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TANAFFOS

Sonography Guided Evaluation of Hemodynamic Indices in Critically Ill Patients Suffering from AKI during CRRT

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Background: Recent pieces of evidence have shown higher efficacy of continuous renal replacement therapy (CRRT) with regard to improvement of survival in critically ill patients by maintaining hemodynamic stability. The present study aimed to assess hemodynamic conditions before and after CRRT with the point-of-care approach.

Materials and Methods: The present interventional before-after study was performed on 20 critically ill patients with unstable hemodynamic status admitted to the ICU at Masih-e-Daneshvari Hospital in Tehran in 2019. They were candidate for CRRT due to acute kidney injury (AKI). The main pointed parameters for assessment before and after CRRT included heart rate, mean arterial pressure (MAP), central vein pressure (CVP), the carotid corrected flow time (FTc), carotid peak systolic velocity (PSV), inferior vena cava collapsibility (cIVC), resistive index (RI), and inferior vena cava (IVC) size and distensibility aided by ultrasonography.

Results: Regarding the changes in ultrasonography parameters after CRRT, except for carotid PSV, heart rate, and carotid area, other parameters showed a significant change. In this regard, IVC size, FTc, MAP, CVP, internal jugular vein (IJV) area, and RI all significantly decreased while IVC distensibility index significantly increased following CRRT. Similar changes were revealed in the subgroup of patients with hypotension, but in another subgroup without hypotension, the decrease in carotid PSV was also meaningful.

Conclusion: Applying CRRT in AKI patients in critically ill situations can effectively balance cardiovascular and hemodynamic parameters and thus lead to more appropriate survival.

Keywords: Acute Renal Failure; Continuous Renal Replacement Therapy; Medical Intensive Care Unit

INTRODUCTION

Acute kidney injury (AKI) has become a serious complication and a major contributor affecting outcomes in critically ill patients admitted to intensive care units (ICUs). Recent reports have suggested an upward trend of AKI in such patients that stage 1 of this complication is expected in over half of the patients in ICUs, while about 10 percent of patients may encounter higher stages requiring renal replacement therapy (RRT) (1-3). However, recent reports have acknowledged high rates of needing

RRT in about 70% of patients with AKI (4,5). Accordingly, the staging and stratifying systems have been developed to early identify the likelihood of AKI and its related survival in such patients. Also, epidemiological studies have attempted to describe AKI-related potential risk factors such as advanced age, history of heart failure, liver dysfunction, anemia, exposure to nephrotoxic agents, major surgeries, sepsis, and requiring mechanical ventilation (6,7). Hence, management strategies and supportive measures have been developed to avoid AKI. However, it should be pointed out that successfully applying these approaches requires continued multidisciplinary team efforts to optimize and standardize AKI management to make a difference in this devastating complication.

Several pieces of evidence have shown the high efficacy and safety of RRT leading to improvement of ICU patients' survival through maintenance of hemodynamic stability as well as modulation of immune response (8,9). In this regard, the use of continuous RRT (CRRT) is preferred even as compared to intermittent hemodialysis (IHD) (10). However, the common CRRT-related complications or the appropriate time for its initiation remains controversial. In other words, the triple concepts of "candidate selecting", "optimal timing", and "hemodynamic condition" for optimal initiating and setting CRRT should be specified to achieve the most appropriate therapeutic outcome (11,12). We believe that hemodynamic status both before and after CRRT sessions can potentially affect patients' outcomes. It should be noted of course that CRRT can be more successful than other supportive approaches in unstable hemodynamic conditions. It would also be much easier to comment on hemodynamic changes during this procedure. The present study aimed to assess hemodynamic conditions aided by sonography before and after CRRT with the point-of-care approach.

MATERIALS AND METHODS

The present interventional before-after study was performed on 20 critically ill patients with unstable hemodynamic status admitted to the ICU at Masih-e-Daneshvari Hospital in Tehran in 2019. They were candidate for CRRT due to AKI. Baseline variables were collected by reviewing the recorded files in the ICU. The main pointed parameters for assessment before and after CRRT included heart rate, mean arterial pressure (MAP), central vein pressure (CVP), the carotid corrected flow time (FTc), carotid peak systolic velocity (PSV), inferior vena cava collapsibility (cIVC), resistive index (RI) and IVC size aided by ultrasonography. In this regard, for the assessment of FTc, a 4.5–12.0 MHz linear array transducer was placed longitudinally on the neck. The long-axis B-mode image of the right common carotid artery was obtained at the level of the lower border of the thyroid cartilage. A pulsed wave Doppler tracing of flow through the artery was obtained with angle correction. The cycle time was obtained by determining the interval between heartbeats at the beginning of the Doppler flow upstroke, and flow time was measured from the beginning of the systolic upstroke to the dicrotic notch. FTc was determined by dividing the flow time by the square root of the cycle time as assessing a single cycle after several consecutive cycles became stable and reached the level of acceptable quality. Carotid PSV was measured from internal carotid artery Doppler waveforms. Angle correction consisted of aligning the angle cursor parallel to the vessel wall. To determine the size and collapsibility of IVC, M mode was applied and cIVC was determined by the ratio of the diameter difference of the inferior vena cava to the least diameter. RI was also defined as the ratio of (peak systolic velocity - end-diastolic velocity) to peak systolic velocity. Finally, all measurements were done once before dialysis and again after dialysis. All the measures performed in this study were following the ethical standards of clinical trials and informed consent was obtained from all guardians.

Descriptive analysis was used to describe the data, including mean ± standard deviation (SD) for quantitative variables. Independent t-test, Mann-Whitney U test, ANOVA test, or Kruskal-Wallis H test were used for

between-group comparing variables. The change in quantitative parameters after CRRT before that was examined by the Paired t-test or Wilcoxon test. For the statistical analysis, the statistical software IBM SPSS Statistics for Windows version 22.0 (IBM Corp. Released 2013, Armonk, New York) was used. P values <0.05 were considered statistically significant.

RESULTS

The baseline characteristics are summarized in Table 1. In total, 20 patients (mean age of 64.97 ± 15.70 years, 65.0% male) were included in the study. The mean time for hemodialysis was 20.10 ± 2.73. Overall, 80.0% needed mechanical ventilation, and increasing the dose of vasopressor was scheduled for 30.0%. The mean frequency of hypotension was 0.85 ± 0.43. Also, the mean initial SOFA and APACHE II scores were 13.60 ± 2.77 and 31.05 ± 5.99, respectively.

Table 1. Baseline characteristics of the study population

Items	Mean (SD) / n (%)
Age, year	64.97±15.7
Gender, Male	13 (65.0)
Requiring Mechanical Ventilation	16 (80.0)
CVVH	4 (20.0)
CVVHDF	14 (70.0)
Vasopressor Dose Increment	6 (30.0)
Time for Hemodialysis	20.10±2.73
SCUF	0.11±0.03
UFRATE	2185±797.4
IO Balance	-332.50±25.5
LVEF	41.0+11.9
SOFA	13.60±2.77
APACHE II	31.05±5.99
Episodes of Hypotension	
0	10(50.0)
1	5(25.0)
2	3(15.0)
3	2(10.0)

As shown in Table 2 regarding the changes in ultrasonography parameters after CRRT, except for carotid PSV, heart rate, and carotid area, other parameters showed a significant change. In this regard, IVC size, FTc, MAP, CVP, internal jugular vein (IJV) area, and RI all significantly decreased while IVC distensibility index significantly increased following CRRT. Similar changes were revealed in the subgroup of patients with hypotension, but in another subgroup without hypotension, the decrease in carotid PSV was also meaningful (Table 2, Figure 1).

In patients with and without hypotension, we showed no difference in mean IVC size before CRRT (1.99 ± 0.56cm versus 1.79±0.49cm, p=0.153) and also after CRRT (1.66±0.56cm versus 1.56±0.47cm, p=0.371). Also, we found no association between the episodes of hypotension and mean IVC size before (p=0.172) and after (p=0.394) CRRT (Table 3). Our study showed a significantly increased IVC distensibility index after CCRT as compared to before that (p<0.001). We revealed a lower IVC distensibility index in patients with episodes of hypotension as compared to those without any evidence of hypotension (14.92±3.88 versus 19.77±3.91, p=0.012) before CRRT, but this discrepancy was not revealed after CRRT (25.86±9.15 versus 27.08±3.98, p=0.704).

As indicated in Table 3, no association was found between the IVC distensibility index and the episodes of hypotension before (p=0.073) and after (p=0.118) CRRT. CRRT could lead to a significantly lower FTc (p<0.001). Before CRRT, the mean FTc was significantly lower in the patients with hypotension compared to those without hypotension (322.01 ± 13.94 versus 340.24±11.94, p=0.005) but was similar between the two groups after CRRT (305.01±17.80 versus 314.76±15.42, p=0.395). The episodes of hypotension were also associated with FTc before CRRT (p=0.021), but not with this parameter after CRRT (p=0.168) (Table 3).

The mean left ventricular ejection fraction (LVEF) did not differ in the hypotensive and normotensive groups (37.50±13.17% versus 44.50±10.12%, p=0.315). We also showed no association between the mean LVEF and the episodes of hypotension (p=0.159). We also showed no difference in carotid PSV between the patients with and without hypotension before CRRT (33.17±6.29 versus 35.21±3.58, p=0.481) as well as after that (33.57±8.33 versus 31.87±3.69, p=0.529). Similarly, in the subgroups with and without hypotension, the mean CVP before CRRT was

14.90±4.06mmHg and 13.50±3.31mmHg respectively reached 9.30±3.16mmHg and 7.50±2.27mmHg. Although CVP significantly reduced after CRRT in both groups (p<0.001), the between-group difference in this parameter was not significant before and after CRRT (p=0.315, p=0.165, respectively). As summarized in Table 3, the mean carotid PSV was considerably lower in those patients with three episodes of hypotension as compared to lower hypotension episodes both before and after CRRT.

Table 2. The overall changes in study parameters after CRRT

Parameter	Before	After	P-Value
Inferior vena cava size	1.9(0.53)	1.61(0.51)	<0.001*
Inferior vena cava distensibility	17.35(4.54)	26.47(6.90)	<0.001 *
Carotid corrected flow time	331.1(15.72)	309.88(16.96)	<0.001*
Carotid peak systolic velocity	34.19(5.10)	32.72(6.35)	0.097
mean arterial pressure	85.74(9.86)	76.43(9.69)	<0.001*
Heart rate	85.62(21.68)	85.95(15.14)	0.947
Central vein pressure	14.2(3.67)	8.4(2.83)	<0.001*
Carotid area	0.34(0.07)	0.31(0.10)	0.160
Internal jugular vein area	1.10(0.63)	0.92(0.48)	<0.001*
Resistive index	0.79(0.04)	0.66(0.05)	<0.001*
Hypotensive group			
Inferior vena cava size	1.99(0.57)	1.66(0.56)	<0.001 *
Inferior vena cava distensibility	14.92(3.88)	25.86(9.15)	0.004*
Carotid corrected flow time	322.0(13.94)	305.01(17.80)	0.004*
Carotid peak systolic velocity	33.17(6.29)	33.57(8.33)	0.788
mean arterial pressure	81.32(3.52)	72.44(9.08)	0.001*
Heart rate	88.25(28.12)	92.70(15.95)	0.646
Central vein pressure	14.90(4.06)	9.30(3.16)	<0.001*
Carotid area	0.34(0.08)	0.30(0.15)	0.097
Internal jugular vein area	1.03(0.81)	0.85(0.61)	0.025
Resistive index	0.79(0.04)	0.66(0.03)	<0.001*
Normotensive group			
Inferior vena cava size	1.79(0.49)	1.55(0.46)	<0.001*
Inferior vena cava distensibility	19.77(3.91)	27.08(3.98)	<0.001*
Carotid corrected flow time	340.24(11.94)	314.76(15.42)	0.004*
Carotid peak systolic velocity	35.21(3.58)	31.87(3.69)	0.001*
mean arterial pressure	90.15(12.23)	80.42(8.97)	<0.001*
Heart rate	83.00(13.62)	79.20(11.30)	0.187
Central vein pressure	13.5(3.31)	7.5(2.27)	<0.001*
Carotid area	0.34(0.05)	0.32(0.04)	0.157
Internal jugular vein area	1.16(0.40)	0.98(0.31)	0.002*
Resistive index	0.78(0.03)	0.66(0.05)	<0.001*

*:Significant at 0.05 level, The results are indicated as mean (SD)

Table 3. The association between the number of hypotension episodes and study parameters

Parameter	Before CRRT					After CRRT				
Episodes	0	1	2	3	P-Value	0	1	2	3	P-Value
IVC size	1.8(0.5)	1.7(0.5)	2.0(0.5)	2.6(0.1)	0.172	1.5(0.4)	1.4(0.5)	1.7(0.6)	2.1(1.5)	0.394
IVC dis.	19.7(3.9)	13.7(5.3)	17.1(0.9)	14.6(0.5)	0.073	27.1(3.9)	30.3(11.3)	21.8(3.8)	20.7(3.8)	0.118
FTc	340(11.9)	319(13.9)	333(3.3)	310(15.0)	0.021*	314(15.4)	301(14.0)	322(11.2)	287(14.8)	0.168
Carotid PSV	35.2(3.5)	34.4(2.7)	37.3(5.8)	23.6(3.5)	0.005*	31.8(3.7)	38.0(6.0)	34.4(5.6)	21.1(1.7)	0.004*
CVP	13.5(3.3)	12.2(4.3)	17.6(0.5)	14.2(3.6)	0.188	7.5(2.2)	7.4(3.5)	10.6(0.6)	12.0(1.4)	0.064

*:Significant at 0.05 level

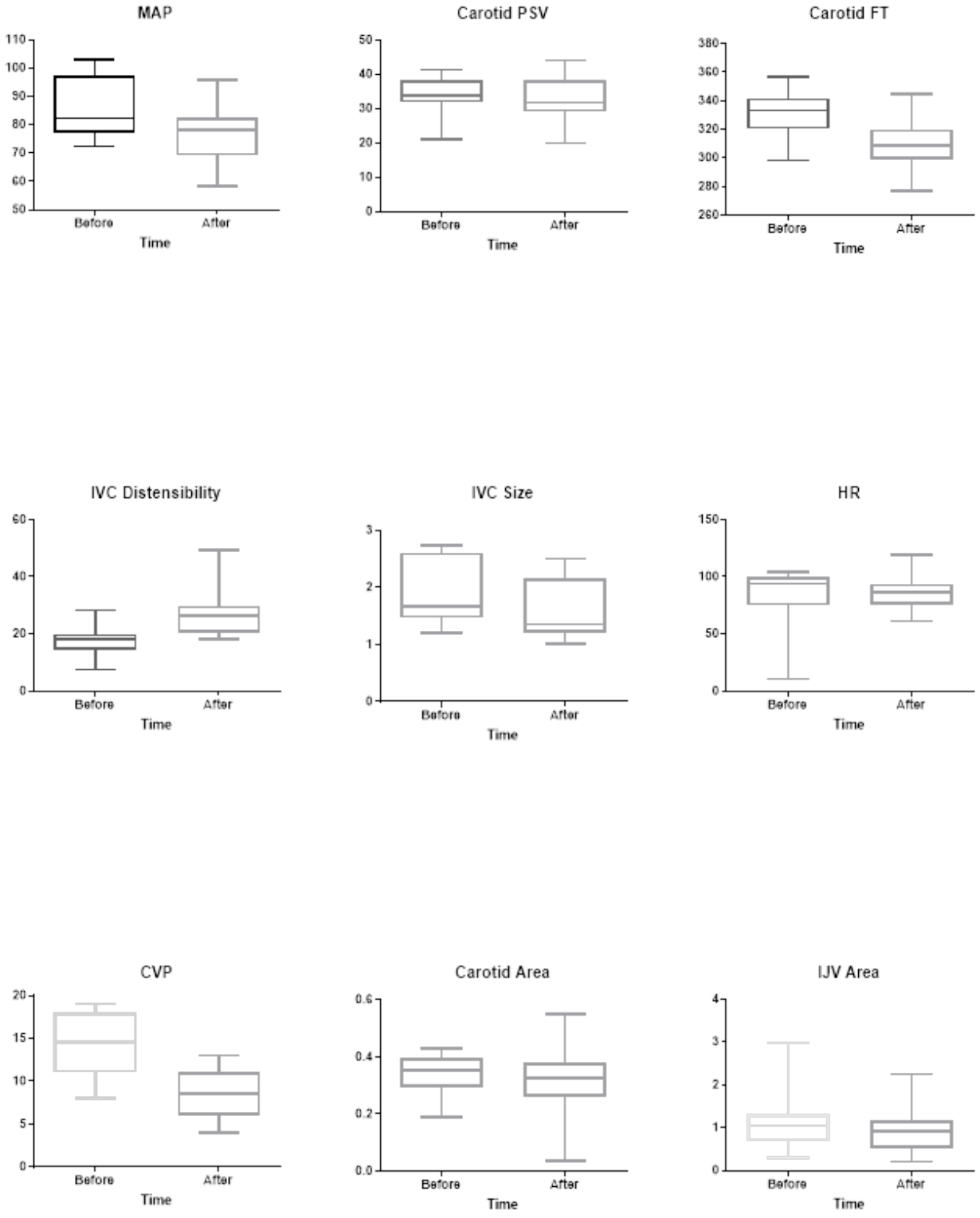

Figure 1. Box plots of the changes of parameters

Table 4. The study parameters in the groups with and without vasopressor increment

Parameter	Increment	Mean (SD)	P value
LVEF	No	46.4 (9.8)	0.002*
	Yes	28.3 (7.5)	
IVC size (before)	No	1.7 (0.4)	0.353
	Yes	2.2 (0.6)	
IVC size (after)	No	1.5 (0.4)	0.274
	Yes	1.9 (0.6)	
IVC distensibility (before)	No	17.9 (5.1)	0.274
	Yes	16.0 (2.8)	
IVC distensibility (after)	No	26.7 (3.9)	0.153
	Yes	25.9 (11.8)	
FTc (before)	No	338.0 (10.6)	0.002*
	Yes	315.1 (14.1)	
FTc (after)	No	314.6 (13.5)	0.091
	Yes	298.7 (20.1)	
Carotid PSV (before)	No	35.7 (3.7)	0.062
	Yes	30.7 (6.3)	
Carotid PSV (after)	No	33.8 (4.8)	0.312
	Yes	30.2 (9.1)	
CVP (before)	No	13.9 (3.2)	0.602
	Yes	14.8 (4.9)	
CVP (after)	No	7.9 (2.2)	0.312
	Yes	9.5 (4.0)	

*:Significant at 0.05 level

Adversely, CVP was not associated with the episodes of hypotension before and after CRRT. Finally, as indicated in Table 4, those patients requiring an increase in the dose of vasopressor had significantly lower LVEF and lower FTc before CRRT when compared to another group, however, this difference was not found after CRRT.

DISCUSSION

Continuously monitoring and managing hemodynamic conditions in AKI patients, particularly when admitted to ICUs and supported by mechanical ventilation is very vital because of the central role of the renal system in stabilizing hemodynamic status. Thus, management can be difficult if hemodynamic instability exists. Recently, CRRT has been introduced as a choice protocol for minutely monitoring and improving hemodynamic and physiologic parameters in AKI patients, especially in ICU setting (13-15). CRRT includes a dialysis protocol that can be provided as a continuous 24-hour treatment protocol. It seems that as compared to the intermittent approach, CRRT can successfully prevent wasting large amounts of water in a short time and thus can effectively help to stabilize hemodynamic indices followed by balancing pressures and volumes in the circulatory system (13). However, it remains unclear how CRRT can balance hemodynamic parameters. As clearly shown in the present study, CRRT can effectively compensate for the changes in some key factors such as IVC size and distensibility index, carotid FTc, CVP, internal jugular vein area, and RI. Most of these compensative changes appear independent of the presence of a hypotensive condition. Thus, as compared to intermittent RTT, CRRT has higher efficacy in normalizing hemodynamic parameters and cardiovascular bed indices in critically ill patients suffering from AKI.

Thus, it cannot be denied the key role of CRRT in the improvement of hemodynamic and circulatory parameters in AKI patients. It has been fully demonstrated that the normal functional status of kidneys depends on both good preload (flow and pressure) and afterload (CVP and venous congestion) (16). A study examining the factors that contribute to the occurrence of AKI in critically ill patients found that direct circulation dysfunction (including hypovolemia and cardiogenic shock)-induced AKI accounts for 47.3% of all cases of AKI (17). Thus, CRRT should balance such parameters appropriately to achieve acceptable renal function and thus patients' survival. As indicated in our study, major critical factors including CVP, carotid FTc, and IVC dynamic were balanced by CRRT. Balancing CVP and maintaining it at less than 8mmHg can reduce the risk of renal damage (18,19). Besides, venous congestion following right-sided heart failure can result in obstructing venous reflux leading to renal vein pressure elevation along with reducing glomerular filtration rate and ultimately leading to the occurrence of AKI (20). In other words, right ventricular dysfunction can be associated with elevated serum

creatinine due to venous congestion, even with a preserved cardiac output. Thus, any therapeutic interventions leading to normalization of cardiovascular flows and pressures can help to preserve and improve renal function in AKI patients, especially in ICUs.

However, it should be taken into consideration that to achieve the most appropriate outcome following CRRT, optimal timing to CRRT initiation is very critical. In some studies, delaying CRRT at least 16.5 hours after occurring AKI could lead to an improvement in survival and considerably reduce the risk of death (21). Adversely, other authors could show that early CRRT may reduce the risk of death and may improve the recovery of kidney function in critically ill patients with AKI (22). However, in a recent systematic review and meta-analysis, compared with the late initiation strategy, early initiation showed no significant advantage in mortality rate and length of the hospital, and ICU stay (23). Thus, the proper timing for CRRT initiation remains uncertain. In total, major indications as well as optimal baseline conditions appropriate for initiating CRRT should be further studied to maximize its clinical benefits.

CONCLUSION

It can be concluded that applying CRRT in AKI patients in critically ill situations can effectively balance cardiovascular and hemodynamic parameters. In this regard, some life-threatening changes in such patients can be modified early after setting CRRT which helps to compensate and balance renal function and thus improve patients' survival.

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