ORIGINAL ARTICLE

# Children on continuous renal replacement therapy: prognostic factors

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**Objectives** To identify prognostic factors in children receiving continuous renal replacement therapy.

**Design** Historical cohort study.

**Setting** Neonatal and paediatric intensive care unit of a Hong Kong hospital.

**Patients** Neonatal or paediatric patients who received continuous renal replacement therapy from January 1998 to December 2008.

**Results** In all, 37 patients who received 39 episodes of continuous renal replacement therapy were identified. The male-to-female ratio was 1.5:1. Among the 39 episodes, 15 (39%) were performed on neonates with a mean birth weight of 2.6 (standard deviation, 0.7; range, 0.9-3.7) kg, and 24 (62%) were performed on paediatric patients with a mean age of 7.9 years (standard deviation, 6.4 years; range, 6 months to 18 years). The overall mortality was 41%; in the neonatal and paediatric groups it was 60% and 29%, respectively. There was no significant difference in the mean and maximal ultrafiltration rate in survivors and non-survivors. Multivariate analysis identified the PRISM III score and fluid overload as independent predictors of mortality. Kaplan-Meier survival analysis showed that patients with pre–continuous renal replacement therapy fluid overload of 5.5% or more was associated with reduced survival in the intensive care unit as compared to those having less severe fluid overload (P=0.011). In neonatal patients, there was a higher proportion with multi-organ failure and severe fluid overload.

**Conclusion** High PRISM III scores and the degree of pre–continuous renal replacement therapy fluid overload were independent predictors of mortality.

**New knowledge added by this study**

- High PRISM III score and fluid overload were independent predictors of mortality in local children receiving continuous renal replacement therapy.
- Fluid overload of 5.5% or more was associated with reduced survival in this cohort of patients.
- Neonatal patients were particularly prone to multi-organ failure and severe fluid overload.

**Implications for clinical practice or policy**

- Early initiation of continuous renal replacement therapy may significantly reduce fluid overload and improve survival in children with acute kidney injury.

**Key words**
Acute kidney injury; Child; Renal dialysis; Renal replacement therapy

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## Introduction

Acute kidney injury in children carries a high risk of mortality. Over the years, continuous renal replacement therapy (CRRT) has evolved into an important modality of treatment in critically ill patients with acute renal injury.[1-3] A 4-year survey conducted in the US reported an increase from 18% to 36% in the number of centres choosing CRRT as the preferred modality of treatment for children with acute kidney injury.[4] Children with acute kidney injury receiving renal support have a high mortality, with rates ranging from 42 to 66%.[4,5]

Identification of potential prognostic factors in this group of patients helps to predict outcome. Various mortality-predicting scoring systems like PRISM III (The third generation of Pediatric Risk of Mortality[6]) score and the Pediatric Index of Mortality have been developed for paediatric patients admitted to intensive care units (ICUs). However, these scoring systems are not specifically targeted at patients having CRRT. Thus, if there are factors that contribute independently to mortality in this group of patients, they should be identified.

## 影響小童接受連續性腎臟替代治療預後的因數

**目的** 研究影響小童接受連續性腎臟替代治療預後的因數。

**設計** 歷史隊列研究。

**安排** 香港一所醫院內的新生嬰兒及兒科深切治療部。

**患者** 1998年1月至2008年12月期間接受連續性腎臟替代治療的新生兒及小兒患者。

**結果** 共有37名（39例）接受連續性腎臟替代治療的患者。男女比例為1.5:1。其中15例（39%）新生兒的平均出生體重為2.6 kg（標準差0.7 kg；介乎0.9至3.7 kg），另24例（62%）小兒患者的平均年齡為7.9歲（標準差6.4歲；介乎6個月至18歲）。總死亡率41%；新生兒及小兒患者的死亡率分別為60%及29%。生存和死亡病例的平均及最大超濾率並無顯著分別。多元分析指出兒童死亡危險度評估表（PRISM III分數）和體液超量均為死亡的獨立預測因子。Kaplan-Meier生存分析顯示，在進行連續性腎臟替代治療前體液超量達5.5%或以上的患者比那些體液超量較少的患者在深切治療部的生存時間為短（P=0.011）。此外，亦觀察到新生兒患者有較高比例的多器官功能衰竭及較嚴重的體液超量情況。

**結論** PRISM III分數高以及在進行連續性腎臟替代治療前有嚴重的體液超量均為死亡的獨立預測因子。


## Methods

This was a retrospective study. Records of patients having CRRT in either the neonatal intensive care unit (NICU) or paediatric intensive care unit (PICU) of Queen Elizabeth Hospital in Hong Kong from January 1998 to December 2008 were analysed. The hospital is a regional referral centre draining a population of around 500 000 inhabitants, and its NICU and PICU receive critically ill patients aged up to 18 years with various surgical, medical, and traumatic conditions. Neonates were defined as babies less than 30 days old.

Patients aged 18 years or younger who had acute kidney injury and received any modality of CRRT in either the NICU or PICU were included. Patients with post-cardiac surgery or in receipt of CRRT for known end-stage renal disease or other reasons not related to acute kidney injury were excluded.

Demographic and clinical data retrieved from the medical records included age, gender, maturity of gestation (for neonates), underlying diagnosis, duration of CRRT, mode and regimen of CRRT, vital signs, and laboratory test results. Potential prognostic factors including the PRISM III score,[6] number of organ failures,[7] percentage of fluid overload, indication of CRRT, pre-CRRT acid-base status, electrolytes, haemoglobin level, and receipt of ventilatory/inotropic support were also logged. The PRISM III score and number of organ failures were calculated separately for every individual episode of CRRT. The following variables were included in the calculation of the PRISM III score: systolic blood pressure, heart rate, temperature, pupillary reflex, Glasgow coma scale, acid-base status, renal function, glucose level, the white blood cell and platelet counts, and the clotting profile.

Percentage fluid overload was calculated for the period starting 24 hours prior to the initiation of CRRT using the following formula[8]:

$$\frac{\text{(Total fluid intake (L) – total fluid output (L)}}{\text{Body weight on admission (kg)}} \times 100\%$$

The primary outcome was death during ICU admission. The secondary outcome was the renal outcome of survivors. Comparisons were made between survivors and non-survivors regarding various demographic and potential prognostic factors.

### Data analyses

Data entry and analysis entailed using the Statistical Package for the Social Sciences (Windows version 16; SPSS Inc, Chicago [IL], US). Distributions for continuous variables were tested for normality. Transformation was attempted for non-normal distributions before applying non-parametric tests. Unpaired *t* tests and the Wilcoxon ranked sum test were used for comparisons between continuous variables, whereas categorical data were compared using the $\chi^2$ test or Fisher's exact test. Prognostic factors were identified by both univariate comparison between survivors and non-survivors, as well as logistic regression. Significant factors identified from the univariate analysis were then selected for multivariate logistic regression analysis. Kaplan-Meier analyses were used for survival analysis. Statistical significance was defined as $P<0.05$.

## Results

### Demographic and outcome data

In all, 38 patients having 41 possible episodes of CRRT during the study period were identified. One potential episode was excluded as the patient died before the CRRT system could be initiated, and another episode treatment was not initiated despite acute renal failure. Hence, eventually there were 37 patients having 39 episodes of CRRT in the final analysis. The male-to-female ratio was 1.5:1. Among these 39 episodes, 15 (39%) involving CRRT were performed on neonates with a mean ± standard deviation (SD) birth weight of 2.6 ± 0.7 (range, 0.9-3.7) kg. By contrast, 24 (62%) of these episodes involving CRRT were performed on paediatric patients with a

mean ± SD age of 7.9 ± 6.4 years (range, 6 months to 18 years) and a mean ± SD body weight of 24.0 ± 15.7 kg. In 25 episodes, continuous venovenous haemofiltration or haemodiafiltration (CVVH/D) were used, and in 16 continuous arteriovenous haemofiltration or haemodiafiltration (CAVH/D) were utilised. Most neonates received CAVH/D (14/16 episodes, 88%), whereas most paediatric patients received CVVH/D (23/25 episodes, 92%). There were two patients who received both CAVH and CVVH.

The indications for CRRT were acute renal failure (10 episodes, 26%), acute-on-chronic renal failure (1 episode, 3%), volume overload unresponsive to other therapies (22 episodes, 56%) and removal of toxins, metabolites/correction of severe acidosis or electrolytes imbalance (6 episodes, 15%). Overall mortality was 41% with 60% (n=9) in neonates versus 29% (n=7) in paediatric patients (P=0.116). Among our 21 patients with multi-organ failure, 14 (67%) did not survive, most of the mortality (88%) being attributable to this problem. Demographic data and clinical variables in the survivors and non-survivors are shown in Tables 1 and 2. There was no statistically significant difference between these groups in terms of age, gender, body weight, maturity (for neonatal patients), the mode of CRRT (CAVH/D vs CVVH/D), or the mean and maximal ultrafiltration rate used. The mean ultrafiltration rate in CAVH/D group was 14.3 mL/kg/h for survivors and 12.9 mL/kg/h for non-survivors (P=0.427), whereas the mean ultrafiltration rate for CVVH/D group was 46.1 mL/kg/h for survivors and 45.7 mL/kg/h for non-survivors (P=0.641).

Concerning renal outcomes upon discharge from the ICU in those survived, 30% (n=7) stopped CRRT with no impairment in renal function, 39% (n=9) stopped it with some impairment of renal function, and 30% (n=7) continued with renal replacement therapy.

## Prognostic factors

In the univariate analysis, the following factors differed significantly in survivors and non-survivors. Non-survivors had higher proportions with multi-organ failure (P<0.001), patients receiving ventilatory support (P=0.029) or inotropic support (P=0.001), severe pre-CRRT fluid overload (P=0.002), and severe metabolic acidosis (P=0.024). They also had lower systolic and diastolic blood pressures on admission (only in the paediatrics group; P=0.016 and 0.042, respectively) and higher mean PRISM III scores (P=0.002) [Table 3]. Significant variables identified during univariate analysis were selected for multivariate analysis. Highly dependent variables identified by correlation coefficients were excluded. Logistic regression identified PRISM III scores and fluid overload as independent predictors of mortality (Table 4).

The relationship of fluid overload with survival was further analysed. The 5.5% sample median for fluid overload was used as the cut-off value, and subjects were then stratified into those with pre-CRRT fluid overload percentages of <5.5% and ≥5.5%. A Kaplan-Meier survival analysis of the two groups revealed that patients with pre-CRRT fluid overload of ≥5.5% had shorter survival times (P=0.011, log-rank test; Fig). There was a statistically significant positive correlation between the log-transformed fluid overload percentage and the time elapsing to initiation of CRRT after ICU admission (Pearson's correlation coefficient=0.352, P=0.041).

## Comparison between neonatal and paediatric patients

There was no statistically significant difference for mortality between paediatric and neonatal patients. Neonatal patients had a higher proportion with

TABLE 1. Demographic backgrounds of survivors and non-survivors

| Variable | Survivor | Non-survivor | Total cohort | P value |
|---|---|---|---|---|
| Sex | | | | |
| Male | 67% (n=14) | 50% (n=8) | 22 | 0.368 |
| Female | 33% (n=7) | 50% (n=8) | 15 | |
| Age* | | | | |
| Neonates (days) | 5.2 ± 5.0 | 9.1 ± 10.9 | 7.5 ± 9.0 | 0.72 |
| Paediatrics (months) | 106.0 ± 78.7 | 66.0 ± 69.8 | 94.3 ± 77.0 | 0.215 |
| Body weight* | | | | |
| Neonates (kg) | 2.7 ± 0.8 | 2.4 ± 0.7 | 2.6 ± 0.7 | 0.443 |
| Paediatrics (kg) | 26.6 ± 16.4 | 17.7 ± 12.7 | 24.0 ± 15.7 | 0.182 |
| Maturity (for neonates) | | | | |
| Term | 83% (n=5) | 44% (n=4) | 9 | 0.287 |
| Pre-term | 17% (n=1) | 56% (n=5) | 6 | |

* Expressed as mean ± standard deviation

TABLE 2. Comparison of clinical variables in survivors and non-survivors

| Variable* | Survivor | Non-survivor | Total cohort | P value‡ |
|---|---|---|---|---|
| Causes | | | | 0.02 |
| Multi-organ failure | 30% (n=7) | 88% (n=14) | 21 | |
| Poor cardiac output | 13% (n=3) | 13% (n=2) | 5 | |
| Primary renal disease | 30% (n=7) | 0% | 7 | |
| Toxin or metabolites overdose | 26% (n=6) | 0% | 6 | |
| Urine output (mL/kg/h)† | 1.4 ± 2.0 | 1.1 ± 2.1 | 1.3 ± 2.0 | 0.26 |
| Pre-CRRT fluid overload (%)† | 2.7 ± 3.5 | 13.3 ± 8.8 | 7.0 ± 8.1 | <0.001 |
| Systolic blood pressure (mm Hg)† | | | | |
| Neonates | 55.0 ± 11.6 | 52.1 ± 12.2 | 53.3 ± 11.6 | 0.409 |
| Paediatrics | 121.4 ± 18.2 | 85.6 ± 22.8 | 111.0 ± 25.4 | 0.004 |
| Diastolic blood pressure (mm Hg)† | | | | |
| Neonates | 35.7 ± 7.1 | 34.7 ± 9.4 | 35.1 ± 8.3 | 0.767 |
| Paediatrics | 67.4 ± 17.9 | 47.0 ± 17.9 | 61.5 ± 19.9 | 0.019 |
| Need of ventilatory support | 70% (16/23) | 100% (16/16) | 32 | 0.029 |
| Need of inotropic support | 52% (12/23) | 100% (16/16) | 28 | 0.001 |
| Pre-CRRT haemoglobin (g/L)† | 92 ± 24 | 103 ± 19 | 97 ± 22 | 0.135 |
| Pre-CRRT urea (mmol/L)† | | | | |
| Neonates | 7.7 ± 7.2 | 6.8 ± 2.7 | 7.1 ± 4.8 | 0.724 |
| Paediatrics | 28.6 ± 14.4 | 20.8 ± 13.2 | 26.3 ± 14.2 | 0.12 |
| Pre-CRRT creatinine (μmol/L)† | | | | |
| Neonates | 160.8 ± 93.0 | 164.6 ± 45.7 | 163.1 ± 65.5 | 0.906 |
| Paediatrics | 499.0 ± 708.5 | 226.1 ± 117.3 | 419.5 ± 607.4 | 0.465 |
| Pre-CRRT potassium (mmol/L)† | 4.6 ± 1.1 | 4.6 ± 1.5 | 4.6 ± 1.3 | 0.951 |
| Pre-CRRT base excess (mmol/L)† | -3.3 ± 10.1 | -10.9 ± 5.6 | -6.4 ± 9.2 | 0.004 |
| Pre-CRRT pH† | 7.4 ± 0.1 | 7.2 ± 0.1 | 7.3 ± 0.1 | 0.001 |
| PRISM III† | 10.4 ± 5.9 | 27.8 ± 15.9 | 17.5 ± 14.0 | 0.001 |
| No. of organ failure† | 3.1 ± 1.6 | 5.4 ± 0.7 | 4.1 ± 1.8 | <0.001 |
| Mean ultrafiltration rate (mL/kg/h)† | | | | |
| CAVH/D | 14.3 ± 20.7 | 12.9 ± 10.6 | 13.5 ± 15.2 | 0.427 |
| CVVH/D | 46.1 ± 24.8 | 45.7 ± 17.1 | 46.0 ± 22.2 | 0.641 |
| Maximal ultrafiltration rate (mL/kg/h)† | | | | |
| CAVH/D | 27.4 ± 30.0 | 31.8 ± 22.3 | 29.9 ± 25.6 | 0.647 |
| CVVH/D | 54.8 ± 30.8 | 54.0 ± 16.1 | 54.5 ± 26.6 | 0.382 |

* CRRT denotes continuous renal replacement therapy, PRISM III 3rd generation of Pediatric Risk of Mortality score, CAVH/D continuous arteriovenous haemofiltration or haemodiafiltration, and CVVH/D continuous venovenous haemofiltration or haemodiafiltration

† Expressed as mean ± standard deviation

‡ Non-survivors had a higher proportion with multi-organ failure, more severe pre-CRRT fluid overload, more severe metabolic acidosis, a higher proportion receiving ventilatory/inotropic support, lower systolic and diastolic blood pressures on admission (in the paediatric group), and a higher mean PRISM III score

multi-organ failure than paediatric patients (80% vs 38%, P=0.041), and a higher mean fluid overload percentage (11.7 vs 4.1%, P=0.004). However, there was no statistically significant difference between the two groups in terms of PRISM III scores, number of failed organs, mean time to initiation of CRRT, and pre-CRRT acid-base status (Table 5).

## Discussion

Acute renal failure in children confers a high mortality and remains a challenge to paediatric nephrologists and intensivists. With the advances in technology and equipment that cater for children and small babies, CRRT has evolved into an important modality of renal replacement therapy for such subjects who develop acute renal failure.[3,4] A number of studies have identified several prognostic factors for mortality in patients receiving CRRT.[7-9] In particular, haemodynamic status and clinical severity as indicated by various mortality-predicting scores are important determinants of survival.[7,9,10] The use of

dialysis element (CAVH/D or CVVH/D) confers better survival according to one report.[9] Besides these, the degree of fluid overload has been suggested as one of the important prognosticators.[3,8,10-13]

Both PRISM III scores and the number of failed organs were identified as significant predictors of mortality in our study cohort. Currently there are several ICU scoring systems to predict patient outcome, but they are not specifically targeted at patients with acute renal failure. Moreover, specific contributing factors such as fluid status and aetiology of renal failure are not addressed when calculating PRISM III scores.

Fluid status plays a critical role in the management of patients with renal failure.[14,15] Fluid overload has been repeatedly shown to be an important predictor of adverse outcomes.[11-13] Fluid overload may reflect the underlying critical condition as well as the fluid management strategy, and may have no direct causative bearing on mortality. However, numerous studies have shown that fluid overload independently contributes to mortality, even after adjustment for severity of illness. Volume overload impairs cardiac output and may even cause heart failure, as described by Starling's myocardial performance curve. It may cause pulmonary congestion and pleural effusions, which could impair gaseous exchange. The detrimental effect of fluid overload on ventilator requirements in critically ill children,[16,17] and the development of lung oedema during peri-operative periods have been extensively studied.[18,19] In adults, a lower degree of fluid overload was associated with shorter durations of artificial ventilation and ICU stays.[20] Others have associated a negative fluid balance with success in weaning patients off ventilators.[21] Earlier initiation of CRRT may therefore be beneficial by reducing the degree of fluid overload, and translate into decreased mortality. This practice has also been reported in the treatment of acute kidney injury among paediatric stem cell transplant recipients.[22] Our study too showed that the mean time to CRRT initiation correlated positively with fluid overload status (Pearson's correlation coefficient=0.352, P=0.041). Moreover, Gillespie et al[12] reported that a higher fluid overload (≥10% vs <10%) was associated with poorer outcomes in children receiving CRRT. In our study cohort, a fluid overload as small as 5.5% (the median fluid overload percentage of the cohort) may have contributed to a difference in mortality. To better understand the relationship between CRRT initiation, fluid overload status, and mortality requires prospective, large-scale randomised controlled trials. However, these may prove difficult to undertake, both from a clinical and ethical perspective.

Acute kidney injury with multi-organ failure conferred a high mortality of 43% in a study reported by the Prospective Pediatric Continuous Renal Replacement Therapy Registry group.[10] Our study sample also revealed high mortality, amounting to 14/21 among such patients; in non-survivors 88% had multi-organ failure. In particular, neonatal patients had a significantly higher proportion of multi-organ failure than the paediatric population (80% vs 38%, P=0.041). Contrarily, primary renal disease and toxin/metabolite-related disease conferred a favourable

TABLE 3. Univariate analysis of prognostic factors

| Variable* | Odds ratio (95% confidence interval) | P value† |
|---|---|---|
| Sex (male vs female) | 0.44 (0.12-1.64) | 0.37 |
| Age-group (neonatal vs paediatric) | 3.64 (0.94-14.16) | 0.12 |
| Body weight (kg) | | |
| Neonates | 0.57 (0.12-2.69) | 0.47 |
| Paediatrics | 0.96 (0.89-1.03) | 0.22 |
| Maturity (term vs prematurity) | 0.16 (0.01-1.98) | 0.27 |
| Multi-organ failure | 16.0 (2.84-90.02) | <0.001 |
| Urine output (mL/kg/h) | 0.91 (0.64-1.28) | 0.58 |
| Pre-CRRT fluid overload (%) | 1.57 (1.17-2.09) | 0.002 |
| Systolic blood pressure (mm Hg) | | |
| Neonates | 0.98 (0.89-1.07) | 0.623 |
| Paediatrics | 0.89 (0.82-0.98) | 0.016 |
| Diastolic blood pressure (mm Hg) | | |
| Neonates | 0.98 (0.86-1.12) | 0.81 |
| Paediatrics | 0.93 (0.86-0.10) | 0.042 |
| Pre-CRRT haemoglobin (g/dL) | 1.29 (0.92-1.80) | 0.14 |
| Pre-CRRT urea (mmol/L) | | |
| Neonates | 0.96 (0.77-1.20) | 0.70 |
| Paediatrics | 0.95 (0.88-1.03) | 0.24 |
| Pre-CRRT creatinine (μmol/L) | | |
| Neonates | 1.00 (0.99-1.01) | 0.91 |
| Paediatrics | 0.98 (0.99-1.00) | 0.30 |
| Pre-CRRT potassium (mmol/L) | 0.98 (0.59-1.65) | 0.95 |
| Pre-CRRT base excess (mmol/L) | 0.87 (0.77-0.98) | 0.024 |
| Pre-CRRT pH | 0.00002 (0.00-0.02) | 0.002 |
| PRISM III | 1.14 (1.05-1.24) | 0.002 |
| No. of organ failure | 4.6 (1.7-12.7) | 0.004 |

* CRRT denotes continuous renal replacement therapy, and PRISM III 3rd generation of Pediatric Risk of Mortality score

† Univariate analysis showed that higher PRISM III score, degree of fluid overload, multi-organ failure, metabolic acidosis, admission blood pressure, and requirement of inotropic / ventilatory support contributes significantly to mortality

TABLE 4. Multivariate analysis of prognostic factors

| Model variable* | Odds ratio (95% confidence interval) | P value† |
|---|---|---|
| Fluid overload (%) | 1.81 (1.05-3.13) | 0.032 |
| PRISM III score | 1.16 (1.02-1.32) | 0.021 |
| Multi-organ failure | 7.14 (0.21-240.84) | 0.273 |

* PRISM III denotes 3rd generation of Pediatric Risk of Mortality score

† Logistic regression showed that PRISM III score and fluid overload are independent predictors of mortality

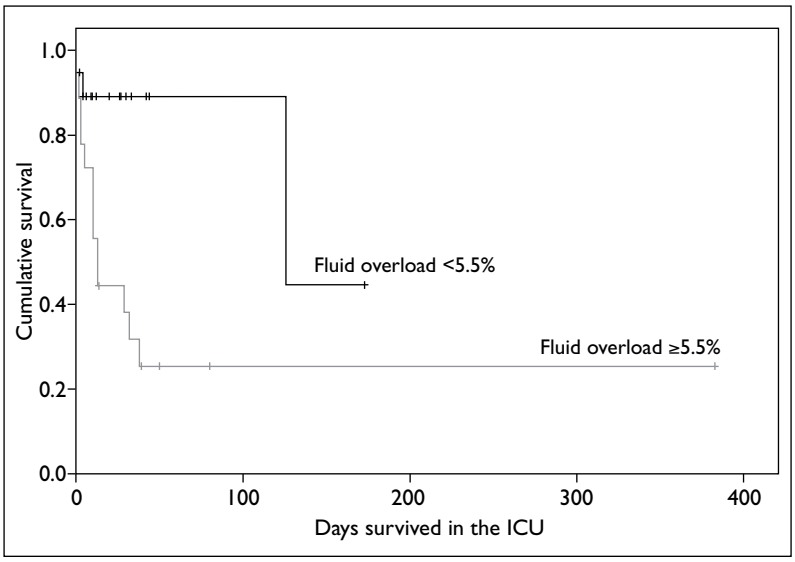


FIG. Kaplan-Meier plot of survival during intensive care unit (ICU) stays based on prior fluid overload status

Patients with pre–continuous renal replacement therapy fluid overload of ≥5.5% had poorer survival during their ICU stay compared to those having less severe fluid overloads (P=0.011). The line for low fluid overload status (<5.5%) is shorter due to discharge from the ICU

TABLE 5. Comparison of clinical variables in neonatal and paediatrics patients

| Variable* | % or mean ± standard deviation | | P value† |
|---|---|---|---|
| | Neonatal | Paediatric | |
| Multi-organ failure | 80% (12/15) | 38% (9/24) | 0.041 |
| Fluid overload (%) | 11.7 ± 9.8 | 4.1 ± 5.2 | 0.004 |
| PRISM III score | 19.6 ± 12.4 | 16.2 ± 14.9 | 0.182 |
| No. of failed organs | 4.6 ± 0.9 | 3.7 ± 2.1 | 0.233 |
| Pre-CRRT pH | 7.3 ± 0.2 | 7.3 ± 0.1 | 0.908 |
| Pre-CRRT base excess (mmol/L) | -4.6 ± 10.5 | -7.6 ± 8.4 | 0.573 |
| Mean time to initiate CRRT (hours) | 45.4 ± 34.9 | 35.8 ± 51.7 | 0.094 |
| Mortality | 60% (9/15) | 29% (7/24) | 0.116 |

* PRISM III denotes 3rd generation of Pediatric Risk of Mortality score, and CRRT continuous renal replacement therapy

† A higher proportion of multi-organ failure and more severe fluid overload were observed in the neonatal group

outcome, as all such patients survived. Receipt of inotropic and ventilatory support, together with severe acidosis, were identified as prognosticators of poor outcomes from the univariate analysis. From our data, overall mortality in this group was 41%, which was comparable to those reported by others.[3,5] Although mortality in neonates (60%) and paediatric patients (29%) did not differ significantly in statistical terms, this could be due to our small sample sizes.

Our study population comprised a relatively high proportion of neonates. A survival rate of 25% in patients less than 3 kg has been reported.[23] Specifically, in our cohort neonatal patients were prone to significantly higher degrees of fluid overload than paediatric patients (11.7 vs 4.1%, P=0.004), and had a longer mean time to initiation of CRRT (though this difference was not statistically significant; 45.4 hours vs 35.8 hours, P=0.094). This may reflect the difficulties in performing dialysis in small babies as opposed to older children, leading to a higher tolerance of fluid overload before CRRT is started. Most of the neonatal patients in our cohort received CAVH/D instead of CVVH/D. This was due to the technical difficulty in securing a good-size double lumen catheter (the smallest available in Hong Kong was a 6.5-Fr double lumen catheter), as well as the lack of CVVH/D equipment to cater for neonates for many years. Nevertheless, using the CAVH mode of dialysis, the mean ultrafiltration rate in our small patients could still reach 40 mL/kg/h, with no complications with respect to lower limb perfusion. The nursing expertise necessary to set-up and monitor CRRT circuits in neonates is also an important determinant of its successful implementation. Whilst it is postulated that early initiation of CRRT in small-size patients appears beneficial, carrying it out on small babies still poses a great practical challenge in NICUs.

The calculation of fluid overload status in this study involved recording the 24-hour fluid balance prior to CRRT treatment, which was an obvious limitation in our study. Insufficient information on fluid management prior to hospitalisation or ICU admission was clearly a problem. Moreover, a 24-hour record may not be sufficient to reflect the real fluid balance status. The small sample size also limited the power of our study. Patients receiving CAVH/D and CVVH/D were analysed as a single group, there being no difference in mortality between the groups receiving different modes of CRRT. Also, CAVH/D was mostly given to neonates (93%), whereas CVVH/D was mostly applied to paediatric patients (96%). Ideally neonatal and paediatric patients should have been separately analysed, but due to the small sample size of our cohort, such subgroup analysis was not feasible.

## Conclusion

High PRISM III scores and fluid overload were important determinants of survival in children with acute kidney injury. If technically feasible, early initiation of CRRT may help prevent significant fluid overload and improve the chances of survival.

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