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Effect of Venous Congestion on Worsening Renal Function in Decompensated Heart Failure Patients

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ABSTRACT

Background: Cardiorenal syndrome has been defined as the simultaneous dysfunction of both the heart and the kidney. In this setting, worsening renal function (WRF) is a common finding.

Objective: The aim of work was to determine whether venous congestion, rather than impairment of cardiac output, is associated with the development of WRF in patients with decompensated heart failure (DHF).

Patients and Methods: This observational prospective study included a total of 30 adult Egyptians patients with DHF, attending at Ain Shams University Hospitals. Inclusion criteria: 1. Left ventricular ejection fraction < 50%. 2. CVP > 10 mmHg. 3. Right ventricular systolic pressure > 40 mmHg. ECHO was done for all subjects. Follow up of s. creatinine, GFR with MDRD equation, CVP, MABP and body weight were done daily for a week.

Results: At follow up, 11 (36.7%) subjects developed WRF and 19 (63.3%) did not. that there was an incremental risk in WRF with increasing baseline CVP, as it appears to be a near linear relationship, because if the baseline CVP reached >19 or > 24 mmHg, we observed a sharp increase in the incidence of WRF approaching 51% and 71%, respectively. Furthermore, the mean baseline CVP was higher in subjects who developed WRF (26.2±3.3 mmHg) versus those who did not (18.1±1.9 mmHg) (p<0.001).We have also noticed that baseline EF was significantly lower in subjects who developed WRF .We also noticed that Subjects who developed WRF had lower eGFR at baseline.

Conclusions: It could be concluded that in the setting of DHF, venous congestion (high CVP) may be the most important driving factor of WRF rather than low COP.

Keywords: Heart failure; Renal function; Kidney; Hemodynamic; Venous congestion.

INTRODUCTION

Heart and kidney performance are strictly interconnected, and communication between these two organs occurs via a variety of pathways, including hemodynamic and nonhemodynamic mechanisms **(1).**

This unique relationship and the interdependence of the kidneys and the heart are well recognized. These critical and dynamic connections between both acute and chronic cardiac dysfunction and acute and chronic kidney disease and the way dysfunction of one organ affects the other have been led to the characterization of the cardiorenal syndrome (CRS) as a separate entity **(2).** Even though decreased forward flow as a result of decreased cardiac output in Acute Decompensated Heart Failure (ADHF) can cause acute deterioration in kidney function, there are many reasons why this mechanism fails to completely explain the development of the CRS. Data from human studies have shown that increased central venous pressure (CVP) and jugular venous pressure (JVP) on examination are associated with worsening in kidney function. as well as increased mortality. Also, renal dysfunction is one of the most important comorbidities in chronic HF patients and is aggravated, or becomes more evident, during episodes of acute heart failure **(3)**.

Aim of the work was to determine whether venous congestion, rather than impairment of cardiac output, is associated with the development of WRF in patients with decompensated heart failure (DHF).

PATIENTS AND METHODS

This observational prospective study included a total of 30 adult Egyptians patients with DHF, attending at Ain Shams University Hospitals. Written informed consent of all the subjects was obtained.

Ethical approval:

The study protocol was reviewed and approved by Ethical Committee of Faculty of Medicine, Ain shams University.

The included 30 patients were ≥ 18 years old, presented with DHF and treated with intravenous or oral loop diuretics while continuing other needed medications as angiotensin converting enzyme inhibitors (ACEIs), beta-blockers (BBs), and spironolactone as tolerated.

Inclusion criteria: patients with Left ventricular ejection fraction (EF) < 50%, CVP > 10 mm Hg, Right ventricular systolic pressure (RVSP) > 40 mmHg).

Exclusion criteria: those on Renal replacement therapy, patients with immunologic or malignant diseases, hepatorenal syndrome, current infections (sepsis), congenital heart disease, intravenous inotropic support on admission and those on mechanical ventilation.

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The included subjects were divided into two groups; **Group (1)** consisted of 11 patients who developed WRF and **Group (2)** consisted of 19 patients without developing WRF.

The patients were subjected to the following: Complete history taking and physical examinations with stress on measurement of CVP on admission, mean arterial blood pressure (MABP) and manifestations of right sided heart failure. Serum **creatinine** on admission with calculation of the estimated glomerular filtration rate **(eGFR)** based on Modification of Diet in Renal Disease Abbreviated Equation (MDRD) [GFR=186× (S.Cr)-1.154× (age)-0.203× (0.742 if female) × (1.210 if African American)]. Measurement of UREA, Na, K, hemoglobin and **Echocardiography (**to asses EF, RVSP) were done. Follow up for s. creatinine, eGFR, CVP, MABP and body weight were done daily for a week. Complete hemodynamic assessment was collected in all subjects all through the study. The systemic blood pressure was measured noninvasively by an automatic cuff sphygmomanometer. The CVP was assessed at end of expiration with the subject in a 45degree supine position. eGFR was estimated daily using the MDRD equation A strict definition of the development of WRF as a rise in serum creatinine an absolute change by ≥ 0.3 mg/dl or a relative rise ≥ 25% from baseline within 72 hours from admission, consistent with several previous studies [4,5]. it considered any significant renal deterioration during the treatment period in the setting of low cardiac output and congestion as defined by the inclusion criteria. Sequential serum creatinine and blood urea nitrogen values were recorded on admission and daily throughout the hospitalization period, including the day of discharge.

Statistical Analysis

Pre-coded data was analysed by the Statistical Package of Social Science Software program, version 21 (SPSS). Data was summarized using range, mean, and standard deviation for quantitative variables and frequency and percentage for qualitative ones (median [interquartile range] for nonparametric data) and as a ratio for categorical data. Univariate comparisons of these variables were performed between baseline and follow-up variables and between subjects who developed WRF versus those who did not. Comparison between groups was performed using independent sample t-test for quantitative variables and Chi square test or Fisher's exact test for qualitative ones. Repeated measures ANOVA test was performed for paired quantitative variables with post hoc Bonferroni test. Pearson correlation coefficients were calculated to signify the association between different quantitative variables. P values less than 0.05 were considered statistically significant, and less than 0.01 were considered highly significant. Graphs were used to illustrate some information.

RESULTS

30 cases were described as follows: males were 18 (60%) and females were 12 (40%) and mean (±SD) age was 51±9.3 years. The comorbidities of the subjects included Hypertension (HTN) 46.7%, Ischemic Heart Disease (IHD) 46.7%, Diabetes Mellitus (DM) 40%, old Cerebrovascular Stroke (CVS) 13.3%, Bronchial Asthma (BA) 10%. Other comorbidities included Peripheral Vascular Disease (PVD), Interstitial Pulmonary Fibrosis (IPF), Deep Vein Thrombosis (DVT) of lower limb, Chronic Obstructive Uropathy and Parkinsonism.

33.3% of the patients who enrolled in the study with eGFR > 60 ml/min on admission, 46.7% with eGFR 30 – 59 ml/min and 20% with eGFR < 30 ml/min calculated by MDRD equation on admission. During the duration of the study after decongestion therapy, Overall, 11 subjects (36.7%) developed WRF and 19 (63.3%) did not. So, the study population divided into 2 groups (those who developed WRF and those who did not). On Comparison of parameters between patients who developed WRF and who did not we found, there was a statistically significant difference between 2 groups as regard mean Baseline Body Weight as it was greater in subjects who developed WRF versus who did not, and also there is more DM patients in group who developed WRF than those who do not. (*Shawn in table 1*). Also, on comparison of baseline measures between subjects who developed WRF and who didn't. Subjects who developed WRF were more likely to have lower (eGFR) at baseline, had also greater serum creatinine both at baseline and at discharge, higher baseline CVP, lower baseline EF in relation to subjects who did not. Regarding medication use there were no statistically significant difference in their usage at admission or during hospitalization to account for the occurrence of WRF. (*table2*)

Table (1): Comparison of parameters between patients who developed WRF and who did not according to baseline patient characteristics.

	Patients with WRF (n=11)		**Patients Without WRF (n=19)**		**P value**
	Mean ±SD		**Mean ±SD**		
Age (Years)	51.6 ±9.8		51.5 ± 9.3		0.8
BW (Kg)	99.5 ± 12.0		87.8± 12.1		**0.03**
	N	**%**	**N**	**%**	
Male	7	63.6	11	58	1.0
Female	4	36.4	8	42	
Smoking history	6	54	10	52	1.0
Other comorbidities					
HTN (mmHg)	7	63.6	7	36.8	0.3
IHD	7	63.6	7	36.8	0.3
DM	8	72.7	4	21.1	**0.009**
CVS	3	9.1	1	5.63	0.1
BA	1	9.1	2	10.5	1.0
PVD	2	18.2	0	0.0	0.4
IPF	0	0.0	1	5.3	1.0
DVT	0	0.0	1	5.3	1.0
Obstructive Uropathy	1	9.1	0	0.0	0.4
Parkinsonism	0	0.0	1	5.3	1.0

Table (2): Comparison of parameters between patients who developed WRF and who did not according to baseline patient comorbidities and medication use.

	Patients with WRF (n=11)	**Patients without WRF (n=19)**	**P value**
Baseline measures			
EF	33.2 ± 5.9	39.8 ± 7.5	**0.03**
RVSP	48. 9 ± 3. 6	46.5 ± 2.7	0.6
CVP	26.2 ± 3.3	18.1 ± 1.9	**<0.001**
Urea (mg/dl)	44.0 ± 14.8	30.2 ± 10.6	**0.01**
Creatinine (mg/dl)	2.1 ± 0.8	1.4 ± 0.4	**0.03**
Na^+ (mEq/L)	136.7 ± 5.6	138.3 ± 4.7	0.5
K^+ (mEq/L)	4.2 ± 0.5	4.3 ± 0.5	0.5
Hemoglobin (g/dl)	10.1 ± 1.1	10.1 ± 1.4	1.0
GFR (mL/min/1.73 m^2)	41.7 ± 24.9	53.7 ± 13.7	**0.03**
UOP	3.6 ± 1.2	3.6 ± 0.9	0.9
Medication	N %	N %	**P value**
ACEI	6 54.5	10 52.6	1.0
Dinitra	3 27.3	4 21.1	1.0
Spironolac	3 27.3	9 47.4	0.4
Digoxin	3 27.3	6 31.6	1.0
BB	6 54.5	11 57.9	1.0
Statin	6 54.5	9 47.4	1.0
Aspirin/ Hep	4 36.4	8 42.1	1.0

On comparing between patients who did and who did not develop WRF regarding mean doses of furosemide and Mean Arterial Pressure during hospitalization. We noticed that on admission the mean dose of furosemide was similar in both groups. However, on follow up the mean dose of furosemide was increased significantly in patients who developed WRF and becomes statistically significant on day 2.

(*table 3*) Regarding Mean arterial pressure (MAP), there were no significant difference between subjects who did and who did not develop WRF during duration of the study. (*table 3*). As regard the progression of renal functions among patients who developed WRF and who did not it remains statistically significant between the two groups during the duration of the study. (*table 4*)**.** We found that the mean CVP was significantly higher among patients who developed WRF versus who did not at both baseline and follow up. *(Table 5).*

Table (3): Comparison of parameters between patients who developed WRF and who did not according to mean dose of furosemide and changes of mean arterial pressure at baseline and follow up.

	Patients with WRF (n=11)			**Patients Without WRF (n=19)**			**P value**
Furosemide day 0	298.2	±	119.1	260.0	±	123.1	0.2
Furosemide day 1	327.3	±	126.3	272.6	±	115.7	0.2
Furosemide day 2	363.6	±	135.6	238.9	±	94.6	**0.02**
Furosemide day 3	312.7	±	136.0	210.5	±	109.8	**0.03**
Furosemide day 4	240.0	±	124.9	171.6	±	117.6	**0.02**
Furosemide day 5	234.5	±	126.2	132.6	±	96.3	**0.002**
Furosemide day 6	216.8	±	134.4	122.4	±	102.1	**0.009**
MAP day 0	94.5	±	12.9	94.7	±	14.6	1.0
MAP day 1	91.7	±	13.7	92.8	±	15.4	0.9
MAP day 2	89.4	±	11.8	89.5	±	12.7	0.9
MAP day 3	87.7	±	11.9	91.4	±	12.7	0.4
MAP day 4	86.1	±	11.9	91.6	±	9.3	0.1
MAP day 5	84.2	±	9.9	91.4	±	10.3	0.1
MAP day 6	85.8	±	11.5	90.4	±	9.6	0.2

Table (4): Comparison of parameters between patients who developed WRF and who didn't according to progress of renal function by time.

	Patients with WRF (n=11)			**Patients Without WRF (n=19)**			**P value**
Creatinine day 0	2.1	±	0.8	1.4	±	0.4	**0.03**
Creatinine day 1	2.1	±	0.7	1.6	±	0.4	**0.05**
Creatinine day 2	2.4	±	0.7	1.5	±	0.4	**0.002**
Creatinine day 3	2.6	±	0.7	1.4	±	0.4	**<0.001**
Creatinine day 4	2.7	±	0.7	1.3	±	0.4	**<0.001**
Creatinine day 5	2.8	±	0.7	1.2	±	0.4	**<0.001**
Creatinine day 6	2.6	±	0.7	1.2	±	0.3	**<0.001**
eGFR day 0	41.7	±	24.9	53.7	±	13.7	**0.03**
eGFR day 6	26.1	±	10.5	70.3	±	22.3	**<0.001**
UREA 0	44.0	±	14.8	30.2	±	10.6	**0.01**
UREA 6	59.8	±	20.5	25.8	±	14.9	**0.001**

Table (5): Comparison of parameters between patients who developed WRF and who did not according to changes of mean CVP by time.

	Patients with WRF (n=11)			**Patients without WRF (n=19)**			**P value**
CVP day 0	26.2	±	3.3	18.1	±	1.9	**<0.001**
CVP day 1	22.6	±	3.4	16.2	±	1.8	**<0.001**
CVP day 2	19.4	±	3.6	14.0	±	1.8	**<0.001**
CVP day 3	16.4	±	3.1	11.8	±	2.1	**<0.001**
CVP day 4	14.0	±	2.2	9.8	±	2.0	**<0.001**
CVP day 5	11.6	±	1.6	8.5	±	1.6	**<0.001**
CVP day 6	10.2	±	1.8	7.5	±	1.4	**<0.001**

We noticed there was an incremental risk in WRF with increasing categories of baseline CVP, with 71% of subjects presented with baseline CVP > 24 mmHg, developed WRF at follow up. Furthermore, the mean baseline CVP was statistically greater in subjects who developed WRF versus those with did not as shown previously in table (5). The development of WRF was associated with a lower baseline eGFR and higher baseline CVP. *(figure 1and2)*. We observed that the mean baseline EF was significantly lower in subjects who developed WRF versus those who did not (33.2 ± 5.9 % vs 39.8 ± 7.5, p = 0.03) . (figure 3)

Table (6): The Correlation between different baseline hemodynamics and baseline estimated glomerular filtration rate.

	GFR at day 0	
	r	**P**
EF	**0. 77**	**<0.001**
RVSP	-0.20	0.28
CVP	-0.28	0.13

Table (7): Hemodynamic variables on admission and follow up in all patients and Stratified according to those who developed WRF (n =11) and those who did not (n=19).

	All patients (n=30)		
	Baseline	**Follow up**	**P value**
EF	37.4 ± 7.6	38.0 ± 7.7	**<0.001**
RVSP	47.3 ± 3.2	46.1 ± 3.2	**<0.001**
CVP	21.1 ± 4.7	8.5 ± 2.0	**<0.001**

	Patients with WRF (n=11)			**Patients Without WRF (n=19)**		
	Baseline	**Follow up**	**P value**	**Baseline**	**Follow up**	**P value**
EF	33. 2 ± 5. 9	33.6 ± 5. 9	**0.003**	39.8 ± 7.5	40.5 ± 7.7	**0.001**
RVSP	48. 9 ± 3. 6	47. 6 ± 3. 6	**0.006**	46.5 ± 2.7	42.8 ± 10.1	**0.001**
CVP	26.2 ± 3.3	10.2 ± 1.8	**0.003**	18.1 ± 1.9	7.5 ± 1.4	**<0.001**

A significant correlation was observed between baseline EF and baseline renal function expressed eGFR **(r = 0.77, p < 0.001)**. We noticed also that there was no statistically significant correlation between baseline CVP or RVSP and baseline renal function could be found (P = NS). However, this correlation between CVP and GFR becomes statistically significant at follow up (r = -0. 79, p < 0.001). (*table 6).* Follow up EF during the duration of the study was almost stationary or with very slight improvement in both groups (p = NS) in contrast to either follow up CVP (p < 0.001) in both groups or follow up eGFR (p < 0.001) in subjects with WRF and in subjects without WRF. *(figure 4and5*)

We observed that hemodynamic alterations demonstrated significant improvements after measures of decongestion as expected (all, p < 0.001). (*table 7*). Also, there was significant drop of follow up Mean Arterial Pressure in relation to baseline (P = 0.007) in patients who developed WRF while there was no significant correlation between MAP and GFR at baseline (r = -0.42, p = 0.2) in subjects who developed WRF versus who did not.

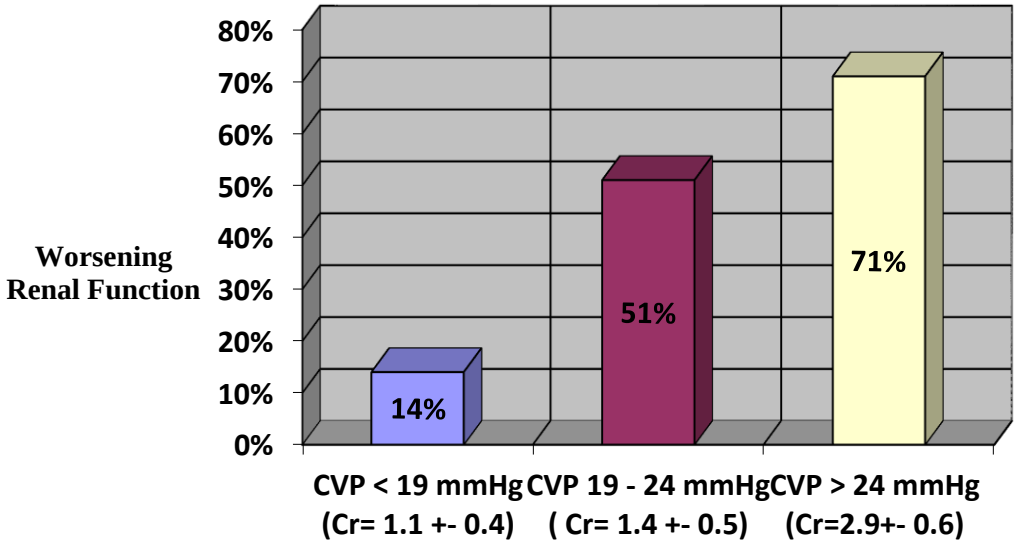

Figure (1): The Correlation between baseline CVP and WRF on follow up.

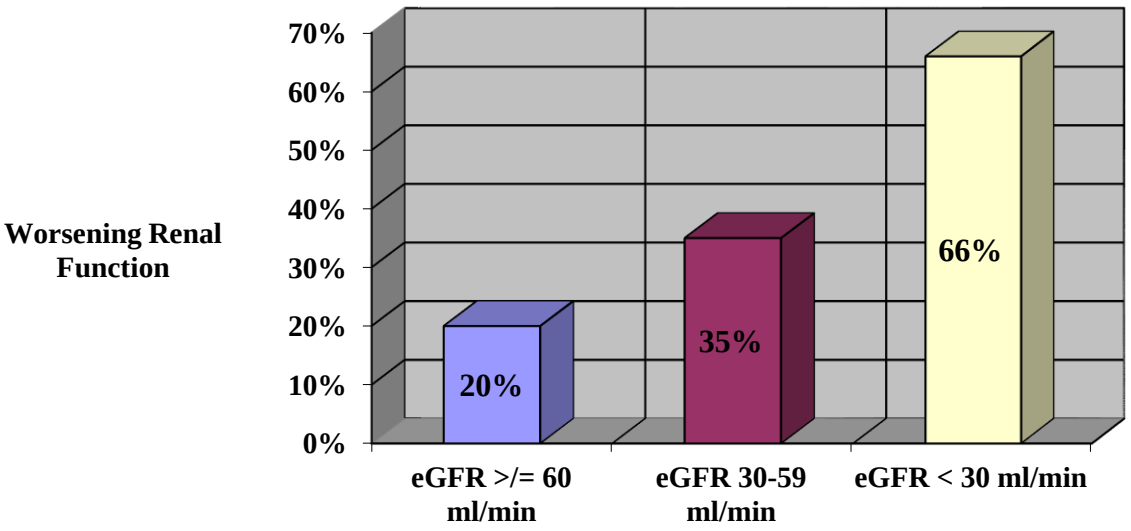

Figure (2): Correlation between baseline eGFR and WRF on follow up.

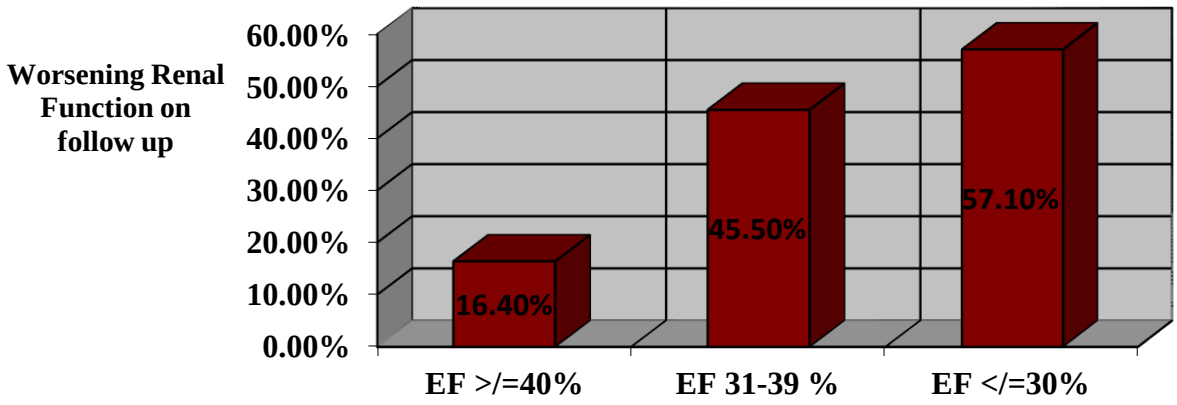

Figure (3): Correlation between baseline EF and WRF on follow up.

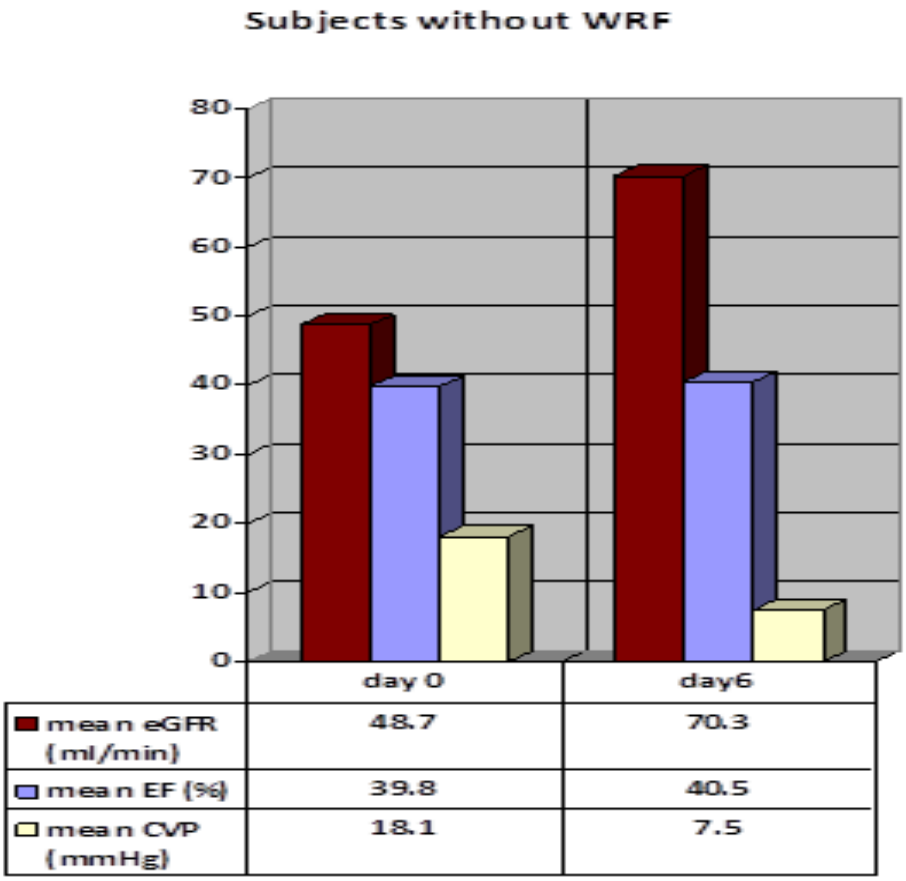

Figure (4): Pattern of changes of mean eGFR, mean EF and mean CVP on day 0 (baseline) and day 6 among subjects with and without WRF.

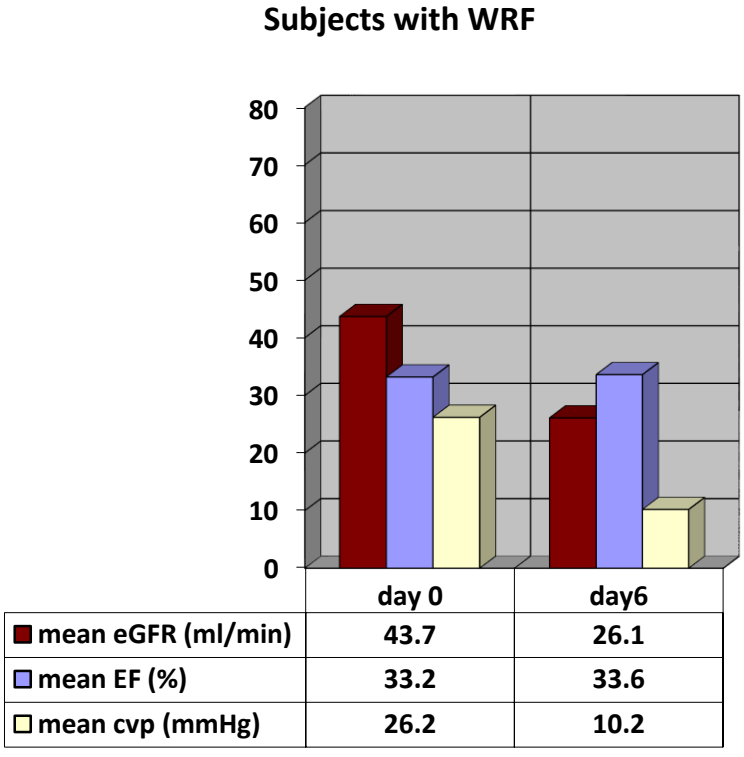

Figure (5): Pattern of changes of mean eGFR, mean EF and mean CVP on day 0 (baseline) and day 6 among subjects who developed WRF.

DISCUSSION

The current study showed that there was an incremental risk in WRF in patients with DHF with increasing values of baseline CVP. Also, we noticed that during treatment for ADHF, persistent venous congestion also posed a very high risk for the development of WRF, indicating that venous congestion evaluated by high central venous pressure may be the most important hemodynamic factor driving WRF in patients with DHF.

Our findings agreed with **Uthoff *et al.*** [6], who found association between a higher CVP and decreasing GFR and they reported that patients with high CVP had a relatively low incidence of WRF within the first 24 h but the highest absolute and relative incidence of WRF between 24 and 96 hours after admission. Although they also noticed that in patients with low SBP, high CVP at presentation and discharge was significantly associated with lower eGFR, while in patients with normal to high SBP; CVP seems to have no effect on eGFR, with no difference between patients with or without WRF**.**

Our finding was also in agreement with **Guglin *et al.*** [7] study**,** who noticed that serum creatinine was significantly higher and GFR was significantly lower in the upper tertile of CVP and PCWP (high cardiac filling pressure), as well as in the lower tertile of renal perfusion pressure. There were no significant differences in GFR across the tertiles of cardiac index (CI) or LVEF. Indicating that Renal dysfunction in heart failure is determined more by passive congestion than by low perfusion. Our findings were also in agreement with **Testani *et al.*** [8] study**.** They suggested that in heart failure patients with congestion assessed by echocardiography, right ventricular failure leads to venous congestion which is strongly associated with renal dysfunction and they also reported that relief of congestion likely drives improvement in renal function**.**

Our findings were also in agreement with **Mullens *et al.*** [9] study, who found that venous congestion evaluated by high CVP is the most important hemodynamic factor driving WRF in patients with decompensated heart failure rather than high CI. Our findings were also in agreement with **Damman *et al.*** [10] study**,** which was a retrospective data review of 2,557 patients with right heart catheterization. They found that only CVP remained associated with renal function in multivariate analysis. Venous congestion and CVP > 6 mm Hg associated with steep decrease in renal function. Confirming that increased CVP was associated with reduced GFR and all-cause mortality in a broad spectrum of cardiovascular patients.

However, our finding disagreed with **Dupont *et al.*** [11] study. They reported that during treatment of ADHF, Blood pressure decrease rather than alterations in cardiac output or central venous pressure were associated with changes in serum creatinine. Although they also found that baseline venous congestion is associated with the development of WRF especially in presence of low cardiac output.

We have noticed also that during decongestion therapy that 36.7% of patients developed WRF and 63.3% did not**.** Our findings agreed with **Mullens *et al.*** [9] study**,** they found that this incidence to be even greater (approaching 40% developed WRF) in a “cold and wet” patient population. Our findings agreed with **Uthoff *et al.*** [6] study, in this cohort study, overall, 140 patients with AHF at presentation, in-hospital WRF was observed in 36% of the patients [6]. We were also in agreement with **Smith *et al.*** [12] study**,** in this systematic review including 16 studies characterizing the association between renal impairment and mortality in 80,098 hospitalized and non-hospitalized HF patients. They reported that WRF occurs in about one-third of patients admitted with ADHF.

Our findings disagreed with **Dupont *et al.*** [11] study, in this retrospective, Worsening (WRF) and improvement (IRF) of RF were defined as a 25% increase or decrease in eGFR from time of admission to pulmonary artery catheter removal, respectively. Of 443 patients, only 46 (10%) experienced WRF and 127 (29%) had IRF. However, these varieties could be explained by different study population. We also noticed that percentage of subjects with Diabetes Mellitus were significant in those who developed WRF versus who did not**.** Our findings were also in agreement with **Chittineni *et al.*** [13] study**.** They reported that elevated admission serum creatinine, DM and lower serum sodium are risk factors and have been associated with the development of WRF.

Regarding medications used during decongestive therapy other than diuretics we found that there was no statistically significant difference in their usage at admission or during hospitalization to account for the occurrence of WRF. Our findings may agree with **Mullens *et al.*** [9] study**,** subjects who developed WRF versus those who did not had comparable baseline medication use on admission, apart from lower spironolactone utilization in those developing WRF are likely due to the relative contraindication of the drug in patients with intrinsic renal diseases. Although the initiation or maintenance of certain classes of drugs like angiotensin-converting enzyme inhibitors and loop diuretics has been linked to WRF, they did not find any difference in their usage at admission or during hospitalization to account for the occurrence of WRF.

Our findings were also in agreement with **Dupont *et al.*** [11] study, they reported that there was no difference in baseline medication or in treatment received during hospitalization account for the occurrence of WRF. However, Our findings were in disagreement with **Uthoff *et al.*** [6] study, they found that b-Blocker medication was significantly associated with WRF during hospitalization in univariate analysis and a

trend was observed with regard to age, diuretic medication, lower hemoglobin, and urea. In the multivariate analysis, no significant association was detected.

We noticed also that dose of furosemide was significantly higher in patients who developed WRF versus who did not. We noticed that on admission the mean dose of furosemide was similar in both groups However, on follow up the mean dose of furosemide was higher significantly in patients who developed WRF. Our findings agreed with **Voors *et al.*** [14] study, they found that higher dose of loop diuretics was also independently related to an increased risk of WRF. Our findings were also in agreement with **Felker *et al.*** [15] (the DOSE study), a prospective, randomized, double-blind, double-dummy, controlled trial. In which 308 patients were enrolled, comparing intravenous low-dose and high-dose loop diuretics, and intermittent bolus and continuous infusion of loop diuretics. The authors suggested that more aggressive decongestive strategies may not translate to better long-term outcomes. In comparing low-dose and high-dose furosemide, the authors found no significance but a trend towards improved efficacy in the high-dose group and no difference in the primary safety endpoint (change in serum creatinine, p=0.21). The high-dose group also demonstrated greater net fluid loss, weight loss, and relief of dyspnea, but a higher proportion of patients developed a deterioration of renal function during the first 72 hours although it was mostly transient [15].

Our findings were, however in disagreement with **Chittineni *et al.*** [13] study, in which they reported that neither diuretic dose nor ACEI/ARBS was associated with increased risk of WRF. We also noticed in our study that subjects who developed WRF were more likely to have lower (eGFR) at baseline, had also greater serum creatinine both at baseline (2.1 ± 0.8 mg/dl vs. 1.4 ± 0.4 mg/dl, P=0.03) and at discharge (2.6 ± 0.7 mg/dl vs. 1.2 ± 0.3 mg/dl, P < 0.001) in relation to subjects who did not. Overall, 66% of the subjects presented with baseline eGFR < 30 ml/min developed worsening renal function on follow up, 35% of subjects with baseline eGFR 30-59ml/min also developed WRF and only 20% of the subjects with baseline eGFR ≥ 60ml/min developed WRF on follow up. Our findings were also in agreement with **Voors *et al.*** [14] study. In this multivariable model, higher age, higher baseline creatinine, and a greater early drop in SBP, but not baseline SBP, remained independent predictors of WRF. Furthermore, WRF was associated with a higher Day 60 and Day 180 mortality. Our findings were also in agreement with **Mullens *et al.*** [9] study**,** as they observed in patients population with low-output decompensated HF that lower baseline eGFR increases the risk of WRF.

Our findings were also in disagreement with **Metra *et al.*** [16], in this Prospective cohort study, 318 patients with AHF were enrolled. They found no significant association of entry GFR with WRF. Apparently, indicating that GFR at presentation does not reliably predicts the reserve of the kidneys to tolerate the therapies initiated during AHF. We have noticed that in the setting of decompensated heart failure, baseline EF was significantly lower in subjects who developed WRF versus those who did not, but it has limited contribution on the pattern of changes in eGFR on follow up which is correlated significantly with CVP. We have noticed also that in decompensated heart failure patients, changes in eGFR were more likely to be related to changes in CVP and RVSP rather than to EF during decongestion therapy. Our findings were also in agreement **Mullens *et al.*** [9] study**,** they found that both increased CVP upon admission, and lack of sufficient reduction of CVP to values <8 mm Hg were associated with a greater incidence of WRF during hospitalization. Our findings were also in agreement with **Dupont *et al.*** [11] study, they found that baseline venous congestion is associated with the development of WRF especially in presence of low cardiac output and the degree of changes in right atrial pressure or CI did not affect the propensity for developing WRF or improved function in patients with decompensated heart failure.

Our findings were also in agreement with **Legrand *et al.*** [17] study, in this retrospective study between 2006 and 2010, included 137 ICU septic patients (69) had new or persistent AKI, they studied the association between the following hemodynamic targets within 24 hours of admission and AKI: CVP, COP, MAP, diastolic arterial pressure (DAP), central venous oxygen saturation (ScvO2) or mixed venous oxygen saturation (SvO2). MAP, ScvO2 and CO was not significantly different between groups. Patients with AKI had lower DAP and higher CVP. The rise in CVP was associated with a sharp increase in new or persistent AKI incidence even after adjustment for fluid balance and positive end-expiratory pressure (PEEP) level. A linear relationship between CVP and the risk of new or persistent AKI was observed. Suggesting a role of venous congestion in the development of AKI rather than CO [17].

CONCLUSION

It could be concluded that in the setting of decompensated heart failure, venous congestion (high CVP) may be the most important driving factor of changes in kidney functions rather than low cardiac output which has a little contribution on that changes and that also the drop of MAP is considered a risk factor for WRF during treatment of DHF.

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