

# Correlation of carotid artery intima–media thickness with parameters of mineral bone disorder in patients with chronic kidney disease

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## ABSTRACT

**Background.** Chronic kidney disease (CKD) is associated with accelerated cardiovascular (CV) risk, particularly due to subclinical atherosclerosis. Carotid intima–media thickness (CIMT) is a non-invasive marker of early atherosclerotic changes. Altered mineral metabolism in CKD, also known as CKD–mineral bone disorder (CKD-MBD), may contribute to vascular damage. We evaluated the association between CIMT and mineral metabolism markers in people with diabetes who had pre-dialysis CKD.

**Methods.** A cross-sectional observational study was done in 110 adults with diabetes and pre-dialysis CKD. Biochemical markers including serum phosphate, intact parathyroid hormone (iPTH), fibroblast growth factor (FGF)-23, and 24-hour urinary phosphate were analyzed. CIMT was measured using B-mode ultrasonography. Correlation and regression analyses were performed.

**Results.** CIMT showed significant positive correlations with serum phosphate, FGF-23, iPTH, and serum creatinine, and negative correlations with nephron index and urinary phosphate excretion. Serum phosphate >6 mg/dl strongly predicted CIMT >0.9 mm.

**Conclusion.** Mineral metabolism markers, particularly serum phosphate and FGF-23, are significantly associated with subclinical atherosclerosis in CKD. Monitoring these parameters may aid in assessment of early CV risk in patients with CKD.

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## INTRODUCTION

Atherosclerosis is the precursor of major cardiovascular (CV) events such as myocardial ischaemia and infarction. Of the two accepted imaging markers of atherosclerosis, i.e. coronary

artery calcium score measured by computed tomography (CT) scan and carotid intima–media thickness (CIMT) measured by ultrasound (US), CIMT has the potential to be widely adopted as a clinical tool.

The easiest way to measure the degree of atherosclerosis is by measuring the thickness of the intimal layer of a large artery usually by US. CIMT is a measurement of the thickness of the tunica intima and tunica media, the innermost two layers of the wall of an artery and is recognized as a marker of subclinical atherosclerosis. It is measured using high-frequency ( $\geq 8$  MHz) US transducers on both carotid arteries. It is recorded at the distal straight 1 cm of the common carotid arteries, the carotid bifurcations and the proximal artery and 1 cm of the internal carotid arteries.

Evaluation of CIMT and plaque formation can improve prediction of risk of coronary heart disease when added to the traditional risk factors. CIMT measurement is a reliable, inexpensive, non-invasive, feasible method and free of radiation. According to the European Society of Cardiology/Hypertension guidelines, CIMT in the normal population is 0.5–0.6 mm and CIMT >0.9 mm is a marker of asymptomatic organ damage.

Salonen and Salonen demonstrated atherosclerotic changes and a close histological relationship between coronary, cerebral and carotid atherosclerotic disease.<sup>1</sup> Increased CIMT is a predictor of adverse CV events and has also been shown to be an independent predictor of CV mortality in patients with chronic kidney disease (CKD) on haemodialysis.<sup>2</sup> The increased arterial stiffness in CKD leads to an isolated increase in systolic blood pressure causing structural and functional cardiac abnormalities and is thus an independent predictor of CV and overall mortality in patients with CKD.

Chhajer *et al.*<sup>3</sup> examined the correlation of CIMT with conventional CV risk factors in different stages of CKD. The mean CIMT in the CKD patients was higher than in healthy age- and sex-matched controls. We examined the vascular changes in people with diabetes with CKD using CIMT and markers of mineral metabolism.

## METHODS

We enrolled 110 patients >18 years of age of both sexes, who had diabetes-related CKD but were not on dialysis. Patients with an estimated glomerular filtration rate (eGFR) <15, those on haemodialysis, who had undergone renal transplant, or had liver disease, thyroid illness, primary hyperparathyroidism, malignancy, vitamin D deficiency or resistance, and those on carbonic anhydrase inhibitors were excluded from the study.

After detailed history and examination, investigations such as kidney function test, serum electrolytes, serum calcium,

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phosphate, serum fibroblast growth factor (FGF)-23, haemoglobin A1c, fasting and post-prandial blood sugar, lipid profile, serum protein, serum intact parathyroid hormone (iPTH) and haemoglobin, urinary albumin and phosphate levels were done.

US whole abdomen was done and CIMT was measured using B-mode and a 7.5 MHz transducer (Fig. 1). CIMT was defined as the distance between the leading edge of the first echogenic line (lumen-intima interface) and the second echogenic line (media-adventitia interface) of the arterial wall. Three measurements were taken at 0.5, 1 and 2 cm below the carotid bifurcation of the common carotid artery on each side and their arithmetic averages were calculated. The CIMT of both the left and right sides was calculated and the average was taken.

### Statistical analysis

Categorical variables were presented in number and percentage (%) and continuous variables as mean (SD) and median. The normality of data was tested by Kolmogorov-Smirnov test. Data not distributed normally was analysed using a non-parametric test.

Pearson correlation coefficient/Spearman rank correlation coefficient was used to correlate nephron index with the degree of atherosclerosis.  $p < 0.05$  was considered statistically significant. Data were entered in MS Excel spreadsheet and analysis was done using the Statistical Package for the Social Sciences version 21.0.

### RESULTS

The mean (SD) age of the patients was 43.8 (15) years. 27 (24.5%) of the participants were 18–30 years of age, 20 (18.2%) were 31–40 years, 26 (22.7%) were 41–50 years, 23 (20.9%) were

51–60 years and 11 (10.0%) were 61–70 years of age. Only 4 (3.6%) were older than 71 years. Age distribution was normal (Shapiro-Wilk test,  $p = 0.054$ ). Seventy (63.6%) of the participants were male (Table 1). CIMT showed a mild inverse relationship with haemoglobin (Fig. 2).

The mean (SD) CIMT was 0.70 (0.17) mm (range 0.38–1.08 mm) and the median (interquartile range) was 0.70 mm (0.58–0.83 mm). Eighty-eight (80%) of the participants had CIMT  $< 0.9$  mm while 22 (20%) had CIMT values  $\geq 0.9$  mm.

### Association between CIMT (mm) and biochemical parameters

The following variables were significantly associated ( $p < 0.05$ ) with CIMT (mm): Age (years), gender, serum creatinine

TABLE 1. Demographic characteristics and baseline values of the patients

Characteristic	Mean (SD)	Range
Age (years)	43.80 (15.08)	18–78
Body weight (kg)	63.36 (8.37)	45–80
Systolic blood pressure (mmHg)	140.04 (17.16)	110–180
Diastolic blood pressure (mmHg)	88.05 (10.64)	64–120
Haemoglobin (g/dl)	9.23 (1.57)	4.1–12.4
Total leucocyte count (cmm)	7493 (2087)	4300–11 600
Serum protein (g/dl)	5.42 (1.00)	2.8–8.0
Blood urea (mg/dl)	92.44 (25.72)	40–180
Serum creatinine (mg/dl)	2.27 (0.70)	1.2–4.7
Serum sodium (mEq/L)	141.05 (6.29)	128–155
Serum potassium (mEq/L)	4.47 (0.58)	3.4–6.1
Serum calcium (mg/dl)	8.57 (0.54)	6.9–9.6
Serum phosphate (mg/dl)	5.46 (0.68)	3.2–7.2
Intact parathyroid hormone (pg/ml)	57.07 (23.87)	27–157
Fasting blood sugar (mg/dl)	186.05 (41.62)	91–269
Post-prandial blood sugar (mg/dl)	223.35 (40.46)	115–299
Glycosylated haemoglobin (%)	7.83 (1.25)	6.1–11
Total cholesterol (mg/dl)	250.01 (73.05)	139–458
Low density lipoprotein (mg/dl)	104.45 (26.27)	68–202

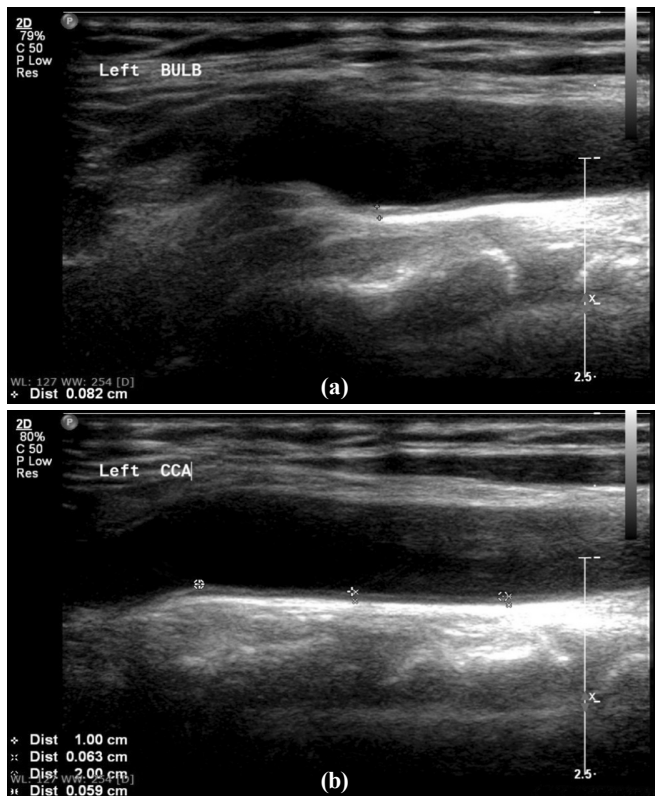


FIG 1. Longitudinal sonograms showing carotid intima-media thickness (CIMT) measured at (a) 0.5 cm and 1 cm, and (b) 2 cm from the bifurcation of the left common carotid artery

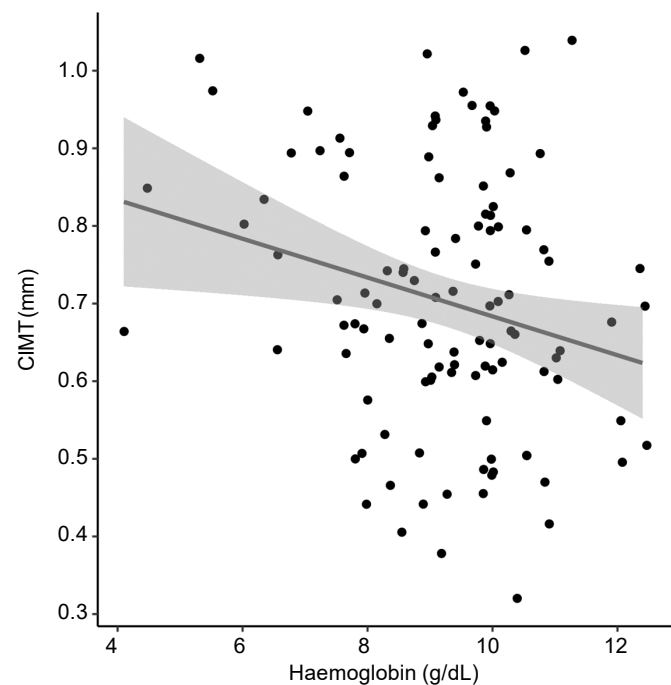


FIG 2. Association between haemoglobin and carotid intima-media thickness (CIMT)

(mg/dl), serum potassium (mEq/L), serum phosphate (mg/dl), iPTH, 24-h urinary phosphate (PO<sub>4</sub>) (mg), FGF-23 (pg/ml), eGFR (ml/minutes/1.73m<sup>2</sup>) and CKD stage (Table 2).

There was a weak positive correlation between serum creatinine (mg/dl) and CIMT (mm), and this correlation was statistically significant ( $\rho=0.24$ ,  $p=0.013$ , Fig. 3). For every 1 unit increase in serum creatinine (mg/dL), the CIMT (mm) increases by 0.05 units (Fig. 3). Conversely, for every 1 unit increase in CIMT (mm), the serum creatinine (mg/dl) increases by 0.84 units. There was slight negative correlation seen with CIMT and serum calcium levels (Fig. 4).

For every 1 unit increase in serum phosphate (mg/dl), the CIMT (mm) increases by 0.06 units. Conversely, for every 1 unit increase in CIMT (mm), the serum phosphate (mg/dl) increases by 1.01 units (Fig. 5). The higher iPTH levels were associated with greater CIMT (Fig. 6). Conversely, for every 1 unit increase in CIMT (mm), the iPTH increases by 51.14 units. An inverse correlation between CIMT and urinary phosphate is depicted in Fig. 7. Conversely, for every 1 unit increase in CIMT (mm), the 24-hour urinary PO<sub>4</sub> (mg) decreases by 1879.90 units. Conversely, for every 1 unit increase in CIMT (mm), the FGF-23 (pg/ml) increases by 326.40 units (Fig. 8). For every 1 unit increase in

TABLE 2. Association between carotid intima-media thickness (mm) and different parameters

Parameter	CIMT (mm)	p value
<i>Age (years)</i>	Correlation coefficient ( $\rho$ )=0.3	0.001*
18-30	0.66 (0.16)	0.010†
31-40	0.64 (0.19)	
41-50	0.67 (0.14)	
51-60	0.78 (0.15)	
61-70	0.79 (0.19)	
71-80	0.86 (0.16)	
<i>Gender</i>		<0.001‡
Male	0.64 (0.16)	
Female	0.81 (0.15)	
Body weight (kg)	Correlation coefficient ( $\rho$ )=-0.05	0.58*
Systolic blood pressure (mmHg)	Correlation coefficient ( $\rho$ )=-0.08	0.42*
Diastolic blood pressure (mmHg)	Correlation coefficient ( $\rho$ )=0.02	0.84*
Haemoglobin (g/dl)	Correlation coefficient ( $\rho$ )=-0.17	0.08*
Total leucocyte count (/cum)	Correlation coefficient ( $\rho$ )=-0.07	0.49*
Serum protein (g/dl)	Correlation coefficient ( $\rho$ )=-0.15	0.13*
Blood urea (mg/dL)	Correlation coefficient ( $\rho$ )=-0.11	0.24*
Serum creatinine (mg/dl)	Correlation coefficient ( $\rho$ )=0.24	0.01*
Serum sodium (mEq/l)	Correlation coefficient ( $\rho$ )=-0.05	0.60*
Serum potassium (mEq/l)	Correlation coefficient ( $\rho$ )=0.21	0.03*
Serum calcium (mg/dl)	Correlation coefficient ( $\rho$ )=-0.15	0.11*
Serum phosphate (mg/dl)	Correlation coefficient ( $\rho$ )=0.23	0.02*
Intact parathyroid hormone	Correlation coefficient ( $\rho$ )=0.40	<0.001*
Fasting blood sugar (mg/dl)	Correlation coefficient ( $\rho$ )=-0.02	0.84*
Post-prandial blood sugar (mg/dl)	Correlation coefficient ( $\rho$ )=-0.1	0.32*
Glycosylated haemoglobin (%)	Correlation coefficient ( $\rho$ )=0.06	0.51*
Total cholesterol (mg/dl)	Correlation coefficient ( $\rho$ )=0.02	0.82*
Low density lipoprotein (mg/dl)	Correlation coefficient ( $\rho$ )=0.03	0.76*
<i>Urine protein</i>		0.39†
Nil	0.68 (0.18)	
1+	0.74 (0.15)	
2+	0.69 (0.15)	
3+	0.76 (0.18)	
<i>Urine red blood cells</i>		0.45†
Present	0.63 (0.18)	
Absent	0.70 (0.17)	
24-hour urinary PO <sub>4</sub> (mg)	Correlation coefficient ( $\rho$ )=-0.42	<0.001*
Fibroblast growth factor-23 (pg/ml)	Correlation coefficient ( $\rho$ )=0.29	0.002*
Nephron index	Correlation coefficient ( $\rho$ )=-0.39	<0.001*
<i>CIMT</i>		<0.001¶
<0.9 mm	0.64 (0.13)	
≥0.9 mm	0.96 (0.05)	
eGFR (ml/minutes/1.73 m <sup>2</sup> )	Correlation coefficient ( $\rho$ )=-0.5	<0.001*
<i>CKD stage</i>		<0.001†
3a	0.56 (0.14)	
3b	0.70 (0.17)	
4	0.77 (0.15)	

\* Spearman correlation † Kruskal-Wallis test ‡ *t*-test ¶ Wilcoxon-Mann-Whitney U-test  
eGFR estimated glomerular filtration rate

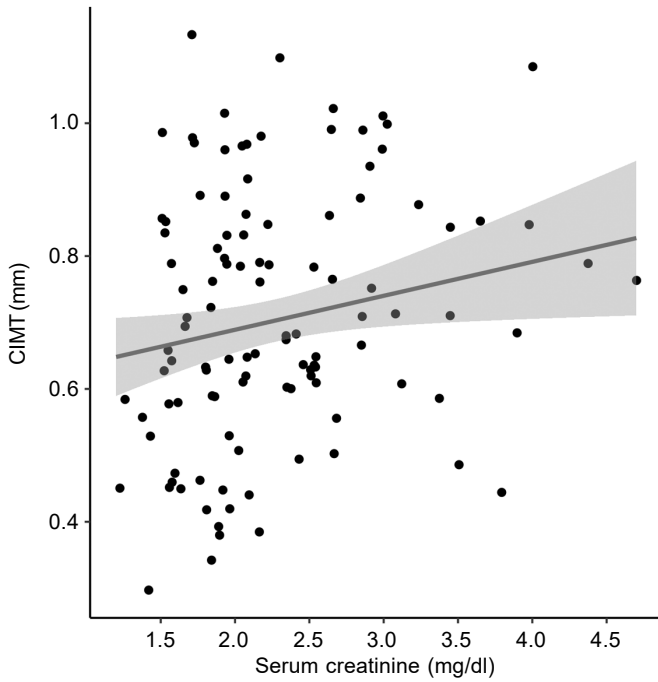


FIG 3. Association between serum creatinine and carotid intima-media thickness (CIMT)

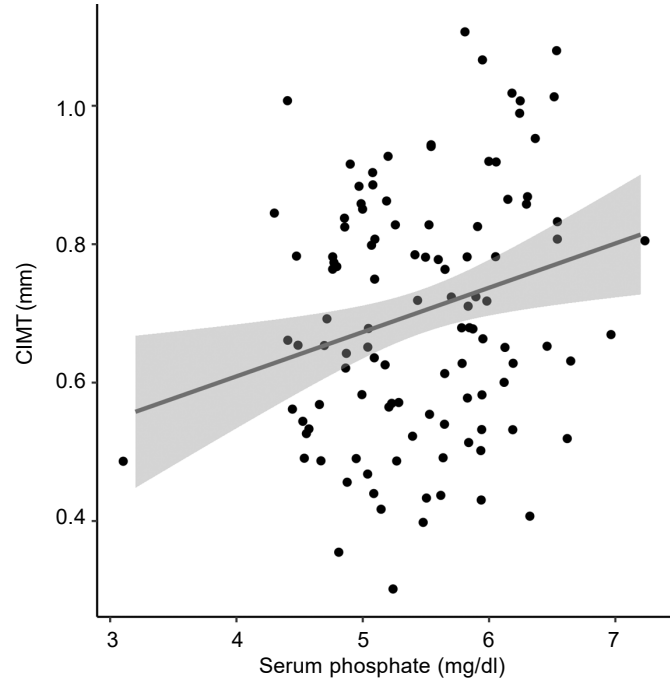


FIG 5. Association between serum phosphate and carotid intima-media thickness (CIMT)

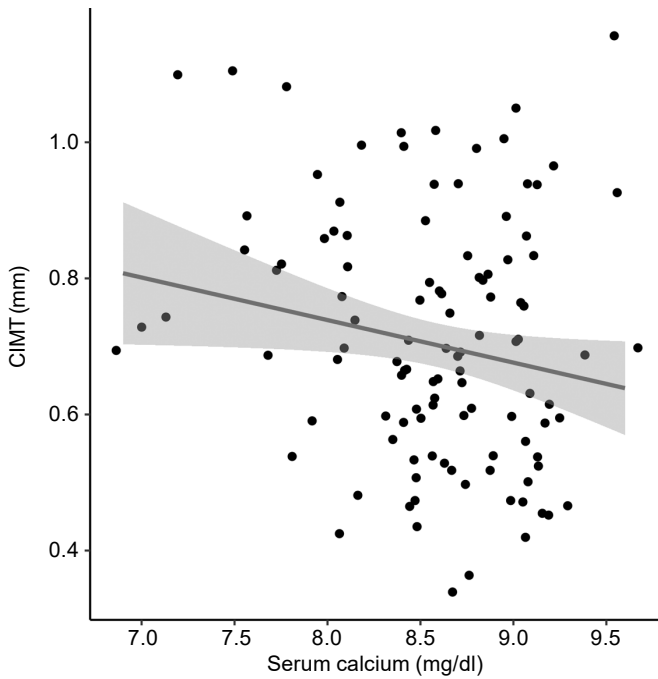


FIG 4. Association between serum calcium and carotid intima-media thickness (CIMT)

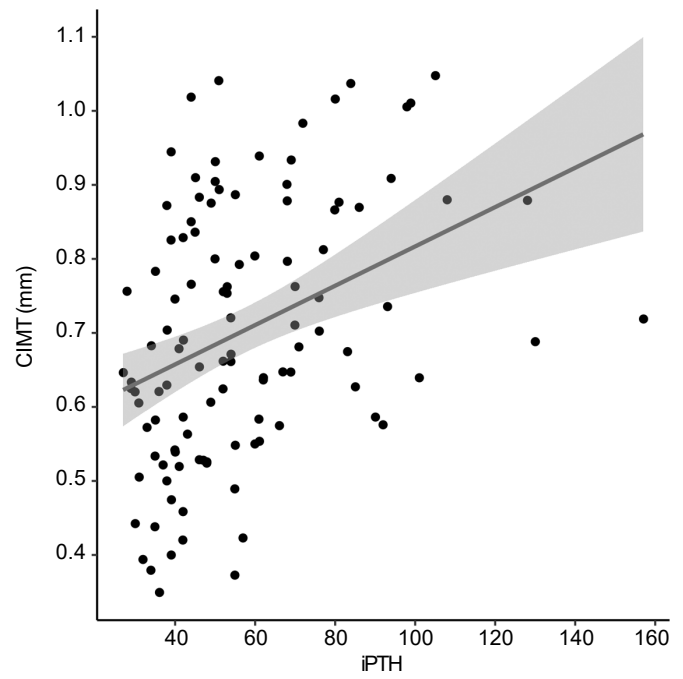


FIG 6. Association between intact parathyroid hormone (iPTH) and carotid intima-media thickness (CIMT)

eGFR (ml/minutes/1.73 m<sup>2</sup>), the CIMT (mm) decreases by 0.01 units (Fig. 9). Conversely, for every 1 unit increase in CIMT (mm), the eGFR (ml/minutes/1.73 m<sup>2</sup>) decreases by 34.67 units. The relation between CKD stage and CIMT is shown in Fig. 10.

*Regression model for CIMT <0.9 mm*

With CIMT <0.9 mm as the dependent variable, regression analysis was done (Table 3).

Serum phosphate was found to be the most important predictor of CIMT <0.9 mm, followed by FGF-23. Performance of study parameters was done for predicting CIMT <0.9 mm versus >0.9 mm. FGF-23 >620 pg/ml, nephron index ≤1.941, 24-hour urinary phosphate ≤404.25 and serum phosphate ≥6 mg/dl were performance predictors of CIMT >0.9 mm. FGF-23 correlated with CIMT. FGF-23 ≥620 pg/ml predicted CIMT ≥0.9 mm with a sensitivity of 64% and a specificity

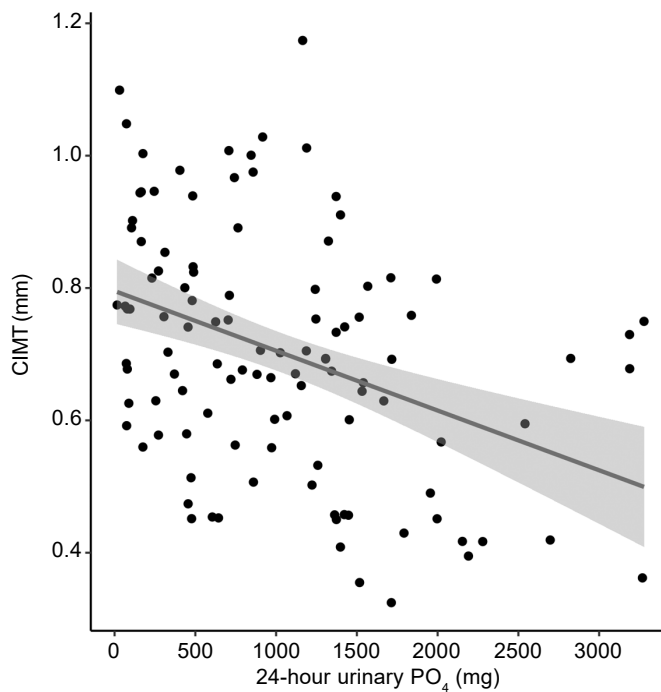


FIG 7. Association between 24-hour urinary phosphate and carotid intima-media thickness (CIMT)

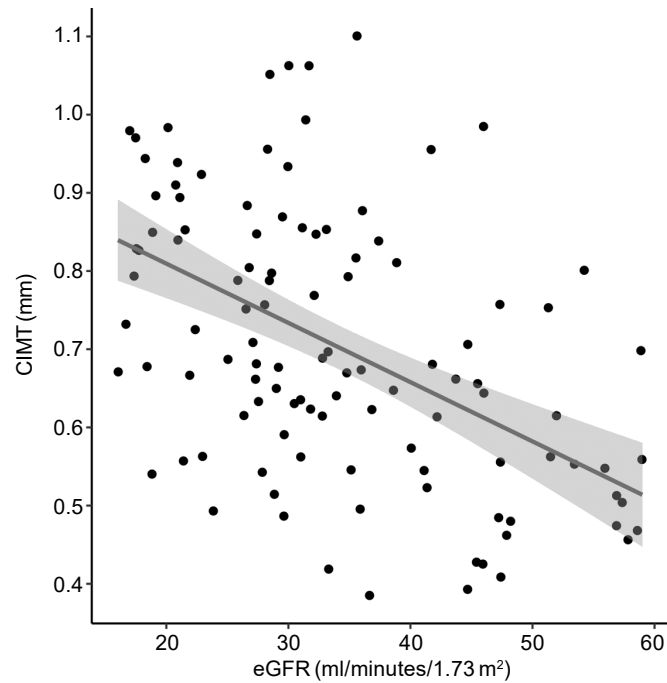


FIG 9. Association between estimated glomerular filtration rate (eGFR) and carotid intima-media thickness (CIMT)

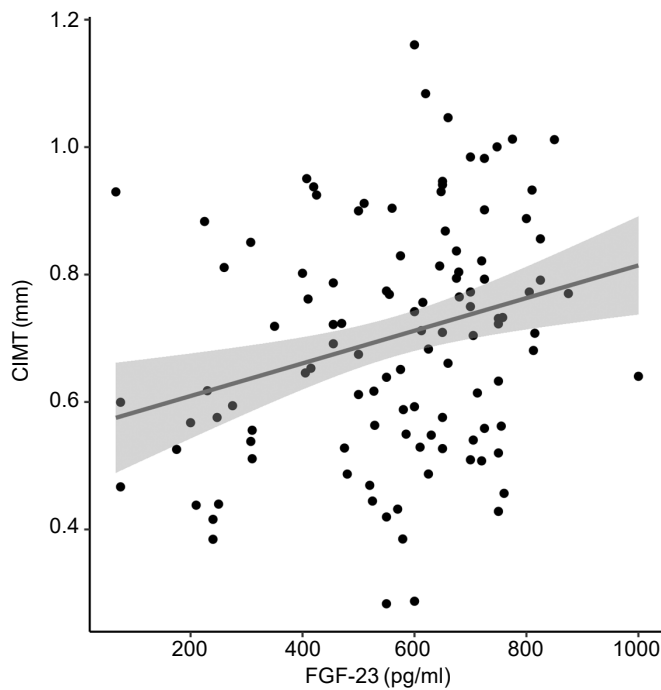


FIG 8. Association between fibroblast growth factor-23 (FGF-23) and carotid intima-media thickness (CIMT)

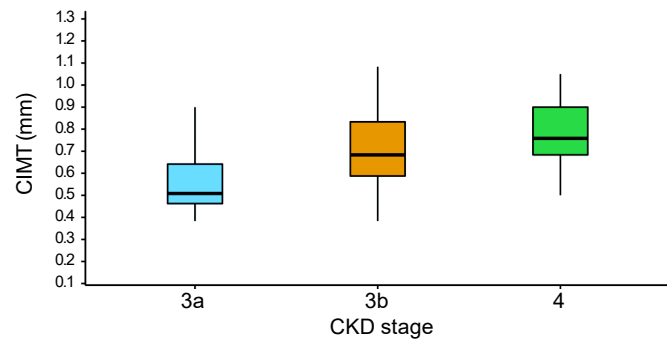


FIG 10. Association between chronic kidney disease (CKD) stage and carotid intima-media thickness (CIMT) (linear plot)

of 58%. The odds ratio (95% CI) for CIMT  $\geq 0.9$  mm when FGF-23 is  $\geq 620$  pg/ml was 1.99 (0.77–5.15). The relative risk (95% CI) for CIMT  $\geq 0.9$  mm when FGF-23 is  $\geq 620$  pg/ml was 1.73 (0.82–3.67).

Serum phosphate was the best parameter to predict atherosclerosis (CIMT  $> 0.9$  mm). Serum phosphate  $> 6$  mg/dl predicted CIMT  $> 0.9$  mm with a sensitivity of 59% and specificity of 83%. The odds ratio (95% CI for CIMT  $> 0.9$  mm), when phosphate  $> 6$  mg/dl, was 6.33 (2.25–17.81). The relative risk

TABLE 3. Regression model for carotid intima-media thickness (CIMT)  $< 0.9$  mm as the dependent variable

Variable	$< 0.9$ mm	$\geq 0.9$ mm	Odds ratio (univariate)	Odds ratio (multivariate)
Serum calcium (mg/dl)	8.6 (0.5)	8.4 (0.7)	0.49 (0.21–1.11, p=0.088)	–
Serum phosphate (mg/dl)	5.4 (0.7)	5.8 (0.6)	2.91 (1.34–6.32, p=0.007)	2.62 (1.12–6.10, p=0.026)
24-hour urinary PO <sub>4</sub> (mg)	1126.8 (814.1)	622.8 (488.2)	1.00 (1.00–1.00, p=0.009)	–
Fibroblast growth factor-23 (pg/ml)	551.4 (201.7)	617.4 (153.5)	1.00 (1.00–1.00, p=0.157)	1.00 (0.99–1.00, p=0.074)
Nephron index	3.2 (3.8)	1.3 (1.5)	0.71 (0.52–0.97, p=0.032)	0.56 (0.34–0.93, p=0.026)

(95% CI for CIMT >0.9 mm), when serum phosphate >6 mg/dl, was 3.78 (1.86–7.43). For every 1 unit increase in serum phosphate, the CIMT (mm) increased by 0.06 units. Conversely, for every 1 unit increase in CIMT, the serum phosphate (mg/dl) increases by 1.01 units. Similar results were reported by Arora *et al.* from India.<sup>4</sup>

## DISCUSSION

CV disease is a major concern in persons with CKD. There continues to be a substantial burden of CV risk factors amongst adults with CKD stages 3–5 and, to a lesser extent, adults with CKD stages 1–2 compared with adults who do not have CKD. Patients with CKD are more likely to die of CV disease than to reach end-stage kidney disease. Stage 3 CKD patients have 20 times higher risk of dying due to CV disease than to progress to end-stage kidney disease.<sup>5,6</sup>

CIMT is correlated with the stage of CKD and CV morbidity and mortality. The degree of CKD can also be used to directly assess the CV risk. There are reports that directly link CV morbidity and mortality with CIMT.<sup>7</sup> It has been proposed that the ratio of urinary protein excretion (mg/day) to FGF-23 is to be used as an index that theoretically represents the number of nephrons (nephron index). If the nephron index indeed reflects the functional nephron number, it should start decreasing in early-stage CKD and be useful to assess the deterioration of renal function and evaluate the risk for complications. Thus, it may aid in making a rational and scientific decision regarding initiation of pre-emptive treatment.

CV disease remains the leading cause of morbidity and mortality in patients with CKD, often surpassing the risk of progression to end-stage kidney disease.<sup>5–7</sup> Our study highlights an important correlation between CIMT—a surrogate marker of subclinical atherosclerosis—and biochemical parameters associated with mineral bone disorder in pre-dialysis CKD patients.

We found that serum phosphate levels had the strongest association with CIMT. A serum phosphate concentration >6 mg/dl was predictive of increased CIMT ( $\geq 0.9$  mm) with high specificity (83%), suggesting that hyperphosphataemia may be a contributor to vascular pathology in CKD. This is consistent with previous reports from Indian cohorts, which have linked elevated phosphate with early atherosclerotic changes in CKD patients.

FGF-23, a hormone involved in phosphate homeostasis, also showed a positive association with CIMT. Although the predictive performance of FGF-23 for elevated CIMT was modest (sensitivity 64%, specificity 58%), the finding supports growing evidence that FGF-23 may play a role in vascular remodelling and calcification. While some studies, have corroborated this link,<sup>7</sup> others have not found a significant association, possibly due to population differences or confounding factors such as vitamin D levels and inflammatory status.<sup>8–10</sup>

iPTH and reduced 24-hour urinary phosphate excretion were also independently associated with higher CIMT values. These findings reinforce the hypothesis that disturbances in mineral metabolism may drive vascular injury through both calcification and impaired phosphate clearance. The nephron index—derived from the ratio of urinary phosphate to FGF-23—was inversely related to CIMT and may serve as a surrogate for nephron functional mass.

Interestingly, traditional CV risk factors such as increased age, male gender, elevated mean arterial pressure, and dyslipidaemia also correlated with higher CIMT, aligning with well-established literature. However, no significant correlation was found between CIMT and serum calcium or low density lipoprotein cholesterol levels, which contrasts with findings in dialysis-dependent populations and may reflect differences in disease stage or treatment exposure.

These observations suggest that even in pre-dialysis CKD, abnormalities in mineral metabolism are intimately linked with vascular changes. Serum phosphate, in particular, emerges as a readily available and clinically actionable marker.<sup>4</sup> Early identification and correction of hyperphosphataemia may offer a strategy to mitigate CV risk in this vulnerable population.

In our study, the severity of atherosclerosis (CIMT) correlated well with the stage of CKD, eGFR, 24-hour urinary phosphate excretion, FGF-23 and parathyroid hormone levels. There have been conflicting reports regarding the relationship between CIMT and FGF-23. Yilmaz *et al.*<sup>7</sup> reported a positive correlation between FGF-23 and CIMT as in our study. However, Kaya *et al.* reported no correlation between FGF-23 and CIMT,<sup>8</sup> even though serum FGF-23 levels increased with progression of CKD. Other studies<sup>10,11</sup> have also reported no correlation between the two.

The cross-sectional nature of our study limits causal inference. Additionally, we did not measure vascular calcification scores or inflammatory markers, which may have provided further insight into the mechanisms underlying increased CIMT. Longitudinal studies are needed to determine whether modifying phosphate or FGF-23 levels can reduce CIMT progression and improve CV outcomes.

*Conflicts of interest.* None declared

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