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CLINICAL STUDY

Can Delivery Dialysis Dose Affect Survival of Acute Kidney Injury Patients?

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Abstract

Intensity of dialysis dose in acute kidney injury (AKI) might benefit critically ill patients. The aim of this study was to evaluate the effect of intermittent hemodialysis (IHD) dose on mortality in patients with AKI. *Methods*: Prospective observational study was performed on AKI patients treated with IHD. The delivered dialysis dose per session was calculated based on single-pool *Kt*/*V* urea. Patients were allocated in two groups according to the weekly delivered median *Kt*/*V*: higher intensity dialysis dose (HID: *Kt*/*V* higher than median) and lower intensity dialysis dose (LID: *Kt*/*V* lower than median). Thereafter, AKI patients were divided according to the presence or absence of sepsis and urine output. Clinical and lab characteristics and survival of AKI patients were compared. *Results*: A total of 121 AKI patients were evaluated. Forty-two patients did not present with sepsis and 45 did not present with oliguria. Mortality rate after 30 days was lower in the HID group without sepsis (14.3% $\times$ 47.6%; $p = 0.045$) and without oliguria (31.8% $\times$ 69.5%; $p = 0.025$). Survival curves also showed that the HID group had higher survival rate when compared with the LID group in non-septic and non-oliguric patients ($p = 0.007$ and $p = 0.003$, respectively). *Conclusion*: Higher dialysis doses can be associated with better survival of less seriously ill AKI patients.

Keywords: dialysis dose, hemodialysis, acute kidney injury, low urine output, sepsis, survival

Acute kidney injury (AKI) is a common finding among hospitalized patients and is associated with high mortality, ranging from 50% to 75%, depending on the severity of AKI and clinical conditions.[1,2] Although the management of AKI has improved, these changes have increased slowly patients' outcomes. The optimal approach to renal replacement therapy, as well as the intensity and timing of such therapy remains unclear.[3,4]

Previous studies have shown that the survival improved when the intensity of dialysis was increased.[5–7] However, recent trials did not confirm this benefit and showed that increasing the intensity of renal replacement therapy did not decrease mortality among patients with AKI.[8–10]

The association of low urine output, fluid overload, and sepsis with worse prognosis of AKI patients has been reported in the literature.[2,11] Paganini et al.,[12] in 1996, reported the association between increased dialysis dose and lower mortality in specific group of patients stratified as having intermediate probability of death.

We performed a prospective observational cohort study to evaluate the effect of delivered dose of daily intermittent hemodialysis (IHD) on mortality in patients with AKI. Thereafter, AKI patients were stratified into subsets based on the presence or absence of sepsis and urine output. Our hypothesis was that a higher delivered dose of IHD would be associated with better patients' outcomes.

PATIENTS AND METHODS

Study Population

This study was conducted from January 2004 to January 2009 in patients enrolled in two Brazilian University Hospitals (Botucatu School of Medicine and Bauru State of Sao Paulo). The protocol was approved by the Institutional Ethical Committee. Written informed consent was obtained from patients or their next of kin.

Patients were eligible for enrollment if they were 18 years of age or older, had acute tubular necrosis (ATN) as etiology of AKI, and were treated with at least two sessions of IHD. AKI was defined as a rising serum creatinine level according to Acute Kidney Network

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Criteria[13] and ATN as a history of prolonged hypotension, severe nephrotoxic drugs overdose, or excess endogenous nephrotoxic pigments (hemoglobinuria, myoglobinuria). Diagnosis was based on clinical history, results of physical examination, relevant blood tests, urinalysis (microscopical examination of urinary sediment), a fractional excretion of sodium that exceeded 1%, and the findings on renal ultrasonography. The indications for dialysis were uremic symptoms, blood urea nitrogen (BUN) level (>100 mg per 100 mL), fluid overload, electrolyte imbalance (potassium >6 mEq/L after clinical treatment), or acid–base refractory disturbances (bicarbonate <10 mEq/L after replacement).

Exclusion criteria were patients with severe chronic kidney disease (baseline serum creatinine higher than 4 mg/dL), kidney transplantation, hemodynamic instability defined as noradrenalin dose higher than 0.5 ucg/kg/min, and other etiologies of AKI.

Methods and Dialysis Dose

All patients included in the study were undergoing IHD according to the protocol already established in the services, as shown below.

AKI severity was determined according to Acute Tubular Necrosis Index Specific Score (ATN-ISS)[14] calculated at the moment of the first nephrology evaluation.

A hemodialysis (HD) machine with volumetric control (*Fresenius 4008F, Gambro K200*) and cellulose acetate dialyzers (*CA 170, 190 or 210*) were used for dialysis treatment.

An IHD session lasted at least 3 h 30 min and sessions were performed 6 times per week. Blood flux ranged from 250 to 350 mL/min and dialysate flux was 500 mL/min. Patients received heparin during the dialysis session (50–80 UI/kg/h). If anticoagulation was contraindicated, patients received 100 mL of saline solution every 1 h.

Bicarbonate, potassium, and sodium dialysate concentrations were adjusted according to individual requirements.

Prescribed Kt/V value was 1.2 per session. The delivered dose was determined by the single-pool Kt/V (spKt/V) value corrected for ultrafiltration but not for reappearance of urea nitrogen.[15]

Anthropometric measurements (weight, height, and body surface area) were obtained before dialysis. Mobile patients were weighed on a digital scale, and weights of immobilized patients were calculated from two variable formulas.[16]

The dialysis was interrupted when there was a partial recovery in renal function defined as urine output higher than 1000 mL per 24 h associated with a progressive drop in creatinine (<4 mg per 100 mL) and BUN levels (<50 mg per 100 mL), change of dialysis method, lack of renal function recovery 30 days after undergoing dialysis, or death.

Patient Allocation

HD adequacy was determined by using urea kinetic modeling based on spKt/V.[15] Thereafter, mean spKt/V per session was calculated for each patient and this value was multiplied by 6, achieving weekly delivered Kt/V for each patient.

Thereafter, patients were divided into

- Higher intensity dialysis (HID) group. Weekly delivered spKt/V higher than the median Kt/V.
- Lower intensity dialysis (LID) group. Weekly delivered spKt/V lower than the median Kt/V.

Thereafter, the same calculations and division into HID and LID were performed for AKI patients stratified into two subsets:

- Sepsis or non-sepsis defined according to *American College of Chest Physicians criteria*.[17]
- Oliguria and non-oliguria defined as urine output lower or higher than 0.5 mL/kg/h for at least 6 h.[13]

Statistical Analysis

Statistical analyses were performed using *SAS for Windows* software, version 9.2 (Cary, NC, USA).

Continuous variables were expressed as mean and SD or median and compared using the Student's t-test for parametric variables and Mann–Whitney test for non-parametric variables. Categorical variables were expressed as proportions and compared with the χ^2-test.

Multiple Cox regression analysis was then conducted to test the relationship between HD dose and mortality, adjusted for sex, age, weekly Kt/V, ATN-ISS, pre-BUN, vasoactive drugs use, mechanical ventilation, sepsis, urine output, clinical or surgical disease, and hypervolemia at the moment of dialysis indication.

At the end of the study, Kaplan–Meier survival curves were presented and compared using log-rank test for each group according to the intensity of HD dose. Statistical significance level was 5% ($p < 0.05$).

RESULTS

A total of 121 patients with ATN requiring HD were studied, of whom 61 were in the LID group and 60 were in the HID group. There was no difference between LID and HID groups in clinical, lab, and dialytical characteristics and outcome (Table 1). Septic patients showed higher ATN-ISS (68.6 ± 21.2 vs. 56.2 ± 22.0; $p = 0.003$) and were older (68.0 years vs. 60.5 years; $p = 0.04$) than non-septic patients.

Table 2 shows data from septic and non-septic patients according to the intensity of delivered dialysis dose. In both populations, there was no difference between LID and HID groups in gender, age, ATN-ISS, vasoactive drug use, mechanical ventilation, oliguria, etiology of AKI, and pre-dialysis BUN levels. However, in non-septic patients, the mortality rate was lower in the HID group than in the LID group at 30 days (14.3% vs. 47.6%; $p = 0.045$) and at the end of follow-up (14.3% vs. 52.4%; $p = 0.020$).

Table 1. Patients characteristics according to the intensity of weekly delivered dialysis dose.

		LID ($Kt/V \leq 5.16$)	HID ($Kt/V > 5.16$)	p
N	121	61	60	
Male sex	60.3	68.8	51.7	0.080
Age (years)	65.0 (53.0–75.0)	64.0 (51.7–75.0)	65.0 (54.0–76.0)	0.582
ATN-ISS	0.68 (0.44–0.79)	0.72 (0.47–0.82)	0.66 (0.41–0.79)	0.129
Vasoactive drugs	67.2	70.5	61.6	0.398
MV	72.2	77.0	65.0	0.199
Oliguria	62.8	68.8	56.6	0.231
Sepsis	65.3	67.2	63.3	0.797
Clinical patients	66.1	65.5	66.6	0.948
Ischemic AKI	70.3	72.2	68.4	0.884
Pre-BUN (mg/dL)	102.5 ± 38.3	101.7 ± 34.2	104.0 ± 42.3	0.749
Number of dialysis sessions	7.0 (4.0–14.0)	7.0 (5.0–11.0)	7.0 (4.0–15.5)	0.963
UF (mL/session)	2044 ± 707.1	2000.0 (1500.0–2500.0)	2211.0 (1705.0–2631.2)	0.199
Weekly Kt/V	5.16 (4.5–6.14)	4.36 ± 0.67	6.26 ± 0.71	<0.001
Mortality after 30 days	57.8	67.2	48.3	0.055
Mortality at the end of follow-up	60.3	68.8	51.7	0.080

Notes: Data are showed in %, mean ± SD, or median. ATN-ISS, Acute Tubular Necrosis Index Specific Score; MV, mechanical ventilation; UF, ultrafiltration; AKI, acute kidney injury; HID, higher intensity dialysis group; LID, lower intensity dialysis group.

Table 2. Septic and non-septic patient characteristics according to the intensity of weekly delivered dialysis dose.

	Sepsis			No sepsis		
	LID ($Kt/V \leq 5.16$)	HID ($Kt/V > 5.16$)	p	LID ($Kt/V \leq 5.2$)	HID ($Kt/V > 5.2$)	p
N	41	38		21	21	
Male sex	68.3	55.2	0.337	71.4	42.8	0.119
Age (years)	62.7 ± 16.6	67.1 ± 14.1	0.203	58.6 ± 18.4	56.8 ± 16.9	0.742
ATN-ISS	0.71 ± 0.20	0.65 ± 0.22	0.176	0.61 (0.37–0.78)	0.55 (0.31–0.72)	0.322
Vasoactive drugs	75.6	71.0	0.845	61.9	42.8	0.354
MV	82.9	71.0	0.308	66.7	47.6	0.444
Oliguria	68.3	63.2	0.808	71.4	42.8	0.119
Clinical patients	68.3	65.8	0.998	57.2	71.4	0.520
Ischemic AKI	75.6	73.7	0.579	61.9	61.9	0.607
Pre-BUN (mg/dL)	105.6 ± 31.7	108.5 ± 40.4	0.330	93.1 ± 37.5	97.0 ± 46.3	0.766
Number of dialysis sessions	7.0 (5.0–11.0)	7.0 (4.0–14.0)	0.933	7.0 (4.0–21.0)	7.0 (7.7–26.2)	0.705
UF (mL/session)	1948.4 ± 782.2	2224.5 ± 673.7	0.098	1989.8 ± 568.0	1962.3 ± 723.4	0.892
Weekly Kt/V	4.53 (3.84–4.96)	6.29 (5.76–6.84)	<0.001	4.4 ± 0.6	6.1 ± 0.7	<0.001
Mortality after 30 days	75.6	68.4	0.645	47.6	14.3	0.045
Mortality at the end of follow-up	75.6	73.6	0.950	52.4	14.3	0.020

Notes: Data are showed in %, mean ± SD, or median. ATN-ISS, Acute Tubular Necrosis Index Specific Score; MV, mechanical ventilation; UF, ultrafiltration; AKI, acute kidney injury; HID, higher intensity dialysis group; LID, lower intensity dialysis group.

Concerning urine output, patients with oliguria showed higher ATN-ISS than patients without oliguria (median ATN-ISS: 75 vs. 58.5; $p < 0.001$).

Table 3 shows data from oliguric and non-oliguric population according to the intensity of delivered dialysis dose. In both populations, there was no difference between LID and HID groups in gender, age, ATN-ISS, vasoactive drug use, mechanical ventilation, sepsis, etiology of AKI, and pre-dialysis BUN levels. However, in non-oliguric patients, the mortality rate was lower in the HID group than in the LID group (31.8% vs. 69.5%; $p = 0.025$).

Multiple Cox regression analysis did not show association between survival of non-septic patients and clinical, lab characteristics, or higher weekly delivered Kt/V ($p = 0.100$; odds ratio (OR) = 0.421). For non-oliguric patients, only the higher weekly delivered Kt/V was associated with better survival ($p = 0.024$; OR = 0.514). Figure 1 shows survival curves of different populations studied.

DISCUSSION

Despite decades of experience, there is still a lack of consensus on how dialysis dose should be utilized to optimally support patients with AKI and only a few studies have discussed IHD.[18,19] Palevsky et al.[8] and Vesconi et al.[18] in multicenter, randomized, and controlled trials showed that increasing the intensity of renal replacement therapy did not decrease mortality among AKI patients. However, in both of them, the

Table 3. Oliguric and non-oliguric patient characteristics according to the intensity of weekly delivered dialysis dose.

	Oliguria			No oliguria		
	LID ($Kt/V \leq 5.1$)	HID ($Kt/V > 5.1$)	*p*	LID ($Kt/V \leq 5.4$)	HID ($Kt/V > 5.4$)	*p*
N	39	37		23	22	
Male sex	64.1	54.0	0.511	73.9	50.0	0.178
Age (years)	61.1 ± 18.3	63.5 ± 15.0	0.534	64.0 (53.0–72.5)	66.5 (45.0–80.0)	0.481
ATN-ISS	0.74 (0.61–0.87)	0.75 (0.46–0.87)	0.432	0.55 ± 0.23	0.55 ± 0.19	0.957
Vasoactive drugs	71.8	67.6	0.880	65.2	54.5	0.665
MV	79.5	64.8	0.321	73.9	63.6	0.509
Sepsis	64.1	72.9	0.559	60.8	59.1	0.855
Clinical patients	69.2	62.2	0.684	60.8	72.7	0.598
Ischemic AKI	66.6	67.6	0.602	78.3	72.8	0.863
Pre-BUN (mg/dL)	99.0 ± 37.7	91.7 ± 38.3	0.405	117.1 ± 32.5	113.5 ± 40.0	0.741
Number of dialysis sessions	7.0 (5.0–11.0)	7.0 (4.0–17.7)	0.992	5.0 (4.0–12.5)	9.0 (4.0–16.0)	0.368
UF (mL/session)	1929.6 ± 684.5	2211.2 ± 691.3	0.079	2099.5 ± 729.1	1911.5 ± 733.2	0.393
Weekly Kt/V	4.2 ± 0.6	6.0 ± 0.7	<0.001	4.6 ± 0.7	6.6 ± 0.5	<0.001
Mortality after 30 days	64.1	59.4	0.857	69.5	31.8	0.025
Mortality at the end of follow-up	66.6	64.8	0.939	69.5	31.8	0.025

Notes: Data are showed in %, mean ± SD, or median. ATN-ISS, Acute Tubular Necrosis Index Specific Score; MV, mechanical ventilation; UF, ultrafiltration; AKI, acute kidney injury; HID, higher intensity dialysis group; LID, lower intensity dialysis group.

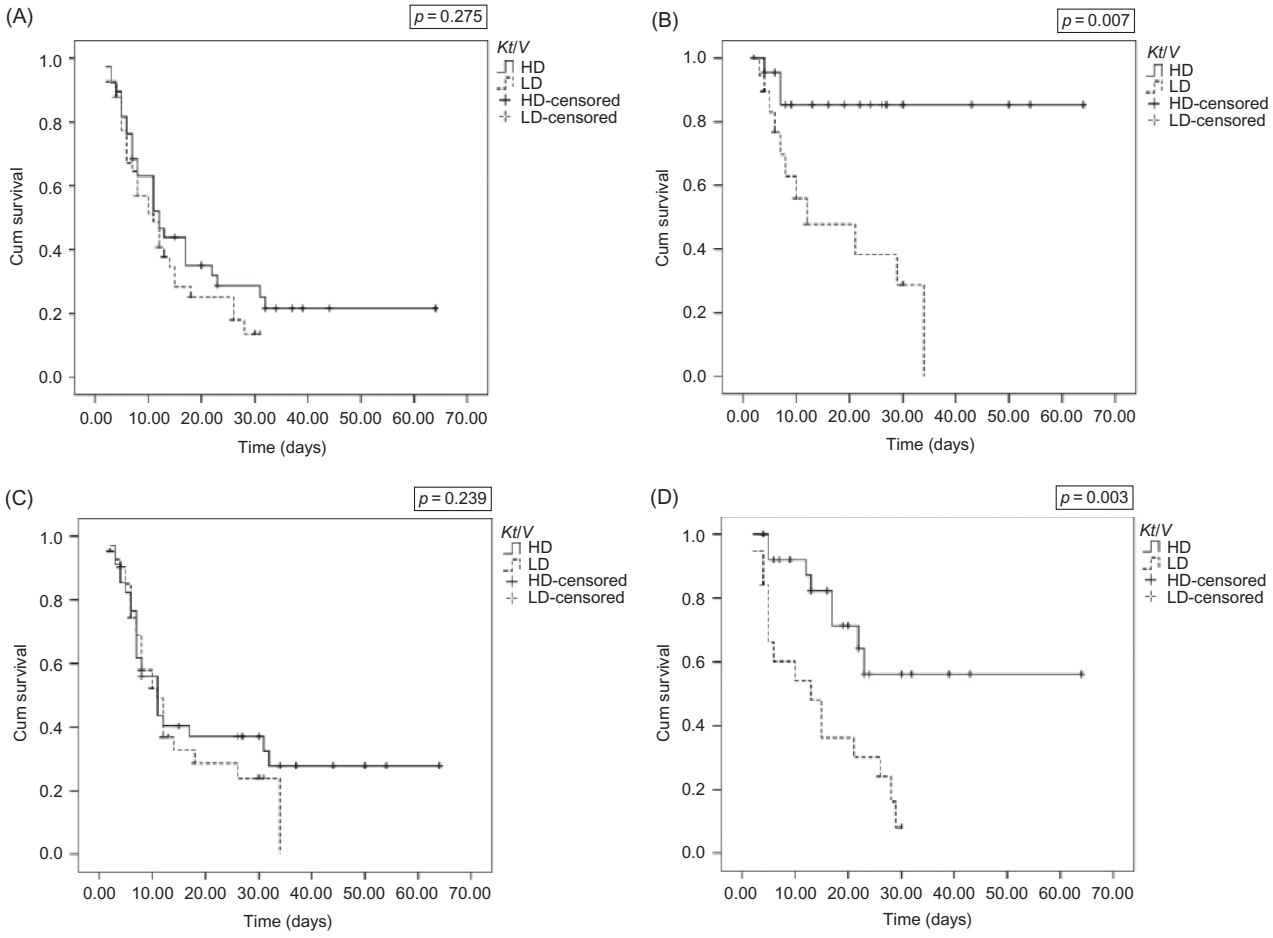

Figure 1. Survival curves of septic and non-septic patients [(A) and (B), respectively] and oliguric and non-oliguric patients [(C) and (D), respectively], according to the intensity of delivered dialysis dose.

intensity of dialysis was defined according to weekly frequency of dialysis sessions, different from this study that defined intensity of dialysis based on delivered dialysis dose, calculated by spKt/V urea.

Palevsky et al.[8] showed that the higher intensity therapy group underwent an average of 5.4 sessions of IHD or sustained low-efficiency dialysis per week, with an average interval between treatments of 1.1 days and

patients receiving lower intensity therapy underwent 3.0 sessions per week, with an average interval of 2.0 days. In both of them, delivered Kt/V urea per session was 1.32. Weekly delivered Kt/V in higher intensity group was around 6 versus 4 in lower intensity group, similar to that described in this study.

Schiffl et al.[6] reported a reduction in 28-day mortality from 46% with alternate day dialysis (weekly delivered $Kt/V = 3.0$) to 28% with daily dialysis (weekly delivered $Kt/V = 5.8$). These results, however, were answered because delivered dialysis dose per session was substantially lower than that recommended for chronic HD patients and the observed mortality rate was lower than that described in literature for similar critically ill patients.

Bouchard et al.[4] recently concluded that dialysis dose still matters and it should be monitored. They suggest the creation of a dose–survival curve, where Kt/V of 0.9 would correspond to the lower portion of the survival curve and Kt/V of 1.2–1.3 would correspond to the higher portion of the survival curve.

There is evidence for the importance of patient-related characteristics such as the severity of acute underlying disease in affecting the delivered dialysis dose, as measured by spKt/V urea.

A number of factors may reduce the delivered dose to AKI ill patients, such as dialysis complications (hypotension and clotting), treatment interrupted by diagnostic investigations, and mainly the imbalance of urea distribution, which reduces the overall effectiveness of urea removal and overestimates delivered dialysis dose.[20] In the Acute Renal Failure Trial Network Study,[8] the dose delivered was 89% of that prescribed for higher intensity treatment.

Evanson et al.[21,22] demonstrated delivered Kt/V of 1.04 versus prescribed Kt/V of 1.25 and concluded that alterations in total body water and its compartmental distribution could explain this discrepancy.

Schiffl[23] also showed that delivered Kt/V urea values in critically ill patients with AKI treated with HD were lower than the prescribed Kt/V urea values (1.28 vs. 0.89) and concluded the importance of patient-related characteristics such as the severity of acute underlying disease in affecting the delivered dose of IHD. The blood flow to some compartments and skeletal muscle is relatively low in septic patients using vasoactive drugs and it can be responsible for the well-described rebound in plasma urea concentration that occurs after dialysis.

Paganini et al.[12] were the first to show that higher delivered dialysis dose ($Kt/V > 1.0$) seems to play a major role in patients with moderated levels of severity.

In this study, non-septic and non-oliguric patients who underwent higher weekly delivered IHD dose showed lower mortality rates after 30 days and better survival curves in univariate analysis. These two groups (HID and LID) were similar in clinical parameters and prognostic scores, such as gender, age, ATN-ISS, vasoactive drug use, mechanical ventilation, etiology of AKI, predialysis BUN levels, presence of sepsis, and urine output.

Therefore, these data lead again to association of delivered dialysis dose and patient outcome and it appears to have more influence among patients with moderate severity scores rather than those patients at either extreme of the scoring system.

Our findings are not consistent with those of recent studies, which did not show difference in mortality of patient groups treated with higher or lower intensity of dialysis dose. Palevsky et al.[8] published results from the ATN and there were no differences in mortality with higher or lower intensity of dialysis dose according to sex, oliguria, sepsis, and sequential organ failure assessment (SOFA) score. Van Wert et al.[24] concluded that there is no difference in mortality of septic patients when compared with non-septic patients treated with different dialysis intensity. However, these two studies evaluated patients treated with intermittent and continuous dialysis methods together, different from this study, which evaluated only patients treated with IHD.

This study had several limitations: the small number of patients, mainly after the division into subsets, which may explain the lack of association between survival and delivered dialysis dose in non-septic patients in multivariate analysis, and the delivered dialysis dose was evaluated only by spKt/V, without considering middle-molecule clearance, fluid removal, fluid overload, and metabolic control.

In summary, this study showed that increasing the intensity of daily HD dose reduces mortality in non-septic and non-oliguric patients and that a minimum HD dose should be delivered to all AKI patients.

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REFERENCES

[1] Dennen P, Douglas IS, Anderson R. Acute kidney injury in the intensive care unit: An update and primer for the intensivist. *Crit Care Med.* 2010;38(1):261–275.

[2] Chertow GM, Soroko SH, Paganini EP, et al. Mortality after acute renal failure: Models for prognostic stratification and risk adjustment. *Kidney Int.* 2006;70:1120–1126.

[3] Palevsky PM. Dialysis modality and dosing strategy in acute renal failure. *Semin Dial.* 2006;19:165–170.

[4] Bouchard J, Macedo E, Mehta RL. Dosing of renal replacement therapy in acute kidney injury: Lessons learned from clinical trials. *Am J Kidney Dis.* 2010;55:570–579.

[5] Ronco C, Bellomo R, Honel P, et al. Effects of different doses in continuous veno-venous hemofiltration on outcomes of acute renal failure: A prospective randomised trial. *Lancet.* 2000;355:26–30.

[6] Schiffl H, Lang SM, Fischer R. Daily hemodialysis and the outcome of acute renal failure. *N Engl J Med.* 2002;346(5):305–310.

[7] Saudan P, Niederberger M, De Seigneux S, et al. Adding a dialysis dose to continuous hemofiltration increases survival in patients with acute renal failure. *Kidney Int.* 2006; 70(7):1312–1317.

[8] Palevsky PM, Zhang JH, O'Connor TZ, et al. Intensity of renal support in critically ill patients with acute kidney injury. *N Engl J Med*. 2008;359(1):1–14.

[9] Faulhaber-Walter R, Hafer C, Jahr N, et al. The Hannover Dialysis Outcome study: Comparison of standard versus intensified extended dialysis for treatment of patients with acute kidney injury in the intensive care unit. *Nephrol Dial Transplant*. 2009;24(7):2179–2186.

[10] Bellomo R, Cass A, Cole L, et al. Intensity of continuous renal-replacement therapy in critically ill patients. *N Engl J Med*. 2009;361(17):1627–1638.

[11] Schrier RW. Fluid administration in critically ill patients with acute kidney injury. *Clin J Am Soc Nephrol*. 2010;5: 733–739.

[12] Paganini EP, Tapolyai M, Goormastic M, et al. Establishing a dialysis therapy/patient outcome link in intensive care unit acute dialysis for patients with acute renal failure. *Am J Kidney Dis*. 1996;28(Suppl. 3):81–89.

[13] Acute Kidney Injury Network Working Group. Improving outcomes from acute kidney injury: Report of an initiative. *Am J Kidney Dis*. 2007;50(1):1–4.

[14] Liano F, Gallego A, Pascual J, et al. Prognosis of acute tubular necrosis: An extended prospectively contrasted study. *Nephron*. 1993;63:21–23.

[15] Daugirdas JT, Blake PG, Ing TS. Physiologic principles and urea kinetic modeling. In: *Handbook of dialysis*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2007:25–58.

[16] Chumlea WC, Guo S, Roche AF, et al. Prediction of body weight for the nonambulatory elderly from anthropometry. *J Am Diet Assoc*. 1988;88(5):564–568.

[17] Trzeciak S, Zanotti-Cavazzoni S, Parrillo J, et al. Inclusion criteria for clinical trials in sepsis: Did the American College of Chest Physicians/Society of Critical Care Medicine consensus conference definitions of sepsis have an impact? *Chest*. 2005;127 (Suppl. 1):242–245.

[18] Vesconi S, Cruz DN, Fumagalli R, et al. Delivered dose of renal replacement therapy and mortality in critically ill patients with acute kidney injury. *Crit Care Med*. 2009;13(2):1–14.

[19] Granado RC, Mehta RL. Assessing and delivering dialysis dose in acute kidney injury. *Semin Dial*. 2011;24:157–163.

[20] Ronco C, Ricci Z, Bellomo R. Current worldwide practice of dialysis dose prescription in acute renal failure. *Curr Opin Crit Care*. 2006;12:551–556.

[21] Evanson JA, Himmelfarb J, Wingard R, et al. Prescribed versus delivered dialysis in acute renal failure patients. *Am J Kidney Dis*. 1998;32(5):731–738.

[22] Evanson JA, Ikizler A, Wingard R, et al. Measurement of the delivery of dialysis in acute renal failure. *Kidney Int*. 1999;55:1501–1508.

[23] Schiffl H. Disease severity adversely affects delivery of dialysis in acute renal failure. *Nephron Clin Pract*. 2007;107:163–169.

[24] Van Wert R, Friedrich JO, Scales DC, et al. High-dose renal replacement therapy for acute kidney injury: Systematic review and meta-analysis. *Crit Care Med*. 2010;38(5):1360–1369.