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ORIGINAL ARTICLE

Association between cardiothoracic ratio and aortic arch calcification with estimated glomerular filtration rate in hypertensive patients

Fransiskus Aryo Pratomo[1]*, Aziza Ghanie Icksan[2], Ikhwanul Hakim Nasution[2]

ABSTRACT

Hypertension remains a leading cause of global cardiovascular mortality, including in Indonesia. Target organ damage, particularly renal impairment, underscores the need for easily obtainable biomarkers for early detection. Although not explicitly recommended in current guidelines, the cardiothoracic ratio (CTR) and aortic arch calcification (AAC), both assessable via chest radiography, represent readily available and cost-effective screening tools. This study aimed to evaluate the association between these radiographic markers and decreased estimated glomerular filtration rate (eGFR) in hypertensive patients at Royal Prima Hospital, thereby addressing a crucial knowledge gap in resource-limited settings with restricted access to advanced imaging modalities. A cross-sectional analysis was conducted involving 175 hypertensive participants, stratified according to their chronic kidney disease (CKD) stage. Baseline demographics, hypertension status, CTR, AAC, and eGFR were assessed. Bivariate correlation and multivariate regression analyses were performed to determine the relationships between CTR, AAC, and eGFR. The study population exhibited a high prevalence of cardiovascular comorbidities, with 82.3% demonstrating cardiomegaly (mild-to-severe CTR) and 61.7% presenting with AAC. Bivariate analysis revealed a strong inverse correlation between eGFR and both CTR ($r = -0.418$, $p < 0.001$) and AAC score ($r = -0.457$, $p < 0.001$). Multivariate regression confirmed that both CTR ($B = -1.738$, $p < 0.001$) and AAC ($B = -16.127$, $p < 0.001$) were significant predictors of eGFR decline. Progressive CKD stages were associated with increased CTR (52.3% in Stage 1 vs. 58.7% in Stage 5) and greater AAC severity (0% advanced calcification in Stage 1 vs. 34% in Stage 5). Age, sex, and blood pressure did not show significant correlations with eGFR ($p > 0.05$). CTR and AAC are strongly associated with renal dysfunction, reinforcing the evidence of an interaction between cardiovascular pathology and CKD progression. These markers have the potential to serve as accessible biomarkers for identifying high-risk patients, thereby facilitating early intervention in resource-limited settings.

Keywords: chronic kidney disease, cardiothoracic ratio, cardiomegaly, aortic arch calcification, glomerular filtration rate

INTRODUCTION

Systemic arterial hypertension is defined as blood pressure exceeding 140/90 mmHg, according to established classification systems.[1] Globally, and in most regions, hypertension is a leading cause of cardiovascular mortality and a significant contributor to preventable disease burden. In 2015, hypertension was implicated in 8.5 million deaths, with 88% of these occurring in low- and middle-income countries.[2] In Indonesia, the prevalence of hypertension among individuals aged ≥18 years reached 658,201, yet only approximately 54.4% of these patients adhere to regular medication regimens.[3] Arterial hypertension is broadly categorized into essential (80–90%) and secondary (10–20%) forms. The etiology of essential hypertension remains unknown but is associated with predisposing factors such as family history, genetic predisposition, smoking, hypercholesterolemia, obesity, and dietary habits, including high salt intake. In contrast, secondary hypertension typically arises from identifiable underlying conditions such as chronic kidney disease, renovascular disease, and endocrine disorders.[4]

Regardless of etiology, poorly controlled hypertension ultimately leads to target organ damage, affecting the brain (cerebrovascular disease), eyes (hypertensive retinopathy), and kidneys (hypertensive

Affiliation
[1]Master's Programme in Clinical Medicine, Universitas Prima Indonesia, Medan, Indonesia
[2]Department of Radiology, Universitas Prima Indonesia, Medan, Indonesia

Correspondence
fransiskus_aryo@yahoo.com

nephropathy).[1] Assessing target organ damage is crucial in the evaluation of hypertensive patients, as it provides vital information regarding hypertension severity and cardiovascular risk stratification. Common markers of target organ damage include renal function, albuminuria, and left ventricular hypertrophy (LVH).[5] Chest radiography is a rapid, cost-effective, and non-invasive tool commonly used to assess aortic arch calcification (AoAC) and the cardiothoracic ratio (CTR) in clinical practice. The CTR, first defined in 1919, has since been accepted as a measure of cardiac size and volume on posteroanterior chest radiographs (PA-CXR). A CTR <0.5 is considered normal, whereas a CTR >0.5 indicates cardiomegaly. The CTR serves as a key prognostic indicator in various conditions, including coronary artery disease, end-stage renal disease, and age-related conditions.[6] Higher CTR values are generally associated with a poorer prognosis, and longitudinal studies in healthy populations have reported a significant correlation between elevated CTR values and cardiovascular mortality.[7] Aortic arch calcification, as identified on chest radiographs, has been linked to pulse pressure, common carotid artery intima-media thickness, cardiovascular events, and an increased risk of cardiovascular mortality in the general population.[8]

Current hypertension guidelines do not recommend routine chest radiography in the evaluation of hypertensive patients.[1] However, for primary care practitioners, echocardiography is often not readily available, necessitating the use of simple and affordable tests to aid in the assessment of hypertensive patients. In this context, we aimed to evaluate the utility of the cardiothoracic ratio and aortic arch calcification in predicting a decline in estimated glomerular filtration rate (eGFR) as a marker of target organ damage, and to assess their correlation with systolic and diastolic blood pressure.

METHOD

This descriptive analytical study employed a cross-sectional design. Patient sampling was conducted retrospectively using consecutive sampling. The study was performed in the Radiology Division of Royal Prima Hospital, Medan. The study population comprised hypertensive patients who underwent examinations in the Radiology Division. The study sample included hypertensive patients referred to the Radiology Division who met the following inclusion criteria: age ≥ 40 years, a diagnosis of hypertension (systolic blood pressure ≥ 140 mmHg), completion of a posteroanterior (PA) chest radiograph, and availability of both creatinine and PA chest radiograph results obtained within a 24-hour period. Exclusion criteria were congenital heart disease, secondary mediastinal widening, pericardial effusion, pleural effusion, and pneumonia.

Hypertension was defined as a systolic blood pressure ≥ 140 mmHg, measured upon the patient's arrival for examination. Hypertension was classified as Stage 1 (≥ 140 mmHg) and Stage 2 (≥ 160 mmHg). The cardiothoracic ratio (CTR) was calculated from PA chest radiographs as the transverse diameter of the cardiac silhouette divided by the transverse thoracic diameter. CTR values of 0.5–0.55 were considered indicative of mild cardiomegaly, 0.55–0.6 as moderate cardiomegaly, and >0.6 as severe cardiomegaly. Aortic arch calcification was assessed on PA chest radiographs using a circumferential grading system: Score 0 indicated no calcification; Score 1, <50% circumferential involvement; Score 2, >50% circumferential involvement; and Score 3, full circumferential involvement.[9] The estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI creatinine formula.

Patient data, including age, sex, blood pressure, and complete blood count results, were retrieved from the electronic medical record system (Transmedic) at Royal Prima Hospital. Chest radiograph images for the same patients were obtained from the PACS application (ZettaPACS). All radiographic interpretations were independently validated by at least two specialist radiologists. Data were processed and analyzed using univariate and bivariate analyses. Pearson correlation was used for parametric data, and Spearman correlation for non-parametric data, to evaluate the relationships between CTR, aortic arch calcification, and eGFR. Statistical analyses were performed using SPSS version 23 for Windows, with a p-value < 0.05 considered statistically significant.

RESULTS

Table 1 summarizes the demographic and clinical characteristics of the 175 individuals included in the study. The majority of respondents (42.29%, n=74) were aged 50–59 years, followed by those aged 60–69 years (32.57%, n=57). The youngest age group, 40–49 years, comprised the smallest proportion (12.00%, n=21). Females constituted a larger proportion of the study population (65.71%, n=115) compared to males

Table 1. Respondent characteristic

Variable	Total (n= 175)	
	n	%
Age (years)		
40-49	21	12,00
50-59	74	42,29
60-69	57	32,57
Gender		
Male	60	34,29
Female	115	65,71
Hypertension		
Stage 1	71	40,57
Stage 2	104	59,43
CTR classification		
Normal	32	18,29
Mild cardiomegaly	61	34,86
Moderate cardiomegaly	52	29,71
Severe cardiomegaly	30	17,14
Aortic arch calcification		
None	67	38,29
Grade 1	78	44,57
Grade 2	25	14,29
Grade 3	5	2,86
CKD stage		
Stage 1	39	22,29
Stage 2	44	25,14
Stage 3A	32	18,29
Stage 3B	18	10,29
Stage 4	10	5,71
Stage 5	32	18,29

(34.29%, n=60). Regarding hypertension status, most participants (59.43%, n=104) were classified as having Stage 2 hypertension, while 40.57% (n=71) had Stage 1 hypertension.

Assessment of cardiac size using cardiothoracic ratio (CTR) classification revealed that 34.86% (n=61) of participants had mild cardiomegaly, 29.71% (n=52) had moderate cardiomegaly, and 17.14% (n=30) had severe cardiomegaly. A smaller proportion (18.29%, n=32) had a normal CTR. The prevalence of aortic arch calcification varied among respondents: 44.57% (n=78) exhibited Grade 1 calcification, 38.29% (n=67) had no calcification, while Grade 2 and Grade 3 calcification were observed in 14.29% (n=25) and 2.86% (n=5) of participants, respectively.

Regarding chronic kidney disease (CKD) stages, Stage 2 was most prevalent (25.14%, n=44), followed by Stage 1 (22.29%, n=39). Both Stage 3A and Stage 5 accounted for 18.29% (n=32) each. Stage 3B and Stage 4 were less common, comprising 10.29% (n=18) and 5.71% (n=10) of participants, respectively.

Respondents were then divided into six groups based on the severity of chronic kidney disease. The characteristics of each group were assessed, and one-way ANOVA was used to analyze trends in these characteristics across the groups.

eGFR (estimated Glomerular Filtration Rate), a key indicator of kidney function, consistently decreased with advancing CKD stages. In Stage 1, the average eGFR was 100.3(±8.6), which progressively dropped to 6.5(±3.2) in Stage 5. This trend is expected, as lower eGFR values signify more severe kidney impairment. The p-value for eGFR is not provided, but its clear progressive decline strongly suggests statistical significance. The average age of respondents ranged from 57.8(±8.6) years in Stage 1 to 62.0(±9.3) years in Stage 4, before slightly decreasing to 58.7(±8.1) years in Stage 5. However, the p-value of 0.567 indicates that there's no statistically significant difference in age across the various CKD stages.

Table 2. Participant characteristics by CKD stage

Characteristic	CKD Stage 1 (n=39)	CKD Stage 2 (n=44)	CKD stage 3A (n=32)	CKD stage 3B (n=18)	CKD stage 4 (n=10)	CKD stage 5 (n=32)	P
eGFR	100,3 (±8,6)	78,4 (±7,5)	54,6 (±4,1)	37,8 (±4,4)	22,1 (±4,1)	6,5 (±3,2)	-
Age (years)	57,8 (±8,6)	59,9 (±7,8)	60,6 (±9,1)	61,0 (±9,5)	62,0 (±9,3)	58,7 (±8,1)	0,567
Sex (M: male, F: female)	M: 16 F: 23	M: 2 F: 42	M: 17 F: 15	M: 7 F: 11	M: 5 F: 5	M: 13 F: 19	< 0,001
Systolic BP (mmHg)	154,4 (±10,8)	152,8 (±10,1)	159,1 (±11,0)	157,6 (±12,9)	157,9 (±12,0)	153,6 (±12,0)	0,161
Diastolic BP (mmHg)	85,4 (±12,3)	86,1 (±10,7)	91,1 (±11,6)	89,8 (±15,1)	90,4 (±10,9)	85,6 (±13,9)	0,298
Cardiothoracic Ratio (CTR)	52,3 (±5,4)	53,8 (±4,7)	56,2 (±6,2)	57,3 (±5,2)	55,8 (±5,5)	58,7 (±5,1)	< 0,001
Cardiomegaly (N: Normal, M: Mild, Mo: Moderate, S: Severe)	N: 13 M: 17 Mo: 5 S: 4	N: 9 M: 18 Mo: 15 S: 2	N: 7 M: 6 Mo: 13 S: 6	N: 2 M: 5 Mo: 5 S: 6	N: 0 M: 7 Mo: 0 S: 3	N: 1 M: 8 Mo: 14 S: 9	< 0,001
Aortic Arch Calcification (Grade)	Grade 0: 23 Grade 1: 14 Grade 2: 2 Grade 3: 0	Grade 0: 25 Grade 1: 14 Grade 2: 5 Grade 3: 0	Grade 0: 14 Grade 1: 13 Grade 2: 5 Grade 3: 0	Grade 0: 4 Grade 1: 10 Grade 2: 3 Grade 3: 1	Grade 0: 0 Grade 1: 7 Grade 2: 2 Grade 3: 1	Grade 0: 1 Grade 1: 20 Grade 2: 8 Grade 3: 3	< 0,001

Note: All values are expressed as mean unless otherwise specified.

Gender distribution showed a notable difference across stages, with a p-value of <0.001, indicating a statistically significant association between gender and CKD stage. In Stage 1, there were more females (23) than males (16). This pattern became more pronounced in Stage 2, with 42 females and only 2 males. However, in later stages (3A, 3B, 4, and 5), the distribution became more balanced, with a closer number of males and females, or even a slightly higher number of males in some cases (e.g., Stage 3A).

Systolic and Diastolic blood pressure generally remained stable across the stages. Systolic blood pressure averaged around 154.4(±10.8) to 159.1(±11.0) mmHg, with no statistically significant difference (p-value of 0.161). Similarly, diastolic blood pressure ranged from approximately 85.4(±12.3) to 91.1(±11.6) mmHg, also without a statistically significant difference (p-value of 0.298). Cardiothoracic Ratio (CTR), an indicator of heart size, showed a statistically significant increase with advancing CKD stages (p-value of <0.001). The average CTR rose from 52.3(±5.4) in Stage 1 to 58.7(±5.1) in Stage 5, suggesting a greater prevalence of cardiac enlargement as kidney disease progresses.

The presence and severity of cardiomegaly also demonstrated a statistically significant relationship with CKD stage (p-value of <0.001). While a higher proportion of respondents in earlier stages had normal heart size, there was a clear shift towards moderate (S) and severe (B) cardiomegaly in later stages. For instance, in Stage 4, no respondents had a normal heart size, and 3 out of 10 had severe cardiomegaly. In Stage 5, 9 out of 32 respondents had severe cardiomegaly.

Finally, aortic arch calcification showed a strong and statistically significant association with CKD progression (p-value of <0.001). In earlier stages (1 and 2), most respondents had no or mild calcification (T0 or T1). As CKD progressed, there was a notable increase in higher grades of calcification (T2 and T3). For example, in Stage 4, no respondents had T0 calcification, while in Stage 5, only 1 respondent had T0, with the majority showing T1, T2, or T3 calcification. This suggests that aortic arch calcification becomes more prevalent and severe with worsening kidney function.

Bivariate analysis was performed to examine the relationships between various variables and the estimated glomerular filtration rate (eGFR). Pearson's correlation coefficient was applied to parametric variables, whereas Spearman's rank correlation was used for nonparametric variables. The variables analy-zed included age, sex, systolic blood pressure, diastolic blood pressure, cardiothoracic ratio (CTR), and aortic arch calcification score. The results of the bivariate correlations with eGFR are summarized in Table 3.

Table 3. Bivariate correlation analysis with eGFR

Characteristic	Correlation Coefficient with eGFR	p
Age	-0,101	0,182
Sex	0,175	0,103
Systolic BP	-0,041	0,586
Diastolic BP	-0.037	0,625
CTR	-0,418	<0,001
Cardiomegaly	-0,398	<0,001
Aortic arch calcification score	-0,457	<0,001

The bivariate analysis revealed that CTR (correlation coefficient= -0.418; p < 0.001), cardiomegaly (correlation coefficient = -0.398; p < 0.001), and aortic arch calcification score (correlation coefficient = -0.457; p < 0.001) were significantly correlated (p < 0.05) with eGFR.

Subsequently, multivariate linear regression analysis was performed to evaluate the relationship between eGFR and the degree of cardiomegaly and aortic arch calcification score. The findings from this analysis are summarized in Table 4.

Table 4. Multivariate regression analysis with eGFR

Characteristic	Unstandardized Coefficient B with eGFR	p
CTR	-1,738 (CI -2,524 ~ -0,953)	<0,001
Aortic arch calcification score	-16,127 (CI -21,93 ~ -10,325)	<0,001

The multivariate analysis indicated that both CTR (unstandardized coefficient B = -1.738; 95% CI: -2.524 to -0.953) and aortic arch calcification score (unstandardized coefficient B = -16.127; 95% CI: -21.93 to -10.325) were significantly associated with eGFR (p < 0.05).

DISCUSSION

The baseline characteristics of the study respondents reflect a population with significant cardiovascular and renal comorbidities, making it an appropriate sample for investigating the relationships among cardiothoracic ratio (CTR), aortic arch calcification (AAC), and reduced estimated glomerular filtration rate (eGFR). The majority of respondents were older adults, consistent with the high prevalence of cardiovascular and renal issues in this age group.[10] There was a notable gender imbalance, with a predominance of female respondents. This aligns with findings from other studies indicating a higher risk of hypertension in women above a certain age, suggesting a potential role of gender in these conditions.[11] Advanced-stage hypertension was also frequently observed, underscoring the high cardiovascular risk profile of this respondent group.

Cardiomegaly, or an enlarged heart, was common among participants, presenting with varying degrees of severity. This highlights the relevance of CTR as an indicator of cardiac burden in this population. Aortic arch calcification was also evident in most participants, particularly in the early stages. This suggests that early-stage vascular calcification may have important clinical implications in renal pathophysiology, although the limited number of advanced calcification cases restricts the generalizability of these findings. The study included a wide spectrum of chronic kidney disease (CKD) severity, encompassing patients with end-stage renal disease. The majority of respondents exhibited moderate to severe eGFR reduction. This distribution strengthens the study's ability to investigate the relationship between CKD progression and cardiovascular markers such as CTR and AAC.

Further analysis stratified by CKD stage revealed significant trends in CTR, aortic arch calcification, and other characteristics, further elucidating their relationship with renal dysfunction. These findings support the concept that markers of cardiac and vascular compromise worsen with declining renal function.[12] The study demonstrated a progressive decline in kidney function (eGFR) from early to late stages of CKD. Age did not show a significant difference across CKD stages, suggesting that the underlying mechanisms linking cardiac and renal health in this population are not predominantly age-dependent. While age does influence renal function, other risk factors such as cardiovascular conditions, dietary patterns, and obesity appear to play a more substantial role in renal decline.[13,14] Gender distribution varied significantly, with a higher proportion of males observed in more advanced CKD stages, despite females constituting the majority of the overall study population. This indicates a potential gender-related difference in CKD progression risk. Other research suggests that women have a higher risk for CKD progression, irrespective of other risk factors, although the exact mechanisms remain unclear.[15]

CTR, cardiomegaly, and aortic arch calcification showed a significant association with CKD stage. The mean CTR increased as renal function declined, reflecting progressive cardiac enlargement. Similarly, the severity of aortic arch calcification increased with advancing CKD stage. In early CKD stages, many patients showed no calcification, whereas in advanced stages, advanced calcification was more common. These trends are consistent with previous research indicating that periaortic vascular calcification and increased CTR are associated with declining renal function, possibly through pathways such as chronic inflammation and hemodynamic stress.[16] Blood pressure, both systolic and diastolic, did not show significant variation across CKD stages. While chronic hypertension is a known risk factor for CKD progression, its long-term impact is more related to uncontrolled blood pressure and treatment resistance. Therefore, the absence of a direct association in this study may reflect differences in antihypertensive management within this population.[17,18]

The stratified analysis of mean eGFR by cardiomegaly classification indicated that eGFR values were progressively lower with increasing severity of cardiomegaly. This result suggests that cardiomegaly classification has the potential to predict CKD severity. Patients with moderate to severe cardiomegaly should be suspected of having CKD Stage 3A or higher, as the mean eGFR in these groups was below the threshold for CKD Stage 3A. Previous studies have not differentiated patients based on cardiomegaly classification but rather categorized its presence or absence.[16,19,20]

Both bivariate and multivariate statistical analyses reinforced the demographic trends of this population and provided strong evidence supporting the association between cardiothoracic ratio (CTR), aortic arch calcification (AAC), and reduced estimated glomerular filtration rate (eGFR). Bivariate analysis revealed a strong inverse correlation between eGFR and both CTR and AAC score, confirming that cardiovascular structural abnormalities increase with declining renal function. These results are consistent with the progressively worsening CTR and AAC observed across CKD stages. Interestingly, age, gender, and blood pressure (systolic/diastolic) did not show significant correlations with eGFR. This suggests that renal dysfunction is more closely linked to hemodynamic and structural changes in the heart and blood vessels, rather than to age and blood pressure alone. Other studies have also found that vascular calcification is a better predictor of renal function decline than age, with groups having vascular calcification experiencing significantly faster eGFR decline.[21] The gender disparity observed in the trend analysis (male dominance in advanced CKD) was not reflected in the correlation analysis, indicating that gender may not be as strong a factor as cardiovascular elements in influencing CKD progression, consistent with prior research.[22]

In multivariate regression, both CTR and AAC score proved to be strong predictors of eGFR. Negative coefficients indicated that each increase in CTR or AAC score correlated with a clinically significant decrease in eGFR. For instance, an increased CTR predicted a substantial eGFR decline, highlighting the profound impact of cardiac structural changes. Similarly, the large coefficient for AAC emphasized that vascular calcification is a major factor in renal impairment, possibly through mechanisms such as arterial stiffness, microvascular injury, or chronic inflammation.[23] These findings align with previous research linking cardiovascular factors (cardiomegaly and vascular calcification) to CKD.[19] Studies in advanced CKD patients have demonstrated a high prevalence of cardiomegaly and vascular calcification.[24] A prior cohort study associated increased CTR and aortic calcification with declining renal function and an elevated risk of cardiovascular mortality.[16] Other research has also found that in CKD patients, higher CTR and advanced aortic calcification increase the risk of cardiovascular mortality, underscoring the importance of these factors in CKD patient management.[25]

This study highlights the utility of CTR and AAC as readily available biomarkers for identifying high-risk CKD patients. Clinically, routine assessment of these markers via chest imaging could prompt earlier interventions. Scientifically, these findings hint at the critical role of cardiac structural changes and vascular calcification in precipitating permanent renal damage, potentially through pathways such as oxidative stress, endothelial dysfunction, or hemodynamic disturbances.

CONCLUSION

This study found a significant correlation between declining renal function (eGFR) and both cardiothoracic ratio (CTR) and aortic arch calcification (AAC) visible on chest imaging in a population of patients at RSU Royal Prima Medan. This strengthens the evidence that CTR and calcification on chest imaging are closely associated with eGFR decline. Clinically, monitoring these cardiovascular markers can serve as early warning signs for chronic kidney disease, thereby facilitating early intervention to ameliorate the vicious cycle between the heart and kidneys. Likewise, patients with chronic kidney disease may benefit from cardiovascular monitoring to mitigate further organ damage. Further research could be conducted longitudinally to determine whether the progression of CTR and AAC precedes or is a consequence of eGFR decline. Additionally, future studies could further explore the classification of cardiomegaly as a predictor of eGFR decline. For institutions, these research findings can serve as a basis for developing management guidelines related to CTR and aortic arch calcification.

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