

Early clinical and laboratory findings associated with mortality during continuous renal replacement therapy in neonates

^{ID}Buse Önen Ocak, ^{ID}Ömer Ertekin, ^{ID}Seray Öztürk, ^{ID}Gülmelek Duranoğlu, ^{ID}Batuhan Yeke, ^{ID}Serdar Balsak, ^{ID}Mustafa Şenol Akın, ^{ID}Davut Bozkaya

Department of Neonatology, Ankara Bilkent City Hospital, University of Health Sciences, Ankara, Türkiye

Corresponding Author: **Buse Önen Ocak**

e-mail: buseonen94@gmail.com

Received : 03.04.2026, Accepted : 26.08.2026

DOI:10.12956/TJPD.2026.1346

ABSTRACT

Objective: The aim of this study was to evaluate the demographic and clinical characteristics, short-term outcomes, and early clinical and laboratory indicators associated with mortality in neonates receiving continuous renal replacement therapy (CRRT) in a tertiary neonatal intensive care unit.

Materials and Methods: This retrospective observational study included neonates younger than 28 days of postnatal age who received CRRT for at least 24 hours between October 1, 2019, and November 30, 2025. Patients were grouped according to in-hospital outcomes as survivors and non-survivors. Demographic, clinical, and laboratory variables were compared between the groups. Continuous variables with non-normal distributions were expressed as medians (IQR) and compared using the Mann-Whitney U test. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate. Given the limited sample size, the analyses were considered exploratory.

Results: A total of 13 neonates were included. The median gestational age was 38 weeks and the median birth weight was 2930 g. Nine patients (69.2%) died during hospitalization, whereas four (30.8%) were discharged. In the mortality group, the serum calcium level at 12 h was lower than that in survivors (8.50 [7.40–9.00] vs. 9.65 [9.13–10.4], $p=0.044$). Lactate levels were higher in the mortality group at both hour 12 (4.00 [4.00–11.0] vs 2.95 [2.67–3.15], $p=0.035$) and hour 24 (4.40 [3.60–13.0] vs 2.40 [1.75–3.05], $p=0.035$). Baseline kidney function tests and most pre-CRRT biochemical parameters were similar between the groups.

Conclusion: In this single-center neonatal CRRT cohort, mortality was high. Lower total calcium at hour 12 and higher lactate levels at hours 12 and 24 were associated with mortality. These findings may reflect persistent physiological instability during the early phase of CRRT. However, because of the small sample size, retrospective design, and absence of multivariable adjustment, these associations should be interpreted as exploratory and hypothesis-generating rather than independent predictors of mortality.

Keywords: Calcium, infant, mortality, newborn, lactic acid, renal replacement therapy

Introduction

Neonatal intensive care units (NICUs) are specialized settings where critically ill patients with hemodynamic instability are closely monitored and managed. In this patient population, clinical conditions associated with acute kidney injury, fluid overload, and the accumulation of toxic metabolites continue to be characterized by high rates of mortality and morbidity. Continuous Renal Replacement Therapies (CRRT) have increasingly been utilized as an important treatment modality, particularly in hemodynamically fragile patients, for the management of fluid overload, severe electrolyte imbalances, multiple organ failure, and the clearance of accumulated toxic metabolites in congenital metabolic disorders (1,2). Although the experience with CRRT in neonates and infants has increased in recent years, the available evidence in this field remains

limited. Current data suggest that outcomes in these patients are influenced not only by renal dysfunction itself but also by the burden of underlying critical illness, hemodynamic status, and early response to treatment (3).

The use of CRRT in infants has long involved important technical challenges because of the limited availability of infant-specific devices, small patient size, the need for relatively large extracorporeal circuit volumes, and the risk of hemodynamic instability at treatment initiation (4). For this reason, CRRT in the neonatal period remains a treatment strategy that can be applied only in experienced centers and is still associated with high mortality rates.

In some pediatric series, mortality rates associated with CRRT have been reported to be as high as 70% (5). Similarly,

in a multicenter cohort study of 405 neonates, the mortality rate was approximately 50% (6). However, the number of studies evaluating CRRT outcomes and factors associated with mortality specifically in neonates remains limited.

Despite the increasing use of CRRT in neonatal intensive care, data specifically addressing the early predictors of mortality in neonates remain limited. Most available studies have focused on overall outcomes, technical feasibility, or broad risk factors, whereas evidence on early dynamic clinical and biochemical changes after CRRT initiation is still scarce. Identification of such early indicators may help improve bedside risk assessment and guide closer monitoring in this highly vulnerable population.

The aim of the present study was to evaluate the demographic and clinical characteristics and short-term outcomes of neonates who underwent CRRT in a level IV neonatal intensive care unit and to investigate early clinical and laboratory indicators that may be associated with mortality.

Materials and Methods

Study population and grouping

In this retrospective observational study, critically ill neonates managed in the Ankara Bilkent City Hospital between October 1, 2019, and November 30, 2025, were identified through the hospital electronic medical record system. To ensure a reliable assessment of CRRT-related outcomes, only patients who received CRRT for at least 24 hours were included. Patients were divided into survivor and non-survivor groups according to their clinical outcomes at hospital discharge.

Inclusion and exclusion criteria

Neonates with a postnatal age of less than 28 days at hospital admission who received CRRT for at least 24 hours during hospitalization were included. Patients with a postnatal age of 28 days or older at admission, those with incomplete clinical data, and those who received CRRT for less than 24 hours were excluded.

Indications for CRRT and timing of treatment

Dialysis treatment was initiated in the presence of fluid overload greater than 10%, life-threatening or medically refractory electrolyte abnormalities, refractory metabolic acidosis, pulmonary edema unresponsive to diuretic therapy, and end-organ involvement related to uremia. Electrolyte abnormalities included hyponatremia (sodium >160 mmol/L), hyponatremia (sodium <120 mmol/L), and persistent hyperkalemia despite conventional treatment (potassium >6.5 mmol/L). Metabolic acidosis was defined as pH <7.1 or bicarbonate level <12 mmol/L (2). In addition, liver failure, severe systemic inflammatory conditions, hyperammonemia (ammonia >350 µmol/L), and accumulation of toxic metabolites due to metabolic diseases were also considered indications for dialysis (1, 7). Acute kidney injury (AKI) was staged according to the neonatal modified Kidney Disease: Improving Global Outcomes (KDIGO) criteria, using changes in serum creatinine from the lowest previous reference value and urine output when available. Stage 2 or higher AKI was defined according to the neonatal modified

KDIGO thresholds. In patients with stage 2 or higher AKI, the decision to initiate CRRT was made by integrating AKI stage with clinical status, fluid overload, refractory electrolyte abnormalities, acid–base disturbance, and evidence of multiorgan involvement (8). The percentage of fluid overload was calculated as follows: $[(\text{pre-dialysis weight} - \text{birth weight}) / \text{birth weight}] \times 100$ (9). Although there was no absolute contraindication for CRRT, caution was exercised in the presence of hypotension and bleeding diathesis.

Continuous renal replacement therapy protocol

Device and filter selection

CRRT was performed using the Prismaflex system (Baxter, Deerfield, IL, USA). The HF20 filter set was used, with an extracorporeal volume of 58 mL.

Selection of treatment modality

Continuous venovenous hemofiltration (CVVH) or continuous venovenous hemodiafiltration (CVVHDF) was used according to the patient's clinical condition, hemodynamic characteristics, and metabolic requirements.

Vascular access

The right internal jugular vein was the preferred vascular access site for CRRT. In cases where this was not feasible, the femoral vein was used as an alternative access route for catheterization. Double-lumen hemodialysis catheters were inserted, and catheter size was selected between 5 and 7 Fr according to patient's weight and vascular anatomy.

Circuit preparation and priming

Before treatment, the filter and circuit were flushed with heparinized saline. Because of the extracorporeal volume of the currently available filters, the circuit was primed with erythrocyte suspension to avoid hemodilution and prepare it for use.

Anticoagulation

Standard heparin was used for anticoagulation purposes. Heparin infusion was generally administered at a dose of 5–40 U/kg/hour. During treatment, activated clotting time was maintained between 180 and 220 seconds, and activated partial thromboplastin time between 80 and 120 seconds. Citrate anticoagulation is not used in our unit. Although some recent data suggest that citrate anticoagulation may be a safe and effective alternative in neonatal CRRT (10), our institutional experience with this method remains limited, and systemic heparin continues to be the standard approach in many centers.

Dialysate and replacement fluid

Multibic-0 solution was used as the dialysate and/or replacement fluid. The Multibic solutions were supplied in 5-L bags and contained sodium 140 mmol/L, calcium 1.5 mmol/L, magnesium 0.5 mmol/L, bicarbonate 35 mmol/L, glucose 5.55 mmol/L, potassium 0 mmol/L, and lactate 0 mmol/L in all formulations. Electrolytes and acid–base status were closely monitored throughout treatment, and adjustments were made as needed.

Blood flow rate

After connection of the circuit to the patient, the blood flow rate was initially set at 8–12 mL/kg/min, taking into account the patient's blood pressure and hemodynamic condition.

Dialysate/replacement fluid flow rate

The replacement fluid flow rate was set at 20–30 mL/kg/hour in most patients. In cases treated for congenital metabolic disease, a higher solute clearance was targeted, and the initial replacement rate was planned as 3000 mL/1.73 m²/day.

Duration of treatment

CRRT was administered continuously. Treatment duration was determined individually according to hemodynamic stability, fluid balance, metabolic control, and laboratory response. Only cases in which CRRT was continued for at least 24 hours were included in the study.

Statistical analysis

IBM Statistical Package for the Social Sciences, version 20.0 (SPSS Inc., Armonk, NY, IBM Corp., USA). Distributional characteristics of continuous variables were assessed with consideration of the limited sample size. Continuous variables not normally distributed were presented as medians (IQR). Comparisons of continuous variables between the two independent groups were performed using the Mann–Whitney U test. As an additional exploratory analysis, lactate clearance was calculated as a derived variable using the formula [(pre-CRRT lactate – follow-up lactate) / pre-CRRT lactate] × 100. Lactate clearance was calculated separately for 12 and 24 hours after CRRT initiation and compared between survivors and non-survivors using the Mann–Whitney U test. Categorical variables were expressed as numbers and percentages, and comparisons between groups were made using the chi-square test or Fisher's exact test when expected cell counts were low. All analyses were based on two-sided p values, and p < 0.05 was considered statistically significant. The primary outcome measure of the study was in-hospital mortality; therefore, analyses were performed by comparing survivors with non-survivors. Because of the limited sample size, statistical analyses were kept at an exploratory level, no multivariable modeling was performed, and the findings were interpreted as hypothesis-generating.

Results

A total of 13 neonates were included in the study. In the overall cohort, the median gestational age was 38 (IQR: 38–38) weeks, and the median birth weight was 2930 g (IQR: 2780–3200). Nine infants (69.2%) died during hospitalization, whereas four (30.8%) were discharged.

In the mortality group, the serum calcium level at hour 12 was lower than that in the discharged group (8.50 [7.40–9.00] vs 9.65 [9.13–10.4], p = 0.044). In addition, lactate levels were higher in the mortality group both at hour 12 (4.00 [4.00–11.0] vs 2.95 [2.67–3.15], p = 0.035) and at hour 24 (4.40 [3.60–13.0] vs. 2.40 [1.75–3.05], p = 0.035). These findings were consistent with persistent tissue hypoperfusion and ongoing metabolic derangement in the early period after

Table 1: Demographic, clinical, and laboratory characteristics of the neonates according to mortality status

Variable	Survivors (n=4)	Mortality (n=9)	p
Gestational age*	39.0 (37.5–40.0)	38.0 (38.0–38.0)	0.398
Birth weight, g*	3400 (2993–3625)	2915 (2780–2940)	0.199
Male sex, n (%)	2 (50.0)	4 (44.4)	1.000
CRRT duration, days*	3.5 (2.5–5.25)	1.0 (1.0–2.0)	0.102
Day of CRRT initiation*	5.0 (3.0–10.0)	4.0 (3.0–5.0)	0.637
Day of CRRT discontinuation*	11.0 (7.5–16.3)	6.0 (5.0–7.0)	0.049
Indication (metabolic)†	2 (50.0)	5 (55.6)	1.000
AKI stage*	3.0 (3.0–3.0)	2.0 (1.0–3.0)	0.512
Presence of fluid overload‡	2 (50.0)	5 (55.6)	1.000
Sepsis†	2 (50.0)	1 (11.1)	0.203
Asphyxia†	2 (50.0)	1 (11.1)	0.203
ECMO†	1 (25.0)	1 (11.1)	1.000
Pre-CRRT urea*	58.0 (29.5–96.3)	23.0 (19.0–58.0)	0.313
Pre-CRRT creatinine*	1.15 (0.71–1.53)	0.83 (0.74–1.07)	0.933
Pre-CRRT Na*	139 (136–144)	135 (130–146)	0.483
Pre-CRRT K*	4.35 (2.67–5.85)	5.30 (4.10–6.50)	0.536
Pre-CRRT Ca*	7.90 (7.53–8.57)	7.90 (7.10–9.00)	0.877
Pre-CRRT P*	5.60 (5.23–7.10)	7.40 (5.60–10.0)	0.699
Pre-CRRT Mg*	1.94 (1.93–2.13)	1.80 (1.70–2.10)	0.588
Pre-CRRT ammonia*	1204 (823–1584)	352 (101–913)	0.429
Pre-CRRT pH*	7.15 (7.10–7.28)	7.18 (6.70–7.30)	0.642
Pre-CRRT HCO ₃ * [‡]	21.0 (17.0–25.3)	10.0 (3.0–19.0)	0.075
Pre-CRRT base deficit*	-3.0 (-5.0 to -3.0)	-16.0 (-30.0 to -4.0)	0.244
Pre-CRRT lactate*	4.15 (3.45–4.67)	10.0 (3.30–13.0)	0.199
Pre-CRRT CRP*	41.5 (2.38–90.3)	14.0 (0.50–16.0)	0.484
Pre-CRRT IL-6*	12.4 (2.70–37.0)	109 (18–347)	0.105
Calcium at hour 12*	9.65 (9.13–10.4)	8.50 (7.40–9.00)	0.044
Lactate at hour 12*	2.95 (2.67–3.15)	4.00 (4.00–11.0)	0.035
Calcium at hour 24*	9.65 (8.95–10.1)	9.50 (8.70–9.90)	0.825
Lactate at hour 24*	2.40 (1.75–3.05)	4.40 (3.60–13.0)	0.035

*: median (IQR), †: n (%), **AKI**: acute kidney injury, **BE**: base deficit, **Ca**: calcium, **CRP**: C-reactive protein, **CRRT**: continuous renal replacement therapy, **ECMO**: extracorporeal membrane oxygenation, **HCO₃**: bicarbonate, **IL-6**: interleukin-6, **K**: potassium, **Mg**: magnesium, **Na**: sodium, **P**: phosphorus

CRRT initiation. In the additional exploratory analysis, lactate clearance at 12 hours was numerically higher in survivors than in non-survivors, but the difference was not statistically significant (median 28.9% vs -5.26%, p = 0.604). Lactate clearance at 24 hours also did not differ significantly between the groups (median 25.1% vs 34.62%, p = 0.940).

In contrast, no statistically significant differences were found between the groups with respect to gestational age, birth weight, day of CRRT initiation, or duration of CRRT. Sex distribution, presence of a metabolic indication, AKI stage, fluid overload, sepsis, asphyxia, ECMO requirement, pre-dialysis kidney function tests, and biochemical parameters were also generally similar. No significant differences were observed in pre-dialysis urea, creatinine, sodium, potassium, calcium, phosphorus, magnesium, ammonia, pH, bicarbonate, base deficit, lactate, CRP, or IL-6 levels.

Nevertheless, although some numerical differences were noted in several variables, these did not reach statistical significance, likely because of the small sample size. The findings are summarized in Table I. Although statistical differences were observed in some additional variables, particularly the day of CRRT discontinuation, these findings were not considered clinically direct or explanatory prognostic indicators and were therefore not discussed in detail.

Discussion

In this single-center neonatal CRRT series, the high mortality rate observed (69.2%) was consistent with outcomes reported in cohorts of critically ill neonates receiving CRRT. In a recent single-center study, for example, the mortality rate among 41 neonates treated with CRRT was reported as 75.6% (3). In addition, our center functions as a level IV referral center, which likely receives transfers of infants with greater disease burdens and more severe clinical conditions requiring advanced supportive therapies. This may partly explain the high mortality rate observed in our cohort.

The need for CRRT in neonates often reflects not only renal dysfunction but also severe systemic illness, hemodynamic instability, electrolyte derangements, and multiorgan involvement. Similarly, in pediatric CRRT cohorts, high illness severity, sepsis, and hemodynamic instability—particularly as reflected by inotropic support requirement—have been reported to be associated with mortality (11). In a recent multicenter US cohort, survival was reported as 63% among 67 infants treated with the infant-specific CRRT device Carpediem; that study also emphasized that CRRT in infants remains technically challenging, complications are common, and prognosis often reflects the underlying burden of critical illness rather than renal dysfunction alone (12). Nevertheless, direct comparison across different series should be made with caution because of differences in device type, anticoagulation strategy, treatment indications, and patient populations. In line with this, studies examining CRRT-related mortality in preterm neonates have identified lower gestational age, lower birth weight, lower Apgar score, greater fluid overload, sepsis, and higher illness severity scores as factors associated with poor outcome (13, 14). In addition, a multicenter European cohort evaluating CRRT outcomes in neonates and infants identified low urine output, greater creatinine elevation, later treatment initiation, and lower blood pressure as the main variables associated with mortality (15). Taken together, these findings suggest that dynamic indicators reflecting physiological severity and treatment response may be more informative prognostically than demographic variables alone.

The most notable finding of our study was that the groups appeared to diverge on early serial measurements rather than on baseline values. Lactate levels at 12 and 24 hours were significantly higher in the mortality group, whereas baseline lactate and other pre-dialysis biochemical parameters were generally similar between groups. In addition, lactate clearance was evaluated as an exploratory measure; however, lactate clearance at 12 and 24 hours did not differ significantly between survivors and non-survivors. This may be related to the small sample size, wide interindividual

variability, and heterogeneity of the underlying diagnoses. Therefore, in the present cohort, persistently higher absolute lactate levels at 12 and 24 hours appeared to be a more consistent early biochemical signal than calculated lactate clearance. Similarly, in the single-center study by Rsovac et al. (3) including 41 neonates, persistent anuria and post-CRRT potassium level were associated with poor prognosis, supporting the view that dynamic clinical and biochemical changes during treatment may be more prognostically relevant than baseline measurements alone in neonates receiving CRRT. In a two-center study of 19 neonates with AKI weighing ≤ 3 kg, lower body weight at CRRT initiation and persistent oliguria at the end of treatment were also associated with poor clinical outcome (16). Likewise, in a multicenter WE-ROCK cohort of infants and children weighing ≤ 10 kg, mortality was reported to be higher particularly among those with lower body weight (17).

Consistent with the literature, Xu et al. (14), in a two-center cohort of 59 neonates, reported that pre-CRRT lactate levels were higher in the non-survivor group; however, this variable was not confirmed as an independent risk factor in multivariable analysis. Similarly, in the study by Chu et al. (18) evaluating CRRT in 52 neonates with multiple organ dysfunction syndrome, pre-treatment lactate levels were elevated, whereas lactate levels declined markedly at 12 and 24 hours. Although baseline lactate was higher in non-survivors, it was not confirmed as an independent determinant in multivariable analysis (18). Taken together, these data suggest that although high lactate levels may be associated with poor prognosis, the role of early lactate clearance in neonatal CRRT remains uncertain and may require larger cohorts for reliable evaluation. In our cohort, persistently high lactate levels at 12 and 24 hours in the mortality group support the possibility that, despite CRRT, persistent hypoperfusion and/or the burden of the underlying critical illness remained insufficiently controlled.

Another noteworthy finding of our study was the lower total calcium level at hour 12 in the mortality group. In critically ill patients, total calcium levels may be influenced by sepsis, inflammation, acid-base disturbances, hyperphosphatemia, and various therapeutic interventions. Early hypocalcemia may also adversely affect myocardial contractility and vascular tone, thereby increasing hemodynamic vulnerability. Therefore, low calcium levels may reflect not only a biochemical abnormality but also an indicator of adverse clinical course. Our findings underscore the importance of close electrolyte monitoring during the first 12 hours in neonates undergoing CRRT and of timely intervention when hypocalcemia occurs.

On the other hand, clinical assessment in neonates receiving CRRT should not be limited to in-hospital mortality alone. A recent study reported that even among survivors, growth failure was common and that higher calorie and protein targets were associated with better growth outcomes (19). Likewise, in a large pediatric CRRT cohort, frequent post-discharge functional impairment and new morbidity further highlighted the importance of long-term multidisciplinary follow-up in this population (20). These findings suggest that neonates treated with CRRT should be followed closely not

only in terms of short-term survival but also with respect to growth, renal function, and overall developmental outcomes.

The main strength of this study is that, within a single-center experience, it offers a clinically relevant message by relating early serial laboratory changes—particularly calcium at hour 12 and lactate at hours 12 and 24—to clinical outcome in neonates receiving CRRT.

Limitations

This study has several important limitations. First, the retrospective design, single-center setting, and very small sample size substantially limited statistical power and generalizability. Because only 13 neonates were included, with four survivors and nine non-survivors, the analyses remained exploratory, multivariable modeling could not be performed, and the observed associations should be regarded as hypothesis-generating rather than independent prognostic indicators. Second, the cohort was heterogeneous in terms of underlying diagnoses, CRRT indications, and illness severity, which limits the generalizability of the findings to specific neonatal CRRT subgroups. Third, standardized illness severity scores such as SNAP-II or SNAPPE-II, detailed vasoactive-inotropic support, and serial hemodynamic variables were not consistently available in the retrospective records. Because mortality in neonatal CRRT is strongly influenced by global illness severity, the absence of these variables limits the interpretation of lactate and calcium findings. Fourth, lactate levels may primarily reflect underlying disease severity, ongoing tissue hypoperfusion, or hemodynamic instability rather than a CRRT-specific treatment response. Fifth, only total serum calcium was available for analysis. Ionized calcium, albumin-adjusted calcium, and concurrent albumin values were not consistently incorporated into the analysis. Although albumin replacement was administered when clinically indicated in patients with hypoalbuminemia, the association between lower total calcium at hour 12 and mortality should still be interpreted cautiously. Sixth, multiple clinical and laboratory variables were compared without adjustment for multiple testing, increasing the possibility of type I error. Finally, detailed CRRT-related variables, including net fluid balance during treatment, ultrafiltration dose, circuit life, ventilation parameters, catheter-related complications, filter clotting, treatment interruptions, and CRRT-related complications, were not uniformly available. The long study period between 2019 and 2025 may also have introduced variability in clinical practice, supportive care strategies, and CRRT management over time. Therefore, these findings need to be confirmed in larger multicenter studies incorporating standardized illness severity assessment, ionized calcium, fluid balance, and dialysis-related technical parameters.

Conclusion

In this single-center neonatal CRRT cohort, lower total calcium at hour 12 and higher lactate levels at hours 12 and 24 were associated with mortality. These findings may reflect persistent physiological instability and underlying disease severity during the early phase of CRRT rather than CRRT-specific treatment failure or independent prognostic effects. Because of the small sample size, retrospective design, lack of multivariable adjustment, and absence of standardized illness severity

measures, the results should be interpreted as exploratory and hypothesis-generating. Larger multicenter studies incorporating illness severity scores, hemodynamic variables, ionized calcium, lactate clearance, fluid balance, and CRRT-related treatment parameters are needed to confirm these preliminary observations.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Bilkent City Hospital (10.12.2025, reference number: TABET 2-25-1724)

Contribution of the authors

Study conception and design: BÖÖ, DB, MŞA; data collection: BÖÖ, ÖE, SÖ, GD, BY, SB; analysis and interpretation of results: BÖÖ, DB, MŞA; draft manuscript preparation: BÖÖ; critical revision of the manuscript: ÖE, SÖ, GD, BY, SB, MŞA, DB. All authors reviewed the results and approved the final version of the article.

Source of funding

The authors declare the study received no funding.

Conflict of interest

The authors declare that there is no conflict of interest.

References

1. Guzzo I, de Galasso L, Bayazit AK, et al. Acute paediatric kidney replacement therapies in Europe: demographic results from the EurAKId Registry. *Nephrol Dial Transplant*. 2022;37:770-80. <https://doi.org/10.1093/ndt/gfab280>
2. Cortina G, Daverio M, Demirkol D, Chanchlani R, Deep A. Continuous renal replacement therapy in neonates and children: what does the pediatrician need to know? An overview from the Critical Care Nephrology Section of the European Society of Paediatric and Neonatal Intensive Care (ESPNIC). *European Journal of Pediatrics*. 2024;183:529-41. <https://doi.org/10.1007/s00431-023-05318-0>
3. Rsovac S, Milosevic K, Spasojevic B, et al. Continuous Renal Replacement Therapy in Critically-Ill Term and Preterm Newborns: A Single-Center Study in Belgrade. *Children (Basel)*. 2025;12(7):828. <https://doi.org/10.3390/children12070828>
4. Slagle C, Askenazi D, Starr M. Recent Advances in Kidney Replacement Therapy in Infants: A Review. *Am J Kidney Dis*. 2024; 83:519-30. <https://doi.org/10.1053/j.ajkd.2023.10.012>
5. Sebeih Y, Al-Turki A, Alfattani A, et al. Continuous kidney replacement therapy outcomes in infants and newborns. *International Journal of Pediatrics and Adolescent Medicine*. 2023;10(3):65-72. https://doi.org/10.4103/ijpam.ijpam_7_24
6. Muff-Luett M, Webb T, Scobell R, et al. Dialysis modality and mortality of the Contemporary Infant and Neonatal Dialysis (COINED) Cohort: a Pediatric Nephrology Research Consortium (PNRC) study. *Pediatric Nephrology*. 2026;41:1731-42. <https://doi.org/10.1007/s00467-025-07082-9>
7. Schaefer F, Straube E, Oh J, Mehls O, Mayatepek E. Dialysis in neonates with inborn errors of metabolism. *Nephrology Dialysis Transplantation*. 1999;14(4):910-8. <https://doi.org/10.1093/ndt/14.4.910>
8. Starr MC, Charlton JR, Guillet R, et al. Advances in Neonatal Acute Kidney Injury. *Pediatrics*. 2021; 148 (5): e2021051220. <https://doi.org/10.1542/peds.2021-051220>
9. Goldstein SL, Currier H, Graf JM, Cosio CC, Brewer ED, Sachdeva R. Outcome in Children Receiving Continuous Venovenous Hemofiltration. *Pediatrics*. 2001; 107(6):1309-12. <https://doi.org/10.1542/peds.107.6.1309>

10. Kaymaz D, Bayhan SA, Kostekci YE, et al. Evaluation of anticoagulant methods in newborns undergoing continuous renal replacement therapy: what is the optimum method? *Eur J Pediatr.* 2025; 184:473. <https://doi.org/10.1007/s00431-025-06314-2>
11. Alrashdi AS, Alshammari JN, Abdullah SK, et al. Outcomes of continuous kidney replacement therapy in pediatric patients: the impact of timing and predictors of mortality. *Pediatr Nephrol.* 2025; 40:2973-80. <https://doi.org/10.1007/s00467-025-06794-2>
12. Slagle CL, Vuong KT, Krallman KA, et al. Infant Renal Replacement Therapy Using Carpediem: A Multicenter Observational Cohort Study from the ICONiC Learning Network. *J Pediatr.* 2026; 288:114838. <https://doi.org/10.1016/j.jpeds.2025.114838>
13. Noh ES, Kim HH, Kim HS, et al. Continuous Renal Replacement Therapy in Preterm Infants. *Yonsei Med J.* 2019; 60(10):984-91. <https://doi.org/10.3349/ymj.2019.60.10.984>
14. Xu J, Chu X, Zhang W, et al. Analysis of risk factors for death in 59 cases of critically ill neonates receiving continuous renal replacement therapy: a two-centered retrospective study. *Eur J Pediatr.* 2023; 182:353-61. <https://doi.org/10.1007/s00431-022-04693-4>
15. Deja A, Deja K, Cappoli A, et al. Predicting mortality and short-term outcomes of continuous kidney replacement therapies in neonates and infants. *Nephrol Dial Transplant.* 2026; 41:489-99. <https://doi.org/10.1093/ndt/gfaf173>
16. Sun Y, Xu J, Chu X, et al. Effectiveness and feasibility of continuous renal replacement therapy for acute kidney injury in neonates weighing 3 kg or less: a two-centre, retrospective study. *BMJ Paediatr Open.* 2024; 8: e002241. <https://doi.org/10.1136/bmjpo-2023-002241>
17. Menon S, Starr MC, Zang H, et al. Characteristics and outcomes of children ≤ 10 kg receiving continuous kidney replacement therapy: a WE-ROCK study. *Pediatr Nephrol.* 2025;40:253-64. <https://doi.org/10.1007/s00467-024-06438-x>
18. Chu X, Xu J, Sun Y, Gong X, Chen D, Cai C. Continuous renal replacement therapy in neonates with multiple organ dysfunction syndrome: clinical utilization effects and outcomes. *Italian Journal of Pediatrics.* 2025; 51:275. <https://doi.org/10.1186/s13052-025-02123-3>
19. Short K, Hanick J, Bickert P, Potts J, Askenazi D. Growth in neonates with congenital kidney failure requiring continuous kidney replacement therapy. *Pediatr Nephrol.* 2025;40:3733-41. <https://doi.org/10.1007/s00467-025-06887-y>
20. Dolan KJ, Gist KM, Basalely A, et al. Functional outcomes in pediatric patients on renal replacement therapy in a worldwide registry. *Nephrol Dial Transplant.* 2025; 40(10):1919-30. <https://doi.org/10.1093/ndt/gfaf067>