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# When CRRT on ECMO Is Not Enough for Potassium Clearance: A Case Report

**Janice A. Tijssen[1] and Guido Filler[1,2,3]**

## Abstract

**Background:** Continuous renal replacement therapy (CRRT) is an excellent method used to remove fluid and solutes. It may also reduce the systemic inflammatory response for patients on extracorporeal membrane oxygenation (ECMO) support. The objective of this report is to describe a case where CRRT in combination with ECMO was insufficient to control hyperkalemia.
**Methods:** We report the case of an adolescent patient with refractory symptomatic hyperkalemia due to substantial rhabdomyolysis in which CRRT insufficiently cleared the patient's excess potassium.
**Results:** Intermittent hemodialysis (IHD) was added and proved successful. The patient was weaned off ECMO, CRRT, and IHD, and his cardiac and renal function eventually normalized.
**Conclusions:** Two important lessons can be learned from this case report: (1) If CRRT is insufficient in achieving a desirable potassium balance, additional IHD should be considered and (2) separate IHD access should be considered to improve efficacy.

## Abrégé

**Contexte :** La thérapie de remplacement rénal continue (TRRC) est une procédure très efficace pour éliminer les solutés et les fluides. Elle peut également contribuer à la diminution de la réponse inflammatoire systémique chez les patients sous oxygénation extracorporelle par membrane (technique ECMO). Ce rapport décrit un cas où la TRRC, en combinaison avec l'ECMO, s'est avérée insuffisante pour réguler l'hyperkaliémie.
**Méthodologie :** Nous rapportons le cas d'un patient adolescent présentant une hyperkaliémie symptomatique réfractaire due à une rhabdomyolyse substantielle chez qui la TRRC s'est avérée insuffisante pour éliminer l'excédent de potassium.
**Résultats :** Chez ce patient, l'ajout de l'hémodialyse intermittente s'est avéré un succès. On a par la suite cessé les traitements (TRRC, EMCO et hémodialyse intermittente) et les fonctions cardiaque et rénale du patient sont revenues à la normale.
**Conclusions :** Ce rapport de cas nous permet de tirer deux conclusions importantes. D'abord, qu'il convient d'envisager l'ajout d'un traitement par hémodialyse intermittente dans les cas où la TRRC est insuffisante pour assurer l'équilibre du potassium. Ensuite, que pour améliorer l'efficacité de l'hémodialyse intermittente, il est important de lui assurer un accès indépendant.

## Keywords

ECMO, CRRT, IHD, hyperkalemia, pediatrics

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## What was known before

While continuous renal replacement therapy (CRRT) is the default renal replacement method in patients with acute kidney injury (AKI) and extracorporeal membrane oxygenation (ECMO), the clearance of potassium and other small solutes is superior on hemodialysis.

## What this adds

Consultation with a nephrologist can assist the intensivist with the choice of the best method for renal replacement

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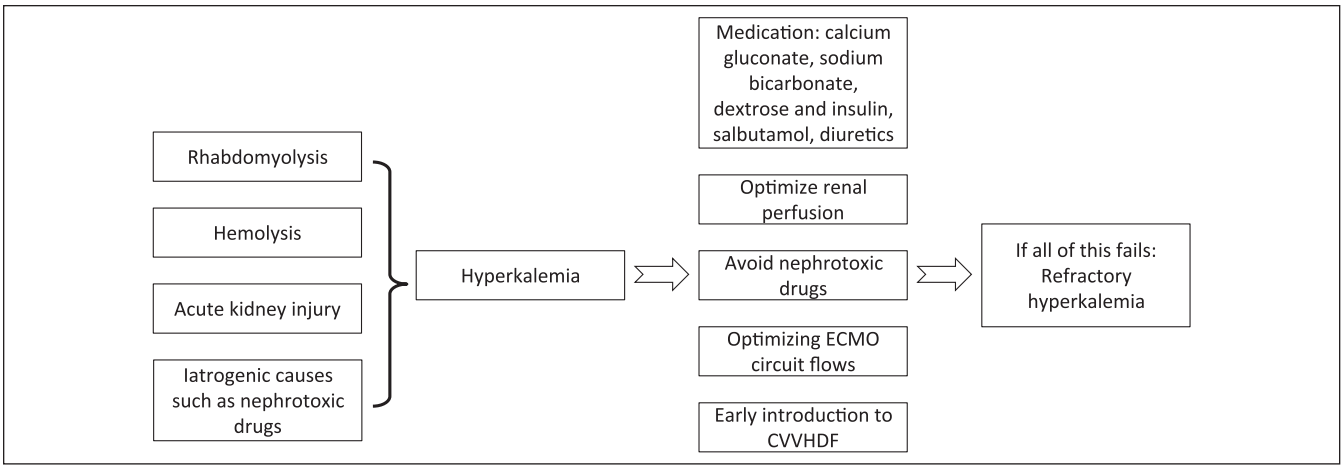


**Figure 1.** Etiology and management of hyperkalemia.
*Note.* ECMO = extracorporeal membrane oxygenation; CVVHDF = continuous venovenous hemodiafiltration.

therapy, specific to the therapeutic goal. CRRT may not always compensate for multiple sources of endogenous potassium and thus may not adequately clear potassium.

## Introduction

Acute kidney injury (AKI) is very common in patients placed on extracorporeal membrane oxygenation (ECMO) for cardiac failure.[1,2] This result is multifactorial: hypoperfusion from a low cardiac output, nonpulsatile renal blood flow in venoarterial-ECMO (VA-ECMO), ischemia/reperfusion injury, systemic inflammatory response syndrome, and hemolysis.[3-5] AKI can also have direct negative effects on the heart.[6] The etiology of the resulting cardiac damage is also multifactorial and includes systemic inflammatory response syndrome with increased vascular permeability, electrolyte disturbances, acidosis, and uremia. These influences lead to a poor prognosis for patients on VA-ECMO for cardiac failure who develop AKI.[2,7-12]

Accordingly, initiating continuous renal replacement therapy (CRRT) early in patients on ECMO may be beneficial as it ameliorates the impact of AKI as shown by Antonucci et al.[13] CRRT is excellent for removing fluid and solutes and may reduce the systemic inflammatory response.[14]

## Methods

We present the case of an adolescent who underwent cardiogenic shock and required VA-ECMO. He developed refractory hyperkalemia and worsening azotemia from multiple causes despite early initiation and optimization of CRRT. Adding intermittent hemodialysis (IHD) successfully led to an improvement in the patient's hyperkalemia and azotemia. There are little data describing the addition of IHD to continuous venovenous hemodiafiltration (CVVHDF) for a patient on VA-ECMO, especially in pediatrics. Informed consent for this report was obtained both from the patient and from his parents.

## Results

A 17-year-old male (47 kg) with a history of Crohn disease presented to our hospital in cardiogenic shock. He was treated with broad-spectrum antibiotics for septic shock and was aggressively supported with fluid boluses, inotropes, vasoactives, and steroids. He also developed a pericardial effusion that required the percutaneous drainage of 150 mL of serous fluid. Within a few hours of admission and despite these interventions, he progressed to cardiac standstill and required extracorporeal cardiopulmonary resuscitation, that is, he was cannulated to a VA-ECMO circuit while cardiopulmonary resuscitation was performed.

Within 24 hours of ECMO cannulation, the patient developed hyperkalemia. The etiology of the hyperkalemia was thought to be multifactorial. The patient had significant hemolysis, rhabdomyolysis, and AKI. The hyperkalemia (8.2 mmol/L) was refractory despite the administration of low-dose dopamine, encouraging urine output with diuretics, optimizing ECMO circuit flows to minimize hemolysis, minimizing nephrotoxic medications, and giving medications for hyperkalemia (sodium bicarbonate, diuretics, insulin and dextrose, and salbutamol, all within a 6-hour window) (Figure 1). The hyperkalemia produced a wide Q wave, R wave, and S wave (QRS) complex (despite the administration of calcium infusions and normal ionized calcium values on point-of-care testing), and the patient developed anuria. His laboratory results at the time were as follows: urea 12.9 mmol/L (normal ≤8.3), creatinine 234 μmol/L (normal = 62-120), phosphate 2.73 mmol/L (normal = 0.8-1.33), creatine kinase >25 300 U/L (normal ≤190), haptoglobin <0.7 g/L (normal = 0.2-2.26), total bilirubin 21.3 μmol/L (normal = 3.4-17.1) and direct bilirubin 6.9 μmol/L (normal = 0-5), lactate dehydrogenase 1219 U/L (normal ≤225), and lactate 7.9 mmol/L (normal ≤2.4).

We introduced CVVHDF to the ECMO circuit proximal to the oxygenator using the Prismaflex system (Figure 2). The CRRT circuit was connected in parallel to avoid high access pressures, with the outflow line originating from the

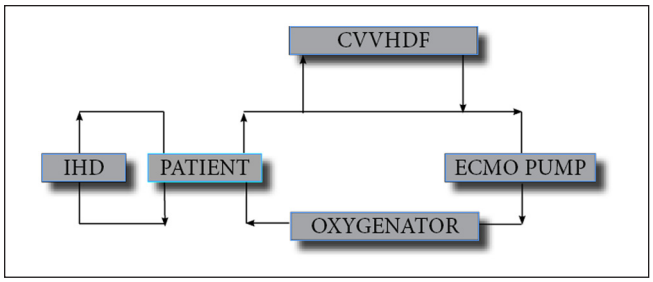


**Figure 2.** Circuit setup.
*Note.* CVVHDF = continuous venovenous hemodiafiltration; IHD = intermittent hemodialysis; ECMO = extracorporeal membrane oxygenation.

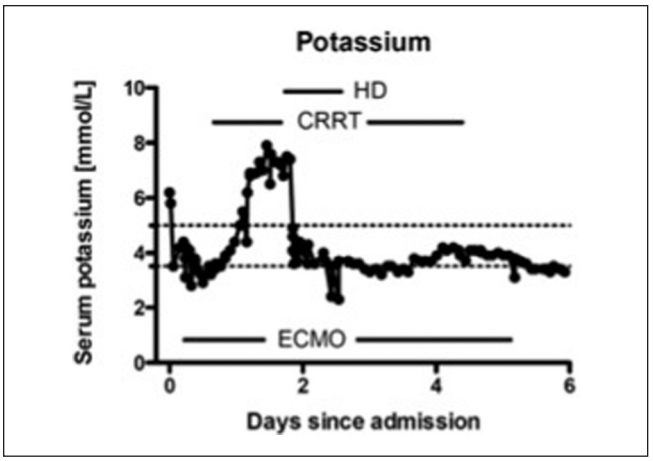


**Figure 3.** Serum potassium in the week following admission.
*Note.* The patient only remained normokalemic on CRRT for a few hours. "Renal dose" dopamine was then started, ventolin was given, nephrotoxic drugs were discontinued, sodium bicarbonate was given via IV, and high doses of furosemide (3 mg/kg in repeated boluses), insulin, and dextrose were administered. As these initiatives were unsuccessful, the dialysis rate of the Prismaflex machine was increased to 6.0 L/h for a total flow rate of 8000 mL/h (dialysate, replacement plus PBP) until day 4 of the CRRT treatment. As none of these concurrent measures were able to lower the patient's potassium level, additional IHD was started with 4-hour treatment cycles interrupted by an 8-hour break; the second treatment caused hypokalemia, so the treatment was stopped. CVVHDF was discontinued after 4.5 days. The patient then received CVVHD until day 13 and was then continued on IHD for a total of 7 weeks, until the AKI resolved. IV = intravenous; CRRT = continuous renal replacement therapy; ECMO = extracorporeal membrane oxygenation; CVVHDF = continuous venovenous hemodiafiltration; CVVHD = continuous venovenous hemodialysis; PBP = pre-blood pump; IHD = intermittent hemodialysis; AKI = acute kidney injury.

patient's venous line and the inflow line attached to the patient's arterial cannula before the oxygenator. Nonetheless, high access pressures from the ECMO circuit limited the blood flow rate, so it was set at 150 mL/min. The pre-blood pump (PBP) was set to 1000 mL/h, which is the rate given on standard preprinted orders. The dialysate (PrismaSol 0) rate was set to 1000 mL/h, which is also the rate on standard preprinted orders, and increased to 6000 mL/h, while the postfilter replacement (PrismaSol 0) rate was set to 1000 mL/h. The Prismaflex has broad flow rate capabilities for blood flows, dialysate, and replacement rates and can deliver up to 8000 mL/h of dialysate (replacement plus PBP). Potassium levels did not improve, so we elected to add IHD to improve the potassium balance (Figure 2). An additional central venous line was inserted for this purpose. His IHD treatment duration was 4 hours, with a dialysate flow rate of 800 mL/min (normal dialysate flow for maintenance dialysis 500 mL/min) and a maximal blood flow rate of 300 mL/min (normal pediatric blood flow rate 4-8 mL/kg/min for maintenance dialysis). The maximum blood flow rate that the dialysis machine that we used could produce was 300 mL/min. The dialyzer employed an F200NR filter to achieve maximum clearance. There was no potassium in the dialysate. We were successful in reducing potassium levels (Figure 3), and the ECG changes normalized with the additional hemodialysis.

After normalization of the hyperkalemia, the patient's CVVHDF was discontinued after 4.5 days and switched to continuous venovenous hemodialysis (CVVHD). The patient was weaned off ECMO after 1 week, came off CVVHD after 13 days, and continued IHD 3 to 4 times per week for a total of 7 weeks until the AKI improved. His cystatin C estimated glomerular filtration rate (eGFR)[15] was 23 (normal ≥ 91) 3 weeks after presentation and is now (6 months later) only mildly impaired at 71 mL/min/1.73 $m^2$(chronic kidney disease [CKD] stage II, normal eGFR at this age 90-135 mL/min/1.73 $m^2$). His urea and creatinine normalized within 3 months of presentation and have remained normal.

## Discussion

The concomitant use of CRRT and ECMO is common. In one case series, 71% of pediatric cardiac patients on ECMO suffered AKI.[2] Of 155 pediatric patients cannulated onto VA-ECMO for heart failure secondary to myocarditis, 42 (27%) patients required dialysis.[12] In a large series of pediatric patients who required ECMO for a variety of indications, 144 (41%) required CRRT.[16] CRRT is typically sufficient for managing fluid overload, acidosis, and electrolyte disturbances. We present a case where CRRT (in this case CVVHDF) was not sufficient in managing severe refractory hyperkalemia despite maximal settings on CVVHDF and ongoing medical management of the hyperkalemia. While we cannot ascertain beyond a doubt that IHD really had to be introduced, the intensivist felt that medical treatment was exhausted and that a higher blood flow (the treatment was already at maximum with 8 L/h) was unacceptable because of the high access pressures. High access pressures on CVVHDF connected to an ECMO circuit are a documented complication.[17] The refractory hyperkalemia was likely due to the patient's continuing massive rhabdomyolysis in addition to hemolysis secondary to ECMO. The addition of IHD rapidly reduced potassium levels in a case of severe ongoing rhabdomyolysis.

It is important for a clinician to consider high-dose CRRT in patients like this when AKI develops, and if this fails, adding IHD to CRRT or replacing CRRT with IHD if a patient

exhibits refractory AKI and persistent hyperkalemia while on ECMO. Early involvement of the nephrology service may have further expedited this process. In our case, the potassium generation exceeded the potassium removal with the maximum treatment delivery of 8 L on CRRT, which is the main reason why IHD was added. In addition to the negative effects of AKI on cardiac function discussed in the "Introduction" section, it is important to reduce severe and persistent hyperkalemia because of its arrhythmogenicity. There is evidence of increased mortality in pediatric patients with cardiac arrhythmias while on ECMO.[12] Our patient's QRS complex widening on ECG resolved with the initiation of IHD.

A good functioning vascular access is an essential component for adequate renal replacement therapy for AKI. We used the existing ECMO access for the CRRT in parallel rather than in series, due to the well-described high access pressures that may be associated with having all blood passing through both ECMO and CRRT in a sequential setup.[17] The parallel setup may, however, have limited the potassium clearance as not all the blood going through the ECMO circuit would be processed by the CRRT.[18] To maximize the efficacy of IHD, we elected to utilize the right jugular vein for separate IHD access as this is the preferred insertion site for a temporary dialysis catheter.[18] We hypothesize that this was an important component of our successful treatment, although we cannot prove that CRRT through separate access might have been more efficient than our chosen setup in removing the potassium. Indeed, the dialysis prescription of the CVVHDF was low, and perhaps the refractory hyperkalemia could have been avoided with had a much higher prescription been introduced earlier.

In the latest Extracorporeal Life Support Organization (ELSO) report, between 35% and 52% of pediatric patients who required ECMO for cardiogenic shock survived to discharge or transfer.[19] The survival rate was lower in those who underwent extracorporeal cardiopulmonary resuscitation (E-CPR) and further decreased in those who developed acute renal failure.[2,12,16,20] Our patient would have had ongoing cardiac insult, undesirable potassium levels despite the use of CRRT, and a lower likelihood of recovery if we had not aggressively added IHD to correct the potassium imbalance. Of course, IHD could have been chosen instead of CRRT, but this has the disadvantage of discontinuous fluid removal. Still, adding IHD can pose some risks. Large fluid shifts in intermittent dialysis can create hemodynamic instability, and there is a risk of catastrophic bleed when inserting a central access line in an anticoagulated patient. We considered using peritoneal dialysis, but it is not as effective as IHD for clearing solutes.

## Conclusion

To conclude, CRRT may not always compensate for multiple sources of endogenous potassium and thus may not adequately clear potassium. We present the unique case of an adolescent on VA-ECMO for cardiogenic shock who survived and regained full renal and cardiac function after IHD was added to CRRT to manage his persistent refractory hyperkalemia. There are 2 important teachable moments: First, in consultation with a nephrologist, consider adding an additional renal replacement modality if the balance of any uremic toxin remains unfavorable despite optimizing CRRT. Given that potassium is best cleared by conventional hemodialysis, our choice was IHD. Second, consider separate vascular access to maximize the efficacy of the additional renal replacement therapy.

### Ethics Approval and Consent to Participate

Ethics approval is waived for a case report in our institution.

### Consent for Publication

Written informed consent to publish was obtained from the patient and the caregivers.

### Availability of Data and Materials

Not applicable.

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### Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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