International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Correlation of Renal Cortical Thickness, Glomerular Filtration Parameters, and Antihypertensive Drug Response in Patients with Essential Hypertension: A Cross-Sectional Observational Study

Dr Anagha A L[1], Dr Rinku Mariam George[2], Dr Abin Augustine[3]

[1]Assistant Professor, Department of Physiology, Azeezia Institute of Medical Sciences and Research, Meeyannoor, Kollam, Kerala, India

[2]Assistant Professor, Department of Anatomy, Azeezia Institute of Medical Sciences and Research, Meeyannoor, Kollam, Kerala, India

[3]Assistant Professor, Department of Pharmacology, Azeezia Institute of Medical Sciences and Research, Meeyannoor, Kollam, Kerala, India.

OPEN ACCESS

Corresponding Author:

Dr Anagha A L
Assistant Professor, Department of Physiology,
Azeezia Institute of Medical Sciences and Research,
Meeyannoor, Kollam, Kerala, India

Received: 20-12-2025
Accepted: 20-01-2026
Available online: 28-02-2026

ABSTRACT

Background: Essential hypertension contributes to early kidney injury. Renal cortical thickness (RCT) on ultrasonography reflects nephron mass and can complement estimated glomerular filtration rate (eGFR).
Objectives: To evaluate associations of RCT with glomerular filtration parameters and antihypertensive response markers in adults with essential hypertension.
Methods: This cross-sectional observational study was conducted in a tertiary-care hospital in Kerala, India (July–December 2025). One hundred adults underwent standardized blood pressure measurement, serum creatinine testing, eGFR estimation, and renal ultrasonography with bilateral RCT measurement. Pearson correlation assessed associations between mean RCT and renal/clinical variables. Group comparisons examined differences by blood pressure control, hypertension duration, and ACE inhibitor/ARB use.
Results: Mean age was 54.8 ± 9.6 years and 58% were men. Mean RCT was 8.7 ± 1.2 mm and mean eGFR was 78.4 ± 18.6 mL/min/1.73 m²; 62% had eGFR ≥90, 28% had 60–89, and 10% had 30–59. Mean RCT correlated positively with eGFR ($r = 0.61$, $p < 0.001$) and inversely with serum creatinine ($r = -0.54$, $p < 0.001$) and hypertension duration ($r = -0.39$, $p = 0.001$). Controlled blood pressure was associated with higher RCT (9.0 ± 1.1 vs 8.2 ± 1.2 mm) and higher eGFR (84.6 ± 15.9 vs 69.8 ± 19.7 mL/min/1.73 m²). RCT was lower with hypertension duration ≥10 years and higher among participants receiving ACE inhibitor/ARB therapy.
Conclusion: RCT showed a moderate association with eGFR and differentiated patients by blood pressure control and treatment patterns. Routine reporting of RCT can support early renal risk stratification in essential hypertension.

Keywords: Essential hypertension; Renal cortical thickness; Ultrasonography; Estimated glomerular filtration rate; Blood pressure control; ACE inhibitors.

INTRODUCTION

Hypertension remains one of the most prevalent noncommunicable diseases worldwide and is a leading contributor to cardiovascular and renal morbidity. Contemporary guidelines emphasize earlier detection, risk-based treatment intensification, and sustained blood pressure control to prevent end-organ damage [1,2]. Kidney involvement in essential hypertension often begins silently, with microvascular remodeling, glomerulosclerosis, and tubulointerstitial ischemia that progress over years before a clinically apparent decline in filtration becomes evident [3]. In clinical practice, renal function monitoring typically relies on serum creatinine and estimated glomerular filtration rate (eGFR), yet these functional indices can lag behind structural injury, and creatinine is influenced by age, sex, and muscle mass [4,5].

Early identification of hypertensive kidney injury is important for risk stratification and therapeutic planning. The KDIGO framework highlights eGFR-based staging for prognosis and follow-up intensity, and recommends integrating clinical context to guide management [6-8]. In parallel, KDIGO blood pressure guidance in CKD supports standardized measurement and tighter systolic targets in appropriate patients to reduce adverse outcomes [3]. Even among individuals without established CKD, early recognition of declining renal reserve can strengthen counseling, adherence support, and rational selection of antihypertensive agents.

Renal ultrasonography is widely accessible, inexpensive, and free of ionizing radiation. Traditional ultrasound descriptors such as renal length and parenchymal echogenicity have been used to support CKD diagnosis and chronicity, but their correlation with renal function varies [10]. Increasing evidence suggests that renal cortical thickness (RCT) is more closely related to filtration capacity than renal length, because the cortex contains the glomeruli and the bulk of proximal tubular mass [6]. Studies in CKD cohorts have reported strong positive relationships between cortical thickness and eGFR and have advocated incorporating RCT into routine reports, alongside other sonographic predictors [6-9].

Beyond diagnosis, ultrasound-derived cortical metrics could help contextualize treatment response in hypertension. Renin–angiotensin–aldosterone system (RAAS) blockade with ACE inhibitors or angiotensin receptor blockers is a cornerstone of nephroprotection in several high-risk settings, with effects that extend beyond blood pressure reduction [12]. Whether current treatment patterns and blood pressure control are reflected in cortical thickness among adults with essential hypertension is clinically relevant, particularly in resource-constrained environments where repeated biochemical monitoring is not always feasible.

The present study was designed to correlate RCT with glomerular filtration parameters and clinically relevant hypertension features. The objectives were (i) to describe renal cortical thickness and eGFR distribution among adults with essential hypertension, (ii) to quantify the correlation between mean RCT and eGFR, serum creatinine, and hypertension duration, and (iii) to compare RCT and eGFR across subgroups defined by blood pressure control status, hypertension duration (<10 vs ≥10 years), and use of RAAS blockade (ACE inhibitor/ARB).

MATERIALS AND METHODS

Study design and setting: This cross-sectional observational study was conducted at Azeezia Institute of Medical Sciences and Research, Kollam, Kerala, India, over 6 months (July 2025 to December 2025). The report was prepared in accordance with STROBE guidance for observational studies .

Participants and sampling: Adults (≥18 years) with a clinician diagnosis of essential hypertension attending outpatient or inpatient services during the study period were screened. A purposive sampling approach was used to enroll 100 eligible participants. Individuals with suspected secondary hypertension, pregnancy, acute kidney injury, known structural renal anomalies, history of renal transplantation, or ongoing renal replacement therapy were excluded. Written informed consent was obtained prior to data collection.

Blood pressure assessment and clinical variables: Blood pressure was measured using a calibrated automated device with an appropriately sized cuff. After at least 5 minutes of seated rest, two readings were recorded 1–2 minutes apart; the average was used for analysis, consistent with guideline recommendations [1,2]. Blood pressure control was defined as <140/90 mmHg, and uncontrolled blood pressure as ≥140/90 mmHg. Duration of hypertension (years) and current antihypertensive prescriptions were documented from medical records and participant interview. Exposure to RAAS blockade was defined as current use of an ACE inhibitor or angiotensin receptor blocker (ARB).

Laboratory measurements and eGFR: Venous blood was collected for serum creatinine estimation using the hospital laboratory's standard enzymatic method. eGFR was calculated using the CKD-EPI creatinine equation [5]. eGFR categories (≥90, 60–89, and 30–59 mL/min/1.73 m²) were defined following CKD staging concepts [14].

Renal ultrasonography and cortical thickness measurement: Renal ultrasonography was performed using a curvilinear 3.5–5 MHz transducer by an experienced radiologist blinded to biochemical results. Both kidneys were scanned in longitudinal and transverse planes. Cortical thickness was measured as the distance from the renal capsule to the base of the medullary pyramid at the mid-pole, avoiding areas with focal scarring; three measurements per kidney were averaged to obtain right and left renal cortical thickness. Mean renal cortical thickness (mean RCT) was calculated as the average of right and left values, consistent with prior sonographic methodology [6-9].

Outcomes and statistical analysis: The primary outcome was the correlation between mean RCT and eGFR. Secondary analyses examined correlations of mean RCT with serum creatinine and hypertension duration, and compared mean RCT and eGFR between predefined subgroups (controlled vs uncontrolled blood pressure; <10 vs ≥10 years hypertension duration; ACE inhibitor/ARB use vs non-use). Continuous variables were summarized as mean ± standard deviation (SD)

and categorical variables as frequency (%). Pearson correlation coefficients (r) were calculated. Independent-samples t-tests compared group means. A two-sided p-value <0.05 was considered statistically significant. Analyses were performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA).

Ethical considerations: Institutional Ethics Committee approval was obtained prior to study initiation. Confidentiality was maintained by de-identifying study data and restricting access to the research team.

RESULTS:

A total of 100 adults with essential hypertension were included in the analysis. Baseline characteristics are summarized in Table 1. The mean age was 54.8 ± 9.6 years, and men constituted 58% of the cohort. The mean duration of hypertension was 7.2 ± 4.1 years. Blood pressure was controlled (<140/90 mmHg) in 64% and uncontrolled in 36% of participants.

Table 1. Baseline characteristics of the study population (n = 100).

Variable	Value
Age (years), mean ± SD	54.8 ± 9.6
Sex, n (%)	Male 58 (58); Female 42 (42)
Duration of hypertension (years), mean ± SD	7.2 ± 4.1
BP controlled (<140/90 mmHg), n (%)	64 (64)
BP uncontrolled (≥140/90 mmHg), n (%)	36 (36)

Renal cortical thickness and renal function parameters are presented in Table 2. Mean renal cortical thickness was 8.7 ± 1.2 mm, with comparable right and left values. Mean eGFR was 78.4 ± 18.6 mL/min/1.73 m²; 62% of participants had eGFR ≥90, 28% had 60–89, and 10% had 30–59 mL/min/1.73 m².

Table 2. Renal cortical thickness and renal function parameters (n = 100).

Parameter	Mean ± SD / n (%)
Right renal cortical thickness (mm)	8.6 ± 1.3
Left renal cortical thickness (mm)	8.8 ± 1.1
Mean renal cortical thickness (mm)	8.7 ± 1.2
eGFR (mL/min/1.73 m²)	78.4 ± 18.6
eGFR category ≥90, n (%)	62 (62)
eGFR category 60–89, n (%)	28 (28)
eGFR category 30–59, n (%)	10 (10)

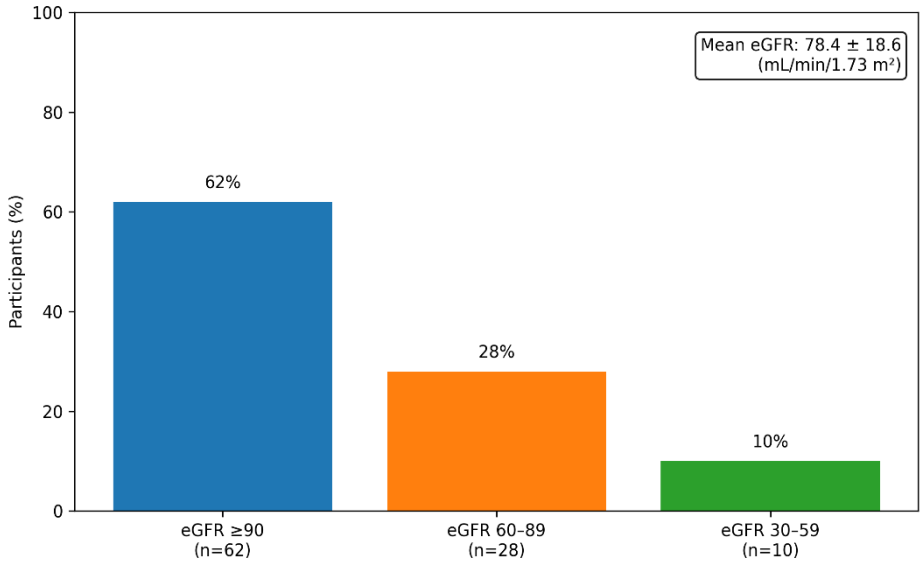

Figure 1: eGFR Distribution and Mean eGFR

Correlation analyses (Table 3) demonstrated a significant positive association between mean renal cortical thickness and eGFR ($r = 0.61$, $p < 0.001$). Mean cortical thickness showed a significant inverse relationship with serum creatinine ($r = -0.54$, $p < 0.001$) and duration of hypertension ($r = -0.39$, $p = 0.001$).

Table 3. Correlation between renal cortical thickness and clinical/renal variables (n = 100).

Variable correlated with mean RCT	Correlation coefficient (r)	p-value
eGFR	0.61	<0.001
Serum creatinine	–0.54	<0.001
Duration of hypertension	–0.39	0.001

Subgroup comparisons are shown in Table 4. Participants with controlled blood pressure exhibited significantly higher mean cortical thickness (9.0 ± 1.1 mm) than those with uncontrolled blood pressure (8.2 ± 1.2 mm; p = 0.004), and also had higher mean eGFR (84.6 ± 15.9 vs 69.8 ± 19.7 mL/min/1.73 m²; p < 0.001). Mean cortical thickness was lower among participants with hypertension duration ≥10 years compared with <10 years (8.1 ± 1.0 vs 9.0 ± 1.1 mm; p = 0.002). Individuals receiving ACE inhibitor/ARB therapy had higher mean cortical thickness than those not receiving RAAS blockade (8.9 ± 1.1 vs 8.3 ± 1.2 mm; p = 0.01).

Table 4. Renal cortical thickness and eGFR by blood pressure control, hypertension duration, and RAAS blockade (n = 100).

Subgroup	Category	n	Mean RCT (mm), mean ± SD	eGFR (mL/min/1.73 m²), mean ± SD	p-value (RCT; eGFR)
Blood pressure control	Controlled (<140/90 mmHg)	64	9.0 ± 1.1	84.6 ± 15.9	0.004; <0.001
Blood pressure control	Uncontrolled (≥140/90 mmHg)	36	8.2 ± 1.2	69.8 ± 19.7	—
Hypertension duration	<10 years	72	9.0 ± 1.1	—	0.002
Hypertension duration	≥10 years	28	8.1 ± 1.0	—	—
RAAS blockade	On ACE inhibitor/ARB	66	8.9 ± 1.1	—	0.01
RAAS blockade	Not on ACE inhibitor/ARB	34	8.3 ± 1.2	—	—

DISCUSSION

This study demonstrates that renal cortical thickness (RCT) measured on routine ultrasonography is meaningfully aligned with renal function in adults with essential hypertension. The observed positive correlation between mean RCT and eGFR (r = 0.61) supports the biological premise that cortical thickness reflects functioning nephron mass. Beland and colleagues reported that cortical thickness was more closely related to eGFR than renal length in CKD and suggested routine inclusion of cortical thickness in ultrasound reporting [6]. Subsequent work has reinforced this concept; Korkmaz et al. found a particularly strong relationship between cortical thickness and eGFR, exceeding the correlation observed with renal length [7]. In our cohort, the correlation was moderate rather than very strong, which is plausible because many participants had preserved eGFR and earlier hypertensive renal involvement rather than advanced CKD.

The inverse association between cortical thickness and serum creatinine further indicates that structural cortical loss tracks functional decline even within a cross-sectional snapshot. Studies in newly diagnosed or mixed-etiology CKD populations have similarly shown that cortical thickness correlates with filtration measures and can complement standard sonographic descriptors [8,9]. Ahmed et al. emphasized the diagnostic value of sonographic markers in CKD and highlighted that multiple ultrasound parameters should be interpreted together [10]. In hypertension clinics, adding RCT provides an additional, easily captured metric that does not require new equipment or patient preparation.

Clinically, blood pressure control status differentiated both RCT and eGFR in this study. Participants with controlled blood pressure had thicker cortex and higher eGFR than those with uncontrolled readings, aligning with guideline priorities that emphasize sustained control to prevent kidney damage [11]. KDIGO guidance also underscores standardized blood pressure measurement and intensive control strategies in suitable CKD populations to reduce adverse renal and cardiovascular outcomes [3]. Although causality cannot be inferred, the observed pattern is consistent with the concept that persistent pressure load accelerates nephrosclerosis and cortical thinning over time. This interpretation is supported by the negative correlation between RCT and hypertension duration, and by the lower RCT among participants with hypertension duration ≥10 years.

In this study, it was observed that higher cortical thickness among individuals receiving ACE inhibitor/ARB therapy. RAAS inhibition is recognized for renoprotective effects that extend beyond blood pressure reduction, particularly through reductions in intraglomerular pressure and proteinuria [12]. In cross-sectional data, this association can also reflect confounding by indication, treatment access, or clinician preference for RAAS blockade in patients perceived to be at renal risk. Even so, the finding reinforces the clinical relevance of documenting current antihypertensive class when interpreting renal ultrasound metrics. Taken together, our results support a pragmatic approach: combine eGFR-based staging with structural RCT assessment, consistent with CKD evaluation principles, to strengthen early risk stratification in essential hypertension [13,14].

LIMITATIONS

This single-center study with purposive sampling limits external validity and introduces selection bias. Antihypertensive exposure was captured from current prescriptions without objective adherence measurement or duration of therapy. Renal ultrasonography is operator dependent, and inter-observer reliability was not assessed. Albuminuria, renal Doppler indices, and detailed comorbidity profiles were not systematically recorded, restricting mechanistic interpretation. The short study window prevented longitudinal assessment of cortical thickness and eGFR trends.

CONCLUSION

In this hospital-based cohort of adults with essential hypertension, mean renal cortical thickness on ultrasonography demonstrated a moderate positive correlation with eGFR and a clear inverse relationship with serum creatinine and hypertension duration. Patients with controlled blood pressure exhibited thicker renal cortex and better filtration indices than those with uncontrolled readings, and cortical thickness was lower after a decade of hypertension. Participants receiving ACE inhibitor/ARB therapy also showed higher cortical thickness, supporting the clinical relevance of RAAS-focused regimens. Routine documentation of renal cortical thickness during ultrasound, alongside eGFR reporting, offers a practical approach to early renal risk stratification and can help refine follow-up intensity in hypertensive care pathways in practice.

REFERENCES

1. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2018 Oct 23;138(17):e484-e594. doi:10.1161/CIR.0000000000000596. PMID:30354654.
2. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018 Sep 1;39(33):3021-3104. doi:10.1093/eurheartj/ehy339. PMID:30165516.
3. Kidney Disease: Improving Global Outcomes (KDIGO) Blood Pressure Work Group. KDIGO 2021 Clinical Practice Guideline for the Management of Blood Pressure in Chronic Kidney Disease. Kidney Int. 2021 Mar;99(3S):S1-S87. doi:10.1016/j.kint.2020.11.003. PMID:33637192.
4. Stevens PE, Levin A; Kidney Disease: Improving Global Outcomes Chronic Kidney Disease Guideline Development Work Group Members. Evaluation and management of chronic kidney disease: synopsis of the kidney disease: improving global outcomes 2012 clinical practice guideline. Ann Intern Med. 2013 Jun 4;158(11):825-830. doi:10.7326/0003-4819-158-11-201306040-00007. PMID:23732715.
5. Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF 3rd, Feldman HI, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 2009 May 5;150(9):604-612. doi:10.7326/0003-4819-150-9-200905050-00006. PMID:19414839.
6. Beland MD, Walle NL, Machan JT, Cronan JJ. Renal cortical thickness measured at ultrasound: is it better than renal length as an indicator of renal function in chronic kidney disease? AJR Am J Roentgenol. 2010 Aug;195(2):W146-W149. doi:10.2214/AJR.09.4104. PMID:20651174.
7. Korkmaz M, Aras B, Güneyli S, Yılmaz M. Clinical significance of renal cortical thickness in patients with chronic kidney disease. Ultrasonography. 2018 Jan;37(1):50-54. doi:10.14366/usg.17012. PMID:28618770.
8. Garg A, Jhobta A, Kapila S, Rathour D. Correlation of Sonographic Parameters with Renal Function in Patients with Newly Diagnosed Chronic Kidney Disease. J Ultrason. 2022 Oct 1;22(91):e216-e221. doi:10.15557/jou.2022.0036. PMID:36483784.
9. Gupta P, Chatterjee S, Debnath J, Nayan N, Gupta SD. Ultrasonographic predictors in chronic kidney disease: A hospital based case control study. J Clin Ultrasound. 2021 Sep;49(7):715-719. doi:10.1002/jcu.23026. PMID:34085292
10. 10.Ahmed S, Bughio S, Hassan M, Lal S, Ali M. Role of Ultrasound in the Diagnosis of Chronic Kidney Disease and its Correlation with Serum Creatinine Level. Cureus. 2019 Mar 12;11(3):e4241. doi:10.7759/cureus.4241. PMID:31131164.

11. Horowitz B, Miskulin D, Zager P. Epidemiology of hypertension in CKD. Adv Chronic Kidney Dis. 2015 Mar;22(2):88-95. doi:10.1053/j.ackd.2014.09.004. PMID:25704344.
12. Berl T. Review: renal protection by inhibition of the renin-angiotensin-aldosterone system. J Renin Angiotensin Aldosterone Syst. 2009 Mar;10(1):1-8. doi:10.1177/1470320309102747. PMID:19286752.
13. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007 Oct 20;370(9596):1453-1457. doi:10.1016/S0140-6736(07)61602-X. PMID:18064739.
14. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024 Apr;105(4S):S117-S314. doi:10.1016/j.kint.2023.10.018. PMID:38490803