

An Impact of Intima Media Thickness of Carotids in Cardiovascular Profile

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ABSTRACT

Low blood pressure indeed includes a worse prognosis than excessive blood strain. This mechanism, bills for the "reverse causation" seen within the haemodialysis' patients, the company of conventional risk elements, such as high blood pressure, hyperlipidemia, and obesity, appear to be the worst diagnosis. Exogenous erythropoietic products can growth blood strain and requirement of antihypertensive tablets. Chronic ECFV overload secondary to activation of renin-angiotensin-aldosterone axis and disturbances inside the stability of vasoconstrictors and the vasodilators contributes to high blood pressure. Improvement in blood pressure can be introduced out with oral sodium restriction, diuretics, and fluid elimination with dialysis. Some patients will continue to be hypertensive notwithstanding of the careful attention to ECFV reputation. LVH is related to reduced endurance of sufferers on hemo/peritoneal dialysis. Lower five-year survival charge in ESRD patients with LVH has a 30% than people missing LVH. This has a look at produces the mean carotid artery intima-medial thickness turned into higher in sufferers with superior CKD. However, it did now not attain statistical significance, probable due to the smaller sample size. It was also observed that carotid intima medial thickness did not correlate with dyslipidemia. Even though the patients had maintained significantly healthy cholesterol and high HDL levels, there was an increase in CIMT. Therefore in CKD patients, CIMT cannot be predicted based on the traditional atherosclerotic risk factors like serum cholesterol and HDL.



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INTRODUCTION

In this section presents the introduction of this research work. Chronic kidney disease (CKD) is normally definite as kidney harm for greater than three months period, characterized through structural or

functional abnormalities of the kidney, with or without lower in the glomerular filtration charge (GFR), appear by way of both pathological abnormalities or markers of kidney damage, which includes abnormalities in the configuration of the blood or urine, or abnormalities in imaging checks, with /without kidney damage. [1, 2]

CKD with comorbidities like diabetes, hypertension can lead to early progression of stage 5 GFR <15ml/min, i.e., end-stage renal disease, causing a higher risk for cardiovascular disease humanity and morbidity also. [3, 4]

The distance for lumen-Intima interface and Media-adventitia interface of the artery wall is defined as Intima-medial thickness. IMT values are 0.5mm in the younger age group (20-30 yrs) and 0.9mm in elders (60-70 yrs). IMT of carotids is useful for mea-

measuring the severity of atherosclerosis which is usually associated with cardiovascular risk factors like diabetes, hypertension and also dyslipidemia. [5, 6]

Association of hypertension represents a danger aspect of cardiovascular sickness found in CKD and almost invariably found in renal failure patients. Sodium retention and beginning of the renin-angiotensin machine have been measured the maximum vital mechanisms worried in the promotion of blood pressure in topics with kidney disease. Hypertension performs a crucial role in cardiac damage in CKD thru left ventricular hypertrophy (LVH). As in different populations in CKD, patients with LVH predicts a worse CV prognosis. [7, 8]

Pulmonary Hypertension is a worry of end level renal sickness. Pulmonary high blood pressure is defined as average pulmonary artery stress which is extra than or once in a while equal to twenty-five mm Hg or Pulmonary artery systolic stress additional than 35 mm Hg. It is usually recognized in cardiology and pulmonary hospital. [9]

In this article represents segment 2 of this article clarifies the aspect of the correlated works. In part 3 represents the materials and methods accepted, and layer 4 describes the particulars of the experimentations and deliberations. Lastly, section 5 accomplishes the article by the allocation of our extrapolations and future strategies Figure 1.

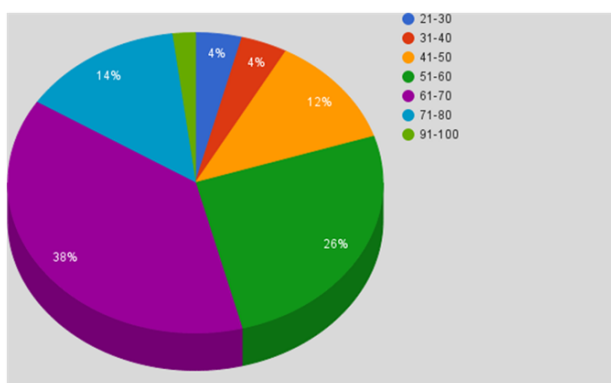


Figure 1: Age Distribution of the Subjects

Related Works

In this segment gives focuses on the associated works of this research paintings. The kidney consists of many tortuous, tightly packed tubules, bound by using a delicate connective tissue wherein the blood vessels, lymphatics and nerves run. A critical part of the kidney is the nephron. It is composed of a renal corpuscle that is constituted by way of the glomerulus and the Bowman's capsule which filters the plasma. It additionally has a renal tubule consisting of the proximal convoluted tubule, loop of Henle, distal convoluted tubule, collecting tubule

and amassing duct which is involved with discerning resorption from the filtrate to form urine. Gathering pipes carry fluid from numerous renal tubules to a terminal papillary pipe establishing into a minor calyx on the apex of a renal papilla. [10]

The kidney consists of numerous tortuous, intently packed tubules, specific by using a sensitive connective tissue in which the blood vessels, lymphatics and nerves run. The nephron is considered as a basic shape. It consists of a renal corpuscle that is constituted with the aid of glomerulus and the Bowman's capsule, which enables in filtering the plasma. It additionally has a renal tubule collected of the proximal convoluted tubule, loop of Henle, distal convoluted tubule, amassing tubule and also accumulating duct which is worried about the formation of urine utilizing selective resorption. Collecting ducts enables in bring fluid from numerous renal tubules to a terminal papillary duct which gets commencing into a minor calyx at the apex of a renal papilla. [11]

Renal arteries supply kidney. Major artery branches into segmental branches which then divide into lobar, interlobar, arcuate and interlobular arteries. The arterial blood enters into the glomeruli through the afferent arteriole which then passes through the efferent arterioles and then converges to form interlobular veins. These interlobular veins pass to the corticomedullary connection before ending in arcuate veins and anastomose with neighbouring veins. They drain into interlobar veins which anastomose to form the renal veins. [12]

In a slightly surprising finding, Vupputuri conducted a study in the US, where it was noted that alcohol and smoking are not related to CKD. [13] A study refuting claims to the knowledge that family history might have a role in the development of CKD, observed no association between family history of CKD and CKD. However, a Southern Chinese study by Wei et al., strongly claimed that first degree relatives are at a higher risk to develop CKD. [14, 15]

MATERIALS AND METHODS

In this segment represents the materials and methods of this research work. Outpatients are attending Internal Medicine and Nephrology OPD and Inpatients admitted under Medical/ Nephrology. Chronic kidney disease patients fulfilling the inclusion criteria were included in the study after obtaining informed consent Table 1.

Detailed history regarding cardiac, renal and gastrointestinal symptoms will be collected using a questionnaire as per the attached proforma. Com-

Table 1: Variable observations of the study

Parameter	Value	Percentage	P Value
Mean Age Of CKD Patients	44.55±16.26 Years		
Patients With Comorbidities	38	43.7%	
Essential Hypertension	27	35%	
Mean BMI Was 18.5-25 Kg/M2	35	68%	
Stage 5 CKD	31	62%	
Hypocalcaemia (< 9 Mg/Dl)	23	46%	
Hyperphosphatemia(4.1-5mg/Dl)	31	62%	
Hyperphosphatemia (>5 Mg/Dl)	11	22%	
High Total Serum Cholesterol Levels >150 Mg/Dl	32	64%	
Mean HDL-C Levels Were >40 Mg/Dl	32	64%	
Pulmonic Hypertension In Stage 5 CKD	6	12%	<0.05

plete blood count, urea, creatinine, liver function tests, fasting lipid Profile, calcium and phosphorus and Ultrasound of abdomen. Assessment of Renal status by blood urea, serum creatinine, USG abdomen Severity of CKD is assessed by Cockcroft – Gault GFR formula and MDRD formula. We are evaluating the cardiac situation by ECG, ECHO, Chest X-ray. Intima medial thickness of carotids Assessment by USG study both in dialysis and non-dialysis patients. Assessment of PAH by ECG, Chest xray, ECHO.

RESULTS AND DISCUSSIONS

In this section focuses the results and discussions of this research work.

This cross-sectional study was conducted over a population of 50 patients, all of whom were diagnosed with CKD. The average age of the subjects was found to be 60±13.87 years with a range of 21-92 years.

In the present study, 50 patients with CKD in the stages, 3-5 (calculated according to MDRD formula) were included. Univariate correlation analysis of CIMT with Age, BMI, Estimated GFR, Serum cholesterol, Triglyceride, HDL, LDL levels, Serum calcium and Serum phosphorus levels were attempted.

The present study did not correlate with AGE and Mean CIMT. A significant association between BMI and CIMT. But our study did not show any relationship between BMI and Mean CIMT. This is probably due to the BMI in Indian population differs from western society.

There were conflicting reports regarding the relation between CIMT and Lipid profile. Reported correlation between reported a non-relationship.

Our study indicated that despite Total cholesterol level average in 88% but still there were increased in CIMT (P-0.0001). Another observation in my research, there was an increase in CIMT (p-0.0001) despite standard and high HDL(>75%). The negative correlation with HDL. The present study did not show any relationship; this may be probably due to new management and imperfect nutrition of our population. In the present study, CKD patients had high calcium phosphorous product. There was no significant correlation (p-0.93160) seen with CIMT and calcium-phosphorus product in CKD patient. Mean CIMT was found higher in the late stages of kidney disease (stage 4 and Stage 5) as compared to early stages (stage 1,2 and 3). Though percentage (60%) of Mean CIMT was found to be higher in late stages of kidney disease (step 4,5) but this is not statistically significant in our study. No significant difference between CKD groups and concluded that increase in CIMT caused by renal disappointment and metabolic abnormalities secondary to renal failure.

Stage 3 to 4 CKD had increased CIMT compared with normotensive volunteers. Lu Xia Zhang et al, in their study, found scene 2-3 CKD patients (i.e., mild and moderate renal insufficiency) found significantly increased CIMT in those patients. They concluded that arterial change might occur in the course of CKD earlier than previously believed. But the present study showed similar observation felt there is increased CIMT in stage 3 and 4. Our study showed a 16% prevalence in CKD patients. Pulmonary hypertension was found in later stages (3-5) which is statistically significant when compared to other steps.

Left ventricular hypertrophy is the most common

ECHO findings in CKD patients (44%), whereas systolic dysfunction was (18%) conducted a prospective study of 161 patients of end-stage disease on dialysis. He observed Left ventricular disease in 105 (65.2%) patients. Only 56(34.8%) had normal echocardiogram, systolic dysfunction in 24 (14.9%), left ventricular hypertrophy in 88 (54.7%) & PAH in 42 (26.1%) patients.

Non-traditional risk factors like Serum homocysteine levels, Serum Parathyroid level lipoprotein (LPA), etc. for atherosclerosis were not studied and compared with CIMT in this study. Inclusion of these variables would have added power to analysis. In the present study, CIMT was measured as a morphological index of atherosclerosis. Measurement of arterial wall difficulty will provide information regarding the effects of renal failure on functional changes of the arterial wall in patients with CKD. Pulse wave velocity is emerging as the most reliable estimate of arterial stiffness (functional measurement) and a strong predictor of the cardiovascular events in ESRD. As our hospital is tertiary /referral centre, most of the patient present in late stages of disease only. Therefore further studies require to throw light on the mechanism of atherosclerosis in early stages of Renal disease also.

The mean age of CKD patients was 44.55 ± 16.26 years. Diabetes was the comorbidity of CKD in 38 (43.7%) patients, essential Hypertension in 27(35%) and both diabetes with hypertension are 22 (25.3%) were other major causes of CKD patients. The mean BMI was 18.5-25 kg/m² in CKD patients are 35(68%). Most of the CKD patients in the present study was in Stage 5 I.e.31(62%). Hypocalcaemia (< 9 mg/dl) was seen in 23(46%) CKD patients. Hyperphosphatemia (4.1-5mg/dl) was seen in 31(62%) and (>5 mg/dl) seen in 11 (22%)CKD patients. High Total serum Cholesterol levels >150 mg/dl was found in 32 patients (64%). The mean HDL-C levels were >40 mg/dl was found in 32 patients (64%). The prevalence of pulmonary hypertension in stage 5 CKD in 6(12%) was statistically significant(p<0.05). Mean CIMT in CKD patients does not significantly correlate with Age (P=0.543389). Body Mass Index (P=0.4255) also not correlated with Mean CIMT. Serum Triglyceride levels (P=0.186) and VLDL-C and LDL levels and with calcium-phosphorous product in CKD patients are not correlated. There is no statistically significant correlation between the stage of kidney disease and CIMT.

CONCLUSIONS

Finally, this work concludes that in the present study, the mean carotid artery intima-medial thickness was higher in a patient with advanced CKD. However, it did not reach statistical significance, perhaps due to the smaller sample size. It was also observed that carotid intima medial thickness did not correlate with dyslipidemia. Even though the patients had maintained significantly healthy cholesterol and high HDL levels, there was an increase in CIMT. Therefore in CKD patients, CIMT cannot be predicted based on the traditional atherosclerotic risk factors like serum cholesterol and HDL. Prevalence of Pulmonary hypertension is significantly higher in stage 5 CKD. None of the patients is having significant dyslipidemia possibly due to medications.

CONFLICT OF INTEREST

None

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REFERENCES

- [1] Yuan Hsu C, McCulloch CE, Darbinian J, Go AS, Iribarren C. Elevated Blood Pressure and Risk of End-stage Renal Disease in Subjects Without Baseline Kidney Disease. *Archives of Internal Medicine*. 2005;165(8):923-923. Available from: [10.1001/archinte.165.8.923](https://doi.org/10.1001/archinte.165.8.923).
- [2] Fogo AB. Mechanisms of progression of chronic kidney disease. *Pediatric Nephrology*. 2007;22(12):2011-2022. Available from: [10.1007/s00467-007-0524-0](https://doi.org/10.1007/s00467-007-0524-0); <https://dx.doi.org/10.1007/s00467-007-0524-0>.
- [3] Kanwar YS, Sun L, Xie P, Liu FY, Chen S. A glimpse of various pathogenetic mechanisms of diabetic nephropathy. *Annual Review of Pathology: Mechanisms of Disease*. 2011;6:395-423.
- [4] Tan ALY, Forbes JM, Cooper ME. AGE, RAGE, and ROS in Diabetic Nephropathy. *Seminars in Nephrology*. 2007;27(2):130-143. Available from: [10.1016/j.semnephrol.2007.01.006](https://doi.org/10.1016/j.semnephrol.2007.01.006).

- [5] Amin R, Turner C, van Aken S, Bahu TK, Watts A, Lindsell DRM, et al. The relationship between microalbuminuria and glomerular filtration rate in young type 1 diabetic subjects: The Oxford Regional Prospective Study. *Kidney International*. 2005;68(4):1740–1749. Available from: [10.1111/j.1523-1755.2005.00590.x](https://doi.org/10.1111/j.1523-1755.2005.00590.x).
- [6] Limardo M, Imbasciati E, Ravani P, Surian M, Torres D, Gregorini G, et al. Pregnancy and Progression of IgA Nephropathy: Results of an Italian Multicenter Study. *American Journal of Kidney Diseases*. 2010;56(3):506–512. Available from: [10.1053/j.ajkd.2010.03.033](https://doi.org/10.1053/j.ajkd.2010.03.033).
- [7] Puri TS, Quigg RJ. The Many Effects of Complement C3- and C5-Binding Proteins in Renal Injury. *Seminars in Nephrology*. 2007;27(3):321–337. Available from: [10.1016/j.semnephrol.2007.02.005](https://doi.org/10.1016/j.semnephrol.2007.02.005).
- [8] Tiengo A, Fadini GP, Avogaro A. The metabolic syndrome, diabetes and lung dysfunction. *Diabetes & Metabolism*. 2008;34(5):447–454. Available from: [10.1016/j.diabet.2008.08.001](https://doi.org/10.1016/j.diabet.2008.08.001).
- [9] Evans AM, Hardie DG, Peers C, Mahmoud A. Hypoxic pulmonary vasoconstriction: mechanisms of oxygen-sensing. *Current Opinion in Anaesthesiology*. 2011;24(1):13–20. Available from: [10.1097/aco.0b013e3283421201](https://doi.org/10.1097/aco.0b013e3283421201).
- [10] Sakao S, Tatsumi K, Voelkel NF. Reversible or Irreversible Remodeling in Pulmonary Arterial Hypertension. *American Journal of Respiratory Cell and Molecular Biology*. 2010;43(6):629–634. Available from: [10.1165/rcmb.2009-0389tr](https://doi.org/10.1165/rcmb.2009-0389tr).
- [11] Abassi Z, Nakhoul F, Khankin E, Reisner SA, Yigla M. Pulmonary hypertension in chronic dialysis patients with arteriovenous fistula: pathogenesis and therapeutic prospective. Ovid Technologies (Wolters Kluwer Health); 2006. Available from: [10.1097/01.mnh.0000232874.27846.37](https://doi.org/10.1097/01.mnh.0000232874.27846.37).
- [12] Libby P. The pathogenesis, prevention, and treatment of atherosclerosis. *Harrison's principle of internal medicine*. 2008;2:2252–2260.
- [13] Halpern SD, Taichman DB. Misclassification of Pulmonary Hypertension Due to Reliance on Pulmonary Capillary Wedge Pressure Rather Than Left Ventricular End-Diastolic Pressure. *Chest*. 2009;136(1):37–43. Available from: [10.1378/chest.08-2784](https://doi.org/10.1378/chest.08-2784).
- [14] Wanner C, Krane V, März W, Olschewski M, Mann JFE, Ruf G, et al. Atorvastatin in Patients with Type 2 Diabetes Mellitus Undergoing Hemodialysis. *New England Journal of Medicine*. 2005;353(3):238–248. Available from: [10.1056/nejmoa043545](https://doi.org/10.1056/nejmoa043545).
- [15] Lins RL, Matthys KE, Billiouw JM, Dratwa M, Dupont P, Lameire NH, et al. Lipid and apoprotein changes during atorvastatin up-titration in hemodialysis patients with hypercholesterolemia: a placebo-controlled study. *Clinical Nephrology*. 2004;62(10):287–294. Available from: [10.5414/cnp62287](https://doi.org/10.5414/cnp62287).

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