Ponce et al., J Nephrol Ther 2018, 8:5
DOI: 10.4172/2161-0959.1000321

Journal of Nephrology & Therapeutics

Research Article Open Access

Fluid Overload is a Predictor of Mortality for Septic Acute Kidney Injury Patients Undergoing Prolonged Hemodialysis

Daniela Ponce[1,2*], **Bianca Ballarin Albino**[1] , **Mariele Gobo-Oliveira**[1] **and André Luís Balbi**[1]

[1]*Botucatu School of Medicine, University São Paulo State-UNESP, Brazil*

[2]*Dentistry College of Bauru, University São Paulo State, Brazil*

[*]**Corresponding author:** Daniela Ponce, Course of Medicine, Dentistry College of Bauru, University São Paulo State, Brazil, Tel: 55-14-3235-8000; E-mail: dponce@fmb.unesp.br

Received date: October 03, 2018; **Accepted date:** October 05, 2018; **Published date:** October 13, 2018

Copyright: © 2018 Ponce D et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Acute kidney injury (AKI) is common in critical patients and positive fluid balance (FB) is associated with increased mortality. We evaluated the influence of FB on mortality in AKI patients treated with prolonged hemodialysis (PHD).

Methods: It was a prospective study that included patients with sepsis-associated AKI admitted to the ICU in use of norepinephrine and treated with PHD. The daily FB was registered at initiation of PHD and after 72 hours.

Results: Among the 194 patients included, the survivors group consisted of 35 patients. Multivariate logistic regression revealed that FBs before PHD initiation and after 72 hours were the only independent risk factors for 28-day mortality (OR=1.6, CI1.04-2.47, p=0.03 and OR=1.54, CI 1.16-2.04, p=0.002, respectively). Area under the receiver operating characteristic (ROC) curve for FB before PHD initiation was 0.69, cut-off point of 2.45l, sensitivity(S) of 77% and specificity(E) of 74%, which is far lower than the ROC curve for FB after three sessions of PHD (AUC=0.79), cut-off point of -0.51 l, S=81% and E=82%. Conclusion: FB measured throughout the dialysis treatment period was good predictor of short-term outcome. Cumulative FB higher than -0.51 after three session of PHD was better predictor of death than cumulative FB before dialysis initiation.

Keywords: Acute kidney injury; Fluid overload

Introduction

Acute kidney injury (AKI) is a common complication in critically ill patients with an incidence of around 35% [1,2]. The main etiology of AKI in patients admitted to intensive care units (ICU) is that associated with septic shock, and carries an observed in hospital mortality as high as 62%, particularly when renal replacement therapy (RRT) is required [3,4].

Early and aggressive volume resuscitation is fundamental in the treatment of hemodynamic instability in critically ill patients and improves patient survival. However, one important consequence of fluid administration is the risk of developing fluid overload (FO). Clinically, FO manifests as an expansion of interstitial space and increased venous pressure, resulting in tissue edema, organ dysfunction, and adverse outcomes as increased need for mechanical ventilation, higher severity of AKI and need for dialysis and increased risk for mortality [5,6].

In a European multicenter trial, Payen et al. observed positive FB as a risk factor for mortality in 60 days in critical AKI patients [7]. Wang et al. [8] in a multicenter ICU study showed the FB was greater in patients with AKI than in patients without AKI and that a higher cumulative FB was an important factor associated with 28-day mortality following AKI. A retrospective analysis of post-operative percentage FO in patients at AKI stage 3 after cardiac surgery showed that FO>7.2% was significantly associated with reduced 90-day survival (p<0.001) [9].

Consequently, instigation of RRT, mainly in the ICU setting, is often triggered by low urine output and FO despite a paucity of data supporting this approach. We aimed to evaluate the influence of FB on mortality in ICU patients with AKI treated with PHD sessions.

Methodology

Studied population

This study was a subanalysis of a larger unpublished study, which evaluated the mortality and recovery of renal function in acute kidney injury patients treated with extended dialysis. It was approved by the medical ethics committee of Plataforma Brasil (Protocol CAAE 28146714.6.0000.5411) and informed consent term was obtained from all participants or their legal representatives. The study was also registered in the ISRCTN Registry under the number ISRCTN33774458 (16th April 2014).

It was a prospective study that evaluated the influence of FB on outcomes among critically ill patients older than 18 years with AKI treated by PHD. Patients were followed up by the AKI Team in a Brazilian University Hospital for 50 consecutive months, from January 2012 until March 2016. The inclusion criteria were age higher 18, presence of sepsis according to the criteria proposed by the International Guideline for Management of Severe Sepsis and Septic Shock [10], AKI according to the criteria of KDIGO [11], and

admission to ICU using noradrenaline in a dose ranged from 0.3 to 0.7 µg/kg/min. Patients using noradrenaline dose lower than 0.3 µck/kg/min were treated by conventional intermittent HD, and using dose higher than 0.7 received continuous RRT.

We excluded from the study patients who were pregnant, treated by a type of dialysis other than PHD, who presented AKI of other etiologies, stage 4 or 5 chronic kidney disease, or a need for chronic RRT (dialysis or kidney transplantation).

HD sessions were accompanied by the same research team until the patient's final clinical outcome (recovery of kidney function or death).

Study protocol

After diagnosing the sepsis-associated AKI and indicating PHD as dialysis method, the central venous catheter was implanted to initiate treatment. We used proportion machines (Fresenius 4008) and filter polysulfone membranes (surface areas of 1.3 and 1.8 m2).

PHD sessions were performed with blood flows of 200 mL/min and dialysate flows of 300 ml/min. We used filters FX-60 or 80 filters, according to the patients' weight. During the sessions, the patients were anti-coagulated with a 50 to 100 IU/kg bolus dose of heparin, and then with 500 to 1000 IU/hour in the following hours. In cases of contraindication to anticoagulation, the system was washed with 50 mL of 0.9% sodium chloride every 30 minutes, throughout the entire procedure. The concentrations of bicarbonate (26-36 mEq/L), potassium (1-3 mEq/ L), sodium (140-145 mEq/L) and calcium (2.5-3.5 mEq/L) of the dialysis bath were adjusted according to the exams and individual needs of the patients. The ultrafiltration rate (UF) did not exceed 500 mL/hour and the bath temperature ranged from 35 to 35.5°C.

To solve hypotension during PHD, we applied protocols, which included saline infusion, decreasing or discontinuing UF and increasing the vasoactive drug, according to the clinical and volume conditions of the patient. Therapy was interrupted when, despite taking these measures, hemodynamic instability persisted and presented risks to the patient.

We collected clinical data (name, sex, age, comorbidities, main diagnosis, etiology of sepsis, score specific for AKI [12] and sequential organ failure assessment (SOFA) [13]. We also evaluated parameters related to PHD sessions as number of sessions, filter used, blood and dialysate flows, ultrafiltration (UF) prescribed and obtained, urea removal rate (URR) and intradialytic complication.

Hypotension was defined as systolic blood pressure (BP) below 90 mmHg, or as a sudden BP drop of 20 mmHg. Filter clotting was defined as the presence of blood clots in the circuit, composed of a dialyzer and lines, precluding the continuation of therapy. When we observed the presence of thrombi and blood clots in the system, we conducted saline flushes or administered an extra dose of heparin to prevent coagulation, according to medical prescription. As post-dialysis complications we evaluated the presence of hypokalemia and hypophosphatemia, characterized by serum levels below 3.5 mEq/L and 3.5 mg/dL, respectively [14,15].

Daily pre and post blood urea nitrogen (BUN), creatinine, sodium, potassium, phosphorus, calcium, venous blood gases) were quantified. The daily FB was recorded, daily and the cumulative FB was registered at pre dialysis moment and at 24, 48, and 72 h. To quantify the cumulative FB over 3 days, we used the following formula: sum of daily (fluid intake [liters]-total output [liters]) [16].

The protocol was interrupted when there were recovery renal function (urine output greater than 1000 mL/24 h associated with the progressive decline of creatinine (<4 mg/dL) and blood urea nitrogen (BUN) values (<50 mg/dL), over 28 days of follow up or the death of the patient [15].

Statistical analysis

We described the variables with normal distribution as mean value ± standard deviation and the variables with non-normal distribution as mean value and interquartile range.

We performed comparisons of the continuous variables between the two groups using Student's t-test for data with normal distribution and the Mann-Whitney test for non-normal data. For the comparative analysis of categorical variables we used the Chi-squared tests. We used univariate and multivariate logistic regression for the association of risk factors for death in 28 days.

We performed a receiver operating characteristic (ROC) analysis in order to derive the predictive FO at pre dialysis moment and after 24, 48 and 72 h RRT initiation and the whole ICU stay for 28-day mortality. We considered a 5% significance level in all of the tests performed.

For data analysis we used the SAS program for Windows, version 9.2 (developed in 2009, in Cary-North Carolina, USA).

Results

A hundred and ninety four patients with sepsis-associated AKI received 531 PHD sessions. Age was 60.8 ± 14.9 years, and 69.5% were male. The main infection focus was pulmonary and hypertension was the most prevalent of the comorbidities (52.5%), ATN-ISS was 0.77 ± 0.1 and SOFA score was 14.2 ± 2.9.

Survivors group (G1) was composed of 35 patients, while non survivors group (G2) was composed of 159 patients. When comparing clinical characteristics, both groups were similar in relation to gender, age, comorbidities as hypertension and diabetes, ATN-ISS and SOFA and need for mechanical ventilation.

The groups presented differences in weight (68.0 ± 18 vs. 76.3 ± 23.2, p=0.04), SOFA (12 ± 2.5 vs. 13.4 ± 3.3 p=0.01), pre and post dialysis FB (2.5 ± 1.5 vs. 3.67 ± 3.3, p=0.002 and -0.48 ± 0.4 vs. 1.27 ± 2.3, p=0.003 respectively), and final vasoactive drug dose (0.53 ± 0.32 vs. 0.74 ± 0.52 p=0.026), as shown in Table 1.

Parameters	General (n=194)	Survivors (n=35)	Non-survivors (n=159)	p value
Age (years)	60.8 ± 14.9	58.1 ± 16.1	61.4 ± 14.6	0.22

Males, n (%)	135(69.5)	21(60)	114(71.6)	0.24
Weight	74.8 ± 22.5	68.0 ± 18	76.3 ± 23.2	0.04
Infectious focus n (%)				
Pulmonary	80(41.2)	17(48.5)	63(39.6)	0.43
Abdominal	69(35.5)	10(28.5)	59(37.1)	0.44
Comorbidities n (%)				
Hypertension	102(52.5)	17(48.5)	85(53.4)	0.73
DM	53(27.2)	8(22.8)	45(28.3)	0.65
CKD	19(9.7)	1(2.8)	18(11.3)	0.22
ATN ISS	0.67 ± 0.1	0.66 ± 0.1	0.66 ± 0.2	0.96
SOFA	12.8 ± 2.9	12 ± 2.5	13.4 ± 3.3	0.01
Pre-dialysis FB(l)	3.36 ± 1.8	2.5 ± 1.5	3.67 ± 2	0.002
Post-dialysis FB(l)	0.88 ± 0.90	-0.48 ± 0.4	1.27 ± 2.3	0.003
Ur(mg/dL)	149.3 ± 65.7	149 ± 77.4	147.3 ± 59.3	0.88
Cr(mg/dL)	3.7 ± 1.5	3.6 ± 1.5	3.7 ± 1.5	0.89
K(mEq/L)	4.7 ± 1	4.3 ± 0.9	4.8 ± 1	0.72
Bic(mEq/L)	19.1 ± 4.6	20.4 ± 4.3	18.8 ± 4.7	0.23
Mechanical ventilation	182(93.8)	30(85.7)	152(95.5)	0.07
Initial vasoactive drug dose	0.55 ± 0.18	0.45 ± 0.23	0.58 ± 0.43	0.07
Final vasoactive drug dose	0.69 ± 0.19	0.53 ± 0.32	0.74 ± 0.52	0.026

Table 1: Clinical and laboratory characteristics of patients with AKI treated with PHD.

Logistic regression was conducted for death and the variables weight, SOFA, pre and post three PHD sessions FB, pre-session potassium, presence of hypotension and hypokalemia were identified as risk factors. After the multivariate analysis, the association was maintained only with FB before and after three PHD sessions (OR=1.6, CI 1.04-2.47, p=0.03 and OR=1.54, CI 1.16-2.04, p=0.002, respectively). These data are shown in Tables 2 and 3.

Parameter	OR	Confidence Interval	p value
Age	1.01	0.99-1.03	0.24
Gender	0.6	0.28-1.28	0.19
Weight	1.01	1.00-1.03	0.04
Infectious focus	0.95	0.33-2.70	0.87
Hypertension	0.84	0.40-1.75	0.64
DM	0.73	0.31-1.74	0.48
CKD	0.22	0.02-1.76	0.15
ATN-ISS	0.94	0.11-7.98	0.95
SOFA	1.22	1.06-1.40	0.004
Pre FB	1.47	1.12-1.93	0.004

Post FB	1.38	1.11-1.72	0.003
Pre Ur	0.99	0.99-1.0	0.15
Post Ur	1	0.99-1.01	0.67
Pre Cr	1.01	0.80-1.28	0.88
CR post	0.94	0.60-1.47	0.78
Pre K	1.79	1.18-2.73	**0.24**
Post K	1.82	0.82-4.00	**0.44**
Pre Bic	0.93	0.85-1.01	0.11
Post Bic	0.9	0.75-1.08	0.27
Pre UF	0.93	0.78-1.12	0.49
Post UF	0.86	0.67-1.12	0.28
RF outcome	1	<0,001->999,999	0.85
Hypotension	0.5	0.23-1.09	0.08
Coagulation	0.76	0.34-1.70	0.5
Hypokalemia	3.7	1.62-8.83	0.002
Hypophosphataemia	1.48	0.66-3.31	0.33
OR: Odds Ratio; AKI: Acute Renal Injury; PHD: Prolonged Hemodialysis; DM: *Diabetes mellitus*; CKD: Chronic Kidney Disease; ATN-ISS: Acute Tubular Necrosis Individual Severity Score; SOFA: Sequential Organ Failure Assessment Score; FB: Fluid Balance; Ur: Urea; Cr: Creatinine; K: potassium; Bic: Bicarbonate, UF: Ultrafiltration, RF: Renal Function; Pre: 1st Session; Post: 3rd Session of PHD			

Table 2: Univariate logistic regression of clinical and laboratory characteristics, and dialysis complications associated with the death of patients with AKI treated with PHD.

Parameter	OR	Confidence Interval	p value
Weight	1.01	0.98-1.04	0.51
SOFA	1.08	0.34-1.38	0.52
Pre FB	1.60	1.04-2.47	0.03
Post FB	1.54	1.16-2.04	0.002
Hypokalemia	1.69	0.39-7.22	0.47
OR: Odds Ratio; AKI: Acute Kidney Injury; HDP: Prolonged Hemodialysis; SOFA: Sequential Organ Failure Assessment Score; FB: Fluid Balance			

Table 3: Multivariate logistic regression of clinical and laboratory characteristics, and dialysis complications associated with the death of patients with AKI treated with PHD.

	Area under curve	p	cutoff	Sensitivity	Specificity	CI (95%)
Pre dialysis FB	0.69	0.004	2.45 l	77%	74%	(0.55 – 0.78)
Post dialysis FB	0.79	0.003	-0.5 l	81%	82%	(0.61- 0.89)
FB: Fluid Balance						

Table 4: Fluid balance sensitivity and specificity in septic AKI patients on prolonged hemodialysis.

The ROC curves of cumulative FB before and after the first threes PHD sessions were employed to predict the 28-day mortality. We observed that cumulative positive FB at initiation of dialysis was predictor of death (AUC=0.69), cut-off point was 2.45 l, sensitivity (S)

was 77% and specificity (E) was 74%, as well as the cumulative FB after three PHD sessions.

With AUC=0.79, cutoff value was -0.5 l, S=81% and E=82%. There was no significant difference in FB between G1 and G2 after the first and second PHD sessions. These data are shown in the Figures 1-3 and Table 4.

Discussion

We investigated the influence of FB on mortality among critically ill AKI patients associated with sepsis treated by PHD in a University Hospital from a developing country. There are few studies in the literature that evaluated this association at the moment of dialysis initiation and during treatment.

Our study has shown that FB during different time periods in septic patients undergoing PHD lead to a worsened prognosis. The mean FB was higher in non-survivors group. According to the multivariable model, the presence of higher cumulative FB at moment of dialysis indication and after 72 h of PHD treatment were independent factor associated with increased 28-day mortality, and these findings are consistent with those of prior studies [8,16-18].

Wang et al. in a multicenter prospective, observation study conducted in 30 ICUs among 28 tertiary hospitals in China showed the FB was greater in patients with AKI than in patients without AKI. Fluid overload (FO) was an independent risk factor for the incidence of AKI and increased the severity of AKI and a higher cumulative FB was an important factor associated with 28-day mortality following AKI [8].

Bouchard et al. [16] evaluated 618 critically adult population with AKI in the PICARD study and found that, at the time of AKI diagnosis, the percentage of fluid accumulation in relation to the patient's weight upon ICU admission was lower among survivors than non-survivors (p=0.01). When the rate of fluid accumulation of all patients was greater than 10%, the mortality at 30 and 60 daysclimbed from 25% to 37% (P=0.02) and from 35% to 48% (P=0.01), respectively. Patients who maintained fluid accumulation after dialysis treatment had higher mortality proportional to fluid buildup (p<0.001).

Kim et al. [17] studied 341 AKI patients treated by continuous HD and concluded that in septic patients and/or high SOFA, the presence of positive FB was associated with lower survival. Silversides et al. [18] carried out a study with 492 AKI patients on dialysis and identified positive FB and presence of hypotension as risk factors for death.

Vaara et al. [19] demonstrated a direct association between cumulative FB at RRT initiation and an increased risk of 90-day mortality.

The relationship of positive cumulative FB and mortality associated with AKI is complex. It is not possible to determine whether the positive FB found in AKI patients was the cause or the consequence of a more severe illness. Maybe there was a higher severity of illness and hypotension among those patients who received more fluids, which are well-known clinical risk factors for mortality. There are many factors that affect the prognosis of AKI patients and we need to perform a propensity analysis to explore the confounding factors. Anyway, positive FB is directly related to death because it can cause consequences such as interstitial edema, oxygen diffusion and impaired metabolites, increased intra-abdominal pressure and higher time in mechanical ventilation, which aggravates the patient's clinical status [5,6].

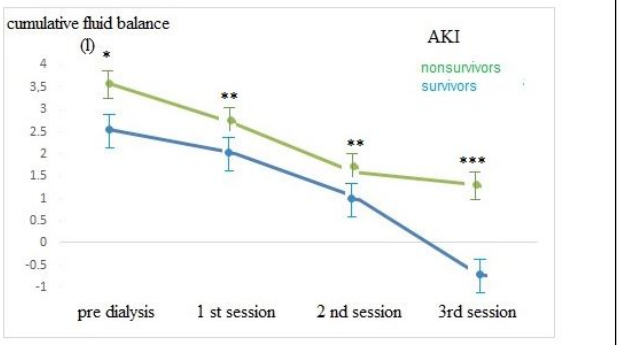

Figure 1: Cumulative fluid balance in acute kidney injury (AKI) survivors and non-survivors at pre-dialysis moment and in the first 3 days of prolonged hemodialysis treatment (mean ± standard error) [*p=0.04, **p>0.05, ***p=0.03].

In fact, several lines of evidence suggest that fluid therapy, rather than preserving renal function, actually precipitates or worsens AKI by causing positive FB. However, the cutoff value is not clear, because an elaborate ROC curve analysis is required.

In our study, the ROC curves of cumulative FB before and after PHD initiation were employed to predict the 28-day mortality. The results suggest that the areas under the ROC for the moment pre dialysis indication and after 72 hours of therapy were relatively larger (around 0.7 and up to 0.75, respectively). In our study, the best cut-off values of FB were 2.45 l (S=77% and E=74%) before dialysis initiation and -0.5 l after three sessions (S and E higher than 80%).

Our findings are in agreement with several other studies also showed positive FB increased mortality among ICU patients [7-9,16-21]. Bagshaw et al. [20] carried out a prospective multicenter observational study in patients with AKI and concluded that a positive FB above 3 liters at initiation of RRT (OR 2.3, 95% CI 1.2-4.5) or FO>5% (OR 2.3, 95% CI 1.2-4.7) were associated with higher mortality.

Xu et al. (21) performed a retrospective analysis in 280 patients with AKI stage 3 after cardiac surgery and the ROC curves of FO within 24 hours, before and after RRT initiation, and FO during the whole ICU stay postoperatively, were employed to predict the 90-day mortality. The results suggest that the area under the ROC for the whole ICU stay period was relatively larger (up to 0.752), with a cut-off point of 7.2%.

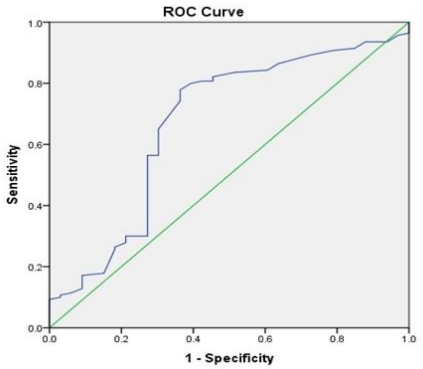

Figure 2: ROC curve performed to analyze the fluid balance before the start of Prolonged Hemodialysis.

Recently, Salahuddin N et al. carried out a single-center prospective, observational study to explore whether FO was an independent risk factor for AKI. Three hundred thirty nine patients were included; AKI developed in 141 (41.6%) patients and FB was significantly higher in patients with AKI. On multivariate regression analysis, a net FB in first 24 h of ICU admission, percentage of fluid accumulation adjusted for body weight, FB in first 24 h of ICU admission, age, vasopressor requirement on ICU day one, and colistin use were significant predictors of AKI [9].

According to a narrative review conducted in the UK, positive FB has been identified as an important short-term effect of AKI, directly related to worsening morbidity and mortality [22]. FO is also directly related to the presence of sepsis and may accelerate the need for the initiation of RRT, which can increase the association with risk for death because it is an invasive procedure [22]. In a meta-analysis performed, Zhang et al. [23] that included 5,095 patients from 12 published studies between 2008 and 2014, a significant association between FO and mortality in patients with AKI was identified, with similar findings for septic and non-septic patients. They also described a significant relationship between mean FB and mortality, considering it a risk factor for death in patients with AKI.

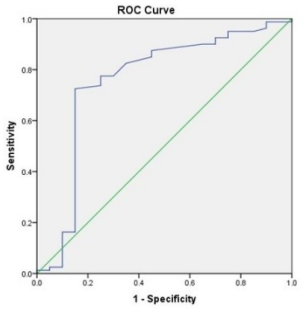

Figure 3: ROC curve performed for fluid balance analysis after three first sessions of prolonged hemodialysis.

Therefore, positive FB before RRT initiation has always been considered to be an important predictor of mortality. However, the higher cumulative FB before RRT initiation can indicate a severe illness status, and this may account for the higher mortality in patients with a higher fluid load before RRT initiation. In the present study, the area under the ROC curve for FB before RRT initiation was 0.69, which is far lower than the area under the ROC curve for FB (area 0.79) after three sessions of PHD. We believe that FB before RRT initiation, which seemingly did not incorporate the effect of subsequent RRT on prognosis, may not be a very good indicator of outcome. The prognosis may be improved if the FB is controlled after RRT treatment, so that the FB during the RRT may be a better predictor of the outcome.

Our study showed that FB can predict outcomes for critically ill septic patients with AKI before and after dialysis initiation. However, it has some limitations. It is important to note that our research was as a single-center study with a relatively small sample size and some bias may have existed. In our study, fluid overload (positive FB/dry weight) was not analyzed. The long-term survival evaluation was also not performed and the predictive values of the FB were only identified until after the third session of PHD.

In agreement with previous studies, our results conclude that higher FB was an important factor associated with 28-day mortality among AKI patients on PHD. FB was higher in the non-survivors AKI group than in the survivors AKI group. FB measured throughout the dialysis treatment period was good predictor of the short-term outcome. Cumulative FB higher than -0.51 after three session of RRT was better predictor of death than cumulative FB before dialysis initiation.

Conclusion

Further studies are needed to investigate the mechanisms underlying the role of FB in patients with AKI and to identify techniques and strategies for volume control in critical patients undergoing HD. Nevertheless, we think that our results provide guidance for the volume control of critically ill septic Aki patients on RRT.

References

1. Md Ralib A, Mat Nor MB (2015) Acute Kidney Injury in a Malasyan intensive care unit: assessment of incidence, risk factors and outcome. J Crit Care 30: 636-642.
2. Chang CH, Fan PC, Chang MY, Tian YC, Hung CC, et al. (2014) Acute kidney injury enhances outcome prediction ability of sequential organ failure assessment score in critically ill patients. PLoS One 9: e109649.
3. Singh S, Patra AK, Patel B, Ramesh GS, Sharma VK, et al. (2016) Acute renal failure in the ICU setting: A prospective observational study. Med J Armed Forces India 72: 236-241.
4. Caires RA, Abdulkader RC, Costa e Silva VT, Ferreira GS, Burdmann EA, et al. (2016) Sustained low-efficiency extended dialysis (SLED) with single-pass batch system in critically-ill patients with acute kidney injury (AKI). J Nephrol 29: 401-409.
5. Nguyen HB, Jaehne AK, Jayaprakash N, Semler MW, Hegab S, et al. (2016) Early goal-directed therapy in severe sepsis and septic shock: insights and comparisons to ProCESS, ProMISe, and ARISE. Crit Care 20: 160.
6. Granado RCL, Metha RL (2016) Fluid overload in the ICU: evaluation and management. BMC Nephrol 17: 109.
7. Payen D, de Pont AC, Sakr Y, Spies C, Reinhart K, et al. (2008) A positive fluid balance is associated with a worse outcome in patients with acute renal failure. Crit Care 12: R74.

8. Wang N, Jiang L, Zhu B, Wen Y, Xi XM, et al. (2015) Fluid balance and mortality in critically ill patients with acute kidney injury: a multicenter prospective epidemiological study. Crit Care 19: 371.
9. Xu J, Shen B, Fang Y, Liu Z, Zou J, et al. (2015) Postoperative fluid overload is a useful predictor of the short-term outcome of renal replacement therapy for acute kidney injury after cardiac surgery. Medicine (Baltimore) 94: e1360.
10. Levy MM, Delinger RP, Townsend SR, Linde-Zwirble WT, Marshall JC, et al. (2010) Surviving sepsis campaign: Results of an international guideline-based performance improvement program targeting severe sepsis. Crit Care Med 38: 367-74.
11. Kidney Disease: Improving Global Outcomes (2013) KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int (Suppl) 3: 1-150.
12. Liano F, Gallego A, Pascual J, García-Martín F, Teruel JL, et al. (1993) Prognosis of acute tubular necrosis: an extended prospectively contrasted study. Nephron 63: 21-23.
13. Ferreira FL, Bota DP, Bross A, Mélot C, Vincent JL (2001) Serial evaluation of the SOFA score to predict outcome in critically ill patients. JAMA 286: 1754-1758.
14. Bellomo R, Baldwin I, Fealy N (2002) Prolonged intermittent renal replacement therapy in the intensive care unit. Crit Care Resusc 4: 281-290.
15. Ponce D, Albino BB, Abrao JMG, Balbi AL (2013) Extended daily dialysis in acute kidney injury patients: metabolic and fluid control and risk factors for death. Plos One 8: e81697.
16. Kim IY, Kim JH, Lee Dw, Lee SB, Rhee H, et al. (2017) Fluid overload and survival in critically ill patients with acute kidney injury receiving continuous renal replacement therapy. Plos One 12: e0172137.
17. Bouchard J, Soroko SB, Chertow GM, Himmelfarb J, Ikizler TA, et al. (2009) Fluid accumulation, vsurvival and recovery of kidney function in critically ill patients withvacute kidney injury. Kidney Int 76: 422-427.
18. Silversides JA, Pinto R, Kuint R, Wald R, Hladunewich MA, et al. (2014) Fluid balance, intradialytic hypotension, and outcomes in critically ill patients undergoing renal replacement therapy: a cohort study. Crit Care 18: 624.
19. Vaara ST, Korhonen AM, Kaukonen KM, Nisula S, Inkinen O, et al. (2012) Fluid overload is associated with an increased risk for 90-day mortality in critically ill patients with renal replacement therapy: data from the prospective FINNAKI study. Crit Care 16: R197.
20. Bagshaw SM, Wald R, Barton J, Burns KE, Friedrich JO, et al. (2012) Clinical factors associated with initiation of renal replacement therapy in critically ill patients with acute kidney injury-a prospective multicenter observational study. J Crit Care 27: 268-275.
21. Haase-Fielitz A, Haase M, Bellomo R, Calzavacca P, Spura A, et al. (2017) Perioperative hemodynamic instability and fluid overload are associated with increasing acute kidney injury severity and worse outcome after cardiac surgery. Blood Purif 43: 298-308.
22. Doyle JF, Forni LG (2016) Acute kidney injury: short-term and long-term effects. Crit Care 20: 188.
23. Zhang L, Chen Z, Diao Y, Yang Y, Fu P (2015) Associations of fluid overload with mortality and kidney recovery in patients with acute kidney injury: A systematic review and meta-analysis. J Crit Care 30: 860.