

Intra-Renal Arterial Resistance and its Association with Different Stages of Chronic Kidney Disease; A Cross Sectional Study

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ABSTRACT

Background

This study was conducted to analyze the association between intrarenal arterial resistive index (RI) measured using renal duplex ultrasonography and different stages of chronic kidney disease (CKD), thereby assessing its ability to predict the severity of CKD.

Methods

This was a hospital-based cross sectional analysis conducted among 100 patients (age >18 years) diagnosed to have CKD at the National Kidney Foundation, Government Medical College, Thrissur, from August 2017 to June 2019 after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

Results

The mean RI measured was 0.53, 0.56, 0.66, 0.74 and 0.91 from stage 1 to stage 5 respectively. A positive correlation was found between rising RI and the progression of CKD stages, and the association was found to be statistically significant ($p < 0.001$).

Conclusion

RI can be used as a valuable sonological marker in predicting the severity of CKD, thereby helping in the early detection of high-risk patients.

Keywords: Intra-Renal Arterial Resistance, Association, Different Stages of Chronic Kidney Disease.

INTRODUCTION

Chronic kidney disease (CKD) is a worldwide public health problem, both in terms of the number of patients and the cost of treatment involved. Globally, CKD is the 12th cause of death and the 17th cause of disability respectively. The approximate prevalence of CKD in India is 800 per million and the incidence of end stage renal disease is 150-200 per million populations.^[1] CKD is defined as kidney damage >3 months, as defined by structural or functional abnormalities of the kidney with or without decreasing GFR, manifested by either pathological abnormalities or markers of kidney damage including abnormalities in the composition of blood or urine or abnormalities in the imaging tests. 2. GFR <60 mL/min/1.73 m² for >3 months with or without kidney damage.^[2] Diabetes and hypertension cause up to two-thirds of CKD^[3] Less common causes include glomerulonephritis, nephrolithiasis, and polycystic kidney disease. In a small proportion of cases, progressive kidney damage leads to end-stage renal disease (ESRD). ESRD patients require dialysis or kidney transplantation to survive. The rate of CKD progression varies between patients depending on disease etiology and pathology.^[4,5] Ultrasound is the ideal imaging modality in CKD because of its non-invasiveness and because it provides easy accessibility and visualization of the kidneys. It can be done

at the bedside to provide clinicians with important anatomical details of the kidney with low interobserver variability.^[6] The safety of the diagnostic procedure using ultrasound is well established. Sonography identifies the renal length, thickness, and echogenicity of renal parenchyma apart from its importance in detailing a dilated collecting system.^[7] These details assist in identifying the extent of the renal parenchymal damage^[8], the possibility of its reversibility and the decision to perform a renal biopsy.^[9] According to a study, abnormal sonographic findings were seen in 67% of cases of CKD.^[10] Renal doppler ultrasonography can detect not only renal macro abnormalities but also changes in the renal vasculature and blood flow. The RI (Resistive Index) is commonly used as an index of intra renal arterial resistance. Progressive chronic renal failure is believed to result in a reduction in the number and area of post glomerular capillaries. Renal scarring ultimately leads to a reduction in the intrarenal vessel area which in turn may be responsible for an increased intra renal vascular resistance. RI increases in various kidney diseases^[11-19] and previous studies have shown the association of RI with renal function and patient prognosis.^[20-26] The relationship between renal histological changes in CKD and RI has been investigated previously. Glomerulosclerosis,^[27] tubulointerstitial damage^[28] and vascular lesions have been reported to correlate with an increase in RI. The serum creatinine level is an endogenous marker that is commonly used to estimate GFR and accordingly the stage of CKD. eGFR derived from the formula such as the MDRD (Modification of Diet in Renal Disease) equation is superior to serum creatinine alone in the diagnosis of CKD.^[29] By calculating eGFR the reserved renal function and in turn the severity of the patient can be evaluated.^[30]

There are studies showing changes in renal echogenicity with different stages of CKD, but there is a scarcity of literature regarding the variation of RI in different stages of CKD. Our study was conducted to find out if there is a significant link between the resistive index in renal duplex ultrasonography and different stages of CKD and if this parameter can be used to measure the severity of CKD.

Aims and Objectives

To analyze the association between intrarenal arterial resistive index (RI) measured using renal duplex ultrasonography and different stages of chronic kidney disease (CKD), thereby assessing its ability to predict the severity of CKD.

METHODS

This was a hospital-based cross sectional analysis study conducted among 100 patients (age >18 years) diagnosed to have CKD at the National Kidney Foundation, Government Medical College, Thrissur, from August 2017 to June 2019 after obtaining clearance from the institutional ethics committee and written informed consent from the study participants.

Inclusion Criteria

The ethical committee of our institution approved this cross-sectional study. According to the National Kidney Foundation's standards, 100 people over the age of 18 who were clinically diagnosed with CKD (calculated by using the Cockcroft-Gault equation) between August 2017 and June 2019 at Government Medical College, Thrissur, were included in the study.

Exclusion Criteria

Patients with hepatic illness diagnosed by ultrasonography, those receiving hemodialysis, peritoneal dialysis, renal transplant patients, patients with renal tumours (both primary and secondary), and patients with hepatic disease. The detailed medical history of patients, including their age, duration of diabetes mellitus if diabetic, duration of hypertension if hypertensive, other causes of chronic renal failure, and treatment history. The most recent serum creatinine values were noted.

Statistical Methods

Data was entered in MS Excel and analyzed using SPSS software. Results were presented as tables.

RESULTS

H/O	No. of Cases	%
DM	43	43.0
HT	26	26.0
DM, HT	18	18.0
None	13	13.0
Total	100	100.0

Table 1: Medical History

The most commonly known cause of CKD in these patients was diabetes mellitus, which was seen in 43 cases (43%) followed by hypertension in 26 cases (26%), and diabetes and hypertension together in 18% of the cases. No provisional cause was made in 13% of the cases.

CKD Stage	Mean RI	SD	Min	Max	Anova F	P-Value
1	0.53	0.05	0.48	0.68	246.24	< 0.001, HS
2	0.56	0.06	0.48	0.69		
3	0.66	0.04	0.5	0.7		
4	0.74	0.04	0.64	0.79		
5	0.91	0.03	0.86	0.96		
Total	0.68	0.14	0.48	0.96		

One Way ANOVA

Stage Compared	Mean Diff.	P-Value
1-2	0.03	0.45, ns
1-3	0.13	0.00**
1-4	0.20	0.00**
1-5	0.38	0.00**
2-3	0.10	0.00**
2-4	0.18	0.00**
2-5	0.35	0.00**
3-4	0.08	0.00**
3-5	0.25	0.00**
4-5	0.17	0.00**

Post-hoc Tukey's Test

Table 2: Resistive Index in Relation to CKD and Stage Wise Comparison

The mean RI values were 0.53 for stage 1 CKD, 0.56 for stage 2, 0.66 for stage 3, 0.74 for stage 4 and 0.91 for stage 5. A statistically significant positive correlation was seen between the CKD stages and the resistive index. As the CKD stage advanced there was an increase in the resistive index ($p < 0.001$). Between CKD stages 1 and 2, no significant change in RI was observed ($p = 0.45$).

CKD Stage	Mean Length (cm)	SD	Min	Max	Anova F	P-Value
1	9.59	0.69	8.3	11	87.48	< 0.001, HS
2	9.41	0.63	8.3	11		
3	8.81	0.62	7.4	9.7		
4	8.86	0.67	7.4	7.3		
5	6.26	0.58	5.2	7.3		
Total	8.54	1.36	5.2	11		

One Way ANOVA

Stages Compared	Mean Diff.	P-Value
1-2	0.03	0.91, ns
1-3	0.13	0.002*
1-4	0.20	0.00**
1-5	0.38	0.00**

2-3	0.10	0.03**
2-4	0.18	0.005**
2-5	0.35	0.00**
3-4	0.08	0.94. ns
3-5	0.25	0.00**
4-5	0.17	0.00**

Post-hoc Tukey's Test

Table 3: Renal length in Relation to CKD Stages and Stage Wise Comparison

The mean renal lengths were 9.59cm for stage 1 CKD, 9.4 cm for stage 2, 8.8cm for stage 3, 8.6cm for stage 4 and 6.26cm for stage 5. The average kidney length measured in the present study was 8.5cm (range = 5.2cm to 11cm; SD=1.36 cm). The kidneys were small in all patients with stage 5 CKD. A statistically significant positive correlation was observed between CKD stage and mean renal length ($p < 0.001$). As the stage increased, there was a reduction in renal length. The size of the kidney was significantly reduced in patients with stage 5 CKD compared with other stages. However, no statistically significant reduction in renal length occurred as the CKD stage advanced from stage 1 to stage 2 and stage 3 to stage 4.

CKD Stage	Mean PT (mm)	SD	Min	Max	Anova F	P-Value
1	16.99	0.81	15.0	18.0	12.06	< 0.001, HS
2	16.56	1.07	14.0	18.0		
3	15.15	1.04	13.0	16.0		
4	14.91	2.02	10.0	18.0		
Total	15.90	1.57	10.0	18.0		

One Way ANOVA

Stages Compared	Mean Diff.	P-Value
1-2	0.43	0.73, ns
1-3	1.84	0.00**
1-4	2.08	0.00**
2-3	1.41	0.007**
2-4	1.65	0.002**
3-4	0.25	0.94. ns

Post-hoc Tukey's Test

Table 4: Parenchymal Thickness Interrelation to CKD Stages and Stage Wise Comparisons

The mean parenchymal thickness obtained in the present study was 15.9mm (range=10mm to 18mm, SD=1.57mm). A statistically significant positive correlation was observed between CKD stage and parenchymal thickness ($p < 0.001$). As the stage of CKD advanced, there was a decrease in the parenchymal thickness. However, no statistically significant reduction in parenchymal thickness occurred as the CKD stage advanced from stage 1 to stage 2 and stage 3 to stage 4.

CKD Stage	Mean CT (mm)	SD	Min	Max	Anova F	P-Value
1	6.92	0.69	6.0	8.0	6.73	< 0.001, HS
2	7.44	0.94	6.0	9.0		
3	6.65	1.02	5.0	8.7		
4	6.17	0.96	4.0	8.0		
Total	6.79	1.00	4.0	9.0		

One Way ANOVA

Stages Compared	Mean Diff.	P-Value
1-2	0.43	0.28, ns
1-3	1.84	0.78, ns
1-4	2.08	0.05*
2-3	1.41	0.04*

2-4	1.65	0.00**
3-4	0.25	0.36, ns
<i>Post-hoc Tukey's Test</i>		
Table 5: Cortical Thickness in Relation to CKD Stages and Stage Wise Comparisons		

The mean cortical thickness obtained in the present study was 6.79mm (range: 4mm to 9mm, SD=1mm). A statistically significant positive correlation was observed between CKD stage and cortical thickness ($p < 0.001$). As the stage of CKD advanced, there was a decrease in cortical thickness. However, no statistically significant reduction in parenchymal thickness occurred as the CKD stage advanced from stage 1 to stage 2 and stage 3 to stage 4.

DISCUSSION

The burden of CKD has dramatically increased and is consuming the resources of both developed and developing economies. For this reason, efforts to reduce the cost of managing this disease are always appreciated.

In the present study, we evaluated the correlation between various ultrasonographic indices such as renal echogenicity, renal length, renal parenchymal thickness, cortical thickness and resistive index with various stages of CKD.

RI in CKD patients is considered as a marker of renal dysfunction. It has been reported that RI can be a useful predictor of the progression of renal dysfunction.^[31,32] RI can be increased by extrinsic factors such as kidney compression, breath holding during the Valsalva maneuver and extreme bradycardia. RI values are also correlated with extrarenal markers of vascular stiffness, indicating that RI might not be an ideal test for renal disease.^[33] Nevertheless, RI can provide diagnostic information for several renal diseases. A RI value exceeding 0.80 is associated with a reduced likelihood of improved renal function after the correlation of renal artery stenosis.^[34] A RI value exceeding 0.80 is also associated with poor allograft survival after renal transplantation.^[35] RI increases in diabetic nephropathy when the kidneys start to shrink and microalbuminuria occurs.^[36] RI decreases with the use of RAS inhibitors in diabetic nephropathy and hypertensive nephrosclerosis, explaining why these drugs are Reno protective. In the present study, we demonstrated that the progression of CKD could be predicted by an RI value. A study done by Parolin et al. showed that an RI of 0.70 or higher is predictive of an unfavorable outcome in patients with CKD. A clinical study done by Hanamura et al. showed that CKD patients with a high normal range RI (0.65-0.70) were at risk for an adverse prognosis.^[37] The mean RI measured in the present study was 0.68 (range: 0.48-0.96, SD = 0.14). This study showed a statistically significant increase in RI as the CKD stage advanced (p -value<.001, HS). This finding is consistent with those of Hanamura et al. who found that RI was the best marker of CKD stage among the ultrasonographic indices and showed that RI increases with CKD stage.

Patients with end-stage kidney disease may have bilateral shrunken kidneys, but at early stages of CKD the kidney length may be within the normal range. Renal length is measured as the longest diameter obtained on a posterior oblique image with a lower limit of normality generally indicated as 9cm. According to Fiorini and Barizzi, renal length under 8cm is definitely reduced and should be attributed to CKD, whereas length between 8 and 9 cm should always correlate to the patient's phenotype particularly height.^[38] The mean renal length measured in the present study was 8.54cm. Patients with stage 5 CKD had small kidneys with a mean length of 6.26+/- 0.58. In the remaining stages the kidneys were of normal size. This correlated well with the finding of Yamashita et al. in which the average renal length was 9.5cm in CKD patients (range;6.99 cm to 13cm). Renal length has traditionally been considered a surrogate marker of renal function. A statistically significant reduction in renal length occurred as the stage of CKD increased. This finding contradicts those of Sidappa et al. and Moccise et al. which showed no significant correlation between renal length and serum creatinine.^[39,40] Additionally, a study by Hanamura et al. showed that there is no established normal range of cortical thickness. A normal range of 8 to 11.5 mm was reported in a small study of transplant donors by Raj et al.^[41] However, El-Reshaid et al. stated that cortical thickness values up to 6mm are also considered normal.^[42] The mean cortical thickness in our study group was found to be 6.79mm (range: 6mm to 8mm). Cortical thickness could not be assessed in stage 5 CKD patients as the renal pyramids could not be identified in USG. In a study conducted by Beland et al.^[43] it was

reported that cortical thickness measured using US was related to eGFR. Those authors measured the RCT and the length of the kidneys in 25 patients with CKD and found that the RCT was more significantly related to eGFR than was the renal length.

Normal parenchymal thickness ranges from 1.5 - 2 cm. The mean parenchymal thickness obtained in the present study was 15.9mm (range: 1cm to 2.35cm; SD=0.3 cm). The average parenchymal thickness was normal in 75% of the cases. In 18% of the cases it was reduced, and in 7% of the cases, it could not be assessed as the cortico-medullary differentiation was lost. These findings correlated well with those of Moghazi et al. who found the mean parenchymal thickness to be 1.71 cm (range: 0.7 cm to 3.3 cm).

Raised renal cortical echogenicity was reported in patients with CKD in this study. Similar echogenicity changes were observed in both kidneys in all cases. Paivansalo et al. also reported that an echogenic cortex was the most common abnormality detected in CKD patients. In the present study, echogenicity was further graded according to the classification proposed by Sidappa et al. 11 cases had grade 0 echogenicity, 25 cases had grade 1 echogenicity, 27 cases had grade 2 echogenicity, 21 cases had grade 3 echogenicity and 16 cases had grade 4 echogenicity. Thus, grade 2 echogenicity had the maximum number of cases. These findings are slightly different from those of Sidappa et al., who found grade 1 echogenicity to be the largest group with 48.3% of the cases in it. Also, corticomedullary differentiation was maintained in 63% of the cases, poorly maintained in 21% of the cases and lost in 16% of the cases. This finding closely resembles the study done by Arvinder Singh et al., who had 77% of cases with maintained corticomedullary differentiation, 16% of cases with poorly maintained corticomedullary differentiation and 7% of cases where the corticomedullary differentiation was lost.

CONCLUSION

RI can be used as a valuable sonological marker in predicting the severity of CKD, thereby helping in the early detection of high-risk patients.

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