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Evaluating the Level of Renal Dysfunction and its Predictors among Adult Cardiovascular Patients, Bahir Dar, Ethiopia

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Abstract

Background: Renal dysfunction has become a significant public health concern and often manifests as a serious complication in patients with cardiovascular disease. This study aimed to assess the prevalence and underlying causes of renal dysfunction. Methods: Comparative cross-sectional study was conducted on 302 adult cardiovascular disease patients and controls at Tibebe Gihon Comprehensive Specialized & Teaching Hospital in Bahir Dar, Ethiopia, from June to September 2024. Five milliliters of venous blood were collected for biochemical analysis, and 10 milliliters random urine were collected to determine albuminuria & analyzed by Dimension RxL 200 analyzer. Renal dysfunction was assessed using the estimated glomerular filtration rate by MDRD equation. Binary and multiple logistic regressions using 95% CI were used with $p < 0.05$ as significant. Results: Participants showed a lower estimated glomerular filtration rate in CVD (77.13 vs. 96.3 mL/min/1.73 m²). The overall prevalence of renal dysfunction was 20.2% and 31.3% for CVD patients. The overall prevalence of positive urinary albumin (UALB) was 39.4% and 61.3% for cardiovascular patients. Individuals with positive UALB results in CVD have an increased likelihood of developing renal dysfunction by about 6.07 times more than negative results. Conclusion: Renal dysfunction was high among cardiovascular disease patients.

Keywords: Renal dysfunction, chronic kidney disease, cardiovascular disease, glomerular filtration Rate

1. Introduction

Renal dysfunction (RD), commonly referred to as kidney dysfunction, is when the kidneys cannot perform their functions effectively. It can be characterized by acute kidney injury (AKI) or chronic kidney disease (CKD) (Chawla et al., 2017; Webster et al., 2017; Johansen et al., 2021). It affects millions of people worldwide. According to the Global Burden of Disease Study, CKD is ranked as the 12th leading cause of death globally, and the occurrence of cardiovascular conditions, are significant contributor to the high morbidity and mortality rates associated with this disease (Roth et al., 2017; Bikbov et al., 2020).

The global burden of kidney disease is on the rise, with prevalence rates ranging from 8% to 16%. Based on reports from the International Society of Nephrology (ISN) and the World Health Organization (WHO), CKD affects approximately 10% of the global population, with an estimated 850 million individuals living with the condition (29, 30). In 2019 alone, CKD was responsible for an estimated 3.1 million deaths,

primarily due to its strong association with CVD. The prevalence of abnormal renal function has increased by 33% from 1990 to 2017, with the most significant growth occurring in low- and middle-income countries (LMICs), where access to healthcare and early diagnosis programs is often limited (Liu et al., 2023; Francis et al., 2024; Dong et al., 2024). In Africa, the situation is alarming as the region confronts a growing burden of RD, particularly CKD, which ranges from 2% to 30%, with an overall average of 13.9% (Stanifer et al., 2014; Thomas et al., 2017; Dong et al., 2024).

Renal Disease is associated more pronouncedly among patients with cardiovascular disease (CVD), a condition that impacts the heart and blood vessels. Factors such as physical inactivity, poor dietary habits, and stress can exacerbate heart function, leading to increased stress on the kidneys, which can lead to diminished renal function (Hill et al., 2016; Crews et al., 2019; Bello et al., 2024). Therefore, disturbances in renal function tests of Albumin, Serum creatinine (SCr), blood urea nitrogen (BUN), and Uric acid (UA). Moreover, imbalances in electrolyte levels of sodium (Na+), potassium (K+), and chloride (Cl-) are regulated by the kidneys (Wasung et al., 2015; Lezaic, 2015; Sunariyanti et al., 2023; Gounden et al., 2024).

Prospective epidemiological data on the risk factors for CKD in patients with heart failure are limited globally. Most associations identified in cohort studies, and to a lesser extent in cross-sectional studies, suggest that age, ethnicity, education, baseline eGFR, body mass index, smoking, and hypertension are predictors of new-onset CKD in the general population. (Kazory & Elkayam, 2014; Tuegel & Bansal, 2017; Zoccali et al., 2012).

Therefore, this research is particularly significant in resource-limited settings such as Ethiopia, where healthcare access remains underdeveloped; thus, early detection and diagnosis could substantially improve patient outcomes. Consequently, the findings from this study may contribute to the formulation of customized healthcare policies and practices, ultimately enhancing the quality of life for patients with CVD. Furthermore, it encourages a deeper understanding of the issue and fosters collaboration among researchers, thereby enhancing the scope and impact of future studies on renal and cardiovascular health should be enhanced.

1.1 Aim: This study aimed to assess renal dysfunction and its predictors among adult cardiovascular patients visiting Bahir Dar University Hospital, Bahir Dar, Ethiopia.

1.1.1 Specific Objectives

1. To compare the mean of renal function tests among adult cardiovascular patients and non-cardiovascular controls.
2. To determine the prevalence of renal dysfunction among adult cardiovascular patients and controls.
3. To identify determinants of renal dysfunction in adult cardiovascular patients and non-cardiovascular controls.

2. Materials and Methods

Study Area and Setting

This study was conducted at Tibebe Gihon Comprehensive Specialized & Teaching Hospital (TGCSTH), Bahir Dar, Ethiopia. It serves as the capital city of the Amhara Regional State and is located approximately 565 km away from Addis Ababa (Kindu et al., 2020). The hospital acts as one of the two referral hospitals in the city, serving a population of more than five million residents (Tesfe et al., 2022).

Study Population and Eligibility Criteria

A hospital-based comparative cross-sectional study was conducted from July to September 2024. Cardiovascular disease patients and healthy controls were considered as the study population. Cardiovascular disease, aged 18 years and older, as cases and healthy individuals were recruited for the study. For the case groups and control, individuals with a history of pre-existing renal abnormalities before the onset of cardiovascular conditions, below one year, were excluded. Additionally, patients who had undergone renal transplantation and those unable to provide informed consent were excluded.

Sample Size and Sampling Techniques

A single population proportion formula was used to calculate the sample size needed to conduct this research by using a level of significance (α = 95%) and 23.9% prevalence from the previous study [23]Considering a 10% non-response rate, approximately 308 participants (154 for cases and 154 for controls) were included in the study.

Data Collection and Laboratory Methods

The study was conducted after the participants had given informed consent. Trained nurse professionals utilized a standardized questionnaire to gather all necessary data. Socio-demographic characteristics, behavioral characteristics, and Clinical characteristics were collected accordingly. Weights were measured using an analog scale on light clothing. Height was measured with a stadiometer, and blood pressure was measured using a manual aneroid sphygmomanometer.

Five milliliters of venous blood were collected from participants using a sterile disposable needle and then transferred into a properly labeled serum separator tube (SST). Then the samples were allowed to clot at room temperature for 30 minutes and centrifuged at 3,000 revolutions per minute for five minutes to separate the serum. Serum Cr, BUN, UA, Na+, K+, and Cl- were analyzed by Dimension RxL 200 analyzer (Siemens Healthcare Diagnostics, USA). Moreover, 10 ml of random urine samples were collected in a clean, dry container for urinary albumin determination. A urine dipstick test was done to determine albuminuria following the manufacturer's instructions. The eGFR was calculated using the Modification of Diet in Renal Disease (MDRD) equation and is utilized to classify participants as having RD.

Data and Laboratory Quality Control

Data control measures were implemented at every stage of the data collection process to ensure proper validation and verification. Completeness, accuracy, and consistency of data were evaluated by analyzing the presence of necessary information. The data were entered using SPSS version 27 statistical software. The exported data were assessed for missing values, cleaned, and verified for completeness. Quality control was conducted every 24 hours before sample analysis to monitor and maintain the precision and accuracy of the test, the functionality of the instruments, reagents, and the overall testing environment. Standard operating procedures (SOPs) for the testing parameters were strictly followed during the testing procedure and laboratory analysis.

Data Analysis and Interpretation

The statistical analysis was done using SPSS 27 software to evaluate the relationship between the factors and the outcome of interest. A non-parametric analysis, specifically the Mann-Whitney U test, was employed to assess their relationship. The model fit for the multivariable logistic regression was evaluated using the Hosmer and Lemeshow test (p-value > 0.05). Binary logistic regression was performed to identify potential candidate variables for multivariable logistic regression. Subsequently, multivariable logistic regression was executed to see the simultaneous relationship between the outcome variable and predictor variables while adjusting for potential confounding factors that could distort the true relationship between the primary predictors and the outcome. A p-value <0.05 for a 95% CI was considered statistically significant.

Ethics Statement

Ethical approval was obtained from the Ethical Review Committee of the School of Biomedical and Laboratory Sciences, College of Medicine and Health Sciences, University of Gondar. A permission letter was obtained from Tibebe Ghion Hospital, College of Medicine and Health Sciences, Bahir Dar University. The study was conducted in accordance with the World Medical Association's Declaration of Helsinki. The confidentiality of participants' records was strictly maintained. Additionally, informed consent was obtained from each study participant, and only voluntary participants were included in the study.

3. Results

Socio-Demographic and Behavioral Characteristics

A chi-square test analysis was done on 302 study participants for characterization of sociodemographic variables. From these variables major findings observed were 154(51.0%) males and 190 (62.9%) living in urban areas, 156 (51.7%) have low monthly income, 133 (44.0%) are educated College & above. For details, see (Table 1).

Table 1: Socio-demographic and Behavioral characteristics among CVD cases and control groups, Bahir Dar, Ethiopia (n=302).

Variables	**Category**	**Cases**	**Controls**	**Total**	*P*-value
		n (%)	**n (%)**	**n (%)**	
Gender	Male	75 (48.7%)	79 (51.3%)	154 (51.0%)	.732
	Female	75 (50.7%)	73 (49.3%)	148 (49.0%)	
Age	18 – 40	61 (47.3%)	68 (52.7%)	129 (42.7%)	.524
	41 – 59	58 (49.2%)	60 (50.8%)	118 (39.1%	
	60 and above	31 (56.4%)	24 (43.6%)	55 (18.2%)	
Residency	Urban	93 (48.9%)	97 (51.1%)	190 (62.9%)	.744
	Rural	57 (50.9%)	55 (49.1%)	112 (37.1%)	
Education level	Unable to read or write	31 (55.4%)	25 (44.6%)	56 (18.5%)	.655
	Elementary	25 (44.6%)	31 (55.4%)	56 (18.5%)	
	High school	30 (52.6%)	27 (47.4%)	57 (19.0%)	
	College& above	64 (48.1%)	69 (51.9%)	133 (44.0%)	
Income	Low	80 (51.3%)	76 (48.7%)	156 (51.7%)	.758
	Medium	25 (45.5%)	30 (54.5%)	55 (18.2%)	
	High	45 (49.5%)	46 (50.5%)	91 (30.1%)	
Occupation	Employed	38 (47.5%)	42 (52.5%)	80 (26.5%)	.856
	Merchant	32 (48.5%)	34 (51.5%)	66 (21.9%)	
	Farming	33 (54.1%)	28 (45.9%)	61 (20.2%)	
	Housewife	34 (52.3%)	31 (47.7%)	65 (21.5%)	
	Student/Others	13 (43.3%)	17 (56.7%)	30 (9.9%)	

Note: *Statistically significant, p-value

Clinical Characteristics of the Study Participants

A chi-square test analysis was done for clinical characteristics (variables) on 302 study participants. Based on the statistical analysis on these study participants, 55 (18.2%) had a previous history of kidney disease, 78 (25.8%) had elevated SBP, 96 (31.8%) had overweight, 132 (43.7%) used NSAIDs, of whom 79 (59.8%) were CVD patients. (*Table 2*).

Table 2: Clinical characteristics among CVD cases and controls attending Tibebe-Ghion Specialized Hospital, Bahir Dar, Ethiopia (n = 302).

Variables	Category	Cases	Controls	Total	p-value
		n (%)	n (%)	n (%)	
FHKD	Yes	28(51.9%)	26(48.1%)	54(17.9%)	.723
	No	122(49.2%)	126(50.8%)	248(82.1%)	
PHKD	Yes	29(52.7%)	26(47.3%)	55(18.2%)	.616
	No	121(49.0%)	126(51.0%)	247(81.8%)	
SBP	Elevated SBP	59(75.6%)	19(24.4%)	78(25.8%)	< .001*
	Normal SBP	91(40.6%)	133(57.6%)	224(74.2%)	
DBP	Elevated DBP	93(67.4%)	45(32.6%)	138(45.7%)	< .001*
	Normal DBP	57(34.8%)	107(65.2%)	164(54.3%)	
Smoking	Yes	16 (61.5%)	10 (38.5%)	26 (8.6%)	.205
	No	13(48.6%)	142 (51.4%)	276 (91.4%)	
Alcohol Use	Yes	3(55.7%)	27 (44.3%)	61 (20.2%)	.289
	No	116 (48.1%)	125 (51.9%)	241 (79.8%)	
NSAIDs use	Yes	79 (59.8%)	53 (40.2%)	132 (43.7%)	.002*
	NO	71 (41.8%)	99 (58.2%)	170 (56.3%)	
Diet	Homemade	96 (51.9%)	89 (48.1%)	185 (61.3%)	.058
	Animal product	35 (40.2%)	52 (59.8%)	87 (28.8%)	
	Processed food	19 (63.3%)	11 (36.7%)	30 (9.9%)	
CVD type	VD	17	----	17 (11.3%)	NA
	CAD	25	----	25 (16.7%)	
	Arrhythmia	21	----	21 (14.0%)	
	HTN	16	----	16 (10.7%)	
	HF	71	----	71 (47.3%)	
Medications	Not Starting	28	----	28(18.7%)	NA
	ACEIs	30	----	30(20.0%)	
	Diuretics	28	----	28(18.7%)	
	B-blockers	36	----	36(24.0%)	
	CCBs	11	----	11(7.3%)	
	MRAs	17 (100)	----	17 (11.3)	
BMI	Overweight	59(61.5%)	37(38.5%)	96(31.8%)	.005*
	Normal	91(44.2%)	115(55.8%)	206(68.2%)	

Note: * statistically significant, NA- Not applicable, HF-heart failure, ACEIs- angiotensin-converting enzyme inhibitors, CCBs-calcium channel blockers, MRAs-mineralocorticoid antagonists, BMI-Body mass index, FHKD-family history of kidney disease, PHKD-previous history of kidney disease, VD- vascular disease, CAD-coronary artery disease, CVD-cardiovascular disease, SBP-systolic blood pressure, DBP-diastolic blood pressure, NSAIDs-nonsteroidal anti-inflammatory drugs, CVD- cardiovascular disease.

Laboratory Parameters of Renal Function Tests

A chi-square test was conducted to assess the abnormal results of laboratory parameters between the two groups of study participants. The statistical analysis revealed that 119 (39.4%) were positive for UALB; from these, 76 (61.3%) were in CVD patients. Moreover, 104 (34.4%) had elevated levels of SCr; of them,

77 (74.0%) were for CVD. Of the total participants, 139 (46.0%) had elevated levels of BUN; of them, 83 (59.7%) were for CVD (*Table 3*).

Table 3: Comparison of Laboratory parameters of renal function test among CVD cases and controls attending Tibebe-Ghion Specialized Hospital, Bahir Dar, Ethiopia (n = 302).

Variables	**Category**	**Cases**	**Controls**	**Total**	**95% CI**
		n (%)	**n (%)**	**n (%)**	
UALB	Positive	73 (61.3%)	46 (38.7%)	119 (39.4%)	33.9-45.2
	Negative	77 (42.1%)	106 (57.9%)	183 (60.6%)	54.8-66.1
SCr	Elevated SCr	77 (74.0%)	27 (26.0%)	104 (34.4%)	29.1-40.1
	Normal SCr	73 (36.9%)	125 (63.1%)	198 (65.6%)	59.9-70.9
BUN	Elevated BUN	83 (59.7%)	56 (40.3%)	139 (46.0%)	40.3-51.8
	Normal BUN	67 (41.1%)	96 (58.9%)	163 (54.0%)	48.2-59.7
UA	Elevated UA	35 (53.0%)	31 (47.0%)	66 (21.9%)	17.3-26.9
	Normal UA	115 (48.7%)	121 (51.3%)	236 (78.1%)	73.1-82.7
Sodium	Lowered Na+	47 (35.9%)	84 (64.1%)	131 (43.4%)	37.7-49.2
	Normal Na+	103 (60.2%)	68 (39.8%)	171 (56.6%)	50.8-62.3
Potassium	Elevated K+	61 (62.2%)	37 (37.8%)	98 (32.5%)	27.2-38.0
	Normal K+	89 (43.6%)	115 (56.4%)	204 (67.5%)	62.0-72.
Chloride	Normal Cl-	69 (42.9%)	92 (57.1%)	161 (53.3%)	47.5-59.0
	Lowered Cl-	81 (57.4%)	60 (42.6%)	141 (46.7%)	41.0-52.5
eGFR	≥60 mL/min/1.73 m²	103 (42.7%)	138 (57.3%)	241 (79.8%)	74.8-84.2
	<60 mL/min/1.73 m²	47 (77.0%)	14 (23.0%)	61 (20.2%)	15.8-25.2

eGFR, estimated glomerular filtration rate; CI, confidence interval; SCr, serum creatinine; BUN, blood urea nitrogen; UA, uric acid; UALB, urinary dipstick albumin.

Comparison of the Mean Level of Renal Function Tests and Clinical Factors

An independent t-test was done on 302 study participants to evaluate the mean difference between continuous variables of the two groups. The statistical analysis output revealed that the mean SCr level was 1.3 ± 0.43 and 1.01 ± 0.21 for CVD patients and controls, respectively, and the mean sodium level was 131.93 ± 5.61 and 135.71 ± 5.8 for CVD patients and controls, respectively. Moreover, the mean eGFR value was 77.13 ± 29.17 and 96.3 ± 25.47 for CVD patients and controls, respectively (*Table 4*). The mean level of these parameters for CVD was significantly higher than control groups ($p<0.001$).

Table 4: Levels of laboratory parameters and clinical characteristics among CVD patients and control study participants (n=302).

Parameters	Total	Cases	Controls	p-value
	$\bar{X}$ ± SD	$\bar{X}$ ± SD	$\bar{X}$ ± SD	
Age	45.79 ± 12.9	46.7 ± 13.2	44.93 ± 12.6	.296
BMI	23.61 ± 3.04	23.9 ± 3.4	23.3 ± 2.6	< .001
eGFR	86.78 ± 28.96	77.13 ± 29.17	96.3 ± 25.47	< .001
SBP	126 ± 15.6	131.2 ± 16.4	121 ± 12.8	< .001
DBP	87.5 ± 11.17	91.6 ± 11.6	83.5 ± 9.2	< .001
SCr	1.15 ± 0.37	1.3 ± 0.43	1.01 ± 0.21	< .001
BUN	23.4 ± 10.76	26.92 ± 12.9	19.95 ± 6.44	< .001
UA	5.4 ± 1.7deletee	5.83(1.7)	5 ± 1.6	< .001
Sodium	133.8 ± 5.99	131.93 ± 5.61	135.71 ± 5.8	< .001
Potassium	4.5 ± 0.74	4.74 ± 0.74	4.32 ± 0.68	< .001
Chloride	97.2 ± 6.84	95.9 ± 6.54	98.6 ± 6.7	< .001

BMI- body mass index, eGFR -estimated glomerular filtration rate, SBP-systolic blood pressure, DBP-diastolic blood pressure, SCr-serum creatinine, BUN-blood urea nitrogen, UA-uric acid.

Magnitude and stage of renal dysfunction

A chi-square test was conducted to assess the magnitude of renal dysfunction between the two groups of study participants. Among the study participants, 161 (53.3%) had a mildly to severely decreased eGFR. The prevalence of renal dysfunction was 61 (20.2%), and of those, 47 (31.3%) were CVD patients (Table 5).

Table 5: Stages of renal dysfunction among CVD cases and controls attending Tibebe-Ghion specialized Hospital, Bahir Dar, Ethiopia, using MDRD-based eGFR (n=302).

eGFR Category (mL/min/1.73m2)		Description	MDRD-based RD		Total n (%)
			Cases n (%)	Controls n (%)	
RD stage	Stage 1(90	Normal or high eGFR	52 (34.7)	89 (58.6)	141 (46.7)
	Stage 2 (60-89)	Mildly decreased eGFR	51 (34.0)	49 (32.2)	100 (33.1)
	Stage 3 (30-59)	Moderately decreased eGFR	41 (27.3)	14 (9.2)	55 (18.2)
	Stage 4 (15-29)	Severely decreased eGFR	6 (4.0)	0 (0.0)	6 (2.0)
Total			150 (100)	152 (100)	302 (100)

eGFR, estimated glomerular filtration rate; MDRD, modification of diet in renal disease; RD, renal dysfunction.

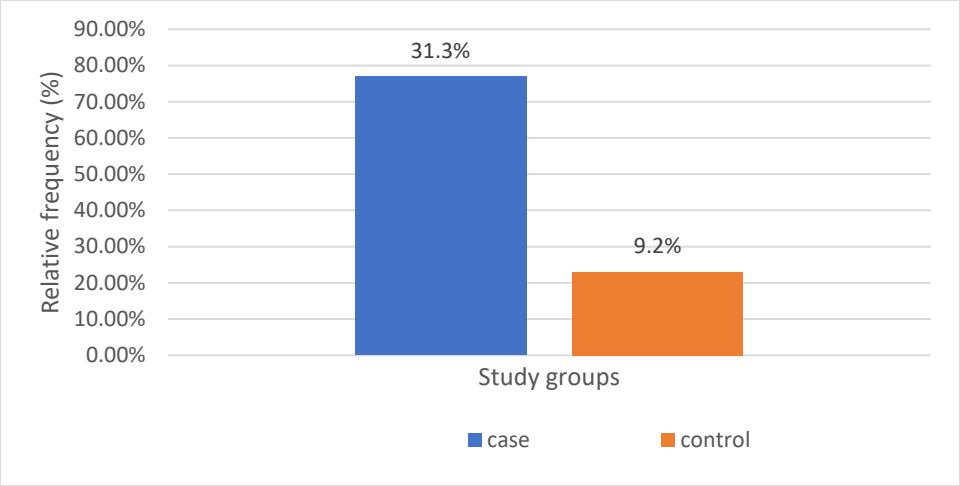

Fig. 1: Comparison of renal dysfunction among CVD cases and non-CVD controls attending Tibebe-Ghion Specialized Hospital, Bahir Dar, Ethiopia.

Factors Associated with Renal Dysfunction among the Study Participants

Binary and multiple logistic regression test was done to evaluate the impact of independent variables on the outcome variable using a reference variable. Based on the statistical result, cardiovascular disease (CVD) increased the presence of RD by 3.7 times compared to controls (CI: 1.343 – 10.416, p = 0.011). Urinary dipstick Albumin in CVD patients was 9.3 times more likely to develop RD than the control group (95% CI: 3.591 – 23.906, p < 0.001). The SBP in CVD was 8.7 times higher to develop RD than the control groups (95% CI: 2.393 – 31.648, p = 0.001). Older age in CVD patients increased the presence of RD by 7.9 times compared to controls (95% CI: 1.025 – 1.136, p = 0.004) (*Table 6*).

Table 6: Factors associated with renal disease among CVD patients and controls at Bahir Dar, Ethiopia (n=302).

Predictor Variables	Variable Category	RD		COR (95% CI)	AOR (95% CI)	p-value
		Yes	No			
Group	Cases	47	103	4.45 (2.35 – 8.608)	3.70 (1.343 – 10.41)	.011*
	Controls	14	138	1	1	
Education	College & above	15	118	1	1	
	Unable to read/write	23	33	5.48 (2.517 – 11.681)	1.43 (.160 – 12.761)	.750
	Elementary school	13	43	2.38(1.047 – 5.403)	.1.04 (.124 – 8.675)	.973
	High school	10	47	1.67 (.702 – 3.99)	.66 (.142 – 3.057)	.594
Occupation	Employed	10	70	1	1	
	Merchant	12	54	1.55 (.625 – 3.869)	.63 (.132 – 2.992)	.559
	Farmer	19	42	3.17 (1.345 – 7.453)	.49 (.046 – 5.146	.551
	Housewife	19	46	2.89 (1.234 – 6.773)	1.36 (.164 – 11.197)	.778
	Student	1	29	.24 (.020 – 1.973)	.24 (.001 – 92.094)	.637
PHKD	Yes	23	32	3.95 (2.089 – 7.479)	1.10 (.375 – 3.248)	.858
	No	38	209	1	1	
NSAIDs use	Yes	39	102	1.43 (.815 – 2.516)	.54 (.207 – 1.386)	.198
	No	22	139	1	1	
UALB	Positive	49	70	9.98 (5.004 – 19.884)	9.30 (3.591 – 23.90)	<.001*

	Negative	12	171	1	1	
Sodium	Lower Na+ level	49	122	3.98 (2.018 – 7.861)	1.84 (.465 – 7.269)	.385
	Normal Na+ level	12	119	1	1	
Potassium	Elevated K+ level	29	69	2.26 (1.271 – 4.014)	.93 (.305 – 2.833)	.898
	Normal K+ level	32	172	1	1	
Chloride	Lower Cl- level	41	100	2.89 (1.598 – 5.229)	.84 (.247 – 2.833)	.775
	Normal Cl- level	20	141	1	1	
SBP	Elevated SBP	36-	42-	6.82 (3.710 – 12.547)	8.70 (2.39 – 31.64)	.001*
	Normal SBP	25	199	1	1	
DBP	Elevated DBP	44	94	4.04 (2.185 – 7.499)	.34 (.093 – 1.244)	.103
	Normal DBP	17	147	1	1	
Age	≥40	45	128	5.24 (1.062 – 1.122)	7.90 (1.025 – 1.136)	.004*
	<40	16	113	1	1	

*-Statistically Significant, COR-crude odds ratio, AOR-adjusted odds ratio, UALB-urinary albumin (dipstick), HPKD-history of previous kidney disease, NSAIDs-nonsteroidal anti-inflammatory drugs, DBP-diastolic blood pressure, SBP-systolic blood pressure, BUN-blood urea nitrogen, UA-uric acid.

Factors Associated with Renal Dysfunction among Cardiovascular Disease Patients

Binary and multiple logistic regression test was done to evaluate the impact of independent variables on the outcome variable for CVD patients only. Based on the statistical analysis, positive UALB results in CVD increased the likelihood of developing RD by about 6.07 times more than negative results (95% CI: 2.02–18.29, p =.001). Abnormal SBP in CVD was 9.48 times higher to develop RD than normal results, and the presence of Arrhythmia was 13 times higher to develop RD in the absence of the disease.*Table 7*).

Table 7: Factors of renal dysfunction among cardiovascular disease patients (cases) (n=150).

Variables	**Category**	**RD**		**COR (95% CI)**	**AOR (95% CI)**	***p*-value**
		Yes	**No**			
UALB	Negative	9	68	1	1	
	Positive	38	35	8.20 (3.57-18.87)	6.08 (2.0-18.29)	.002*
Sex	Male	28	47	1.756 (.872-3.54)	2.52 (.65-10.09)	.201
	Female	19	56	1	1	
Residence	Rural	22	35	1.71 (.85-3.45)	.062 (.003-1.21)	.067
	Urban	25	68	1	1	
Education Level	No formal Education	17	14	4.34 (1.72-10.91)	10.29 (.144-25.6)	.155
	Elementary school	7	18	1.39 (.48-3.99)	4.9 (.26-9.21)	.288
	High school	9	21	1.53 (.57-4.08)	1.13 (.21-6.04)	.888
	College+	14	50	1	1	
Occupation	Employed	10	28	1	1	
	Merchant	9	23	1.096 (.38-3.15)	.524 (.10-2.8)	.451
	Farmer	15	18	2.33 (.86-6.31)	.1.36 (.11-16.5)	.808
	Housewife	12	22	1.53 (.56-4.18)	3.19 (.30-33.71)	.335
	Student/labor	1	12	.233 (.03-2.03)	.03 (.001-1.27)	.066
PHKD	Yes	16	13	3.57 (1.55-8.26)	1.32 (.35-5.03)	.991
	No	31	90	1	1	
Sodium	Lower Na+ level	39	64	2.97 (1.26-7.01)	3.66 (.68-19.66)	.130

	Normal Na+ level	8	39	1	1	
Potassium	Elevated K+ level	25	36	2.115 (1.05-4.26)	1.83 (.50-6.68)	.362
	Normal K+ level	22	67	1	1	
Chloride	Lowered Cl- level	31	50	2.054 (1.03-4.20)	.70 (.16-3.06)	.636
	Normal Cl- level	16	53	1	1	
CVD	VD	4	13	1.51 (.42-5.45)	.19 (.03-1.35)	.096
	CAD	13	12	5.32 (1.96-14.48)	4.01 (1.03-15.7)	.046*
	Arrhythmia	10	11	4.47 (1.56-12.87)	13.12 (2.51-68.60)	.001*
	HTN	8	8	4.91 (1.55-15.68)	2.11 (.33-13.5)	.429
	HF	12	59	1	1	
NSAIDs use	Yes	29	52	1.58 (.80-3.19)	.80 (.263-2.43)	.696
	No	18	51	1	1	
SBP	Elevated SBP	31	28	5.19 (2.47-10.91)	9.48 (2.30-39.70)	.002*
	Normal SBP	16	75	1	1	
DBP	Elevated DBP	39	54	4.42 (1.90-10.38)	1.46 (.38-5.59)	.557
	Normal DBP	8	49	1	1	

*Statistically significantly, OR-adjusted odds ratio, CI-confidence interval, CVD-cardiovascular disease, HF-heart failure-HTN, hypertension, NSAIDs-nonsteroidal anti-inflammatory disease, RD-renal dysfunction, UALB-urinary dipstick albuminuria, VD-vascular disease.

4. Discussion

Early detection and management of renal dysfunction in CVD patients allow for timely interventions that can slow the progression of RD and improve cardiovascular outcomes. Understanding these risk factors enables the development of comprehensive treatment plans that address cardiovascular and renal health, ultimately reducing morbidity and mortality and enhancing the quality of life.

According to this study, the mean value for an eGFR in CVD patients was 77.13 ± 29.17, indicating compromised kidney function in these patients compared to their controls, whose mean value was 96.3 ± 25. Additionally, Serum creatinine levels were significantly higher in CVD patients than in controls (1.3 ± 0.43 vs 1.01 ± 0.21). The mean blood Urea nitrogen levels were also higher among CVD patients compared to controls (26.92 ± 12.9 vs 19.95 ± 6.4), reflecting that cardiovascular disease is interconnected with renal dysfunction and poor outcomes. It indicates that CVD with poor cardiac output and venous congestion markedly affects renal perfusion, resulting in reduced renal blood flow with impaired waste filtration, leading to renal hypoperfusion and tubular congestion manifested by rising serum creatinine and BUN and low eGFR (Deferrari et al., 2021; Dundar et al., 2021). Inflammation and oxidative stress due to CVD can elevate UA levels due to impaired kidney excretion and increased purine breakdown. Oxidative stress from CVD produces free radicals that further damage blood vessels. Medications like diuretics used for CVD can also increase UA levels by limiting their excretion, leading to endothelial dysfunction and accelerating RD (Waring et al., 2000; Zoccali et al., 2023).

There was an electrolyte imbalance between the two groups; lower sodium levels were seen in CVD patients compared to controls (131.93 ± 5.61 vs 135.71 ± 5.8). Potassium levels were also higher in CVD patients compared to controls (4.74 ± 0.74 vs 4.32 ± 0.68), while chloride levels were lower in CVD patients compared to controls (95.9 ± 6.54 vs 98.6 ± 6.7). Impaired cardiovascular function leads to fluid retention, and activating the RAAS causes sodium and water retention, which results in diluting sodium and causing hyponatremia (Deferrari et al., 2021; Zhao et al., 2023). Additionally, increased antidiuretic hormone (ADH) further promotes water retention without increasing sodium, increasing the dilution. Hyperkalemia arises from impaired renal function, where the kidneys fail to excrete potassium properly, and from

medications like ACE inhibitors and potassium-sparing diuretics that increase potassium retention. Diuretics also contribute to hypochloremia by promoting chloride excretion and, in some cases, inducing metabolic alkalosis, which shifts chloride from the bloodstream into cells. These mechanisms result in significant alterations in the range of various electrolytes seen among CVD patients (Urso et al., 2015; Zandijk et al., 2021).

In the current study, the prevalence of RD was 20.2% among the total study participants, 31.3% among CVD patients, and 9.2% among controls. This overall prevalence (20.2%) was in line with studies conducted in Ethiopia, Addis Ababa-Ethiopia (23.9%), Israel (19.8%), and Spain (18.9%) (Amenós et al., 2010; Rozenbaum et al., 2016; Chala et al., 2019), The possible reason for this similarity in CKD prevalence across different regions listed above could be attributed to common risk factors such as hypertension, overweight, and aging populations for the occurrence of RD. These shared risk factors contribute significantly to the development of RD and may lead to comparable prevalence rates globally (Kazancioğlu, 2013). Contrary to this, the magnitude of RD in this study was lower than in the studies conducted in Egypt (31.5%) and China (34.1%) (Yang et al., 2010; Khamis et al., 2020). This disparity between the current study and those conducted in Egypt and China could be in population demographics, such as age distribution, genetic predisposition, and lifestyle factors that might contribute to varying RD rates. Furthermore, variations in diagnostic criteria, methods used to assess, and study design might be the reason for the observed disparities (Nicholas et al., 2022; Sawaf et al., 2023).

Moreover, the overall prevalence of RD in this study (20.2%) was higher than the studies conducted in the United Kingdom (3.8%) and the United Arab Emirates (11.4%) (Elsayed et al., 2007; Al-Shamsi et al., 2018). The observed significant difference in the reported prevalence rate can be attributed to advanced healthcare infrastructure in areas with lower prevalence rates. Early detection and diagnosis of renal abnormalities will significantly reduce the prevalence of RD. Additionally, differences in study design might be the possible cause of the observed differences. Our study uses a cross-sectional study design, which captures a snapshot of RD at a specific time, which may reflect acute or chronic cases. In contrast, a longitudinal study in the United Kingdom might track changes over time, potentially identifying fewer cases as it may focus on stable or less severe and chronic cases, leading to lower prevalence. A retrospective study design was used in a study conducted in the United Arab Emirates. This study design may lead to underreporting due to selection bias, as it relies on previously recorded data, which might not comprehensively capture all cases of RD.

Older age was a significant predictor 7.90 times (P=0.004) for the development of RD. This might be due to physiological changes, a decrease in nephron number and GFR in older ages, and the kidney becoming less efficient in filtering blood. In addition, an accumulation of comorbidities and increased inflammation through oxidative stress and endothelial dysfunction in older adults is also attributed to considering older age as a risk factor for RD (Carracedo et al., 2020; Li & Lindholm, 2023).

Elevated SBP also increases the risk factor of having kidney disease by 8.72 times (P=0.001) in CVD patients, which can lead to nephrosclerosis, characterized by vascular changes in the kidney that impair blood flow and filtration capacity. High blood pressure also damages endothelial cells in renal vasculature, leading to reduced nitric oxide availability and impaired vasodilation. This results in progressive loss of nephron function and lowers GFR, renal ischemia, and dysfunction (Carracedo et al., 2020; Deferrari et al., 2021).

The presence of positive UALB in this study increases the presence of kidney disease by 9.25 times (p=001) in CVD patients. It indicates that glomerular injury, or increased permeability, is often a precursor to RD. The leakage of albumin into urine reflects underlying nephron damage which reduces the kidney's filtering ability. It is also linked to systemic inflammation and oxidative stress, both of which can accelerate kidney injury (Carracedo et al., 2020; Deferrari et al., 2021).

Based on our study, the presence of CVD in certain individuals significantly increases the risk of developing RD by 3.691 times (p<0.011). This is due to interconnected mechanisms like the presence of

shared common risk factors, which can compound the effect on the development of RD. Alteration of hemodynamics, including reduced renal perfusion due to compromised cardiac output, neurohormonal activation through which RAAS is activated and causes vasoconstriction and sodium retention, lowering eGFR and further aggravating RD (Deferrari et al., 2021; Li & Lindholm, 2023).

However, alcohol users had a probability of developing RD 0.191 times (p=.027), which showed a protective effect for the progression of RD in the presence of CVD. This protective effect might be due to moderate alcohol consumption by the users, which has been associated with improving cardiovascular health by increasing high-density lipoprotein cholesterol and enhancing endothelial function, and alcohol reduces inflammation due to the presence of polyphenols and antioxidants in it (Lai et al., 2019; Yoon et al., 2020; Shao et al., 2024). Even though alcohol consumption has a protective effect on the development of RD in the presence of CVD, as in our study.

5. Conclusion

There was a significant difference in the mean levels of renal function test parameters between the CVD patients and the control group. The eGFR value was low, and Scr, BUN, and UA levels were high among CVD patients. There were lower sodium, chloride, and higher levels of potassium in patients with CVD. The overall magnitude of RD among the study participants was high (20.2%). The prevalence of RD was significantly higher among cardiovascular patients than in control groups. The presence of cardiovascular disease, positive urinary protein, elevated systolic blood pressure, and older age were predictors for the presence of RD. Scholars are needed to expand the study scope as longitudinal studies will be required to explore causal relationships between CVD and RD progression over time.

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7. References

Al-Shamsi, S., Regmi, D., & Govender, R. D. (2018). Chronic kidney disease in patients at high risk of cardiovascular disease in the United Arab Emirates: A population-based study. *PLOS ONE, 13*(6), e0199920.

Amenós, A. C., González-Juanatey, J. R., Gutiérrez, P. C., Gilarranz, A. M., & Costa, C. G. (2010). Prevalence of chronic kidney disease in patients with or at high risk of cardiovascular disease. *Revista Española de Cardiología, 63*(2), 225–228.

Bello, A. K., Okpechi, I. G., Levin, A., Ye, F., Damster, S., Arruebo, S., Donner, J.-A., Caskey, F. J., Cho, Y., & Davids, M. R. (2024). An update on global disparities in kidney disease burden and care across world regions. *The Lancet Global Health, 12*(3), e382–e395.

Bikbov, B., Purcell, C. A., Levey, A. S., Smith, M., Abdoli, A., Abebe, M., Adebayo, O. M., Afarideh, M., Agarwal, S. K., & Agudelo-Botero, M. (2020). Global, regional, and national burden of chronic kidney disease, 1990–2017. *The Lancet, 395*(10225), 709–733.

Carracedo, J., Alique, M., Vida, C., Bodega, G., Ceprián, N., Morales, E., et al. (2020). Mechanisms of cardiovascular disorders in patients with chronic kidney disease. *Frontiers in Cell and Developmental Biology, 8*, 185.

Chala, G., Sisay, T., & Teshome, Y. (2019). Chronic kidney disease and associated risk factors among cardiovascular patients. *International Journal of Nephrology and Renovascular Disease*, 205–211.

Chawla, L. S., Bellomo, R., Bihorac, A., Goldstein, S. L., Siew, E. D., Bagshaw, S. M., Bittleman, D., Cruz, D., Endre, Z., & Fitzgerald, R. L. (2017). Acute kidney disease and renal recovery: Consensus report. *Nature Reviews Nephrology, 13*(4), 241–257.

Crews, D. C., Bello, A. K., & Saadi, G. (2019). Burden, access, and disparities in kidney disease. *Brazilian Journal of Medical and Biological Research, 52*(3), e8338.

Deferrari, G., Cipriani, A., & La Porta, E. (2021). Renal dysfunction in cardiovascular diseases and its consequences. *Journal of Nephrology, 34*(1), 137–153.

Dong, J., Zhang, Z., Sun, J., Yang, X., & Zhang, W. (2024). Cardiovascular disease burden attributable to kidney dysfunction. *European Heart Journal – Quality of Care and Clinical Outcomes*, qcae088.

Dundar, Z. D., Kucukceran, K., & Ayranci, M. K. (2021). Blood urea nitrogen to albumin ratio as predictor of mortality. *The American Journal of Emergency Medicine, 46*, 349–354.

Elsayed, E. F., Tighiouart, H., Griffith, J., Kurth, T., Levey, A. S., Salem, D., et al. (2007). Cardiovascular disease and subsequent kidney disease. *Archives of Internal Medicine, 167*(11), 1130–1136.

Francis, A., Harhay, M. N., Ong, A., Tummalapalli, S. L., Ortiz, A., Fogo, A. B., Fliser, D., Roy-Chaudhury, P., Fontana, M., & Nangaku, M. (2024). Chronic kidney disease and global public health agenda. *Nature Reviews Nephrology*, 1–13.

Gounden, V., Bhatt, H., & Jialal, I. (2024). Renal function tests. In *StatPearls*. StatPearls Publishing.

Hill, N. R., Fatoba, S. T., Oke, J. L., Hirst, J. A., O'Callaghan, C. A., Lasserson, D. S., & Hobbs, F. R. (2016). Global prevalence of chronic kidney disease: A systematic review. *PLOS ONE, 11*(7), e0158765.

Johansen, K. L., Chertow, G. M., Foley, R. N., Gilbertson, D. T., Herzog, C. A., Ishani, A., Israni, A. K., Ku, E., Tamura, M. K., & Li, S. (2021). US renal data system annual data report. *American Journal of Kidney Diseases, 77*(4), A7–A8.

Kazancioğlu, R. (2013). Risk factors for chronic kidney disease: An update. *Kidney International Supplements, 3*(4), 368–371.

Kazory, A., & Elkayam, U. (2014). Cardiorenal interactions in acute heart failure. *Journal of Cardiac Failure, 20*(12), 1004–1011.

Kindu, M., Angelova, D., Schneider, T., Döllerer, M., Teketay, D., & Knoke, T. (2020). Monitoring urban growth patterns in Ethiopia. *ISPRS International Journal of Geo-Information, 9*(9), 548.

Lai, Y.-J., Chen, Y.-Y., Lin, Y.-K., Chen, C.-C., Yen, Y.-F., & Deng, C.-Y. (2019). Alcohol consumption and risk of chronic kidney disease. *Nutrients, 11*(9), 2121.

Lezaic, V. (2015). Albuminuria as a biomarker of renal disease. In V. Patel & V. R. Preedy (Eds.), *Biomarkers in kidney disease* (pp. 1–18). Springer.

Li, X., & Lindholm, B. (2023). Cardiovascular risk prediction in chronic kidney disease. *American Journal of Nephrology, 53*(10), 730–739.

Liu, W., Zhou, L., Yin, W., Wang, J., & Zuo, X. (2023). Burden of CKD attributable to high sodium intake. *Frontiers in Nutrition, 10*, 1078371.

Nicholas, S. B., Ford, C. L., & Norris, K. C. (2022). Race and genetic risk for kidney failure. *Cell Reports Medicine, 3*(8).

Rozenbaum, Z., Leader, A., Neuman, Y., Shlezinger, M., Goldenberg, I., Mosseri, M., et al. (2016). Unrecognized renal dysfunction in acute coronary syndrome. *The American Journal of Medicine, 129*(2), 187–194.

Shao, L., Chen, Y., Zhao, Z., & Luo, S. (2024). Alcohol consumption and mortality risks. *Medicine, 103*(27), e38857.

Stanifer, J. W., Jing, B., Tolan, S., Helmke, N., Mukerjee, R., Naicker, S., & Patel, U. (2014). CKD epidemiology in sub-Saharan Africa. *The Lancet Global Health, 2*(3), e174–e181.

Sunariyanti, E., Andayani, T. M., Endarti, D., & Puspandari, D. A. (2023). Cost analysis of CKD patients. *ClinicoEconomics and Outcomes Research*, 621–629.

Thomas, B., Matsushita, K., Abate, K. H., Al-Aly, Z., Ärnlöv, J., Asayama, K., Atkins, R., Badawi, A., Ballew, S. H., & Banerjee, A. (2017). Cardiovascular and renal outcomes of reduced GFR. *Journal of the American Society of Nephrology, 28*(7), 2167–2179.

Urso, C., Brucculeri, S., & Caimi, G. (2015). Acid–base and electrolyte abnormalities in heart failure. *Heart Failure Reviews, 20*, 493–503.

Waring, W., Webb, D., & Maxwell, S. (2000). Uric acid as a cardiovascular risk factor. *QJM, 93*(11), 707–713.

Webster, A. C., Nagler, E. V., Morton, R. L., & Masson, P. (2017). Chronic kidney disease. *The Lancet, 389*(10075), 1238–1252.

Yang, J.-G., Li, J., Lu, C., Hasimu, B., Yang, Y., & Hu, D. (2010). CKD and mortality among cardiovascular patients. *Journal of Atherosclerosis and Thrombosis, 17*(4), 395–401.

Yoon, S.-J., Jung, J.-G., Lee, S., Kim, J.-S., Ahn, S.-K., Shin, E.-S., et al. (2020). Alcohol consumption and cardiovascular disease. *BMC Public Health, 20*, 1–9.

Zandijk, A. J., van Norel, M. R., Julius, F. E., Sepehrvand, N., Pannu, N., McAlister, F. A., Voors, A. A., & Ezekowitz, J. A. (2021). Chloride in heart failure. *ESC Heart Failure, 9*(12), 904–915.

Zhao, K., Zheng, Q., Zhou, J., Zhang, Q., Gao, X., Liu, Y., Li, S., Shan, W., Liu, L., & Guo, N. (2023). Serum electrolyte and heart failure outcomes. *Annals of Medicine, 55*(1), 155–167.

Zoccali, C., Leonardis, D., Enia, G., Postorino, M., D'Arrigo, G., Tripepi, G., & Mallamaci, F. (2012). Risk of kidney failure progression. *Journal of Nephrology, 25*(Suppl 19), 20–27.

Zoccali, C., Mallamaci, F., Adamczak, M., de Oliveira, R. B., Massy, Z. A., Sarafidis, P., Agarwal, R., Mark, P. B., Kotanko, P., & Ferro, C. J. (2023). Cardiovascular complications in CKD. *Cardiovascular Research, 119*(11), 2017–2032.