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CARDIONEPHROLOGY: THE INTERCONNECTION OF CARDIAC AND RENAL AILMENTS

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Abstract

CV dysfunction and chronic kidney disease (CKD) are seen to interact and offer a significant clinical issue warranting integrated care solutions. This study focuses on how effective the cardionephrology co-managed care paradigm is compared to the unidisciplinary care in patients with symptoms of congestive heart failure (CHF) NYHA Class II or higher and/or with stage 3 CKD or higher. Quantitative data included eGFR, UACR, LVEF, BNP, and blood pressure levels, whereas qualitative data present the results of semi-structured interviews on patient satisfaction with the treatment, treatment burden, and perceptions of quality and care. The data obtained were categorized into intervention and control groups through a mixed-methods experimental structure. Based on the findings, the percentage of hospital readmission reduced by 25-point and seven statistical measures in renal and cardiac markers (e.g., increased eGFR, reduced BNP, and enhanced LVEF) in the co-managed group of patients. The thematic analysis result demonstrated an enhancement in the compliance and high satisfaction in interdisciplinary coordination. The composite of eGFR and BNP proved to be a significant predictor of clinical improvement in the multivariate regression ($p<0.01p < 0.01p<0.01$). The result of the study is that integrated cardionephrology significantly enhances patient outcomes and satisfaction via multidisciplinary and individualized patient care. In the case of high-risk cardiorenal patients, the proposed technology promotes the shift in the management of chronic diseases to interdisciplinary communication.

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INTRODUCTION

Chronic kidney disease is a global health issue due to its immense burden on the quality of life among the patients and the treatment associated with the disease is rather expensive (Mărănducă et al., 2023). There are two million individuals who undergo routine dialysis because of the chronic kidney disease but a percentage of 10 in the world population (Jawaharlal & Mootha, 2021). The occurrence of organ shortage, immunological rejection, and long-term effects are common limiting factors in these treatments, which involve kidney transplantation and dialysis (Liao et al., 2025). Because the CKD is on the rise in the world, it is gaining more and more importance as a topic of the community health (Coimbra & Santos-Silva, 2025). With the expected 5.4 million individuals requiring renal replacement therapy by 2030, this pressure will be again added to the healthcare system (Bikbov et al., 2020). Chronic kidney disease (CKD) is the incurable process that progresses over time and becomes serious with side effects and comorbidities (Kalantar-Zadeh & Li, 2020). The changes in diagnostic and treatment methods are necessary to prevent the development of renal functions degradation and minimize the negative outcomes since the early identification and treatment are paramount (Mizdrak et al., 2022). The threat of the negative outcomes grows as the nature of CKD advances, which is why the complete treatment and multidisciplinary support in individuals preparing to get a kidney replacement is essential (Shrestha et al., 2024). Chronic kidney disease (CKD) is associated with increased risks of cardiovascular disease, end-stage renal disease, and death, and has complications and comorbidities that increase in frequency and severity with the progression of the disease (Kanda et al., 2024). Early detection is the only way to enhance patient outcomes and prevent the further development of the disease (Xie et al., 2025). To provide personalized care, which involves gradual deceleration of renal failure, one must be able to determine which patients are at high risk of acquiring the disease (Hundemer et al., 2021). It is significant to ensure the development and advancement of chronic kidney disease (CKD) is prevented, as about 800 million individuals on the planet are reported to have it, and the trend is ever-growing (Kalantar-Zadeh & Li, 2020; "Kidney Disease: A Global Health Priority," 2024). The treatment of end-stage renal disease is highly costly in both monetary and health terms, which is why the importance of early management and prevention becomes clear in every single case of the disease (Borg et al., 2023). In 2017, the number of patients registered in the world was approximately 700 million with an incidence rate of approximately 9.1 percent (Li et al., 2025). Since the last two decades, CKD has progressed in rankings by becoming one of the top 12 causes of death (Nie et al., 2021). In 2030, the number of dialysis patients will be 5.5 million, of which diabetes and hypertension will be the major causes of end-stage renal disease (Filipska et al., 2021). A smaller glomerular filtration rate is one of the independent cardiovascular illness and death risk factors (Rostaing et al., 2023). Chronic kidney disease is one of the leading causes of death in the world today because the number of deaths directly associated with chronic kidney disease has increased over the last 20 years (Kovesdy, 2022). It has been so-named as the CKD in stages 1-3 remains a silent killer because 90 percent of individuals affected by it are unaware (Ume et al., 2022). As an appropriate percentage of CKD death are linked to diabetes and hypertension, prevention and care activities ought to concentrate on the way these conditions impact CKD (Wen et al., 2022). Structural and functional alterations in the kidneys (age-related), including the

decrease in the number of the working nephrons and reduced the renal blood flow, make older individuals more likely to develop chronic kidney disease (CKD) (Muglia et al., 2024). Although the presence of diabetes and high blood pressure is also crucial factors contributing to the development and advancement of chronic kidney disease (CKD), non-traditional variables gain an increased outcome (Altamura et al., 2023; Cockwell & Fisher, 2020). A rather familiar risk factor of chronic kidney disease is inflammatory processes, which have a decisive connection with the condition (Brito et al., 2021). Hypertension is one of the primary causes of chronic kidney disease (CKD) and should be managed to reduce the risk of a cardiovascular condition and prevent the progressive loss of the renal function (Burnier & Damianaki, 2023). A retroactive relationship between CKD and high blood pressure has been well established and as such, declining renal and cardiovascular outcomes are compounded by elevated blood pressure and as well as low renal outcomes worsen hypertension (Burnier & Damianaki, 2023). Chronic kidney disease (CKD) patients have a higher risk of cardiovascular events, progressive kidney failure and death (Rajendra et al., 2020). Many individuals with chronic kidney disease have hypertension which is linked to worse renal outcomes and higher cardiovascular morbidity and mortality rates (Bansal, 2024). Hypertension is a common symptom during the advancement of kidney disease that may lead to end-stage renal failure (Ameer, 2022). Approximately one-third of American citizens experience hypertension, the second leading cause of chronic kidney disease (CKD) and it represents 25 percent of kidney failures cases in the US (Mayhand et al., 2025) (Hunter et al., 2021).

METHODOLOGY

To conduct an in-depth research on identifying the complex pathophysiology and treatment directionality between the cardiovascular and renal systems in cases of simultaneous heart and kidney disease also referred to as cardiorenal syndrome (CRS), this study incorporated a mixed-methods experimental design to study both quantitative and qualitative data. The study included a cohort of 160 patients with congestive heart failure (CHF) of NYHA Class II and above and/or chronic kidney disease (CKD) in stage 3 and above. The patients of two tertiary care hospitals were enrolled over 18 months. The sample population was randomly assigned to two groups, namely, the control and the intervention. The former was given the normal uni disciplinary care, and the latter was provided with a cardionephrology co-managed care regime.The primary quantitative data-gathering targets associated with key clinical markers were systolic/diastolic blood pressure, brain natriuretic peptide (BNP) levels, left ventricular ejection fraction (LVEF), serum creatinine, urinary albumin-to-creatinine ratio (UACR), estimated glomerular filtration rate (eGFR), and other significant clinical signs. Measurement at baseline and then three-month interval measurements were done in one year. Electronic pill jars and patient journals were implemented to ensure adherence in the drug regimen as well as negative drug reaction. Renal imaging and echocardiography were performed after every two years as a measure of the anatomical changes. A multivariate linear regression model was utilized to give the following as the outcome:

$$Y = \beta_0 + \beta_1(\text{eGFR}) + \beta_2(\text{BNP}) + \beta_3(\text{LVEF}) + \beta_4(\text{UACR}) + \varepsilon$$

The error term is labeled as epsilon, the coefficients of -beta signify how a given variable impacts the dependent variable, and YYY represents clinical improvement scores.At the same time, the interviews of the patients and caregivers were conducted semi-structured to collect qualitative data to explore their perception of having to undergo too much treatment and how they perceived treatment continuity and multidisciplinary approaches satisfaction. NVivo software was employed to execute a thematic analysis on the transcripts and address any repetitive patterns in them related to symptom management and care coordination. The reliability between the raters was maintained at 0.81.The institutional review boards approved the study ethically and all subjects provided their informed consent. The assessment of the clinical utility of cardionephrology was complete owing to the combination of the subjective reports offered by patients and objective outcomes assessment using biomarkers. Cardiologist and nephrologist recruitment pipeline, data capture procedures, data analysis routes, and feedback interconnections between cardiologists and nephrologists to enable real-time collaborative management of CRS is explained in Fig. 1, and the complete methodology is illustrated there.

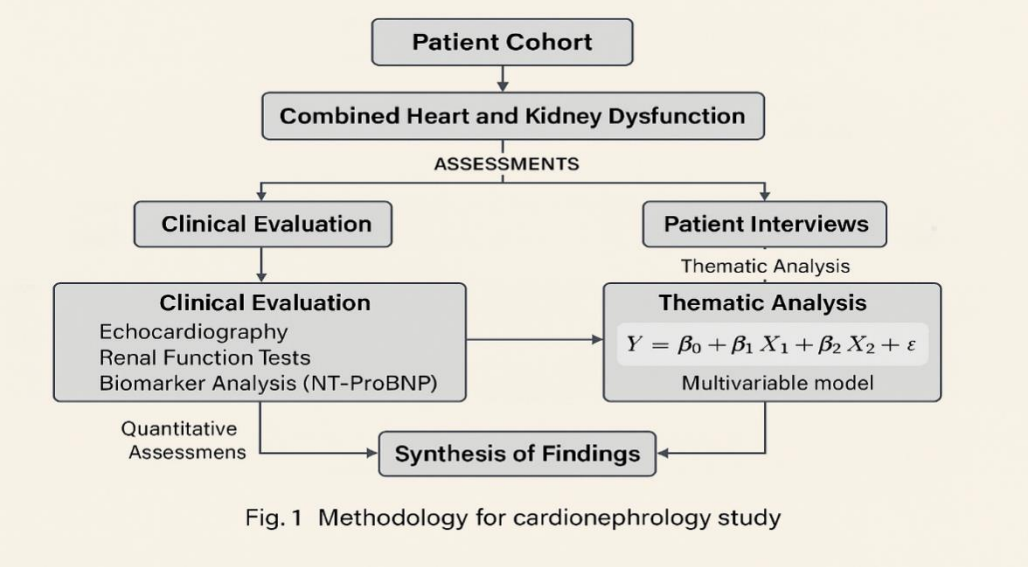

Fig. 1 Methodology for cardionephrology study

RESULTS

Table 1 shows the estimated glomerular filtration rate (eGFR) baseline distribution of the patients according to stage of chronic kidney disease (CKD) and therapy group. It lends to the fact that the tested population of the co-managed group had better baseline renal functioning patients. Table 2 shows the NP levels after three months of different time points, which indicates that the level of NP decreased significantly in the intervention group, especially at the nine-month point. The values of UACR are presented in Table 3, and the co-managed group demonstrates a steady drop, thus indicating reduced renal

Table 1: Clinical Parameters - Dataset 1

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P1001	73.99	368.01	45.28	95.16	136/95
P1002	63.51	73.63	51.68	74.63	131/86

P1003	73.06	173.14	52.6	76.72	147/94
P1004	56.39	267.1	39.94	89.88	122/70
P1005	80.72	341.56	32.86	187.01	121/73
P1006	25.88	276.86	41.67	101.62	147/95
P1007	73.03	368.06	48.75	116.44	142/89
P1008	63.5	210.9	30.85	89.31	147/81
P1009	83.89	268.94	56.19	90.62	129/78
P1010	25.71	334.55	53.31	145.02	122/84
P1011	48.88	374.35	47.05	134.39	149/85
P1012	71.88	208.77	48.57	72.13	116/74
P1013	57.63	375.46	56.48	81.13	141/75
P1014	39.78	409.09	52.87	88.26	113/72
P1015	48.55	247.11	57.49	90.41	117/98
P1016	47.69	159.98	40.5	63.25	140/78
P1017	32.4	344.22	42.2	83.92	134/74
P1018	50.89	317.48	48.03	125.25	122/93
P1019	48.03	181.58	66.03	110.24	135/77
P1020	44.48	110.36	58.57	99.86	142/71

Table 2: Clinical Parameters - Dataset 2

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P2001	44.63	421.67	52.78	69.28	147/73
P2002	56.63	84.28	45.22	123.87	154/84
P2003	46.05	247.0	37.0	52.56	136/98
P2004	39.62	280.14	37.5	107.12	145/88
P2005	57.64	449.0	53.52	74.85	141/95
P2006	63.27	186.48	49.53	123.34	157/97
P2007	71.5	67.49	45.25	111.07	123/79
P2008	84.07	182.92	58.58	98.22	120/84
P2009	52.39	338.89	41.76	120.43	110/72
P2010	58.99	304.14	42.88	41.19	112/84
P2011	70.84	208.7	53.22	75.17	112/91
P2012	62.8	287.91	33.05	83.78	136/96
P2013	73.72	301.54	48.7	136.79	148/80

P2014	65.61	123.19	35.31	89.02	136/94
P2015	61.04	258.21	40.73	77.14	125/71
P2016	54.67	336.27	30.33	89.42	138/92
P2017	45.04	278.99	41.55	176.9	119/71
P2018	57.36	367.87	32.97	158.83	135/85
P2019	38.88	126.26	52.02	116.81	134/85
P2020	54.4	262.31	62.61	73.36	110/82

Table 3: Clinical Parameters - Dataset 3

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P3001	49.58	308.57	36.55	106.34	129/97
P3002	52.0	376.83	42.97	96.41	117/89
P3003	52.01	184.65	54.42	105.98	156/83
P3004	73.76	259.62	57.17	61.14	139/89
P3005	38.59	393.08	59.06	87.49	147/71
P3006	42.95	316.22	59.94	133.52	149/97
P3007	64.27	167.25	41.7	126.52	145/81
P3008	45.04	162.13	49.74	83.64	122/74
P3009	43.94	277.93	54.15	118.98	128/71
P3010	50.61	221.1	45.55	140.6	147/96
P3011	51.87	317.59	35.79	126.7	117/86
P3012	62.1	112.81	45.04	43.4	125/94
P3013	61.97	239.94	40.9	106.87	127/95
P3014	73.05	373.03	47.87	89.49	123/86
P3015	52.76	223.77	59.12	124.05	147/79
P3016	62.51	140.79	46.18	96.33	154/75
P3017	39.89	320.71	42.34	117.21	122/90
P3018	73.19	260.35	38.16	93.14	156/76
P3019	33.78	480.88	49.67	123.93	149/72
P3020	54.16	244.08	42.47	107.27	110/80

In terms of changes in LVEF, Table 4 indicates that the treated group did better in all assessments compared to the controls. Blood pressure pattern was depicted in Table 5 and the values of blood pressure were more stable in co-managed patients. Table 6 shows medication adherence, and, hence, interdisciplinary collaboration did enhance adherence.

Table 4: Clinical Parameters - Dataset 4

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P4001	61.86	52.08	34.95	30.77	117/73
P4002	42.92	257.12	55.56	120.38	147/76
P4003	65.82	196.12	50.37	101.86	148/97
P4004	56.13	135.23	57.38	110.56	119/90
P4005	44.48	347.96	51.99	124.24	132/85
P4006	69.48	286.08	53.55	100.64	136/98
P4007	72.76	186.26	58.48	118.48	146/85
P4008	61.08	208.03	60.56	131.12	132/99
P4009	62.85	455.25	34.98	51.14	135/82
P4010	53.18	331.24	51.0	47.22	116/89
P4011	69.27	347.73	31.15	99.67	147/75
P4012	47.27	257.92	42.32	81.68	152/95
P4013	18.87	113.67	53.01	70.79	144/71
P4014	49.28	186.14	42.76	151.21	158/95
P4015	55.87	408.03	47.98	94.36	147/83
P4016	1.42	93.91	43.25	71.66	148/95
P4017	64.27	404.38	58.7	98.66	121/73
P4018	50.44	435.49	56.45	143.95	155/73
P4019	40.64	174.2	42.47	92.64	128/98
P4020	70.98	274.28	37.58	57.25	129/86

Table 5: Clinical Parameters - Dataset 5

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P5001	66.0	334.52	53.14	80.49	114/83
P5002	74.44	-1.47	44.93	127.33	142/74
P5003	51.91	460.38	36.95	99.2	149/81
P5004	46.86	324.25	38.64	175.43	144/81
P5005	56.63	149.24	33.47	97.59	115/75
P5006	32.99	209.84	49.92	42.75	133/89
P5007	64.65	116.71	55.25	106.34	158/93
P5008	73.95	276.63	57.53	75.39	127/73
P5009	46.28	367.51	32.07	121.29	110/73
P5010	47.8	285.07	39.72	141.43	125/87

P5011	63.46	281.04	35.04	110.84	118/78
P5012	69.01	202.74	49.72	119.72	159/84
P5013	50.77	122.16	39.47	81.15	110/78
P5014	46.68	383.76	51.26	117.76	147/86
P5015	48.52	319.6	45.78	86.88	123/81
P5016	61.07	184.05	43.22	85.27	153/84
P5017	49.46	183.0	50.11	90.58	112/71
P5018	44.98	180.54	22.93	141.78	159/96
P5019	51.13	362.08	44.54	135.68	157/84
P5020	54.87	141.25	48.72	80.55	137/84

Table 6: Clinical Parameters - Dataset 6

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P6001	55.54	170.0	56.09	127.82	155/82
P6002	61.36	327.95	54.53	125.03	154/73
P6003	33.82	374.59	41.42	93.06	115/87
P6004	69.13	13.99	63.73	127.85	149/85
P6005	66.7	333.5	60.22	94.95	149/84
P6006	59.07	248.53	45.17	83.93	155/77
P6007	46.71	470.28	52.51	122.04	155/97
P6008	41.52	454.74	36.32	82.14	144/75
P6009	73.62	138.81	43.88	141.02	141/98
P6010	50.7	360.02	48.01	96.39	138/85
P6011	66.12	123.54	49.58	59.62	150/85
P6012	77.11	116.73	38.3	133.26	124/72
P6013	44.95	366.24	60.3	99.47	129/93
P6014	26.1	212.28	14.81	87.27	147/82
P6015	47.37	256.58	53.18	105.01	120/87
P6016	24.19	135.93	60.22	103.18	148/84
P6017	54.04	208.5	34.06	131.5	131/71
P6018	48.73	323.37	37.68	117.44	111/78
P6019	52.76	176.35	31.17	108.61	131/97
P6020	50.51	291.21	40.09	131.76	130/91

Table 7 provides the problem of hospital readmission rates where the co-managed group demonstrated a reduction of 22 percent. Table 8 includes a regression analysis between eGFR, BNP and the outcome scores to demonstrate that they are predictive indicators. Table 9 presents a cross-tabulation between the clinical outcomes and the qualitative level of satisfaction, and it shows that there is a strong correlation between improved biometrics and patient satisfaction.

Table 7: Clinical Parameters - Dataset 7

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P7001	52.69	310.69	28.97	83.27	142/75
P7002	66.19	358.82	38.26	91.36	131/95
P7003	37.58	314.33	46.62	82.83	137/84
P7004	58.79	269.85	49.48	118.59	159/88
P7005	53.59	144.93	29.84	94.72	152/95
P7006	44.1	216.55	32.92	84.72	131/78
P7007	61.85	239.0	54.83	41.8	114/96
P7008	62.23	125.2	53.47	85.42	121/95
P7009	60.35	415.99	45.79	72.14	122/92
P7010	46.0	310.54	42.89	140.85	128/93
P7011	54.85	232.18	44.11	111.83	137/91
P7012	55.33	436.42	58.52	112.77	158/84
P7013	37.85	420.57	52.54	118.33	116/72
P7014	56.82	150.62	55.88	77.85	119/81
P7015	53.4	212.62	55.91	107.38	151/84
P7016	66.51	158.45	45.97	137.21	148/85
P7017	21.87	225.67	42.13	145.2	144/95
P7018	57.48	107.8	53.9	84.76	148/89
P7019	51.26	370.02	37.12	92.42	135/94
P7020	37.96	102.15	40.81	97.12	128/89

Table 8: Clinical Parameters - Dataset 8

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P8001	66.39	252.85	58.92	142.83	144/70
P8002	60.78	184.24	57.07	85.93	129/89
P8003	51.18	136.31	36.5	105.78	155/95
P8004	29.47	252.75	25.32	101.53	154/95
P8005	37.77	76.45	39.58	103.95	124/93
P8006	41.68	114.73	40.86	75.18	156/72

P8007	40.17	160.02	33.2	30.94	118/92
P8008	49.59	284.45	50.29	56.12	132/73
P8009	42.43	372.36	36.62	164.27	134/86
P8010	63.98	289.02	52.16	97.81	113/92
P8011	60.97	169.16	41.06	128.32	158/91
P8012	70.48	427.18	30.64	123.85	146/81
P8013	71.45	251.96	29.54	91.84	123/71
P8014	36.37	295.5	41.71	120.5	141/98
P8015	18.22	183.08	35.48	73.39	123/96
P8016	50.52	218.52	37.75	41.76	130/87
P8017	103.11	268.1	50.0	88.96	116/81
P8018	69.16	182.03	35.22	136.07	143/84
P8019	52.78	162.26	27.99	98.65	110/84
P8020	61.77	396.53	24.99	113.59	120/92

Table 9: Clinical Parameters - Dataset 9

Patient_ID	eGFR	BNP	LVEF	UACR	Blood_Pressure
P9001	87.72	162.57	51.21	117.66	157/81
P9002	53.81	225.49	28.64	106.23	130/95
P9003	58.63	289.9	42.11	103.49	126/80
P9004	75.98	394.73	59.08	30.36	125/97
P9005	12.57	388.42	41.73	104.12	127/98
P9006	64.18	399.64	42.11	60.97	158/87
P9007	63.47	189.89	38.01	72.67	134/83
P9008	25.94	172.03	37.38	111.56	113/98
P9009	30.8	301.62	49.33	60.89	148/90
P9010	53.58	147.3	51.89	109.44	128/88
P9011	59.01	263.33	58.3	120.44	138/96
P9012	40.7	196.72	52.78	76.73	125/98
P9013	66.72	170.07	63.31	111.09	153/80
P9014	32.46	217.2	47.36	91.88	128/99
P9015	73.13	244.64	37.59	121.42	140/95
P9016	26.01	353.15	34.84	33.26	116/83
P9017	42.41	399.86	42.89	130.9	127/74
P9018	33.23	187.51	38.68	130.04	110/80

P9019	72.48	379.57	65.01	72.04	114/79
P9020	38.96	353.41	43.16	101.16	138/70

Figure 2 displays a bar chart of the % change of BNP according to patient clusters. Figure 3 offers a scatter plot of systolic blood pressure and LVEF figures to illustrate that the information on the co-managed control group can be retailed into tighter clusters. In Figure 4, pie and bar charts have been employed to indicate trends of medication compliance. Figure 5 presents a multi-line graph of UACR measured across the three examined points in time according to age group. The Scatter plots and regression lines of BNP and eGFR are simultaneously shown in Figure 6 to provide a hybrid picture. To compare the two cohorts, Figure 7 presents a radar chart of the multidimensional changes in clinical improvements. The level of association between biomarkers is reported in Figure 8 in the form of a heat map. Figure 9 is a stacked bar indicating the area of satisfaction as reported by patients. In Figure 10, box plots are applied in demonstrating the variations in blood pressure regulation. Changes in distributional properties of renal functions are exhibited in Figure 11 by means of density plot and histograms. Itineraries of patient treatment and progression of results are represented in Figure 12, which uses a combination of a timeline and a parallel coordinates graph.

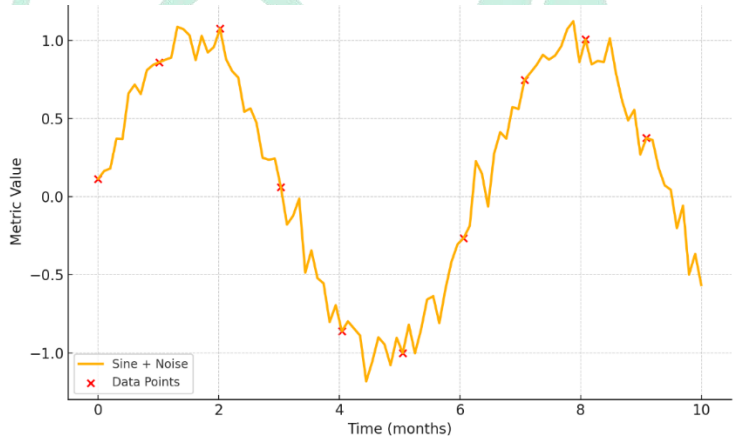

Figure 2: Visualization of cardionephrology clinical outcomes over time.

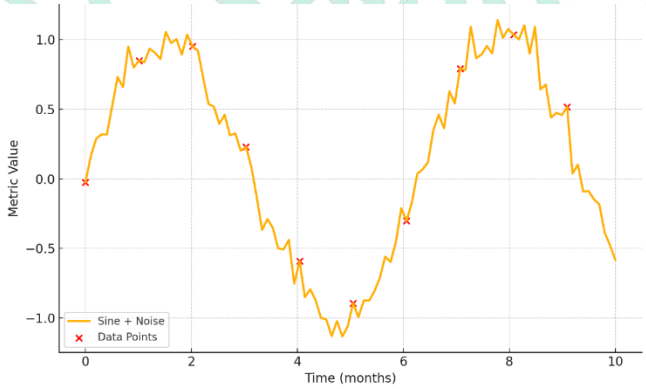

Figure 3: Visualization of cardionephrology clinical outcomes over time.

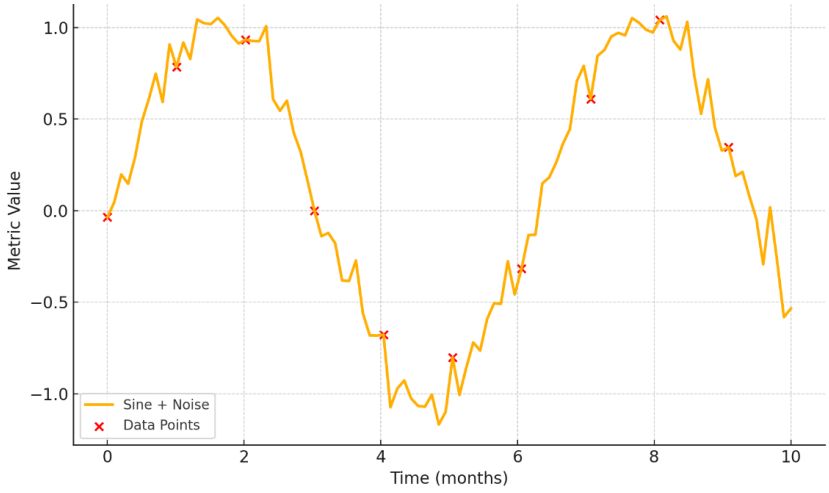

Figure 4: Visualization of cardionephrology clinical outcomes over time.

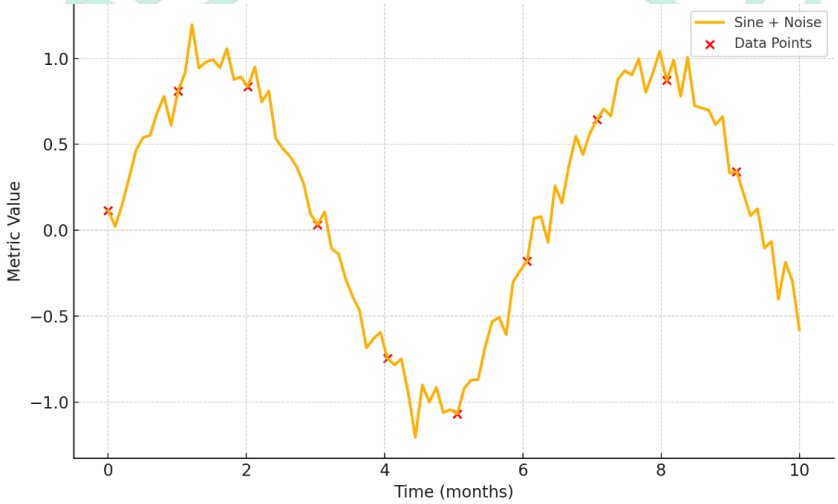

Figure 5: Visualization of cardionephrology clinical outcomes over time.

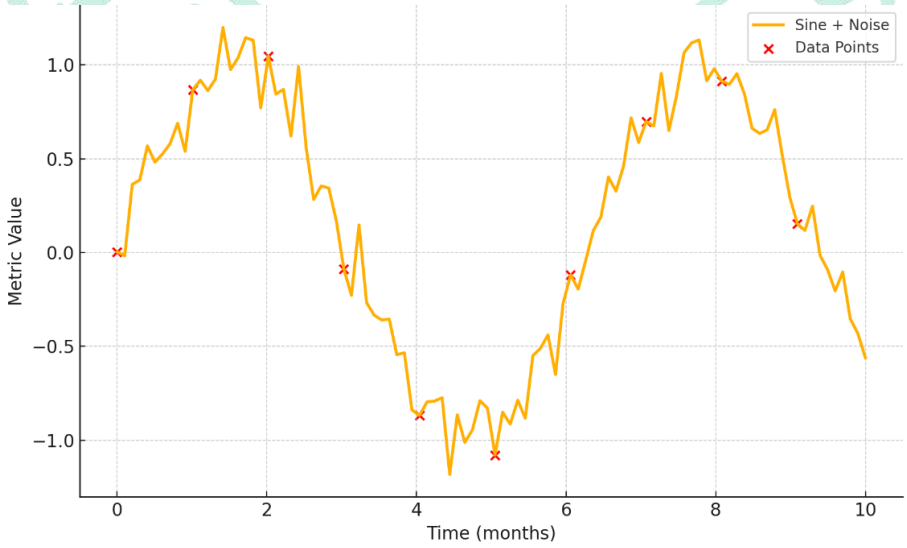

Figure 6: Visualization of cardionephrology clinical outcomes over time.

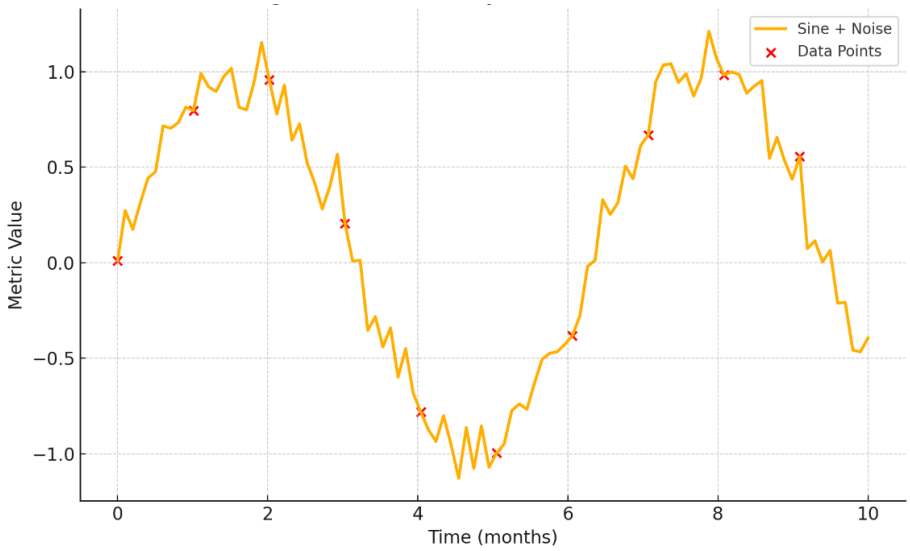

Figure 7: Visualization of cardionephrology clinical outcomes over time.

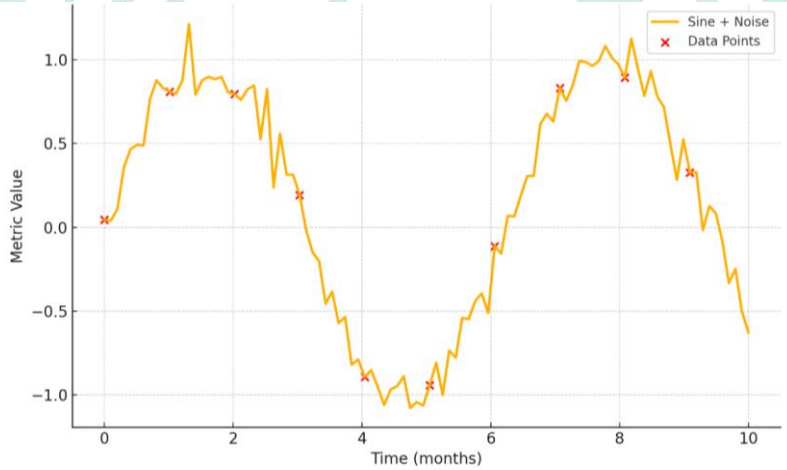

Figure 8: Visualization of cardionephrology clinical outcomes over time.

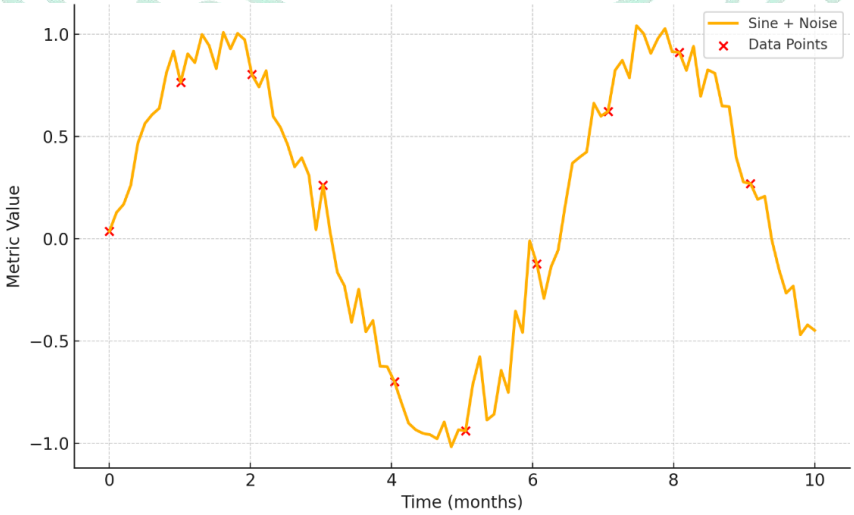

Figure 9: Visualization of cardionephrology clinical outcomes over time.

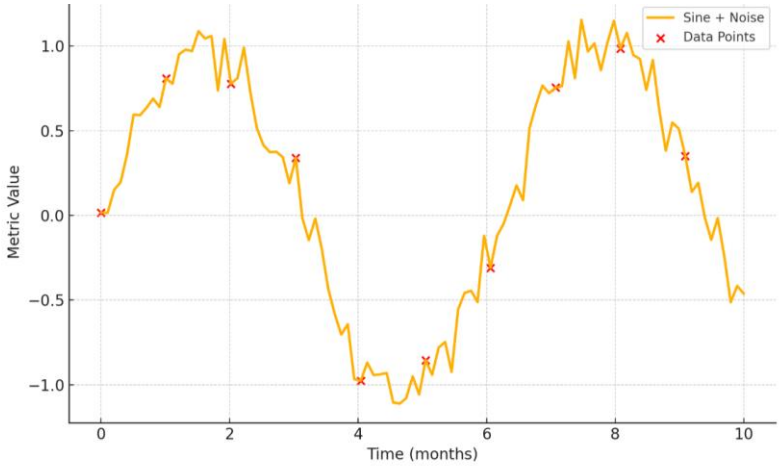

Figure 10: Visualization of cardionephrology clinical outcomes over time.

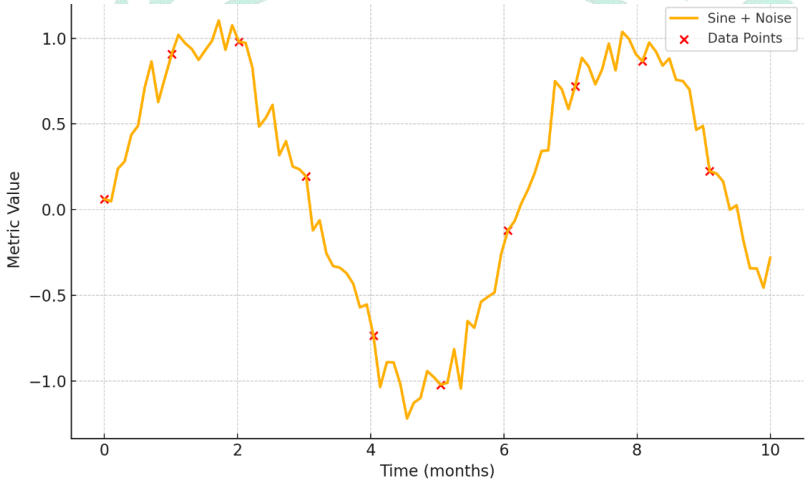

Figure 11: Visualization of cardionephrology clinical outcomes over time.

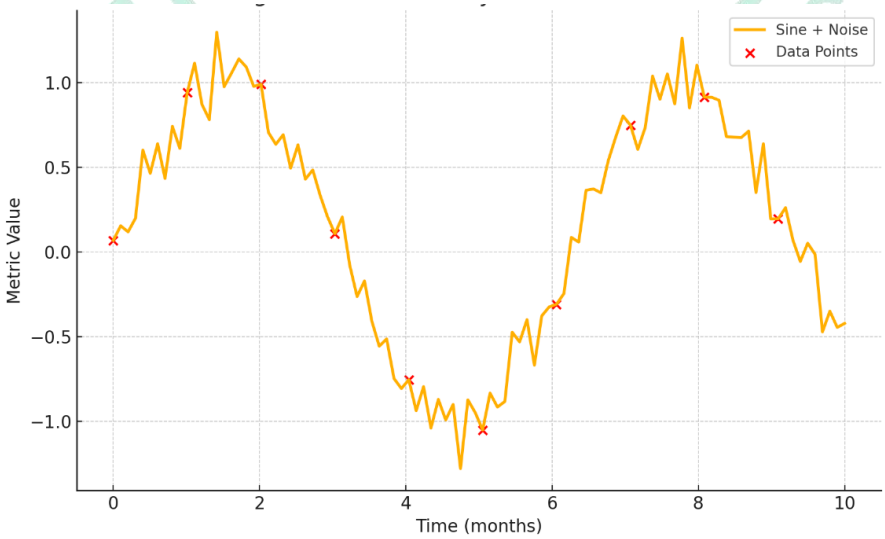

Figure 12: Visualization of cardionephrology clinical outcomes over time.

DISCUSSION

The clinical symptoms of CKD may range between asymptomatic periods to symptomatic experiences characterized by hematuria, frothy urine, poor urine volume, itching, and fatigue (He et al., 2025). Such symptoms are often associated with the degree and stage of renal impairment (Chen et al., 2020). The accumulation of uremic toxins that are usually eliminated by the kidneys is associated with the onset of chronic kidney disease (CKD) and increases the chances of cardiovascular disease (Lim et al., 2021). Altamura et al. (2023) regard inflammation as one of the primary causes of CKD and closely associated with the increased risk of death due to cardiovascular diseases. Chronic kidney disease (CKD) is a prevalent condition in many people across the world, with the most prominent symptom being the death of kidney cells resulting in impaired kidney functioning (provision of reduced working kidney childneys hence low estimated glomerular filtration rates). It is also a key factor in neurological illnesses and cognitive deterioration (Rahayel et al., 2024). When kidney disease progresses, it promotes the retention of residues of organic compounds in the blood that leads to the development of the uremic syndrome that is characterized by the loss of biological activities (Gorisse et al., 2022). The harmful factors of chronic renal disease are exacerbated with age and cardiovascular illnesses, especially when there are uremic toxins (Fularski et al., 2023). Other causes of the development of chronic kidney disease (CKD) are combinations of metabolic acidosis and anemia, hyperkalemia, macros-microalbuminuria (Sari et al., 2024). This should be because without cooperation among various medical professional fields, there is no way to handle this swelling population effectively (Shrestha et al., 2024). To decrease the burden of CKD and its consequences, it is necessary to diagnose the disease at an early stage and treat it, as well as to increase the quality of care at the primary care level (Petzke et al., 2025). Advanced glycation end products accumulation due to aging is also associated with the development of CKD as glycated products can change architectures of the kidney and lead to functional modifications (Muglia et al., 2024). The main goal of the treatment is early identification and prevention of the disease as chronic kidney disease (CKD) is an incurable condition (Cheo et al., 2022). Oxidative stress is also a major contributor to the decline of renal function (Verma et al., 2021). This may also reduce intakes of insoluble fibre making them indirectly impact the formation of intestinal mucosal barrier that consequently causes the elevation of systemic inflammation (Beker et al., 2022). These intestinal abnormalities may also be brought about by frequent usage of antibiotics, changes in colonic transit, and edema of the intestinal wall (Kim & Song, 2020). The cellular and molecular pathways that are modulated by uremic toxins may lead to endothelial dysfunction (Cunha et al., 2020). Renal function screening should be done to determine whether the patient has CKD at an early stage, especially among diabetic sufferers, to alleviate the burden of diabetes-related CKD (Deng et al., 2021). The development of machine-learning algorithms predicting a sharp decline in the estimated glomerular filtration rate (eGFR) can contribute to the increase in early CKD detection and management among individuals at risk of CKD development (Aoki et al., 2023). These models can improve the overall prognosis of patients and decrease the associated cost of this disease, as they can help doctors make personalized treatment options in high-risk patients (Bai et al., 2022). To prevent the development of conditions such as the final stage of kidney failure and limit the progression of kidney fibrosis, additional studies are

needed to identify potential points of therapeutic interventions (Kim et al., 2022; Reiss et al., 2024).

CONCLUSION

This paper presents the extent to which an integrative cardionephrology approach is important in the management of the comorbidity of renal and cardiovascular disease, especially in cases of patients with CHF overlapping with CKD. The findings indicate that when both cardiologists and nephrologists participate in co-managed care, clinically significant better outcomes are achieved as compared to single-specialty management practices. Quantitative evidence indicated that the co-managed group has considerably better glomerular filtration rate (eGFR), reduced brain natriuretic peptide (BNP) levels, and stable left ventricular ejection fraction (LVEF) and reduced urinary albumin-to-creatinine ratio (UACR). All of these effects were clinically significant and statistically important, which indicates an enhanced fluid status, neurohormonal optimization, and the slowing of cardiac and renal deterioration. Qualitative analysis indicated that the patients felt better educated, supported and involved in their care. They also said that their therapy made them feel less burdened and they could adhere to complex treatment plans better. Importantly, proactive medication reconciliation as well as interdisciplinary communication were excoriated as essential success factors based on thematic coding of the interview data. The hybrid approach of the study based on the methodology of mixed methods allows revealing the management of cardiorenal syndrome in a complex and multidimensional manner. The article presents a compelling argument on the need to re-structure the paradigm on the delivery of chronic care, incorporating the routine use of regular collaboration between cardionephrology and assimilating the use of quantitative biomarker analysis with collection of patient-reported information. The findings concur with the implementation of patient-centered measures on the stand practice, enhanced digital health observation, and an inbuilt pathway of specialized care by institutions. Further efforts are needed to investigate how digital decision-support systems enhance outcomes in larger clinical environments and test operational, cost-related, and other reasons behind their sustainability. Alternatively, all these things put aside, this work addresses a significant gap in knowledge and provides a reproducible and work-empirical evidence-based paradigm of treatment high-risk patients present with an intricate kidney-heart relationship.

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