Original Article

Renal Dysfunction in Patients with Heart Failure

Jawad K. Manuti* **FICMS**

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Summary:

Background: The coexistence of renal and heart failure carries an extremely bad prognosis. The exact cause of deterioration of kidney function and the mechanism underlying this interaction are complex, multifactorial in nature, and still not completely understood. Both the heart and the kidney act in tandem to regulate blood pressure, vascular tone, diuresis, natriuresis, etc.

Patients and methods: Sixty patients mean age 65.5 year were complaining heart failure duo to different causes assessed for renal function (blood urea &creatinine) and cardiac function by echocardiography in day 0 and 10 day after treatment of heart failure.

Results: The mean value (±S.D) of blood urea and serum creatinine on day 0 were 64.17mg/dL (±30.72) and 1.75mg/dL(±0.68) respectively. There is significant correlation between the severity of heart failure and the degree of renal impairment (P.value <0.05). after 10 days of treatment there was improvement in symptoms of heart failure but no significant correlation was found between the improvement of cardiac function and renal failure after 10 days treatment(51% patients has high blood urea>43mg/dL after 10 days&58% patients has high creatinine after 10 days although improvements in ejection fraction)(P.value >0.05).

Conclusion: Renal dysfunction is common in heart failure and has strong prognostic value. the classical treatment of heart failure dose not improve renal dysfunction duo to intrinsic renal disease, renal vascular disease , nephron loss (age, renal disease), inadequate renal perfusion, hypovolemia, inadequate cardiac output (vasoconstriction/pump failure), hypotension , abnormally high central venous pressure and by drug induced e.g.(ACEI, ARB). So patients were advice to take adenosine, A1receptor blocker, vasopressin antagonists, other intervention include earlier use of dialysis and ultrafiltration and left ventricular assist devices.

Keywords: Renal impairment, heart failure, ejection fraction, urea, creatinine.

Introduction:

Renal insufficiency (RI) is a common co-morbidity among patients with heart failure (HF) and confers excess mortality. Renal insufficiency is a major contributor to progressive cardiac damage, whereas HF is often associated with a rapid deterioration of renal function. (1) The coexistence of kidney and heart failure carries an extremely bad prognosis. The exact cause of deterioration of kidney function and the mechanism underlying this interaction are complex, multifactorial in nature, and still not completely understood. both the heart and the kidney act in tandem to regulate blood pressure, vascular tone, diuresis, natriuresis, etc. (2) Heart failure can lead to excessive and inappropriate activation of the renin-angiotensin system (RAS), which has been implicated in many ways in the progression of renal disease. (3)Despite growing recognition of the frequent presentation of combined cardiac and renal dysfunction, or "cardiorenal syndrome," its underlying pathophysiology is not well understood, and no Consensus as to its appropriate management has been achieved. Because patients with heart failure are surviving longer and dying less frequently from primary arrhythmia, we expect that the cardiorenal syndrome will become more common (4). In ambulatory heart failure patients, the presence of concomitant renal dysfunction consistently has been one of the strongest risk factors for mortality. This risk becomes evident even at serum creatinine clearance levels >1.3 mg/dL and estimated creatinine clearance values 60 to 70 mL/min. (5) Furthermore, renal function is at least as powerful an adverse prognostic factor as most clinical variables, including ejection fraction and New York Heart Association function class. Although renal dysfunction predicts all-cause mortality, it is most predictive of death from progressive heart failure, which suggests that it is a manifestation of and/or exacerbating factor for left ventricular dysfunction.(6).

Patients and methods:

A prospective study was done for 60 patients (35 male &25 female) mean age 65.5year with symptomatic heart failure admitted to the medical ward in AL-Nahrain College of Medicine in AL-Kadhmiya Teaching Hospital during the period from the June 2008 to the September 2009. Full

*Department Of Medicine, Medical College, AL-Nahrain University, Alkadhmiya Teaching Hospital.

clinical examination was done including blood pressure, change in pulse volume, rate, rhythm, raised jugular venous pressure, palpable liver, leg oedema, basal crepitation and ascitis. Heart failure was graded on day 0 according to New York Heart Association (NYHA) classification of heart failure.(7) Investigation was done in day 0 for evaluation of the heart failure include electrocardiography, chest X ray, echocardiography study (to evaluate the severity of ventricular dysfunction by measuring ejection fraction and fractional shortening and looking for any evidence of valvular lesion, segmental hypokinesia and feature of cardiomyopathy) Other investigation was done to determine renal function include general urine examination, blood urea ,creatinine and ultrasonic abdominal exam. Patients with hypertension and diabetes were enrolled only when they had normal test of renal function prior to the development heart failure. Patients who were excluded from this study include patients with prostatic hypertrophy, obstructive uropathy by (stone, stricture, masses or adhesion), bilateral small kidney, and patients with heart failure and other systemic disease or complication causing renal impairment from 0 day such as bleeding, dehydration, repeated vomiting and diarrhea. All the patients received antifailure measures including diuretic and vasodilator such as ACI inhibitor some of them receive anti-ischemia medication also. A 10 days period of follow up was done looking for changes in heart failure and renal function status by checking changes in grade of heart failure, echocardiography and blood urea and serum creatinine. All information which mentioned previously were taken on day 0 and on day 10.The relationship between cardiac and renal parameter were studied by statistical analysis using modified t- test, correlation coefficient ratio and regression analysis.

Results:

The survey included 60 consecutive patients hospitalized with a Presumptive diagnosis of heart failure depending on history, physical examination and investigation as mentioned previously the baseline characteristics of this study as in Table1.

Characteristics	percentages
Hypertension	70%
Ischemic heart disease	74%
Corpulmonal	22%
Valvular heart disease	18%
Cardiomyopathy	14%
Diabetes mellitus	46%
Previous stroke	10%
LV Ejection fraction ≥50%	28%
LV Ejection fraction 40---49%	25%
LV Ejection fraction 30---39%	27%
LV Ejection fraction <30%	18%

*LV= left ventricle

Descriptive statistic for all parameters, evaluated in the study including the blood urea, serum creatinine, ejection fraction, fractional shortening and grade of heart failure on both day 0 and day10 were applied in Table 2

parameter	Day 0			Day 10		
	mean	S.E	S.D	mean	S.E	S.D
Blood urea	64.17mg/dL	4.79	30.72	49.09mg/dL	3.72	23.85
S. creatinine	1.74 mg/dL	0.10	0.67	1.32 mg/dL	8.58	0.54
E.fraction	45.70%	2.37	15.21	48.78%	2.30	14.72
F. Shortening	21.78%	1.40	8.97	24.39%	1.35	8.65
Grade	3.12	0.12	0.81	2.19	0.13	0.87

S.E= standard error, S.D=standard deviation,
E.fraction=ejection fraction,
F. shortening = fractional shortening

Table 3 shows the difference in blood urea and creatinine in day 0&10

parameter	Range	%	S.D
Urea on day 0	26--80mg/dL		64.17±30.72
Urea >43mg/dL on day 0		75%	
Increase urea in 10 day		17%	
Same urea in 10 day		4%	
Urea >43mg/dL on 10 day		51%	
S. creatinine on day 0	0.7--4 mg/dL		1.7±0.67
S. creatinine >1.2 mg/dl on day 0		70%	
Progressive increase in creatinine		14%	
Same creatinine in 0&10 day		5%	
S.creatinine >1.2mg/dL on 10 day		58%	

Table 4 the correlations between the grad of heart failure, ejection fraction and blood urea &creatinine on day 0&10

parameters	P. value in day o	P. value in day 10
Blood urea & grade	0.002	0.589
S. creatinine & grade	0.017	0.093
Blood urea & ejection F.	0.019	0.111
S. creatinine & ejection F.	0.039	0.150
Blood urea &fractional S.	0.025	0.016
S.creatinine & fractional S.	0.012	0.042

Discussion:

Worsening renal function during treatment of acute decompensated heart failure (ADHF) often complicates the treatment course of heart failure. Furthermore, the development of worsening renal function is a strong independent predictor of long-term adverse outcomes. Sometimes referred to as "cardio-renal syndrome," the definition varies widely, and the overall understanding of pathogenesis is limited (8). The study showed that there is

significant correlation between blood urea on day 0 and the grade of heart failure, ejection fraction, and fractional shortining (P. <0.05). Also the study showed that there is significant correlation between serum creatinine on day 0 and the grade of heart failure, ejection fraction and fractional shorttining (P. <0.05).These changes explained by the Pathophysiologic link between heart failure and renal insufficiency duo to intrinsic renal disease, renal vascular disease (pre-renal, intrarenal), nephron loss (age, renal disease), inadequate renal perfusion, hypovolemia, inadequate cardiac output (vasoconstriction/pump failure), hypotension , abnormally high central venous pressure and by drug induced e.g.(ACEI, ARB). Other possible causes of renal dysfunction include neurohumoral changes e.g. arginine vasopressin, rennin-angiotesin aldestrone system and natriuretic peptides or metabolic (hypoxia inflammatory nephropathy)(9). There was no significant changes of renal parameters (blood urea &serum creatinine) and the changes of the cardiac parameters (grade, EJ, FS) after 10 days of treatment (P.value>0.05) may be duo to short period of treatment in the hospital and follow up or duo to homodynamic changes to heart failure which induce with time many structural changes affecting the kidney including thickening the basement membrane with increase permeability to protein, mesangeal expansion and glomerular sclerosis. In our study the prevalence of renal dysfunction in heart failure is high but is similar to other study such as in Tarantini" (Moderate-to-severe renal dysfunction was diagnosed in 59% of patients at hospital admission and 61% at discharge. These patients were older and had a higher prevalence of diabetes, anemia, history of hypertension, myocardial infarction and hospitalization for heart failure than those with normal or mildy impaired renal function) (10). In studies by Frederick" that reported prevalence, 29% of patients had moderate to severe renal impairment, and 63% had at least mild renal insufficiency in heart failure (11). Potentially promising pharmacological approaches include selective adenosine, A1 receptor blockers, which have a variety of effects on intrarenal hemodynamics and tubular function, and vasopressin antagonists, other interventions include the earlier use of dialysis and ultrafiltration and, ultimately, left ventricular assist devices to manage these patients effectively, at least in the short term.

Early use of vasodilators (nesiritide) shown to reduce filling pressures, improve symptoms and reduce diuretic needs in decompensated congestive heart failure (CHF). aggressive diuretic therapy can lead to diuretic resistance and worsening creatinine and glomerular filitration rate(GFR).

Conclusion:

Renal dysfunction is common in heart failure and has strong prognostic value . the classical treatment of heart failure dose not improve renal dysfunction duo to intrinsic renal disease, renal vascular disease , nephron loss (age, renal disease), inadequate renal perfusion, hypovolemia, inadequate cardiac output (vasoconstriction/pump failure), hypotension , abnormally high central venous pressure and by drug induced e.g.(ACEI, ARB). so patients were advice to take adenosine, A1receptor blocker, vasopressin antagonists, other intervention include earlier use of dialysis and ultrafiltration and left ventricular assist devices.

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