia is common among patients undergoing CRRT.[2] In the following study, magnesium decreased by an average of 17% in 68 patients and increased by 18% in other 43. This means that in our research, CRRT did not lead to new hypo-or hypermagnesemia.

Citrate is the anticoagulant of choice in many parts of the world. Citrate chelates calcium, leading to a decrease in ionized calcium levels, so calcium replacements may be required to prevent hypocalcemia. In the present study, we did not use regional citrate anticoagulation, and all patients received unfractionated heparin (UFH) as anticoagulant. None of the studied patients developed new-onset hypocalcemia as a complication. Akhoundi *et al.* reported decreased level of ionized calcium as the most common clinically significant electrolyte disturbance in patients who underwent CRRT (22%), followed

by increased level of ionized calcium (23%) and hyperphosphatemia (44%).[8] The reason for the difference in the results between the two studies can be related to the mode of anticoagulation used.

CRRT can also play a significant role in managing acidemia or alkalemia by continuous removal of accumulated acid and providing bicarbonate replacement. However, it can lead to metabolic acidosis or alkalosis. In our study, acidemia resolved in most of the patients, and while 83% of patients had acidemia before CRRT, acidemia resolved in 78%. Eleven patients had alkalemia before CRRT, which was subsequently resolved after the completion of therapy.

CRRT can potentially increase the risk of infection, primarily due to the use of catheters, vascular access devices, or circuit contamination. None of the patients in our study showed evidence of infection at the insertion site. Also, we did not detect any signs or symptoms of infection, such as fever, hypotension, or changes in laboratory parameters (such as procalcitonin) during the treatment.

One of the challenges in performing CRRT is maintaining hemodynamic stability (especially avoiding hypotension). Hypotension can occur due to factors such as rapid fluid removal, blood volume shifts, and changes in vascular tone. In this study, CRRT resulted in the cessation or reduction of the need and dependency on vasopressor in 78% of patients. In 27%, the vasopressor was discontinued, and in 50%, the dose of vasopressor was reduced to more than 10% of the initial dose.

CONCLUSION

This study showed a noticeable decrease in platelet counts after CRRT. Patients who require CRRT are prone to a drop in their platelet counts during or after CRRT. In the investigation and analysis of electrolytes and mineral changes, we did not find clinically important new-onset hypo- or hyperkalemia, hypo- or hyper-natremia, hypo- or hyperphosphatemia, hypo- or hypermagnesemia, or calcemic disorders.

The findings of the present study showed that potassium and phosphorus reductions were not statistically significant; there was no significant clinically valuable new-onset electrolyte disorders requiring intervention. CRRT resulted in a significant decrease in vasopressor dose. Finally, in this study, two-thirds of patients were discharged from the hospital following treatment with CRRT.

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ETHICAL CONSIDERATION

This study has been registered with the Ethics Committee of Shahid Beheshti University of Medical Sciences, and based on this approval, it has the authorization to publish the data (IR.SBMU.MSP.REC.1399.752).

AUTHOR CONTRIBUTIONS

Kianoush Kashani contributed to the study design, data analysis and critically revised the manuscript. Amir Ahmad Nassiri and Monir Sadat Hakemi were involved in the study conception, data collection, and manuscript writing and interpretation in nephrology. Antoine Schneider contributed to data interpretation and reviewed the manuscript. Tahereh Sabaghian provided input in the study design and manuscript revision. Azadeh Ahmadi Koomleh contributed to data analysis and interpretation in nephrology. Mir Mohammad Miri assisted with the critical care aspects of the study and reviewed the manuscript. Kiana Entezarmahdi contributed to data collection in the ICU setting and manuscript revision. Taymaz Yousefzad provided expertise in nephrology and contributed to manuscript editing. All authors approved the final manuscript.

CONFLICT OF INTEREST DISCLOSURES

The authors declare that they have no competing interests.

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CONSENT FOR PUBLICATION

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