"CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD - A CASE CONTROL STUDY IN TERTIARY CARE CENTER TAMAKA KOLAR"

By:

DR .TUNGALA LEELA PAVAN

Dissertation submitted to the

Sri Devaraj Urs Academy of Higher Education and Research,

Tamaka, Kolar, Karnataka,

IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

DOCTOR OF MEDICINE (M.D.)

IN

GENERAL MEDICINE

Under The Guidance Of

Dr PRABHAKAR.K **MBBS, MD (MEDICINE)**

Professor & Head Of Unit

DEPARTMENT OF GENERAL MEDICINE

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR, KARNATAKA

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"CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD A CASE CONTROL STUDY IN TERITIARY CARE CENTRE TAMAKA KOLAR"

Abstract

Introduction: In the general population, carotid adventitial thickness (CIMT) forecasts future vascular events. But little research has been done on how chronic kidney disease (CKD) stages and conventional cardiovascular risk factors relate to CIMT. High-resolution B-mode ultrasonography was used to evaluate CIMT in order to compare it to old-style cardiovascular risk variables such age, body weight (BMI), lipid abnormalities , and different stages of CKD patients.

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Results: In the study majority of participants in stage 4 and 5 of patients suffering from the disease belonged to the age group of lesser than48 years and 49 to 64 years while in age groups greater than 64 years majority of participants stage 1 of CKD. There was an increase in CIMT

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"CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD A CASE CONTROL STUDY IN TERITIARY CARE CENTRE TAMAKA KOLAR" Abstract Introduction: In the general population, carotid adventitial thickness (CIMT) forecasts future vascular events. But little research has been done on how chronic kidney disease (CKD) stages and conventional cardiovascular risk factors relate to CIMT. High-resolution B-mode ultrasonography was used to evaluate CIMT in order to compare it to old-style cardiovascular risk variables such age, body weight (BMI), lipid abnormalities , and different stages of CKD patients. Method of collection of data: At RL Jalappa Hospitals and Research Centre, Kolar, patients diagnosed with various stages of CKD who were treated were randomly chosen patients. Age, gender, duration of diabetes, BMI, and outcomes were associated independently for each patient in two distinct groups. 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In CKD patients with CIMT, the study discovered a marginally favorable connection between blood triglyceride levels and total cholesterol levels.In the study in group 1 which includes participants with CKD, the median of haemoglobin was 9.00 and it was 15.00 in group 2. Conclusions: Despite the fact that CIMT was somewhat higher in individuals with advanced CKD, no statistically significant link was discovered between CIMT and eGFR. However, risk factors such as dyslipidemia was moderately associated with CKD patients with CIMT. Key words:"carotid intima-media thickness; chronic kidney disease; glomerular filtration rate". INTRODUCTION: Around 8-16% of people worldwide have the disease (CKD), which is indicated by a rate of glomerular filtration (GFR) about< sixty mL/min/1.73m2 that has persisted for more than 3 months. Compared to high income nations, low and medium income countries have a heavier burden from CKD 1.From modest renal dysfunction to total failure, there are 5 stages of kidney disease in CKD. Individuals with CKD in Stage 3 or 4 have a greater chance of dying or developing end-stage renal disease 2. Global Burden of Disease indicates estimates there were 1.4 million CKD related deaths in the world in 2019 which is a 20% increase from 2010. The main factors responsible for increased burden of CKD in the global are obesity, hypertension, diabetes and cardiovascular diseases 3. Along with the above listed variables, further explanations for the prevalence of CKD in South Asian nations like India include environmental pollutants and high levels of fluoride underground 4. India recorded a 38% rise in the percentage of renal failure-related mortality between 2001-03 and 2010-135. CKD is a substantial risk factor for cardiovascular disease (CVD). Even in the early stages of renal illness, the risk of heart disease is higher6. Early stages of CKD are associated with a 2 to 4 times increased risk of cardiovascular mortality7. Nearly 7.6percent of total of cardiovascular deaths worldwide in 2017 were caused by CKD- related illnesses, and CKD is also responsible for 25.3 million CVD -related disability adjusted life years 8. The dysregulation of minerals such as phosphorous amd calcium, vessel calcification, anaemia, and hyperhomocysteinemia that CKD promotes raises the risk of cardiovascular disease9. CKD raises the likelihood of heart disease without increasing the risk from other traditional cardiovascular disease risk factors6. Richard Bright made the initial suggestion in 1836 that cardiovascular disease and kidney illness are connected. The level of proteinuria and renal impairment, as well as the rate that these changes take place, all influence the risk of heart disease in individuals with CKD. "Albuminuria, anemic, thyroid dysfunction, metabolic bone disease, hyperhomocysteinaemia, malnourishment, apolipoprotein variants, inflammation, endothelial dysfunction, and oxidative stress are among the non-traditional CKD associated heart disease risk factors that are ureamia specific". These variables increase the rate of atherosclerosis and the progression of CKD in addition to established risk factors such advancing age, hypertension, dyslipidemia, diabetes, smoking, and obesity10. The absence of the traditional trifecta of ischemic symptoms, high cardiac biomarker levels, and electrocardiogram (ECG) alterations makes it difficult to diagnose heart disease in CKD patients11. Due to the absence of adequate medical facilities in underdeveloped nations like India When glomerular filtration rate ("gfr is below 15mL/min/1.73 m2", 50percent of CKD patients receive their first diagnosis12. Even mild renal failure significantly raises the risk of cardiovascular illness due to a number of factors, including aberrant myocardium reorganization, ventricular hypertrophy, arrhythmia, and cardiac arrest. In contrast to people with normal kidney function, atherosclerotic disease is overrepresented across the full range of CKD patients13. The increased prevalence of coronary artery disease in CKD patients is attributed to the presence of a chronic inflammatory state, disturbances in calcium-phosphate metabolism, oxidative stress, malnutrition, anaemia, hypervolemia, volatility in systemic fluid volume, instabilities in the coagulation system, buildup of active compounds, and unknown toxic agents. 14. It is noted that composition of atherosclerotic plaques in CKD patients is different from those present in sclerotic subjects with standard kidney function. CKD subjects have atherosclerotic plaques with more calcium deposits, reduced collagen fibres and smooth muscle cells, and more inorganic substances 15. Aging process of vascular and peripheral blood cells is one of many factors contributing to the complicated phenomena of vascular injury in CKD patients16. A direct inspection of the vessel wall is necessary to diagnose atherosclerosis since it is asymptomatic unless it is severe. Common carotid artery carotid-intima media thickness measurement using B-mode Early atherosclerotic process monitoring using ultrasound is non- invasive17. But because CIMT values change depending on the imaging side, the measurement spot, the measuring angle, and the cardiac cycle phase, evaluating the severity of atherosclerosis using this technique requires complex ultrasound techniques. Single-site CIMT assessments have been found to have lesser sensitivity. Carotid artery evaluation using imaging from a single viewpoint is not possible in three dimensions18. The non-invasive tools for detection of 5 atherosclerosis including typical symptoms, electrocardiogram, and cardiac stress testing are found to be less reliable in CKD subjects 19. Serum biomarkers measurements used in non-CKD subjects for diagnosis of cardiovascular diseases cannot be used in CKD subjects as in these subjects the biomarkers are elevated without myocardial infarction and myocardial apoptosis 20. Recently eGFR measurements are being considered for diagnosis of atherosclerosis. Kidney disease may now be easily detected thanks to the development of an equation for estimating estimate GFR (eGFR) from blood creatinine based on equations that take into account a variety of patient variables. This method converts the serum creatinine value into physiologic units of GFR21. Among

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"correlation of carotid intimal medial thickness with eGFR in patients with various stages of CKD A case control study in teritiry care centre Tamaka Kolar"** being investigated by **DR. TUNGALA LEELA PAVAN**, Dr. Prabhakar H in the Department of Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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ACKNOWLEDGEMENT

I sincerely thank my respected teacher, mentor and guide, **Dr. K. PRABHAKAR** for his guidance, constant encouragement, help and valuable advice which enabled me to complete this work successfully. His vast experience, knowledge, supervision were a constant source of inspiration during the entire course of my study.

I thank **Dr B.N. RAGHAVENDRA PRASAD, Head of department**, Department of **GENERAL MEDICINE,** for his constant guidance and advices.

I would like to express my heartfelt gratitude to **Dr.Raveesha.A and Dr VIDYASAGAR C.R. and DR. SRINIVASA S.V. and DR ANITHA A** for their valuable lessons and support.

To all my teachers throughout my life for having made me what I am today.

My deep felt gratitude to my dear parents, **T VISWESWARA RAO, T NAGALAKSHMI** whose countless sacrifices and blessings have made me who I am today.

I would like to thank my sister **CHANDANA.NIRUPAMA** and brother in law **CHANDANA. PAWAN KUMAR** for their love and constant encouragement.

I thank my best friend and brother **DR MANOHAR GOWDA B G** for constant support and help throughout.

Special thanks to **DR PRAVEEN P** sir who has been my constant support and mentor through my course

I thank my seniors **Dr SASI SEKHAR, DR DHRUVA NANDAN , DR SREENATH , DR SANMITHA ,DR KISHORE , DR RAKESH, DR DEEPAK D , DR JAVERIA AND DR ATISHAYA**

I am also thankful to my friends **DR.AMULYA, DR.SUJITHA, DR.TRISALI, DR. MANASA, DR. MANOHAR, DR. KAVYA, DR. POONGULALI, DR. INBA PRAVEEN, DR. KRUTHI, DR. BALA, DR. SANJANA, DR.RUPA, DR. MANI MOHAN, DR.LAKWAN, Dr. LAKSHMISHA** fellow postgraduate colleagues, seniors, juniors for their constant motivation and countless help.

I thank all my interns and nurses for their help and support.

Last but not least, I thank all my patients involved in this study, without whose co-operation, this study would not have been possible.

Dr TUNGALA LEELA PAVAN

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ABBREVIATIONS

Glossary	Abbreviations
CIMT	Carotid Adventitial Thickness
CKD	Chronic Kidney Disease
BMI	Body mass index
GFR	Glomerular Filtration
CVD	Cardiovascular Disease
ECG	Electrocardiogram
MDRD	Modification of Diet in Renal Function
CGN	chronic nephropathy
ATN	Acute Tubular Necrosis
ASE	American Society of Electrocardiography

ABSTRACT

Introduction: In the general population, carotid adventitial thickness (CIMT) forecasts future vascular events. But little research has been done on how chronic kidney disease (CKD) stages and conventional cardiovascular risk factors relate to CIMT. High-resolution B-mode ultrasonography was used to evaluate CIMT in order to compare it to old-style cardiovascular risk variables such age, body weight (BMI), lipid abnormalities , and different stages of CKD patients**.**

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Key words: carotid intima-media thickness; chronic kidney disease; glomerular filtration rate.

INTRODUCTION

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Around 8 to 16% of people worldwide have chronic kidney disease (CKD), which is indicated by a rate of glomerular filtration (GFR) about < sixty mL/min/1.73m2 that has persisted for more than 3 months. Compared to high income nations, low and medium income countries have a heavier burden from CKD [1]. From modest renal dysfunction to total failure, there are 5 stages of kidney disease in CKD. Individuals with CKD in stages 3 or 4 have a greater chance of dying or developing end-stage renal disease [2]. According to global burden of disease estimates there were 1.4 million CKD related deaths in the world in 2019 which is a 20% increase from 2010. The main factors responsible for increased burden of CKD in the global are obesity, hypertension, diabetes and cardiovascular diseases [3]. Along with the above listed variables, further explanations for the prevalence of CKD in South Asian nations like India include environmental pollutants and high levels of fluoride underground [4].

India recorded a 38% rise in the percentage of renal failure-related mortality between 2001-03 and 2010-13[5]. CKD is a significant contributor to the risk of cardiovascular disease (CVD). Even in the early stages of renal illness, the risk of heart disease is higher[6]. Early stages of CKD are associated with a 2 to 4 times increased risk of cardiovascular death [7].

Nearly 7.6percent of total of cardiovascular deaths worldwide in 2017 were caused by CKD-related illnesses, and CKD is also responsible for 25.3 million CVD -related disability adjusted life years [8]. The dysregulation of minerals such as phosphorous amd calcium, vessel calcification, anaemia, and hyperhomocysteinemia that CKD promotes raises the risk of cardiovascular disease [9]. CKD raises the likelihood of heart disease without increasing the risk from other traditional cardiovascular disease risk factors [6]. Richard Bright made the initial suggestion in 1836 that cardiovascular disease and kidney illness are connected. The level of proteinuria and renal impairment, as well as the rate that these changes take place, all

influence the risk of heart disease in individuals with CKD. Albuminuria, anemic, thyroid dysfunction, metabolic bone disease, increased Homocysteine, inflammation, malnourishment, endothelial atrophy, and antioxidant damage are among the non-traditional CKD associated heart disease risk factors that are ureamia specific. These variables increase the rate of atherosclerosis and the progression of CKD in addition to established risk factors such advancing age, hypertension, dyslipidemia, diabetes, smoking, and obesity[10]. The absence of the traditional trifecta of ischemic symptoms, high cardiac biomarker levels, and electrocardiogram (ECG) alterations makes it difficult to diagnose heart disease in CKD patients [11].

Due to the absence of adequate medical facilities in underdeveloped nations like India When glomerular filtration rate gfr is below 15mL/min/1.73 m2, 50percent of CKD patients receive their first diagnosis [12]. Even mild renal failure significantly raises the risk of cardiovascular illness due to a number of factors, including aberrant myocardium reorganization, ventricular hypertrophy, arrhythmia, and cardiac arrest. In contrast to people with normal kidney function, atherosclerotic disease is overrepresented across the full range of CKD patients[13]. The presence of a chronic inflammatory state, disturbances in calcium-phosphate breakdown, antioxidant damage, malnourishment, anaemia, hypervolemia, disruptions in the coagulation system, buildup of active compounds, and unknown lethal agents all contribute to the increased occurrence of coronary artery illness in CKD patients [14]. It is noted that composition of atherosclerotic plaques in CKD patients is different from those present in sclerotic subjects with standard kidney function. CKD subjects have atherosclerotic plaques with more calcium deposits, reduced collagen fibres and smooth muscle cells, and more inorganic substances [15]. Aging process of vascular and peripheral blood cells is one of many factors contributing to the complicated phenomena of vascular injury in CKD patients [16].

A direct inspection of the vessel wall is required to diagnose atherosclerosis since it is asymptomatic unless it is severe. Common carotid artery carotid-intima media thickness measurement using B-mode Early atherosclerotic process monitoring using ultrasound is non-invasive [17]. But because CIMT values change depending on the imaging side, the measurement spot, the measuring angle, and the cardiac cycle phase, evaluating the severity of atherosclerosis using this technique requires complex ultrasound techniques. Single-site CIMT assessments have been found to have lesser sensitivity. Carotid artery evaluation using imaging from a single viewpoint is not possible in three dimensions [18]. The non-invasive tools for detection of atherosclerosis including typical symptoms, electrocardiogram, and cardiac stress testing are found to be less reliable in CKD subjects [19]. Serum biomarkers measurements used in non-CKD subjects for diagnosis of cardiovascular diseases cannot be used in CKD subjects as in these subjects the biomarkers are elevated without myocardial infarction and myocardial apoptosis [20]. Recently eGFR measurements are being considered for diagnosis of atherosclerosis. Kidney disease may now be easily detected thanks to the development of an equation for estimating estimate GFR (eGFR) from blood creatinine based on equations that take into account a variety of patient variables. This method converts the serum creatinine value into physiologic units of GFR [21]. Among various equations available the 4 variables Modification of Diet in Renal Function (MDRD) equation is widely used for calculation of eGFR. There are certain limitations even with this equation which have been recently overcome by introduction of another equation by Chronic kidney Disease Epidemiology collaboration. In the region of low creatinine levels and higher GFRs, this equation has been proven to give a more precise estimation of GFR. This study seeks to measure carotid adventitial medial thickness in individuals with various stages of CKD and investigate the relationship between carotid adventitial medial thicknesses and eGFR in these patients.

Need for the study:

The burden of CKD is gradually increasing in India. Diabetes and hypertension are major causes attributable to development of CKD in developed countries [22]. In recent years the burden of diabetes and hypertension has increased in India and it is projected that 40 to 60% of CKD cases found in the country are related to these causes [23]. According to data from the Indian Council of Medical Research, the occurrence of diabetes and hypertension among Indian adults is 7.1% and 17%, respectively. The prevalence of CKD would inevitably rise in India along with the burdens of hypertension and diabetes. Screening for CKD related cardiovascular diseases is highly challenging in India as majority of population lack access to proper well equipped medical care facilities. Diagnosis and screening for CKD patients with risk of developing atherosclerosis using carotid intima media thickness measurement may not be possible in resource limited settings. Even if available it will be a burden for majority of CKD patients to get CIMT measurements done using ultrasound after getting diagnostic test done for CKD. It would be highly advantageous if single eGFR measurement which reflects kidney damage status also helps in assessment of risk of atherosclerosis in CKD patients. Given the aforementioned information, the goal of this study is to measure the carotid adventitial medial thickness in CKD patients at various stages and investigate the relationship between these measurements and eGFR.

AIMS AND OBJECTIVES:

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1)To determine the carotid intimal medial thickness in various stages of CKD patients

2)To study the correlation of carotid intimal medial thicknesses with eGFR in various stages of CKD patients

REVIEW OF LITERATURE:

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Chronic kidney disease:

Definition:

A persistent alteration in renal architecture or efficiency for more than three months is considered to be chronic renal disease[24]

Glomerular filtration:

The excretory, hormonal, and metabolic activities of the kidney are only a few of its many roles. One facet of excretory function is glomerular filtration. Due to the reduction in rate of glomerular filtration following structural or functional injury to the kidney, it is regarded as an overall indicator of renal function.

The rate of glomerular filtration is a crucial indicator of renal health. GFR (Estimated glomerular filtration) measures the volume of fluid filtered into Bowman's capsule from glomerular capillaries per time unit [25]. GFR levels are predicted to be 120 and 130 ml/min/1.73 m2 in young men and women, respectively, and depend on age, sex, and body surface [26].

Table 1: The following table give glomerular filtration rate classes in CKD:

GFR category	GFR (ml/min/1.73m^2)	Terms
G1	Greater 90	standard or high
G2	60 to 89	Slightly decreased
G3a	40 to 59	Slight to modest decrease
G3b	30 to 44	Modest to severe decrease
G4	15 to 29	Sternly decreased
G5	Greater 15	Kidney failure

eGFR: Approaches for translating the plasma creatinine value into the GFR result have been developed through the development of formula-based eGFR calculations. For the purpose of calculating eGFR, the following 4-variable Modulation of Dietary in Renal Impairment equation is frequently used:

eGFR (millilitres) = 186 (S.Cr in mol/l 0.011312) -1.154 (age) -0.203 (0.742 if female) (1.212 if African-American Black).

A new equation was created once it was discovered that the previously mentioned one had several shortcomings. The reviewed equation was tested down to serum creatinine concentrations of 62 mol/L in women and 80 mol/L in men (in men). In the spectrum of decreased plasma creatinine levels and higher GFRs, CKD-EPI was proven to be a more accurate measure of GFR [27]. The following are the equations:

For women with creatinine lesser than 62 µmol/L:

$eGFR\ (mL/min/1.73m^2) = 144 \times (Cr/61.6)^{-0.329} \times (0.993)^{Age}$

For women with creatinine greater than 62 µmol/L:

$eGFR\ (mL/min/1.73m^2) = 144 \times (Cr/61.6)^{-1.209} \times (0.993)^{Age}$

For men with creatinine lesser 80 µmol/L":

$eGFR\ (mL/min/1.73m^2) = 141 \times (Cr/79.2)^{-0.411} \times (0.993)^{Age}$

For men with creatinine greater 80 µmol/L:

$eGFR\ (mL/min/1.73m^2) = 141 \times (Cr/79.2)^{-1.209} \times (0.993)^{Age}$[27].

Epidemiology:

In 2010, it was reported that among those under 20 years old, the age-standardized worldwide incidence of CKD stages 1 to 5 was 10.4% for males and 11.8% for women[1]. According to more current data, the incidence of CKD in the world is 10.6% for stages 3-5 and 13.4% for

stages 1–5 [28]. The occurrence of each stage of CKD was 3.5% for stage 1, 3.9% for stage 2, 7.6% for stage 3, 0.4% for stage 4, and 0.1% for stage 5[28]. According to research looking at the occurrence of CKD worldwide, 843.6 million people are now estimated to be afflicted by Stages of ckd 1 to 5 globally[29].

The estimated incidence of CKD in various parts of India varied from 1% to 13%, and most recently, statistics from the Kidney Disease Data Center Study of the International Society of Nephrology revealed a prevalence of 17%[30]. In 2017, there have been 697 million new cases of CKD globally. Two countries, India and China, were home to about 1/3rd of the patients with CKD.

In 2017, it was projected that 91% of people worldwide had CKD, with stages 1-2 accounting for 50%, stages 3 for 39%, stages 4 for 06%, stages 5 for 07%, dialysis accounting for 04%, and kidney transplantation accounting for 01%. Females had a 129 times greater time of life prevalence of CKD than males did. Males had a 147 times higher age-standardized incidence of hemodialysis and transplant than females. Males had a 139 times higher time of life CKD mortality rate per 100,000 people than females did. The average prevalence of CKD nationwide was 89%.[31]

Aetiology:

Between industrialised and developing nations, there are notable differences in the aetiology of CKD, as well as potential variations in the processes driving CKD development. Although type 2 diabetes and hypertension are among the most frequent causes of CKD in industrialised and many emerging nations, chronic nephropathy (CGN) has been described as the primary cause in several developing nations, such as China, with diabetes and hypertension coming in second and third [32].

Prerenal Illness

Chronic prerenal illness, which increases the likelihood of numerous events of an inherent kidney damage such as tubular atrophy, occurs in individuals with longstanding cardiac problems or hepatitis with chronically diminished renal perfusion (ATN). Over time, this results in a gradual decrease of renal function [33].

Disease of the intrinsic renal arteries

The most common chronic renal vascular illness is nephrosclerosis, which causes prolonged vasculature damage to the nephrons [34].

Glomerular Disease that is Internal (Nephritic or Nephrotic)

infectious endocarditis, shunt nephritis, lupus nephritis, Post-streptococcal GN, Goodpasture syndrome, IgA nephritis, and vasculopathy are the most frequent causes [35,36]

Postrenal (Obstructive Nephropathy)

The most prevalent causes of chronic blockage include prostatic illness, and nephrolithiasis. One uncommon cause of prolonged ureteral blockage is retroperitoneal fibrosis. [33]

Classification:

The 2012 KDIGO CKD classification divides the disease into 6 groups based on glomerular filtration rate and offers recommendations on the source of the condition (G1 - G5 with G3 split into 3a and 3b).

The six categories consist of:

• G1: GFR more than 90 mlper min per 1.73 m2; G2: GFR between 60 and 89 ml per min per 1.73 m2; and G3a: GFR between 45 and 59 ml/min per 1.73 m2

• G5: GFR just under 15 ml per min per 1.73 m2 or therapy with dialysis; G3b: GFR 30 to 44 millilitres per 1.73 m2; G4: GFR 15 to 29 ml per min in 1.73 m [37,38.]

Pathophysiology:

Nearly 400 ml/100 g of tissue of blood go through the kidneys/ minute. In compared to other well-perfused arterial beds including the liver, heart,and brain, this flow rate is significantly higher. As a result, more potentially hazardous circulating chemicals are exposed to renal tissue. Because glomerular filtration needs high intra- and transglomerular pressure, renal capillaries are more susceptible to hemodynamic damage than other capillaries. Both renal hypertension and hyperfiltration play a major role in the progression of CKD. Disruption of the glomerular filtration membrane's electrostatic barrier is the cause of many types of glomerular damage. The glomerular damage is disseminated to the tubulointerstitial compartment by the sequential arrangement of the nephron's microvasculature and the downstream location of the tubule. Each individual glomerulus component is essential to its correct operation. Any portion that is damaged will impact the others.

- Mechanisms of glomerular, tubular, and vascular damage are involved in the pathophysiology of CKD.

- Glomerular dysfunction: Injuries caused by the immune system, as well as metabolic and mechanical stress, cause acquired glomerular disease. The 3 kinds of glomerular disorders are as follows:Non proliferative glomerular disease: Glomerular pathological conditions wit inflammatory glomerular and with no immunoglobulin deposition , or with immunoglobulin deposition albeit without nephron inflammatory response, most likely as a result of immunoglobulin lamina propria clustering.

- Immunoglobulin accumulation and progressive glomerular disorders with elevated cellularity (IgA nephropathy, severe glomerular damage and inflammation, without immunoglobulin deposition, or, lupus nephritis, anti-GBM, relatively nontoxic GN).
- Heterogeneous category of glomerular disorders in systemic illnesses such hyperglycemia, paraproteinemia and amyloidosis.[39]

Renal damage that is not immunologic.

- Glomerular impairment can be brought on either on its own or in combination with immunological processes by hemodynamic, metabolic, and chemical damage.[39]
- Glomerular damage may come from systemic hypertension that affects the glomeruli as well as renal hypertension brought on by specific alterations in glomerular hemodynamics. Normal autoregulation serves to protect the kidney from systemic hypertension, but it can be overridden by high blood pressure, which means that systemic hypertension directly damages the glomerular filtration barrier. Chronic hypertension causes secondary sclerosis, glomerular and tubulointerstitial atrophy, and arteriolar vasoconstriction and sclerosis[39].

Whatever the source, glomerular hypertension is often a nephron's way of adapting to the increased workload brought on by nephron loss. Due to the buildup of ECM, the persistent intraglomerular hypertension stimulates the formation of mesangial matrix and causes glomerulosclerosis.[39].

Tubulointerstitial impairment:

A number of distinctive characteristics of tubulointerstitial impairment include an infiltrate of inflammatory cells which is caused by both the stimulation of occupant proinflammatory

cytokines and the enrollment of circulating immune cytokines; an increase in intercellular fibroblasts as a result of increased emergence and decreased cell death of resident interstitial cells; and the presence of myofibroblasts that express the cytoskeletal protein. Proinflammatory, vasoconstrictive, and profibrotic factor expression is known to increase as fibrosis progresses.[39]

Diagnosis:

Screening may be essential for early illness detection because the most of CKD patients show no symptoms. The Renal Foundation developed a kidney profile test that evaluates both creatinine clearance for estimating GFR and urine ACR.. Generally speaking, risk-based screening is advised for the identification of CKD. Screening is recommended for individuals above 60 years with diabetes or hypertension. Screening is also recommended if individuals have any of the following mentioned risk factors:

Clinical risk factors:

• Nephrotoxic drugs; • Diabetes; • High blood pressure; • Autoimmune conditions; • Systemic infections (hepatitis B and C virus HIV);

Malignancy, obesity, renal mass loss, kidney stones, recurrent urinary tract infections, blockage of the urinary system

Smoking, having a history of severe renal damage, and using intravenous drugs (eg, heroin, cocaine)

• Kidney illness in the family

Risk factors for demographical changes include:

Age greater than 60 years, non-white race, low wage, and poor education

Genetic factors

- Sickle cell disease and its risk genes
- Congenital abnormalities of the urinary and renal system, Alport syndrome, polycystic renal disease and other family causes

Management:

Lowering the risk of heart disease: Since CKD patients have a greater chance of evolving the illness, lowering this risk is a key part of care. This covers quitting smoking, keeping blood pressure at 140/90 mm Hg, and treating over 50 years of age with moderate and low doses of statins.[40]

Treatment of hypertension: Individuals with diabetes and a urine albumin to creatinine ratio of 30 mg every 24hrs or adults without diabetes but with a urine albumin to creatinine ratio of 300 mg per 24 hours should employ the renin-angiotensin-aldosterone system with whichever an ACE-I or an ARB. Subjects who have reduced ejection fraction, resilient hypertension, or albuminuria must get aldosterone receptor opponents [41,42].

Diabetes care: Blood glucose control slows the course of CKD, making diabetic management crucial. The dosage of oral hypoglycemic medications must be changed.

Nephrotoxins: CKD patients must avoid nephrotoxin usage. Some generally used nephrotoxins are Non-steroidal anti-inflammatory drugs,[43] herbal remedies, phosphate based bowel preparations, [44] proton pump inhibitors[45] Proton pump inhibitors are found to increase risk of atherosclerosis.

Dosage modifications are frequently necessary for CKD patients. The majority of antimicrobials, direct oral anticoagulants, anticonvulsants , oral anti diabetic medicines, insulin, anti - cancer agents, and opiates are common treatments that call for dose reductions.[46, 47]

Dietary maintenance: According to the KDIGO recommendations, protein consumption should be decreased in people with CKD stages G4 to G5 to very little than 0.8 g/kg daily and in other adult ckd patients who are at risk of advancement to fewer than 1.3 g/kg each day[47,48]. Limiting dietary protein may provide benefits, but they must be balanced against the possibility of causing protein malnutrition or dietary deficiency.[49] Patients with high blood pressure, proteinuria, or hypervolemia are advised to follow low-sodium diets, which typically contain less than 2 grams per day. [46]

Complications:

Drug toxicity : Kidney disease (CKD) affects the way medications are eliminated by the kidneys and increases the risk of prescription medications. This requires dosage adjustment of drugs and errors in dosage in CKD patients results in systemic toxicity.

Metabolic and endocrine complications: CKD causes loss of endocrine or exocrine functions of kidney resulting in anaemia, acidosis, malnutrition, bone and mineral disorders. GFR of less than 60 ml/min/1.73 m2 increases the risk of all causes and cardiovascular death. [24]

Table 2: The following table give tests and management of CKD related complications:

Complication	Relevant tests	Frequency of testing	Management
Anaemia	Haemoglobin	Stages G1 and G2- when clinically indicated. Stage G3 – once a year. Stages G4 and G5- twice a year	Consider iron supplementation after ruling out other causes of anaemia like iron deficiency, vitamin B12 deficiency, folate deficiency, occult bleeding[50].
Mineral and bone disorder	Serum calcium phosphate, parathyroid hormone, 25-hydroxy vitamin D	Serum calcium phosphate in Stage G3- every 6 to 12 months. Stage G4- every 3 to 6 months. Stage G5- every one to three months	Consider phosphate lowering therapy and vitamin D supplementation[50].

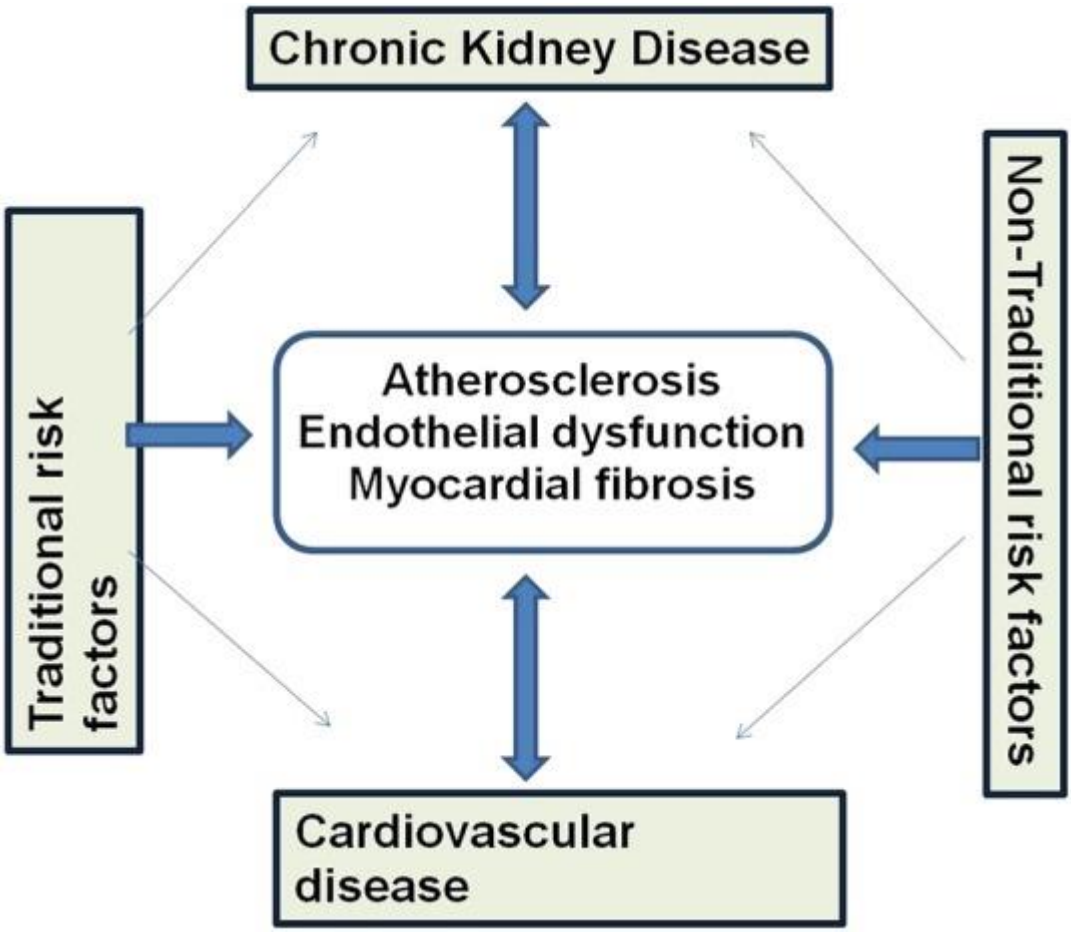
Association of CKD to CVD

Any heart disease is twice as common in CKD patients. The risk of death due to cardiovascular rose by 5% for every ml per 1.73 m2 decline in eGFR, according to a study[51]. The classical risk factors for atherosclerosis in over-all population are different from risk factors for CKD patients[52].

Table 3: The following table describes classical risk factors and risk factors in CKD subjects for atherosclerosis:

Classical risk factors in general population	Emerging risk factors in CKD patients
Age	Mineral bone metabolism
Male gender	Vascular calcification
Hypertension	Uraemic toxins
Left ventricular hypertrophy	Abnormal lipid modifications
Smoking	Inflammation
Diabetes	Oxidative stress
Dyslipidaemia	Endothelial dysfunction [52]
Physical inactivity [52]	

Figure 1: The following figure shows complex relationship between traditional risk factors and emerging risk factors, cardiovascular disease and CKD:



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Pathophysiology of Cardiovascular illness in CKD:

Subjects with CKD develop cardiovascular disease due to two main processes. One is the kidney's secretion of hormones, enzymes, and cytokines in reaction to renal damage, which causes recognisable alterations in the vasculature. The second mechanism is that heart injury is caused by CKD-related mediators and hemodynamic changes [53].

Old-styled risk factors of vascular illness in CKD:

These include insulin resistance, hypertension, dyslipidaemia and smoking which cause atherosclerotic vascular sequelae and CKD progression [54].

Other risk factors of vascular disease in CKD:

Smooth muscle vascular cells in the median layer of vessels shift from having a contractile gene to having a more synthetic character as a result of CKD's hemodynamic alterations [55]. The major mechanism that causes CKD is inflammation. Numerous infections, such as gum disease, reactive oxygen species (ros brought on by the buildup of advanced glycation products, hypokalemia, decreased chemokines clearance, insulin sensitivity, post-translational modifications of bloodborne molecules, and epigenetic factors are all included in proinflammatory processes[56].

Carotid-intima media thickness (CIMT):

Definition:

CIMT is defined as the area of tissue starting at the luminal-intimal interface and the media-adventitia interface of CCA [57].

The method for CIMT evaluation has been standardised by the American Society of Electrocardiography (ASE) inside a consensus statement. [58] Following the recommended imaging technique and paying great attention to the apparatus are essential for CIMT assessment since minor mistakes might place patients in various risk groups. CIMT employs transducers that generate acoustics or sound waves for ultrasound imaging. In ultrasonic imaging, sector staged and linear phased-array transducers are employed. Due to their superior picture quality, linear phased-array transducers (versions A and B) outperform sector phased-array. With increased picture quality, ergonomics, and trapezoidal imaging formats, these transducers are currently assisting physicians in making correct diagnoses[59].

CIMT assessment in cardiovascular risk prediction:

The common carotid artery is the first of four segments that make up the carotid artery. The ECA and ICA are formed by the carotid bulb, which arises from the common carotid artery. At the carotid bulb's outer wall, where shear forces are moderate and fluctuations in shear force are large, atherosclerosis tends to occur. CIMT measurement aids in the early diagnosis of occlusive atherosclerosis [18]. A non-invasive technique for tracking the early stages of the atherosclerotic process is B-mode ultrasound measurement of the carotid-intima media cross - sectional area of the CCA [17].

The side of the imaging field, the measurement point, the measurement angle, and the stage of the cardiac cycle all affect CIMT measurements differently. The sensitivity is decreased by CIMT measurement at a single location. A single-angle imaging does not provide a three-dimensional assessment of the carotid artery. Systolic luminal dimension expansion causes the CIMT to thin during systole, resulting in lower CIMT values at the end of systole than at the end of diastole. For a thorough assessment of the degree of atherosclerotic load and to see a treatment's effects, extensive ultrasonography procedures are needed.

CIMT readings between 0.6 to 0.7 mm were deemed typical in healthy middle-aged people, but CIMT of 1 mm or higher has been linked to a considerably higher absolute risk of CHD60 [60].

The reported average and highest CIMT readings for healthy Indian individuals were close to 1 and 0.70 millimeters, respectively. The evaluation of CIMT fluctuates with age, and in younger populations, readings >1.0 mm are deemed abnormal and have a generally higher risk of CHD. [61,62,63.]

Advantages of CIMT assessment:

In clinical practise, ultrasonographic measurement of CIMT provides a number of benefits over angiography for monitoring atherosclerotic vascular alterations and the onset of atherosclerosis [64].

CIMT has no negative effects on the individuals and may be applied frequently and reproducibly. As opposed to indirect biomarkers like low-density lipoprotein cholesterol or sophisticated biomarkers like hsCRP or membrane protein alkaline phosphatase A2 (Lp-PLA2), CIMT directly visualises the vasculature and can detect atheromatous illnesses in initial and no symptom stages. CIMT monitoring protocol can discern atherosclerotic illnesses in early and show no symptoms stages.

It allows in determining the exact area of CVD instead of only lumen and aids in measuring the lesion unlike CT-CAG

• Unlike some other assessment techniques, such as the coronary artery calcification score, CIMT does not depend on the calcium deposition of the lesion.

• CIMT is safer than other imaging procedures like carotid interrogation since it contains no radiation [64].

Disadvantages of CIMT assessment: [65,66,67]

- Because there is no established technique for measuring CIMT, follow-up studies and assessments of treatment interventions' effects on the measured CIMT may result in erroneous assessments of the CIMT's progression and regression.

- The value of the measured CIMT may vary according on which carotid artery is used to test it, including the common carotid, bifurcation, internal carotid, and combination CIMT. In order to increase repeatability and reduce reader variance, edge detection application software were built because the atherosclerotic approach is confined to the intimal layer of the artery wall.

- Since CAD represents the most common reason for fatal cardiovascular events, CIMT only offers a hazy assessment of the possible atherosclerotic burden in the coronary vessels since it depicts major blood vessels instead of coronary. The carotid artery might not even genuinely contain atherosclerosis, despite the fact that CIMT discovered it there.

Relevant studies:

- Rizikalo, A., et al.[68] assessed the association between CIMT and GFR in subjects without diabetic CKD . During the 4-year follow-up, this study showed a strong correlation between GFR and CIMT in quasi stage 2 and stage 4 CKD.
- Nitesh, C., et al. [69] the association between CIMT and conventional cardiovascular risk variables such age, BMI, hyperlipidemia, and different phases of CKD patients. Despite the fact that CIMT was somewhat higher in individuals with advanced CKD, no statistically relevant link was discovered between CIMT and eGFR.
- Kawamoto, R., et al.[70] examined the relationship between carotid atherosclerosis and the glomerular filtration rate. The study discovered that in both men and women, regardless of common cardiac risk factors, eGFR was linked to an augmented incidence of carotid atherosclerosis.
- Polak, J, F., et al.[71] studied connotation amognst the thickness of carotid intima media and CVD. The highest maximum and mean thickness of common carotid artery intima-media together predict cardiovascular events, according to the study's findings.

- Benedetto, A, F., et al. [72]found that IMT in dialysis subjects is linked to LV eccentric enlargement and is a standalone predictor of mortality rates, based on the study's findings. IMT may be applied to the dialysis population to help with risk classification.

- Nishizawa, Y., et al.[73] investigated if thickness of carotid artery intima-media may predict risk of coronary heart disease in individuals with ESRD. According to the study's findings, elevated CA-IMT is a reliable indicator of cardiovascular death among patients receiving hemodialysis.

- Hinderliter, A., et al. [74] study found that increased maximal IMT was an accurate indicator of cardiac disease and mortality among individuals with end staged CKD pre-dialysis and was linked with conventional coronary risk factors, subclinical CVD, and CVD alone as the indicators.

- Lyngdoh, L., et al. [75] The non-invasive sensitive instrument for CIMT assessment is B-mode ultrasound. This method may help us identify individuals with atherosclerosis load since CKD is linked to faster atherosclerosis and a subsequent increase in cardiovascular death.

Lacunae in literature:

Chronic kidney disease (CKD), which significantly contributes to fatalities and disability from non-communicable diseases, should be vigorously treated if the UN's Sustainable Development Goal of lowering early mortality from non-communicable illnesses by the a third by 2030 is to be met. CVD, which is the leading cause of early death and disability-adjusted life years, is significantly increased by CKD. Opportunities for secondary and primary CKD prevention are frequently missed in underdeveloped countries like India. In

order to further the body of information on correlation, this study aims to assess carotid adventitial medial width in patients with various stages of CKD and look into the relationship among carotid adventitial median thicknesses and eGFR on the same individuals. This will make it easier to calculate the eGFR needed to forecast atherosclerotic risk in those with chronic renal disease.

MATERIALS AND METHODS

MATERIALS AND METHODS

Source of data:. This is an case control study in patients at various stages of CKD patients treated at RL JALAPPA HOSPITALS, KOLAR

Study design: A Case Control Study

Study period: 1 year January 2021 to December 2022

Method of collection of data: Patients admitted with diagnosis of various stages of CKD treated at RL Jalappa Hospitals and Research Centre, Kolar were selected in a randomized manner

Inclusion Criteria:

1) After receiving approval from the institute's ethics committee between the years of 2020 and 2023, all subsequent CKD patients as well as a similar number of controls who were of a similar age and sex were enrolled in the study.

2) age >12 years

3) Patients with type 2DM, Hypertension are also included in the study and are considered as confounding factors

Exclusion Criteria:

1) Individuals with a diagnosis of acute renal damage and those with a history of carotid operation were exempted from the trial.

2. Patients on drugs causing dyslipidaemia and dysglycemia like Diuretics, beta blockers, steroids, anti-psychotics.

3. Conditions affecting hemoglobin- methemoglobinemia, anemia, polycythemia

Methodology:

- They were explained about the procedure and their consent was taken and they were subjected to blood investigations and carotid Doppler.

- Clinical, laboratory and sociodemographic data was elicited and recorded in a predefined proforma

  1.Socio demographic data

  - Age
  - Gender

  2.Clinical data

  - Height
  - Body weight
  - Clinical examination
  - BMI

  3.Laboratory data

- The patients will be age, gender, period of diabetes ,BMI matched and their results were correlated in two different groups separately.
- In **GROUP ONE**, which consists of 39 patients, eGFR in CKD was correlated with carotid intima media thickness and the results were analysed.
- In **GROUP TWO**, which consists of 39 patients, eGFR IN non CKD was correlated with carotid intima media thickness and the results were analysed separately.

Sample size:

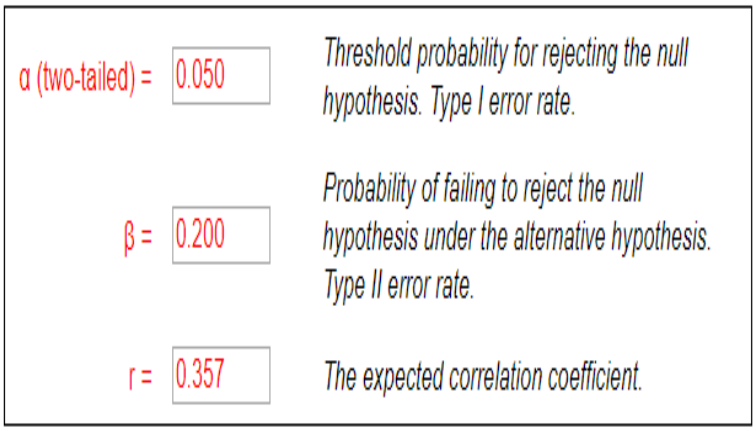
Sample size was estimated by using correlation coefficient (r) of eGFR with CIMT as 0.357 (i.e. r = 0.357) from the study by Kavita Bendwal et al. Using these values at 95% confidence level and 80% power and substituting in the below formula, sample size of 29 was obtained.

Considering 10% Non response rate a sample size of 39 + 39 = 78 subjects with CKD in various stages

Sample size overall = N = [(Z+Z)/C] 2 + 3

The typical deviation for = Z = 1.960

The standard normal deviation for r = Correlation coefficient = 0.357 C = 0.5 * ln[(1+r)/(1-r)] = 0.373 is equal to = Z = 0.842 r = 0.357



N = 39

Investigations :

- CAROTID DOPPLER
- CBC
- eGFR USING MDRD equation
- BLOOD PRESSURE
- RENAL FUNCTION TEST

eGFR is calculated by using MDRD GFR in mL/min per 1.73 m^2 = 175 x $SerumCr^{-1.154}$ x $age^{-0.203}$ x 1.212 (if patient is black) x 0.742 (if female)

Statistical Methods

Comorbidities, lab results, investigational parameters, etc. were all taken into consideration as the main outcome variables. CKD stage and EGFR were thought to be the main explanatory factors. Primary characteristics such as age, gender, and BMI were thought to be significant to the investigation.

For quantitative variables, the standard deviation and mean were used in the descriptive analysis, while frequency and percentage were used for categorical variables.

Additionally, the data was shown utilising the relevant designs, such as pie and bar charts.

Using the Chi square test, categorical results were compared between research groups.

Using Independent sample t- test (2 groups) and ANOVA, the mean values for normal distributions quantitative parameters were compared across study groups (3 groups). Using the Mann Whitney u test (2 subgroups) and the Kruskal-Wallis test, measures of central tendency and interquartile (IQR) for quantitative parameters with non-normal distribution were examined between study groups (3groups). By computing the spearmans rank coefficient of correlation and plotting the data in a scatter diagram, we were able to determine the relationship between the quantitative explanatory factors and the outcome variables. Statistical significance was defined as a P value 0.05. The coGuide Statistics programme, Version 1.0, handles data analysis.

RESULTS

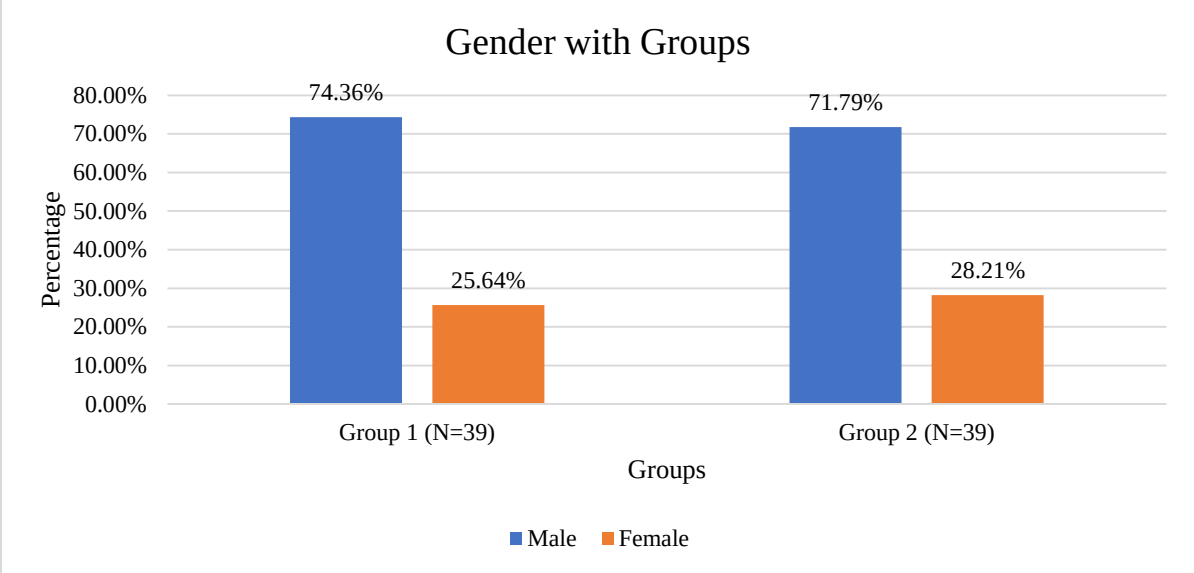
RESULTS

A whole of 78 subjects were considered into the study.

Table 4: Gender distribution as per the study gorups (N=78)

Gender (M/F)	Groups		Chi square value	P value
	Group 1 (N=39)	Group 2 (N=39)		
Male	29 (74.36%)	28 (71.79%)	0.07	0.7985
Female	10 (25.64%)	11 (28.21%)		

Figure 2: Grouped bar graph of Gender with Groups



In Group 1, 29 (74.36%) were male and 10 (25.64%) were female. In Group 2, 28 (71.79%) were male, and 11 (28.21%) were female. The difference in gender between groups was found to be insignificant with a P- value of 0.7985. (Table 4 & Figure 2)

Table 5: Comparison of Age (in year) with Groups in the study population (N=78)

Age	Groups		Significance Value (P)
	Cluster 1 (N=39)	Cluster 2 (N=39)	
	58.05 ± 12.35	56.28 ± 13.14	0.5419

There was insignificance difference in mean of age between two study samples statistically 58.05 ± 12.35 and 56.28 ± 13.14 in group 1 and group 2 resepectively. (Table 5)

Table 6: Comparison of Age (in year) across CKD stages (N=78)

Age (in year)	Stage of CKD				Chi square value	P value
	Stage 1 (Normal)	Stage 3	Stage 4	Stage 5		
<=48 (N = 20)	11 (55.00%)	3 (15.00%)	1 (5.00%)	5 (25.00%)	2.00	0.9199
49-64 (N = 31)	15 (48.39%)	3 (9.68%)	4 (12.90%)	9 (29.03%)		
>64 (N = 27)	13 (48.15%)	5 (18.52%)	2 (7.41%)	7 (25.93%)		

In age group <=48 years, 11 (55.00%) participants were in stage 1, 3 (15.00%) participants were in stage 3, 1 (5.00%) participant were in stage 4 and 5 (25.00%) participants were in stage 5. In age group 49-64 years, 15 (48.39%) participants were in stage 1, 3 (9.68%) participants were in stage 3, 4 (12.90%) participants were in stage 4 and 9 (29.03%) participants were in stage 5. In age group >64 years, 13 (48.15%) participants were in stage 1, 5 (18.52%) participants were in stage 3, 2 (7.41%) participants were in stage 4 and 7 (25.93%) participants were in stage 5. The difference in proportion of age group among stages of CKD was reported as insignificant since the P- value was 0.9199. (Table 6)

Table 7: Association of BMI with CKD different Stages (N=78)

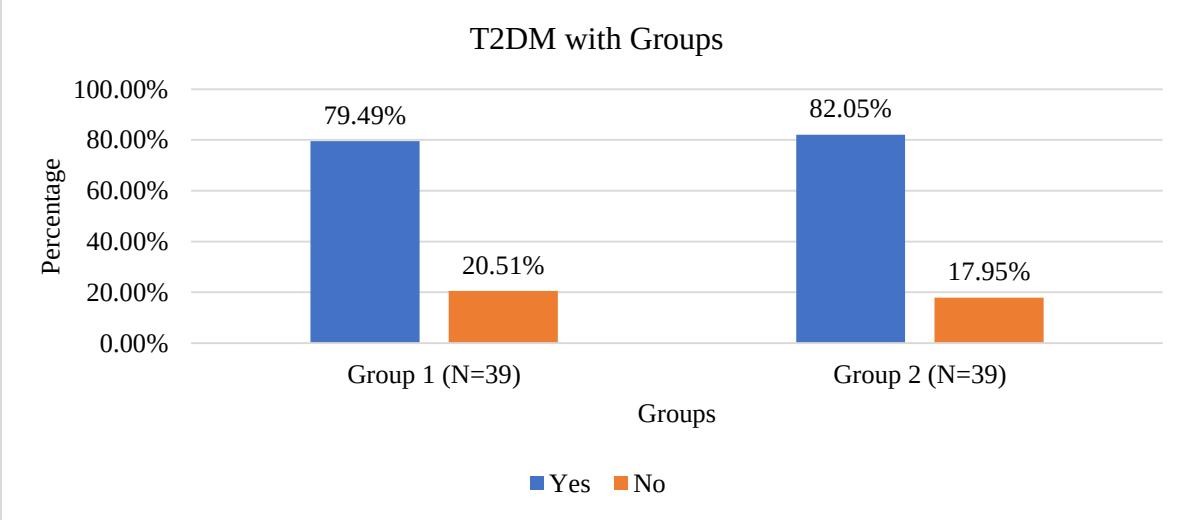
BMI	Stage of CKD				P Value [One way ANOVA]
	Stage 1 (Normal) (N=39)	Stage 3 (N=11)	Stage 4 (N=7)	Stage 5 (N=21)	
	24.59 ± 7.15	23.18 ± 2.04	25.14 ± 3.13	28.00 ± 4.69	0.0946

The mean bmi was 24.59 ± 7.15 in stage 1, 23.18 ± 2.04 was in stage 3, 25.14 ± 3.13 was in stage 4 and it was 28.00 ± 4.69 in stage 5, the difference in BMI among the stages of CKD indicated no statistical significance where the P value was 0.0946. (4th Table)

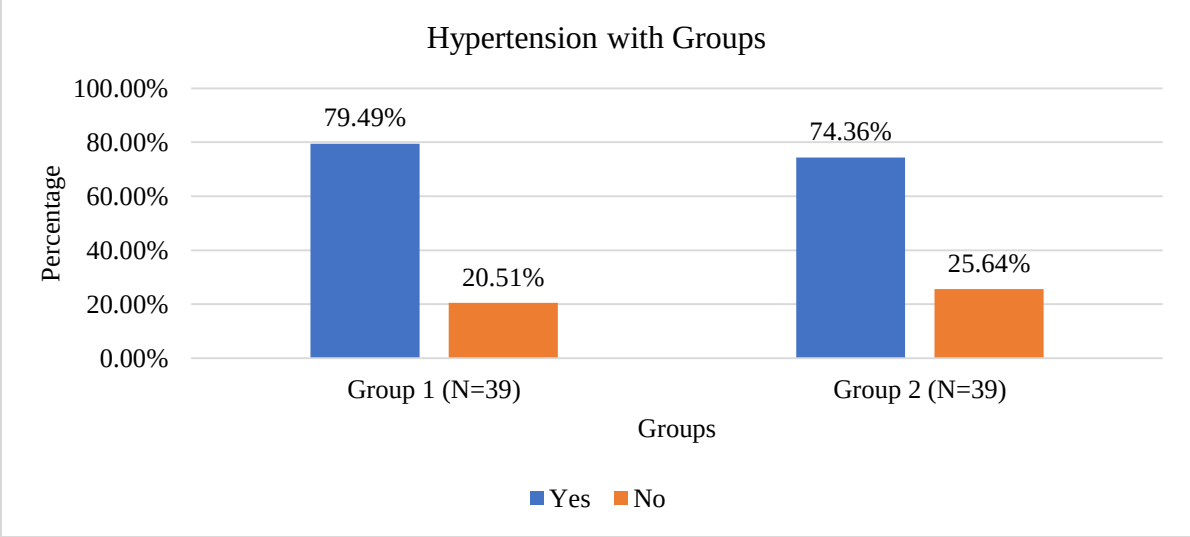
Table 8: Summary of Comorbidities in two study groups (N=78)

Comorbidities	Groups		Chi square	P value
	Cluster 1 (N=39)	Cluster 2 (N=39)		
T2DM				
Yes	31 (79.49%)	32 (82.05%)	0.08	0.7739
No	8 (20.51%)	7 (17.95%)		
Hypertension				
Yes	31 (79.49%)	29 (74.36%)	0.29	0.5909
No	8 (20.51%)	10 (25.64%)		

Chart 3: Grouped bars depicting T2DM with Groups



Graph 4: Hypertension distribution in each group with clustered bars



In the group 1, 31 (79.49%) people had T2DM and 31 (79.49%) people had hypertension. The difference in proportion of T2DM between groups was indicated as insignificant because the P- value was 0.7739. In the group 2, 32 (82.05%) people had T2DM and 29 (74.36%) people had hypertension. The difference in proportion of hypertension between groups reported insignificance since P- value = 0.5909. (Table 8 & Figure 3, 4)

Table 9: Lab parameters as per the study samples in two groups (N=78)

Lab parameters	Groups		P (Significance value)
	Cluster 1 (N=39)	Cluster 2 (N=39)	
Pulse	80.05 ± 9.03	81.87 ± 9.05	0.7739
Blood Pressure			
SBP	125.85 ± 20.76	127.64 ± 19.01	0.6915
DBP	77.69 ± 13.27	78.46 ± 12.04	0.7893

The mean pulse was 80.05 ± 9.03 in group 1 and in group2 81.87 ± 9.05, there was no significant difference in pulse rate in two groups of the study where P value= 0.7739. The mean systolic blood pressure (SBP) was 125.85 ± 20.76 in group 1 and it was 127.64 ± 19.01 in group 2. The mean diastolic blood pressure (DBP) was 77.69 ± 13.27 in group 1 and it was 78.46 ± 12.04 in group 2. the difference in SBP and also DBP indicated no difference in two samples of study significantly since the P value was>0.05. (Table 9)

Table 10: Summary of Investigations between Groups along with comparison (N=78)

Investigations Parameters	Groups		P Value
	Cluster 1 (N=39)	Cluster 2 (N=39)	
Hemoglobin	9.00(8.0 to 9.8)	15.00(14.0 to 16.0)	<0.001‡
WBC	8.00 ± 2.01	7.94 ± 2.05	0.9024†
PLT	156.00(140.0 to 170.0)	298.00(265.0 to 337.5)	<0.001‡
BI urea	79.00(67.5 to 128.0)	20.00(18.0 to 25.0)	<0.001‡
Sr. Creat	5.00(1.9 to 6.9)	0.60(0.4 to 0.7)	<0.001‡
Sodium	132.18 ± 3.74	135.75 ± 3.74	<0.001†
Potassium	4.60(4.3 to 4.8)	4.00(3.6 to 4.6)	<0.001‡

Note: †-Independent t test, ‡-Mann Whitney test

In group 1 and 2, the median of hemoglobin was 9.00 (IQR 8.0 to 9.8), 15.00 (IQR 14.0 to 16.0) respectively where the difference in HB values identified as significant statistically in two groups of the study (P Value <0.001). The mean WBC was 8.00 ± 2.01 in group 1 and in the other group 7.94 ± 2.05, but no statistical significance reported between the groups. (P value 0.9024). The difference of PLT, BI urea, Sr. creatinine and potassium between groups reported significance as per the P Value <0.001. The mean sodium was 132.18 ± 3.74 in group 1 and it was 135.75 ± 3.74 in group 2, P value <0.001 indicated the statistical significance in sodium values in two samples of the groups. (Table 10)

Table 11: Association of CIMT (Carotid artery intimal medial thickness) with Groups in the study population (N=78)

CIMT	Groups		P Value [Mann Whitney Test]
	Cluster 1 (N=39)	Cluster 2 (N=39)	
Left CIMT	7.50(5.25 to 8.45)	0.80(0.55 to 1.15)	<0.001
Right CIMT	7.30(5.55 to 8.2)	0.82(0.7 to 1.1)	<0.001
MEAN CIMT	7.70(5.88 to 8.75)	0.80(0.62 to 1.25)	<0.001

The difference in the (Left, Right, Mean) CIMT (Carotid artery intimal medial thickness) was statistically significant in the groups of the study (p value <0.05). (Table 11)

Table 12: EGFR compared between two groups (N=78)

EGFR	Groups		P Value [Mann Whitney Test]
	Cluster 1 (N=39)	Cluster 2 (N=39)	
	14.10(10.0 to 34.55)	172.00(137.5 to 264.0)	<0.001

In group 1, the median EGFR was 14.10 (IQR 10.0 to 34.55) and it was 172.00 (IQR 137.5 to 264.0) in group 2. EGFR showed significant difference in the study groups (P Value <0.001). (Table 12)

Table 13: Comparison of CIMT with CKD stages in the study participants (N=78)

CIMT	Non CKD	Stage of CKD			P Value [One way ANOVA]
	Normal (N=39)	Stage 3 (N=11)	Stage 4 (N=7)	Stage 5 (N=21)	
Left CIMT	1.23 ± 1.33	4.02 ± 0.92	6.56 ± 0.61	8.93 ± 1.77	<0.001
Right CIMT	1.12 ± 1.12	4.15 ± 0.97	6.76 ± 0.68	8.87 ± 1.89	<0.001
MEAN CIMT	1.18 ± 1.12	4.60 ± 1.40	7.52 ± 1.42	9.10 ± 2.00	<0.001

The mean of left CIMT was 1.23 ± 1.33 in nonCKD, 4.02 ± 0.92 in stage 3, 6.56 ± 0.61 in stage 4 and it was 8.93 ± 1.77 in stage 5. The mean of right CIMT was 1.12 ± 1.12 in nonCKD, 4.15 ± 0.97 in stage 3, 6.76 ± 0.68 in stage 4 and it was 8.87 ± 1.89 in stage 5. The mean of CIMT was 1.18 ± 1.12 in nonCKD, 4.60 ± 1.40 in stage 3, 7.52 ± 1.42 in stage 4 and it was 9.10 ± 2.00 in stage 5. The difference in proportion of CIMT (Left, Right, Mean) among stages of CKD was found to be significant with a P- value reported as <0.001. (13th Table)

Table 14: Correlation of CIMT with EGFR in the study population (N=78)

CIMT	EGFR	P Value (spearman)
	rs Value (spearman)	
L CIMT	-0.87	<0.001
R CIMT	-0.86	<0.001
MEAN CIMT	-0.86	<0.001

There was a strong negative correlation between left CIMT and HDL (r_s value: -0.87, P value: <0.001). There was a strong negative correlation between right CIMT and HDL (r_s value: -0.86, P value: <0.001). There was a strong negative correlation between mean CIMT and HDL (r_s value: -0.86, P value: <0.001). (Table 14 & Figure 5)

Figure 5: Scatter chart of camparison of CIMT mean value with EGFR (N=78)

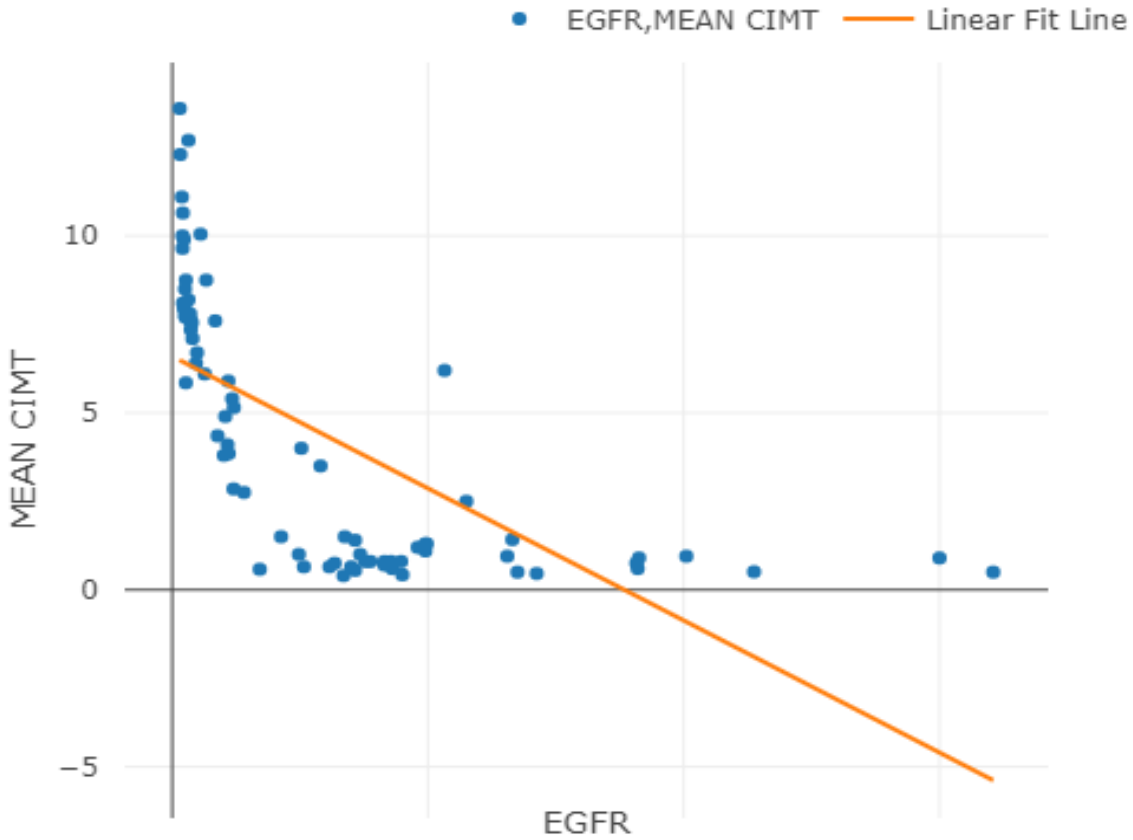


Table 15: Comparison of CIMT with EGFR in the study population (N=78)

CIMT	EGFR				Non CKD	P Value [One way ANOVA]
	Kidney failure (<15) (N=21)	Severe GFR (15-29) (N=7)	Moderate GFR (30-59) (N=11)	Kidney damage with mild GFR(60-89) (N=2)		
L CIMT	8.93 ± 1.77	6.56 ± 0.61	4.02 ± 0.92	1.16 ± 0.63	1.24 ± 1.37	<0.001
R CIMT	8.87 ± 1.89	6.76 ± 0.68	4.15 ± 0.97	0.92 ± 0.67	1.13 ± 1.14	<0.001
MEAN CIMT	9.10 ± 2.00	7.52 ± 1.42	4.60 ± 1.40	1.04 ± 0.65	1.18 ± 1.14	<0.001

The mean of left CIMT was 8.93 ± 1.77 in kidney failure (<15) EGFR, 6.56 ± 0.61 in severe GFR (15-29), 4.02 ± 0.92 in moderate GFR (30-59), 1.16 ± 0.63 kidney damage with mild (60-89) and it was 1.24 ± 1.37 in non-CKD. The mean of right CIMT was 8.87 ± 1.89 in

kidney failure (<15) EGFR, 6.76 ± 0.68 in severe GFR (15-29), 4.15 ± 0.97 in moderate GFR (30-59), 0.92 ± 0.67 kidney damage with mild (60-89) and it was 1.13 ± 1.14 in non-CKD. The mean CIMT was 9.10 ± 2.00 in kidney failure (<15) EGFR, 7.52 ± 1.42 in severe GFR (15-29), 4.60 ± 1.40 in moderate GFR (30-59), 1.04 ± 0.65 kidney damage with mild (60-89) and it was 1.18 ± 1.14 in non-CKD. The mean difference of CIMT (Left, Right, Mean) among EGFR was significant with a P- value <0.001. (15th Table)

Table 16: CIMT comparison with TGL in the study population (N=78)

CIMT	TGL	P Value (spearman)
	rs Value (spearman)	
L CIMT	0.40	<0.001
R CIMT	0.41	<0.001
MEAN CIMT	0.45	<0.001

There was a weak positive association between left CIMT and TGL (r_s value: 0.4, P value: <0.001). There was a weak positive (correlation) between right CIMT and TGL (r_s value: 0.41, P value: <0.001). There was a weak positive correlation between mean CIMT and TGL (r_s value: 0.45, P value: <0.001). (Table 16)

Table 17: Comparison of CIMT with TC in the study population (N=78)

CIMT	TC	P Value (spearman)
	rs Value (spearman)	
L CIMT	0.26	0.0221
R CIMT	0.26	0.0211
MEAN CIMT	0.28	0.0130

There was a weak positive correlation between left CIMT and TC (r_s value: 0.26, P value: 0.0221). There was a weak positive correlation between right CIMT and TC (r_s value: 0.26, P value: 0.0211). There was a weak positive correlation between mean CIMT and TC (r_s value: 0.28, P value: 0.0130). (Table 17)

Table 18: Comparison of CIMT with HDL (N=78)

CIMT	HDL	P Value (spearman)
	rs Value (spearman)	
L CIMT	-0.50	<0.001
R CIMT	-0.54	<0.001
MEAN CIMT	-0.53	<0.001

There was a weak negative correlation between left CIMT and HDL (r_s value: -0.5, P value: <0.001). There was a moderate negative correlation between right CIMT and HDL (r_s value: -0.54, P value: <0.001). There was a moderate negative correlation between mean CIMT and HDL (r_s value: -0.53, P value: <0.001). (Table 18)

Table 19: Comparison of CIMT with LDL (N=78)

CIMT	LDL	P Value (spearman)
	rs Value (spearman)	
L CIMT	-0.12	0.2938
R CIMT	-0.11	0.3194
MEAN CIMT	-0.08	0.4859

There was a weak negative correlation between left CIMT and LDL (r_s value: -0.12, P value: 2938). There was a weak negative correlation between right CIMT and LDL (r_s value: -0.11, P value: 3194). There was a weak negative correlation between mean CIMT and LDL (r_s value: -0.08, P value: 0.4859). (Table 19)

DISCUSSION

DISCUSSION:

With a 13.4% global prevalence, chronic renal disease is a hazard for public health on a global scale. Risk of CVD is more in chronic disease patients due to high prevalence of CVS risk factors in them and two other mechanisms of CKD also add on to the risk. The two mechanisms are characteristic changes in vasculature brought about by kidney hormones, enzymes and cytokines released in response to kidney injury [76]and cardiac damage by chronic kidney disease associated mediators and heamodynamic alterations [53]. In those with chronic renal disease, a lower eGFR has been identified as a significant and substantial risk factor for heart disease [77]. Early detection and treatment of in those with chronic renal disease, a lower eGFR has been identified as a significant and substantial risk factor for heart disease. CVD risk factors in CKD subjects is the key strategy to reduce CVD related morbidity and mortality in them [78]. Diagnosis of cardiovascular disease risk in CKD subjects is vital because early initiation of therapies to reduce cardiovascular damage will help in reducing mortality. This study aims to aims to determine the thickness of carotid intimal medial at all stages of CKD subjects and correlate of carotid intimal medial thicknesses with eGFR in several phases of CKD subjects.

Final analysis included 78 subjects. They were grouped into two groups 1 and 2 each consisting of 39 participants. Group 1 consisted of subjects with chronic kidney disease and group 2 had subjects with normal kidney function. There was no substantial difference in age and gender in between the groups. The mean age was 58.05 ± 12.35 in group 1 and it was 56.28 ± 13.14 in group 2. This observation of mean age of CKD subjects being more than 55 years is similar to that observed in similar studies including in a study by Margekar, V., et al. [79] in which the mean age of CKD subjects ranged between 30 to 60 years and in a study by Hinderliter et al.[74] in which the mean age of CKD patients was 61 ± 14 years. This

observation is slightly different from that observed in study by Kaiser, A, M., et al.[80] in which the mean age of CKD participants was 36.1±9.5 years and in another study by Jha., et al.[81] observed that in contrast to the considerably older age groups afflicted in affluent nations, CKD typically affects young individuals seen between ages of twenty and 55 years old in emerging regions of various parts of the world.

In group 1 participants with CKD, in the age group of <=48 years, (55.00%) participants were in stage 1, (15.00%) participants were in stage 3, (5.00%) participants were in stage 4 and 5 (25.00%) participants were in stage 5. In age group 49-64 years, (48.39%) participants were in stage 1, (9.68%) participants were in stage 3, (12.90%) participants were in stage 4 and 9 (29.03%) participants were in stage 5. In age group >64 years, (48.15%) participants were in stage 1, (18.52%) participants were in stage 3, (7.41%) participants were in stage 4 and 7 (25.93%) participants were in stage 5 of CKD. In the study majority of subjects in stage 4 and 5 of CKD were in the age group of < 48 yrs and 49 to 64 years while in age groups greater than 64 years majority of participants stage 1 of CKD. The fact that chronic glomerulonephritis, one of the most common causes of CKD in Southeast Asia and Sub-Saharan Africa, is one of the most likely reasons of CKD in the developing world, lends weight to the finding that individuals in younger age groups are in the disease's later stages [81].

Multiple cardiovascular risk factors bring about changes in arterial walls of heart and measurement of carotid intima thickness gives understanding about these changes[58]. Hence carotid intima thickness measurement which is a noninvasive and reproducible method is used for prediction of cardiovascular disease risk and in identifying and quantifying subclinical CVD in individuals[82].

In present study the difference in the (Left, Right, Mean) CIMT (Carotid artery intimal medial thickness) between groups was statistically significant. The mean of left CIMT was 1.23 ± 1.33 in non-CKD participants of Group 1, 4.02 ± 0.92 in stage 3, 6.56 ± 0.61 in stage

4 and it was 8.93 ± 1.77 in stage 5. The mean of right CIMT was 1.12 ± 1.12 in non-CKD participants, 4.15 ± 0.97 in stage 3, 6.76 ± 0.68 in stage 4 and it was 8.87 ± 1.89 in stage 5. The mean of CIMT was 1.18 ± 1.12 in non-CKD, 4.60 ± 1.40 in stage 3, 7.52 ± 1.42 in stage 4 and it was 9.10 ± 2.00 in stage 5. These observations indicate increase in CIMT thickness in CKD patients in comparison with normal individuals and increase in CIMT thickness with progression of CKD disease. From these observations CKD can be considered as a risk factor for development of cardiovascular disease. This observation of increased risk of CVD which was reflected by increase in Carotid artery intima medial thickness in CKD patients was also reported in studies by Lawal, O, M., et al.[83], Zoungas, S., et al. [84], Brzosko, S., et al.[85], Kawagishi, T., et al.[86], Lahoti, S., et al.[87], Szeto, C., et al.[88], Nakashima, A., et al.[89], Yilmaz, M., et al.[90].

The observation of increase in CIMT thickness with progression in stages of CKD is similar to that reported in studies by Lahoti, S., et al.[87], Ponna, A, K., et al.[91],

Table 20: The following table gives comparison of CIMT thickness in CKD patients and in normal individuals from different studies:

Study	Mean CIMT in CKD patients (in mm)	Mean CIMT in normal individuals (in mm)
Present study	Left CIMT: 4.02 ± 0.92 in stage 3 6.56 ± 0.61 in stage 4 8.93 ± 1.77 in stage 5 Right CIMT: 4.15 ± 0.97 in stage 3 6.76 ± 0.68 in stage 4 8.87 ± 1.89 in stage	Left CIMT: 1.23 ± 1.33 Right CIMT: 1.12 ± 1.12
Lawal, O, M., et al.[83]	Right CIMT: 1.1 ± 0.38 Left CIMT: 1.1 ± 0.43	Right CIMT: 0.70 ± 0.10 Left CIMT: 0.70 ± 0.11
Zoungas, S., et al. [84]	0.89 ± –0.17	0.73 ± –0.13
Lahoti, S., et al.[87]	0.80 ± 0.28	0.64 ± 0.16
Szeto, C., et al.[88]	0.808 ± 0.196	-
Brzosko, S., et al.[85]	0.76 ± 0.14	0.55 ± 0.07
Nakashima, A., et al.[89]	0.746 ± 0.142	-
Yilmaz, M., et al.[90]	0.9	0.6

The mean of left CIMT was 8.93 ± 1.77 in kidney failure (<15) EGFR, 6.56 ± 0.61 in severe GFR (15-29), 4.02 ± 0.92 in moderate GFR (30-59), 1.16 ± 0.63 kidney damage with mild (60-89) and it was 1.24 ± 1.37 in non-CKD. The mean of right CIMT was 8.87 ± 1.89 in kidney failure (<15) EGFR, 6.76 ± 0.68 in severe GFR (15-29), 4.15 ± 0.97 in moderate GFR (30-59), 0.92 ± 0.67 kidney damage with mild (60-89) and it was 1.13 ± 1.14 in non-CKD. These observations indicate significant negative correlation between CIMT and stages of CKD or glomerular filtration rate. Some studies including a study by Rizikalo, A., et al. [68] have shown similar undesirable association amongst CIMT and GFR, in another study by Buscemi et al. [92] in individuals with near average or modestly impaired renal function, a strong negative connection between GFR and CIMT was discovered; however, Desbien et al investigation .'s found that the influence of further CKD advancement produced different results.[93] There has been substantial evidence linking CIMT to diminished renal function. Arroyo et al. observed lower CIMT in individuals with more advanced CKD, however some investigations, including one by Chhajed et al, identified no significant link among CIMT with eGFR in CKD patients.[69] [94]

In the study a strong negative association among the mean CIMT and HDL levels was observed in CKD patients similar to that observed in studies by Shoji., et al.[95] and Lahoti, S., et al[87]. Chronic renal disease is linked with alterations in lipoprotein metabolism, which may result in hypertriglyceridemia and the buildup of atherogenic components [96]. The most common changes seen in CKD patients are hypertriglyceridemia, elevated LDL cholesterol, lipoprotein(a) components, and ldl (Apo B)-containing lipoproteins, as well as low levels of HDL. In addition to contributing to the decline in HDL levels, chronic renal disease alters the lipoprotein's composition by, for example, lowering plasma levels of its 2 main constituents, apoA-I and apoA-II [97]. The low levels of HDL concentration resulting in CKD may be one of

the factors responsible for increased CIMT. The antiatherosclerosis properties of HDL include maintaining endothelium barrier function, hinder platelet initiation, collection, and formation of thrombus[98].

The study found a weak +ve correlation among serum triglyceride levels and total cholesterol levels in CKD patients with CIMT. In studies by Lahoti, S., et al.[87], Brzosko, S., et al.[85] and Kawagishi, T., et al. [86] greater level of serum triglycerides and total cholesterol were observed in CKD patients. The presence of more chylomicron remains, which can enter vascular endothelium and cause atherosclerosis, is indicated by higher blood triglyceride levels [99]. In the current investigation, individuals with CKD showed a limited connection among LDL levels and CIMT.

In the study in group 1 which includes participants with CKD, the median of haemoglobin was 9.00 and it was 15.00 in group 2. Anaemia, a prevalent and predictable side effect of chronic renal disease. Reduced synthesis of erythropoietin is strongly correlated with decreased haemoglobin (Hb) levels, which are also a recognised risk factor for unfavourable cardiovascular outcomes[100].

CONCLUSION

CONCLUSION

This study found augmented carotid intima thickness in participants with CKD in comparison with normal individuals. The research discovered a significant inverse relationship between CIMT and eGFR. The study also found other factors like increased levels of total cholesterol, triglycerides, low LDL and HDL and declined haemoglobin levels in CKD patients in comparison with normal individuals which also contribute increased CIMT in them.

Recommendation:

This study recommends CIMT measurement even in early stage for CKD patients for prediction of CVD risk in them and initiation of a scientifically supported medical procedure to lower cardiovascular risk, which would probably lower future CVD-related morbidity and death in CKD patients.

Limitation:

This study is a single center study based on small sample size and requires data from similar multicentre large sample-based studies for generalization of results.

SUMMARY

SUMMARY

- In the study majority of participants in stage 4 and 5 of CKD were in the age group of < 48 years and 49 to 64 years while in age groups greater than 64 years majority of participants stage 1 of CKD.
- The study found increase in CIMT thickness in CKD subjects in comparison with normal individuals and increase in CIMT thickness with progression of CKD disease.
- The study discovered a strong inverse relationship between glomerular filtration rate or CKD stages and CIMT.
- In the research, individuals with CKD showed a significant negative connection between median CIMT and HDL values. In CKD patients with CIMT, the study discovered a marginally favorable connection between blood triglyceride levels and total cholesterol levels.
- In the study in group 1 which includes participants with CKD, the median of haemoglobin was 9.00 and it was 15.00 in group 2.

BIBLIOGRAPHY

REFERENCES

1. Mills KT, Xu Y, Zhang W, Bundy JD, Chen C-S, Kelly TN, et al. A systematic analysis of worldwide population-based data on the global burden of chronic kidney disease in 2010. Kidney Int. 2015;88(5):950–7.
2. Zahmatkesh M, Tamadon MR. World kidney day 2018; chronic kidney disease in women. J Nephropathol. 2017;7(1):4–6.
3. Levey AS, Atkins R, Coresh J, Cohen EP, Collins AJ, Eckardt K-U, et al. Chronic kidney disease as a global public health problem: approaches and initiatives - a position statement from Kidney Disease Improving Global Outcomes. Kidney Int. 2007;72(3):247–59.
4. Singh NP, Gupta AK, Kaur G, Khanna T. Chronic Kidney Disease of Unknown Origin - What do we know? J Assoc Physicians India. 2020;68(2):76–9.
5. Dare AJ, Fu SH, Patra J, Rodriguez PS, Thakur JS, Jha P. Renal failure deaths and their risk factors in India 2001-13: nationally representative estimates from the Million Death Study. Lancet Glob Heal. 2017;5(1):e89–95.
6. Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation. 2003;108(17):2154–69.
7. Matsushita K, van der Velde M, Astor BC, Woodward M, Levey AS, de Jong PE, et al. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. Lancet (London, England). 2010;375(9731):2073–81.
8. Carney EF. The impact of chronic kidney disease on global health. Nat Rev Nephrol.

2020;16(5):251.

9. Goodman WG, London G, Amann K, Block GA, Giachelli C, Hruska KA, et al. Vascular calcification in chronic kidney disease. Am J Kidney Dis. 2004;43(3):572–9.

10. Gansevoort RT, Correa-Rotter R, Hemmelgarn BR, Jafar TH, Heerspink HJL, Mann JF, et al. Chronic kidney disease and cardiovascular risk: Epidemiology, mechanisms, and prevention. Lancet. 2013;382(9889):339–52.

11. Subbiah AK, Chhabra YK, Mahajan S. Cardiovascular disease in patients with chronic kidney disease: a neglected subgroup. Heart Asia. 2016;8(2):56–61.

12. Varughese S, John GT, Alexander S, Deborah MN, Nithya N, Ahamed I, et al. Pre-tertiary hospital care of patients with chronic kidney disease in India. Indian J Med Res. 2007;126(1):28–33.

13. Kon V, Linton MF, Fazio S. Atherosclerosis in chronic kidney disease: the role of macrophages. Nat Rev Nephrol. 2011;7(1):45–54.

14. Malyszko J, Bachorzewska-Gajewska H, Malyszko J, Iaina-Levin N, Kobus G, Dobrzycki S. Markers of kidney function in the elderly in relation to the new CKD-EPI formula for estimation of glomerular filtration rate. Arch Med Sci. 2011;7(4):658–64.

15. Kono K, Fujii H, Miyoshi N, Kawamori H, Shite J, Hirata K, et al. Coronary plaque morphology using virtual histology-intravascular ultrasound analysis in hemodialysis patients. Ther Apher Dial. 2011;15(1):44–50.

16. Merino A, Portolés J, Selgas R, Ojeda R, Buendia P, Ocaña J, et al. Effect of different dialysis modalities on microinflammatory status and endothelial damage. Clin J Am Soc Nephrol. 2010;5(2):227–34.

17. Handa N, Matsumoto M, Maeda H, Hougaku H, Ogawa S, Fukunaga R, et al. Ultrasonic evaluation of early carotid atherosclerosis. Stroke. 1990;21(11):1567–72.

18. Peters SAE, den Ruijter HM, Palmer MK, Grobbee DE, Crouse III JR, O'Leary DH, et al. Extensive or restricted ultrasound protocols to measure carotid intima-media thickness: analysis of completeness rates and impact on observed rates of change over time. J Am Soc Echocardiogr. 2012;25(1):91–100.

19. Shroff GR, Frederick PD, Herzog CA. Renal failure and acute myocardial infarction: clinical characteristics in patients with advanced chronic kidney disease, on dialysis, and without chronic kidney disease. A collaborative project of the United States Renal Data System/National Institutes. Am Heart J. 2012;163(3):399–406.

20. Freda BJ, Tang WHW, Van Lente F, Peacock WF, Francis GS. Cardiac troponins in renal insufficiency: review and clinical implications. J Am Coll Cardiol. 2002;40(12):2065–71.

21. Lamb EJ, Tomson CR V, Roderick PJ. Estimating kidney function in adults using formulae. Ann Clin Biochem. 2005;42(Pt 5):321–45.

22. Snyder S, Pendergraph B. Detection and evaluation of chronic kidney disease. Am Fam Physician. 2005;72(9):1723–32.

23. Rajapurkar MM, John GT, Kirpalani AL, Abraham G, Agarwal SK, Almeida AF, et al. What do we know about chronic kidney disease in India: first report of the Indian CKD registry. BMC Nephrol. 2012;13:10.

24. Chapter 1: Definition and classification of CKD. Kidney Int Suppl. 2013;3(1):19–62.

25. McCANCE RA, ROBINSON JR. Evaluation of renal clearances. Proc R Soc Med. 1949;42(7):475–80.

26. Stevens LA, Coresh J, Greene T, Levey AS. Assessing kidney function--measured and estimated glomerular filtration rate. N Engl J Med. 2006;354(23):2473–83.

27. Mula-Abed W-AS, Al Rasadi K, Al-Riyami D. Estimated Glomerular Filtration Rate (eGFR): A Serum Creatinine-Based Test for the Detection of Chronic Kidney Disease

and its Impact on Clinical Practice. Oman Med J. 2012;27(2):108–13.

28. Hill NR, Fatoba ST, Oke JL, Hirst JA, O'Callaghan CA, Lasserson DS, et al. Global Prevalence of Chronic Kidney Disease - A Systematic Review and Meta-Analysis. PLoS One. 2016;11(7):e0158765.

29. Jager KJ, Kovesdy C, Langham R, Rosenberg M, Jha V, Zoccali C. A single number for advocacy and communication-worldwide more than 850 million individuals have kidney diseases. Kidney Int. 2019;96(5):1048–50.

30. Ene-Iordache B, Perico N, Bikbov B, Carminati S, Remuzzi A, Perna A, et al. Chronic kidney disease and cardiovascular risk in six regions of the world (ISN-KDDC): a cross-sectional study. Lancet Glob Heal. 2016;4(5):e307–19.

31. Bikbov B, Purcell CA, Levey AS, Smith M, Abdoli A, Abebe M, et al. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395(10225):709–33.

32. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet (London, England). 2013;382(9888):260–72.

33. Vaidya SR, Aeddula NR. Chronic Renal Failure., Treasure Island (FL): 2022.

34. Textor SC. Ischemic nephropathy: where are we now? J Am Soc Nephrol. 2004;15(8):1974–82.

35. Khanna R. Clinical presentation & management of glomerular diseases: hematuria, nephritic & nephrotic syndrome. Mo Med. 2011;108(1):33–6.

36. Aeddula NR, Baradhi KM. Reflux Nephropathy., Treasure Island (FL): 2022.

37. Luttropp K, Lindholm B, Carrero JJ, Glorieux G, Schepers E, Vanholder R, et al. Genetics/Genomics in chronic kidney disease--towards personalized medicine? Semin

Dial. 2009;22(4):417–22.

38. Levey AS, Coresh J. Chronic kidney disease. Lancet (London, England). 2012;379(9811):165–80.

39. Matovinović MS. 1. Pathophysiology and Classification of Kidney Diseases. EJIFCC. 2009;20(1):2–11.

40. Anderson TJ, Grégoire J, Pearson GJ, Barry AR, Couture P, Dawes M, et al. 2016 Canadian Cardiovascular Society Guidelines for the Management of Dyslipidemia for the Prevention of Cardiovascular Disease in the Adult. Can J Cardiol. 2016;32(11):1263–82.

41. Whelton PK, Carey RM, Aronow WS, Casey DEJ, 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 P. J Am Coll Cardiol. 2018;71(19):e127–248.

42. Bakris GL, Agarwal R, Chan JC, Cooper ME, Gansevoort RT, Haller H, et al. Effect of Finerenone on Albuminuria in Patients With Diabetic Nephropathy: A Randomized Clinical Trial. JAMA. 2015;314(9):884–94.

43. Inker LA, Astor BC, Fox CH, Isakova T, Lash JP, Peralta CA, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for the evaluation and management of CKD. Am J Kidney Dis Off J Natl Kidney Found. 2014;63(5):713–35.

44. Markowitz GS, Perazella MA. Acute phosphate nephropathy. Kidney Int. 2009;76(10):1027–34.

45. Lazarus B, Chen Y, Wilson FP, Sang Y, Chang AR, Coresh J, et al. Proton Pump Inhibitor Use and the Risk of Chronic Kidney Disease. JAMA Intern Med.

2016;176(2):238–46.

46. Lamb EJ, Levey AS, Stevens PE. The Kidney Disease Improving Global Outcomes (KDIGO) guideline update for chronic kidney disease: evolution not revolution. Clin Chem. 2013 Mar;59(3):462-5.

47. Chan KE, Giugliano RP, Patel MR, Abramson S, Jardine M, Zhao S, et al. Nonvitamin K Anticoagulant Agents in Patients With Advanced Chronic Kidney Disease or on Dialysis With AF. J Am Coll Cardiol. 2016;67(24):2888–99.

48. Menon V, Kopple JD, Wang X, Beck GJ, Collins AJ, Kusek JW, et al. Effect of a very low-protein diet on outcomes: long-term follow-up of the Modification of Diet in Renal Disease (MDRD) Study. Am J Kidney Dis. 2009;53(2):208–17.

49. Goraya N, Simoni J, Jo C, Wesson DE. Dietary acid reduction with fruits and vegetables or bicarbonate attenuates kidney injury in patients with a moderately reduced glomerular filtration rate due to hypertensive nephropathy. Kidney Int. 2012;81(1):86–93.

50. Chen TK, Knicely DH, Grams ME. Chronic Kidney Disease Diagnosis and Management: A Review. JAMA. 2019;322(13):1294–304.

51. Manjunath G, Tighiouart H, Coresh J, Macleod B, Salem DN, Griffith JL, et al. Level of kidney function as a risk factor for cardiovascular outcomes in the elderly. Kidney Int. 2003;63(3):1121–9.

52. Valdivielso JM, Rodríguez-Puyol D, Pascual J, Barrios C, Bermúdez-López M, Sánchez-Niño MD, et al. Atherosclerosis in chronic kidney disease: more, less, or just different? Arterioscler Thromb Vasc Biol. 2019;39(10):1938–66.

53. Fujii H, Goto S, Fukagawa M. Role of uremic toxins for kidney, cardiovascular, and bone dysfunction. Toxins (Basel). 2018;10(5):202.

54. Wilson PWF, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB.

Prediction of coronary heart disease using risk factor categories. Circulation. 1998;97(18):1837–47.

55. Brunet P, Gondouin B, Duval-Sabatier A, Dou L, Cerini C, Dignat-George F, et al. Does uremia cause vascular dysfunction. Kidney Blood Press Res. 2011;34(4):284–90.

56. Zoccali C, Vanholder R, Massy ZA, Ortiz A, Sarafidis P, Dekker FW, et al. The systemic nature of CKD. Nat Rev Nephrol. 2017;13(6):344–58.

57. Liviakis L, Pogue B, Paramsothy P, Bourne A, Gill EA. Carotid intima-media thickness for the practicing lipidologist. J Clin Lipidol. 2010;4(1):24–35.

58. Stein JH, Korcarz CE, Hurst RT, Lonn E, Kendall CB, Mohler ER, et al. Use of carotid ultrasound to identify subclinical vascular disease and evaluate cardiovascular disease risk: a consensus statement from the American Society of Echocardiography Carotid Intima-Media Thickness Task Force. Endorsed by the Society for Vascul. J Am Soc Echocardiogr Off Publ Am Soc Echocardiogr. 2008;21(2):90–3.

59. Mooney MG, Wilson MG. Linear array transducers with improved image quality for vascular ultrasonic imaging. Hewlett Packard J. 1994;45:43.

60. Jacoby DS, Mohler ER, Rader DJ. Noninvasive atherosclerosis imaging for predicting cardiovascular events and assessing therapeutic interventions. Curr Atheroscler Rep. 2004;6(1):20–6.

61. Hansa G, Bhargava K, Bansal M, Tandon S, Kasliwal RR. Carotid intima-media thickness and coronary artery disease: an Indian perspective. Asian Cardiovasc Thorac Ann. 2003;11(3):217–21.

62. O'Leary DH, Bots ML. Imaging of atherosclerosis: carotid intima-media thickness. Eur Heart J. 2010;31(14):1682–9.

63. Greenland P, Bonow RO, Brundage BH, Budoff MJ, Eisenberg MJ, Grundy SM, et al. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium

scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: a report of the American College of Cardiology Foundation C. J Am Coll Cardiol. 2007;49(3):378–402.

64. Cobble M, Bale B. Carotid intima-media thickness: knowledge and application to everyday practice. Postgrad Med. 2010;122(1):10–8.

65. Gepner AD, Korcarz CE, Aeschlimann SE, LeCaire TJ, Palta M, Tzou WS, et al. Validation of a carotid intima-media thickness border detection program for use in an office setting. J Am Soc Echocardiogr. 2006;19(2):223–8.

66. Iglesias del Sol A, Bots ML, Grobbee DE, Hofman A, Witteman JCM. Carotid intima-media thickness at different sites: relation to incident myocardial infarction; The Rotterdam Study. Eur Heart J. 2002;23(12):934–40.

67. Salonen JT, Salonen R. Ultrasonographically assessed carotid morphology and the risk of coronary heart disease. Arterioscler Thromb a J Vasc Biol. 1991;11(5):1245–9.

68. Rizikalo A, Coric S, Matetic A, Vasilj M, Tocilj Z, Bozic J. Association of Glomerular Filtration Rate and Carotid Intima-Media Thickness in Non-Diabetic Chronic Kidney Disease Patients over a 4-Year Follow-Up. Life. 2021;11(3).

69. Chhajed N, Subhash Chandra B, Shetty M, Shetty C. Correlation of carotid intimal-medial thickness with estimated glomerular filtration rate and cardiovascular risk factors in chronic kidney disease. Saudi J Kidney Dis Transplant. 2014;25(3):572–6.

70. Kawamoto R, Ohtsuka N, Kusunoki T, Yorimitsu N. An association between the estimated glomerular filtration rate and carotid atherosclerosis. Intern Med. 2008;47(5):391–8.

71. Polak JF, Pencina MJ, Pencina KM, O'Donnell CJ, Wolf PA, D'Agostino RB. Carotid-Wall Intima–Media Thickness and Cardiovascular Events. N Engl J Med. 2011;365(3):213–21.

72. Benedetto FA, Mallamaci F, Tripepi G, Zoccali C. Prognostic value of ultrasonographic measurement of carotid intima media thickness in dialysis patients. J Am Soc Nephrol. 2001;12(11):2458–64.

73. Nishizawa Y, Shoji T, Maekawa K, Nagasue K, Okuno S, Kim M, et al. Intima-media thickness of carotid artery predicts cardiovascular mortality in hemodialysis patients. Am J Kidney Dis. 2003;41(3 Suppl 1):S76-9.

74. Hinderliter A, Padilla RL, Gillespie BW, Levin NW, Kotanko P, Kiser M, et al. Association of carotid intima-media thickness with cardiovascular risk factors and patient outcomes in advanced chronic kidney disease: the RRI-CKD study. Clin Nephrol. 2015;84(1):10–20.

75. Lyngdoh L, Banerjee B, Chowdhury S, Mukherjee R, Naiya SB, Bhattacharya R. Evaluation of atherosclerosis in patients with chronic kidney disease by measuring carotid intima media thickness: An observational study from a tertiary care center in India. Asian J Med Sci. 2021;12:50–7.

76. Nasrallah R, Hassouneh R, Hébert RL. PGE2, kidney disease, and cardiovascular risk: beyond hypertension and diabetes. J Am Soc Nephrol. 2016;27(3):666–76.

77. Drury PL, Ting R, Zannino D, Ehnholm C, Flack J, Whiting M, et al. Estimated glomerular filtration rate and albuminuria are independent predictors of cardiovascular events and death in type 2 diabetes mellitus: the Fenofibrate Intervention and Event Lowering in Diabetes (FIELD) study. Diabetologia. 2011;54(1):32–43.

78. Carracedo J, Alique M, Vida C, Bodega G, Ceprián N, Morales E, et al. Mechanisms of cardiovascular disorders in patients with chronic kidney disease: a process related to accelerated senescence. Front Cell Dev Biol. 2020;8:185.

79. Margekar V, Thakur S, Jatav OP, Tiwari D, Gupta M, Yadav P. Carotid intimal medial

thickness (CIMT) in patients of chronic kidney disease (CKD) and its association with CKD staging. Int J Contem Med Res. 2020;7(3):C7-C9

80. Kaiser AM, Islam RN, Mozaffor M, Feroz S, Rahman MM. Correlation of Carotid Artery Intima-Media Thickness with Age, Body Mass Index and Glomerular Filtration Rate in Chronic Kidney Disease Patients. Med Today. 2021;33(2):147–51.

81. Jha V, Wang AY-M, Wang H. The impact of CKD identification in large countries: the burden of illness. Nephrol Dial Transplant. 2012;27(suppl_3):iii32–8.

82. Taylor AJ, Merz CNB, Udelson JE. 34th Bethesda Conference: Executive summary--can atherosclerosis imaging techniques improve the detection of patients at risk for ischemic heart disease? J Am Coll Cardiol. 2003;41(11):1860–2.

83. Lawal OM, Balogun MO, Akintomide AO, Ayoola OO, Mene-Afejuku TO, Ogunlade O, et al. Carotid Intima-Media Thickness: A Surrogate Marker for Cardiovascular Disease in Chronic Kidney Disease Patients. Clin Med Insights Cardiol. 2019;13:1179546819852941.

84. Zoungas S, Cameron JD, Kerr PG, Wolfe R, Muske C, McNeil JJ, et al. Association of carotid intima-medial thickness and indices of arterial stiffness with cardiovascular disease outcomes in CKD. Am J Kidney Dis. 2007;50(4):622–30.

85. Brzosko S, Lebkowska U, Malyszko J, Hryszko T, Krauze-Brzosko K, Mysliwiec M. Intima media thickness of common carotid arteries is associated with traditional risk factors and presence of ischaemic heart disease in hemodialysis patients. Physiol Res. 2005;54(5):497–504.

86. Kawagishi T, Nishizawa Y, Konishi T, Kawasaki K, Emoto M, Shoji T, et al. High-resolution B-mode ultrasonography in evaluation of atherosclerosis in uremia. Kidney Int. 1995;48(3):820–6.

87. Lahoti S, Kumar S, Agrawal S. Study of carotid intimal medial thickness in chronic

kidney disease at rural teaching hospital. Ann Med Health Sci Res. 2017;

88. Szeto C-C, Chow K-M, Woo K-S, Chook P, Kwan BC-H, Leung C-B, et al. Carotid intima media thickness predicts cardiovascular diseases in Chinese predialysis patients with chronic kidney disease. J Am Soc Nephrol. 2007;18(6):1966–72.

89. Nakashima A, Yorioka N, Asakimori Y, Ito T, Masaki T, Shigemoto K, et al. Different risk factors for the maximum and the mean carotid intima-media thickness in hemodialysis patients. Intern Med. 2003;42(11):1095–9.

90. Yilmaz MI, Qureshi AR, Carrero JJ, Saglam M, Suliman ME, Caglar K, et al. Predictors of carotid artery intima-media thickness in chronic kidney disease and kidney transplant patients without overt cardiovascular disease. Am J Nephrol. 2010;31(3):214–21.

91. Ponna AK, Farishta F. Carotid intima–Media thickness in patients with chronic kidney disease. Indian J Appl Res. 2016;6:714–8.

92. Buscemi S, Geraci G, Massenti FM, Buscemi C, Costa F, D'Orio C, et al. Renal function and carotid atherosclerosis in adults with no known kidney disease. Nutr Metab Cardiovasc Dis. 2017;27(3):267–73.

93. Desbien AM, Chonchol M, Gnahn H, Sander D. Kidney function and progression of carotid intima-media thickness in a community study. Am J Kidney Dis Off J Natl Kidney Found. 2008;51(4):584–93.

94. Arroyo D, Betriu A, Martinez-Alonso M, Vidal T, Valdivielso JM, Fernández E. Observational multicenter study to evaluate the prevalence and prognosis of subclinical atheromatosis in a Spanish chronic kidney disease cohort: baseline data from the NEFRONA study. BMC Nephrol. 2014;15:168.

95. Shoji T, Emoto M, Tabata T, Kimoto E, Shinohara K, Maekawa K, et al. Advanced atherosclerosis in predialysis patients with chronic renal failure. Kidney Int.

2002;61(6):2187–92.

96. Bulbul MC, Dagel T, Afsar B, Ulusu NN, Kuwabara M, Covic A, et al. Disorders of Lipid Metabolism in Chronic Kidney Disease. Blood Purif. 2018;46(2):144–52.

97. Vaziri ND. Causes of dysregulation of lipid metabolism in chronic renal failure. Semin Dial. 2009;22(6):644–51.

98. Linton MRF, Yancey PG, Davies SS, et al. The Role of Lipids and Lipoproteins in Atherosclerosis. [Updated 2019 Jan 3]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK343489/

99. Reiss AB, Voloshyna I, De Leon J, Miyawaki N, Mattana J. Cholesterol Metabolism in CKD. Am J Kidney Dis. 2015;66(6):1071–82.

100. Jing Z, Wei-jie Y, Nan Z, Yi Z, Ling W. Hemoglobin targets for chronic kidney disease patients with anemia: a systematic review and meta-analysis. PLoS One. 2012;7(8):e43655.

ANNEXURES

PATIENT INFORMATION SHEET

Name of the study - "**CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD A CASE CONTROL STUDY IN TERITIARY CARE CENTRE TAMAKA KOLAR**"

Chronic kidney disease (CKD) encompasses a spectrum of pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR).CKD in INDIA are associated with increased morbidity and mortality, decreased quality of life and increased healthcare expenditures,at Sri Devaraj Urs Academy of Higher Education & Research has decided to undertake a study on this regard.

We are inviting the patients with CKD to take part in this study, however based on criteria list, eligible participants will be chosen among the interested ones.

Your participation in this research is entirely voluntary. It is your choice whether to participate or not. If you agree to participate in this study, you will undergo carotid doppler. We will collect blood samples to test CBC & eGFR using MRMD & also assess your balance. You can take your regular antidiabetic and hypertensive medications during the exercise sessions.

By participating in this research you will benefit by improved strength, balance required to do your daily activities effectively. Your participation will also help us to use the outcomes of this study for future subjects. Your participation in this study will not put you at any risk.

All information collected from you will be strictly confidential & will not be disclosed to any outsider. This information collected will be used for research purpose. This information will not reveal your identity & this study have been reviewed by central ethical committee.

There is no compulsion to participate in this study, further you are at the liberty to withdraw from the study at any time if you wish to do so. Your treatment aspect will not be affected if you not wish to participate. You are required to sign only if you voluntarily agree to participate in proposed study. A copy of this document will be given to you for your information.

INFORMED CONSENT FORM

Name of the study - "CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD A CASE CONTROL STUDY IN TERITIARY CARE CENTRE TAMAKA KOLAR".

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.

Print Name of Participant_____

Signature of Participant _____ Date _____

For illetrate -

I have witnessed the accurate reading of the consent form to the potential participant, and the individual has had the opportunity to ask questions. I confirm that the individual has given consent freely.

Print name of witness_____ AND Thumb print of participant

Signature of witness _____ Date _____

Statement by the researcher/person taking consent

I have accurately read out the information sheet to the potential participant with the best of my ability.I confirm that the participant was given an opportunity to ask questions about the study, and all the questions asked by the participant have been answered correctly and to the best of my ability. I confirm that the individual has not been coerced into giving consent, and the consent has been given freely and voluntarily.

A copy of this ICF has been provided to the participant.

Print Name of Researcher taking the consent_____

Signature of Researcher taking the consent_____

Date_____

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಹೆಸರು - ''ಸಿಕೆಡಿಯ ವಿವಿಧ ಹಂತಗಳೊಂದಿಗೆ ರೋಗಿಗಳಲ್ಲಿ ಇಜಿಎಫ್ಆರ್ನೊಂದಿಗೆ ಕ್ಯಾರೊಟಿಡ್ ಇಂಟಿಮಲ್ ಮೀಡಿಯಲ್ ಥಿಕ್ನೆಸ್ನ ಕೊರತೆ ತೃತೀಯ ಕೇರ್ ಸೆಂಟರ್ ತಮಕಾ ಕೋಲಾರ್ನಲ್ಲಿ ಕೇಸ್ ಕಂಟ್ರೋಲ್ ಅಧ್ಯಯನ

ದೀರ್ಘಕಾಲದ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆ (ಸಿಕೆಡಿ) ಅಸಹಜ ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯಚಟುವಟಿಕೆಗೆ ಸಂಬಂಧಿಸಿದ ರೋಗಶಾಸ್ತ್ರೀಯ ಪ್ರಕ್ರಿಯೆಗಳ ವರ್ಣಪಟಲವನ್ನು ಒಳಗೊಂಡಿದೆ ಮತ್ತು ಗ್ಲೋಮೆರುಲರ್ ಶೋಧನೆ ದರದಲ್ಲಿ (ಜಿಎಫ್ಆರ್) ಪ್ರಗತಿಶೀಲ ಕುಸಿತವಾಗಿದೆ .ಇಂಡಿಯಾದಲ್ಲಿ ಸಿಕೆಡಿ ಹೆಚ್ಚಿದ ಕಾಯಿಲೆ ಮತ್ತು ಮರಣ, ಜೀವನದ ಗುಣಮಟ್ಟ ಮತ್ತು ಆರೋಗ್ಯ ರಕ್ಷಣೆಯ ವೆಚ್ಚಗಳೊಂದಿಗೆ ಸಂಬಂಧಿಸಿದೆ , ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ಅಕಾಡೆಮಿ ಆಫ್ ಹೈಯರ್ ಎಜುಕೇಶನ್ & ರಿಸರ್ಚ್ನಲ್ಲಿ ಈ ನಿಟ್ಟಿನಲ್ಲಿ ಅಧ್ಯಯನವನ್ನು ಕೈಗೊಳ್ಳಲು ನಿರ್ಧರಿಸಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಾವು ಸಿಕೆಡಿ ಹೊಂದಿರುವ ರೋಗಿಗಳನ್ನು ಆಹ್ವಾನಿಸುತ್ತಿದ್ದೇವೆ, ಆದರೆ ಮಾನದಂಡಗಳ ಪಟ್ಟಿಯನ್ನು ಆಧರಿಸಿ, ಆಸಕ್ತರಲ್ಲಿ ಅರ್ಹ ಭಾಗವಹಿಸುವವರನ್ನು ಆಯ್ಕೆ ಮಾಡಲಾಗುತ್ತದೆ.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆ ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದೆ. ಭಾಗವಹಿಸಬೇಕೆ ಅಥವಾ ಬೇಡವೇ ಎಂಬುದು ನಿಮ್ಮ ಆಯ್ಕೆಯಾಗಿದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ, ನೀವು ಶೀರ್ಷಧಮನಿ ಡಾಪ್ಲರ್ಗೆ ಒಳಗಾಗುತ್ತೀರಿ. ಎಮ್ಆರ್ಐಂಡಿ ಬಳಸಿ ಸಿಬಿಸಿ ಮತ್ತು ಇಜಿಎಫ್ಆರ್ ಅನ್ನು ಪರೀಕ್ಷಿಸಲು ನಾವು ರಕ್ತದ ಮಾದರಿಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ ಮತ್ತು ನಿಮ್ಮ ಸಮತೋಲನವನ್ನು ಸಹ ನಿರ್ಣಯಿಸುತ್ತೇವೆ. ವ್ಯಾಯಾಮದ ಅವಧಿಯಲ್ಲಿ ನಿಮ್ಮ ನಿಯಮಿತ ಆಂಟಿಡಿಯಾಬೆಟಿಕ್ ಮತ್ತು ಅಧಿಕ ರಕ್ತದೊತ್ತಡದ ations ಷಧಿಗಳನ್ನು ನೀವು ತೆಗೆದುಕೊಳ್ಳಬಹುದು.

ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸುವ ಮೂಲಕ ನಿಮ್ಮ ದೈನಂದಿನ ಚಟುವಟಿಕೆಗಳನ್ನು ಪರಿಣಾಮಕಾರಿಯಾಗಿ ಮಾಡಲು ಅಗತ್ಯವಾದ ಸುಧಾರಿತ ಶಕ್ತಿ, ಸಮತೋಲನದಿಂದ ನೀವು ಪ್ರಯೋಜನ ಪಡೆಯುತ್ತೀರಿ. ಭವಿಷ್ಯದ ಅಧ್ಯಯನದ ಈ ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳನ್ನು ಬಳಸಲು ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ನಮಗೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಯು ನಿಮಗೆ ಯಾವುದೇ ಅಪಾಯವನ್ನುಂಟು ಮಾಡುವುದಿಲ್ಲ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯು ಕಟ್ಟುನಿಟ್ಟಾಗಿ ಗೌಪ್ಯವಾಗಿರುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಸಂಶೋಧನಾ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಮಾಹಿತಿಯು ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ ಮತ್ತು ಈ ಅಧ್ಯಯನವನ್ನು ಕೇಂದ್ರ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ, ಮುಂದೆ ನೀವು ಬಯಸಿದರೆ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯುವ ಸ್ವಾತಂತ್ರ್ಯವಿದೆ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನಿಮ್ಮ ಚಿಕಿತ್ಸೆಯ ಅಂಶವು ಪರಿಣಾಮ ಬೀರುವುದಿಲ್ಲ. ಉದ್ದೇಶಿತ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಸಹಿ ಮಾಡಬೇಕಾಗುತ್ತದೆ. ನಿಮ್ಮ ಮಾಹಿತಿಗಾಗಿ ಈ ಡಾಕ್ಯುಮೆಂಟ್‌ನ ನಕಲನ್ನು ನಿಮಗೆ ನೀಡಲಾಗುವುದು.

ಮಾಹಿತಿ ಕಾನ್ಸೆಂಟ್ ಫಾರ್ಮ್

ಅಧ್ಯಯನದ ಹೆಸರು - ''ಸಿಕೆಡಿಯ ವಿವಿಧ ಹಂತಗಳೊಂದಿಗೆ ರೋಗಿಗಳಲ್ಲಿ ಇಜಿಎಫ್ಆರ್ನೊಂದಿಗೆ ಕ್ಯಾರೊಟಿಡ್ ಇಂಟಿಮಲ್ ಮೀಡಿಯಲ್ ಥಿಕ್ನೆಸ್ನ ಕೊರತೆ, ತೃತೀಯ ಕೇರ್ ಸೆಂಟರ್ ತಮಕಾ ಕೋಲಾರ್ನಲ್ಲಿ ಕೇಸ್ ಕಂಟ್ರೋಲ್ ಅಧ್ಯಯನ''.

ನಾನು ಮೇಲಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿದ್ದೇನೆ, ಅಥವಾ ಅದನ್ನು ನನಗೆ ಓದಲಾಗಿದೆ. ಅದರ ಬಗ್ಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳುವ ಅವಕಾಶ ನನಗೆ ಸಿಕ್ಕಿದೆ ಮತ್ತು ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರ ಮುದ್ರಣ ಹೆಸರು _____

ಭಾಗವಹಿಸುವವರ ಸಹಿ _____ ದಿನಾಂಕ _____

ಇಲ್ಲೆಟ್ರೇಟ್ಗಾಗಿ -

ಸಂಭಾವ್ಯ ಪಾಲ್ಗೊಳ್ಳುವವರಿಗೆ ಒಪ್ಪಿಗೆಯ ರೂಪವನ್ನು ನಿಖರವಾಗಿ ಓದುವುದಕ್ಕೆ ನಾನು ಸಾಕ್ಷಿಯಾಗಿದ್ದೇನೆ ಮತ್ತು ವ್ಯಕ್ತಿಯು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳುವ ಅವಕಾಶವನ್ನು ಹೊಂದಿದ್ದಾನೆ. ವ್ಯಕ್ತಿಯು ಮುಕ್ತವಾಗಿ ಒಪ್ಪಿಗೆ ನೀಡಿದ್ದಾನೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ.

ಸಾಕ್ಷಿಯ ಮುದ್ರಣ ಹೆಸರು _____ ಮತ್ತು ಭಾಗವಹಿಸುವವರ ಹೆಬ್ಬೆರಳು ಮುದ್ರಣ

ಸಾಕ್ಷಿಯ ಸಹಿ _____ ದಿನಾಂಕ _____

ಒಪ್ಪಿಗೆ ಪಡೆಯುವ ಸಂಶೋಧಕ / ವ್ಯಕ್ತಿಯ ಹೇಳಿಕೆ

ಸಂಭಾವ್ಯ ಭಾಗವಹಿಸುವವರಿಗೆ ನನ್ನ ಸಾಮರ್ಥ್ಯದಿಂದ ನಾನು ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ನಿಖರವಾಗಿ ಓದಿದ್ದೇನೆ. ಭಾಗವಹಿಸುವವರಿಗೆ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ಅವಕಾಶ ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ, ಮತ್ತು ಭಾಗವಹಿಸುವವರು ಕೇಳಿದ ಎಲ್ಲಾ ಪ್ರಶ್ನೆಗಳಿಗೆ ಸರಿಯಾಗಿ ಮತ್ತು ಉತ್ತರಿಸಲಾಗಿದೆ ನನ್ನ ಸಾಮರ್ಥ್ಯದ ಅತ್ಯುತ್ತಮ. ಒಪ್ಪಿಗೆ ನೀಡುವಂತೆ ವ್ಯಕ್ತಿಯನ್ನು ಒತ್ತಾಯಿಸಲಾಗಿಲ್ಲ ಮತ್ತು ಒಪ್ಪಿಗೆಯನ್ನು ಮುಕ್ತವಾಗಿ ಮತ್ತು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ಖಚಿತಪಡಿಸುತ್ತೇನೆ.

ಭಾಗವಹಿಸುವವರಿಗೆ ಈ ಐಸಿಎಫ್ ನಕಲನ್ನು ಒದಗಿಸಲಾಗಿದೆ.

ಒಪ್ಪಿಗೆ ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಮುದ್ರಣ ಹೆಸರು _____

ಒಪ್ಪಿಗೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಸಹಿ _____ ದಿನಾಂಕ _____

PROFORMA for CORRELATION OF CAROTID INTIMAL MEDIAL THICKNESS WITH eGFR IN PATIENTS WITH VARIOUS STAGES OF CKD A CASE CONTROL STUDY IN TERITIARY CARE CENTRE TAMAKA KOLAR

Name:

Age:

Sex:

Occupation:

UHID number:

Phone number:

Address:

Complaints with duration:

Previous history:

Family history:

Past history:

GENERAL PHYSICAL EXAMINATION:

Built and nourishment:

Pallor/Cyanosis/Icterus/Clubbing/edema/Generalized lymphadenopathy

VITAL DATA:

Pulse:

Temperature:

BP:

Respiration rate:

Systemic examination:

Per abdomen:

Respiratory system:

Cardio vascular system:

Central nervous system:

INVESTIGATIONS

COMPLETE HEMOGRAM

BLOOD UREA

SERUM CREATININE

SERUM ELECTROLYTES

SPECIFIC PARAMETER

CAROTID DOPPLER

eGFR using MRMD EQUATION:

USG ABDOMEN AND PELVIS:

MASTER CHART

S.NO	Gender	AGE	T2DM	HTN	Pulse	SBP	DBP	CVS	RS	P/A	CNS	HB	WBC	PLT	BI urea
1	Male	61	Yes	Yes	80	90	40	S1S2+.	B/LAE+	SOFT	NFND	9	5.6	150	70
2	Female	71	Yes	Yes	72	130	80	S1S2+.	B/LAE+	SOFT	NFND	10	7.8	175	68
3	Male	71	No	Yes	71	130	80	S1S2+.	B/LAE+	SOFT	NFND	8	8.9	168	65
4	Female	44	Yes	No	60	80	60	S1S2+.	B/LAE+	SOFT	NFND	7	8.5	144	78
5	Female	56	No	Yes	82	130	90	S1S2+.	B/LAE+	SOFT	NFND	8.5	7	188	90
6	Male	72	Yes	Yes	86	120	80	S1S2+.	B/LAE+	SOFT	NFND	9.3	8	194	112
7	Male	80	No	No	72	170	100	S1S2+.	B/LAE+	SOFT	NFND	8	9	123	79
8	Male	64	Yes	Yes	82	180	80	S1S2+.	B/LAE+	SOFT	NFND	10	10	134	80
9	Male	56	Yes	No	86	140	80	S1S2+.	B/LAE+	SOFT	NFND	7	4	156	70
10	Male	66	Yes	Yes	82	150	80	S1S2+.	B/LAE+	SOFT	NFND	6	6	166	68
11	Female	72	Yes	Yes	84	118	70	S1S2+.	B/LAE+	SOFT	NFND	8	7	188	55
12	Female	52	Yes	Yes	94	120	80	S1S2+.	B/LAE+	SOFT	NFND	10	12	190	76
13	Female	34	No	Yes	95	130	70	S1S2+.	B/LAE+	SOFT	NFND	9	9	150	74
14	Female	40	Yes	Yes	88	140	60	S1S2+.	B/LAE+	SOFT	NFND	8	5	140	88
15	Male	40	Yes	Yes	92	110	70	S1S2+.	B/LAE+	SOFT	NFND	7	6	130	145
16	Male	50	Yes	Yes	69	110	90	S1S2+.	B/LAE+	SOFT	NFND	9	7.8	120	133
17	Male	73	Yes	Yes	70	150	100	S1S2+.	B/LAE+	SOFT	NFND	8	9.9	140	190
18	Male	63	Yes	No	90	100	70	S1S2+.	B/LAE+	SOFT	NFND	9.5	5	150	120
19	Male	70	Yes	Yes	92	140	90	S1S2+.	B/LAE+	SOFT	NFND	8.7	7.8	170	133
20	Female	61	Yes	Yes	100	130	80	S1S2+.	B/LAE+	SOFT	NFND	9.6	10	160	144
21	Male	40	Yes	Yes	76	110	70	S1S2+.	B/LAE+	SOFT	NFND	10	11	150	133
22	Male	51	Yes	Yes	75	100	80	S1S2+.	B/LAE+	SOFT	NFND	9	12	170	155
23	Male	42	Yes	Yes	86	110	70	S1S2+.	B/LAE+	SOFT	NFND	8.9	8.9	180	123
24	Male	52	No	Yes	76	130	80	S1S2+.	B/LAE+	SOFT	NFND	9.9	7.7	140	133
25	Female	55	Yes	Yes	80	130	90	S1S2+.	B/LAE+	SOFT	NFND	10	8.8	130	88
26	Female	72	Yes	Yes	84	130	80	S1S2+.	B/LAE+	SOFT	NFND	7.9	4.7	140	67
27	Male	58	Yes	No	86	100	70	S1S2+.	B/LAE+	SOFT	NFND	8.5	6.6	150	56
28	Male	50	Yes	No	72	120	80	S1S2+.	B/LAE+	SOFT	NFND	8.9	7.6	130	67
29	Male	70	Yes	Yes	80	110	70	S1S2+.	B/LAE+	SOFT	NFND	10	8.8	156	53
30	Male	60	Yes	No	80	160	120	S1S2+.	B/LAE+	SOFT	NFND	8	6	170	88
31	Male	65	Yes	Yes	65	140	80	S1S2+.	B/LAE+	SOFT	NFND	9	5	176	134

32	Male	60	No	Yes	70	110	70	S1S2+.	B/LAE+	SOFT	NFND	8.6	6.7	187	76
33	Male	68	Yes	Yes	82	110	70	S1S2+.	B/LAE+	SOFT	NFND	9.8	7.4	199	65
34	Male	70	Yes	Yes	84	110	70	S1S2+.	B/LAE+	SOFT	NFND	7.9	8	156	34
35	Male	44	Yes	No	65	130	80	S1S2+.	B/LAE+	SOFT	NFND	10	9	170	55
36	Male	42	No	Yes	72	120	60	S1S2+.	B/LAE+	SOFT	NFND	11	8.9	156	66
37	Male	64	Yes	Yes	86	140	70	S1S2+.	B/LAE+	SOFT	NFND	9	9.5	143	83
38	Male	70	Yes	Yes	76	140	80	S1S2+.	B/LAE+	SOFT	NFND	9.2	10	155	79
39	Male	35	No	Yes	80	140	90	S1S2+.	B/LAE+	SOFT	NFND	9.8	11	130	144
40	Male	70	Yes	Yes	92	140	90	S1S2+.	B/LAE+	SOFT	NFND	12	7.8	298	17
41	Male	61	Yes	Yes	100	130	80	S1S2+.	B/LAE+	SOFT	NFND	13	10	287	12
42	Male	40	No	Yes	76	110	70	S1S2+.	B/LAE+	SOFT	NFND	14	11	356	23
43	Male	51	Yes	No	75	100	80	S1S2+.	B/LAE+	SOFT	NFND	15	12	378	24
44	Male	42	Yes	Yes	86	110	70	S1S2+.	B/LAE+	SOFT	NFND	16	8.9	324	26
45	Male	52	Yes	Yes	76	130	80	S1S2+.	B/LAE+	SOFT	NFND	13	7.7	412	19
46	Male	55	No	No	80	130	90	S1S2+.	B/LAE+	SOFT	NFND	17	8.8	364	20
47	Male	72	Yes	Yes	84	130	80	S1S2+.	B/LAE+	SOFT	NFND	14	4.7	312	29
48	Male	58	Yes	Yes	86	100	70	S1S2+.	B/LAE+	SOFT	NFND	13	6.6	298	17
49	Male	50	Yes	No	72	120	80	S1S2+.	B/LAE+	SOFT	NFND	14	7.6	267	19
50	Male	70	Yes	Yes	80	110	70	S1S2+.	B/LAE+	SOFT	NFND	17	8.8	287	19
51	Female	60	Yes	Yes	80	160	120	S1S2+.	B/LAE+	SOFT	NFND	16	6	234	20
52	Male	65	No	Yes	65	140	80	S1S2+.	B/LAE+	SOFT	NFND	18	5	265	21
53	Male	60	Yes	Yes	70	110	70	S1S2+.	B/LAE+	SOFT	NFND	15	6.7	276	22
54	Female	68	Yes	Yes	82	110	70	S1S2+.	B/LAE+	SOFT	NFND	19	7.4	265	26
55	Male	70	Yes	Yes	84	110	70	S1S2+.	B/LAE+	SOFT	NFND	15	8	226	28
56	Male	44	No	Yes	65	130	80	S1S2+.	B/LAE+	SOFT	NFND	14	9	224	17
57	Male	42	Yes	Yes	72	120	60	S1S2+.	B/LAE+	SOFT	NFND	17	8.9	233	28
58	Male	64	Yes	Yes	86	140	70	S1S2+.	B/LAE+	SOFT	NFND	13	9.5	231	23
59	Female	70	Yes	Yes	76	140	80	S1S2+.	B/LAE+	SOFT	NFND	15	10	321	27
60	Female	35	No	No	80	140	90	S1S2+.	B/LAE+	SOFT	NFND	14	11	321	19
61	Female	72	Yes	Yes	86	120	80	S1S2+.	B/LAE+	SOFT	NFND	17	8	28	26
62	Female	80	Yes	Yes	72	170	100	S1S2+.	B/LAE+	SOFT	NFND	13	9	338	19
63	Male	64	Yes	Yes	82	180	80	S1S2+.	B/LAE+	SOFT	NFND	14	10	337	20
64	Male	56	Yes	No	86	140	80	S1S2+.	B/LAE+	SOFT	NFND	15	4	356	16
65	Male	66	No	Yes	82	150	80	S1S2+.	B/LAE+	SOFT	NFND	13	6	367	18
66	Male	72	Yes	Yes	84	118	70	S1S2+.	B/LAE+	SOFT	NFND	14	7	387	18

67	Male	52	Yes	Yes	94	120	80	S1S2+.	B/LAE+	SOFT	NFND	15	12	327	19
68	Female	34	Yes	No	95	130	70	S1S2+.	B/LAE+	SOFT	NFND	16	9	345	16
69	Male	40	Yes	No	88	140	60	S1S2+.	B/LAE+	SOFT	NFND	14	5	239	18
70	Male	40	Yes	Yes	92	110	70	S1S2+.	B/LAE+	SOFT	NFND	14	6	336	23
71	Male	50	Yes	No	69	110	90	S1S2+.	B/LAE+	SOFT	NFND	18	7.8	376	26
72	Female	73	Yes	Yes	70	150	100	S1S2+.	B/LAE+	SOFT	NFND	12	9.9	276	24
73	Male	63	Yes	Yes	90	100	70	S1S2+.	B/LAE+	SOFT	NFND	13	5	300	28
74	Female	70	Yes	Yes	92	140	90	S1S2+.	B/LAE+	SOFT	NFND	14	7.8	18	19
75	Male	34	Yes	No	95	130	70	S1S2+.	B/LAE+	SOFT	NFND	15	9	287	13
76	Female	40	Yes	No	88	140	60	S1S2+.	B/LAE+	SOFT	NFND	17	5	278	17
77	Male	40	No	Yes	92	110	70	S1S2+.	B/LAE+	SOFT	NFND	16	6	300	26
78	Female	50	Yes	Yes	69	110	90	S1S2+.	B/LAE+	SOFT	NFND	19	7.8	200	21

S.NO	Sr. Creat	Sodium	potassium	L CIMT	R CIMT	MEAN CIMT	EGFR	USG	STAGE OF CKD	Stage of CKD	Groups	BMI	TGL	TC	HDL	LDL
1	1.9	132	4.6	3.9	4	5.9	43.9	GRADE 3	3B	Stage 3	Group 1	25	533	233	43	100
2	1.5	134	5	3	3.9	4.9	41.5	GRADE 2	3B	Stage 3	Group 1	24	200	267	40	163
3	1.8	136	4.9	4.8	5.7	7.6	33.6	GRADE 2	3B	Stage 3	Group 1	26	367	256	34	110
4	2.5	138	3.8	5.6	6.7	6.1	25.4	GRADE 2	Four	Stage 4	Group 1	27	246	189	37	130
5	3.6	140	4.7	7	7.2	7.1	15.9	GRADE 3	Four	Stage 4	Group 1	31	256	176	35	150
6	5.4	129	4.8	8.4	8.7	12.7	12.7	GRADE 3	Five	Stage 5	Group 1	34	600	346	34	80
7	2.8	133	3.8	5.9	5.7	8.75	26.6	GRADE 2	Four	Stage 4	Group 1	24	401	256	29	116
8	3.4	128	4.5	6.6	6.9	10.05	22.2	GRADE 2	Four	Stage 4	Group 1	25	478	199	37	114
9	1.6	127	4.8	3.7	3.9	3.8	40.4	GRADE 2	3B	Stage 3	Group 1	21	234	150	42.4	90
10	1.9	132	4.2	4	4.2	4.1	43.2	GRADE 2	3B	Stage 3	Group 1	22	345	160	45	92
11	1.3	133	4.7	2.9	2.8	2.85	48	GRADE 2	3A	Stage 3	Group 1	23	167	183	39	99
12	1.6	129	4.9	3.8	3.9	3.85	44.2	GRADE 2	3B	Stage 3	Group 1	20	233	156	45	175
13	1.5	130	4.6	4.9	5.4	5.15	48.2	GRADE 2	3A	Stage 3	Group 1	21	245	199	50	85
14	1.9	133	4.5	4.3	4.4	4.35	35.5	GRADE 2	3B	Stage 3	Group 1	24	213	176	37	105
15	8	140	4.5	10	9.8	9.9	9.1	GRADE 3	Five	Stage 5	Group 1	34	356	301	26.3	98
16	7.6	130	5	8	7.9	7.95	9.2	GRADE 3	Five	Stage 5	Group 1	35	298	213	56.1	86
17	7.7	125	4.6	8.2	8	8.1	8.4	GRADE 3	Five	Stage 5	Group 1	36	312	200	57	110
18	8.8	133	4.8	10.5	10.8	10.65	8.3	GRADE 3	Five	Stage 5	Group 1	34	367	245	64	193
19	6.7	136	4.3	8.6	8.4	8.5	10	GRADE 3	Five	Stage 5	Group 1	20	297	199	26.3	56

20	5.9	138	4.7	8.7	8.8	8.75	10.7	GRADE 3	Five	Stage 5	Group 1	27	312	277	60	45
21	8.9	135	4.9	9.8	10.2	10	8	GRADE 3	Five	Stage 5	Group 1	30	277	167	38	108
22	9	130	4.6	11	11.2	11.1	7.6	GRADE 3	Five	Stage 5	Group 1	31	356	176	41.5	53
23	8.8	138	3.9	9.8	9.5	9.65	8.1	GRADE 3	Five	Stage 5	Group 1	26	289	125	38	65
24	7	132	5	8	7.5	7.75	10.1	GRADE 3	Five	Stage 5	Group 1	26	178	178	56	75
25	3.7	131	4.3	7.3	7.8	7.55	15.4	GRADE 3	Four	Stage 4	Group 1	21	256	345	32	57
26	3	129	4.7	6.6	6.2	6.4	18.6	GRADE 3	Four	Stage 4	Group 1	24	314	267	36	120
27	5	127	4.6	7.7	7	7.35	14.5	GRADE 3	Five	Stage 5	Group 1	25	233	345	36	89
28	4.4	129	3.9	6.9	6.8	6.7	19.4	GRADE 3	Four	Stage 4	Group 1	24	276	234	38.2	108
29	5	128	3.8	7.7	7.9	7.8	14	GRADE 3	Five	Stage 5	Group 1	22	278	218	28	110
30	5.6	133	3.9	8.5	7.9	8.2	12.7	GRADE 3	Five	Stage 5	Group 1	28	312	256	29.3	95
31	10	132	4	12	12.6	12.3	6.4	GRADE 3	Five	Stage 5	Group 1	26	456	289	26.3	110
32	6.5	132	4.6	5.8	5.9	5.85	10.7	GRADE 3	Five	Stage 5	Group 1	23	213	243	34.5	96
33	5	133	5	7.9	7.3	7.6	14.1	GRADE 3	Five	Stage 5	Group 1	25	276	213	26	110
34	1.5	132	3.9	3	2.5	2.75	56.1	GRADE 2	3A	Stage 3	Group 1	26	187	234	40	95
35	1.9	133	4.6	5.9	4.9	5.4	46.9	GRADE 2	3A	Stage 3	Group 1	23	200	213	50	96
36	5.7	128	4.7	7.9	7.5	7.7	13.3	GRADE 3	Five	Stage 5	Group 1	28	198	194	37.9	120
37	6.8	129	4.3	7.9	8	7.95	10	GRADE 3	Five	Stage 5	Group 1	28	176	134	36	69
38	6.4	130	4.7	7.5	7.9	7.7	10.5	GRADE 3	Five	Stage 5	Group 1	29	127	178	35	86
39	12	138	5	13.7	13.5	13.6	5.9	GRADE 3	Five	Stage 5	Group 1	21	300	176	112	96
40	0.5	137	3.6	1.2	1.4	1.3	199	NORMAL	One	Stage 1	Group 2	18	176	245	99	96
41	0.6	136	3.6	0.6	0.8	0.7	166	NORMAL	One	Stage 1	Group 2	20	154	215	89	120
42	0.3	133	4.1	0.9	1	0.95	402	NORMAL	One	Stage 1	Group 2	23	128	178	45	110
43	0.8	139	4.6	0.5	0.8	0.65	123	NORMAL	One	Stage 1	Group 2	2	233	123	167	96
44	0.4	137	4.6	0.4	0.52	0.46	285	NORMAL	One	Stage 1	Group 2	5	245	199	123	98
45	0.7	136	4.6	0.6	0.5	0.55	143	NORMAL	One	Stage 1	Group 2	27	333	213	90	105
46	0.4	131	3.5	0.5	0.5	0.5	270	NORMAL	One	Stage 1	Group 2	24	199	134	24	130
47	0.5	136	4.2	1.1	1.1	1.1	198	NORMAL	One	Stage 1	Group 2	21	316	287	126	120
48	0.7	136	4.1	0.6	0.7	0.65	140	NORMAL	One	Stage 1	Group 2	26	298	213	187	95
49	0.6	135	3.9	0.4	0.8	0.6	172	NORMAL	One	Stage 1	Group 2	19	134	175	90	102
50	0.9	127	3.9	3	5	4	101	NORMAL	One	Stage 1	Group 2	24	312	193	40	103
51	1	140	3.5	0.71	0.45	0.58	68.5	NORMAL	One	Stage 1	Group 2	26	356	200	50	98
52	0.3	138	4.2	0.5	0.7	0.6	364	NORMAL	One	Stage 1	Group 2	29	193	123	233	110
53	0.6	132	4.9	0.8	0.8	0.8	166	NORMAL	One	Stage 1	Group 2	28	213	199	180	102
54	0.4	138.3	4.6	1.1	1.3	1.2	192	NORMAL	One	Stage 1	Group 2	21	256	212	123	89

55	0.7	140	3.7	1.6	1.4	1.5	135	NORMAL	One	Stage 1	Group 2	28	256	199	127	85
56	0.8	134	3.4	0.8	0.7	0.75	127	NORMAL	One	Stage 1	Group 2	38	167	190	190	110
57	0.6	139	4	0.7	0.9	0.8	179	NORMAL	One	Stage 1	Group 2	32	212	211	43.8	104
58	0.3	135	3.8	0.8	1	0.9	365	NORMAL	One	Stage 1	Group 2	39	342	170	89	98
59	0.5	130	5.45	1	1	1	147	NORMAL	One	Stage 1	Group 2	31	312	213	79	98
60	0.7	130	3.7	0.79	0.82	0.8	155	NORMAL	One	Stage 1	Group 2	36	167	177	89	92
61	0.7	138	4.6	1.1	0.9	1	99	NORMAL	One	Stage 1	Group 2	34	178	123	87	11
62	0.5	139	4.1	1.6	1.2	1.4	143	NORMAL	One	Stage 1	Group 2	29	312	289	90	86
63	0.4	136	4.7	0.8	1.1	0.95	262	NORMAL	One	Stage 1	Group 2	21	234	178	56	89
64	0.2	138	4.9	0.6	1.2	0.9	600	NORMAL	One	Stage 1	Group 2	23	256	189	99	98
65	0.3	137	3.4	0.9	0.6	0.75	363	NORMAL	One	Stage 1	Group 2	26	216	190	78	95
66	0.5	133	3.8	1.5	1.1	1.3	198	NORMAL	One	Stage 1	Group 2	28	212	199	123	102
67	0.6	136	3.5	0.8	0.8	0.8	171	NORMAL	One	Stage 1	Group 2	25	199	189	245	103
68	0.7	135	3.7	6	1	3.5	116	NORMAL	One	Stage 1	Group 2	26	233	176	199	98
69	0.2	130	5.48	0.5	0.5	0.5	642	NORMAL	One	Stage 1	Group 2	20	187	190	38	110
70	0.7	136	3.6	0.8	0.8	0.8	151	NORMAL	One	Stage 1	Group 2	18	256	217	78	102
71	0.5	136	3.9	6.1	6.3	6.2	213	NORMAL	One	Stage 1	Group 2	19	218	366	89	89
72	0.8	136	3.5	1.6	1.4	1.5	85.2	NORMAL	One	Stage 1	Group 2	26	245	278	112	85
73	0.9	143	3.4	0.5	0.8	0.65	103	NORMAL	One	Stage 1	Group 2	24	356	245	29	110
74	0.3	135	3	1.44	1.4	1.42	266	NORMAL	One	Stage 1	Group 2	27	267	213	96	104
75	0.5	136	4	4	1	2.5	230	NORMAL	One	Stage 1	Group 2	21	189	190	178	98
76	0.7	138	4.2	0.4	0.4	0.4	134	NORMAL	One	Stage 1	Group 2	25	129	126	198	98
77	0.6	145	4.7	0.41	0.44	0.425	180	NORMAL	One	Stage 1	Group 2	26	199	134	34	92
78	0.2	128	4.3	0.47	0.55	0.51	455	NORMAL	One	Stage 1	Group 2	24	213	201	190	11