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Cardiorenal Syndrome: An Important Prognostic Factor in Patient With Congestive Heart Failure

Medical Science

KEYWORDS : Cardiorenal, GFR, Heart failure, CKD

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ABSTRACT

Cardiorenal syndrome is defined as concomitant dysfunction of the heart and the kidney in which acute or chronic dysfunction in one organ may result in acute or chronic dysfunction in another organ. Until recently renal insufficiency was not seen as a significant problem in patient with heart failure, largely because serum creatinine was the benchmark of renal function. Calculated GFR is much more precise indicator of renal disease.

In our article we will be stressing the point that while managing a case of heart failure we must keep a close watch on pre-existing renal failure for the optimum benefit of the patient.

INTRODUCTION: Cardiorenal syndrome is defined as concomitant dysfunction of the heart and the kidney in which acute or chronic dysfunction in one organ may result in acute or chronic dysfunction in another organ (1). Until recently renal insufficiency was not seen as a significant problem in patient with heart failure, largely because serum creatinine was the benchmark of renal function. Calculated GFR is much more precise indicator of renal disease. The prevalence of chronic kidney disease(CKD),defined as GFR less than 60 ml/min/1.73 m has ranged from 26-57% in clinical trials like CHARM,SOLVD,MERIT-HF and 35-56% in observational studies that have enrolled ambulatory HF patients like Go et al, de Silva et al, McAlister et al etc.

REVIEW OF TEXT: The prevalence of Chronic kidney disease (CKD) in acute decompensated heart failure (ADHF) at hospital admission had been studied extensively in the ADHF National registry (ADHERE) study done on >1 lakh patients. At admission only 9% had normal renal function (GFR >90 ml/min/m), 27.4% had mild renal dysfunction (GFR= 60-90 ml/min/m) 43.5% had moderate renal dysfunction (GFR = 30-59 ml/min/m), 13.1% had severe renal dysfunction (GFR= 15-29 ml/min/m) and 7% had kidney failure (GFR < 15ml/min/m). In hospital mortality was correlated with CKD and found increased from 1.9% for normal renal function to 7.6% for patient with severe renal dysfunction & kidney failure (2).

Worsening renal function (WRF) defined as an increase in serum creatinine level or a corresponding decrease in GFR occurs in approximately 25% of ADHF patients during hospitalization. The WRF was associated with increased mortality, prolonged hospital stay and higher percentage of readmission rates (3).

Prognosis of worsening renal function:

There is consistent and powerful evidence that renal dysfunction adversely affect prognosis in CHF. Hampton & colleagues (4) analyzed 1906 patients in the 2nd prospective randomized study of Ibopamine on mortality and efficacy by evaluating the mortality in four quartiles of the study group based on the estimated GFR. Mortality in the lowest renal function quartile had a survival of less than 50% in 2 years. In another outpatient study (5) blood urea nitrogen was the most important predictor in a cohort of >800 HF patient with reduced ejection fraction. Fonarow & colleagues (6) using data from over 60,000 admission created a mortality model and concluded that a BUN greater than 43 mg/dl was the most powerful predictor of inpatient mortality followed by low systolic blood pressure and serum creatinine.

Pathophysiology of cardiorenal syndrome:

The kidney receives 20% of cardiac output thus the function of the heart and kidney are closely intertwined. For example change in volume & pressure in the cardiac atria initiate atrial-renal reflex which increases dieresis through suppression of ADH arginine vasopressin via vagus nerve stimulation. In patient having heart failure with decrease in CO and fall in BP lead to activation of RAAS. Activation of RAAS have both positive and negative effect. Its activation leads to immediate acute hemodynamic improvement but the chronic activation leads to increased preload to heart, increased in cardiac afterload, increase in myocardial workload. An increased aldosterone level leads to extracellular potassium shift posing risk for arrhythmia and also causes endothelial dysfunction thus posing risk for thrombogenesis. These all can worsen the cardiac out come.

Evidence based facts related to the treatment of CRS:

Diuretics: Loop diuretics are very helpful in patient having acute pulmonary edema or systemic edema and provide immediate symptomatic relief and short term survival advantage also. But higher dose of loop diuretics are associated with higher serum creatinine and reduced survival (7). Patient taking loop diuretics also have increased risk of hospitalization and death related to CHF compared to those not taking these medication (8). Among potassium sparing diuretic spironolactone and eplerenone are currently the only diuretics which have been shown to reduce mortality in HF and this is probably not related to the agent's diuretic effect (9). Their utility in HF is to counteract potassium wasting and hypokalemic metabolic alkalosis associated with loop diuretics and thiazide and it also helps by reducing the raised aldosterone level associated with HF.

ACE Inhibitors & ARBs: In patient with both symptomatic and asymptomatic myocardial dysfunction long term administration of ACE-I reduces symptoms morbidity and mortality from HF associated with reduced ejection fraction (10). Use of ACEI & ARBs in HF can lead to decreased renal function initially but in the long term will lead to survival benefits. Decreased renal function in SOLVD study was defined as a rise in serum creatinine by >0.5 mg/dl. ACEI & ARBs may cause 10-20% rise in serum creatinine and this range is acceptable. RAAS blockade should be continued as mortality is very high when ACEI/ARBs are stopped because of renal concern and when these drugs have to be stopped, is a poor prognostic indicator (11).

Following facts decide the decrease in renal function:

- If ACEI/ARBs is used in person having low mean arterial pressure

- HF with volume depleted state
- B/L renal artery stenosis
- Stenosis of a dominant or a single kidney renal artery
- Extensive atherosclerotic disease in smaller preglomerular vessels
- Patient taking NSAIDs or COX-2 inhibitors
- Older age, concomitant diuretic therapy and Diabetic patients are more prone

Role of elevated central venous pressure:

Renal perfusion as in all organ is influenced by the pressure gradient across its vascular bed. Mullen in a study of severely ill HF patients found that CVP was the best predictor of the percentage who would go to develop renal insufficiency (14). Some researchers also have shown that reduction of intra abdominal pressure either by parancentesis when ascites is present or by ultrafiltration may at time result in improved renal function.

Role of ultrafiltration on diuretic resistance and Cardiorenal Syndrome:

Ultrafiltration as supportive care in patients with cardiorenal syndrome is emerging as a useful therapeutic strategy in both in elective and emergency situation (13).The UNLOAD Trial(ultrafiltration v/s IV diuretics for patients hospitalized for ADHF) has demonstrated that early treatment with ultrafiltration in patients with acute HF safely produces greater weight and fluid loss than intravenous diuretics with a 44% reduction in re hospitalization for heart failure in the following three months.

Conclusion:

When dealing with the management of HF renal function should be closely monitored and one should be very precise in selecting and continuing the drug which can lead to renal compromise and if yet Heart Failure is refractory to conventional treatment, ultrafiltration might be of great value.

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