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Beyond the usual: A focus on infrequent complications of CRRT

Gonzalo Ramírez-Guerrero [a,b,*], Cristian Pedreros-Rosales [a,c], David Ballesteros [d], Mitchell Rosner [e], Claudio Ronco [b,f]

[a] Nephrology Service, Hospital Las Higueras, Talcahuano 4270918, Chile
[b] International Renal Research Institute of Vicenza (IRRIV), Vicenza, Italy
[c] Departamento de Medicina Interna, Facultad de Medicina, Universidad de Concepción, Concepción 4070386, Chile
[d] Division of Nephrology, Department of Internal Medicine, Universidad del Cauca, Hospital Universitario San José de Popayan, Colombia
[e] Division of Nephrology, University of Virginia Health System, 1300 Jefferson Park Avenue, Charlottesville, Virginia, USA
[f] Department of Medicine (DIMED), Università degli Studi di Padova, Padova, Italy

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ABSTRACT

Continuous renal replacement therapy (CRRT) is a cornerstone in the management of critically ill patients with acute kidney injury and fluid-electrolyte disturbances. While widely adopted and considered safe, CRRT is not exempt from complications—some of which, though infrequent, can have serious consequences for patient outcomes and circuit performance. In this review, we present a focused analysis of underrecognized but clinically relevant complications associated with CRRT, including hypertriglyceridemia-related circuit failure, euglycemic ketoacidosis, cold agglutinin-mediated filter clotting, hypophosphatemia-induced weaning failure, hypothermia, and citrate-induced magnesium depletion. Each section highlights the pathophysiology, risk factors, clinical implications, and preventive strategies for these events. Notably, we emphasize the importance of early recognition and targeted monitoring, as many of these complications may develop silently or mimic more common issues. Practical recommendations are provided to guide clinicians in optimizing CRRT delivery and minimizing the risk of adverse events. By shedding light on these rare complications, we aim to improve awareness, encourage proactive surveillance, and ultimately enhance the safety and effectiveness of extracorporeal therapy in critical care settings.

1. Introduction

Continuous renal replacement therapy (CRRT) has become an essential modality for managing critically ill patients with acute kidney injury, providing gentle solute clearance and hemodynamic stability. Despite its advantages over intermittent dialysis, CRRT is associated with a diverse array of complications that can undermine treatment efficacy and patient outcomes. While the most commonly recognized issues include circuit clotting, bleeding, and electrolyte disturbances, a growing body of evidence highlights less frequent but clinically significant complications that may remain underappreciated in daily practice. These uncommon events—such as severe hypertriglyceridemia-related circuit failure, euglycemic ketoacidosis, profound hypophosphatemia, hypomagnesemia, and inadvertent hypothermia—pose unique diagnostic and therapeutic challenges (Fig. 1). This review aims to synthesize current knowledge on these infrequent or overlooked complications, elucidate their pathophysiological mechanisms, and outline preventive and management strategies to improve the safety and effectiveness of CRRT.

Although the reported incidence of hypophosphatemia, hypomagnesemia, and hypothermia during CRRT can be substantial (30–80 % depending on study design and CRRT prescription), these complications remain underrecognized at the bedside, frequently misattributed to more common issues or insufficiently monitored. We therefore included them alongside truly rare events such as hypertriglyceridemia-induced circuit failure and cold agglutinin disease to highlight phenomena that are either infrequent or underappreciated in daily practice yet carry significant consequences for patient outcomes.

The discussion is organized to begin with complications that directly affect the circuit (hypertriglyceridemia, cold agglutinin disease), then move to systemic disturbances (hypothermia, euglycemic ketoacidosis), and end with electrolyte disorders (hypophosphatemia, hypomagnesemia).

* Corresponding author at: Alto Horno 777, 4260000 Talcahuano, Chile.
E-mail address: ramirezguerrero.g@gmail.com (G. Ramírez-Guerrero).

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2. Hidden in plain sight: hyperlipidemia as a cause of CRRT inefficacy

While circuit failure in CRRT is often attributed to well-recognized factors such as inadequate anticoagulation, catheter malfunction, or high filtration fraction, hypertriglyceridemia represents a rare yet increasingly reported cause of premature filter clotting. Evidence to date is limited almost exclusively to isolated case reports and small case series, with a recent review identifying 11 published cases of CRRT circuit clotting attributed to hypertriglyceridemia, most related to propofol infusion and, less frequently, to parenteral nutrition [1]. Accordingly, the true incidence remains undefined but is likely underestimated. This phenomenon is typically underrecognized, particularly when it arises in the context of lipid-based therapies routinely administered in the intensive care unit.

Hypertriglyceridemia may impair CRRT performance through a combination of mechanisms, including increased plasma viscosity, formation of lipid-fibrin microaggregates, and physical obstruction of the hemofilter fibers by lipid droplets. Electron microscopy studies have confirmed the presence of triglyceride-rich deposits and fibrin strands in occluded filters, providing a structural correlate to this functional complication [1,2].

The most frequently implicated therapy is propofol, a lipid-based sedative whose prolonged or high-dose administration can significantly elevate serum triglyceride levels. Beyond its lipid content, propofol may induce mitochondrial dysfunction by impairing β-oxidation of free fatty acids and inhibiting electron transport chain activity, leading to an accumulation of unmetabolized lipids in the plasma [3–5]. This metabolic disturbance contributes to hyperlipidemia and may further exacerbate systemic toxicity, as seen in propofol infusion syndrome (PRIS), a rare but life-threatening entity characterized by severe metabolic acidosis, cardiac arrhythmias, cardiovascular collapse, rhabdomyolysis, and renal failure [3]. The syndrome is more likely to occur with infusion rates exceeding 4–5 mg/kg/h and durations beyond 48 h, particularly in the presence of additional risk factors such as critical illness, high-dose vasopressor use, corticosteroids, and young age [3,6–8]. However, other common ICU interventions such as total parenteral nutrition (TPN) and intravenous lipid emulsions (ILE) have also been associated with severe hypertriglyceridemia and CRRT dysfunction. A recent case series found that nearly one-third of reported cases were unrelated to propofol, highlighting that any lipid-rich infusion poses a risk [1].

Clinically, this complication often manifests as recurrent circuit clotting or high transmembrane pressures despite adequate anticoagulation, accompanied by the visual cue of lipemic or "milky" blood within the circuit tubing. Most reported cases have occurred with serum triglyceride levels exceeding 1000 mg/dL, and resolution was typically observed only after discontinuation of the offending agent, with or without additional interventions such as insulin, heparin, or lipopheresis (Fig. 2) [2,9–11].

Given the increasing use of lipid-based therapies in critically ill patients, routine monitoring of triglyceride levels is advisable, particularly when facing unexplained circuit dysfunction. Early recognition may avoid unnecessary filter losses, optimize solute clearance, and support timely adjustments in sedation or nutritional strategies.

The treatment of hypertriglyceridemia in critically ill patients should begin with immediate discontinuation of exogenous lipid sources, such as propofol, intravenous lipid emulsions, or total parenteral nutrition. Intravenous insulin infusion is the cornerstone of therapy, even in non-diabetic patients, due to its ability to stimulate lipoprotein lipase and enhance triglyceride clearance. Typical regimens include 0.05–0.1 U/kg/h with concomitant glucose administration to maintain euglycemia [12]. Heparin may transiently increase circulating lipoprotein lipase but is no longer routinely recommended due to rebound hypertriglyceridemia and enzyme depletion. In cases of extremely elevated triglycerides (>1000–2000 mg/dL) or when complications such as pancreatitis or CRRT circuit failure occur, therapeutic plasma exchange may be considered. However, recent prospective studies have failed to demonstrate a consistent benefit in organ failure resolution or mortality, and its use should be individualized [12].

Practical Recommendations

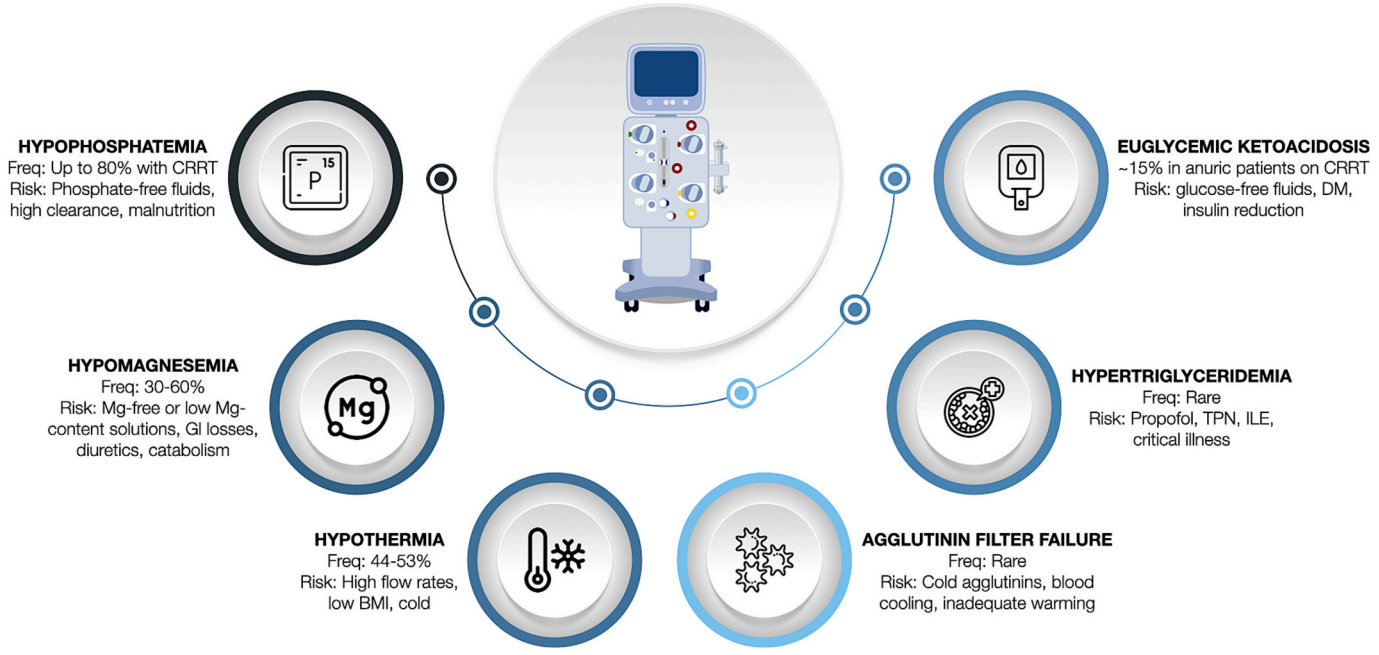

Fig. 1. *Summary of infrequent complications associated with continuous renal replacement therapy (CRRT).*
CRRT: Continuous Renal Replacement Therapy, DM: Diabetes Mellitus, ILE: Intravenous Lipid Emulsions, TPN: Total Parenteral Nutrition, Mg: Magnesium, GI: Gastrointestinal, BMI: Body Mass Index.

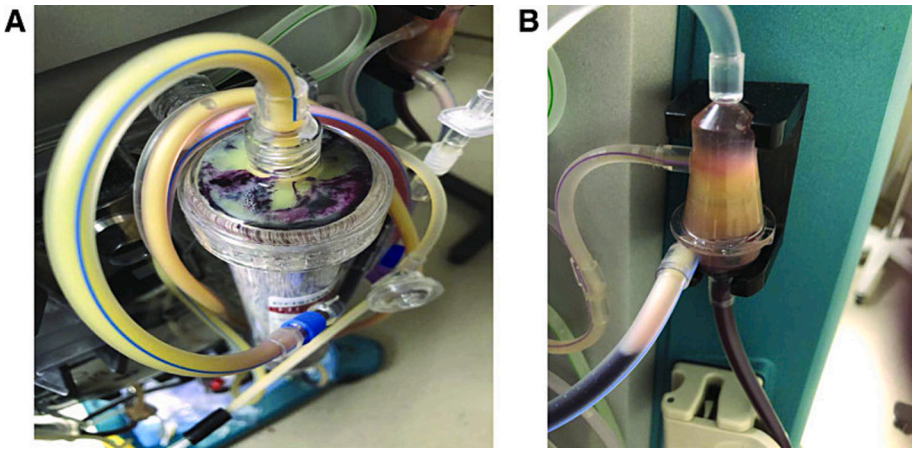

Fig. 2. *Lipoid material clogging the CRRT circuit in a patient with hypertriglyceridemia related to propofol infusion.*
The image demonstrates lipid deposition within the filter (A) and deaeration chamber (B), leading to recurrent clotting despite adequate regional citrate anticoagulation. Reproduced with permission from Wolters Kluwer Health, Inc.: Whitlow M, Rajasekaran A, Rizk D. Unusual Cause for Continuous Renal Replacement Therapy Filter Clotting. *Kidney360*. 2020;1(3):235–237. https://doi.org/10.34067/KID.0000092019

1. **Monitoring:** Check serum triglyceride levels regularly in critically ill patients receiving lipid-based therapies (e.g., propofol, TPN, ILE), particularly when facing unexplained circuit dysfunction.
2. **Recognition:** Suspect hypertriglyceridemia when recurrent circuit clotting or high transmembrane pressures occur despite adequate anticoagulation, especially if lipemic or "milky" blood is visible in the tubing.
3. **Management:** Immediately discontinue exogenous lipid sources. Initiate intravenous insulin infusion (0.05–0.1 U/kg/h with glucose supplementation) to enhance clearance. Avoid routine heparin use due to rebound effects.
4. **Escalation:** Consider therapeutic plasma exchange for extremely high triglycerides (>1000–2000 mg/dL) or when associated complications such as pancreatitis or refractory CRRT dysfunction develop.

3. A hidden agglutination: cold agglutinin disease triggering early CRRT circuit failure

Cold agglutinin disease (CAD) is a rare form of autoimmune hemolytic anemia characterized by the presence of cold-reactive autoantibodies, typically immunoglobulin M (IgM), which bind to erythrocytes at temperatures substantially below core body temperature—commonly between 0 and 4 °C [13,14]. Less frequently, IgG- or IgA-mediated cases have been described. CAD can arise idiopathically or as a secondary phenomenon triggered by infections (e.g., *Mycoplasma pneumoniae*, *Legionella pneumophila*, Epstein-Barr virus, hepatitis B or C, cytomegalovirus), lymphoproliferative disorders, or drug exposures [13,15].

The pathogenic process is predominantly complement-mediated hemolysis, which can occur intravascularly or extravascularly. As blood circulates through cooler peripheral tissues (temperatures as low as 28–30 °C), cold agglutinins transiently bind to erythrocyte membranes, leading to agglutination and subsequent hemolysis [15]. While this phenomenon is classically associated with cold-induced symptoms in the extremities, it can have unrecognized implications during extracorporeal therapies, particularly CRRT.

In patients requiring extracorporeal support for AKI, the exposure of circulating blood to non-physiologic temperatures within the circuit may precipitate acute hemagglutination. This process increases filter resistance, elevates transmembrane pressures, and can rapidly culminate in circuit clotting. A representative example involved a 60-year-old man with severe CAD who required therapeutic plasma exchange and continuous venovenous hemodiafiltration (CVVHDF). A few minutes after initiating therapy, which included pre-filter replacement fluids at room temperature (approximately 23 °C), the patient developed sudden high filter pressures and visible agglutination in the extracorporeal tubing, prompting abrupt cessation of treatment (Fig. 3). Subsequent implementation of inline blood warming to 36–37 °C and avoidance of room-temperature solutions enabled successful continuation of extracorporeal support [16,17].

Although infrequent, CAD should be considered in cases of recurrent, unexplained circuit clotting—particularly when accompanied by visible erythrocyte aggregates or rapid filter thrombosis early during therapy. Early recognition and targeted interventions can prevent repeated circuit loss and ensure continuity of renal replacement support.

Practical Recommendations

Monitoring and recognition: Suspect CAD in patients with recurrent, unexplained circuit clotting, especially if accompanied by visible red cell aggregates or early filter thrombosis.

1. **Temperature management:** Pre-warm the blood outlet line to 36–37 °C prior to passage through the filter, using heating devices that may be external (e.g., inline warmers) or adapted from integrated systems in older CRRT machines, since this option is not available in most new generation platforms.
2. **Fluid strategy:** Avoid room-temperature replacement fluids (e.g., pre-filter solutions), as they exacerbate cold-induced agglutination.
3. **Underlying condition:** Address the primary disease process (e.g., infection or lymphoproliferative disorder) to reduce cold agglutinin titers over time

4. A silent acidosis: euglycemic ketoacidosis during CRRT

Euglycemic ketoacidosis (EKA) is an underrecognized but potentially life-threatening complication of CRRT, particularly when using glucose-free dialysate or replacement solutions. It is defined by the triad of normoglycemia (glucose <250 mg/dL), high anion gap metabolic acidosis, and elevated serum ketones, typically β-hydroxybutyrate [18,19]. While often associated with sodium–glucose cotransporter-2 (SGLT2) inhibitors, fasting, or pregnancy, EKA has increasingly been reported in critically ill patients undergoing CRRT without these classical triggers. In a prospective ICU cohort, EKA occurred in 18 patients—approximately 15 % of those treated with phosphate-containing glucose-free solutions [20]. A separate series of eight diabetic patients reported similar findings when caloric intake was inadequate during glucose-free CRRT [18]. Beyond these cohorts, the literature consists of multiple isolated case reports describing EKA in diverse clinical settings, including patients on glucose-containing CRRT solutions when

Fig. 3. *Cold agglutinin-mediated hemagglutination in the CRRT circuit.*
The image shows early circuit clotting triggered by cold agglutinin disease. Notably, the patient also developed peripheral livedo reticularis due to cold-induced microvascular agglutination. Prompt recognition and the use of blood warming and warmed solutions are critical to prevent recurrent filter loss.

nutritional support or insulin supplementation was insufficient [17,19,21,22]. Taken together, these data suggest that while rare, EKA may be underrecognized, and vigilance is warranted when persistent high anion gap metabolic acidosis is observed despite adequate solute control during CRRT.

The pathophysiology of EKA in this setting is multifactorial. First, nutritional deficits are common early in critical illness, as enteral feeding is often delayed due to hemodynamic instability or gastrointestinal intolerance. Second, CRRT continuously removes glucose and other low-molecular-weight solutes, with glucose losses ranging from 40 to over 100 g per day depending on the prescription and dialysate composition [20,21]. This contributes to a negative caloric balance and depletion of glycogen stores, promoting ketogenesis. Third, in patients with diabetes mellitus or critical illness–induced insulin resistance, relative insulin deficiency further tilts the hormonal milieu toward a catabolic state marked by increased glucagon-to-insulin ratio, lipolysis, and hepatic ketone body production (Fig. 4) [23].

Notably, EKA has been observed even with the use of glucose-containing dialysates, especially when caloric intake remains suboptimal or insulin therapy is reduced due to normoglycemia [22]. The clinical presentation may be subtle. In multiple case series, patients demonstrated persistent high anion gap metabolic acidosis despite improving lactate and uremic parameters, prompting further workup and eventual diagnosis of ketosis based on β-hydroxybutyrate levels above 1.5–3.0 mmol/L [18,19,24].

Several case series have highlighted the reproducibility and clinical significance of this phenomenon. In a report by Ting et al., eight critically ill patients with diabetes mellitus developed euglycemic ketosis during CRRT using glucose-free solutions, despite concurrent improvements in lactate and urea levels [18]. The patients exhibited a median β-hydroxybutyrate of 3.7 mmol/L and were receiving suboptimal caloric intake (median 15 kcal/kg/day) prior to the onset of ketosis. The condition was reversed with dextrose infusion and insulin titration, allowing caloric intake to rise to approximately 25 kcal/kg/day. Similarly,

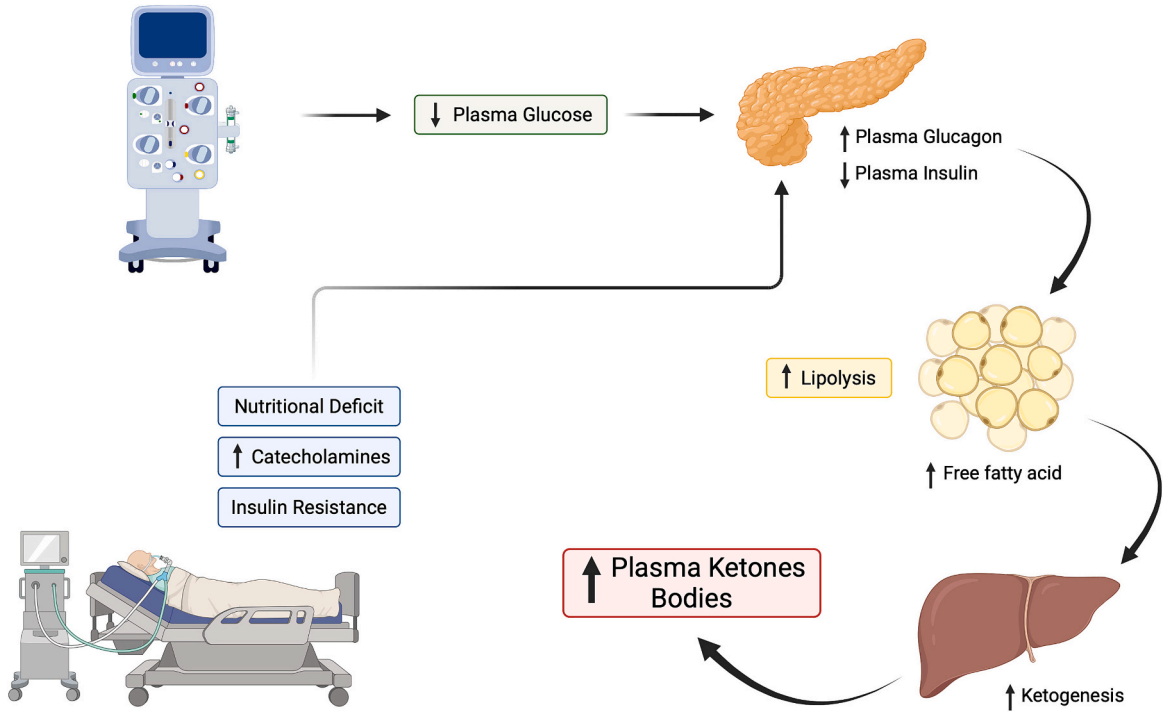

Fig. 4. *Pathophysiological mechanisms of euglycemic ketoacidosis during CRRT.*
Illustration of the multifactorial contributors to ketone body accumulation in critically ill patients, including glucose losses in dialysate, nutritional deficits, insulin deficiency, and increased catabolism leading to high anion gap metabolic acidosis despite normoglycemia.

Coutrot et al. prospectively identified 18 patients (15 % of all anuric patients on CRRT) who developed EKA while receiving phosphate-buffered glucose-free fluids [20]. Most had poor enteral intake and were not on insulin, suggesting a convergence of insulin deficiency, catabolism, and dialytic glucose loss. In both cohorts, the median time to EKA onset ranged from 43 to 72 h after CRRT initiation, underscoring the need for early metabolic surveillance in high-risk individuals.

Management requires prompt recognition. Treatment includes initiating intravenous dextrose (usually 5–10 %) to offset CRRT-associated glucose losses (estimated at 40-100 g over 24 h), thereby permitting safe and continuous insulin infusion, which facilitates suppression of ketogenesis and promotes glucose utilization. In all reported cases, ketosis resolved within 12–24 h following this intervention. Additionally, switching to CRRT fluids with glucose concentrations between 90 and 110 mg/dL and optimizing enteral feeding to reach at least 25–30 kcal/kg/day are critical to prevent recurrence [18,20].

EKA should be suspected in any patient on CRRT who develops new or persistent high anion gap acidosis without elevated lactate, uremia, or drug-related causes. This includes non-diabetic patients and those with apparent normoglycemia. Regular assessment of β-hydroxybutyrate and closer nutritional monitoring may help unmask this complication early. In the differential diagnosis, citrate accumulation related to regional citrate anticoagulation should also be considered.

As awareness of EKA in the setting of CRRT grows, it is essential for clinicians to monitor caloric intake and glucose balance proactively, particularly when using low-glucose or individualized CRRT solutions in patients with high catabolic stress or diabetes.

Practical Recommendations

1. **Monitoring:** Suspect EKA in patients with persistent high anion gap acidosis despite adequate solute control. Measure β-hydroxybutyrate when lactate and uremia do not explain the gap.
2. **Prevention:** Ensure early and adequate caloric intake (≥25–30 kcal/kg/day) and consider CRRT solutions with physiological glucose concentrations when feasible.
3. **Management:** Initiate IV dextrose (5–10 %) to compensate for CRRT-associated glucose losses (≈40–100 g/24 h) and allow safe continuous insulin infusion. Adjust nutritional support accordingly.
4. **Differential diagnosis:** Always rule out citrate accumulation and other drug-related causes of metabolic acidosis.

5. A hidden chill: hypothermia during CRRT

Hypothermia represents a frequent but sometimes overlooked complication of CRRT. Defined as a core body temperature < 36 °C (or ≤ 35 °C in some series), hypothermia arises from sustained extracorporeal heat losses and is reported in up to 44–53 % of critically ill patients receiving CRRT (Fig. 5) [25,26,27]. While this phenomenon has long been acknowledged, its true incidence is likely underestimated due to inconsistent temperature monitoring practices [26].

During CRRT, blood is continuously circulated through extracorporeal tubing and filters, which are exposed to ambient temperatures (typically 20–23 °C), resulting in convective and conductive heat losses [25,27,28]. This thermal gradient is further amplified by high effluent and blood flow rates, the use of unheated dialysate or replacement fluids, and prolonged treatment duration [25,27,28]. Studies have demonstrated that higher dialysate flows and lower body mass index are associated with greater heat dissipation, whereas more advanced blood warming technologies can partially mitigate these effects [25,29]. Yagi et al. prospectively measured blood temperatures in CRRT circuits and confirmed that increasing dialysate flow rates produce a direct and measurable decline in venous return temperatures, underscoring the impact of treatment parameters on thermal balance [28].

Hypothermia is not a benign occurrence. Even moderate reductions in core temperature can impair coagulation, promote arrhythmias, increase oxygen consumption, and precipitate hemodynamic instability [25–27]. Additionally, it may mask infectious fevers or mimic sepsis, complicating diagnostic evaluation [27]. In a large cohort study, lower core temperatures and greater temperature variability were independently associated with higher ICU and 30-day mortality [26]. Multivariate analyses have identified several predisposing factors for CRRT-related hypothermia. These include older age—particularly patients over 70 years, who exhibit nearly a threefold increase in risk—along with lower body weight, which has a protective effect for each kilogram of increase. Other contributors include higher positive fluid balance in the 24 h preceding CRRT initiation, higher CRRT dose prescriptions, and the use of older-generation blood warmers that lack precise thermal

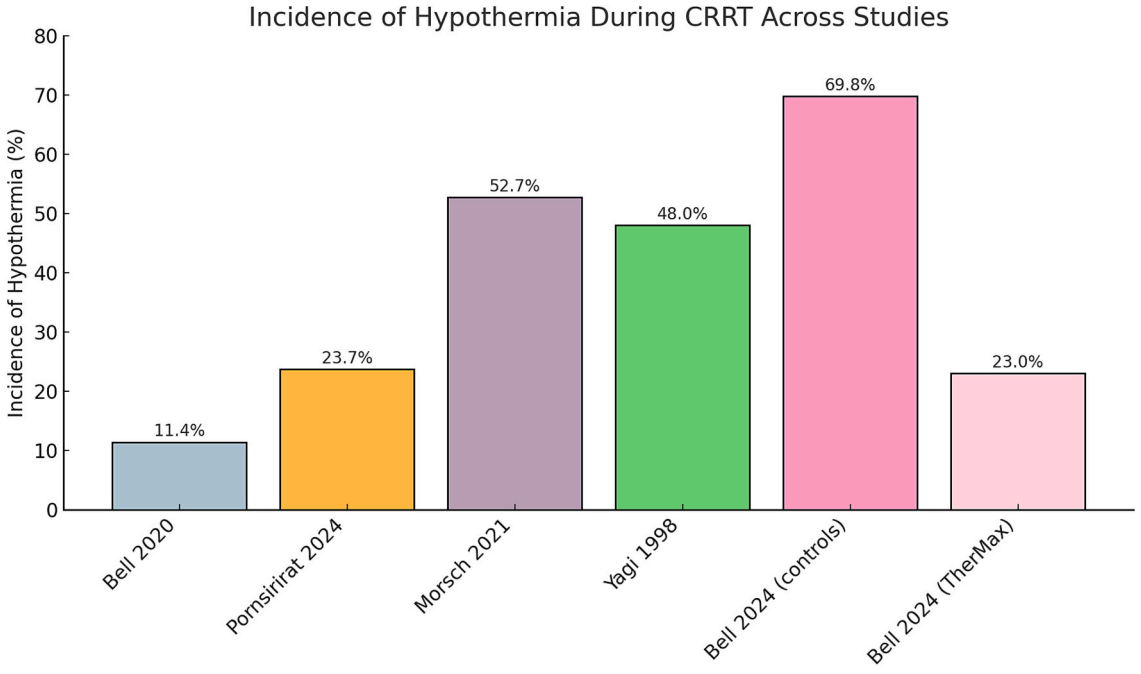

Fig. 5. Incidence of Hypothermia During CRRT Across Studies.

control. Furthermore, the choice of modality also influences risk: CVVHDF has been associated with higher incidence of hypothermia compared to continuous venovenous hemodialysis (CVVHD) (Table 1) [26,27,29].

Core temperature should be measured with reliable techniques, such as bladder or esophageal probes with thermistors, which correlate closely with pulmonary artery values and are widely available in ICUs [30,31]. Nonetheless, practice variability persists, and reliance on peripheral measurements may still lead to underrecognition of CRRT-related hypothermia [30]. Importantly, mild reductions in core temperature reduce oxygen consumption and carbon dioxide production by up to 50 %, a mechanism that underlies the rationale for therapeutic hypothermia in selected clinical settings [32]. Whether such metabolic benefits might confer protection during CRRT remains speculative, as in most critically ill patients the risks of arrhythmia, coagulopathy, and masking of infectious fever outweigh potential advantages.

Preventive strategies include proactive temperature monitoring, warming of dialysate and replacement solutions, use of modern CRRT platforms with integrated precise warmers (such as the PrisMax-TherMax system), thermal blankets, and frequent adjustment of set warmer temperatures [25,29]. Prospective multicenter data have demonstrated that the adoption of next-generation warming devices can reduce the mean hypothermia duration from nearly 7 h to less than 1 h per day of CRRT [29].

Practical Recommendations

- **Routine monitoring**: Measure core temperature regularly to detect and promptly manage hypothermia.
- **Preventive strategies:** Use warming blankets and adjust the room temperature. Pre-warm CRRT fluids or blood lines when feasible using CRRT-integrated technology or approved external devices.
- **Preferred technology**: When feasible, prefer modern CRRT platforms equipped with advanced blood warming systems, as these have shown improved precision and reduced risk of inadvertent hypothermia compared with older devices

6. The hidden depletion: hypophosphatemia-induced respiratory failure in CRRT

Hypophosphatemia is a frequently overlooked complication of CRRT, with reported incidences ranging from 27 % to 78 % depending on dialysis intensity and duration [33,34]. The continuous clearance of phosphate, especially in high-dose or prolonged CRRT regimens, can result in a negative phosphate balance and severe systemic depletion, even in patients who initially present with hyperphosphatemia.

Phosphate plays a pivotal role in cellular energy metabolism, diaphragmatic contractility, red blood cell function, and myocardial performance [35,36]. Profound hypophosphatemia (serum phosphate $<$0.32 mmol/L) has been directly linked to acute respiratory failure through impaired ATP generation in respiratory muscles and defective diaphragmatic function [37,38]. Early studies using nuclear magnetic resonance spectroscopy confirmed sustained energy deficits in skeletal muscle despite serum phosphate correction, suggesting intracellular depletion persists beyond biochemical normalization [36].

In CRRT patients, hypophosphatemia has been independently associated with failure to wean from mechanical ventilation and prolonged respiratory support. In a cohort of 66 ICU patients undergoing ventilatory support, lower serum phosphate levels significantly correlated with weaning failure (1.06 vs 1.18 mmol/L, $p = 0.008$) [39]. Similarly, Demirjian et al. observed in a large prospective study ($n = 321$) that patients with dialysis-induced hypophosphatemia had a 1. 8-fold increased risk of requiring tracheostomy due to prolonged respiratory failure, though mortality at 28 days was unaffected [40].

Table 1
Summary of Hypothermia Incidence, Risk Factors, and Associated Complications in CRRT Across Selected Studies.

Study / Author	Design / N	Modalities	Reported Incidence of Hypothermia	Identified Risk Factors	Associated Complications
Bell et al., 2020 [20]	Observational comparative	Not reported	11.43 % of treatment time < 36 °C with Prismaflex	Use of older-generation systems without precise thermal control	Not specifically reported
Pornsirirat et al., 2024 [21]	Multicenter retrospective / 300 patients	CVVH 276 patients (92.6 %) CVVHDF 7 patients (2.4 %) SCUF 17 Patients (5.7 %)	23.7 % of patients during the first 24 h	Age > 70, low body weight, positive fluid balance, higher CRRT dose prescriptions	Increased ICU mortality
Morsch et al., 2021 [22]	Prospective / 186 patients	CVVHD 96 patients (51.6 %) CVVHDF 90 patients (48.4 %)	52.7 % of patients	Hemodiafiltration, shock	Higher rates of mechanical ventilation, vasopressor use, septic shock, arrhythmias, hemodynamic instability, and increased mortality
Yagi et al., 1998 [23]	Retrospective + prospective study / 72 + 27 patients	Retrospective: SCUF 4 sessions (4.4 %) CAVH 10 sessions (11 %) CVVH 15 sessions (16.5 %) CAVHD 10 sessions (11 %) CVVHD 52 sessions (57.1 %) Prospective: CVVHD all sessions	48 % of patients developed hypothermia (<35.5 °C) during CVVHD; mean duration 2.6 days	Lower body weight, venovenous modalities, higher dialysate flow rates	Prolonged hypothermia; no significant difference in mortality in this cohort
Bell et al., 2024 [24]	Prospective multicenter comparative / 100 patients (TherMax) vs 86 controls	Not reported	77 % of patients remained free from hypothermia (<36 °C); mean duration 0.66 h vs 6.92 h in controls	Use of modern warming technology (TherMax) significantly reduced hypothermia; no additional patient-related risk factors studied	Not reported; study focused on incidence and warming performance

CRRT, Continuous Renal Replacement Therapy; SCUF, Slow Continuous Ultrafiltration; CAVH, Continuous Arteriovenous Hemofiltration; CVVH, Continuous Venovenous Hemofiltration; CAVHD, Continuous Arteriovenous Hemodialysis; CVVHD, Continuous Venovenous Hemodialysis; CVVHDF, Continuous Venovenous Hemodiafiltration; ICU, Intensive Care Unit.

The mechanism is particularly relevant in critically ill patients with baseline malnutrition, sepsis, insulin resistance, or prolonged fasting, all of which compound phosphate loss. Additionally, parenteral nutrition, vasopressor use, and low serum calcium levels have been identified as independent predictors of hypophosphatemia during CRRT [33,40]. Refeeding syndrome should also be considered, as it not only represents a differential diagnosis but may further exacerbate or accentuate phosphate depletion in malnourished patients undergoing CRRT [41,42].

To mitigate this risk, phosphate supplementation strategies have evolved. Multiple studies support the addition of phosphate to dialysate and replacement solutions, as this not only prevents hypophosphatemia but avoids the logistical challenges of intravenous repletion. Pistolesi et al. demonstrated that phosphate-enriched CRRT solutions (1.2 mmol/L) combined with regional citrate anticoagulation effectively maintained normophosphatemia in 88 % of patients without increasing circuit complications [38]. Similarly, Song et al. showed that supplementing phosphate at concentrations of 2.0–3.0 mmol/L reduced the incidence and duration of hypophosphatemia, with serum phosphate normalization within 1–2 days and without significant adverse effects [33]. Concerns regarding calcium–phosphate precipitation within CRRT replacement or dialysate solutions have been evaluated both in vitro and clinically. Available data demonstrate that the addition of phosphate to these fluids remains chemically stable for the duration of clinical use, without evidence of precipitation or alterations in calcium–phosphate balance within the solution bags. [43].

Despite increasing awareness, current clinical practice varies widely, with many centers still relying on reactive supplementation. Recent editorials have emphasized the need for preemptive phosphate strategies, particularly beyond the first 48 h of CRRT [34]. These include standardized repletion protocols and greater adoption of commercially available phosphate-containing solutions, such as Phoxilium® or custom additives.

Clinicians should monitor phosphate levels closely in all CRRT patients, particularly those receiving intensive therapy, and consider proactive supplementation to prevent life-threatening complications like ventilatory failure. Integrating phosphate into CRRT protocols is not only safe but essential for comprehensive electrolyte management in the critically ill.

The target phosphate concentration in CRRT solutions can be titrated to approximately 1.0–1.2 mmol/L, as demonstrated by Troyanov et al. [43], who confirmed in vitro and clinical stability at these levels. Monitoring of serum phosphate should be performed at least every 6–12 h in critically ill patients at risk of rapid shifts. For example, to supplement a 5-liter phosphate-free replacement fluid bag to a final concentration of 1.2 mmol/L, 6 mmol of phosphate are required. With our local monopotassium phosphate 15 % (10 mL ampoules providing 11.02 mmol phosphate and 11.02 mmol potassium), this corresponds to ≈ 5.5 mL per 5-liter bag. The exact volume should be adjusted according to the commercial formulation available and the associated potassium load [43].

Practical Recommendations

1. **Monitoring:** Check serum phosphate regularly (at least every 12 h) in patients on CRRT, with the goal of preventing hypophosphatemia rather than reacting once it occurs.
2. **Replacement strategy:** Prefer CRRT solutions already containing phosphate to avoid manipulation. If unavailable, supplement replacement or dialysate fluids with phosphate to reach ~1.2–2.4 mmol/L in the solution, aiming to prevent a decline in serum phosphate.
3. **Special considerations:** Account for refeeding syndrome, the patient's serum potassium level if using monopotassium phosphate, and other risk factors such as vasopressor therapy or low serum calcium.

7. The hidden loss: magnesium depletion and chelation during CRRT

Magnesium is a crucial cofactor in cellular homeostasis, contributing to enzymatic reactions, myocardial stability, and neuromuscular excitability. In critically ill patients undergoing CRRT, hypomagnesemia is common and clinically significant, yet often under-recognized [44]. Clinically, deficiency may present with neuromuscular symptoms (tremor, tetany, seizures), cardiovascular complications (QT prolongation, ventricular arrhythmias, atrial fibrillation), and refractory hypokalemia or hypocalcemia, all of which can worsen outcomes in the ICU [45]. The extracorporeal circuit introduces substantial magnesium losses, especially when RCA is used, as citrate chelates magnesium in a manner similar to calcium.

Clinical studies report that approximately 30–60 % of patients undergoing citrate-based CRRT experience low serum magnesium levels at some point during therapy, particularly in the context of high-effluent therapies or magnesium-poor solutions, although most cases are mild and manageable with replacement [46,47]. Under normal physiological conditions, serum magnesium exists in three forms: about 55–70 % as biologically active ionized magnesium (Mg^{2+}), 20–30 % bound to proteins (mainly albumin), and the rest complexed with anions such as phosphate or citrate. Notably, ionized magnesium is the physiologically relevant form, but clinical practice typically relies on total serum magnesium, which often poorly reflects the active fraction—particularly during critical illness or citrate exposure. Studies have shown that ionized and total magnesium correlate poorly, and relying solely on total magnesium may fail to identify functionally significant deficits [48].

The extent of magnesium removal during CRRT depends on the effluent dose, anticoagulation strategy, and, critically, the magnesium content of replacement and dialysate solutions. Most standard CRRT fluids contain only 0.50 mmol/L (1.22 mg/dL) of magnesium, which is frequently inadequate to maintain balance in RCA protocols. Using solutions with higher concentrations (e.g., 0.75 mmol/L or 1.82 mg/dL) can better support homeostasis and reduce the need for intravenous supplementation (Table 2).

In a pivotal study by Brain et al., magnesium loss during CVVHDF was significantly greater with citrate circuits compared to heparin (median 26.2 vs. 17.3 mmol/day) [49]. Arterial magnesium levels dropped rapidly after supplementation, with a median half-life of just 4.7 h, often leaving prolonged periods of subtherapeutic concentrations—highlighting how once-daily monitoring may be insufficient.

These findings are reinforced by case reports such as DePriest et al., where a patient required up to 64 g of magnesium sulfate over 12 days of RCA-CRRT, despite protocolized replacement using low-magnesium fluids [50]. This illustrates how solution composition can critically impact magnesium balance.

Unlike calcium, magnesium is not replaced continuously, and monitoring typically relies on total serum levels, even though both total and ionized magnesium decline with citrate chelation. The lack of routine ionized magnesium measurement further complicates management. Clinically, deficiency may manifest as neuromuscular irritability, arrhythmias, or impaired recovery, and observational data associate hypomagnesemia with increased risk of atrial fibrillation and mortality [51].

Emerging evidence suggests that increasing magnesium concentrations in dialysate or replacement fluids to 0.75–1.0 mmol/L can reduce intravenous supplementation needs and better maintain serum levels. However, caution is warranted, as higher concentrations may elevate citrate requirements and the risk of citrate accumulation, especially in hepatic dysfunction. Until validated protocols become widely available, a pragmatic approach combining individualized supplementation with close monitoring is advisable, consistent with empirical recommendations outlined by Hansen and colleagues (Table 3) [45].

Practical Recommendations

Table 2
Citrate Solutions and Fluid Composition in CRRT Protocols.

CITRATE SOLUTION	HYPERTONIC SODIUM		ISOTONIC SODIUM		
	Sodium citrate 4 % (Ci-Ca® Fresenius)	ACD-A (Various brands)	Prismocitrate 10/2® (Gambro)	Prismocitrate 18/0® (Gambro)	Citra-HF Pre® (Dirinco)
Trisodium citrate (mmol/L)	136	74,8	10	18	13,3
Citric acid (mmol/L)	N/A	38,1			0
Sodium (mmol/L)	408	224	136	140	140
Chloride (mmol/L)	0	0	106	86	104
Potasium (mmol/L)					3
Magnesium (mmol/L)					0,5
Glucose (mmol/L)					5
Volume (mL)	1000–1500	500–1000	5000	5000	5000
DIALYSIS FLUID	Na 133; K 2-4; Ca 0; **Mg 0,75**-1,0; Cl 116; HCO3 20; Glucose 1 g/L	Different according to protocols	Na 140; K 0; Ca 0; **Mg 0.5**; CI 106; HCO 32, lactate 3 mmol/L	Na 140; K 4; Ca 0; **Mg 0.75**; CI 120.5; HCO 22; lactate 3; Glucose 6.1 mmol/L	N/A
REPLACEMENT SOLUTION	N/A	Different according to protocols	Na 140; K 2; Ca 1.75; MG 0.5; Cl 111.5; HCO 32; lactate 3; Glucosa 6.1 mmol/L	Na 140; K 4; Ca 1.25; MG 0.6; Cl 116; HCO 30; Phosphate 1.2 mmol/L	Predilution only CVVH (*citrate buffered solution Citra-HF-Pre ®*)

Table 3
Nomogram for Intravenous Magnesium Replacement in CRRT Patients. This table presents an institutional protocol for intravenous magnesium repletion based on serum magnesium levels. Thresholds are provided in both millimoles per liter (mmol/L) and milligrams per deciliter (mg/dL) to support clinical decision-making. Dosing recommendations are stratified by severity of hypomagnesemia, and follow-up timing is specified to ensure effective correction and prevent under-recognition of ongoing losses. The protocol emphasizes early recognition and targeted therapy, especially in patients undergoing CRRT with high magnesium losses.

Serum Magnesium Level (mmol/L /mg/dL)	IV Magnesium Dose (Peripheral or Central Line)	Repeat Magnesium Measurement
0.65–0.74 mmol/L (1.57–1.80 mg/dL)	2 g in 50 mL over 1 h	With routine CRRT lab panel
0.55–0.64 mmol/L (1.32–1.56 mg/dL)	4 g total: 2 g in 50 mL over 1 h, q1h × 2	4 h after infusion completion
< 0.55 mmol/L (< 1.32 mg/dL)	6 g total: 2 g in 50 mL over 1 h, q1h × 3; call nephrologist	2 h after infusion completion

1. **Monitoring:** Measure total magnesium at least every 24 h (ideally every 12 h). If available, prefer ionized magnesium, as it better reflects the physiologically active fraction.
2. **Thresholds:** Consider supplementation when Mg ≤1.9 mg/dL (0.78 mmol/L), especially in RCA circuits.
3. **Replacement strategy:** Use replacement or dialysate fluids with higher magnesium concentrations (≥0.75 mmol/L) in high-risk patients. If serum levels remain low, initiate intravenous magnesium sulfate with dose adjustments based on severity and recheck within 2–4 h.
4. **Special considerations:** Interpret values in the context of recent supplementation, as serum magnesium may decline rapidly during CRRT. A structured nomogram for IV magnesium replacement is provided in Table 3.

8. Conclusion

As the utilization of CRRT expands globally, clinicians must remain vigilant not only for common complications but also for rare, underrecognized phenomena that can have serious consequences. Hypertriglyceridemia, euglycemic ketoacidosis, severe hypophosphatemia, hypomagnesemia, and hypothermia exemplify the spectrum of challenges that may silently compromise circuit performance, metabolic homeostasis, and patient stability. Early identification, proactive monitoring, and tailored preventive strategies are paramount to mitigating their impact. Incorporating systematic surveillance protocols, standardized nutritional support, and modern CRRT technologies with advanced thermal and biochemical controls will be essential steps toward reducing the burden of these complications and optimizing outcomes in critically ill patients requiring extracorporeal renal support.

CRediT authorship contribution statement

Gonzalo Ramírez-Guerrero: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Cristian Pedreros-Rosales:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Methodology, Investigation, Data curation. **David Ballesteros:** Writing – original draft, Visualization, Validation, Investigation, Data curation. **Mitchell Rosner:** Writing – review & editing, Visualization, Project administration, Conceptualization. **Claudio Ronco:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Formal analysis, Conceptualization.

Ethics approval and consent to participate

Not applicable.

Statement of ethics

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Declaration of competing interest

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Appendix A. Supplementary data

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