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Carotid Intima-Media Thickness as a Cardiovascular Risk Factor in Patients with Chronic Kidney Disease

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ABSTRACT

Background: Chronic kidney disease (CKD) is a well-established risk factor for cardiovascular disease (CVD), with increased mortality due to accelerated atherosclerosis. Carotid intima-media thickness (CIMT) is a reliable marker of subclinical atherosclerosis and cardiovascular risk.

Objective: This study aimed to investigate the effect of CKD on CIMT and its correlation with CVD risk factors.

Patients and methods: Over a six-month period from January to June 2022, 200 participants aged 30 to 60 years were enrolled in a case-control study at Ain Shams University Hospitals. The case group comprised 150 CKD patients with varying degrees of renal impairment (eGFR ranging from 17 to 88 mL/min), while the control group included 50 healthy subjects with eGFR > 90 mL/min. CIMT was measured using duplex ultrasonography, and both clinical characteristics and laboratory findings were analyzed.

Results: CKD patients had significantly increased CIMT compared to controls (1.10 ± 0.16 mm vs. 0.49 ± 0.15 mm, P < 0.001). CIMT increased with CKD severity, being highest in patients with eGFR 15–29 mL/min (1.30 ± 0.08 mm). CIMT positively correlated with age (r = 0.377, P < 0.001), systolic blood pressure (SBP) (r = 0.311, P < 0.001), creatinine (r = 0.868, P < 0.001), and total cholesterol (TC) (r = 0.387, P < 0.001), while it negatively correlated with eGFR (r = -0.960, P < 0.001). Diabetic and hypertensive CKD patients had significantly higher CIMT than non-diabetic and normotensive counterparts (P = 0.001, P = 0.005, respectively).

Conclusion: CIMT is significantly increased in CKD patients, correlating with declining renal function and traditional cardiovascular risk factors.

Keywords: Chronic kidney disease, Carotid intima-media thickness, Cardiovascular risk, Atherosclerosis, Ultrasonography.

INTRODUCTION

Atherosclerosis is a well-established contributor to adverse cardiovascular outcomes, including myocardial infarction (MI), ischemic strokes and transient ischemic attacks (TIAs). It is a widespread arterial pathology that commonly involves multiple vascular beds. From an etiological perspective, atherosclerotic involvement across various arterial beds often stems from a common cluster of traditional risk factors. As a result, patients with atherosclerotic involvement in one vascular bed are often found to have concurrent disease in additional arterial regions. Accordingly, the presence of atherosclerosis within one arterial compartment may serve as an indicator of broader systemic vascular involvement [1].

Furthermore, carotid artery intima-media thickness (IMT) has emerged as a reliable surrogate marker for MI risk, with substantial evidence indicating a significant correlation between increased IMT and underlying coronary artery disease (CAD) [2].

The notion that carotid artery atherosclerosis signifies overall atherosclerotic disease is well-supported. Autopsy studies and carotid ultrasonography have revealed a significant association with coronary artery atherosclerosis. Interestingly, advancing carotid artery stenosis (CAS) has been identified as a more potent predictor of imminent MI than of subsequent stroke events [3].

There is a well-documented association between chronic kidney disease (CKD) and an increased incidence of major cardiovascular disease (CVD) complications. CVD-related mortality in patients with end-stage renal disease (ESRD) undergoing dialysis is estimated to be 10 to 30 times higher than that of the general population. Moreover, individuals with even mild to moderate renal impairment face a substantially increased risk of cardiovascular events. Emerging evidence from prospective, population-level research suggests that mild-to-moderate renal dysfunction is associated with an increased risk of cardiovascular events and death [4]. In patients with CKD, the burden of cardiovascular mortality outweighs the risk of transitioning to end-stage renal failure [5].

This study aimed to investigate the effect of CKD on CIMT, considering its role as a surrogate marker for cardiovascular risk in this patient population.

PATIENTS AND METHODS

Design and population:

This case-control investigation was carried out at Ain Shams University Hospitals over the period from January to June 2022, involving 200 participants aged 30–60 years, categorized into two study arms. The case group included 150 patients with varying degrees of renal impairment who were not on dialysis. Their eGFR ranged from 17 to 88 mL/min, with a mean age of 46.58 ± 8.67 years, and 49.3% were male. Based on their eGFR levels, this group was stratified into three distinct subgroups: Subgroup 1 (eGFR 60–89 mL/min) included 19 patients, subgroup 2 (eGFR 30–59 mL/min) included

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109 patients, and subgroup 3 (eGFR 15–29 mL/min) included 22 patients. The control group included 50 subjects with eGFR values above 90 mL/min, indicating normal renal function, the mean age was 44.08 ± 9.13 years, and males represented 46% of the group.

Exclusion criteria: Patients were excluded if they had a history of cardiovascular disease, ischemic heart disease (including previous percutaneous coronary intervention, coronary artery bypass grafting, or myocardial infarction) and cerebrovascular disease, or kidney transplantation.

Clinical and laboratory assessments: Informed consent was secured from all subjects, followed by a detailed clinical history taking that encompassed demographic data, smoking status, duration and presence of hypertension (HTN) and diabetes mellitus (DM), type of antidiabetic therapy, and previous diagnosis of dyslipidemia. A standardized physical examination protocol was applied, involving the measurement of heart rate (HR), blood pressure (BP), body height and weight, and subsequent computation of body mass index (BMI). Laboratory investigations included hemoglobin levels, kidney function tests (serum urea and creatinine), eGFR calculation using the Cockcroft-Gault formula [6], and a full lipid profile (total cholesterol (TC), triglycerides (TGs), high-density lipoprotein [HDL], and low-density lipoprotein [LDL]).

Carotid ultrasonography and IMT measurement: Carotid artery ultrasonography was conducted using Siemens ACUSON X700 system, utilizing a high-frequency linear transducer operating within the 4.0–12.0 MHz range. Bilateral scanning was conducted for the extracranial common carotid artery (CCA), carotid bulb, and internal carotid artery (ICA). For optimal imaging, patients were positioned in either the overhead position, where the examiner sat beyond the patient's head and used both hands for scanning, or the lateral sitting position, where the examiner used the right hand for both carotid arteries. The overhead position was preferred for Doppler ultrasonography due to its wider sonic window and clearer visualization, particularly from the posterolateral projection. Patients were positioned with their heads tilted approximately 45° away from the examined artery, ensuring neck relaxation to minimize muscle contractions that could interfere with imaging [7].

Ultrasound imaging methodology: Carotid ultrasonography was conducted in two planes: The short-axis view, useful for detecting vascular lesions, and the long-axis view. To compensate for potential limitations in a single viewing direction, short-axis scanning was performed from both anterior and lateral (posterior) projections. The study covered the CCA, carotid bulb, and ICA bilaterally to evaluate IMT and the presence of plaques.

Measurement of CIMT: The intima-media complex (IMC) consists of two layers: A hyperechoic layer adjacent to the vascular lumen and a hypoechoic layer beneath it. The maximum IMT was measured bilaterally in the CCA, carotid bulb, and ICA, excluding the external carotid artery (ECA). Due to ultrasound imaging limitations, anterior wall measurements were sometimes challenging, necessitating reliance on posterior wall (far wall) measurements. The smallest measurable IMT unit was 0.1 mm, with images magnified to reduce measurement errors. Both short-axis and long-axis views were used for IMT assessment.

Mean IMT was calculated from measurements taken at multiple points along the right and left CCAs, excluding the carotid bulb. Reference IMT values based on age categories were 0.59 mm, 0.67 mm, and 0.70 mm in women and 0.62 mm, 0.72 mm, and 0.80 mm in men for the age groups 30–39, 40–49, and 50–59 years, respectively [8].

CASES

Representative cases from the study illustrate the variations in CIMT among different patient groups.

- **Case 1:** A 50-year-old male patient from subgroup 2 of the case group (eGFR = 40 mL/min) had a CIMT measurement of 1.0 mm (Figure 2).
- **Case 2:** A 59-year-old female patient from subgroup 3 of the case group (eGFR = 20 mL/min) exhibited a CIMT of 1.3 mm, demonstrating further vascular thickening with declining renal function (Figure 3).
- **Case 3:** A 35-year-old male patient from the control group (eGFR = 100 mL/min) had a CIMT of 0.5 mm, which is significantly lower than those observed in CKD patients (Figure 4).

Additionally, figure (1) represented a longitudinal ultrasound image of the distal CCA, demonstrating the imaging technique used for CIMT assessment.

Ethical considerations: Ethical approval for the study was granted by the Research Ethics Committee of Ain Shams University. Prior to participation, all subjects provided written informed consent, which included explicit permission for inclusion in the study and publication of anonymized data. Confidentiality and privacy were strictly maintained. This study adhered to the ethical principles outlined in the Declaration of Helsinki.

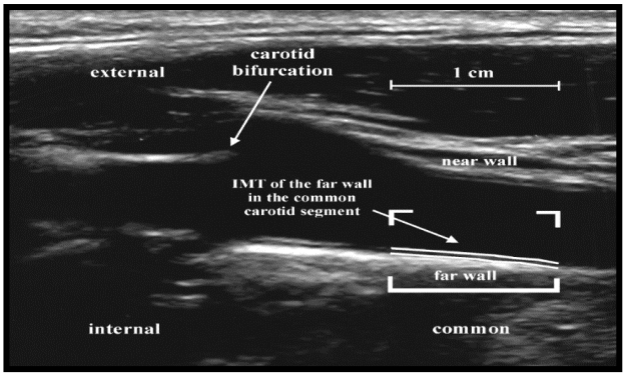

Figure (1): Longitudinal ultrasound image of the distal common carotid artery [9].

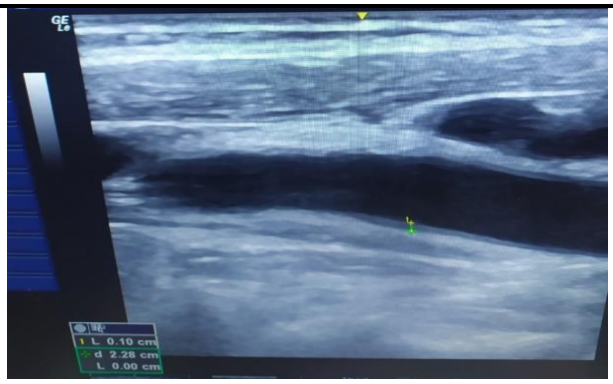

Figure (2): Case No. 20 from subgroup 2 of the case group. A 50-year-old male patient with an eGFR of 40 mL/min and a CIMT of 1 mm.

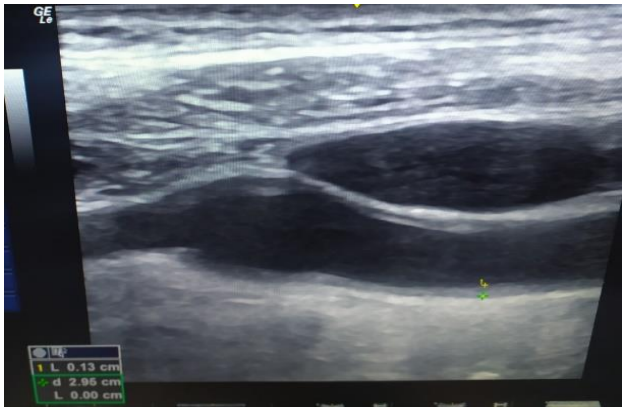

Figure (3): Case No. 10 from subgroup 3 of the case group. A 59-year-old female patient with an eGFR of 20 mL/min and a CIMT of 1.3 mm.

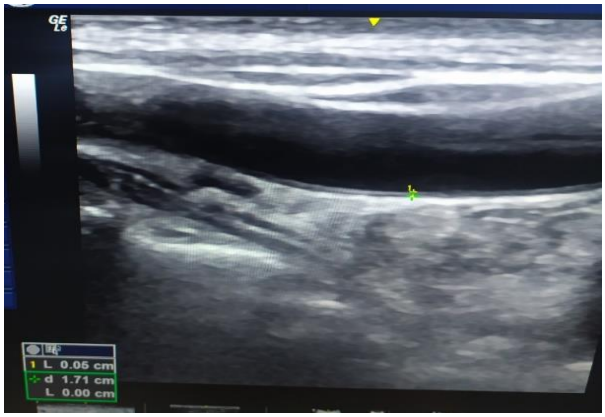

Figure (4): Case No. 22 from the control group. A 35-year-old male patient with an eGFR of 100 mL/min and a CIMT of 0.5 mm.

Statistical analysis

IBM SPSS statistics (Version 28; Armonk, NY, USA) was used for data analysis and management. The normal distribution of quantitative data was examined using the Shapiro-Wilk test in conjunction with visual exploratory methods. According to normality distribution, quantitative data were summarized as means and standard deviations (SD) for normally distributed variables or as medians and interquartile ranges (IQR) for non-normally distributed variables. Categorical data were described using frequencies and percentages. Continuous variables were analyzed using the independent t-test under the assumption of normal distribution, whereas the Mann-Whitney U test was utilized for data that did not meet normality. Categorical comparisons were performed using either the Chi-square test or Fisher's exact test, depending on the data distribution. To compare quantitative variables across CKD subgroups, one-way analysis of variance (ANOVA) was employed for data following a normal distribution, whereas the Kruskall–Wallis test was utilized for non-normally distributed variables. Post-hoc analysis was conducted in case of a significant overall effect, with Bonferroni correction applied for multiple comparisons. Correlations were done using Pearson's and Spearman's correlations. Two-sided tests were applied throughout the analysis, and statistical significance was defined by a p-value ≤ 0.05.

RESULTS

Patients with CKD had a significantly lower BMI compared to controls (26.04 ± 3.33 vs. 27.44 ± 2.80 kg/m², P = 0.008). Among diabetic patients, oral hypoglycemic agents were used significantly less frequently in the CKD group compared to the control group (67.3% vs. 100.0%, *P* = 0.013), while insulin use was exclusive to CKD patients. Additionally, patients with CKD exhibited significantly higher urea (31.97 ± 10.49 vs. 15.42 ± 3.42 mg/dL, P < 0.001) and creatinine levels (2.02 ± 0.52 vs. 0.67 ± 0.14 mg/dL, P < 0.001). Lipid profile parameters were also significantly altered, with CKD patients demonstrating elevated TC (247.35 ± 37.74 vs. 179.56 ± 25.06 mg/dL, P < 0.001), LDL cholesterol (130.58 ± 35.25 vs. 110.96 ± 17.87 mg/dL, P < 0.001), and TGs (267.63 ± 71.16 vs. 150.88 ± 50.33 mg/dL, P < 0.001), while HDL levels were markedly lower (39.71 ± 6.21 vs. 36.28 ± 4.98 mg/dL, P = 0.001). Other variables, including age (P = 0.083), sex (P = 0.683), smoking status (P = 0.555), DM (*P* = 0.484), duration of diabetes (P = 0.141), HTN (P = 0.431), duration of HTN (P = 0.11), dyslipidemia history (P = 0.43), SBP (*P* = 0.489), diastolic blood pressure (DBP) (P = 0.455), HR (P = 0.468), and hemoglobin levels (P = 0.674), did not show substantial differences between the two groups (Table 1).

Table (1): Demographic, clinical, and laboratory characteristics of the studied groups

		Cases (n =150)	Controls (n =50)	P-value
Age (years)	Mean ±SD	46.58 ±8.67	44.08 ±9.13	0.083
Sex				
Female	n (%)	76 (50.7)	27 (54.0)	0.683
Male	n (%)	74 (49.3)	23 (46.0)	
BMI	Mean ±SD	26.04 ±3.33	27.44 ±2.80	**0.008***
Smoking	n (%)	58 (38.7)	17 (34.0)	0.555
DM	n (%)	50 (33.3)	14 (28.0)	0.484
DM duration (years)	Median (IQR)	9 (5 – 15)	5.5 (5 – 9)	0.141
DM treatment				
Oral	n (%)	33 (67.3)	14 (100.0)	**0.013***
Insulin	n (%)	16 (32.7)	0 (0.0)	
HTN	n (%)	60 (40.3)	17 (34.0)	0.431
HTN duration (years)	Median (IQR)	9 (5 – 10)	5 (5 – 8)	0.11
Dyslipidemia history	n (%)	25 (16.7)	6 (12.0)	0.43
SBP (mmHg)	Mean ±SD	121.03 ±16.06	118.88 ±25.90	0.489
DBP (mmHg)	Mean ±SD	78.55 ±10.79	79.80 ±8.45	0.455
HR (bpm)	Mean ±SD	75.86 ±9.76	77.02 ±9.85	0.468
Hb (g/dl)	Mean ±SD	12.54 ±1.34	12.46 ±0.67	0.674
Urea (mg/dl)	Mean ±SD	31.97 ±1.49	15.42 ±3.42	**<0.001***
Creat (mg/dl)	Mean ±SD	2.02 ±0.52	0.67 ±0.14	**<0.001***
TC (mg/dl)	Mean ±SD	247.35 ±37.74	179.56 ±25.06	**<0.001***
LDL (mg/dl)	Mean ±SD	130.58 ±35.25	110.96 ±17.87	**<0.001***
HDL (mg/dl)	Mean ±SD	39.71 ±6.21	36.28 ±4.98	**0.001***
TG (mg/dl)	Mean ±SD	267.63 ±7.16	150.88 ±5.33	**<0.001***

DM: Diabetes Mellitus, IQR: Interquartile Range, HTN: Hypertension, BMI: Body Mass Index, SBP: Systolic Blood Pressure, HR: Heart Rate, DBP: Diastolic Blood Pressure, SD: Standard Deviation, Hb: Hemoglobin, TC: Total Cholesterol, LDL: Low-Density Lipoprotein, TG: Triglycerides, HDL: High-Density Lipoprotein, Creat: Creatinine, Urea: Blood Urea, *: Significant P-value, n: number.

Patients with CKD exhibited significantly increased CIMT relative to controls (1.10 ± 0.16 mm vs. 0.49 ± 0.15 mm, P < 0.001) (Figure 5)

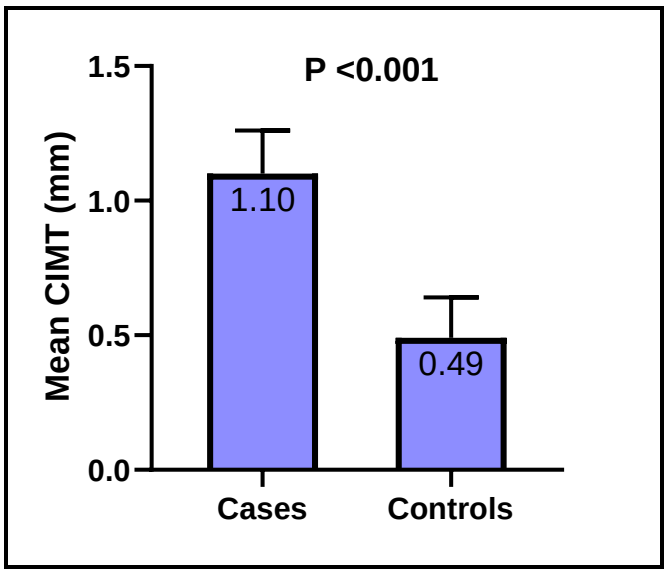

Figure (5): CIMT (mm) between the studied groups.

Three eGFR-based subgroups were established within case group for further analysis: Group I: eGFR 60-89 mL/min (n = 19), group II: eGFR 30-59 mL/min (n = 109), and group III: eGFR 15-29 mL/min (n = 22). Age significantly differed among the CKD subgroups (P < 0.001). Post hoc analysis indicated that it was substantially lower in group I (40.21 ± 7.89 years) compared to group II (46.26 ± 8.38 years) and group III (53.68 ± 5.39 years). Additionally, age was notably lower in group II relative to group III. HTN duration significantly differed among the CKD subgroups (P = 0.033). Post-hoc analysis revealed that it was markedly lower in group I (4 [3 – 5] years) compared to group II (8 [5 – 10] years) and group III (10 [6 – 15] years). Moreover, it was significantly lower in group II compared to group III. Other variables, including sex (P = 0.418), smoking status (P = 0.562), DM (P = 0.253), diabetes duration (P = 0.271), HTN prevalence (P = 0.054), and history of dyslipidemia (P = 0.335), did not show substantial variations among the three groups (Table 2).

Table 2: Demographic and clinical characteristics among CKD subgroups

		Group I (n =19)	Group II (n =109)	Group II (n =22)	P- value
Age (years)	Mean ±SD	40.21 ± 7.89 [2,3]	46.26 ± 8.38 [1,3]	53.68 ± 5.39 [1,2]	**<0.001***
Sex					
Female	n (%)	9 (47.4%)	53 (48.6%)	14 (63.6%)	0.418
Male	n (%)	10 (52.6%)	56 (51.4%)	8 (36.4%)	
Smoking	n (%)	6 (31.6%)	45 (41.3%)	7 (31.8%)	0.562
DM	n (%)	4 (21.1%)	36 (33.0%)	10 (45.5%)	0.253
DM duration (years)	Median (IQR)	5 (4.5 – 7.5)	8 (5 – 16)	10 (7 – 20)	0.271
HTN	n (%)	3 (15.8%)	46 (42.6%)	11 (50.0%)	0.054
HTN duration (years)	Median (IQR)	4 (3 – 5) [3]	8 (5 – 10) [3]	10 (6 – 15) [1,2]	**0.033***
Dyslipidemia history	n (%)	5 (26.3%)	18 (16.5%)	2 (9.1%)	0.335

*Significant P-value; 1: Significantly different from group I, 2: Significantly different from group II, 3: Significantly different from group III, n: number, eGFR: Estimated Glomerular Filtration Rate, DM: Diabetes Mellitus, IQR: Interquartile Range, HTN: Hypertension, SD: Standard Deviation.

CIMT significantly differed among the CKD subgroups (P < 0.001). Post hoc analysis highlighted that CIMT was substantially lower in group I (0.83 ± 0.09 mm) compared to group II (1.11 ± 0.10 mm) and group III (1.30 ± 0.08 mm). Additionally, CIMT was significantly lower in group II compared to group III (Figure 6).

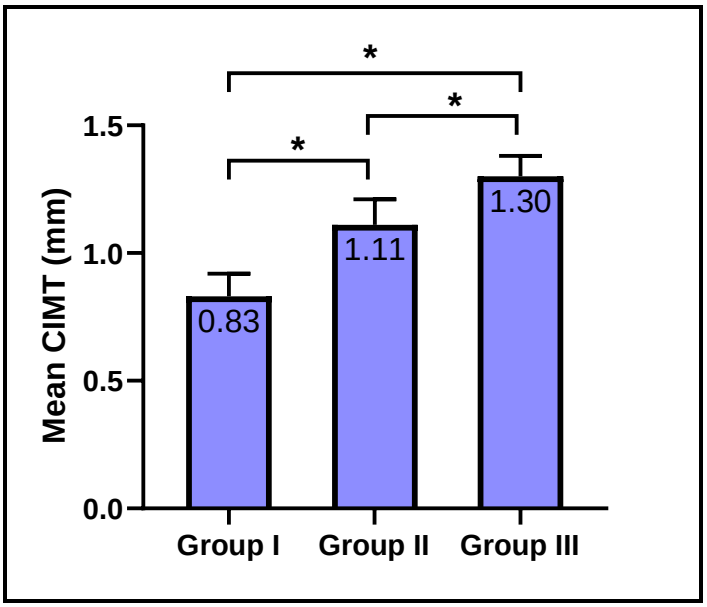

Figure (6): CIMT (mm) among the three case groups.

CIMT revealed substantial positive correlations with age (r = 0.377, P < 0.001), SBP (r = 0.311, P < 0.001), DBP (r = 0.328, P < 0.001), creatinine (r = 0.868, P < 0.001), urea (r = 0.187, P = 0.022), and TC (r = 0.387, P < 0.001). In contrast, CIMT showed a notable negative correlation with GFR (r = -0.960, P < 0.001). No substantial correlations were observed between CIMT and BMI (P = 0.068), LDL (P = 0.317), and HDL (P = 0.144) (Table 3).

Table (3): Correlation of CIMT with other studied parameters among all patients with renal impairment

	CIMT	
	r	**P- value**
Age (years)	**0.377**	**<0.001***
BMI (kg/m²)	-0.149	0.068
SBP (mmHg)	**0.311**	**<0.001***
DBP (mmHg)	**0.328**	**<0.001***
Creat (mg/dL)	**0.868**	**<0.001***
Urea (mg/dL)	**0.187**	**0.022***
GFR (mL/min/1.73m²)	**-0.960**	**<0.001***
TC (mg/dL)	**0.387**	**<0.001***
LDL (mg/dL)	-0.082	0.317
HDL (mg/dL)	-0.120	0.144

BMI: Body Mass Index, CIMT: Carotid Intima-Media Thickness, SBP: Systolic Blood Pressure, Creat: Creatinine, GFR: Glomerular Filtration Rate, HDL: High-Density Lipoprotein, DBP: Diastolic Blood Pressure, Urea: Blood Urea, TC: Total Cholesterol, LDL: Low-Density Lipoprotein, r: Correlation Coefficient, *: Significant P-value.

CIMT was significantly higher in patients with DM compared to those without DM (1.16 ± 0.14 mm vs. 1.07 ± 0.16 mm, P = 0.001). Similarly, CIMT was markedly increased in patients with HTN relative to those without HTN (1.15 ± 0.14 mm vs. 1.07 ± 0.16 mm, P = 0.005) (Table 4).

Table (4): Relation for CIMT with the presence of DM and HTN

	Mean ± SD	**P-value**
Diabetes mellitus		
Yes	1.16 ± 0.14	**0.001***
No	1.07 ± 0.16	
Hypertension		
Yes	1.15 ± 0.14	**0.005***
No	1.07 ± 0.16	

CIMT: Carotid Intima-Media Thickness, SD: Standard Deviation, *: Significant P-value.

DISCUSSION

Patients with CKD are at a significantly higher risk of developing CVD. CKD and CVD share many common risk factors, such as diabetes and HTN. Evidence from longitudinal studies indicates that cardiovascular events are more prevalent than renal outcomes in individuals with CKD, with mortality rates exceeding the incidence of progression to ESRD [10]. Globally, CVD stands as the foremost cause of mortality, with a heightened prevalence among patients with high-risk comorbidities such as DM and CKD [11]. Accordingly, comprehensive cardiovascular risk assessment is vital and can be achieved through vascular imaging in conjunction with traditional risk factor evaluation.

CIMT is increasingly utilized as a non-invasive and widely available marker of atherosclerotic burden [11]. Extensive evidence supports a strong relationship between carotid artery wall changes and CVD, with CIMT and plaque presence serving as valuable predictors of cardiovascular events among the general population [2]. This study aimed to assess the impact of CKD on CIMT and evaluate CIMT's potential utility as a surrogate indicator of CVD risk in affected patients.

This case-control study was carried out at Ain Shams University Hospital and included 200 participants aged between 30 and 60 years. The study population was stratified into two main groups: a control group of 50 individuals with normal renal function (eGFR > 90 mL/min), and a case group of 150 non-dialysis CKD patients (eGFR 15–89 mL/min), which was further stratified into three subgroups according to eGFR values. The results revealed that patients with CKD had substantially elevated CIMT values (mean: 1.10 ± 0.16 mm) compared to the control group (mean: 0.49 ± 0.15 mm), despite similar incidences of other atherosclerotic risk factors such as diabetes, HTN, smoking, and dyslipidemia.

Our findings align with those of **Shoji *et al.*** [12], who measured CIMT in 110 non-diabetic pre-dialysis CKD patients (serum creatinine ≥1.5 mg/dL, CIMT: 0.88 mm), 345 non-diabetic patients with ESRD on maintenance hemodialysis (CIMT: 0.86 mm), and 302 healthy controls (CIMT: 0.68 mm). Their study concluded that CIMT was substantially greater in CKD patients than in controls, whereas CIMT values did not differ significantly between CKD and hemodialysis groups. Additionally, no substantial correlation was detected between CIMT and the duration of hemodialysis.

Our CIMT values were slightly higher than those reported by **Roumeliotis *et al.*** [13], who included 142 type 2 diabetes mellitus (T2DM) patients at various CKD stages (CIMT: 0.86 mm), and **Szeto *et al.*** [14], who studied 203 Chinese patients with CKD stage 3–4 (CIMT: 0.81 mm). These variations could be due to heterogeneity in the study populations, especially differences in ethnicity (Greek and Chinese) and the proportion of patients at various CKD stages.

When analyzing CKD patients based on eGFR levels, we observed a progressive increase in CIMT with worsening kidney function. CIMT showed a clear trend of gradual elevation as eGFR decreased. At baseline, patients with eGFR <75 mL/min exhibited higher CIMT values (>0.7 mm) than those with eGFR ≥75 mL/min. Using CIMT as a stratification parameter, **Roumeliotis *et al.*** [13] categorized 142 patients with T2DM and differing CKD stages into two subgroups, noting that those with CIMT exceeding 0.86 mm had greater age and more progressed kidney dysfunction.

In the scope of our study, CIMT was positively correlated with several clinical parameters, including age, BP, serum urea, creatinine, and TC, and negatively correlated with eGFR in our study cohort. In addition, patients with coexisting diabetes and HTN demonstrated markedly increased CIMT levels relative

to non-diabetic and normotensive individuals, corroborating results from previous studies. **Shoji *et al.*** [12] reported a positive correlation between CIMT, age, and BP in their study population. Similarly, **Szeto *et al.*** [14] observed that CIMT was positively associated with age and was significantly higher in diabetic patients than in non-diabetic individuals.

LIMITATIONS

Our study had several limitations. First, participants were recruited from a single hospital, and the sample size was relatively small, limiting the generalizability of findings to the broader population of CKD patients not on dialysis in Egypt. Second, we did not follow up with patients to assess the long-term impact of increased CIMT on cardiovascular events. Third, ultrasonographic assessment of CIMT lacks a standardized, universally accepted scanning protocol, which may introduce variability. Additionally, interobserver and intraobserver reproducibility can be affected by factors such as the type of ultrasound scanner, the sonographer's experience, and the millimeter-scale precision of CIMT measurements, where small trackball movements may lead to significant measurement errors. Future prospective, multicenter studies with standardized imaging protocols and larger, more representative samples are needed to confirm the association between eGFR and CIMT.

CONCLUSION

CIMT is significantly increased in CKD patients, correlating with declining renal function and traditional cardiovascular risk factors. These findings highlight the importance of early cardiovascular risk assessment and intervention in CKD patients.

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Conflict of Interest: None.

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