

Effect of hypertension on kidney Volume: A Case-control Study

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Abstract: Background: According to world health organization (WHO) blood pressure is the force exerted by circulating blood against the walls of the body's arteries, the major blood vessels in the body. According to the WHO, hypertension is a major risk factor for cardiovascular disease (CVD, excluding congenital CVD) as well as renal impairment, peripheral vascular disease, and blindness. **Material and Method:** This is a prospective, descriptive, cross sectional and observational study. Department of Physiology, General Medicine and Radiology, Index Medical College, Hospital and Research center Indore. A curvilinear probe with transducer frequency of 2–8 MHz of a Sonoace X6 ultrasound machine was used. Each individual was laid supine on the couch with the abdomen adequately exposed from upper abdomen to the symphysis pubis. **Result:** The mean right renal cortical thickness in case group 1.5 ± 0.32 cm and in control group 1.4 ± 0.38 cm. On the other hand, left renal cortical thickness in case group 1.6 ± 0.32 cm and in control group 1.4 ± 0.37 cm. The mean right renal volume in case group 82.58 ± 1.47 cm and in control group 78.16 ± 1.02 cm. The left renal volume in case group 93.19 ± 1.221 cm and in control group 79.27 ± 0.91 cm. **Conclusion:** The cortical size for both kidneys was greater in our study group compared to cortical size in normotensive subjects. In agreement with published studies, our study showed that renal volume is higher in the left than in the right kidney for both sexes.

Keywords: Renal cortical thickness, Renal volume, Hypertension.

INTRODUCTION

According to world health organization (WHO) blood pressure is the force exerted by circulating blood against the walls of the body's arteries, the major blood vessels in the body. ^[1] Prevalence of Hypertension according to WHO An estimated 1.28 billion adults aged 30-79 years worldwide have hypertension, most (two-thirds) living in low- and middle-income countries. ^[2] In the year 2021 the prevalence of hypertension in India was 16.32% among men and 11.56% among

women. Persons in the urban location (12.5%, 95% CI 12.25% to 12.80%) had a marginally higher prevalence than persons in rural location (10.6%, 95% CI 10.50% to 10.78%). The proportion of population suffering from hypertension varied greatly between states, with a prevalence of 8.2% in Kerala to 20.3% in Sikkim. ^[3]

Renal volume is an important parameter in clinical evaluation and management of patients with kidney diseases such as congenital anomalies, renal cystic diseases, kidney stones, renal artery stenosis, recurrent urinary tract infections, vesicoureteral reflux, chronic kidney disease, kidney tumors and kidney transplants both in the paediatric and adult population. ^[4]

New imaging modalities have achieved an increasingly important role in the clinical workup of kidney diseases. They allow minimally invasive measurement of the kidney volume and the loss of functional parenchyma, as well as renal blood flow (RBF) and glomerular filtration rate (GFR). ^[5] Sonography is a standard low-cost modality and is easily accessible. However, it is operator dependent, estimated volumes suffer from poor reproducibility and low accuracy, and 3D ultrasound equipment is not widely available. ^[6] CT provides precise measurements, also in 3D, but the patients are exposed to ionized radiation and iodinated contrast agents, which may be contraindicated for use. ^[7] MRI may emerge as a good alternative by acquiring high-resolution 3D images without radiation exposure. Generally, MRI provides good tissue contrast that facilitates segmentation of the kidney and extraction of volumetric information from the images. ^[8]

MATERIAL AND METHOD

This is a prospective, descriptive, cross sectional and observational study. Department of Physiology, General Medicine and Radiology, Index Medical College, Hospital and Research center Indore.

Age 41-60 years of either gender hypertensive subjects according to JNC VIII (Systolic BP >140mmHg and Diastolic BP >90 mmHg) was inclusion criteria of cases. Age 41-60 years of either gender normotensive subjects according to JNC VIII and no renal diseases was inclusion criteria of control. Patients with renal tumours, kidney failure and hydronephrosis was excluded. Pregnant and lactating women was excluded.

A curvilinear probe with transducer frequency of 2–8 MHz of a Sonoace X6 ultrasound machine was used. Each individual was laid supine on the couch with the abdomen adequately exposed from upper abdomen to the symphysis pubis.

Longitudinal, coronal, and transverse scans of the kidneys were obtained in the supine, supine-oblique, and prone positions. Renal dimensions including length, width, anteroposterior thickness as well as renal cortical thickness and renal parenchymal volume/echogenicity/echotexture was assessed.

Cortical echogenicity was assessed using the supine views only and graded as follows:

Grade 0 = normal, renal cortical echogenicity less than the echotexture of the liver on the right and spleen on the left.

Grade 1 = Renal cortical echogenicity equal to echotexture of the liver on the right and spleen on the left.

Grade 2 = Renal cortical echogenicity greater than echotexture of the liver on the right and spleen on the left but less than the renal sinus echo.

Grade 3 = Renal cortical echogenicity equal to the renal sinus.

Using electronic calipers, the renal length (L) was taken as the longest distance between the renal poles on the longitudinal scan and the renal width (W) as the maximum transverse diameter on the transverse scan. The renal thickness or depth (D) was taken as the average of the maximum distance between the anterior and posterior walls of the midportion of the kidney in the longitudinal and transverse scans (D1 and D2). The kidney volume was obtained using the prolate ellipsoid formula ($L \times W \times D1 + D2/2 \times 0.523$).^[9]

Cortical thickness was assessed on a longitudinal scan as the perpendicular distance from the base of a pyramid to the renal capsule, 2 cm away from the renal poles and at the midportion of the kidney.

The renal parenchymal volume was obtained by using longitudinal and transverse scans. The maximum longitudinal, transverse, and anteroposterior dimensions of the kidney and the central sinus echo was obtained, the ellipsoid formula was used to calculate the renal volume and the volume of the central sinus echo, respectively. The renal parenchymal volume is the volume obtained by subtracting the volume of the central sinus echo from the renal volume. All measurements were done by one observer, the values were measured three times, and the average value was taken to reduce intraobserver errors.

Statistical analysis

The collected responses were converted into excel sheets and converted into SPSS software version 25 for analysis. The study used simple frequency distribution and unpaired t-test for testing the framed hypothesis.

RESULT

In table 1, the age group of case in the study was 41 to 60 years. The mean age of case group was 51.02 years with standard deviation of 4.29. The mean age of control group was 49.21 years with standard deviation of 4.83.

Table 1: Age Statistics of case and control group

(Age in years)	Case	Control
Mean	51.02	49.21
Std. Deviation	4.29	4.83
Minimum	41	41
Maximum	60	60

Table 2: Case and control group of Body Weight Changes

Study Subjects	Weight (kg/m ²) Mean ±SD	p- Value
Case group	67.96 ± 8.92	<0.05
Control group	65.94 ± 8.54	

It is observed from Table 2 that; Mean Body Weight of subject case and control group are 67.96 ± 8.92 kg (Mean±SD) and 65.94 ± 8.543 kg respectively. This difference in Body Weight between case and control group is found to be statistically significant. (P value < 0.01).

Table 3: Case and Control Group of BMI Changes

Study Subjects	BMI (kg/m ²) Mean ±SD	p- Value
Case group	23.35 ± 2.27	<0.05
Control group	22.01 ± 2.20	

It is observed from Table 3 that; case group Mean BMI is 23.35 ± 2.27 kg/m² (Mean±SD) and control group Mean BMI is 22.01 ± 2.20 kg/m² (Mean±SD). The difference of these values shows statistically significant reduction in BMI. (P value < 0.01).

Table 4: Case and Control Group of WC Changes

Study Subjects	WC (cm) Mean \pm SD	p- Value
Case group	84.58 \pm 8.31	<0.05
Control group	82.99 \pm 8.98	

It is observed from Table 4 that, Mean Waist-Circumference is difference from case group of 84.58 \pm 8.31 cm and control of 82.99 \pm 8.98 cm. This difference in Waist-Circumference is statistically significant. (P value < 0.01).

Table 5: Case and control group of WHR Changes

Study Subjects	WHR Mean \pm SD	p- Value
Case group	0.87 \pm 0.03	<0.05
Control group	0.86 \pm 0.03	

It is observed from Table 5 that, Mean WHR is difference in case group of 0.87 \pm 0.03 (Mean \pm SD) and control group of 0.86 \pm 0.03 (Mean \pm SD). This difference in Waist to Hip ratio is statistically significant. (P value < 0.01).

Table 6: Case and Control Group of Pulse Rate Changes

Study Subjects	Pulse Rate (beats/minute) Mean \pm SD	p- Value
Case group	81.79 \pm 5.04	<0.001
Control group	74.24 \pm 4.99	

It is observed from Table 6 that; the case group Mean Pulse Rate is 81.79 \pm 5.04 beats/min (Mean \pm SD) and control group Mean Pulse Rate of 74.24 \pm 4.99 beats/min (Mean \pm SD). The difference in Pulse Rate is statistically significant. (P value < 0.01).

Table 7: Case and Control Group of Right renal Cortical thickness

Study Subjects	Right renal Cortical thickness (cm), Mean \pm SD	p- Value
Case group	1.5 \pm 0.32	<0.05
Control group	1.4 \pm 0.38	

Furthermore, mean right renal cortical thickness in case group 1.5 \pm 0.32 cm and in control group 1.4 \pm 0.38 cm in table 7.

Table 8: Case and Control Group of Left renal Cortical thickness

Study Subjects	Left renal Cortical thickness (cm), Mean \pm SD	p- Value
Case group	1.6 \pm 0.32	<0.05
Control group	1.4 \pm 0.37	

On the other hand, left renal cortical thickness in case group 1.6 \pm 0.32 cm and in control group 1.4 \pm 0.37 cm in table 8.

Table 9: Case and Control Group of Right renal volume

Study Subjects	Right renal volume (cm ³), Mean \pm SD	p- Value
Case group	82.58 \pm 1.47	<0.001
Control group	78.16 \pm 1.02	

Moreover, mean right renal volume in case group 82.58 \pm 1.47 cm and in control group 78.16 \pm 1.02 cm in table 9.

Table 10: Case and Control Group of Left renal volume

Study Subjects	Left renal volume (cm ³), Mean \pm SD	p- Value
Case group	93.19 \pm 1.22	<0.001
Control group	79.27 \pm 0.91	

On the other hand, left renal volume in case group 93.19 \pm 1.22 cm and in control group 79.27 \pm 0.91cm in table 10.

DISCUSSION

The assessment of renal disease using biochemical assay is often carried out by the estimation of serum electrolyte, urea and creatinine (E/U/Cr) in blood and also through the determination of the amount endogenous or exogenous substances present in urine (urinalysis, 24 hours Creatinine or iohexol Clearance). Renal function can also be determined from the GFR by estimating endogenous creatinine clearance using the Cockcroft Gault equation. ^[10]

Ultrasonography (US) has therefore become the standard imaging modality in the investigation of renal diseases because it is accurate, non-invasive, cost effective, easily available, convenient and provides excellent anatomical details. Ultrasonography requires no special patient preparation neither does it require the use of X-radiation or contrast agents which are potentially harmful. ^[11]

In this study, the Mean age of the case group subjects was 51.02 \pm 4.29 years (Mean \pm SD) and control group subjects was 49.21 \pm 4.83 years. It is observed from Table 3, that in case group Mean Body Weight was 67.96 \pm 8.92 kg (Mean \pm SD) and control group was 65.94 \pm 8.54 kg. The difference in these values was found statistically significant (P value < 0.01). That means hypertension leads to significant weight increase. Similarly, other studies have reported weight gain during the Hypertension (Mansi KMS 2007). ^[12] A few study sheds light on the renin-angiotensin system – the hormone system that regulates blood pressure – may also promote excess weight gain. Peripheral RAS

activity is increased, there are high levels of the RAS hormone angiotensin circulating in the rest of the body it reduces resting metabolism, causing weight gain. [13]

The present study indicates that case group Mean BMI was $23.35 \pm 2.27 \text{ kg/m}^2$ and control group Mean BMI was $22.01 \pm 2.20 \text{ kg/m}^2$. The difference in these values was statistically significant (P value < 0.01), as evident from Table 4. That means hypertension causes significant increase in BMI. Mechanisms of hypertension-related increase in BMI include insulin resistance, sodium retention, increased sympathetic nervous system activity, activation of renin–angiotensin–aldosterone, and altered vascular function. This result is in line with the findings reported by Ziaee V et al (2006). [14]

In the present study, it is evident from Table 5 that, case group Waist-Circumference of $84.58 \pm 8.31 \text{ cm}$ (M±SD) and control group Waist-Circumference of $82.99 \pm 8.98 \text{ cm}$ (M±SD). This increase in Waist-Circumference was found to be statistically significant. The result is similar to that observed by Saleh SM (2005). [15] However, few studies reported no change in Waist-Circumference [Yucel A et al 2004]. [16]. Although the biological mechanisms responsible for the association between WC and cardiovascular complications have been hypothesized including genetic predisposition, activation of the sympathetic nervous system, insulin resistance, and metabolic products of intra-abdominal adipose tissue such as inflammatory adipokines, angiotensinogen, or cortisol. [17]

Also, there was significant increase in Waist to Hip ratio (WHR) after hypertension as evident from Table 6, where Mean WHR was difference from 0.87 ± 0.03 to 0.86 ± 0.03 . Yucel A et al (2004) reported change in WHR. [17] Waist-Circumference and WHR are the reflection of abdominal obesity. This region of fat deposition provides a reasonable indication of the accumulation of intra-abdominal (visceral) adipose tissue. As there is progressive of fat depots during the hypertension. [18]

Furthermore, mean right renal cortical thickness in case group $1.5 \pm 0.32 \text{ cm}$ and in control group $1.4 \pm 0.38 \text{ cm}$. On the other hand, left renal cortical thickness in case group $1.6 \pm 0.32 \text{ cm}$ and in control group $1.4 \pm 0.37 \text{ cm}$. This is in agreement with a study conducted by Habbal et al. (1998) [19] The cortical size in our study group for both sides are greater than the cortical size in normoxic area. This variation is reflection of the fact that short kidneys with stocky infundibuli has thicker cortex than kidneys with elongated spidery infundibula (Adibi, 2008). [20]

It is observed from this study, there is a significant increase in Pulse Rate in case group. In case group Mean Pulse, Rate was $81.79 \pm 5.04 \text{ beats/min}$ and

control group Mean Pulse Rate was $74.24 \pm 4.998 \text{ beats/min}$. Similar result is reported by Mansi KSA (2007). [21] The issues of heart rate increase and hypertension control are magnified in patients with kidney disease. Decreased renal function is associated with elevated heart rate, hypertension, and significantly increased risk for cardiovascular disease. In patients with kidney disease, elevated heart rate is associated with increased risk for cardiovascular disease. [22]

Moreover, mean right renal volume in case group $82.58 \pm 1.47 \text{ cm}$ and in control group $78.16 \pm 1.02 \text{ cm}$. On the other hand, left renal volume in case group $93.19 \pm 1.22 \text{ cm}$ and in control group $79.27 \pm 0.91 \text{ cm}$. Our observation of increase in left renal volume is supported by the findings of Athar S, Habib M (1994). [23] Increased blood flow in the left renal artery may result in a relatively increase in volume of the left kidney. [24]

CONCLUSION

The cortical size for both kidneys was greater in our study group compared to cortical size in normotensive subjects. In agreement with published studies, our study showed that renal volume is higher in the left than in the right kidney for both sexes. The female patients have smaller kidney size compared to males.

REFERENCE

1. Griffin KA, Picken M, Giobbie-Hurder A, Bidani AK. Low protein diet mediated renoprotection in remnant kidneys: renal autoregulatory vs. hypertrophic mechanisms. *Kidney Int.* 2003; 63: 607–616.
2. van Rodijnen WF, van Lambalgen TA, Tangelder GJ, van Dokkum RP, Provoost AP, ter Wee PM. Reduced reactivity of renal microvessels to pressure and angiotensin II in fawn-hooded rats. *Hypertens.* 2002; 39: 111–115.
3. Fogo AB. Glomerular hypertension, abnormal glomerular growth, and progression of renal diseases. *Kidney Int.* 2000; 57: S15–S21.
4. Kriz W, Elger M, Mundel P, Lemley KV. Structure-stabilizing forces in the glomerular tuft. *J Am Soc Nephrol.* 1995; 5: 1731–1739.
5. Pavenstadt H, Kriz W, Kretzler M. Cell Biology of the glomerular podocyte. *Physiol Rev.* 2003; 83: 253–307.
6. Grantham JJ, Torres VE, Chapman AB, Guay-Woodford LM, Bae KT, King BF Jr, et al.; CRISP Investigators. Volume progression in polycystic kidney disease N Engl J Med 2006;18;354(20):2122-30.
7. Lawson CR, Doulton TW, MacGregor GA. Autosomal Dominant Polycystic Kidney Disease: Role of the Renin-Angiotensin

- System in Raised Blood Pressure in Progression of Renal and Cardiovascular Disease. *J Renin-Angiotensin-Aldosterone System* 2006;7:139-45.
8. Cadnapaphornchai MA, McFann K, Strain JD, Masoumi A, Schrier RW. Increased left ventricular mass in children with autosomal dominant polycystic kidney disease and borderline hypertension. *Kidney Int* 2008;74(9):1192-6.
9. Hernández del Rey R, Armario P, Martín-Baranera M, Sánchez P, Coca A, Pardell H. Afectación orgánica en hipertensos con efecto de bata blanca inversa. *Estudio Hospitalet. Hipertensión* 2000;17(extra):127.
10. Björklund K, Lind L, Zethelius B, Andrén B, Lithell H. Isolated ambulatory hypertension predicts cardiovascular morbidity in elderly men. *Circulation* 2003;107:1297-302.
11. Hernández del Rey R, Armario P, Martín-Baranera M, Sánchez P, Almendros MC, Coca A, et al. Cardiac damage in hypertensive patients with inverse white coat hypertension. *Hospitalet study. Blood Pressure* 2001;12:89-96.
12. Ohkubo T, Kikuya M, Metoki H, Asayama K, Obara T, Hashimoto J, et al. Prognosis of masked hypertension and white coat hypertension detected by 24h ambulatory blood pressure monitoring: 10 year follow-up from the Ohasama study. *J Am Coll Cardiol* 2005;46:508-15.
13. Mancia G, Facchetti R, Bombelli M, Grassi G, Sega R. Long-term risk of mortality associated with selective and combined elevation in office, home, and ambulatory blood pressure. *Hypertension* 2006;47:846-53.
14. Mancia G, De Backer G, Dominiczak A, Cifkova R, Fagard R, Germano G, et al. 2007 Guidelines for the Management of Arterial Hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *J Hypertens* 2007;25:1105-187.
15. Oyuela-Carrasco J, Rodríguez-Castellanos F, Kimura E, Delgado- Hernández R, Herrera-Félix JP. Longitud renal por ultrasonografía en población mexicana adulta. *Nefrología* 2009;29(1):30-4.
16. Sega R, Corrao G, Bombelli M, Beltrame L, Facchetti R, Grassi G, et al. Blood pressure variability and organ damage in a general population: results from the PAMELA study (Pressioni Arteriose Monitorate E Loro Associazioni). *Hypertension* 2002;39:710-4.
17. Staessen JA, Wang J, Bianchi G, Birkenhager WH. Essential hypertension. *Lancet*. 2003;361:1629–1641.
18. Barker DJ. Developmental origins of adult health and disease. *J Epidemiol Community Health*. 2004;58:114–115.
19. Drake AJ, Walker BR. The intergenerational effects of fetal programming: non-genomic mechanisms for the inheritance of low birth weight and cardiovascular risk. *J Endocrinol*. 2004;180:1–16.
20. McMillen IC, Robinson JS. Developmental origins of the metabolic syndrome: prediction, plasticity, and programming. *Physiol Rev*. 2005; 85:571– 633.
21. Lifton RP, Gharavi AG, Geller DS. Molecular mechanisms of human hypertension. *Cell*. 2001;104:545–556
22. Hughson M, Farris AB, Douglas-Denton R, Hoy WE, Bertram JF. Glomerular number and size in autopsy kidneys: The relationship to birth weight. *Kidney Int*. 2003;63:2113–2122.
23. Hoy WE, Douglas-Denton RN, Hughson MD, Cass A, Johnson K, Bertram JF. A stereological study of glomerular number and volume: preliminary findings in a multiracial study of kidneys at autopsy. *Kidney Int*. 2003;83(suppl);S31–S37.
24. Hoy WE, Hughson MD, Bertram JF, Douglas-Denton R, Amann K. Nephron number, hypertension, renal disease, and renal failure. *J Am Soc Nephrol*. 2005;16:2557–2564.