Section: General Medicine

Original Research Article

CARDIORENAL SYNDROME IN PATIENTS WITH HEART FAILURE: A PROSPECTIVE OBSERVATIONAL STUDY AT A TERTIARY CARE HOSPITAL

Sandeep Tarapatla[1], Preema Dsa[2], Aditya Kumar Mishra[3], Santhosh Pai B H[4], Amal Shameer[5], Abdul Muizz Moosa Hamza[6]

[1]Associate Consultant, Jayam Hrudayalaya, Hospet, Karnataka, India
[2]Senior Resident, Yenepoya Medical College Hospital, Mangalore, Karnataka, India
[3]Assistant Professor, Yenepoya Medical College Hospital, Mangalore, Karnataka, India
[4]Professor and Head of the Department, Yenepoya Medical College Hospital, Mangalore, Karnataka, India
[5]Junior Resident, Yenepoya Medical College Hospital, Mangalore, Karnataka, India
[6]Junior Resident, Yenepoya Medical College Hospital, Mangalore, Karnataka, India

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Corresponding Author:
Dr. Preema Dsa,
Email: preemadsa3@gmail.com

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ABSTRACT

Background: This study was done with the aim of evaluating the clinical profile and outcomes of patients with cardiorenal syndrome, with special focus on morbidity and mortality among heart failure patients. **Materials and Methods:** This study was a prospective observational cross-sectional study which was conducted over a period of 1 year from June 1, 2013 to May 31, 2014 among patients of heart failure admitted at a tertiary care hospital. All consenting adult patients admitted with heart failure, with or without renal dysfunction, who met the inclusion criteria, were enrolled. Clinical parameters and outcomes, including duration of hospital stay and mortality in hospital were recorded according to the proforma. **Result:** Our study found a significant association between prolonged hospital stay in heart failure patients with cardio renal syndrome, compared to those who did not have renal dysfunction. These findings indicate that the presence of cardiorenal syndrome in patients admitted with heart failure is associated with substantially worse outcomes. Heart failure patients with concomitant renal dysfunction also experienced longer in-hospital mortality compared with those without renal impairment. **Conclusion:** This study highlights how kidney impairment is associated with poor outcomes in people with heart failure, emphasizing the importance of prompt detection and specific treatment plans for this vulnerable group.

INTRODUCTION

Heart failure (HF) remains a major global public health challenge and is among the leading causes of hospitalization worldwide1. Its prevalence rises sharply with age, affecting approximately 1% of individuals between 50 and 59 years and exceeding 10% in those older than 80 years.[1] Renal dysfunction is frequently observed in patients with HF and contributes substantially to adverse clinical outcomes and the complexity of management.[1]

The interplay between cardiac and renal impairment has become increasingly recognized as a defining component of HF pathophysiology. Cardiorenal syndrome (CRS) refers to a group of conditions in which dysfunction of the heart or kidneys, whether acute or chronic, directly contributes to injury of the other organ. Five subtypes of CRS have been described.[2] Type 1, the acute form, is the most frequently encountered and involves sudden deterioration in cardiac function leading to acute kidney injury (AKI). Newer renal biomarkers, including neutrophil gelatinase-associated lipocalin (NGAL) and cystatin C, offer the potential for earlier recognition of AKI compared with traditional creatinine-based measures, as they reflect both structural and functional renal changes.[3,4]

Type 2 CRS occurs when longstanding heart failure gradually impairs kidney function, affecting roughly a quarter of chronic HF patients.[5] Reduced forward flow and renal venous congestion both contribute to declining renal performance.[6] Type 3 CRS describes the reverse sequence—primary AKI triggering acute cardiac complications through mechanisms such as volume overload, electrolyte imbalance, and inflammation.[7]

Type 4 CRS highlights the impact of chronic kidney disease (CKD) on cardiovascular structure and

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function. CKD promotes left ventricular hypertrophy, diastolic abnormalities, and a markedly increased risk of cardiovascular events. Even modest reductions in glomerular filtration rate are associated with poorer cardiovascular outcomes. In addition, suboptimal risk-factor management in CKD contributes to the disproportionately high cardiovascular mortality observed in this population.[8]

Type 5 CRS describes concurrent cardiac and renal dysfunction triggered by systemic illnesses. Severe sepsis is the most common acute systemic condition affecting both organs simultaneously. Management of such patients requires a coordinated, multidisciplinary approach involving nephrology, cardiology, and critical care medicine.[9]

MATERIALS AND METHODS

This prospective observational study evaluated the prevalence, predictors, and short-term outcomes of cardiorenal syndrome (CRS) among patients with heart failure (HF). The study was conducted at Rajarajeshwari Medical College and hospital, Bengaluru, over a period of 1 year from 1st June 2013 to 31st May 2014. Out of 78 patients who were screened, 50 patients met the inclusion criteria and were enrolled in the study. The study was approved by the Scientific Review Board and the Institute Ethics Committee (IEC) of Rajarajeshwari Medical College and Hospital.

Inclusion Criteria

Adult patients (>18 years of age) admitted to the hospital with HF, with or without CRS, were included.

Exclusion Criteria

Patients meeting the following criteria were excluded:

- Chronic kidney disease,
- Renal artery stenosis
- Diabetic nephropathy with proteinuria >300 mg/24 h,
- History of chronic NSAID use,
- Serum creatinine >5 mg/dL,
- Hospital stay <24 hours, or
- Active infection at admission.

Data Collection

All patients who met the inclusion criteria were subjected to the following data collection:

1. **Demographic data:** age, sex, education level, socioeconomic status, and address.
2. **Clinical status:** A detailed analysis was conducted on presenting symptoms such as dyspnea on exertion (graded by NYHA class), leg swelling, abdominal bloating, chest pain, palpitations and fatigue. Additionally, comprehensive medical histories and family history were recorded.

A thorough physical assessment prioritized the cardiovascular, respiratory, digestive, and neurological systems. Heart failure was identified using ACC/AHA guidelines. Specific risk factors were categorized as follows: obesity as defined by Body Mass Index (BMI), dyslipidemia through high cholesterol levels, tobacco use by the smoking index, and alcohol abuse defined as daily consumption exceeding 100ml for at least 3 months.

3. **Biochemical investigations:** This study recorded glucose levels (at admission, fasting and post meal), serum urea and creatinine, with serial creatinine checks at 24 hours, 48 hours and discharge. The Cockcroft-Gault formula was used to determine creatinine clearance. Myocardial enzymes, thyroid function and fasting lipids were also included. Participants with proteinuria more 300gm in 24 hours or active urinary sediments were excluded from the study.
4. **Radiological investigations:** All patients underwent 2D and color Doppler echocardiography. Left ventricular ejection fraction (LVEF) was used to assess systolic function (systolic dysfunction defined as LVEF <50%). Diastolic function was graded (I–III) using Doppler indices.

All data were recorded in an organized proforma and then transcribed into an MS Excel worksheet.

Participants were categorized into two distinct sub groups: those with isolated heart failure and those presenting with heart failure complicated by cardiorenal syndrome. The CRS group was identified with the help of clinical markers which included serum creatinine more than 1.4mg/dl, a creatinine clearance below 60ml/min/1.73m2, or a minimum 25% increase in serum creatinine (> 0.3mg/dl).

Patient Follow-Up

All 50 patients were followed until hospital discharge and subsequently for a minimum of two months. Functional status, symptom progression, and in-hospital outcomes were documented. For patients who died, information was obtained from family members, with careful notation of the circumstances of death.

Statistical Analysis: Statistical data processing was conducted via IBM SPSS Statistics version 19. We evaluated the data distribution using the Kolmogorov – Smirnov test; normally distributed continuous variables are shown as mean ± SD, while non-normal data are reported as median (IQR). Categorical datasets were summarized by frequency and proportion. Group differences were analyzed using Pearson's Chi-square or Fisher exact tests. To isolate independent predictors of prolonged hospital stays, variables yielding a $p<0.10$ in univariate analysis were progressed to a multivariate logistic regression model. The threshold for statistical significance was established at $p<0.05$.

RESULTS

50 patients were included in the study out of which 64% (n=32) were males. Eighteen patients (36%) met criteria for heart failure with Cardiorenal syndrome

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(CRS) out of which 33.3% (n= 6) were females. Of the 32 patients with isolated heart failure, 62.5% (n=20) were male and 37.5% were female [Table 1].

Age distribution

The CRS group had a higher median age (58 years, range 18–72) than the HF-alone group (48 years, range 18–65), a statistically significant difference (p = 0.0014). CRS patients were predominantly aged 50–70 years, while the HF-alone cohort was younger (40–60 years).

Table 1: Sex distribution

Gender	HF Alone (n=32)	%	CRS (n=18)	%
Male	20	62.5	12	66.7
Female	12	37.5	6	33.3
Total	32	100	18	100

Etiology of heart failure: There were no significant disparities between the two groups regarding the primary causes of heart failure. Ischemic heart disease accounted for approximately half of the cases in each group, while rheumatic heart disease contributed to about one-quarter of the cases in both groups, as shown in [Figure 1].

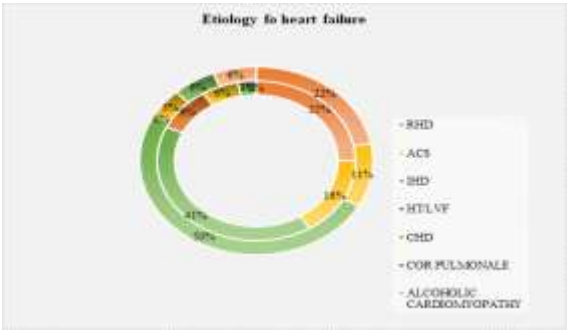

Figure 1: Etiology of heart Failure

Functional Status and BMI

In terms of functional capacity, 48 out of the 50 patients (96%) were identified as NYHA class III or IV with only two patients (4%) exhibiting class II symptoms. Body mass index was comparable between the two groups. The mean BMI was 22.97 kg/m² in the heart-failure-alone group and 23.33 kg/m² in the CRS group, with no statistically significant difference between them (p = 0.6896).

Analysis of risk factors

Statistical analysis identified diabetes mellitus (p = 0.0176), smoking (p=0.0352), and left ventricular diastolic dysfunction (p<0.0001) as significant correlates for the development of CRS within the heart failure population. Conversely, systemic hypertension, deranged lipid profile, alcohol consumption, and systolic left ventricular dysfunction (EF <50%) demonstrated no significant correlation with the study outcome [Table 2].

Table 2: Risk Factors Associated with CRS

Risk Factor	CRS (n=18)	HF Alone (n=32)	p value	Relative Risk
Diabetes Mellitus	13	11	0.0176	2.81
Hypertension	12	15	0.2410	NS
Dyslipidemia	11	18	0.7742	NS
Smoking	11	9	0.0352	2.36
Alcoholism	9	14	0.7709	NS
Systolic Dysfunction	11	14	0.3772	NS
Diastolic Dysfunction	17	12	<0.0001	12.31

NS = Not significant

Blood sugar levels, total cholesterol, and cardiothoracic ratio were comparable between patients with heart failure alone and those with CRS (p>0.05 for all). As shown in [Table 3], the mean ejection fraction was markedly lower in the CRS group than in the heart failure alone group (41.17 ± 10.08% vs. 50.81 ± 6.30%; p=0.0001), suggesting that cardiorenal involvement is linked to more advanced systolic dysfunction.

Table 3: Laboratory and Echocardiographic Parameters

Parameter	HF Alone (n=32)	CRS (n=18)	p value
Blood Sugar (mg/dL)	114.34 ± 47.58	104.77 ± 27.32	0.438
Total Cholesterol (mg/dL)	208.44 ± 45.67	211.22 ± 46.04	0.789
Cardiothoracic Ratio	0.559 ± 0.033	0.560 ± 0.035	0.904
Ejection Fraction (%)	50.81 ± 6.30	41.17 ± 10.08	0.0001*

*Significant difference

Serum creatinine and creatinine clearance

Mean admission serum creatinine levels differed significantly between groups (p < 0.0001), with 1.91 mg/dl (95% CI: 1.52-2.30), in the CRS group compared to 0.78 mg/dl (95% CI: 0.7-0.84) in those with heart alone. Similarly, creatinine clearance showed a statistically significant difference with a mean value of 100.81 ml/min/1.73m2 (95% CI: 90–111) in the heart failure alone group and 40.01 ml/min/1.73m2 (95% CI: 32.54–47.48) in the CRS group (p < 0.0001) as shown in [Table 4].

Morbidity and Mortality

The duration of functional improvement and duration of hospitalization were used for the evaluation of morbidity. Patients in the CRS cohort experienced significantly longer hospitalizations than those with

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isolated heart failure (11.06 vs. 6.78 days; $p < 0.001$). As illustrated in Figure 2, this mean difference of 3.28 days suggests a higher burden of morbidity associated with CRS.

Table 4: Serum Creatinine and Creatinine Clearance

Parameter	HF Alone (n=32)	CRS (n=18)	p value
Serum Creatinine (mg/dL)	0.78 ± 0.19	1.91 ± 0.78	<0.0001
Creatinine Clearance (mL/min/1.73 m²)	100.82 ± 28.52	40.01 ± 15.02	<0.0001

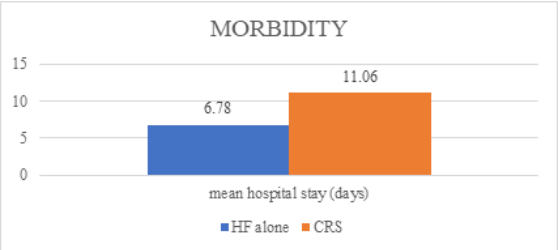

Figure 2: Mean hospital stay of the patients

In-hospital mortality rates were markedly elevated in the CRS cohort; 16.66% (n = 3) among patients with CRS compared to 6.25% (n = 2) in the isolated heart failure group.

Follow-up and Outcome: During the follow-up period, the mortality rate was significantly higher in the CRS cohort at 28% (5 of 18 patients), compared to 9.3% (3 of 32 patients) in the heart failure alone group. Clinical recovery was also poor in the CRS patients; only 16% attained complete recovery as compared to the heart failure alone group (60%). Only 54% of the patients attained partial recovery in the CRS group, as shown in [Figure 3].

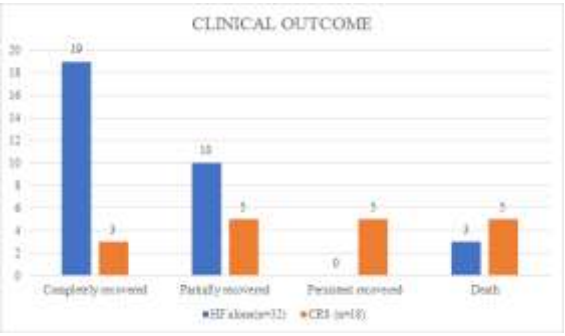

Figure 3: Clinical outcomes of the patients

DISCUSSION

In patients of heart failure; impaired renal function emerged as an independent predictor of poor clinical outcomes like prolonged hospitalization and increased mortality. Data from various meta-analyses demonstrate that mortality risk more than doubles in the presence of moderate renal insufficiency; characterized by an eGFR below 60ml/min/1.73m2 or a serum concentration of at least 1.5mg/dl.[10]

In this study conducted at Rajarajeshwari Medical College and Hospital, Bengaluru, 36% of the patients admitted with acute or chronic heart failure had cardiorenal syndrome. This finding of our study is in accordance with earlier studies, demonstrating the regular coexistence of renal and cardiac dysfunction in routine clinical practice. In our study; primary focus was on classical CRS types I and II, where cardiac dysfunction precipitates renal injury; recent literature emphasizes that the heart-kidney interaction is bidirectional, encompassing reno-cardiac syndromes as well.[11]

Multivariate regression analysis showed that high smoking index, diastolic dysfunction, prior history of two or more hospitalizations, prior CRS, diabetes mellitus and advancing age were found to be independent predictors of adverse outcomes. Isolated hypertension was not found to be a significant predictor of CRS which might be due to suboptimal recognition or under diagnosis of chronic non communicable diseases in the community setting. 90% of the patients had echocardiographic evidence of highlighting the pivotal role of venous congestion, elevated filling pressures and impaired renal perfusion in the pathophysiology of CRS.[12]

While baseline renal insufficiency is an established risk factor, the acute development of worsening renal function (WRF) during hospitalization serves as a more powerful independent predictor of adverse outcomes than admission values alone. These findings underscore the need of rigorous renal surveillance to identify high risk individuals. Future prospective randomized studies are required to determine whether early risk stratification and individualized therapeutic strategies can favorably modify outcomes in this high-risk population.

Therapeutic management of HF complicated by renal dysfunction remains challenging. Despite limited representation of patients with advanced renal impairment in major trials, renin–angiotensin–aldosterone system inhibitors (RAASi) continue to form the cornerstone of treatment in left ventricular systolic dysfunction. An initial rise in serum creatinine following RAASi initiation often reflects a reversible hemodynamic effect rather than intrinsic renal injury and should not automatically preclude continued therapy, provided renal function stabilizes and, hyperkalaemia does not occur.[13]

Overall, our findings reaffirm that detailed analysis of the cardiorenal dynamics across CRS subtypes is essential for improving diagnostic strategies, selecting appropriate therapies, and reducing the overall burden of cardiorenal complications.

Limitations of the study

A primary limitation of this study is its modest sample size, which reduces statistical power and potentially constrains the generalizability of our results. Furthermore, as the data was gathered in a tertiary care environment, the findings may

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specifically reflect a population with more advanced disease stages than those seen in primary care. This may have contributed to an overestimation of CRS prevalence.

Second, the absence of a uniform definition for worsening renal function (WRF) and CRS complicates direct comparison with other studies. Prior literature has variably defined WRF using absolute or relative changes in serum creatinine or eGFR, leading to significant heterogeneity in reported prevalence and outcomes.[14]

Third, treatment strategies were not standardized, as patients were managed by different physicians using individualized protocols. This therapeutic variability may have influenced outcomes and introduced residual confounding.

Finally, our analysis was restricted to CRS resulting from primary cardiac dysfunction (types I and II). Renocardiac syndromes, where renal disease precedes cardiac dysfunction, were not evaluated. Consequently, the overall burden of combined cardiorenal and renocardiac dysfunction may have been underestimated.

CONCLUSION

Cardiorenal Syndrome was observed in 36% of patients hospitalized with heart failure, highlighting a substantial overlap between cardiac and renal dysfunction. Advancing age and a history of two or more prior hospitalizations were strong predictors of CRS, with affected patients being approximately a decade older than those without CRS. Additional independent risk factors included diabetes mellitus, smoking, and left ventricular diastolic dysfunction.

The development of CRS extended the duration of hospital stay, delayed clinical recovery, increased readmissions, and nearly three-fold higher short-term mortality compared with HF patients without CRS. These findings suggest that CRS represents not merely a comorbidity but a marker of advanced, high-risk heart failure, potentially reflecting progression toward stage D disease.

Given the complex and bidirectional nature of heart–kidney interactions, optimal management of CRS likely requires a multidisciplinary approach involving cardiology, nephrology, and critical care. Further inquiry is essential to bridge the gap in our understanding of these mechanisms, ultimately paving the way for more individualized and evidence-based management strategies. Until such data are available, careful clinical judgment, judicious drug use, and close follow-up remain essential.

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