“CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME - DHKD SYNDROME" - A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL AND MRC”

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
AGEs	Advanced glycation end-products
BBs	Beta-blockers
BMI	Body mass index
BP	Blood pressure
CKD	Chronic kidney disease
CRF	Chronic renal failure
CVA	Cerebrovascular accident
CVD	Cardiovascular disease
DHKD	Diabetic hypertension-kidney disease
DKD	Diabetic kidney disease
DM	Diabetes mellitus
DN	Diabetic nephropathy
eGFR	Estimated glomerular filtration rate
eNOS	Endothelial nitric oxide synthase
ESRD	End-stage renal disease
GFR	Glomerular filtration rate
HT	Hypertension
IQR	Interquartile range
IHD	Ischemic heart disease
MDRD	Modified diet in renal disease
PVD	Peripheral vascular disease
RAAS	Renin-angiotensin-aldosterone system
RAS	Renin-angiotensin system
RRT	Renal replacement therapy
UACR	Urine albumin creatinine ratio
ACR	Albumin to creatinine ratio

ABSTRACT

Introduction: Diabetes Mellitus and hypertension remain one of the most common causes of structural and functional kidney abnormalities leading to chronic kidney disease. Diabetes Hypertension Kidney Disease Syndrome is a new term introduced in medical terminology. The present study was conducted to examine clinical & laboratory profile of diabetes hypertension kidney disease syndrome – "DHKD syndrome" over a period of one year**. Materials and Methods:** Hospital-based observational cross-sectional was done in the department of general medicine & nephrology among 120 patients with diabetes & hypertension in combination with kidney disease, with any duration of Diabetes > 2 years & any duration of hypertension >2 years presenting to the OPD. Renal function tests performed include serum creatinine, Blood urea nitrogen, serum electrolytes. Urinalysis includes urine protein and urine albumin to creatinine ratio whichwere calculated by taking the ratio between urinary albumin or urine protein & urinary creatinine in random spot urine. The patients were then scored based on MDRD formula and CKD EPI formula to calculate the estimated glomerular function rate & placed into various stages of CKD. **Results**: A total of 120 subjects were included in the final analysis. Mean age was 63.64 ± 10.80 in the study population.72.5% of the study participants were males. Mean years of diabetes was 16.15 ± 7.5 in the study population. Among the study population, 39 (32.5%) had a past history of dialysis & 80 (66.7%) had a present history of dialysis. In study population among no albuminuria group , 1 (50%) had gfr 30-44 (grade 3 ckd) and 1 (50%) had gfr<=15 (grade 5), among microalbuminuria group , 1 (4.45%) had gfr 60-89 (grade 2) & 1 had gfr 45-59 (grade 3a), 3 (13.64%) had gfr 30-44 (grade 3b), 9 (40.91%) had gfr 15-29 (grade 4) , 8 (36.36%) had gfr<=15 (grade 5) , among macroalbuminuria group , 4 (4.6%) had gfr 45-59 (grade

3a), 8 (9.2%) had gfr 30-44 (grade 3b) , 12 (13.79%) had gfr 15-29 (grade 4) and 63 (72.41%) had gfr<=15 (grade 5) . Majority had macro albuminuria. The proportion of the difference between SBP & macroalbuminuria was statistically significant. (P value<0.05). The proportion of the difference between insulin usage with macroalbuminuria was statistically significant. (P value<0.05)

Conclusion: Our study delivers sufficient evidence endorsing high relationship between Diabetes, Hypertension, & kidney disease, which is like a bond. It is essential to coin the term “Diabetic Hypertension-Kidney disease” for creating consciousness among patients & doctors for early detection of Diabetic nephropathy, at a stage when it can be competently treated.

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INTRODUCTION

One of the prime reasons of mortality & morbidity is chronic illnesses. Previously measured to be a health issue only in urbanized nations, four out of five chronic illness deaths now ensue in low revenue & middle revenue countries.[1]In our country assessed amount of total deaths due to chronic illnesses has increased from over thirty-seven lakhs in 1990 to a predictable seventy-six lakhs by year 2020.[2] Array of disease mortality & morbidity throughout the world is ever-altering equally in urbanized & developing countries of the world. During the twentieth century, infective diseases were the chief cause of death & disability. Nonetheless, in the present era, non-communicable, non-infectious diseases have become the main reason for mortality & morbidity around the world.[3]

Conventionally, health platforms for the anticipation of chronic diseases have mostly dedicated on Diabetes Mellitus, hypertension, & Cardiovascular Disease (CVD); nonetheless, upsurge in the occurrence of 'Chronic kidney disease'(CKD) continuing to 'end-stage renal disease' (ESRD) & subsequent monetary challenges of 'renal replacement therapy' (RRT) in both urbanized along with undeveloped states has emphasized position of CKD and its risk factors.[4]

CKD, with its high prevalence, is a significant public health problem. With less than 3 percent of land area, Our country hordes 17 percent of the world's population. Large numbers of patients below the poverty line, low monetary allocations, & low GDP for health care have led to negligible outcomes. Moreover, CKD & other non-communicable diseases have often been ignored in the aspect of persistent challenges from & competition for resources for communicable diseases , high infant & maternal mortality.[5]

CKD is a frequent cause of mortality & morbidity due to renal sources. 'WHO' has recognized renal diseases as 17th & 12th main reason of disability & death globally, correspondingly.[6] New study appraised that 'age-adjusted' occurrence rate of CKD in Our country is two hundred twenty-nine per ten lakh populations, & greater than a lakh new patients go in 'renal replacement program' each year in Our country.[7]

Numerous reasons of CKD with hypertension, diabetes & chronic glomerulonephritis among shared reasons are there. Overall, these reasons progressively lead to 'End stage renal disease' (ESRD), & the above syndrome is identified by anemia, hypertension , reduced quality of life, renal bone disease, & lessened life expectation. With criteria suggested by National Kidney Foundation, 2002, CKD is shared into five stages, classified conferring to the degree of patient's kidney function. END-STAGE RENAL DISEASE is a main public health problem globally & may be related with substantial mortality & morbidity.[8] It's been predictable that occurrence of END-STAGE RENAL DISEASE will rise over following periods, driven by populace aging, & rising prevalence of hypertension & diabetes.[9]

Diabetic kidney disease, a medical condition categorized of consistently high arterial blood pressure, albuminuria, persistent reduction of 'Glomerular Filtration Rate' (GFR), & a related high probability of cardiovascular mortality & morbidity. In diabetic subjects, hypertension upsurges possibility for renal & CVD mortality & morbidity.[10] Development of hypertension in diabetic nephropathy is multifaceted, partly understood & includes of electrolyte disparities, stimulation of 'Renin-Angiotensin-Aldosterone System' (RAAS), 'Endothelial Cell Dysfunction', & augmented oxidative tension.[11]

Nowadays key reason of end-stage renal failure is hypertension & diabetes. Due to the unexpected rate of progression of the disease, it is expected that the number of patients with diabetes will increase by 2 times in the next 25 years. This may lead to a subsequent increase in the number of patients with 'Chronic renal disease' & those requiring treatment for 'end-stage renal failure' mainly by hemodialysis.[12]

Hypertension & Diabetes mellitus which are two major reasons for 'Chronic renal disease' and both expanding similar to an epidemic in our country & also in other emerging nations.[13] 'ICMR' study stated in 2011 revealed 64.4 & 77.2 million persons had diabetes & pre-diabetes correspondingly. It is foretold that by 2030 our country's diabetic populace would be approximately eight crores. Likewise, the occurrence of hypertension has gradually risen at rapid speed. The 2010 'WHO' report on the worldwide position of non-communicable diseases, the occurrence of hypertension was raised from sixteen percent in 2004 to thirty-seven percent in 2008.[14,15]

Furthermore, Jaipur heart watch study on Indians existing in cities showed the occurrence of hypertension raised from thirty percent in 1994 to fifty-one percent in 2003. Above fifty percent of diabetics in Our country are ignorant of possible kidney damage prior to appearance of kidney ailment because of lack of testing serum creatinine & urine albumin or urine protein. Therefore, CKD goes unnoticed during initial stages and progresses to further advanced stages. It may be a grave error & therefore essential to use in practice "Diabetic Hypertension-Kidney disease (DHKD) Syndrome" for generating alertness amongst general population & doctors for timely recognition of DHKD, a phase that may be competently detected and cured.[16]

'Chronic kidney disease' along with diabetes or diabetic kidney disease (DKD), exhibits clinically as albuminuria, reduced 'Glomerular Filtration Rate' (GFR), or both.[17] Natural history of DKD usually has been described as progressive albuminuria followed by a steady loss of GFR. Nonetheless, this natural history may have changed over the last 2 decades. In particular, GFR loss has been detected prior to the development of albuminuria, a reduced GFR without albuminuria has been often described, & albuminuria has been detected to be temporary or reversible.[18]

Changes in diabetes management over time comprise increased use of intensive glycemic control, improved blood pressure control, & augmented use of renin-angiotensin-aldosterone system (RAAS) inhibitors.[19]

To recognize the early phases of CKD is tough as affected persons are usually asymptomatic. The chronic renal disease frequently rests unnoticed till there's great damage to renal function. 'Chronic kidney disease' is a curable disease, & if recognized early & suitably treated, then the progression of CKD may be lessened. The decline in kidney function may be noticed over a predictable hematological test that quantifies serum creatinine, a urinalysis calculating urine albumin creatinine ratio.[20]

Hypertension & Diabetes together play a significant role in progression to 'Chronic kidney disease'. Approximately 1 out of 3 adults with diabetes & 1 out of 5 adults with Hypertension develop CKD. Persons having Hypertension are 5 times greater expected to have 'Chronic kidney disease' when compared to those who don't have it. Due to this, heavy monetary burden will be placed on nations, including the price of managing of end-stage kidney failure. Therefore, this is therapeutically & frugally vital for consciousness about the disease and for mitigation programs that

have been planned in emerging nations. Rigorous act from worldwide organizations, administrations, health facility workers, & doctors will be needed for this.[20,21]

Need of study

'Chronic kidney disease' is a disease of huge importance in our country, & by a growing diabetic load, Hypertension, & mounting ageing populace it's heading for upsurge even more. Money capitalized at the present time in founding a prevention program for CKD is certainly aimed at giving effects in time to come & eventually in future it may prove profitable. Nevertheless, it necessitates a great deal of statistics & long term follow up.[22]

Diabetes & Hypertension are the ever-mounting problem in a developing country like our country & there's a significant association of both with CKD. Since much of the focus is given to Diabetes kidney disease & impact of Hypertension in Diabetic Kidney Disease & Chronic Kidney Disease, focus on DHKD as a syndrome is not yet studied in detail. Like diabetes, CKD is common, harmful, & treatable. If noticed early & coped suitably, DHKD progression may be prevented or delayed. Considering their similarities & differences may be helpful in research, clinical practice, & public health to address fundamental unsolved problems in CKD. There are only a few studies which have evaluated clinical as well as laboratory parameters of Diabetes Hypertension kidney Disease Syndrome.

Diabetes Hypertension Kidney Disease Syndrome is a new term introduced in medical terminology. Diabetes Hypertension Kidney Disease Syndrome has not yet been thoroughly investigated in the literature. There are insufficient studies that are intended to observe the incidence of Diabetes mellitus (DM) as a cause for 'Chronic kidney disease, the relation between CKD & diabetes and hypertension. Furthermore, the calculation has to be made on how often diabetes is made complex by chronic

renal disease, the occurrence of the relation between diabetes mellitus , hypertension & kidney disease. With this experience, the present study was conducted to examine the clinical & laboratory profile of diabetes hypertension kidney disease syndrome - DHKD syndrome" over a period of one year**.**

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AIMS AND OBJECTIVES

- To study clinical as well as laboratory profile in patients with diabetes hypertension kidney disease.

REVIEW OF LITERATURE

'Chronic Kidney Disease':

a) Definition:

"'Chronic kidney disease' is well-defined by an estimated 'Glomerular Filtration Rate'(eGFR) less than sixty mL/min/1.73m^3 for more than 3 months, with or without the presence of kidney damage.[23] This one may likewise be well-demarcated by functional & structural aberrations of the kidney, which should be existing for more than 3 months, as pathological aberrations & markers of renal damage that may or not reduce eGFR".[24]

Criteria for diagnosis:

Criteria for definition of CKD are objective & can be established by means of simple laboratory tests without identification of the cause of disease, thereby allowing detection of CKD by non-nephrologist doctors & supplementary health professionals.

Duration >3 Months-Kidney diseases may be acute or chronic. The basis for defining chronicity is to differentiate CKD from acute kidney diseases (such as acute Glomerulonephritis), together with AKI, that may necessitate different interventions, & have different etiologist & outcomes.[25] Length of renal disease can be recognized otherwise concluded founded on the clinical setting. Resolving over days to weeks would endorse the finding of AKI. The judgment of evaluation rest on clinical acumen, with earlier assessment for subjects supposed of having AKI & earlier assessment for patient expected to have CKD.

Reversibility- Maximum renal diseases don't show any signs till late in its progression & noticed once they turn chronic. Maximum reasons of CKD are irretrievable with a life-long sequence, & management is aimed at decelerating

progress to kidney failure. Yet, chronicity may not be identical with irreversibility. In a few instances, CKD may totally be revocable, both instinctively & with treatment, & in additional cases, treatment may lead to limited reversion of renal damage & functional growth.

Decreased GFR- GFR is widely accepted as the best overall index of kidney function because it's generally reduced after widespread structural damage & most other kidney functions decline in parallel with GFR in CKD. A GFR <60 ml/min/1.73m2 is less than half of the normal value in young adult men & women of approximately 125 ml/min/ 1.73m2. A GFR <60 ml/min/1.73m2 can be detected by routine laboratory testing. Current estimating equations for GFR (eGFR) based on serum creatinine (SCr), but not SCr alone, is sensitive for detecting measured GFR.[27] A decreased eGFR using SCr can be confirmed by GFR estimation using an alternative filtration marker (cystatin C) or GFR measurement, as necessary. A GFR <60 ml/min/1.73m2 is associated with a higher risk of complications of CKD than in subjects with CKD & conserved GFR.[26]

Figure 1: Criteria for 'chronic kidney disease.[26]

Markers of kidney damage (one or more)	Albuminuria (AER ≥ 30 mg/24 hours; ACR ≥ 30 mg/g [≥ 3 mg/mmol]) Urine sediment abnormalities Electrolyte and other abnormalities due to tubular disorders Abnormalities detected by histology Structural abnormalities detected by imaging History of kidney transplantation
Decreased GFR	GFR < 60 ml/min/1.73 m^2 (GFR categories G3a–G5)

Abbreviations: CKD, chronic kidney disease; GFR, glomerular filtration rate

b) Clinical presentation:

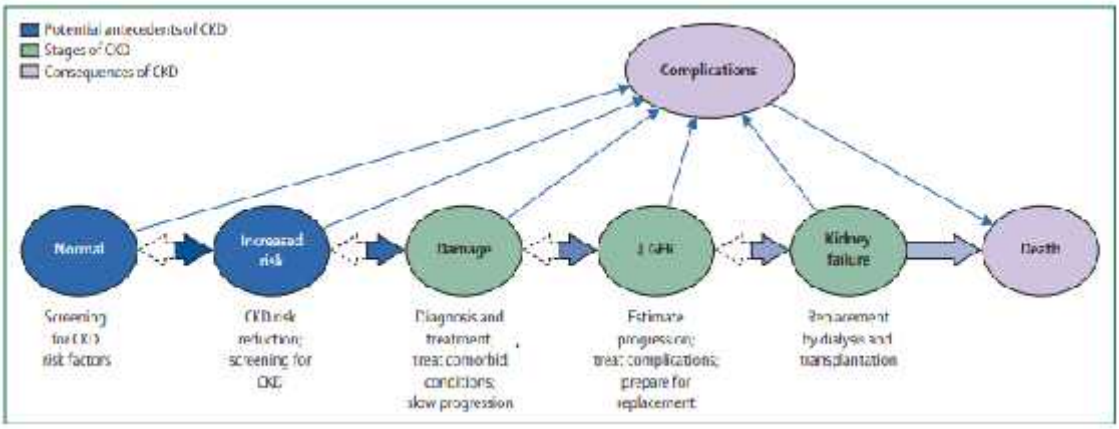
Kidney injury is demonstrated by the constant manifestation of haematuria, albuminuria, proteinuria, or structural aberrations noticed by imaging tests. 'Chronic renal disease' advances gradually & subtly for a long time.[20,27]Chronic renal disease is

normally recognized by routine screening with serum renal function profile & urine studies. Less frequently, patients may have symptoms, for example, undefined hematuria, "foamy urine", renal angle tenderness, nocturia, or decreased urine. In advanced stages, patients may show tiredness, little appetite, metallic taste, nausea, vomiting, pruritus, weight loss, altered mental status, dyspnoea, peripheral oedema.[28]

In assessing the patient by recognized/supposed CKD, clinicians should question their supplementary symptoms that may propose a systemic source otherwise urinary blockade. Furthermore, patients should be measured intended for risk elements of renal disease, comorbidities, history of nephrolithiasis, with former exposure to nephrotoxins (analgesics in the form of nsaids), or repeated UTI, family history of renal disease, and genetic 'risk factors'.[28]

Findings of arterio-venous nicking or retinopathy on fundoscopy establish diabetes or Hypertension. In those with abdominal bruits might show renovascular disease. Renal angle tenderness or distended kidneys may point towards contemplation of obstructive uropathy, pyelonephritis, polycystic kidney disease, nephrolithiasis. Neuropathy may be because of diabetes or vasculitis. Dermatological findings may include telangiectasias, rashes, palpable purpura, or extensive sclerosis. In those with progressive CKD can exhibit altered mental status, pallor, myoclonic jerks, skin excoriations, muscle wasting, asterixis, & pericardial rub.[29]

Figure 2: Conceptual model for 'Chronic kidney disease' Continuum of development, progression, & complications of 'Chronic kidney disease' (CKD) & strategies to improve outcomes. Thick arrows between circles represent development, progression, & remission of CKD. Complications refer to all complications of CKD, including complications of decreased 'Glomerular Filtration Rate' (GFR) & cardiovascular disease. Complications might also arise from the adverse effects of interventions to prevent or treat disease. Horizontal arrows pointing from left to right represent progressive nature of CKD. Dashed arrowheads signify that remission is less frequent than progression.[30]



c) Types

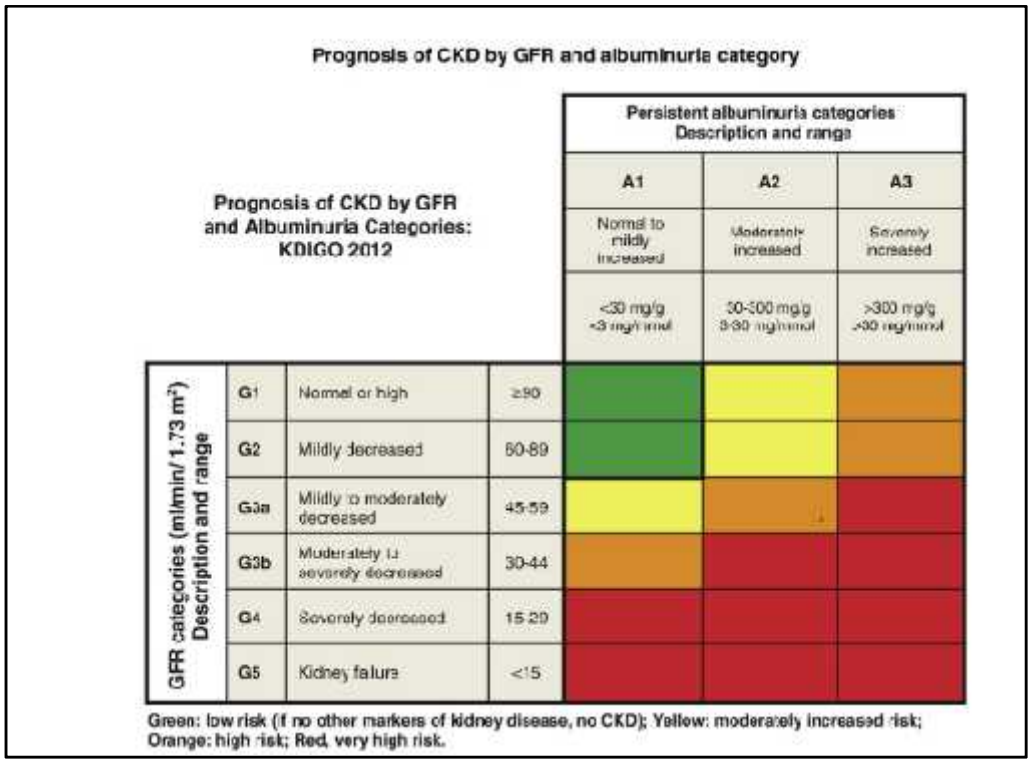
'Chronic kidney disease' is categorized as 5 stages basing on kidney function & destruction.[20]

Figure 3: Classification of Chronic Kidney Disease

Stage	Estimated GFR (mL/min/1.73 m²)	Comment
1	≥90	Normal GFR w/ proteinuria
2	60–89	Age-related decline in GFR w/proteinuria
3A 3B*	30–59	Low risk of progression to kidney failure
4	15–29	High risk of progression to kidney failure
5 5D 5T	<15	Kidney failure

*Because of greater cardiovascular disease risk and risk of disease progression at lower *eGFRs*, CKD Stage 3 is sub-divided into Stages 3A (45–59 ml /min/1.73 m²) and 3B (30–44 ml /min/1.73 m². CKD Stage 5 includes patients that may require or are undergoing kidney replacement therapy. Designations 5D and 5T indicate end stage renal disease patients who undergo chronic dialysis (5D) treatment or have undergone kidney transplantation (5T).

Figure 4: "Staging of CKD: Green: low risk (if no additional markers of kidney disease, no CKD); yellow: moderately increased risk; orange: high risk; red: very high risk. CKD, chronic kidney disease. GFR, 'Glomerular Filtration Rate'; KDIGO, Kidney Disease: Improving Global Outcomes."



d) Epidemiology:

CKD prevalence Stages one to five was 13.4 percent & 10.6 percent in stages three to five worldwide. CKD is more widespread in females than in males.[31] Industrialized countries such as USA, Europe, Australia & Canada had an advanced occurrence of CKD in contrast to regions where markets are rising like Our country, Sub-Saharan Africa etc.[32] Populace of our country surpasses 130 crores besides is likely to develop a large pool of chronic diseases like hypertension & diabetes. Subsequently, twenty to forty percent of them may progress to CKD, ESRD load will grow & health care organization may want to take caution of them. Our country stays facing a quick health change by huge & increasing loads of chronic diseases, that is

estimated for fifty-three percent of all deaths & forty-four percent of disability-adjusted life years gone by 2005.'[21]

Changes of lifestyle & development caused in hypertension, obesity, & diabetes that are associated with the augmented danger of CKD. The particular occurrence of CKD in our country isn't flawless in skiving of regular national data & given by few observational studies or individual experiences & quality of data being irregular. Supposing uniform occurrence in the entire nation, approximately 2 lakh new END-STAGE RENAL DISEASE patients would need 'renal replacement therapy'(RRT) each year in our country.[7]

e) Causes, Risk Factors, Pathogenesis:

Glomerulonephritis, Renal vascular disease, & genetic disease of kidney, diabetic nephropathy are important reasons for CKD.[33]

As Hypertension & Diabetes is connected with a considerably increased risk of evolving into CKD. Together these are major reasons of CKD.[34] Irrespective of nature of underlying nephropathy, the progression of CKD is seen with progressive sclerosis of glomeruli, tubulointerstitial fibrosis, & vascular sclerosis which can be initiated by endothelial, mesangial, or epithelial cell injury or damage. Kidney reacts to injury by adaptive changes that lead to remodelling evolving to their healing & functional recovery or scarring with loss of kidney function & progressive CKD. It occurs chiefly in AKI when acutely damaged tubules recover from initial insult and replace lost tubular cells to reconstitute integrity of tubules & to reinstate kidney function.[35]

f) Diagnosis

Understanding the definition & staging of CKD is vital to correctly identify people with the condition in clinical practice. Such information is also a key to

properly advise patients about their kidney health & estimate their future risk. According to KDIGO CKD guidelines, a patient is recognized with CKD if abnormalities of kidney structure or function were present for a minimum of 3 months. In practice, in primary care, the most important measures to recognize CKD are eGFR resulting from serum creatinine & ACR obtained from the urine. NICE endorses that certain populations should be obtainable testing for CKD using eGFR& ACR.[33]

Diagnostic criteria for CKD:[33]

1 of these criterias needs to be existing for at least 3 months:

a)Reduced eGFR (<60 mL/min/1.73 m2)

b)1or more marker of kidney damage:

i. Albuminuria (urinary albumin-to-creatinine ratio [ACR] ≥30 mg/g.
ii. Structural abnormalities (from imaging).
iii. Urine sediment abnormalities (hematuria, red or white blood cell casts, oval fat bodies or fatty casts, granular casts, & renal tubular epithelial cells).
iv. Electrolyte & other abnormalities due to tubular disorders.
v. Histological abnormalities.
vi. Previous history of kidney transplantation.

g) Complications

Complications of 'Chronic kidney disease' disturb every organ system. Patients with a GFR below 60 mL per minute per 1.73 m^2 should go through periodic monitoring for complications. Clinical evaluation may identify gastrointestinal, neurologic, dermatologic, & musculoskeletal complications in advanced stages of

chronic kidney disease. Gastrointestinal symptoms may sign the onset of uremia, indicating the need for kidney replacement therapy. Laboratory tests detect complications such as electrolyte abnormalities, disordered calcium or phosphorus metabolism, & anemia. Patients with nephrotic-range proteinuria are at risk for hypo-albuminemia& immune dysfunction due to loss of immunoglobulins. Episodic monitoring of total serum protein level & albumin level is directed in these patients. Nutritional status should be evaluated because malnutrition adversely affects prognosis.[36]

h) Treatment

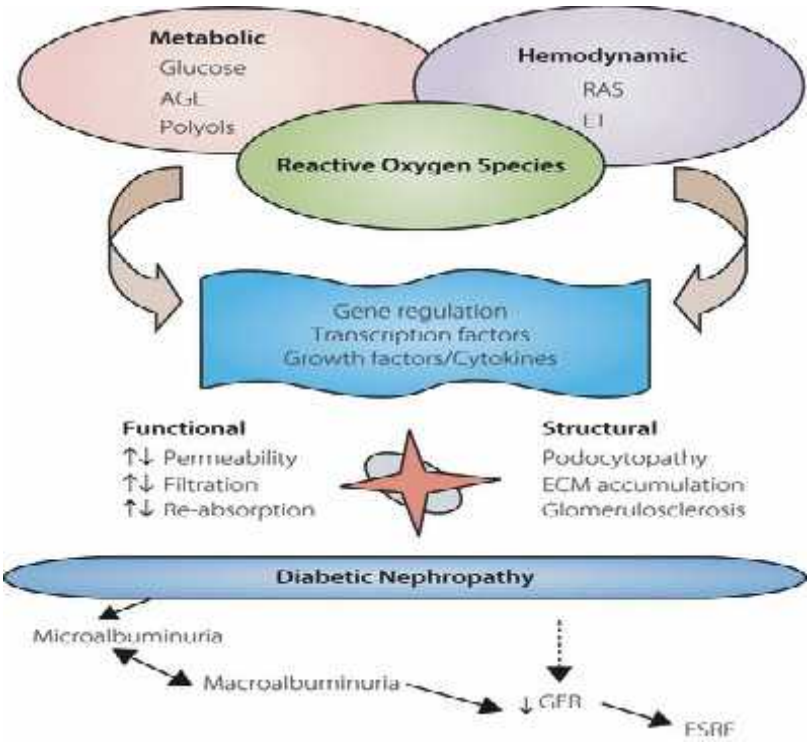
The goal of managing is to stop or defer the evolution to ESKD, decrease cardiovascular risk, & upsurge the quality of life. Treatment approaches for CKD include pharmacological & non-pharmacological interfaces. Still, when one reaches End Stage Kidney Disease, he needs to take choice with the management choices, that are basically conservative or renal replacement therapy.[37] Control of Blood pressure (BP) is important for those having CKD, primarily to delay the development of CKD & then to decrease the possibility of CVD. In diabetics, decent blood sugar control may delay the development of CKD. Non-pharmacological mediations mainly aim at lifestyle changes, endure an important aspect in the management of CKD, irrespective of medical therapy.[20]

1. ASSOCIATION BETWEEN DIABETES & 'CHRONIC KIDNEY DISEASE'

The pathophysiology of diabetic nephropathy is most likely to be as a result of metabolic and hemodynamic abnormalities, as seen in diabetes, interacting with each other and with various reactive oxygen species-dependent pathways. Both gene

regulation and activation of transcription factors are influenced by the interactions between metabolic stimuli, hemodynamic factors and reactive oxygen species generation in diabetes. The consequences of this molecular activation or inhibition are functional and structural changes leading to the classical hallmarks of diabetic nephropathy

Figure: 5Schema of pathogenesis of diabetic nephropathy.



(AGE -advanced glycation end-products. ECM -extracellular protein production. ESRF-end-stage renal failure. ET- endothelin)[38]

DM is a metabolic disorder which leads to kidney failure, & kidney failure increases the need for insulin in diabetics. Insulin resistance in skeletal muscle tissues is mainly seen because of aggregation of uremic toxins & increased parathyroid hormone levels in patients with chronic renal failure (CRF). This results in

interruption of insulin binding to its receptors, that interrupts glucose and glycogen metabolic pathways.[39]

It likewise appears that anemia caused by 'chronic renal failure' may influence on insulin resistance, & its correction by administration of erythropoietin has been revealed to improve insulin sensitivity. Insulin secretion is also decreased in patients with CRF, which seems to be due to metabolic acidosis, raised levels of parathyroid hormone, and reduced level of vitamin D. It should be noted that in spite of the decreased insulin secretion and impaired tissue sensitivity to insulin that happens in patients with CRF, most nondiabetic CRF patients do not have hyperglycemia unless they are genetically predisposed.[40]

Once 'Glomerular Filtration Rate' turns out to be below than 15 - 20 cc/min, in advanced stages of CRF, break down of insulin in kidney & renal clearance of insulin, both reduces which is clinically important for treatment of diabetics . Though resistance of insulin raises insulin need, reduced degradation of insulin decreases the necessity for insulin administration in diabetics with advanced CRF or even decreases for diabetics. The hypoglycaemic risk raises with decreased insulin need.'Renal replacement therapy', peritoneal dialysis & haemodialysis moderately solve the difficulty in maximum people & insulin requirements change based on the quality of clinical improvement. Insulin requirements alter with the enhanced appetite & food consumption resultant from replacement therapy & lessening of uremic symptoms.[41]

For prevention of nephropathy, Control of diabetes & timely treatment for risk factors of diabetes are vital. Drugs to control diabetes and maintain strict glycemic control is important . 'End-Stage Renal Disease' occurrence in diabetics is reduced

with the drugs especially angiotensin II receptor antagonists & ACEIs as they are considered to be renoprotective .[41]

Also, some newer modalities , as well as consumption of inhibitors of advanced glycation end-products (AGEs), antifibrotic agents, endothelin receptor antagonists, growth factors & protein kinase C; receptor antagonists of advanced glycation end-products; glycosaminoglycans; oxidase inhibitors; NADPH has shown positive effects in stopping development of diabetic nephropathy. Current scientific research suggested that glycemic control be considered as a chief therapeutic aim in the treatment of diabetics with 'End-Stage Renal Disease'.[42]

2. ASSOCIATION BETWEEN HTN & CKD

Prevalence of hypertension continues to increase & approximately 74.5 million people ages 20 years & older have hypertension. Hypertension often goes together with evolving CKD, & it's wrongly expected as the reason of CKD rather than the result of CKD. Actually, many of the patients develop HTN from CKD than get CKD from HTN, i.e., hypertensive nephrosclerosis.[43]

Afro-Americans are affected with DHKD far more often than Caucasians. Genetic vulnerability to hypertensive nephropathy related with gene polymorphisms (e.g., APOL1, MYH9) in Afro-Americans may add to this risk.[44] Furthermore, hypertensive kidney disease in Afro-Americans is not alone ascribed to high BP & may imitate an underlying glomerular defect. Suspect these in non-diabetic persons identified with hypertensive nephropathy once their Urine Protein Creatinine are >1g or UACRs are greater than 300mg/g. Afro-Americans also respond lesser than Caucasian patients to monotherapy with ACEIs (angiotensin-converting–enzyme inhibitors), beta-blockers (BBs), &/or ARBs (angiotensin–receptor blockers). Nevertheless, ethnicity related alterations in the therapeutic reaction are usually

cancelled by associated diuretic therapy. For instance, response to combination use of thiazide diuretic with ACEI/ARB therapy is equivalent among various ethnicities. So, no specific drug groups should be evaded in patients of African American origins.

Asnon-diabetic CKD patients will have a similar probability for the occurrence of CVD as diabetics deprived of chronic renal disease, CV protective arrangements besides antihypertensive therapy should be measured. As a person's Blood Pressure feedback to high sodium consumption is not predictable, restriction of sodium should usually be imposed in CKD patients, i.e., <1.5 g sodium (65 mEq Na) per day.[45]

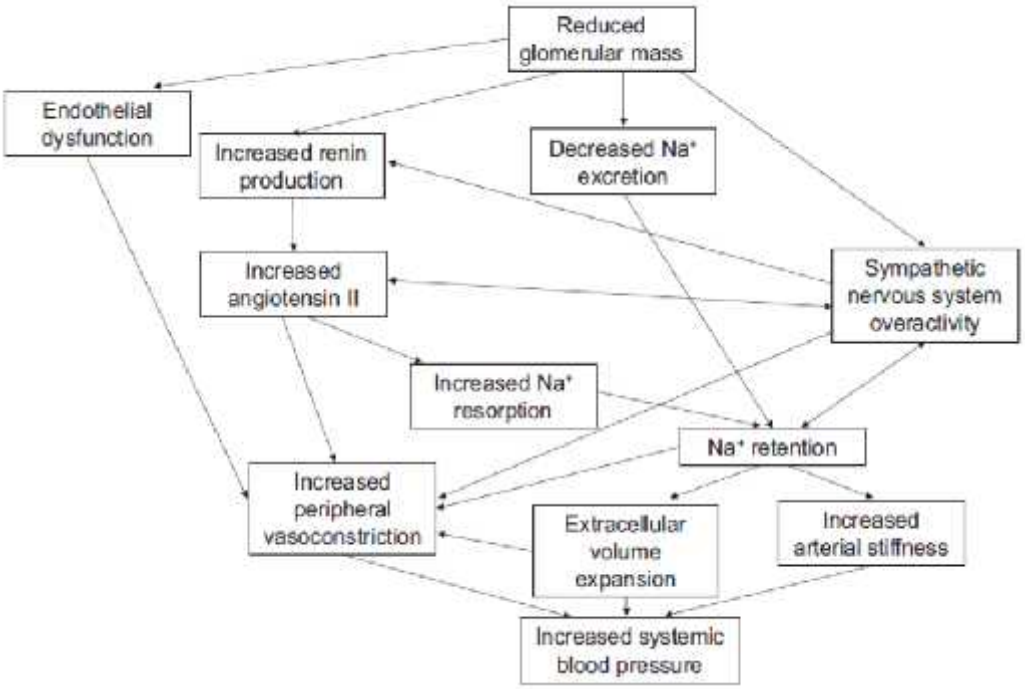
Blood Pressure Profile for Hypertension in CKD is measured by default as "resistant HTN", i.e., adjusted doses of three or greater antihypertensive agents are used for treatment & one of them which should be a diuretic. Typical BP outline is a Systolic greater than Diastolic, manifested as a raised pulse pressure (>55 mmHg). Either Systolic BP or pulse pressure may be elevated in hypervolemic or edematous persons who should regularly be given diuretics.

Proteinuria Assessment for & quantification of albuminuria/proteinuria is advocated when there's a family history of CKD or eGFR is less than 60 mL/min/1.73 m^2. To achieve an optimum target blood pressure, mainly systolic BP, will require 2 or greater antihypertensive medications in maximum cases, if SBP is ≥150 mmHg initially, 2 antihypertensive agents must be in use, an anti-renin–angiotensin–aldosterone system (RAAS) drug like an ACEI or ARB & whichever a diuretic (e.g., thiazide or loop diuretic, latter predicated by GFR) or CCB (calcium channel blocker).Ultimately, non-dihydropyridine calcium channel blockers (NDHPCCBs: diltiazem, verapamil) are ideal in proteinuric CKD, along with beta-blocker therapy, keeping in mind about bradycardia.[46]

3. CLINICAL PROFILE OF DIABETIC CKD WITH HTN &CKD - HTN PRESENTING WITH DIABETES

Hypertension is exceedingly seen in persons with DHKD & happens two times than in normal public. Particularly, the occurrence of Hypertension upsurges from thirty-six percent in CKD stage 1 to 84percent in more progressive CKD four & five stages. There is a constant link among decline in 'Glomerular Filtration Rate' (GFR) & occurrence of acute coronary syndromes. It's not merely mortality that's of importance, but also illness & prices of care connected to DHKD. It's a big role in consequence of the strong link of DKD with CVD results such as stroke, heart failure, Myocardial infarction, & 'End-Stage Renal Disease'.[47]

Figure 6: Hypertension Pathophysiology in DHKD:[48]



Patients with type 1 diabetes have albuminuria or obvious nephropathy, which usually results in Hypertension.[49] Nevertheless, in patients with diabetes type 2, Hypertension in many patients comes before the appearance of albuminuria

&estimated GFR (eGFR) reduces because of multiple 'risk factors', which comprises dyslipidemia, cardio-renal metabolic syndrome, & obesity. There are various mechanisms in the progress of Hypertension in patients with DKD, together which involves incorrect stimulation of 'renin-angiotensin-aldosterone system' & stimulation of sympathetic nervous system, peripheral vasoconstriction, volume expansion due to increased sodium reabsorption, up-regulation of endothelin 1, inflammation species, & decreased levels of Nitric Oxide.[11] A great deal of these issues speeds up the occurrence of renal disease & raise the risk for CVD in persons with Hypertension & diabetes.

Therefore, understanding pathophysiology targets risk alleviation of CKD. Though, optimum BP for managing of Hypertension is not as clear; a small number of randomized trials have evaluated dissimilar Blood Pressure levels in patients with Diabetic Nephropathy, & 1 was unsuccessful in proving an advantage of lower Blood Pressure on CV risk lessening. There's a discussion regarding 2014 Expert Panel report for the handling of Hypertension in those with Diabetes, in addition to those with renal disease. The report stated that those with Hypertension & diabetes should try for a BP target of less than 140/90 mmHg. But nowadays various organizations recommend for a target SBP of less than 130 mmhg for patients having CKD .[26,50]

4. LABORATORY PROFILE OF DIABETIC CKD WITH HTN &CKD - HTN PRESENTING WITH DIABETES

Diabetic Kidney Disease is identified on the basis of 'Glomerular Filtration Rate' (GFR) measurement & UACR to identify by albumin in urine. Clinically it's recognized by "estimated GFR" below sixty ml/min/1.73 m^2 or constant excretion of albumin in the urine (albuminuria). GFR reduced in Type 1 DM & Type 2 DM

despite having normal Urine Albumin excretion. Consequently, it's essential to quantify creatinine levels yearly in diabetics. There are some formulas for measurement of estimated GFR like 'Chronic kidney disease Epidemiology Collaboration' (CKD-EPI) equation which reflected weight, transplant, & diabetes as potential variables. CKD-EPI equation categorises patients as high risk (eGFR<90 ml/min/1.73 m2) & low risk (eGFR>90 ml/min/1.73 m2) category. A formula of "Modified Diet in Renal Disease" (MDRD) which computes GFR based on serum creatinine & few other features of patients. CKD-EPI equation was seen to be better compared to the MDRD equation in precisely stratifying CKD risk. American diabetes association guidelines endorse screening for nephropathy in a patient with more than five years of type 1 diabetes & in every Type 2 DM patients regardless of the time of onset of diabetes.[51]

5. MOST RELEVANT STUDIES –

a) GLOBAL:

FasilWagnew et al[52] **(2018),** conducted a systematic review & meta-analysis to find out Diabetic nephropathy & HT in diabetes patients of sub-Saharan countries. They included a total of 27 studies for meta-analysis. The pooled overall prevalence of diabetic nephropathy was seen to be 35.3 (95percent CI 27.46–43.14). In sub-group analyses by types of diabetes & regions, prevalence was 41.4percent (95percent CI 32.2–50.58percent) in type-2 diabetes mellitus & 29.7percent (95percent CI 14.3–45.1percent) in Eastern Africa. They concluded that Diabetic nephropathy is a common complication in diabetic patients & is significantly higher in hypertensive patients. They proposed that a preventive strategy should be adopted or planned to

reduce diabetes mellitus & its complication of neuropathy, particularly in hypertensive.

Abraham et al[4] (2017), conducted a study to find out the relationship between HT, diabetes & 'Chronic kidney disease' in Arab countries. They concluded that escalation of the social burden of (pre-) HT, (pre-) diabetes, END-STAGE RENAL DISEASE & CVD, as mediated by obesity, in the Arab region cannot be ignored. They also put forward a suggestion that these policies & systems should include planning, funding & deployment of necessary preventive, clinical & rehabilitative services & workforce needed to deliver care.

Simon DS Fraser et al[33] (2016), conducted a review to encapsulate dynamic role that primary care shows in predialysis CKD care & chief considerations in its monitoring, identification & clinical management. They proposed that CKD is often asymptomatic in its early stages, & clinicians working in primary care have a vital role to play in its identification, risk stratification, & monitoring. Primary care also has a pivotal role in the prevention of complications & progression in managing 'risk factors' such as high BP & prevention in AKI. CKD often occurs in conjunction with other chronic disease comorbidities, & primary care clinicians are best placed to take a holistic view of care in mild-to-moderate CKD & empower patients. They suggested that in alignment with principles of the World Health Organization World Health Report 2008, CKD is a good exemplar of why primary health care is needed now more than ever.

Roberto Pecoits Filho et al[53] (2016), in their review tried to find out the relationship between diabetes & kidney disease based on a report of discussions from an interdisciplinary group of experts in areas of endocrinology, diabetology& nephrology. They concluded that understanding renal physiology & pathophysiology of DKD is essential to all specialties treating diabetic patients.

Nathan R. Hill et al[54] **(2016)** conducted a Systematic Review & Meta-Analysis on Global Prevalence of Chronic Kidney Disease. Of 5,842 potential articles, 100 studies of diverse quality were included, comprising 6,908,440 patients. Global mean (95percentCI) CKD prevalence of 5 stages 13.4 percent (11.7–15.1percent), & stages 3–5was 10.6 percent) (9.2–12.2percent). Assessing by study quality did not affect prevalence estimates. They found out that CKD prevalence by stage was Stage-1 (eGFR>90 +ACR>30): 3.5percent (2.8–4.2percent); Stage-2 (eGFR 60–89+ACR>30): 39percent (2.7–5.3percent); Stage-3 (eGFR 30–59): 7.6percent (6.4–8.9percent); Stage-4 = (eGFR 29–15): 0.4percent (0.3–0.5percent); & Stage-5 (eGFR<15): 0.1percent (0.1–0.1percent). CKD has a high global prevalence with a consistent estimated global CKD prevalence of between 11 to 13percent with majority stage 3.

SeyedBahmanGhaderian et al[55] **(2014),** from their study on 'role of diabetes mellitus & Hypertension' in 'Chronic kidney disease' concluded that although diabetic nephropathy & hypertensive nephrosclerosis as frequent reasons of end-stage renal disease in emerging countries, however, possibly because of unawareness of patients with CKD & late recommendation of patients with CKD to nephrologists, causes of END-STAGE RENAL DISEASE insignificant percent of patients in developing countries are still unknown & therefore everyone with 'risk factors' of CKD such as high blood pressure, diabetes mellitus, metabolic syndrome, family history of CKD & proteinuria have to be educated about benefits of early identification of disease & subsequent kidney protection through appropriate interventions.

Rieokada et al[56] **(2014),** conducted a study to find out the number of metabolic syndrome components as a good risk indicator for both early- & late-stage kidney

damage. In their study, a vast number of subjects were included numbering to 2, 05,382 people aged 40-74 years who underwent Specific Health Checkups in Aichi Prefecture, Japan. Prevalence of renal hyper filtration [estimated 'Glomerular Filtration Rate' (eGFR) above age-/sex-specific 95th percentile] & hypo filtration (eGFR below 5th percentile) was compared according to number of MetS components. They found that the prevalence of both hyper filtration & hypo filtration increased with an increasing number of MetS components. Renal hyper filtration was associated with prehypertension & pretension, & overt diabetes. In their study, they concluded that a number of MetS components is a good risk indicator of early- & late-stage kidney damage. Therefore, kidney function should be monitored in subjects with MetS components. They advocated that MetS components should be treated as early as possible to prevent the development of kidney damage & cardiovascular diseases in people with hyper filtration, regardless of their body weight.

George L. Bakris et al[57] **(2011**), conducted a study of pathophysiology in Nephropathy in Patients with Diabetes Mellitus Type 2 and various treatment modalities in the same. He concluded that Nephropathy is a usual microvascular complication within type 2 DM patients & the main reason for renal failure. It categorized by macroalbuminuria (≥300 mg/d) & a decreased 'Glomerular Filtration Rate' & often seen at detection of diabetes after a kidney has been subjected to long term hyperglycemia during the initial diabetic phase. A low 'Glomerular Filtration Rate' (<60 mL/min/1.73 m2) is an independent contributing factor in CV mortality. Diagnosing diabetic nephropathy during its early stages has scope for prior therapeutic interferences to prevent or delay complications & bad outcomes. Systematic management of multiple factors is the method which is needed that targets all risk determinants at the same time. The approach should comprise lifestyle

changes along with target achievement of blood pressure, blood sugars, & lipid targets that are evidence-based.

Van Buren et al[11] **(2011),** described that diabetes is related with a prevalence of CKD of 8.9 percent (stage I), 12.8percent (stage II), 19.4percent (stage III), & 2.7percent (stage IV & V combined).

Robert c. Atkins et al[12] **(2005),** conducted a study to find out the epidemiology of chronic kidney disease. In his, he proposed that Diabetes is a magnanimous disease, & its prevalence may increase two-fold in the coming twenty-five years, chiefly in emerging countries. This will place an enormous financial burden on countries, with the cost of management of end-stage renal failure. He stated that hence, it's medically & frugally important for awareness, discovery, & preventive programs to be professed across the world, chiefly in developing countries. This will require concentrated action from global organizations, governments, health service providers, & medical doctors.

b) INDIAN

Salman Hussain et al[51] **(2020),** in their study assessed the prevalence, 'risk factors', & biomarkers in Diabetic kidney disease. They proposed that major obstacle in early diagnosis is limited information, lack of routine screening. Well-timed diagnosis & correct mediation are foremost plans to deal with this disastrous health situation. The quick analysis may have lifetime advantage by controlling the development of the disease, increasing life expectation, decreasing humanistic & financial burden. Even after all these benefits; DKD cases are identified when patient's health deteriorates. Unavailability of diagnostic biomarkers was said by them to be the main obstacle to early diagnosis of DKD.

Suresh Chandra Dash et al[22] **(2019**), in their study assessed incidence of Diabetes mellitus as a reason 'Chronic kidney disease'(CKD), connotation between diabetic-CKD (diabetics who then developed CKD as complication), hypertension (HT) & obesity in 6175 patients attending Delhi & other in Bhubaneswar. They concluded that Diabetes had been a greater cause (62.3percent) of CKD in our country. At demonstration association of diabetic-CKD with HT was noted higher (78.7percent) in our country, 54.4percent of diabetics reporting to medicine Out Patient Department were not tested by any physician or GP for CKD because urine albumin & serum creatinine tests were absent. Thus, advance to CKD in most patients went ignored. They were one of the first to use syndrome "DHKD", (complex of diabetes, HT & kidney disease).

Rahul Sudan et al[9] **(2018),** in their study assessed outline of 'Chronic kidney disease'(CKD) patients giving in a tertiary care center across Srinagar, Jammu & Kashmir, Our country. They concluded from their study that is necessary to diagnose patients of CKD in early asymptomatic stages. At these stages it's feasible to slow down the reduction of nephrons by controlling underlying factors like diabetes & Hypertension but once the stage of END-STAGE RENAL DISEASE sets in, renal transplantation as the preferred modality of 'renal replacement therapy'.

Varughese et al[5] **(2018),** in their study, tried to elucidate burden & future changes required to manage CKD in Our country. They proposed that India has unique situations & challenges that influence early diagnosis & management of CKD. Prevention & early detection of CKD mandate involvement of doctors at all levels. Most patients with CKD can be managed by their primary doctors with timely nephrology referrals.

Verma et al[58] **(2016),** conducted a prospective cohort study to examine the link between albuminuria, Hypertension & estimated 'Glomerular Filtration Rate' (eGFR) in T2DM people in Our country on 824 patients. They found for Diabetic Kidney Disease; HT was an independent risk factor. Comparative elevation in serum creatinine levels &eGFR was found with an occurrence of Hypertension in Diabetic Nephropathy.

SwarajSathyan et al[59] **(2016),** conducted a study in a tertiary care referral center in South India on Clinical & epidemiological profile of 'Chronic kidney disease'. In their study of over three hundred patients included in the study, 217 (65percent) were males & 116 (35percent) were women. Most of the patients aged between 21-60 years were there. Chronic glomerulonephritis was the most shared etiological diagnosis (51percent) followed by Diabetic Nephropathy (22percent) & 'hypertensive nephrosclerosis' (7percent). About 24 percent had Diabetes while 84 percent of patients had Hypertension. 'Dyspnea' (75.68percent), symptoms indicative of volume overload in 242 (72.7percent) & 'Oliguria' (69percent) were the main complaint. An overwhelming majority of patients in the study presented in stage 5 CKD (264, 79.2percent). 167 (50.15percent) patients were found to exhibit cardiovascular disease. Cigarette smoking was seen in 32.7 percent, alcohol consumption in 6.91 percent, NSAID use in 5.1 percent & herb minerals in 4.5 percent. Mean hemoglobin in the study was 8.42 g/dl. Mean phosphate level in study was 5.94 mg/dl. They found that there was an important statistical correlation between hemoglobin levels & stage of CKD & also between serum phosphate level & stage of CKD. They suggested that early detection of CKD by a screening of 'high-risk persons' will go a long way in decreasing development of end-stage renal disease & help in lessening huge load due to mismatch between demand & availability of resources for 'renal replacement

therapy 'in developing countries like Our country, especially for patients belonging to a lower socioeconomic group.

Shashank R. Joshi et al[60] **(2012**), in their study on Prevalence of Diagnosed & Undiagnosed Diabetes & HT in India, found out that substantial burden of diabetes & HT is on rising in India & that Patient awareness & timely diagnosis & intervention hold the key to limiting this double epidemic after they screened from 2009–2010, in total, 15,662 eligible patients (54.8percent males; mean age, 48.9 – 13.9 years) from eight states. They found that Diabetes was prevalent in 5,427 (34.7percent) patients, & 7,212 (46.0percent) patients had hypertension. Diabetes & HT were coexistent in 3,227 (20.6percent) patients. Among those whose disease status was not known at enrollment, 7.2percent (793 of 11,028) & 22.2percent (2,408 of 10,858) patients were newly diagnosed with diabetes & HT, respectively; additionally, 18.4percent (2,031 of 11,028) were classified as having prediabetes& 60.1percent (6,521 of 10,858) as having pre-HRT.

Agarwal et al[61] **(2011),** have studied three hundred recently diagnosed diabetes type II & have found an occurrence of 17.5percent of nephropathy & stated Hypertension as a vitally related factor contributing to progress to renal disease.

Agarwal et al[22] **(2009)** tried to elucidate Challenges & Solutions in various regions of Our country for managing Chronic Kidney Disease. They concluded that in Our country there's quite a long way to go with respect to CKD & 'til then, in Our country, screening of high-risk persons for CKD & 'risk factors' as finest option.

Murugesan Ram Prabahar et al[21] **(2008),** in their study on the Epidemic of 'Chronic kidney disease' in Our country put forward a few suggestions. They proposed that prevention of CKD must be the aim of the medical community, general public & government of India, & that there are already many effective & attractive

interferences for treatment & prevention of CKD exist & many more confidently may be developed.

6. DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME –

a) Terminology – history – when it is used, officially recognized by any organization

Ideal treatment of patients with diabetic hypertensive kidney disease (DHKD) needs right interpretation, markers use & designating phases of CKD, stratifying diabetics and Hypertensives, speedy disease recognition, & team work between nephrologists & primary care doctors. Since multiple terms can be confusing for 'Chronic kidney disease'(CKD), e.g., ''Chronic renal disease'', chronic renal insufficiency, & chronic renal failure, National Kidney Foundation Kidney Disease Outcomes Quality Initiative™ (NKF KDOQI™) has defined all-inclusive term, CKD.[62] Using kidney rather than renal increases awareness for families, patients, healthcare workers, & lay public. The above term includes the continuation of kidney dysfunction from mild kidney damage to kidney failure, & it also encompasses the term 'end-stage renal disease' (ESRD).

b) Interpretation & Definition

"DHKD syndrome is defined with persistent severely elevated albuminuria of >300 mg/24 hour or urinary albumin to creatinine ratio [UACR] of > 300 mg/g, a relentless decline in 'Glomerular Filtration Rate', raised arterial blood pressure & enhanced cardiovascular morbidity." DHKD syndrome is not yet recognized globally.

An epidemiological study conducted by Dash et al[63] (2018), provides sufficient evidence endorsing high link between Diabetes, hypertension & Kidney Disease is like a connection. More than fifty percent of diabetics in India are ignorant of the presence of kidney dysfunction prior to presentation due to a lack of simple

urine albumin test & or serum creatinine estimation. Therefore 'CKD' at key initial stage go unobserved, which advances in stages with more severity. This is a fault & so it is need of the hour for usage of "Diabetic Hypertension-Kidney Disease (DHKD) syndrome" in making attentiveness among subjects & physicians for early recognition of Diabetic Nephropathy, at a stage when it may be efficiently treated.

c) Epidemiology- Global, India, (HTN in diabetic CKD or DM in Hypertensive CKD)

'Chronic kidney disease' in India:

There is no data entry regarding renal diseases in India. Hence the actual status of 'CKD/end-stage renal disease' in our populace is unknown. From the population-based survey from Northern Indian region, by 'multistage cluster sampling method' where serum creatinine & urine samples were checked among patients calculated, the prevalence of CKD stage three was found in 0.79percent among 4,972 examined.[64] Collective source of CKD in this 'population-based study' was Diabetic Nephropathy responsible for 41percent cases. In a Southern Indian study incidence in impaired kidney function was found to be 8.6per 1000 among twenty-five thousand & then 13.9per 1000 among another twenty-one thousand. Prevalence of other kidney diseases was seen in 0.68percent & CKD was seen in 0.16percent in the first study.[65] Urban Central Indian study showed a comparable disease status as inferred disease status from a Northern Indian survey.[7] Diabetes & Hypertension suspected to be the source for CKD in these 'hospital-based studies' approximated 30percent among all. From a new 'hospital-based study', from hospitals, occurrence in stage 3 CKD & beyond noted in 0.8percent.[7] In a pilot project that was initiated by nephrologists the information source on 'CKD', in particular, CKD patients coming to hospitals was given. It later received validation from 'National Society of Nephrology'. In 2005

Indian CKD Registry, was started to know the various comorbidities and causes for CKD with information of thirty thousand subjects procured till date. Male subjects are 70percent in this study, with an average of ≈45–50 years with mostly in stages four and three. In nearly 30percent of patients, CKD is due to DM, with T2DM in ≈97 percent, having Diabetes for <10 years for ≈40percent. 'CVD' observed frequently as stages of CKD progressed; ≈0.7percent in stages 1 to 43percent in stages 5. For now, there's no continuation statistics. Additional data produced about 'CKD' in Our country is from 'Screening & Early Valuation of Kidney Disease' (SEEK), initiated by nephrologists in America 2006. 'SEEK' has set various goals to understand the various disease aspects of CKD. 'SEEK' data has been obtainable at 'Annual Conference of Indian Society of Nephrology' for over 2 years. 'Serum creatinine' & urine examination could be attained in 93percent of patients. It's noteworthy that in the majority of studies cited above; 'eGFR' was calculated by MDRD formula, that has yet to be applied in Indian populace. eGFR is used to still for GFR measurement in 'community-based studies' in Our country. Moreover, with the raising life expectancy, the DHKD may also rise. A 'multicentric survey', is proposed with both rural & urban populaces for better understanding of the disease.[22]

d) Clinical presentation & lab diagnosis

Detailed history, physical examination, duration of diabetes & hypertension, prior records of renal function tests should be examined to classify individuals into DHKD syndrome.. Renal function testsinclude serum creatinine, Blood urea nitrogen , serum electrolytes . Urinalysis includes urine protein and urine albumin to creatinine ratio whichwere calculated by taking the ratio between urinary albumin or urine protein & urinary creatinine in random spot urine.

"Serum creatinine ≥ of 1.2mg/dL & Serum creatinine should be measured on greater than 3 occasions in six months. Proteinuria may be considered into normal albuminuria [<30mg/g] or micro-albuminuria [30-300mg/g] macro-albuminuria [>300mg/g]".[63]

"Renal damage is defined by these findings: a) 'pathologic kidney abnormalities' b) 'persistent proteinuria' c) other urine 'abnormalities, e.g., renal hematuria d) imaging abnormalities e) eGFR<60 mL/min/1.73 m2 on two occasions separated by ≥90 days' & that's not seen with a 'transient, reversible condition such as volume depletion'." Only five & ten percent of 'general population' experiences a 'screening urinalysis' or a 'Serum Cr', correspondingly. Anemia in CKD & 'CKD-Mineral & Bone Disorder (CKD-MBD)' are seen in third Stages. Hypertension is seen in third to fifth Stages of CKD & 'acid-base balance, dyslipidemia, & glucose homeostasis' turn unstable late stages. To facilitate the low eGFR, reduced dosages of medicine are given in third to fifth stages of CKD. Disease areas of 'Hypertension, urine protein excretion, & increased lipid levels may occur at any stage & therapy should be beset adjusted accordingly. Patients with CKD alone have 2-4 times the risk of CVD. In patients with CKD &hypertension & diabetes, there is a 4-8-fold chance of CVD happening. CVD risk worsens through CKD third to fifth Stages, and also there is increased mortality in these patients.

Treatment:

Managing CKD as well as DHKD syndrome requires thorough comprehension of its definition as suggested in 'National Kidney Foundation'.[46] An well-versed understanding of 'estimated Glomerular Filtration Rate (eGFR)' is necessary, as GFR is a standard tool to define kidney function in normal persons as well as patients.

Categorization of CKD to five stages is important. Consequently, for patients during periodic healthcare meetings, clinical 'causative factors' for commencement &/or advancement of CKD should be determined. 'eGFRs' should be assessed frequently in those at risk of CKD and required measures be taken in those with greater risk.

Hypertension & Diabetes are 2 important ''risk factors'' of CKD, and together they constitute 72 percent for the 'end-stage renal disease'. Along with these 'insulin resistance', 'obesity',& 'metabolic syndrome' were added as 'risk factors'. Nearly 24 percent of 'end-stage renal disease' subjects may have a relative with CKD, that's seen much more in Afro-Americans than Caucasians. Other CKD 'risk factors' comprise following: 'AKI/ARF, urinary tract obstruction, stones, nephrotoxins (analgesics, aminoglycosides, amphotericin, radiocontrast), reduced kidney mass (solitary kidney), autoimmunity (SLE), low birth weight, preeclampsia, socio-demographics (older age, reduced access to healthcare, male gender, low income/education level, hazardous chemical or environmental exposures' should be noted.[46]

'Diabetic kidney disease'/ 'diabetic nephropathy'., people who have Diabetes & 'Chronic kidney disease' constitute DKD, that show 'increased urine albumin excretion' or low 'Glomerular Filtration Rate' (GFR) / together.[66] This tweaks the usual process of 'elimination of waste products' & excess fluid from the body and hamper renal function. 'Albuminuria, frequent urination at night, increased weight attainment, swelling of ankle & legs, sick feeling, anemia, & high blood pressure' are the Sign & symptoms.[63] DKD may lead to CKD & end-stage renal disease.[67] It is also dissimilarly growing among developing nations and still not identified by the organizations.[51] Kidney disease augmented mortality risk by 31.1 percent diabetics

&it augments with the seriousness of the disease.[68] Moreover, DHKD may demand a lot of capital as it is a health burden. DKD in prior stages is often undiagnosed until appearances of ill-fated complications.[69] Due to limited knowledge, lack of screening early diagnosis may be not possible.[51]

Higher incidence of DHKD is seen among 'Indo-Asians in UK, Mexican-Americans, African-American, in USA & Pima Indians'.[70] This may be due to genetic predisposition among them.

Pathophysiologically DHKD is related with hypertension through the process of 'salt & water retention, ischemic damage to renal medulla'. The genetic basis was established with Diabetes & Hypertension. Together they also cause 'renal, retinal microagiopathy'.[71,72] 'Genetical link: (i) Angiotensinogen: Amongst many candidate genes connecting Hypertension to Diabetic nephropathy genes of the renin-angiotensin system (RAS), has been accredited. Pro-renin, renin, ACE & angiotensin level are raised in diabetic nephropathy.[73] (ii) Furthermore, studies have implicated genes of the renin-angiotensin system to be determinants for both Hypertension & diabetic kidney damage along with the cardiovascular disease. (iii) Linkage of M235T polymorphism gene has been definite in essential Hypertension. (iv) Though there are contradictory reports, one study has clearly shown association TT genotype with raised blood pressure in people with diabetic nephropathy'.[16]

LACUNAE IN LITERATURE:

There is sufficient data to show an association between diabetes, hypertension, ‘‘Chronic renal disease’’. Many studies have been done to identify the association between CKD, DKD, & hypertension. However, the relationship between all three is inadequate. Clinical & lab findings are not studied till date for DHKD syndrome. A thorough understanding of clinical features & laboratory parameters will aid in understanding syndrome better. Very few studies have been done in India & across the world. Our study is one of the firsts to establish a base for DHKD syndrome.

MATERIALS AND METHODS

Study location: The present study was carried at the department of general medicine & nephrology at Dr KLE Prabhakar KoreHospital & MRC.

Study population:subjectswith diabetes & hypertension in combination with kidney disease with any duration of Diabetes > 2 years & any duration of hypertension >2 years. Attended to the department of General Medicine &nephrology at Dr. KLE Prabhakar KoreHospital & MRC were considered as the study population.

Study design: the current study was a hospital-based observational cross-sectional study

Sample size:

Sample size- 120

$$n=Z^2pq/d^2$$

(n= sample size, z= confidence interval, p=prevalence being 88 percent in one of Indian study q=100-p, d=absolute error being 6)

Sampling method: All eligible subjects were selected into study consecutively by convenient sampling till sample size is achieved.

Study duration: data collection for the study was done between January 2019 to December 2019 for a period of one year.

Inclusion Criteria:

- Subjectswith diabetes & HT in combination with kidney disease with any duration of Diabetes > 2 years& any duration of HT >2 years.
- Patients age more than 25 yrs.

Exclusion criteria:

- Acute kidney injury.
- 'Chronic kidney disease' of other etiology including membranous nephropathy, mpgn (membranous proliferative glomerulonephritis), fsgs(focalsclerosingglomerulonephritis) and CKD of etiology other than diabetes & Hypertension.
- Isolated Hypertension / Isolated diabetes / Isolated kidney disease / type 1 diabetes mellitus.

Ethical considerations: Study was approved by the institutional human ethics committee. Informed written consent was obtained from all study participants & only those participants willing to sign informed consent were included in the study. Risks & benefits involved in study & voluntary nature of participation were explained to participants before obtaining consent. Confidentiality of study participants was maintained.

Data collection tools: All relevant parameters were documented in a structured study proforma'.

Methodology: All patients fulfilling inclusion criteria & willing to participate were included in the study. Informed consent was obtained & then the patient was enrolled for the study.

DHKD syndrome is defined with persistent severely elevated albuminuria of >300 mg/24 hour or urinary albumin to creatinine ratio [UACR] of > 300 mg/g, a relentless decline in 'Glomerular Filtration Rate', raised arterial blood pressure & enhanced cardiovascular morbidity. Detailed history, physical examination, duration

of diabetes & hypertension, prior records of renal function tests were analyzed. . Renal function testsinclude serum creatinine, Blood urea nitrogen, serum electrolytes. Urinalysis includes urine protein and urine albumin to creatinine ratio whichwere calculated by taking the ratio between urinary albumin or urine protein & urinary creatinine in random spot urine. 'Serum creatinine ≥ of 1.2 mg/dL (as per our lab standard) &Serum creatinine were measured on more than 3 occasions in 6 months (after correction of acute kidney injury factors if any).Proteinuria categorised into normal albuminuria [<30 mg/g] or micro albuminuria [30 to 300 mg/g] or macro albuminuria [> 300 mg/g]'.

Patients were then scored based on MDRD [modified diet in renal disease] formula to calculate estimated glomerular function rate & placed into various stages of CKD. Using serum creatinine levels, estimated 'Glomerular Filtration Rate' for each patient was computed according to the equation of MDRD FORMULA [modification of renal diet] & CKD EPI FORMULA ['Chronic kidney disease' epidemiology collaboration equation] & AVERAGE Were TAKEN.

MDRD EQUATION:

GFR (mL/min/1.73 m^2) = 175 × $(S_{cr})^{-1.154}$ × $(Age)^{-0.203}$ × (0.742 if female) where eGFR is measured in ml/min per 1.73m2, creatinine is in mg/dl, & age in years.

- **CKD EPI EQUATION**

$GFR = 141 \times \min(Scr/\kappa, 1)^{\alpha} \times \max(Scr/\kappa, 1)^{-1.209} \times 0.993^{Age} \times 1.018[\text{if female}] \times 1.159[\text{if black}]$

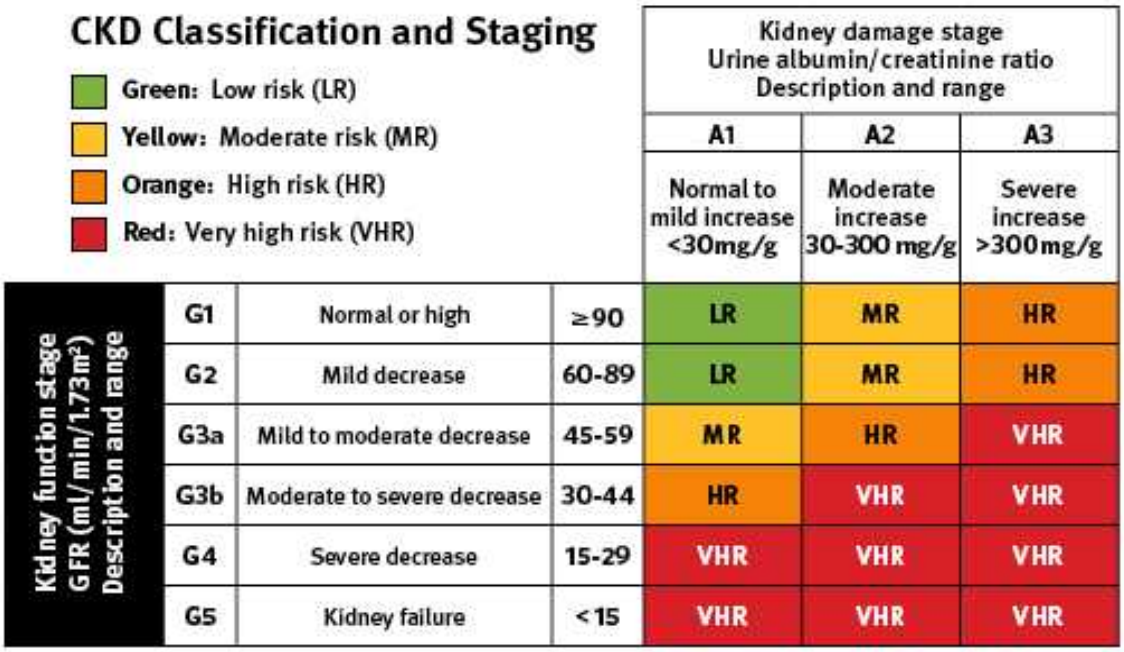
$\kappa = 0.7$ if female
$\kappa = 0.9$ if male

$\alpha = -0.329$ if female
$\alpha = -0.411$ if male

min = The minimum of Scr/κ or 1
max = The maximum of Scr/κ or 1

Scr = serum creatinine (mg/dL)

Figure 7: CKD classification and staging



Patients were then categorized to have CKD if eGFR was < 60 ml/min per 1.73m2 & not to have CKD if eGFR was ≥ 60 ml/min per 1.73m2.

USG abdomen, including the size of kidney & grading for ckd. Regular BP Recording to categorize into hypertension if BP IS >130/80 mm/hg as per American heart association guidelines 2017

- **Diabetes is defined according to ADA**

Type 2 Diabetes ADA Diagnosis Criteria

Diagnostic criteria by the American Diabetes Association (ADA) include the following:

- A fasting plasma glucose (FPG) level of 126 mg/dL (7.0 mmol/L) or higher, *or*
- A 2-hour plasma glucose level of 200 mg/dL (11.1 mmol/L) or higher during a 75-g oral glucose tolerance test (OGTT), *or*
- Random plasma glucose of 200 mg/dL (11.1 mmol/L) or higher in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis

Whether a haemoglobin A1C (HbA1c) level of 6.5percent or higher should be a primary diagnostic criterion. UACR is urine albumin is to urine creatinine ratio is available by laboratory testing.

STATISTICAL METHODS:

MDRD, 'Chronic kidney disease'&'Glomerular Filtration Rate' (GFR) were considered as primary outcome variables. Micro albuminuria, Macro albuminuria was considered as Primary explanatory variable. Age, Gender, decreased urine output etc were considered as other explanatory variables.

Descriptive analysis was carried out by mean & standard deviation for quantitative variables, frequency & proportion for categorical variables. None normally distributed quantitative variables were summarized by median & interquartile range (IQR). Data was also represented using appropriate diagrams like a bar diagram, pie diagram & box plots.

All Quantitative variables were checked for normal distribution within each category of an explanatory variable by using visual inspection of histograms & normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as a normal distribution.For normally distributed Quantitative parameters mean values were compared between study groups using Independent sample t-test (2 groups) / ANOVA (>2 groups).

Categorical outcomes were compared between study groups using Chi square test /Fisher's Exact test (If the overall sample size was < 20 or if the expected number in any one of cells is < 5, Fisher's exact test was used.)

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.

RESULTS

A total of 120 subjects were included in the final analysis.

Table 1: Descriptive analysis of age (years) in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Age (Years)	63.64 ± 10.80	64.0	40.0	89.0	61.7	65.6

The mean age was 63.64 ± 10.80 years in the study population, minimum and maximum was 40 and 89 in the study population with (95% C. I 61.7 to 65.6). (Table 1)

Table 2: Descriptive analysis of age group (years) in the study population (N=120)

Age Group (years)	Frequency	Percentages
40-49	14	11.67%
50-59	26	21.67%
60-69	40	33.33%
70-79	31	25.83%
80-89	9	7.50%

Among the study population, 14 (11.67%) participants were aged between 40 to 49 years, 26 (21.67%) were aged between 50 to 59 years, 40 (33.33%) were aged between 60 to 69 years, 31 (25.83%) were aged between 70 to 79 years and 9 (7.50%) were aged between 80 to 89 years. (Table 2 & Figure 8)

Figure 8: Pie chart of age group in the study population (N=120)

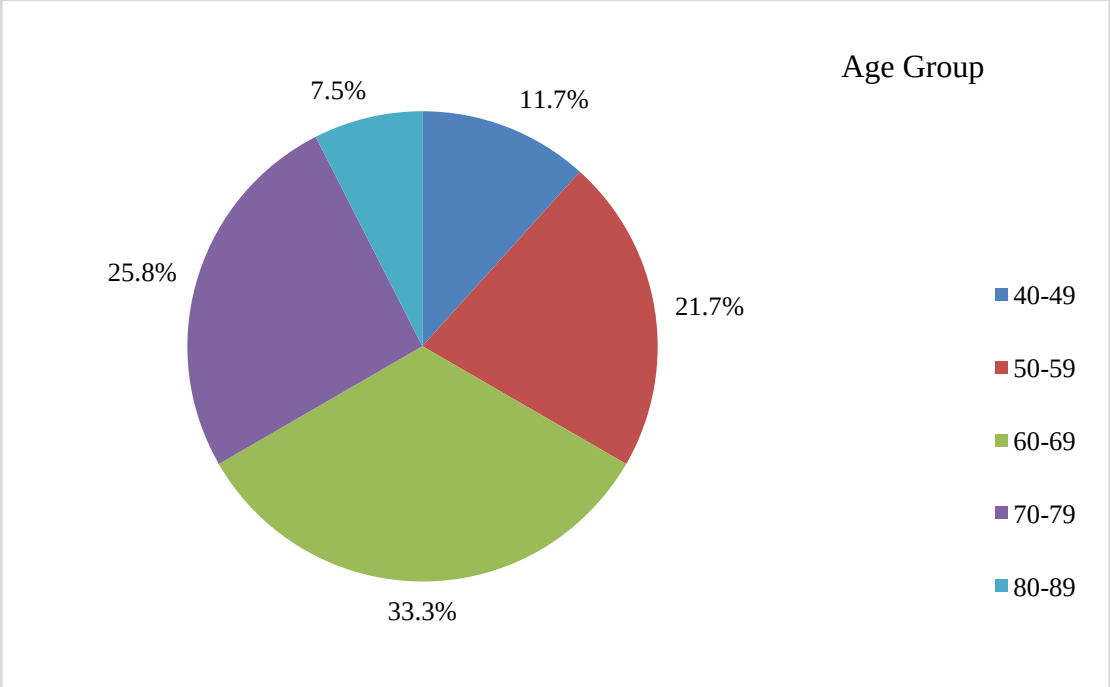


Table 3: Descriptive analysis of gender in the study population (N=120)

Gender	Frequency	Percentages
Male	87	72.50%
Female	33	27.50%

Among the study population, 87 (72.50%) were male and 33 (27.50%) were female. (Table 3 & Figure 9)

Figure 9: Bar chart of sex in the study population (N=120)

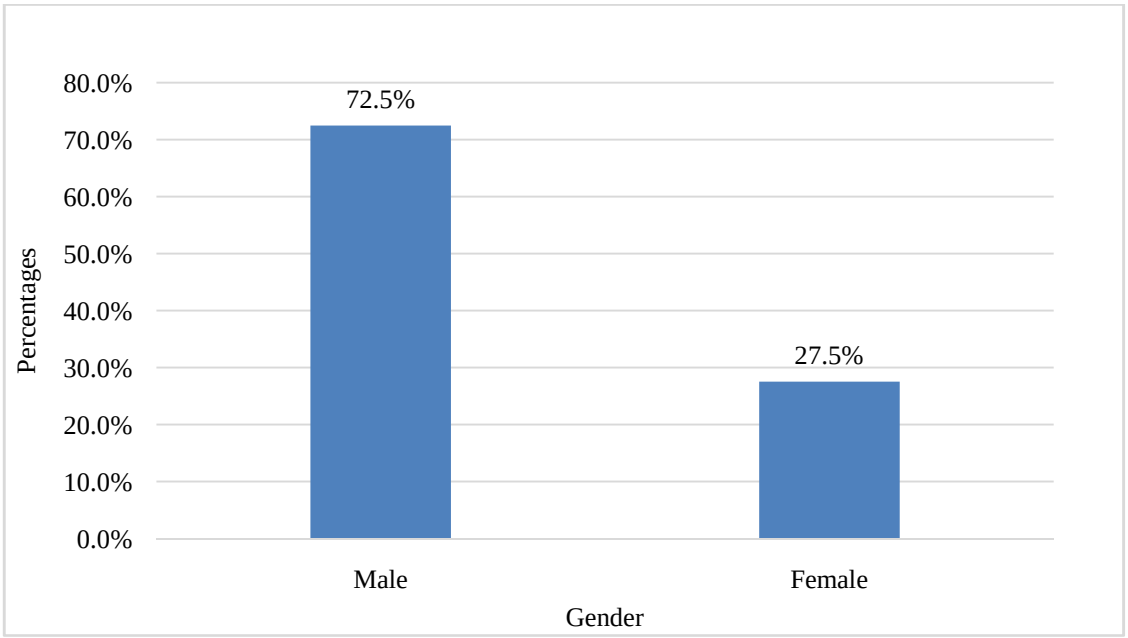


Table 4: Descriptive analysis of generalized weakness in the study population (N=120)

Generalized Weakness	Frequency	Percentages
Yes	94	78.33%
No	26	21.67%

Among the study population, 94 (78.33%) had generalized weakness. (Table 4 & Figure 10)

Figure 10: Bar chart of generalized weakness in the study population (N=120)

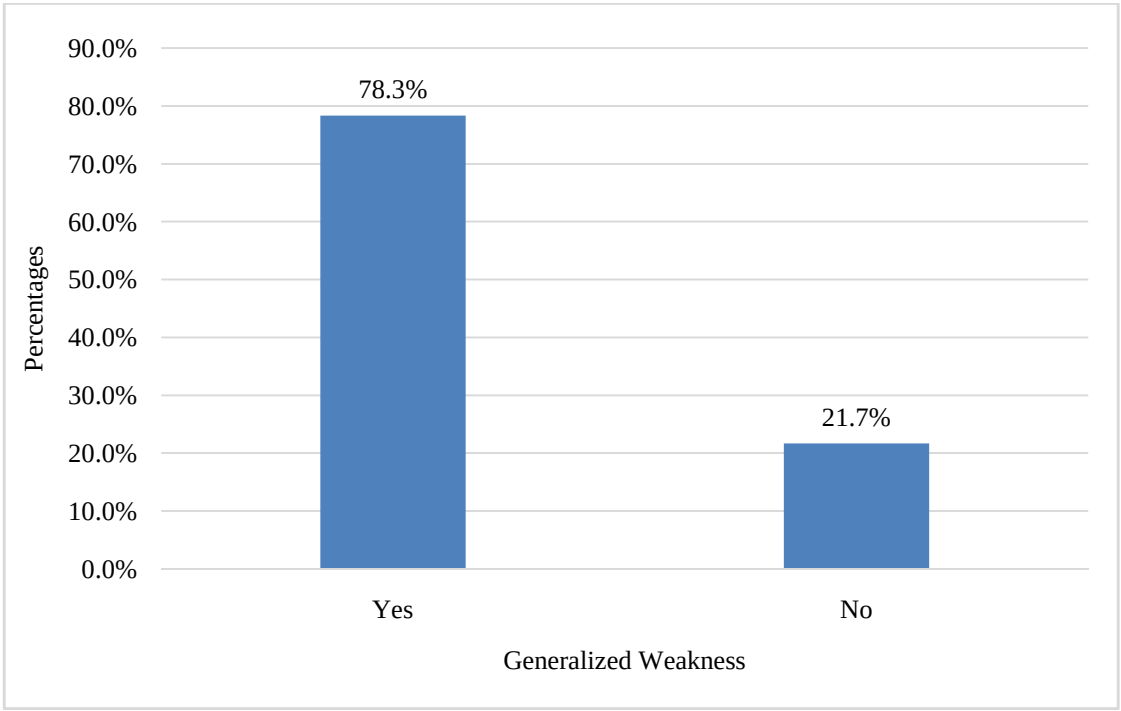


Table 5: Descriptive analysis of Peripheral edema in the study population (N=120)

Peripheral Edema	Frequency	Percentages
Yes	86	71.67%
No	34	28.33%

Among the study population, 86 (71.67%) had peripheral Edema.

(Table 5 & Figure 11)

Figure 11: Bar chart of Peripheral edema in the study population (N=120)

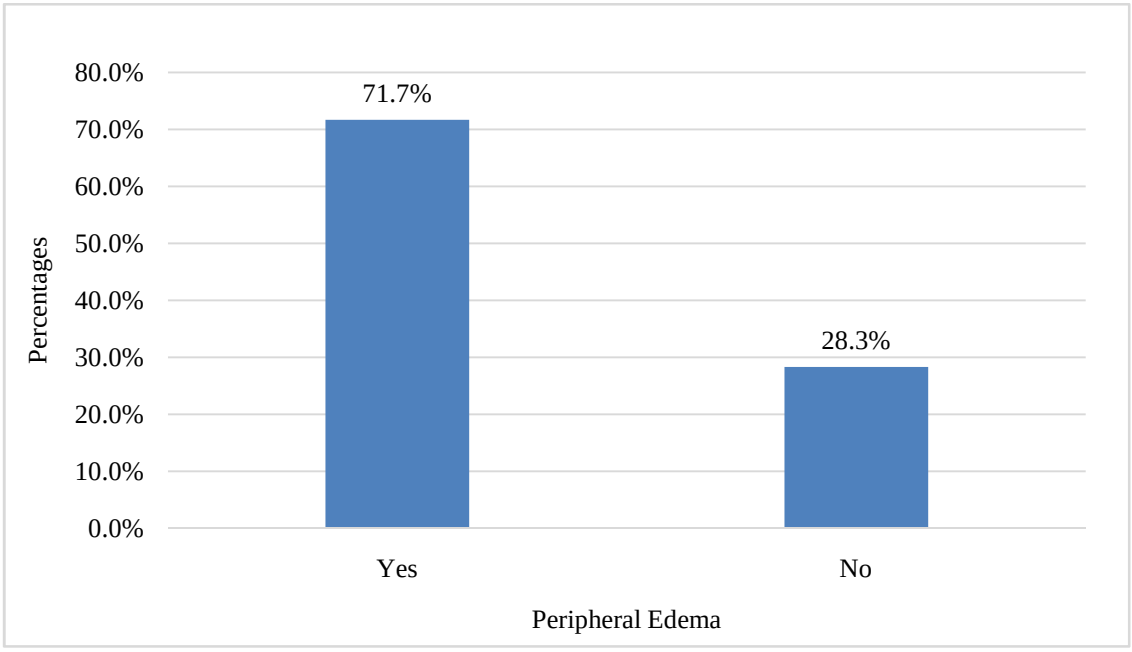


Table 6: Descriptive analysis of complaints at presentation in the study population (N=120)

Complaints at presentation	Frequency	Percentages
Decreased urine output		
Yes	57	47.50%
No	63	52.50%
Loss of appetite		
Yes	58	48.3%
No	62	51.7%
Nausea and Vomiting		
Yes	21	17.5%
No	99	82.5%
Dyspnoea		
Yes	71	59.17%
No	49	40.83%
Weight Loss		
Yes	30	25.00%
No	90	75.00%
Change in mental status		
Yes	13	10.8%
No	107	89.2%

Among the study population, 57 (47.50%) had decreased urine output, 58 (48.3%) had loss of appetite, 21 (17.5%) had nausea and vomiting, 71 (59.17%) had dyspnoea, 30 (25.00%) had weight loss and 13 (10.8%) had change in mental status. (Table 6 & Figure 12)

Figure 12: Bar chart of Complaints at presentation in the study population (N=120)

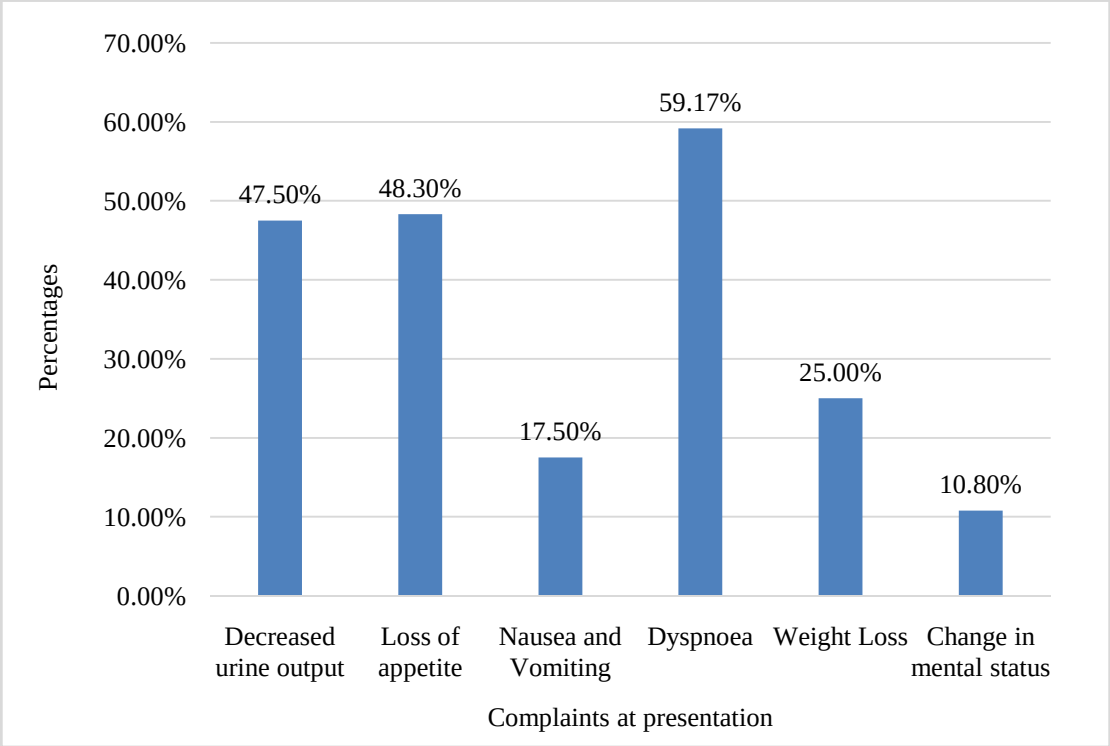


Table 7: Descriptive analysis of duration of Diabetes in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Diabetic Since How Long (years)	16.15 ± 7.5	15.0	2.0	35.0	14.8	17.5

The mean years of diabetes was 16.15 ± 7.5 in the study population, minimum and maximum was 2.0 and 35.0 in the study population with (95% C. I from 14.8 to 17.5). (Table 7)

Table 8: Descriptive analysis of diabetic medications in the study population (N=120) (Changed the categories)

Diabetic medications	Frequency	Percentages
OHA		
Metformin	32	26.7%
Sulfonylureas	35	29.26%
Thiazolidinediones	3	2.50%
Dpp4 inhibitors	31	25.83%
Alpha glucosidase inhibitors	2	1.70%
Not taking medication	57	47.50%
Insulin		
Yes	69	57.5%
No	39	32.5%
Not taking now	12	10%
Both Insulin and OHA		
Yes	25	20.83%
No	95	79.17%

Among the study population, 32 (26.7%) were taking metformin, 35 (29.26%) were taking sulfonylureas, 69 (57.5%) took insulin and 25 (20.83%) took both insulin and OHA. (Table 8)

Table 9: Descriptive analysis of 1st degree relatives being diabetic or hypertensive in the study population (N=120)

Are Your 1St Degree Relatives Diabetic or Hypertension	Frequency	Percentages
Yes	56	46.67%
No	53	44.17%
Don't know	11	9.17%

Among the study population, 56 (46.67%) participants' 1st degree relatives were either diabetic or hypertensive. (Table 9 & Figure 13)

Figure 13: Bar chart of are your 1st degree relatives diabetic or hypertension in the study population (N=120)

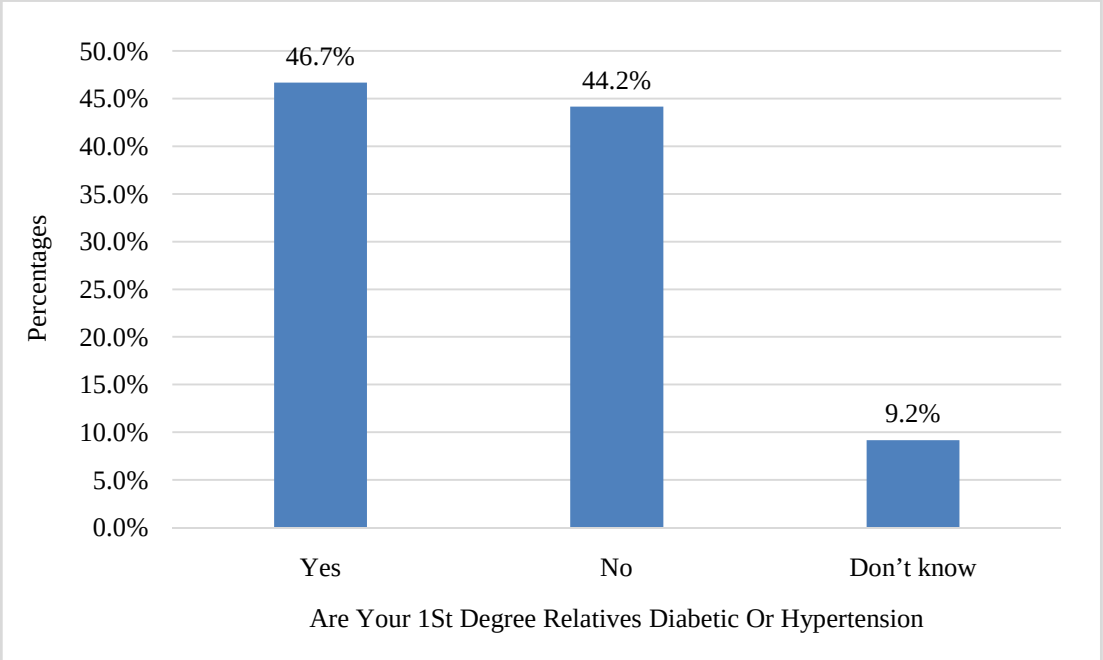


Table 10: Descriptive analysis of duration of hypertension in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Hypertensive Since How Long (years)	14.16 ± 7.35	15.0	2.0	35.0	12.8	15.5

The mean years of hypertension was 14.16 ± 7.35 in the study population, minimum and maximum was 2.0 and 35.0 in the study population with (95% C. I from 12.8 to 15.5). (Table 10)

Table 11: Descriptive analysis of antihypertensive Medications in the study population (N=120)

Antihypertensive Medications	Frequency	Percentages
ACEI/ARBs		
Yes	32	26.67%
No	88	73.33%
Beta Blockers		
Yes	51	42.5%
No	69	57.5%
CCB		
Yes	66	55%
No	54	45%
Nitrates		
Yes	6	5%
No	114	95%
Diuretic		
Yes	21	17.5%
No	99	82.5%%
Others-Prazosin and Clonidine		
Yes	24	20%
No	96	80%

Among the study population, 33 (27.5%) used ACEI/ARBs, 51 (42.5%) used beta blockers, 65 (54.2%) used CCB, 6 (5%) used Nitrates, 24 (20%) used Prazosin and clonidine and 21 (17.5%) were using diuretic. (Table 11 & Figure 14)

Figure 14: Bar chart of Medications in the study population (N=120)

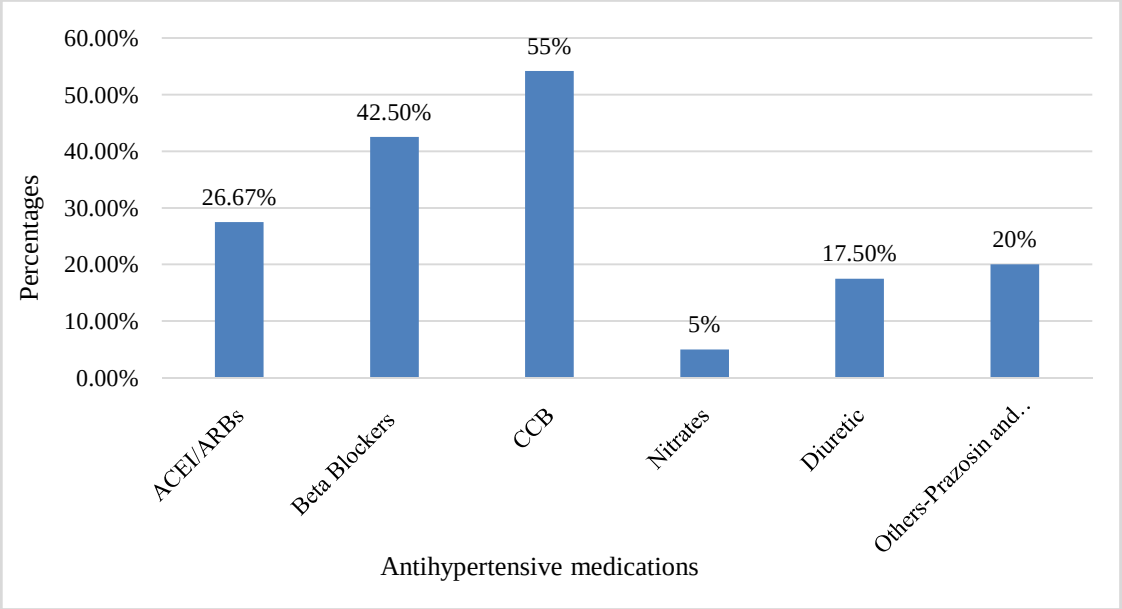


Table 12: Descriptive analysis of previously admitted for any kidney/ urine related problems in the study population (N=120)

Previously Admitted for Any Kidney/ Urine Related Problems	Frequency	Percentages
Yes	47	39.17%
No	73	60.83%

Among the study population, 47 (39.17%) were previously admitted for kidney/ urine relate problems. (Table 12)

Table 13: Descriptive analysis of approximate Average Urine Output In ml in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Approximate AverageUrine Output in ml	602.58 ± 324.4	575.0	0.0	1400.0	544.0	661.2

The mean Urine output (in ml) was 602.58 ± 324.4 in the study population, minimum and maximum was 0 and 1400 in the study population with (95% C. I from 544 to 661.2), (Table 13)

Table 14: Descriptive analysis of use of NSAIDS previously in the study population (N=120)

Use of NSAIDS Previously	Frequency	Percentages
Yes	18	15.00%
No	102	85.00%

Among the study population, 18 (15.00%) participants used NSAIDS previously. (Table 14 & Figure 15)

Figure 15: Bar chart of use of NSAIDS previously in the study population (N=120)

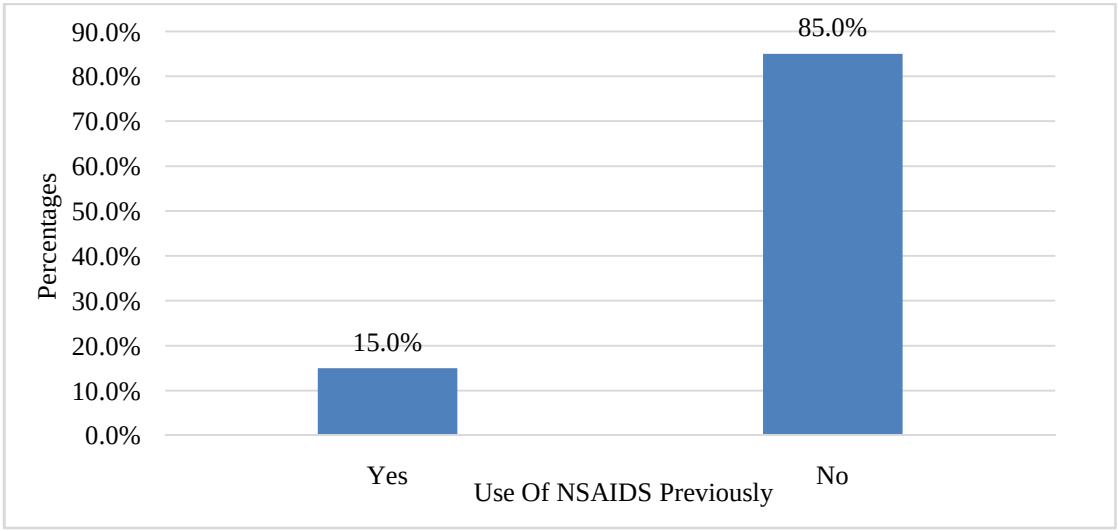


Table 15: Descriptive analysis of history of dialysis in the study population (N=120)

History of Dialysis	Frequency	Percentages
Past		
Yes	39	32.50%
No	81	67.50%
Present		
Yes	80	66.7%
No	40	33.3%

Among the study population, 39 (32.5%) had past history of diabetes and 80 (66.7%) had present history of diabetes. (Table 15 & Figure 16)

Figure 16: Bar chart of History of dialysis in the study population (N=120)

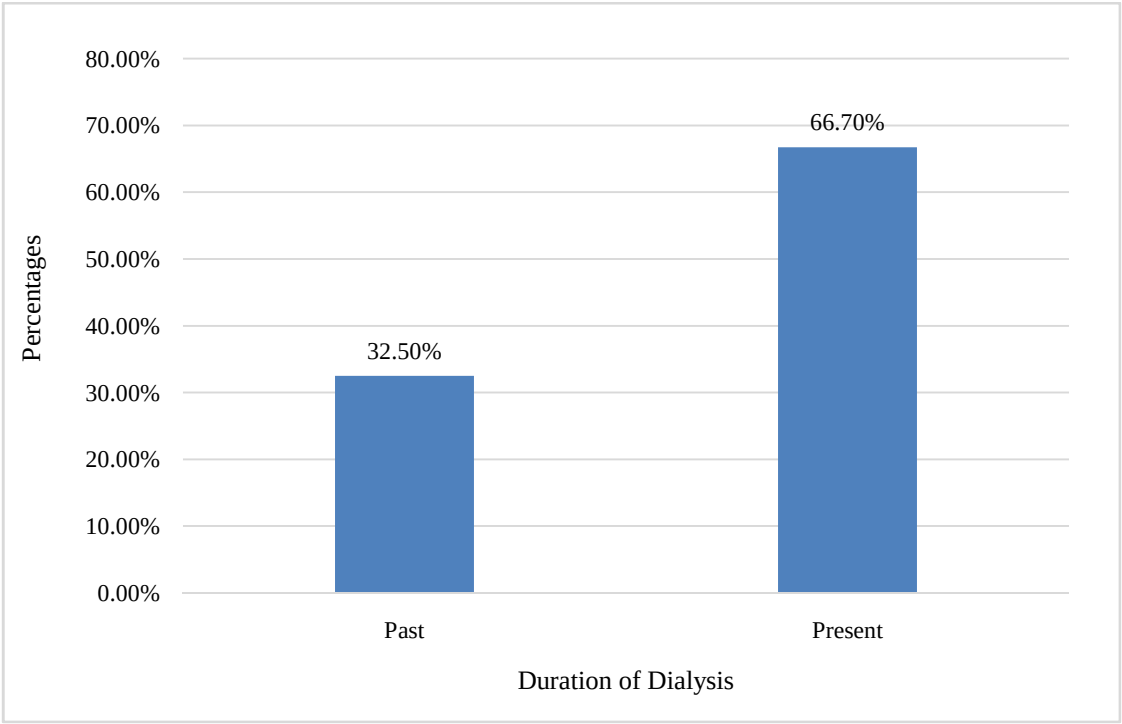


Table 16: Descriptive analysis of duration of dialysis in study population (N=39)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Duration of Dialysis (years)	3.85 ± 2.67	3.00	0.60	12.00	2.99	4.72

The mean duration of dialysis was 3.85 ± 2.67 years in the study population, minimum and maximum was 0.60 and 12.00 in the study population with (95% C. I from 2.99 to 4.72) (Table 16)

Table 17: Descriptive analysis of frequency of dialysis in the study population (N=120)

Frequency of Dialysis	Frequency	Percentages
2 times per week	11	9.17%
3 times per week	38	31.67%
emergency HD	31	25.83%
No	40	33.33%

Among the study population, 11 (9.17%) underwent dialysis 2 times per week, 38 (31.67%) underwent 3 times per week and 31 (25.83%) underwent emergency hemodialysis. (Table 17)

Table 18: Descriptive analysis of awareness of diabetic complications in the study population (N=120)

Analysis of awareness of diabetic complications	Frequency	Percentages
Nephropathy		
Yes	81	67.50%
No	39	32.50%
Retinopathy		
Yes	69	57.5%
No	51	42.5%
Neuropathy		
Yes	42	35%
No	78	65%
IHD		
Yes	107	89.2%
No	13	10.8%
PVD		
Yes	35	29.2%
No	85	70.8%
CVA		
Yes	55	45.8%
No	65	54.2%

Among the study population, 81 (67.50%) knew about Nephropathy, 69 (57.5%) knew about Retinopathy, 42 (35%) knew about Neuropathy, 107 (89.2%) knew about IHD, 35 (29.2%) knew about PVD and 55 (45.8%) knew about CVA. (Table 18 & Figure 17)

Figure 17: Bar chart of analysis of awareness of diabetic complications in the study population (N=120)

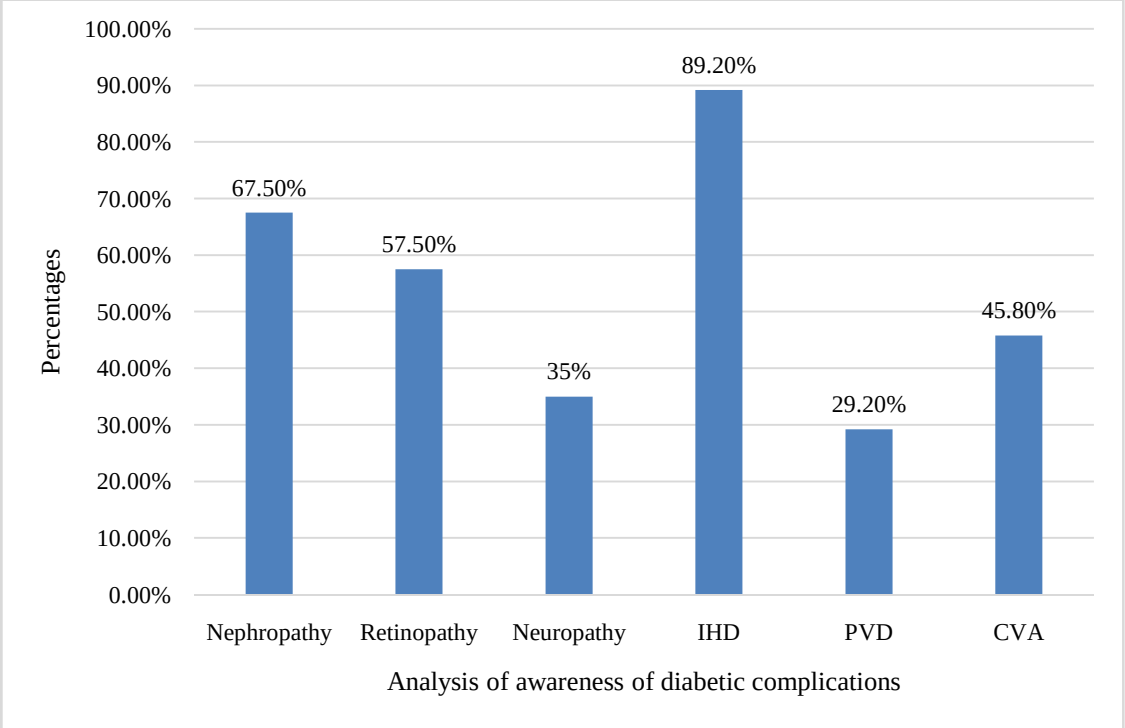


Table 19: Descriptive analysis of social habits in the study population (N=120)

Social habits	Frequency	Percentages
Smoking		
Yes	15	12.50%
No	105	87.50%
Alcohol		
Yes	19	15.8%
No	101	84.2%
Tobacco chewing		
Yes	20	16.7%
No	100	83.3%

Among the study population, 15 (12.50%) were smoking, 19 (15.8%) consumed Alcohol and 20 (16.7%) were chewing tobacco. (Table 19 & Figure 18)

Figure 18: Bar chart analysis of social habits in the study population (N=120)

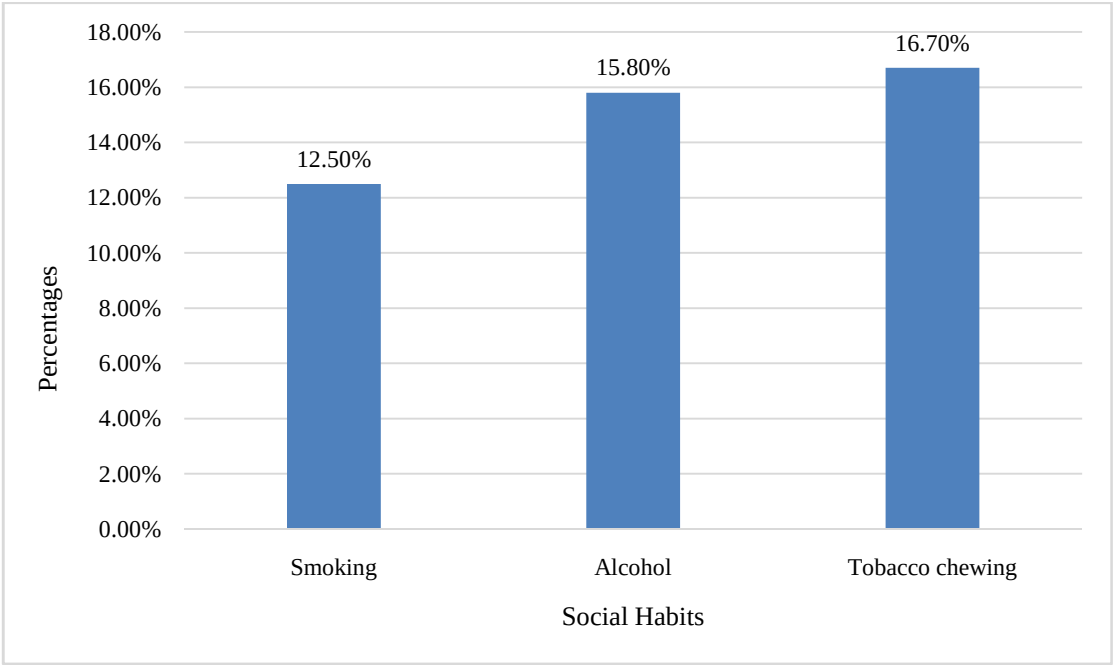


Table 20: Descriptive analysis of physical examination in the study population (N=120)

Physical examination	Frequency	Percentages
Pallor		
Yes	72	60%
No	48	40%
Edema		
Yes	89	74.2%
No	31	25.8%
Muscle wasting		
Yes	38	31.7%
No	82	68.3%
Altered mental status		
Yes	11	9.2%
No	109	90.8%

Among the study population, 72 (60%) had Pallor, 89 (74.2%) had Edema, 38 (31.7%) had muscle wasting and 11 (9.2%) had altered mental status. (Table 20 & Figure 19)

Figure 19: Bar chart analysis of physical examination in the study population (N=120)

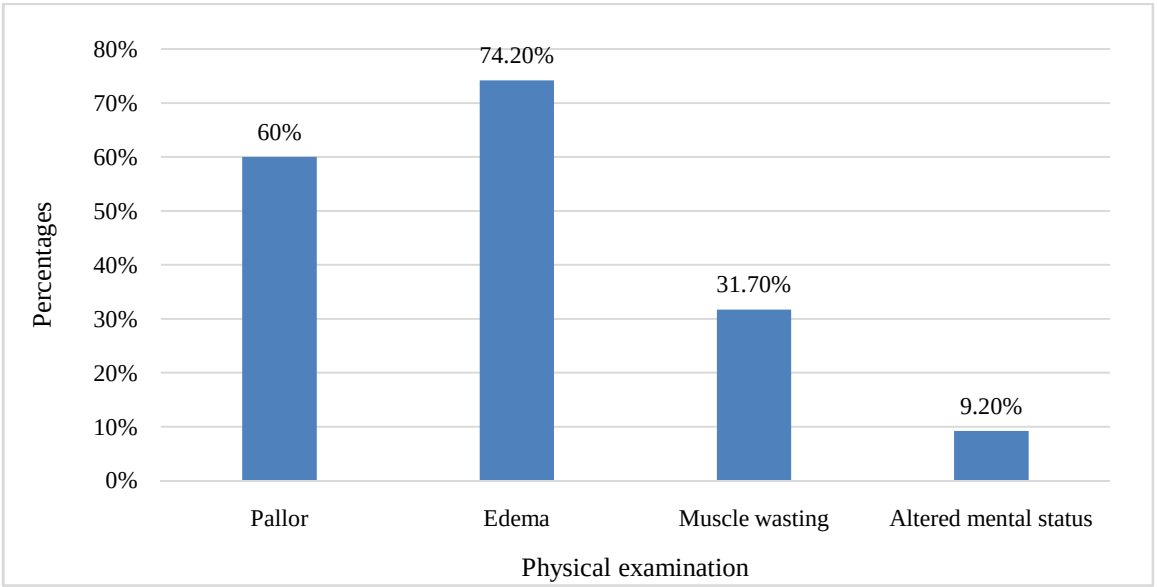


Table 21: Descriptive analysis of vital signs in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Systolic Bp mm Hg	149 ± 28.5	150.00	100.00	220.00	143.85	154.15
Diastolic Bp mm Hg	84 ± 14.05	80.00	60.00	120.00	81.46	86.54

The mean systolic BP was 149 ± 28.5 in the study population, minimum and maximum was 100 and 220 in the study population with (95% C. I from 143.85 to 154.15). The mean Diastolic BP was 84 ± 14.05 in the study population, minimum and maximum was 80 and 120 in the study population with (95% C. I from 81.46 to 86.54). (Table 21)

Table 22: Descriptive analysis of anthropometric in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Height (cm)	168.3 ± 6.73	168.00	154.00	186.00	167.08	169.52
Weight (kg)	70.2 ± 11.33	70.50	48.00	105.00	68.15	72.25
BMI ($kg/m^{2)}$	24.77 ± 3.88	24.40	17.70	42.10	24.07	25.47

The mean Height was 168.3 ± 6.73 (in cm) in the study population, minimum and maximum was 154 and 186 in the study population with (95% C. I from 167.08 to 169.52). The mean Weight was 70.2 ± 11.33 (in kg) in the study population, minimum and maximum was 48 and 105 in the study population with (95% C. I from 68.15 to 72.25). The mean BMI was 24.77 ± 3.88 in the study population, minimum and maximum was 17.70 and 42.10 in the study population with (95% C. I from 24.07 to 25.47).(Table 22)

Table 23: Descriptive analysis of respiratory system examination in the study population (N=120)

Respiratory System Examination	Frequency	Percentages
Normal breath sounds	67	55.83%
Bilateral crepitations	53	44.17%

Among the study population, 67 (55.83%) had normal breath sounds and 53 (44.17%) Had bilateral crepitations in lungs. (Table 23 & Figure 20)

Figure 20: Bar chart of respiratory system examination in the study population (N=120)

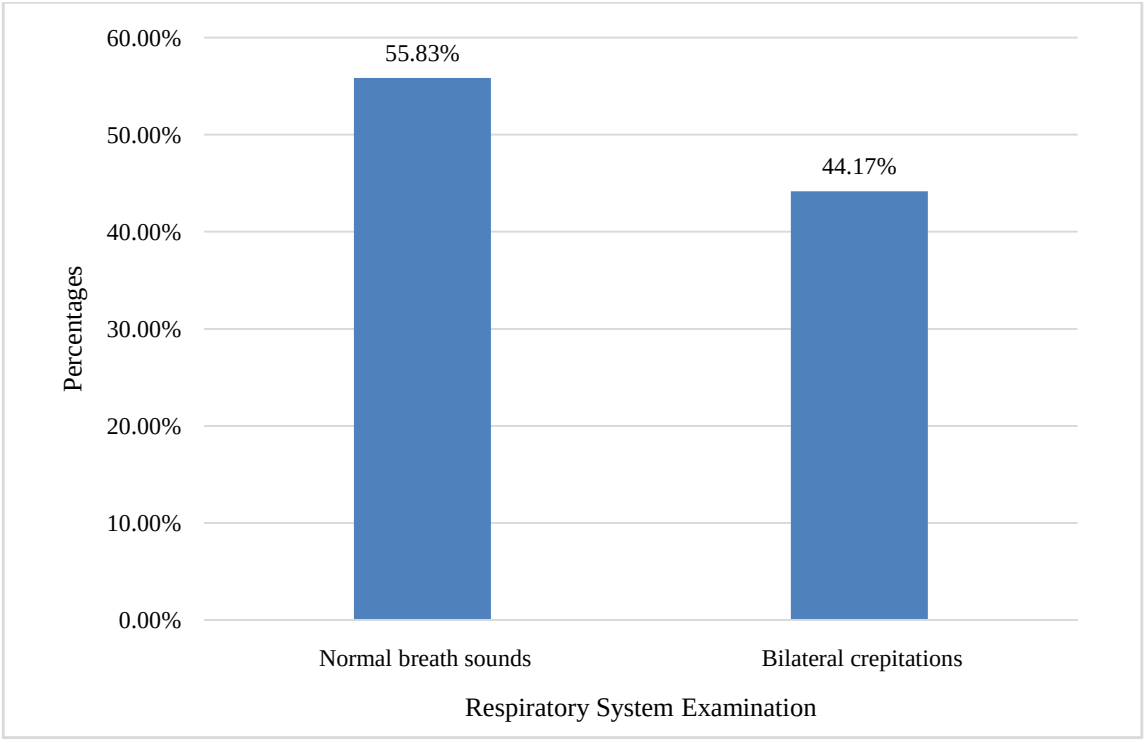


Table 24: Descriptive analysis of lab investigations in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C.I	
					Lower	Upper
Haemoglobin (g/dl)	10.43 ± 1.6	10.25	7.40	14.10	10.14	10.72
FBS (mg/dl)	193.9 ± 86.97	180.50	70.00	542.00	178.18	209.62
PPBS (mg/dl)	219.21 ± 86.77	196.00	79.00	511.00	203.52	234.89
Hba1C	8.11 ± 2.13	7.60	5.20	15.60	7.73	8.50
Creatinine (mg/dl)	6.11 ± 3.52	5.75	1.20	16.36	5.47	6.74
Urea (mg/dl)	90.59 ± 42.66	92.00	27.00	209.00	82.88	98.30
Sodium (meq/L)	135.49 ± 5.32	136.00	117.00	148.00	134.53	136.45
Potassium(meq/L)	4.99 ± 0.79	4.95	3.35	7.20	4.84	5.13
Calcium (mg/dl)	8.52 ± 0.94	8.40	6.20	12.20	8.35	8.69
Phosphorous (mg/dl)	4.71 ± 1.71	4.25	1.70	10.10	4.40	5.02

The mean Haemoglobin was 10.43 ± 1.6 in the study population, minimum and maximum was 7.40 and 14.10 in the study population with (95% C. I from 10.14 to 10.72). The mean FBS was 193.9 ± 86.97 in the study population, minimum and maximum was 70 and 542.00 in the study population with (95% C. I from 203.52 to 234.89). The mean PPBS was 219.21 ± 86.77 in the study population, minimum and maximum was 79 and 511 in the study population with (95% C. I from 10.14 to 10.72). The mean Hba1C was 8.11 ± 2.13 in the study population, minimum and maximum was 5.20 and 15.60 in the study population with (95% C. I from 7.73 to 8.50). The mean Creatinine was 6.11 ± 3.52 in the study population, minimum and maximum was 1.20 and 16.36 in the study population with (95% C. I from 5.47 to 6.74). The mean Urea was 90.59 ± 42.66 in the study population, minimum and maximum was 27.00 and 209.00 in the study population with (95% C. I from 82.88 to 98.30). The mean Sodium was 135.49 ± 5.32 in the study population, minimum and maximum was 117.00 and 148.00 in the study population with (95% C. I from 134.53 to 136.45). The mean Potassium was 4.99 ± 0.79 in the study population, minimum and maximum was 3.35 and 7.20 in the study population with (95% C. I from 4.84 to 5.13). The mean Calcium was 8.52 ± 0.94 in the study population, minimum and

maximum was 6.20 and 12.20 in the study population with (95% C. I from 8.35 to 8.69). The mean Phosphorous was 4.71 ± 1.71 in the study population, minimum and maximum was 1.70 and 10.10 in the study population with (95% C. I from 4.40 to 5.02) (Table 24)

Table 25 (a): Descriptive analysis of serum electrolytes in the study population (N=120)

Parameters	Frequency	Percentages
Sodium (meq/L)		
<135	46	38.33%
135-145	73	60.83%
>145	1	0.83%
Potassium (meq/L)		
<3.5	3	2.50%
3.5-5.5	86	71.67%
>5.5	31	25.83%
Calcium (mg/dl)		
<9	85	70.83%
9-11	34	28.33%
>11	1	0.83%
Phosphorous (mg/dl)		
<2.5	7	5.83%
2.5-4.5	59	49.17%
>4.5	54	45.00%

Among the sodium range in the study population, 46 (38.33%) had <135, 73 (60.83%) had 135-145 and 1 (0.83%) had >145. Among the potassium range in the study population, 3 (2.5%) had <3.5, 86 (71.67%) had 3.5-5.5 and 31 (25.83%) had >5.5. Among the calcium range in the study population, 85 (70.83%) had <9, 34 (28.33%) had 9-11 and 1 (0.83%) had >11. Among the phosphorous range in the study population, 7 (5.83%) had <2.5, 59 (49.17%) had 2.5-4.5 and 54 (45%) had >4.5 (Table 25a).

Table 26: Descriptive analysis of urine protein (spot urine) in the study population (N=120)

Urine protein (Spot Urine)	Frequency	Percentages
1+	30	25.00%
2+	38	31.67%
3+	28	23.33%
4+	7	5.83%
Traces	6	5.00%
Negative	2	1.67%
No urine output	9	7.50%

Among the study population of urine protein, 30 (25%) had 1+, 38 (31.67%) had 2+, 28 (23.33%) had 3+, 7 (5.83%) had 4+, 6 (5%) had traces, 2 (1.67%) were negative and 9 (7.50%) had no urine output. (Table 26)

Table 27: Descriptive analysis of urine glucose (spot urine) in the study population (N=120)

Urine Glucose (spot urine)	Frequency	Percentages
1+	27	22.50%
2+	24	20.00%
3+	15	12.50%
4+	14	11.67%
Traces	12	10.00%
Negative	19	15.83%
No urine output	9	7.50%

Among the study population of urine glucose, 27 (22.50%) had 1+, 24 (20%) had 2+, 15 (12.50%) had 3+, 14 (11.67%) had 4+, 12 (10%) had traces, 19 (15.83%) were negative and 9 (7.50%) had no urine output. (Table 27)

Table 28: Descriptive analysis of EGFR in the study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
MDRD (ml/Min)	15.28 ± 13.27	9.5	2.8	65.0	12.9	17.7
CKD Epi (ml/Min)	14.41 ± 13.08	9.0	3.0	68.0	12.1	16.8

The mean MDRD (ml/Min) was 15.28 ± 13.27 in the study population, minimum and maximum was 2.8 and 65.0 in the study population with (95% C. I from 12.9 to 17.7). The mean CKD EPI (ml/Min) was 14.41 ± 13.08 in the study population, minimum and maximum was 3.0 and 68.0 in the study population with (95% C. I from 12.1 to 16.8) (Table 28)

Table 29: Descriptive analysis of urine albumin creatinine ratio (spot urine) in the study population (N=111)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
Urine albumin creatinine ratio (spot urine)	2967.69 ± 4185.35	1485.3	15.0	31454.0	2180.4	3755.0

*Note: 9 patients did not have any urine output

The mean Urine albumin creatinine ratio was 2967.69 ± 4185.35 in the study population, minimum and maximum was 15.0 and 31454.0 in the study population with (95% C. I from 2180.4 to 3755.0) (Table 29)

Table 30: Descriptive analysis of proteinuria in the study population (N=120)

Proteinuria	Frequency	Percentages
Normal		
Yes	1	0.83%
No	110	91.67%
No Urine	9	7.50%
microalbuminuria		
Yes	22	18.33%
No	89	74.17%
No Urine	9	7.50%
macroalbuminuria		
Yes	85	70.83%
No	26	21.67%
No Urine	9	7.50%

Among the study population, 1 (9.83%) were normal, 22 (18.33%) had micro albuminuriaand 85 (70.83%) had macroalbuminuria. (Table 30 & Figure 21)

Figure 21: Bar chart analysis of proteinuria in the study population (N=120)

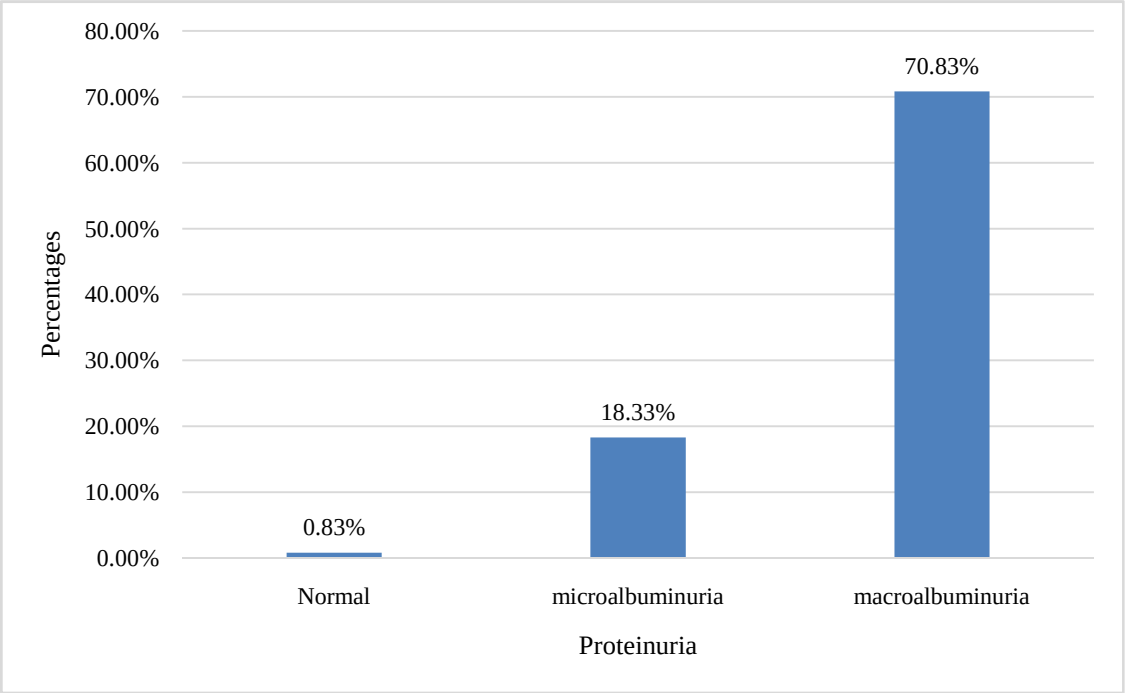


Table 31: Descriptive analysis of USG KUB parenchymal changes in the study population (N=120)

USG KUB parenchymal changes	Frequency	Percentages
Normal	34	28.33%
Grade 1 RPC	45	37.50%
Grade 2 RPC	26	21.67%
Grade 3 RPC	15	12.50%

Among the study population, 34 (28.33%) were normal, 45 (37.50%) were under Grade 1 RPC, 26 (21.67%) were under Grade 2 RPC and 15 (12.50%) were under grade 3 RPC. (Table 31 & Figure 22)

Figure 22: Bar chart of USG KUB parenchymal changes in the study population (N=120)

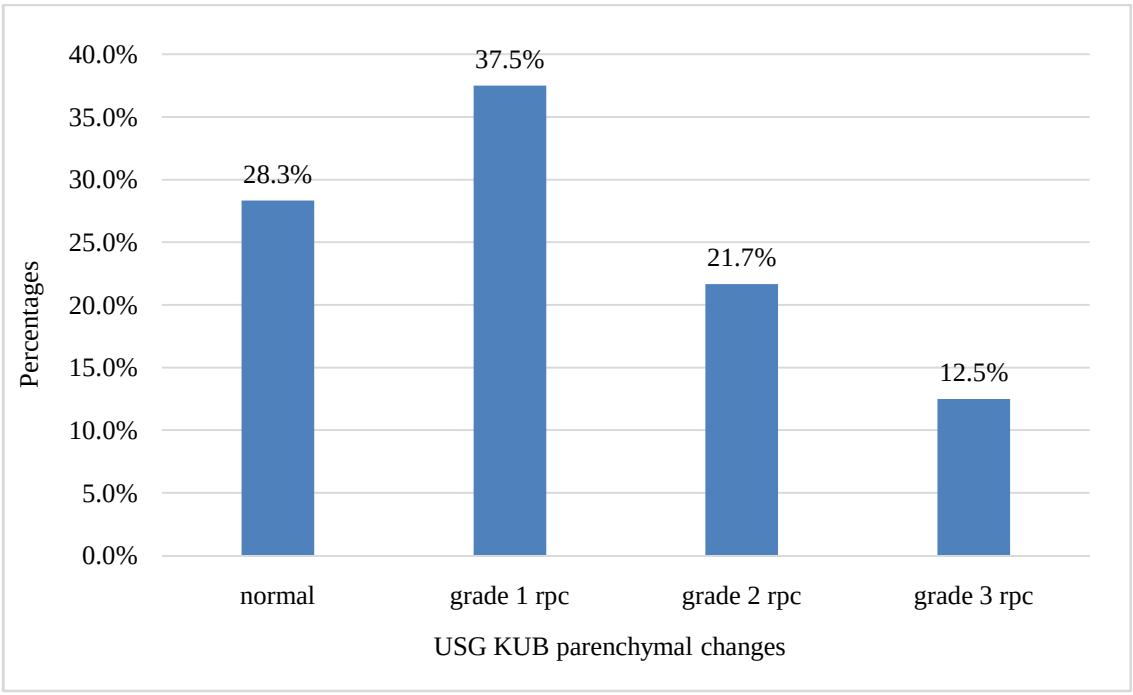


Table 32: Descriptive analysis of diabetic retinopathy in the study population (N=120)

Diabetic retinopathy		Frequency	Percentages
Mild NPDR			
Yes		37	30.8%
No		83	69.2%
Moderate NPDR			
Yes		24	20%
No		96	80%
Severe NPDR			
Yes		11	9.2%
No	109	90.8%	
PDR			
Yes	4	3.3%	
No	116	96.7%	

Among the study population, 37 (30.8%) had Mild NPDR, 24 (20%) had Moderate NPDR, 11 (9.2%) had severe NPDR and 4 (3.3%) had PDR. (Table 32 & Figure 23)

Figure 23: Bar chart of diabetic retinopathy in the study population (N=120)

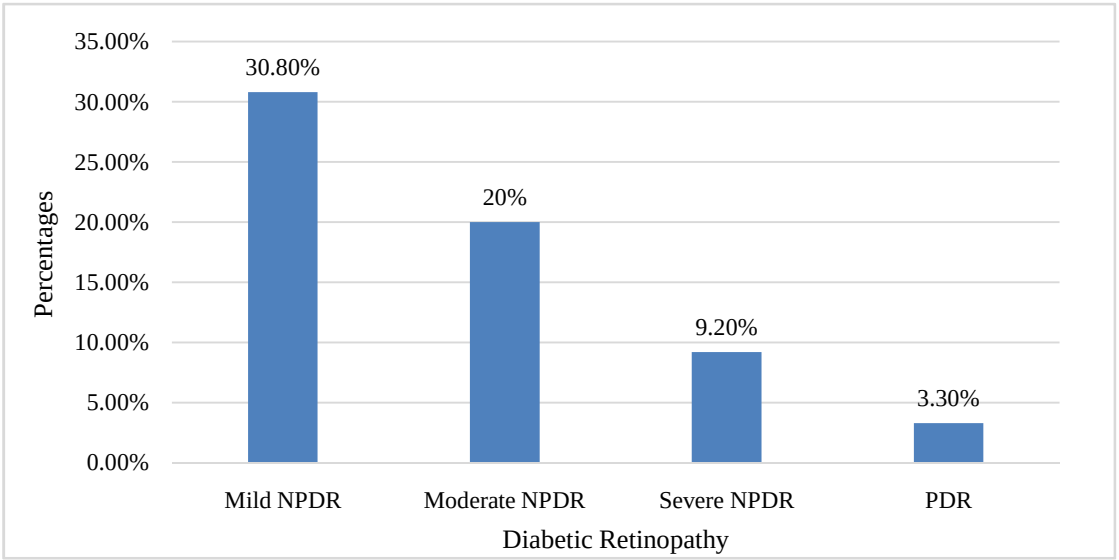


Table 33: Descriptive analysis of hypertensive retinopathy in the study population (N=120)

Hypertensive retinopathy	Frequency	Percentages
Grade 1	4	3.33%
Grade 2	5	4.17%
Grade 3	1	0.83%
No	110	91.67%

Among the study population, 4 (3.33%) were under grade 1, 5 (4.17%) were under grade 2 and 1 (0.83%) were under grade 3. (Table 33 & Figure 24)

Figure 24: Bar chart of hypertensive retinopathy in the study population (N=120)

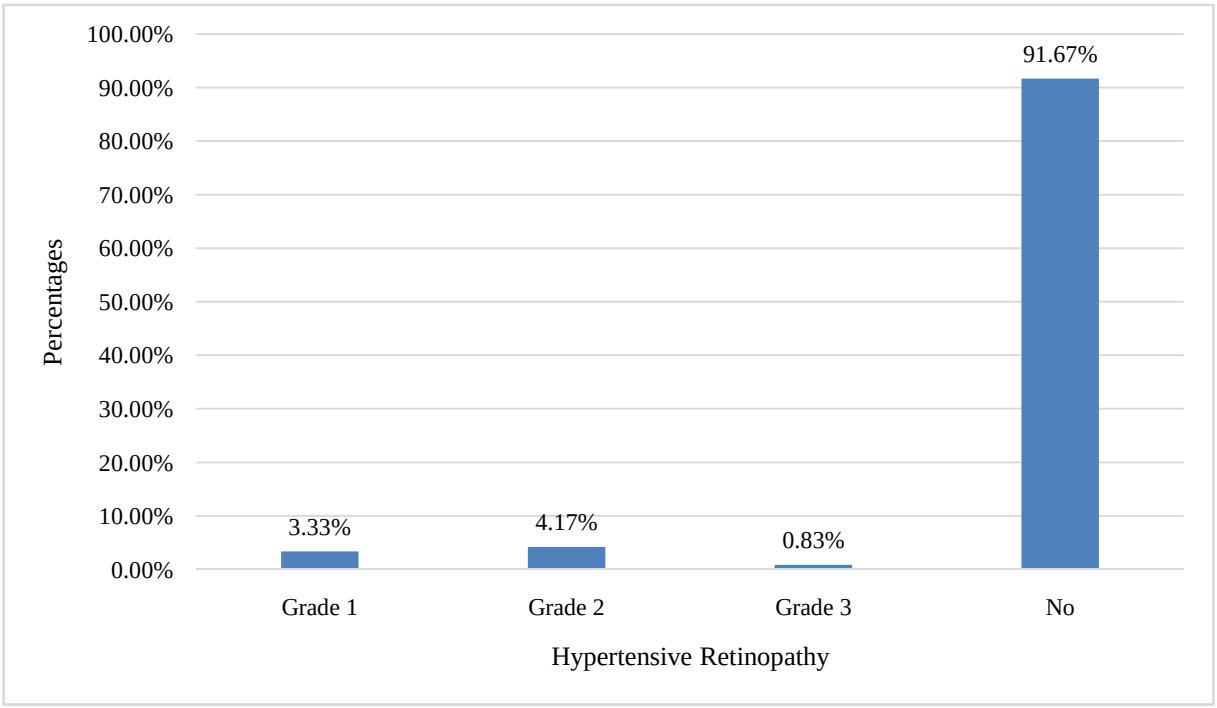


Table 34: Descriptive analysis of ECG Findings in the study population (N=120)

ECG Findings	Frequency	Percentages
Normal		
Yes	115	95.8%
No	5	4.2%
Abnormal		
Low voltage	1	0.8%
LVH	4	3.4%
No	115	95.8%

Among the study population, 115 (95.8%) were normal, and in the abnormal 1 (0.8%) had low voltage, and 4 (3.4%) had LVH changes. (Table 34)

Table 35: Descriptive analysis of combined anti hypertensives in the study population (N=120)

Combined Anti Hypertensives	Frequency	Percentages
Ace inhibitors or arbs	32	26.67%
Beta blockers	51	42.50%
Calcium channel blocker	66	55.00%
Nitrates	6	5.00%
Diuretics	22	18.33%
Others	25	20.83%
Not taking medication	3	2.50%

Among the study population the frequency of combined anti hypertensives were , 32 usedAce inhibitors or arbs, 51 used beta blockers, 66usedCalcium channel blocker, 6 used nitrates, 22 used diuretics, and 25used other medications. (Table 35)

Table 36: Descriptive analysis of hypertensive medications in the study population (N=120)

Hypertensive Medications	Frequency	Percentages
Yes	117	97.50%
No	3	2.50%

Among the study population, 117 (97.50%) had hypertensive medications. (Table 36 & Figure 25)

Figure 25: Bar chart of hypertensive medications in the study population (N=120)

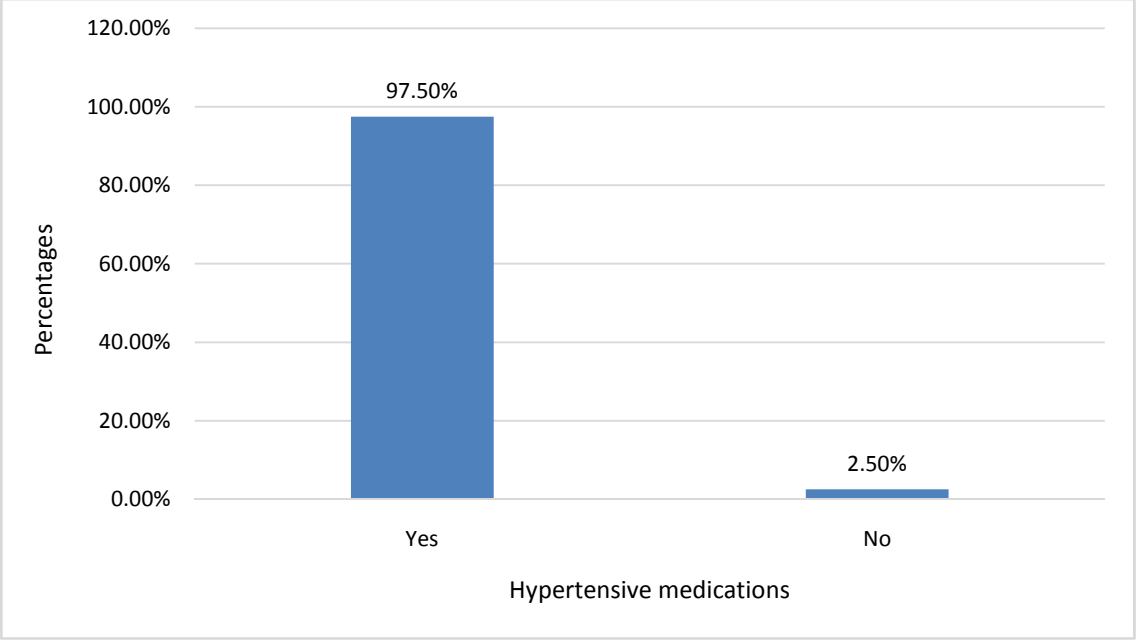


Table 37: Correlation between Number of years of diabetes and EGFR parameters in the study population (N=120)

Parameter	Pearson correlation (r)	P value
MDRD (ml/min)	-0.084	0.361
CKD EPI (ml/min)	-0.104	0.257

There was a weak negative correlation between MDRD (ml/min) and Number of years of diabetes (r value: -0.084, P value: 0.361). There was a weak negative correlation between CKD EPI (ml/min) and Number of years of diabetes (r value: -0.104, P value: 0.257) (Table 37 & Figure 26, 27)

Figure 26: Scatter plot between Number of years of diabetes and MDRD (ml/min) in the study population (N=120)

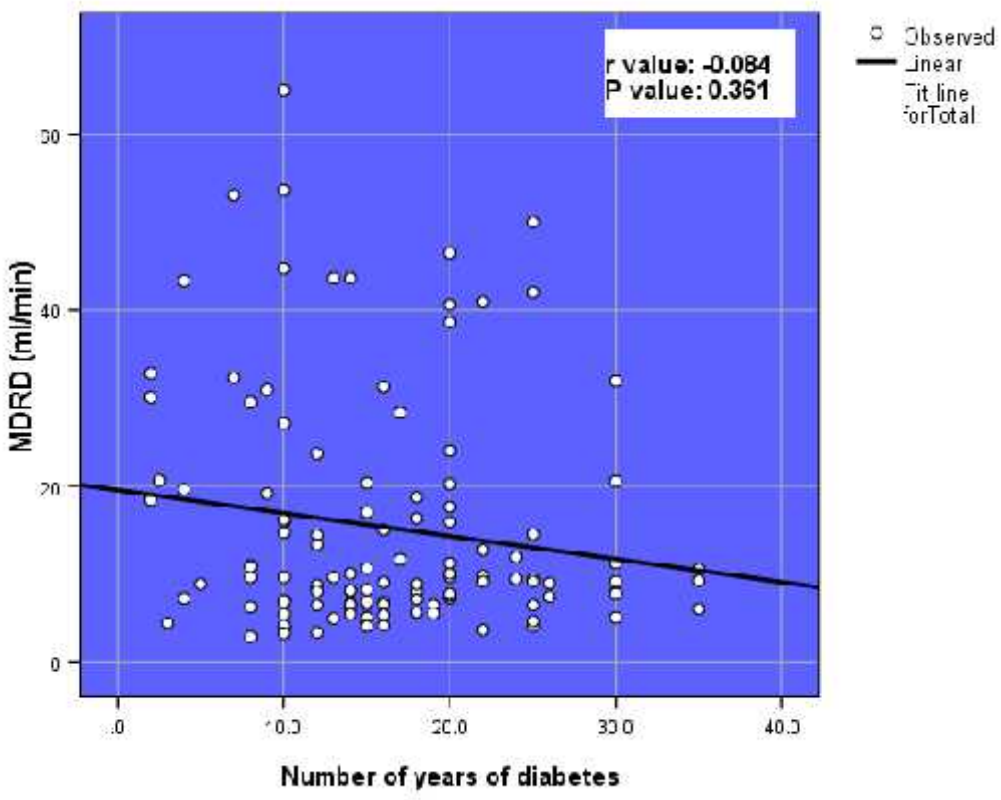


Figure 27: Scatter plot between Number of years of diabetes and CKD EPI (ml/min) in the study population (N=120)

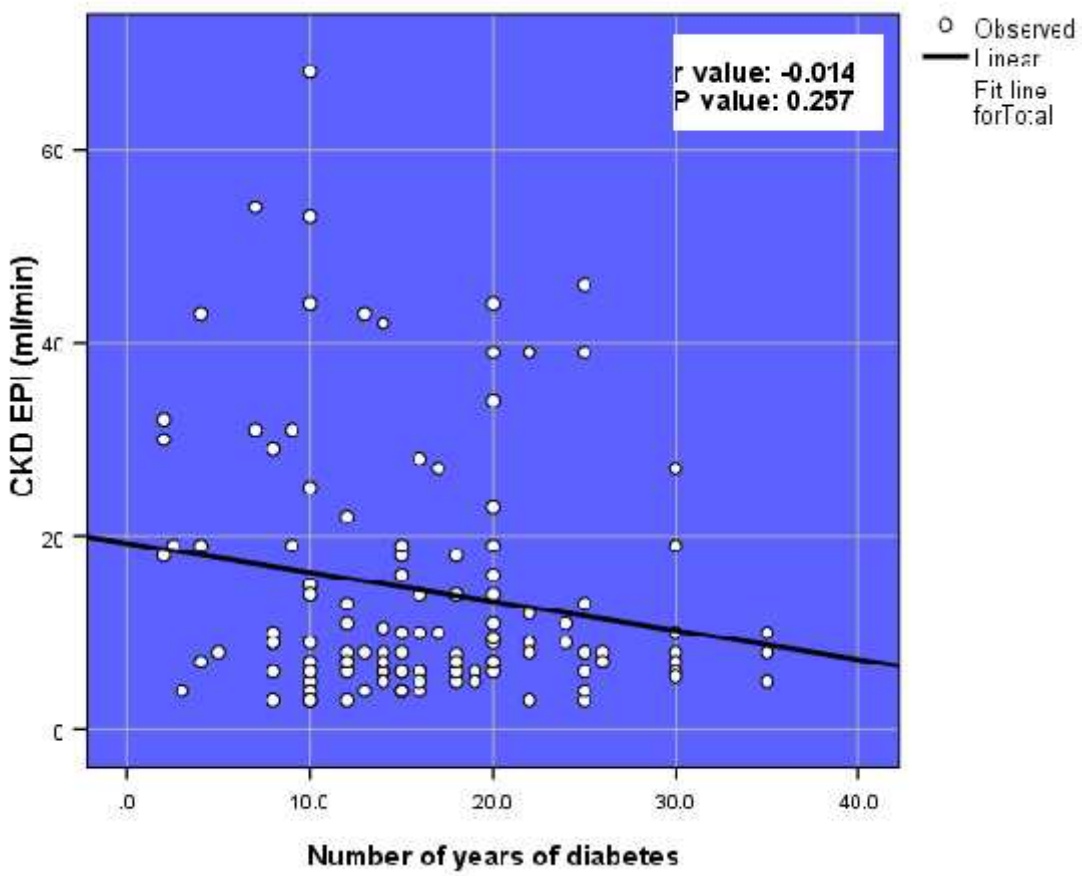


Table 38: Comparison of Urine albumin creatinine ratio and history of dialysis past & Present (N=111)

Parameter	Urine albumin creatinine ratio	Mann Whitney U test (P value)
History of Dialysis (Past)		
Yes	1247.65 (545.33,2328.75)	0.318
No	1712.7 (491.1,3726)	
History of Dialysis (Present)		
Yes	1755 (683,3385.2)	0.322
No	1392.15 (246.25,3576.98)	

The median Urine albumin creatinine ratio was 1247.65 (IQR 545.33,2328.75) in past history of dialysis and it was 1755 (IQR 683,3385.2) in the present history of dialysis. The difference between Urine albumin creatinine ratio and history of dialysis was not statistically significant. (P value>0.05) (Table 38)

Table 39: Descriptive analysis of GFR in study population (N=120)

Parameter	Mean ± SD	Median	Minimum	Maximum	95% C. I	
					Lower	Upper
GFR	14.85 ± 13.17	9.25	2.90	66.50	12.47	17.23

The mean GFR was 14.85 ± 13.17 in the study population, minimum and maximum was 2.0 and 66.50 in the study population with (95% C. I from 12.47 to 17.23) (Table 39)

Table 40: Descriptive analysis of GFR in the study population (N=120)

GFR	Frequency	Percentages
<=60	119	99.17%
>60	1	0.83%

Among the GFR in study population, 119 (99.17%) were <=60 and 1 (0.83%) were >60. (Table 40 & Figure 28)

Figure 28: Bar chart of GFR in the study population (N=120)

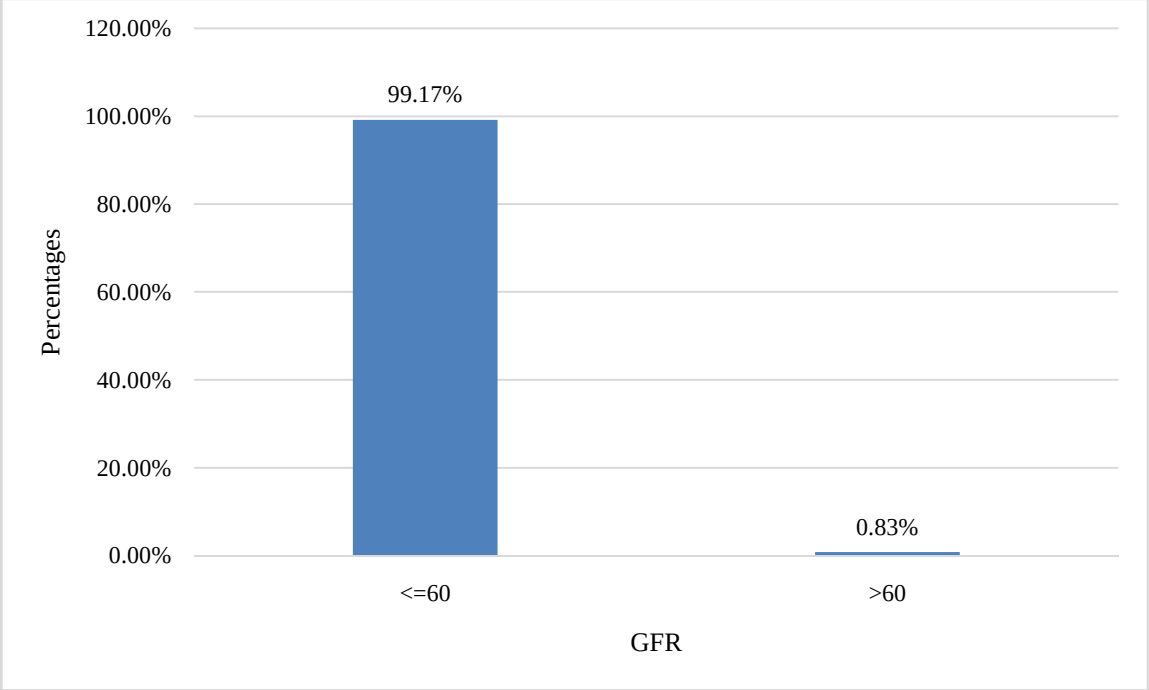


Table 41: Comparison of demographic and laboratory parameters between GFR (N=120)

Parameters	GFR	
	<=60 (N=119)	**>60 (N=1)**
Age (Mean± SD)	63.84 ±10.63	0 ± 0
Age group (Male) (N=87)		
40-49	11 (12.79%)	1 (100%)
50-59	17 (19.77%)	0 (0%)
60-69	29 (33.72%)	0 (0%)
70-79	23 (26.74%)	0 (0%)
80-89	6 (6.98%)	0 (0%)
Age group (Female) (N=33)		
40-49	2 (6.06%)	0 (0%)
50-59	9 (27.27%)	0 (0%)
60-69	11 (33.33%)	0 (0%)
70-79	8 (24.24%)	0 (0%)
80-89	3 (9.09%)	0 (0%)
UACR		
30-299	21 (19.44%)	1 (100%)
>=300	87 (80.56%)	0 (0%)
BMI		
<24.9	62 (52.1%)	0 (0%)
25-29.9	46 (38.66%)	1 (100%)
>=30	11 (9.24%)	0 (0%)
DM Duration (Mean± SD)	16.20±7.51	0±0
Hba1C		
<6.49	27 (22.69%)	0 (0%)
6.5-7.49	25 (21.01%)	0 (0%)
7.5-8.49	28 (23.53%)	0 (0%)
8.5-9.49	14 (11.76%)	1 (100%)
>=9.5	25 (21.01%)	0 (0%)
FBS		
<126	29 (24.37%)	0 (0%)
>=126	90 (75.63%)	1 (100%)
PPBS		
<200	63 (52.94%)	0 (0%)
>=200	56 (47.06%)	1 (100%)
DBP		
<80	36 (30.25%)	0 (0%)
>=80	83 (69.75%)	1 (100%)
SBP		

<130	22 (18.49%)	0 (0%)
>=130	97 (81.51%)	1 (100%)
Ace-I/Arbs		
Yes	32 (26.89%)	1 (100%)
No	87 (73.11%)	0 (0%)
B Blocker		
Yes	51 (42.86%)	0 (0%)
No	68 (57.14%)	1 (100%)
Insulin		
Yes	69 (57.98%)	0 (0%)
No	38 (31.93%)	1 (100%)
Not Taking Now	12 (10.08%)	0 (0%)
Diuretic		
Yes	21 (17.65%)	0 (0%)
No	98 (82.35%)	1 (100%)
Nsaids		
Yes	18 (15.13%)	0 (0%)
No	101 (84.87%)	1 (100%)
Hypertensive Retinopathy		
Grade 1	4 (3.36%)	0 (0%)
Grade 2	5 (4.2%)	0 (0%)
Grade 3	1 (0.84%)	0 (0%)
No	109 (91.6%)	1 (100%)
Mild NPDR		
Yes	37 (31.09%)	0 (0%)
No	82 (68.91%)	1 (100%)
Moderate NPDR		
Yes	24 (20.17%)	0 (0%)
No	95 (79.83%)	1 (100%)
Severe NPDR		
Yes	11 (9.24%)	0 (0%)
No	108 (90.76%)	1 (100%)
PDR		
Yes	4 (3.36%)	0 (0%)
No	115 (96.64%)	1 (100%)
Urine output (ml) (Mean± SD)	600.08±324.6	0±0

Among the GFR <=60, the mean age was 63.84 ±10.63, 11 (12.79%) male participants were aged between 40t o 49 years, 17 (19.77%) male participants were aged between 50 to 59 years, 29 (33.72%) male participants were aged between 70 to 79 years, 6 (6.98%) male participants were aged between 80 to 89 years, 2 (6.06%)

female participants were aged between 40 to 49 years, 9 (27.27%) female participants were aged between 50 to 59 years, 11 (33.33%) female participants were aged between 70 to 79 years, 3 (9.09%) female participants were aged between 80 to 89 years and so on. Among the GFR >60, the mean age was 0 ± 0, 1 (100%) male participants were aged between 40 to 49 years, 1 (100%) participants were under UACR and so on. (Table 41)

Table 42: Comparison of EGFR across albuminuria (N=111)

EGFR	Albuminuria		
	A1 (N=2)<30	A2 (N=22)30-299	A3 (N=87)>=300
60-89	0 (0%)	1 (4.55%)	0 (0%)
45-59	0 (0%)	1 (4.55%)	4 (4.6%)
30-44	1 (50%)	3 (13.64%)	8 (9.2%)
15-29	0 (0%)	9 (40.91%)	12 (13.79%)
<=15	1 (50%)	8 (36.36%)	63 (72.41%)

*No statistical test was applied- due to 0 subjects in the cells

Among A1 Albuminuria, 1 (50%) were in the range 30-44 in EGFR and 1 (50%) were <=15 in EGFR, among A2 Albuminuria, 1 (4.45%) were in the range 60-89 & 45-59 in EGFR, 3 (13.64%) were in the range 30-44 in EGFR, 9 (40.91%) were in the range 15-29 in EGFR, 8 (36.36%) Were in <=15 in EGFR, among A3 Albuminuria, 4 (4.6%) were in the range 45-59 in EGFR, 8 (9.2%) were in the range 30-44 in EGFR, 12 (13.79%) were in the range 15-29 in EGFR and 63 (72.41%) were in the range <=15 in EGFR. (Table 42 & Figure 29)

CKD Classification and Staging

- Green: Low risk (LR)
- Yellow: Moderate risk (MR)
- Orange: High risk (HR)
- Red: Very high risk (VHR)

Kidney function stage GFR (mL/min/1.73m²) Description and range				Kidney damage stage Urine albumin/creatinine ratio Description and range		
				A1	A2	A3
				Normal to mild increase <30mg/g	Moderate increase 30-300 mg/g	Severe increase >300mg/g
	G1	Normal or high	≥90	LR	MR	HR
	G2	Mild decrease	60-89	LR	MR	HR
	G3a	Mild to moderate decrease	45-59	MR	HR	VHR
	G3b	Moderate to severe decrease	30-44	HR	VHR	VHR
	G4	Severe decrease	15-29	VHR	VHR	VHR
	G5	Kidney failure	<15	VHR	VHR	VHR

Figure 29: Staked bar chart of comparison of EGFR between Albuminuria (N=109)

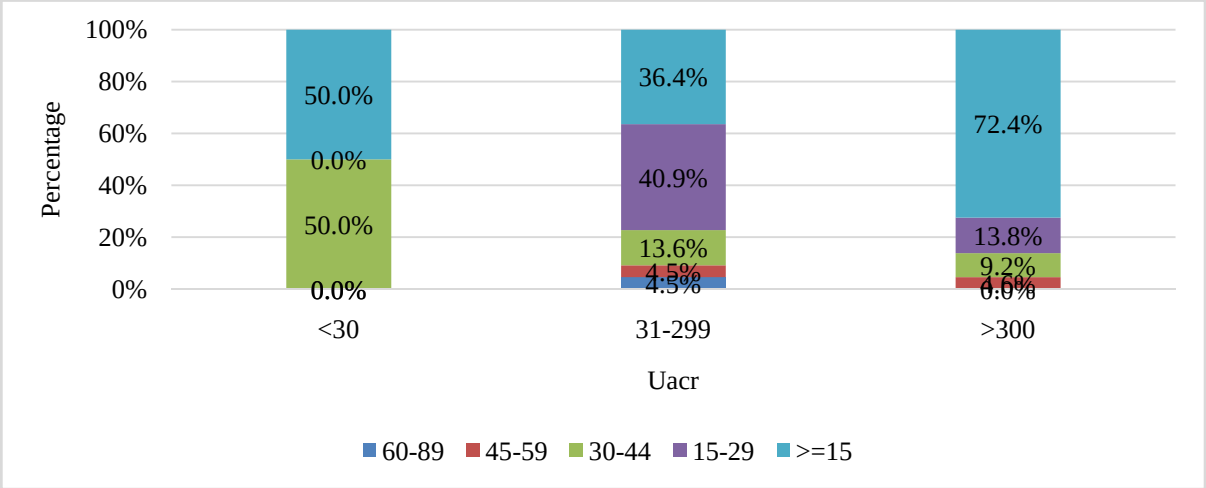


Table 43: Comparison of mean of GFR between gender(N=119)

Parameter	Gender (Mean± SD)		P value
	Male (N=86)	Female (N=33)	
GFR	15.1 ± 12.96	12.61 ± 10.46	0.325

The mean GFR in male was 15.1 ± 12.96 in the study population and it was 12.61 ± 10.46 in female. The difference between GFR and Gender was statistically not significant. (P value > 0.05) (Table 43)

Table 44: Comparison of mean GFR across the study groups (N=119)

Age Group	GFR Mean ± SD	Mean difference	95% CI		P value
			Lower	Upper	
40-49 years	13.21 ± 14.11				
50-59 years	15.41 ± 12.25	2.20	-6.23	10.63	0.606
60-69 years	13.82 ± 11.36	0.60	-7.31	8.52	0.880
70-79 years	14.77 ± 13.85	1.56	-6.63	9.76	0.706
80-89 years	14.67 ± 10.85	1.46	-9.29	12.22	0.788

The mean GFR was 13.21 ± 14.11 in 40 to 49 years age group, it was 15.41 ± 12.25, 13.82 ± 11.36, 14.77 ± 13.85 and 14.67 ± 10.85 in 50 to 59 years, 60 to 69 years, 70 to 79 years and 80 to 89 years age groups respectively. The difference between GFR and age groups was statistically not significant (P value > 0.05). (Table 44)

Table 45: Comparison of mean GFR across the study groups (Male) (N=87)

Age Group (Male)	GFR Mean ± SD	Mean difference	95% CI		P value
			Lower	Upper	
40-49 years	18.74 ± 20.78				
50-59 years	17.06 ± 13.6	1.68	-8.99	12.35	0.755
60-69 years	13.61 ± 11.62	5.13	-4.58	14.84	0.296
70-79 years	15.12 ± 14.1	3.62	-6.46	13.70	0.477
80-89 years	18.06 ± 12.03	0.68	-13.47	14.83	0.924

The mean GFR was 18.74 ± 20.78 in 40 to 49 years age group of males, it was 17.06 ± 13.6, 13.61 ± 11.62, 15.12 ± 14.1 and 18.06 ± 12.03 in 50 to 59 years, 60 to 69 years, 70 to 79 years and 80 to 89 years age groups of males respectively. The difference between GFR and age groups of males was statistically not significant (P value > 0.05). (Table 45)

Table 46: Comparison of mean GFR across the study groups (Female) (N=33)

Age Group (Female)	GFR Mean ± SD	Mean difference	95% CI		P value
			Lower	Upper	
40-49 years	6.7 ± 5.09				
50-59 years	12.3 ± 9.1	5.60	-11.83	23.03	0.516
60-69 years	14.36 ± 11.15	7.66	-9.47	24.80	0.367
70-79 years	13.79 ± 14	7.09	-10.54	24.71	0.417
80-89 years	7.9 ± 2.39	1.20	-19.15	21.55	0.905

The mean GFR was 6.7 ± 5.09 in 40 to 49 age group of females, it was 12.3 ± 9.1, 14.36 ± 11.15, 13.79 ± 14 and 7.9 ± 2.39 in 50 to 59, 60 to 69, 70 to 79 and 80 to 89 age groups of females respectively. The difference between GFR and age groups of females was statistically not significant (P value > 0.05). (Table 46)

Table 47: Comparison of demographic and laboratory parameters between micro albuminuria (N=111)

Parameters	Micro Albuminuria		Chi square	P value
	Yes (N=22)	No (N=89)		
Age Group				
40-49	1 (4.55%)	12 (13.48%)	6.954	0.138
50-59	6 (27.27%)	20 (22.47%)		
60-69	4 (18.18%)	33 (37.08%)		
70-79	8 (36.36%)	20 (22.47%)		
80-89	3 (13.64%)	4 (4.49%)		
BMI				
<24.9	9 (40.91%)	46 (51.69%)	2.538	0.281
25-29.9	12 (54.55%)	33 (37.08%)		
>=30	1 (4.55%)	10 (11.24%)		
FBS				
<126	8 (36.36%)	16 (17.98%)	3.519	0.082
>=126	14 (63.64%)	73 (82.02%)		
PPBS				
<200	14 (63.64%)	43 (48.31%)	1.658	0.198
>=200	8 (36.36%)	46 (51.69%)		
Hba1C				
<6.49	5 (22.73%)	18 (20.22%)	7.866	0.097
6.5-7.49	4 (18.18%)	21 (23.6%)		
7.5-8.49	9 (40.91%)	16 (17.98%)		
8.5-9.49	3 (13.64%)	12 (13.48%)		
>=9.5	1 (4.55%)	22 (24.72%)		
SBP				
<130	9 (40.91%)	12 (13.48%)	8.650	0.006
>=130	13 (59.09%)	77 (86.52%)		
DBP				
<80	8 (36.36%)	26 (29.21%)	0.424	0.515
>=80	14 (63.64%)	63 (70.79%)		
Ace/Arbs				
Yes	8 (36.36%)	24 (26.97%)	0.759	0.384
No	14 (63.64%)	65 (73.03%)		
B Blockers				
Yes	8 (36.36%)	38 (42.7%)	0.292	0.589
No	14 (63.64%)	51 (57.3%)		
Insulin				
Yes	9 (40.91%)	56 (62.92%)	4.629	0.099
No	10 (45.45%)	29 (32.58%)		

Not Taking Now	3 (13.64%)	4 (4.49%)		
Diuretic				
Yes	6 (27.27%)	13 (14.61%)	1.995	0.204
No	16 (72.73%)	76 (85.39%)		
NSAIDS				
Yes	1 (4.55%)	17 (19.1%)	2.751	0.117
No	21 (95.45%)	72 (80.9%)		
Hypertensive Retinopathy				
Grade 1	1 (4.55%)	3 (3.37%)	0.071	0.965
Grade 2	1 (4.55%)	4 (4.49%)		
No	20 (90.91%)	82 (92.13%)		
Mild NPDR				
Yes	6 (27.27%)	30 (33.71%)	0.333	0.564
No	16 (72.73%)	59 (66.29%)		
Moderate NPDR				
Yes	3 (13.64%)	20 (22.47%)	0.838	0.557
No	19 (86.36%)	69 (77.53%)		
UACR				
<30	0 (0%)	2 (2.25%)	*	*
31-299	22 (100%)	0 (0%)		
>300	0 (0%)	87 (97.75%)		
USG Abdomen				
Normal	11 (50%)	22 (24.72%)	5.741	0.125
Grade 1 Rpc	7 (31.82%)	37 (41.57%)		
Grade 2 Rpc	3 (13.64%)	19 (21.35%)		
Grade 3 Rpc	1 (4.55%)	11 (12.36%)		
Urine Output				
Yes	9 (40.91%)	41 (46.07%)	0.190	0.663
No	13 (59.09%)	48 (53.93%)		

*No statistical test was applied- due to 0 subjects in the cells
* 9 participants are no urine patients

Among the micro albuminuria, 1 (4.55%) were aged between 40 to 49 years, 6 (27.27%) were aged between 50 to 59 years, 4 (18.18%) were aged between 60 to 69 years, 8 (36.36%) were aged between 70 to 79 years, 3 (13.64%) were aged between 80 to 89 years, 9 (40.91%) were in <24.9 BMI, 12 (54.55%) were in 25 to 2.99 range of BMI, 1 (4.55%) were in >=30 BMI and so on. The proportion of difference between SBP and Micro Albuminuria was statistically significant. (P value<0.05) (Table 47 & Figure 24)

Table 48: Comparison of demographic and laboratoryparameters between macro albuminuria (N=111)

Parameters	Macro Albuminuria		Chi square	P value
	Yes (N=85)	No (N=26)		
Age Group				
40-49	11 (12.94%)	2 (7.69%)	4.202	0.379
50-59	20 (23.53%)	6 (23.08%)		
60-69	31 (36.47%)	6 (23.08%)		
70-79	19 (22.35%)	9 (34.62%)		
80-89	4 (4.71%)	3 (11.54%)		
BMI				
<24.9	42 (49.41%)	13 (50%)	1.525	0.466
25-29.9	33 (38.82%)	12 (46.15%)		
>=30	10 (11.76%)	1 (3.85%)		
FBS				
<126	14 (16.47%)	10 (38.46%)	5.682	0.017
>=126	71 (83.53%)	16 (61.54%)		
PPBS				
<200	41 (48.24%)	16 (61.54%)	1.410	0.235
>=200	44 (51.76%)	10 (38.46%)		
Hba1C				
<6.49	16 (18.82%)	7 (26.92%)	5.878	0.208
6.5-7.49	20 (23.53%)	5 (19.23%)		
7.5-8.49	16 (18.82%)	9 (34.62%)		
8.5-9.49	12 (14.12%)	3 (11.54%)		
>=9.5	21 (24.71%)	2 (7.69%)		
SBP				
<130	12 (14.12%)	9 (34.62%)	5.453	0.041
>=130	73 (85.88%)	17 (65.38%)		
DBP				
<80	25 (29.41%)	9 (34.62%)	0.254	0.614
>=80	60 (70.59%)	17 (65.38%)		
Ace/Arbs				
Yes	23 (27.06%)	9 (34.62%)	0.554	0.457
No	62 (72.94%)	17 (65.38%)		
B Blockers				
Yes	37 (43.53%)	9 (34.62%)	0.652	0.419
No	48 (56.47%)	17 (65.38%)		
Insulin				
Yes	55 (64.71%)	10 (38.46%)	7.952	0.019
No	27 (31.76%)	12 (46.15%)		

Not Taking Now	3 (3.53%)	4 (15.38%)		
Diuretic				
Yes	13 (15.29%)	6 (23.08%)	0.850	0.379
No	72 (84.71%)	20 (76.92%)		
NSAIDS				
Yes	15 (17.65%)	3 (11.54%)	0.547	0.557
No	70 (82.35%)	23 (88.46%)		
Hypertensive Retinopathy				
Grade 1	3 (3.53%)	1 (3.85%)	0.039	0.981
Grade 2	4 (4.71%)	1 (3.85%)		
No	78 (91.76%)	24 (92.31%)		
Mild NPDR				
Yes	29 (34.12%)	7 (26.92%)	0.470	0.493
No	56 (65.88%)	19 (73.08%)		
Moderate NPDR				
Yes	19 (22.35%)	4 (15.38%)	0.589	0.443
No	66 (77.65%)	22 (84.62%)		
Severe NPDR				
Yes	7 (8.24%)	1 (3.85%)	0.574	0.678
No	78 (91.76%)	25 (96.15%)		
UACR				
<30	0 (0%)	2 (7.69%)	*	*
31-299	0 (0%)	22 (84.62%)		
>300	85 (100%)	2 (7.69%)		
USG Abdomen				
Normal	20 (23.53%)	13 (50%)	7.607	0.055
Grade 1 Rpc	35 (41.18%)	9 (34.62%)		
Grade 2 Rpc	19 (22.35%)	3 (11.54%)		
Grade 3 Rpc	11 (12.94%)	1 (3.85%)		
Urine Output				
Yes	39 (45.88%)	11 (42.31%)	0.103	0.749
No	46 (54.12%)	15 (57.69%)		

*No statistical test was applied- due to 0 subjects in the cells
* 9 participants are no urine patients

Among the macro albuminuria, 11 (12.94%) were aged between 40 to 49 years, 20 (23.53%) were aged between 50 to 59 years, 31 (36.47%) were aged between 60 to 69 years, 19 (22.35%) were aged between 70 to 79 years, 4 (4.71%) were aged between 80 to 89 years, 42 (49.41%) were in <24.9 BMI, 33 (38.82%) were in 25 to 2.99 range of BMI, 10 (11.76%) were in >=30 BMI and so on. The proportion of difference between SBP with Macro Albuminuria was statistically significant. The proportion of

difference between insulin administration and macro albuminuria was statistically significant. (P value<0.05) (Table 48 & Figure 42)

Table 49: Comparison of demographic and laboratory parameters across EGFR (N=120)

Parameters	EGFR				
	60-89 (N=1)	45-59 (N=5)	30-44 (N=12)	15-29 (N=23)	<=15 (N=79)
Age Group (Male) (N=87)					
40-49	1 (100%)	1 (25%)	1 (10%)	1 (5.88%)	8 (14.55%)
50-59	0 (0%)	1 (25%)	2 (20%)	6 (35.29%)	8 (14.55%)
60-69	0 (0%)	0 (0%)	4 (40%)	5 (29.41%)	20 (36.36%)
70-79	0 (0%)	2 (50%)	1 (10%)	4 (23.53%)	16 (29.09%)
80-89	0 (0%)	0 (0%)	2 (20%)	1 (5.88%)	3 (5.45%)
Age Group (female) (N=33)					
40-49	0 (0%)	0 (0%)	0 (0%)	2 (8.33%)	0 (0%)
50-59	0 (0%)	1 (50%)	2 (33.33%)	6 (25%)	0 (0%)
60-69	0 (0%)	1 (50%)	3 (50%)	7 (29.17%)	0 (0%)
70-79	1 (100%)	0 (0%)	1 (16.67%)	6 (25%)	1 (100%)
80-89	0 (0%)	0 (0%)	0 (0%)	3 (12.5%)	0 (0%)
Age					
40-49	1 (100%)	1 (20%)	1 (8.33%)	1 (4.35%)	10 (12.66%)
50-59	0 (0%)	1 (20%)	3 (25%)	8 (34.78%)	14 (17.72%)
60-69	0 (0%)	0 (0%)	5 (41.67%)	8 (34.78%)	27 (34.18%)
70-79	0 (0%)	3 (60%)	1 (8.33%)	5 (21.74%)	22 (27.85%)
80-89	0 (0%)	0 (0%)	2 (16.67%)	1 (4.35%)	6 (7.59%)
Creatinine	1.3± 5.95	1.43 ± 1.19	1.89 ± 0.5	3.22 ± 0.26	7.95 ± 0.08
BMI					
<24.9	0 (0%)	2 (40%)	8 (66.67%)	10 (43.48%)	42 (53.16%)
25-29.9	1 (100%)	3 (60%)	4 (33.33%)	11 (47.83%)	28 (35.44%)
>=30	0 (0%)	0 (0%)	0 (0%)	2 (8.7%)	9 (11.39%)
DM Duration	10 ± 56.2	14.4 ± 11.24	14 ± 4.68	13.8 ± 2.44	17.34 ± 0.71
Hba1C					
<6.49	0 (0%)	0 (0%)	0 (0%)	6 (26.09%)	21 (26.58%)
6.5-7.49	0 (0%)	1 (20%)	2 (16.67%)	4 (17.39%)	18 (22.78%)
7.5-8.49	0 (0%)	1 (20%)	3 (25%)	9 (39.13%)	15 (18.99%)
8.5-9.49	1 (100%)	0 (0%)	3 (25%)	1 (4.35%)	10 (12.66%)

>=9.5	0 (0%)	3 (60%)	4 (33.33%)	3 (13.04%)	15 (18.99%)
FBS					
<126	0 (0%)	0 (0%)	3 (25%)	7 (30.43%)	19 (24.05%)
>=126	1 (100%)	5 (100%)	9 (75%)	16 (69.57%)	60 (75.95%)
PPBS					
<200	0 (0%)	1 (20%)	5 (41.67%)	15 (65.22%)	42 (53.16%)
>=200	1 (100%)	4 (80%)	7 (58.33%)	8 (34.78%)	37 (46.84%)
SBP					
<130	0 (0%)	0 (0%)	2 (16.67%)	8 (34.78%)	12 (15.19%)
>=130	1 (100%)	5 (100%)	10 (83.33%)	15 (65.22%)	67 (84.81%)
DBP					
<80	0 (0%)	1 (20%)	4 (33.33%)	9 (39.13%)	22 (27.85%)
>=80	1 (100%)	4 (80%)	8 (66.67%)	14 (60.87%)	57 (72.15%)
Ace-I/Arbs					
Yes	1 (100%)	4 (80%)	6 (50%)	9 (39.13%)	13 (16.46%)
No	0 (0%)	1 (20%)	6 (50%)	14 (60.87%)	66 (83.54%)
B Blocker					
Yes	0 (0%)	2 (40%)	3 (25%)	9 (39.13%)	37 (46.84%)
No	1 (100%)	3 (60%)	9 (75%)	14 (60.87%)	42 (53.16%)
Diuretic					
Yes	0 (0%)	1 (20%)	2 (16.67%)	7 (30.43%)	11 (13.92%)
No	1 (100%)	4 (80%)	10 (83.33%)	16 (69.57%)	68 (86.08%)
Insulin					
Yes	0 (0%)	2 (40%)	6 (50%)	12 (52.17%)	49 (62.03%)
No	1 (100%)	3 (60%)	6 (50%)	9 (39.13%)	20 (25.32%)
Not Taking Now	0 (0%)	0 (0%)	0 (0%)	2 (8.7%)	10 (12.66%)
CCBS					
Yes	0 (0%)	1 (20%)	7 (58.33%)	11 (47.83%)	46 (58.23%)
No	1 (100%)	4 (80%)	5 (41.67%)	12 (52.17%)	33 (41.77%)
Nsaids					
Yes	0 (0%)	1 (20%)	2 (16.67%)	3 (13.04%)	12 (15.19%)
No	1 (100%)	4 (80%)	10 (83.33%)	20 (86.96%)	67 (84.81%)
Microalbuminuria					
Yes	1 (100%)	1 (20%)	3 (25%)	9 (39.13%)	8 (10.13%)
No	0 (0%)	4 (80%)	9 (75%)	12 (52.17%)	64 (81.01%)
No Urine	0 (0%)	0 (0%)	0 (0%)	2 (8.7%)	7 (8.86%)
Macroalbuminuria					
Yes	0 (0%)	4 (80%)	7 (58.33%)	12 (52.17%)	62 (78.48%)
No	1 (100%)	1 (20%)	5 (41.67%)	9 (39.13%)	10 (12.66%)
No Urine	0 (0%)	0 (0%)	0 (0%)	2 (8.7%)	7 (8.86%)

Hypertensive Medications					
Yes	1 (100%)	5 (100%)	11 (91.67%)	22 (95.65%)	78 (98.73%)
No	0 (0%)	0 (0%)	1 (8.33%)	1 (4.35%)	1 (1.27%)
Urine Output	900 ± 48598.03	980 ± 9719.61	925 ± 4049.84	937.83 ± 2112.96	428.35 ± 615.17
Hypertensive Retinopathy					
Grade 1	0 (0%)	0 (0%)	1 (8.33%)	0 (0%)	3 (3.8%)
Grade 2	0 (0%)	0 (0%)	1 (8.33%)	1 (4.35%)	3 (3.8%)
Grade 3	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.27%)
No	1 (100%)	5 (100%)	10 (83.33%)	22 (95.65%)	72 (91.14%)

Among the EGFR 60-89, 1 (100%) male participants were aged between 40 to 49 years, 1 (100%) female participants were aged between 70 to 79 years, 1 (100%) participants were aged between 40 to 49 years, among the EGFR 45-59, 1 (25%) male participants were aged between 40 to 49 years, 1 (25%) male participants were aged between 50 to 59 years, 2 (50%) male participants were aged between 70 to 79 years, 1 (50%) female participants were aged between 50 to 59 years, 1 (50%) female participants were aged between 60 to 69 years, (20%) were aged between 40 to 49 years, 1 (20%) were aged between 50 to 59 years, 3 (60%) were aged between 70 to 79 years, among the EGFR 0-44, 1 (10%) male participants were aged between 40 to 49 years, 2 (20%) male participants were aged between 50 to 59 years, 4 (40%) male participants were aged between 60 to 69 years, 1 (10%) male participants were aged between 70 to 79 years, 2 (20%) male participants were aged between 80 to 89 years, 2 (33.33%) female participants were aged between50 to 59 years, 3 (50%) female participants were aged between 60 to 69 years, 1 (16.67%) female participants were aged between 70 to 79 years, 1 (8.33%) were aged between 40 to 49 years, 3 (25%) were aged between 50 to 59 years, 5 (41.67%) were aged between 60 to 69 years, 1 (8.33%) were aged between 70 to 79 years, 2 (16.67%) were aged between 80 to 89 years and so on. (Table 49 & Figure 60)

DISCUSSION

The patients with 'hypertension, diabetes, & Chronic kidney disease' are a distinctive group of Diabetic Kidney Disease populace, that is acknowledged by increased albuminuria or decreased 'Glomerular Filtration Rate' (GFR) or together.[4] This interrupts renal function & changes the way kidney can excrete waste products and maintain fluid balance in body .[51] Patients with DHKD exhibit multiple symptoms and signs most commonly pedal edema, generalized weakness, easy fatiguability, loss of apetite and weight loss sometimes.[59] CKD & end-stage renal disease are caused by mainly DHKD. DHKD is slowly raising unnoticed in all the urbanized and also the developing nations. DHKD is associated with an increased death toll. Renal disease increased the morbidity and mortality in other conditions.[1] this calls for an immediate action from the governments and health organizations as this puts a socioeconomic crisis on the nations.[74] Hence the early diagnosis is advised in the DHKD.[51]

We did a hospital-based observational cross-sectional in the department of general medicine & nephrology was conducted on 120 patients with diabetes & hypertension in combination with kidney disease, with any duration of Diabetes > 2 years& any duration of hypertension >2 years Presenting to OPD. The study has been done from Jan 2019 - December 2019 intended, for one year after getting ethical clearance. MDRD, 'Chronic kidney disease'&'Glomerular Filtration Rate' (GFR) were considered as primary outcome variables. Micro albuminuria, Macro albuminuria was considered as Primary explanatory variable. Objectives of our study were similar to that of Suresh Chandra Dash et al.[16] (2018) Rahul Sudan et al.[9] (2018) Anand Verma et al.[58] (2016).

CLINICAL PROFILE IN DHKD:

Socio-demographic Variables:

Male sex has been found to be a risk factor in the development of CKD. In our study, of 120 patients, 72.50 percent were of male sex which was concordant with CKD registry of India report where males constituted 68percent of total CKD patients & CMC Vellore study where 62percent were males, probably reflects a quicker reduction in GFR in males as compared to females due to hormonal influence.[59] Because of documented age-related debility in GFR, the prevalence of CKD rises with age. This was seen in our study too, with most of the subjects were in groups of age 41-60 years with a mean of 63.64 years. Mean age in the CKD registry of India report was 48.3±16.6 years.

Clinical Variables:

'Chronic kidney disease' generally is recognized by 'routine screening with renal function tests & urinalysis' as an accompanying finding. Less commonly, patients may present with indications such as evident hematuria, "foamy urine" (a sign of albuminuria), increased micturition at night, renal angle tenderness, or reduced urine output. If CKD is advanced, patients complain of fatigue, reduced appetite, nausea, vomiting, metallic taste, changes in mental status, dyspnea, unintentional weight loss, pruritus, or peripheral edema'.[29] Among study population, 94 (78.33percent) had generalized weakness, 86 (71.67%) had peripheral Edema, 57 (47.50%) had decreased urine output, 58 (48.3%) had loss of appetite, 21 (17.5%) had nausea & vomiting, 71 (59.17%) had dyspnea, 30 (25.00%) had weight loss & 13 (10.8%) had change in mental status. Among study population, 72 (60%) had Pallor, 89 (74.2%) had Edema, 38 (31.7%) had muscle wasting & 11 (9.2%) had altered mental status. Thorough 'physical examination' may give the clues of CKD & should

comprise assessment of volume status of the patients. Poor oral intake, diarrhoea, vomiting, or excessive micturition may be shown as Signs of volume depletion, but signs of volume overload may be due to 'decompensated heart failure, liver failure, or nephrotic syndrome'. They are thus highlighting the need for a high index of suspicion even in patients giving with symptoms related to other systems.

Habits:

Smoking may be one of risk factor leading to diabetic kidney disease.[20] Association of smoking was shown in many studies with higher micro albuminuria & macro albuminuria & reduced GFR (<60 mL/min/1.73 m2) among patients with type 2 diabetes.[20] Alcohol consumption & diet are also recognized high-'risk factors' for diabetes & Hypertension.[60] 'Structural & functional' kidney alterations have been identified in diabetics who smoke, but the mechanism is not yet known. Among study population, 15 (12.50%) had smoking, 19 (15.8%) consumed Alcohol & 20 (16.7%) were chewing tobacco.

Duration:

Mean years of diabetes was 16.15 ± 7.5 in the study population, minimum & maximum was 2.0 & 35.0 in the study population with (95% C. I from 14.8 to 17.5). In CKD registry of India report, 40.7% of diabetics had a duration of <10 years & 16.9%, less than 5 years. This stresses the value of checking for the presence of micro albuminuria & proteinuria at the time of diagnosis of type 2 Diabetes mellitus.

LAB PROFILE IN DHKD:

'HYPERTENSION':

Among the patients of DHKD, the occurrence of Hypertension upsurges by retention of sodium & augmented 'peripheral vascular resistance'.[35] Numerous 'single nucleotide polymorphisms in genes such as ACE, eNOS, etc.' are seen in

Hypertension & DKD. 'ACE is a key enzyme in the renin-angiotensin system, which causes the conversion of angiotensin I to angiotensin II.[50]nsertion (I)/deletion (D) polymorphism of this gene has been established to be linked with Hypertension & DKD in several studies. Endothelial Nitric Oxide Synthase (eNOS) produces Nitric Oxide (NO) from L-arginine. NO has a prime role in the modulation of vascular tone & in the balance of blood pressure. Consequently, a mutation in eNOS alters NO production & leads to hypertension'.[11] Mean systolic BP was 149 ± 28.5, Diastolic BP was 84 ± 14.05, Height was 168.3 ± 6.73, Weight was 70.2 ± 11.33, BMI was 24.77 ± 3.88. Among the study population, 67 (55.83%) had normal breath sounds & 53 (44.17%) had bilateral crepitations. Among the study population, 56 (46.67%) participants' 1st-degree relatives were either diabetic or hypertensive. Among study population, 33 (27.5%) had ACEI/ARBs, 51 (42.5%) had beta-blockers, 65 (54.2%) had CCB, 6 (5%) had Nitrates, 24 (20%) had Prazosin & clonidine & 21 (17.5%) had prazosin.

DIABETES:

Diabetic nephropathy occurs by a complex mechanism. 'Initial hemodynamic changes of glomerular hyper perfusion & hyper filtration are followed by leakage of albumin from glomerular capillaries & structural alterations such as glomerular basement membrane thickening, glomerulosclerosis, glomerular hypertrophy, mesangial cell expansion, & podocyte injury & loss'.[57] Clinical manifestations of diabetic nephropathy comprise a decrease in 'Glomerular Filtration Rate' (GFR) & an escalation in levels of urinary albumin excretion.[51,57] In present study mean hemoglobin was 10.43 ± 1.6, 193.9 ± 86.97, PPBS was 219.21 ± 86.77, Hba1C was 8.11 ± 2.13, Creatinine was 6.11 ± 3.52, Urea was 90.59 ± 42.66, Sodium was 135.49

± 5.32, Potassium was 4.99 ± 0.79, Calcium was 8.52 ± 0.94, Phosphorous was 4.71 ± 1.71.

'Hyperglycemic damage' to vascular glycocalyx may distort its role as a barrier between blood & endothelium & its role in modulating vascular permeability to macromolecules, adhesion of circulating cells, & flow-mediated dilatation. Other factors include reduced levels of vitamin D that contribute to increased vascular calcifications'.[57] Among study population of urine glucose, 27 (22.50%) had 1+, 24 (20%) had 2+, 15 (12.50%) had 3+, 14 (11.67%) had 4+, 12 (10%) had traces, 19 (15.83%) were negative & 9 (7.50%) had no urine output. Van Buren et al. stated that diabetes is associated with a frequency of CKD of 8.9% in first stages, 12.8% in second stages, 19.4% in third stages, & 2.7% last stages.[11]

Anand verma et al[58], in the study, observed that 60.7% normotensive TYPE 2 DIABETES patients had DKD while in hypertensive TYPE 2 DIABETES patients incidence of DKD had increased to 73.6%. Agarwal et al., have studied 300 newly diagnosed type II diabetes & have found an occurrence of 17.5% of nephropathy & reported Hypertension as key factor causative to the development of kidney disease.[22]Among study population of 'urine protein, 30 (25%) had 1+, 38 (31.67%) had 2+, 28 (23.33%) had 3+, 7 (5.83%) had 4+, 6 (5%) had traces, 2 (1.67%) were negative & 9 (7.50%) had no urine output. In an enormous cross-sectional pathway study among, microabluminuria was reported in 731(24.62%) out of 2969 type 2 diabetes mellitus. Of these 731 patients, HT was present in 44.9% of patients. [58] HT in diabetes mellitus may be due to metabolic syndrome, secondary to complications of diabetes mellitus, endocrine disorders or coincidental (essential arterial HT, isolated systolic HT). Among study population, 63 (52.5%) took OHA, 69 (57.5%) took insulin & 25 (20.83%) took both insulin & OHA. Among the study population, the

majority had 39 (32.5%) had a past history of diabetes & 80 (66.7%) had a present history of diabetes'.

KIDNEY DISEASE:

Hypertension, Diabetes are risk issues for ill-fated complications like CKD & CVD, which put the massive monetary burden on patients & states for treatment. Current study witnessed significant higher link between diabetes, Hypertension & CKD.

'Once an evaluation of CKD has been made, the further thing is to decide staging, which is based on GFR, albuminuria, & cause of CKD. Staging of GFR is classified as G1 (GFR ≥90 mL/min/1.73 m2), G2 (GFR 60-89 mL/min/1.73 m2), G3a (45-59 mL/min/1.73 m2), G3b (30-44 mL/min/1.73 m2), G4 (15-29 mL/min/1.73 m2), & G5 (<15 mL/min/1.73 m2).[29] Modification of Diet in Renal Disease (MDRD) equation may be used to calculate eGFR from serum creatinine value. This equation takes into account variables of age, sex, & ethnicity, which are important determinants of serum creatinine.[63] For example, in elderly patients, age-related decrease in GFR is not equal to the increase in serum creatinine levels because of the expected age-related decline in creatinine generation.' There was a weak negative correlation between MDRD (ml/min) & Number of years of diabetes. Mean MDRD (ml/Min) in the present study was 15.28 ± 13.27. Mean CKD EPI (ml/Min) was 14.41 ± 13.08. Mean Urine albumin creatinine ratio was 2967.69 ± 4185.35.

Excretion of albumin in the urine is a chief causative factor for the advancement of kidney disease in people having diabetes. It's characterized by increased excretion of albumin/g creatinine in urine denoted to as microalbuminuria (30–300mg/g) or

macro-albuminuria (>300mg/g). Microabluminuria was seen in majority 85 (70.83%) in the present study.

Among the study population, the majority showed renal parenchymal changes.Non proliferative diabetic retinopathy among study population, 37 (30.8%) had Mild NPDR, 24 (20%) had Moderate NPDR, 11 (9.2%) had severe NPDR & 4 (3.3%) had proliferative diabetic retinopathy.Hypertensive retinopathy was seen only in <10% in the present study. Less than 5% showed ECG changes. Among the study population, the majority underwent dialysis on emergency HD. Similar observations were done by Fasil Wagnew et al[52], in their metanalysis of sub-Saharan countries, where they suggested that Diabetic nephropathy complication is significantly higher in hypertensive patients. A preventive strategy should be adopted or planned to reduce diabetes mellitus & its complication of neuropathy, particularly in hypertensive.

Difference between Urine albumin creatinine ratio & history of dialysis was not statistically significant. Median Urine albumin creatinine ratio was 1247.65 (IQR 545.33, 2328.75) in the past history of dialysis & it was 1755 (IQR 683, 3385.2) in the present history of dialysis.

In the literature, it is accepted that a combination of diabetes & hypertension leads to a greater occurrence of nephropathy.[11] Same was revealed by our study. Among GFR in study population, majority 119 (99.17%) were <=60 & 1 (0.83%) were >60. Among GFR <=60, mean age was 63.84 ±10.63. Among A1 Albuminuria, 1 (50%) were in range 30-44 in EGFR & 1 (50%) were <=15 in EGFR, among A2 Albuminuria, 1 (4.45%) were in the range 60-89 & 45-59 in EGFR, 3 (13.64%) were in the range 30-44 in EGFR, 9 (40.91%) were in the range 15-29 in EGFR, 8 (36.36%) Were in <=15 in EGFR,These findings were regular with observations made in the CKD registry of India report, where 50.3% presented in stage 5, 24% in stage 4

& 19.1% in stage 3. This exhibits short of awareness about CKD among public & failure of medical practitioners to identify and screen at-risk people & to diagnose CKD at an early stage, which would suffice suitable treatment to be instituted so as to prevent or decrease the rate of progression of CKD.

Among the sodium range in the study population, 38.33% had <135 mEq/L, 60.83% had 135-145 mEq/L and 0.83% had >145 mEq/L. Among the potassium range in the study population, 2.5% had <3.5 mEq/L, 71.67% had 3.5-5.5 mEq/L and 25.83% had >5.5 mEq/L. Among the calcium range in the study population, 70.83% had <9 mEq/L, 28.33% had 9-11 mEq/L and 0.83% had >11 mEq/L. Among the phosphorous range in the study population, 5.83% had <2.5 mEq/L, 49.17% had 2.5-4.5 mEq/L and 45% had >4.5 mEq/L. Majority of the study population showed levels of sodium 135-145 mEq/L (60.83%), potassium 3.5-5.5 mEq/L (71.67%), calcium <9 mEq/L (70.83%), phosphorous 2.5-4.5 mEq/L (49.17%). The present study's observation is in accordance with the study of Rahul Sudan et al[9] where they also observed a majority of the study population showed potassium 3.5-5.5 mEq/L (71.67%), however phosphorous levels 4.5- 6mg/dl (42%), calcium 4-8 mg/dl (65%), this is in contrast with the present study where lower levels were observed. This could be explained as few had ESRD in the above study and our study population constituted those with all the stages of CKD. Sodium(Na) balance certainly was seen in the pathophysiology of the onset of hypertension seen in Diabetes with kidney disease. Total exchangeable body Na was seen increased even in the absence of increased systemic RAAS activity in diabetics.[50] In early diabetes, the increased renal RAAS activity may enable increased sodium reabsorption independent of GFR. The situation was theorized as a reaction to the osmotic diuretic effects of tubular hyperglycemia, and that the typical GFR increase seen in early diabetes protects

against this Na avid state. Na loading does not appropriately suppress systemic RAAS in DM2, enabling hypertension to occur in the context of a high sodium diet. Na sensitivity occurs in patients with DM2 and is associated withalbuminuria. Insulin reduces renal Na excretion independent of serum glucose levels. This can be explained by the increased prevalence of Hypertension prior to onset of DM2, rather than DM1. As GFR gradually drops, the kidney's ability to excrete Na and water further diminishes. Volume sensitivity persists as a mechanism that facilitates further Na excretion at the expense of higher blood pressure in the context of a Na load.[11]

Mean GFR in male was 15.1 ± 12.96 years in study population & it was 12.61 ± 10.46 in the female. No statistically significant changes were observed in the present study with regard to GFR & Gender, GFR & age groups, GFR & age groups of males, females. The proportion of the difference between SBP & Micro Albuminuria was statistically significant. The proportion of the difference between SBP & insulin with Macro Albuminuria was statistically significant. (P value<0.05).

Table 50: Comparison of Socio-demographic findings among various studies.

Socio-demographic findings	Present study	Sathyan et al.[59]	Rahul Sudan et al.[9]	Suresh Chandra Dash et al.[16]
Sample size	120	333	300	3050
Age	64	60	52	56
Gender- Male Gender-Female	87 33	65% 35%	66 34	62 38

Table 51: Comparison of clinical findings among various studies.

Clinical findings	Present study	Sathyan et al.[59]	Anand Verma et al.[58]	Rahul Sudan et al.[9]
Duration of diabetes	15	12		
Duration of hypertension	15	5.2		
CVD	4.2%	50.15%		
1St Degree Relatives	46.67%	23%		
Dyspnoea	59.17%	75.6%		
Decreased urine output	47.50%	69.36%		
Loss of appetite	17.5%	66.97%		
Weight Loss				
Change in mental status	10.8%	50.45%		
Use of NSAIDS Previously	15.00%	5.10%		
Hemoglobin g/dl	10.25	8.42		9.02
Smoking	12.50%	32.73		48%
Alcohol	15.8%	6.00		8%
Tobacco chewing	16.7%	6.90		
vital signs- SBP DBP	150.00, 80.00			
Height	168.00			
Weight	70.50			
BMI	24.40		24.59±5.79	
HbA1c	7.60		7.58±1.4	

FBS	180.50			
PPBS	196.00			
Hba1C	7.60			
Creatinine	5.75		1.97±1.78	
Urea	92.00			
Sodium	136.00			
Potassium	4.95			5.01
Calcium	8.40			
Phosphorous	4.25	5.94		5.24
Urine albumin creatinine ratio	1485.3			
gfr	9.25	10.81.		
Proteinuria	0.83%		6.85 ± 0.97	
microalbuminuria	18.33%			
macroalbuminuria	70.83%			
Diabetic retinopathy	30.8%			
Hypertensive retinopathy	7.5%			

CONCLUSION

1. A hospital based observational cross-sectional in department of general medicine & nephrology was conducted on 120 patients with diabetes & Hypertension in combination with kidney disease, with any duration of Diabetes > 2 years & any duration of hypertension>2 years presenting to OPD.
2. Among study population, patients with DHKD present mainly with vague complaints such as generalized weakness, breathlessness, peripheral edema and decreased urine output as the chief presenting complaints.
3. Most of the patients with DHKD were found to have pallor and their mean hemoglobin was found to be 10.43 mg/dl.
4. In my study correlation between systolic blood pressure and macroalbuminuria was statistically significant showing that strict control of blood pressure is required to prevent DHKD.
5. DHKD patients show uncontrolled fasting blood glucose levels and this is detrimental in leading to worsening albuminuria as showed by our study. This potrays the importance of strict glycemic control.
6. The above study shows that DHKD patients have electrolyte imbalances mainly of potassium, calcium and phosphorous levels and they need to be corrected to prevent electrolyte disorders.
7. Study shows that there was no much correlation between number of years of diabetes or hypertension on progression to DHKD instead strict glycemic control and BP control are essential.
8. The study also projects the awareness among patients about complication of diabetes and hypertension as most patients commonly think IHD, nephropathy,and

retinopathy are the main complications and very few are aware of neuropathy, PVD and CVA.

9. Majority of DHKD population had macro albuminuria and high urine albumin creatinine ratio.This needs early detection. Drugs which help in decreasing albuminuria like ACE inhibtors or ARBS should be used in younger age groups.
10. Study shows that calculation of estimated GFR in every patient of diabetes & hypertension is important. In our study we found that more than half of patients were in kidney failure stage requiring renal replacement therapy. And rest were having high risk of progression to end stage renal failure. Out of 120 patients, 117 patients were in very high risk category with 80 of them requiring renal replacement -therapy.

LIMITATION & RECOMMENDATION

The current study is a cross-sectional study. Consequently, the incidence of hypertension & diabetic nephropathy may be different. Hence temporality of association is always in doubt. Simply referral cases were recruited for the study. Our study was only a hospital-based study leading to biased representation in community & generalization of our results is difficult. Incidence may be completely different when this type of study is done in a general population.

SUMMARY

Our study delivers sufficient evidence endorsing high relationship between diabetes, hypertension & kidney disease is like a bond. Many diabetics are ignorant of the persistence of renal damage prior to appearance due to the absence of urine albumin test & or serum creatinine estimation. Thus, DHKD at a crucial early stage goes unnoticed, leading to more advanced stages. This is a grave error & so it's essential to use term "Diabetic Hypertension-Kidney disease" for creating consciousness among patients & doctors for early detection of Diabetic Nephropathy, at a stage when it may be competently treated.

BIBLIOGRAPHY

1. WHO. Global status report on noncommunicable diseases 2010 [Internet]. 2010. World Health Organisation. [cited 2020 Sept 11]. Available From: https://www.who.int/nmh/publications/ncd_report_full_en.pdf
2. WHO. Preventing Chronic Disease: A Vital Investment [Internet]. 2005. World Health Organisation. [cited 2020 Sept 11]. Available From: https://www.who.int/chp/chronic_disease_report/en/
3. Yach D, Hawkes C, Gould Cl, Hofman Kj: The global burden of chronic diseases: Overcoming impediments to prevention and control. JAMA.2004.291(21):2616–22.
4. Abraham I, Kurdi S, MacDonald K. The hypertension, diabetes and chronic kidney disease triangle in Arab countries. J Hum Hypertens. 2017;31(6):373–5.
5. Varughese S, Abraham G. Chronic kidney disease in India: A clarion call for change. Clin J Am Soc Nephrol. 2018;13(5):802–4.
6. WHO. Analysis and use of health facility data [Internet]. 2012. World Health Organisation. [cited 2020 Sept 12]. Available From: https://www.who.int/healthinfo/tools_data_analysis_routine_facility/en/
7. Modi GK, Jha V. The incidence of end- stage renal disease in India: A population based study. Kidney Int. 2006; 70(12): 2131-3.
8. Grassmann A, Gioberge S, Moeller S, Brown G. ESRD patients in 2004: Global overview of patient numbers, treatment modalities and associated trends. Nephrol Dial Transplant. 2005; 20(12): 2587–93.
9. Sudan R, Ahmed M, Wani IA, Wani MM, Banday KA, Gupta G. Profile of Chronic Kidney Disease (CKD) patients presenting in a tertiary care center in north India 2018;5(8):72–80.

10. Hypertention in Diabetes Study (HDS):I.Prevalence of hypertension in newly presenting type 2 diabetic patients and the association with risk factors for cardio-vascular and diabetic complications. J Hypertens. 1993;11:309–17.

11. Van Buren PN, Toto R. Hypertension in diabetic nephropathy: epidemiology, mechanisms and management. Adv Chronic Kidney Dis.2011;18(1):28-41.

12. Atkins RC. The epidemiology of chronic kidney disease. Kidney Int Suppl. 2005;67(94):14–8.

13. WHO. Global Health Risks. Mortality and burden of disease attributable to selected major risks[Internet]. 2009. World Health Organisation. [cited 2020 Sept 23]. Available From: https://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf

14. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R, et al. Prevalence of diabetes and prediabetes (impaired fasting glucose and/or impaired glucose tolerance) in urban and rural India: phase I results of the Indian Council of Medical Research-INdia DIABetes (ICMR-INDIAB) study. Diabetologia. 2011;54(12):3022-7.

15. Shaw JE, Sicree RA, Zmmet PZ. Global estimates of, Clin prevalence of diabetes for 2010 and 2030. Diabetes Res Clin Pract.2010;87(1)87:4-14.

16. Dash SC, Agarwal SK, Panigrahi A, Mishra J, Dash D. Diabetes, hypertension and kidney disease combination “DHKD syndrome” is common in India. J Assoc Physicians India. 2018;66(3):30–3.

17. de Boer IH, Rue TC, Hall YN, Heagerty PJ, Weiss NS, Himmelfarb. Temporal trends in the prevalence of diabetic kidney disease in the United Status.JAMA.2011; 305(24):2532–2539.

18. Costacou T, Ellis D, Fried L, Orchard TJ. Sequence of progression of albuminuria and decreased GFR in persons with type 1 diabetes: a cohort study. Am J Kidney Dis. 2007 Nov;50(5):721–32

19. L. Sheng, M. Christopher AM. 乳鼠心肌提取 HHS Public Access. Physiol Behav. 2016;176(1):100–106.

20. Res a. P Rimary H Ealth C Are Ry H Ealt Th C Are 2007;(September):157–74.

21. Prabahar MR, Chandrasekaran V, Soundararajan P. Epidemic of chronic kidney disease in India -what can be done? Saudi J Kidney Dis Transpl. 2008 Sep;19(5):847–53.

22. Agarwal SK, Srivastava RK. Chronic kidney disease in India: challenges and solutions. Nephron Clin Pract. 2009;111(3):c197–203

23. Johnson DW, Atai E, Chan M, Phoon RK, Scott C, Toussaint ND, et al.; KHA-CARI. KHA-CARI guideline: Early chronic kidney disease: detection, prevention and management. Nephrology (Carlton). 2013 May;18(5):340–50

24. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guidelines for the Evaluation and Management of Chronic Kidney Disease. Kidney International Supplements. 2013; 3 1-150.

25. James MT, Hemmelgarn BR, Wiebe N, Pannu N, Manns BJ, Klarenbach SW, et al.; Alberta Kidney Disease Network. Glomerular filtration rate, proteinuria, and the incidence and consequences of acute kidney injury: a cohort study. Lancet. 2010 Dec;376(9758):2096–103

26. Ckd DO, Graded N. Chapter 1: definition and classification of CKD. Kidney Int Suppl (2011). 2013 Jan;3(1):19–62

27. Webster AC, Nagler EV, Morton RL, Masson P. Chronic kidney disease.

Lancet. 2017 Mar;389(10075):1238–52

28. Skorecki K, ChertowGM, Marsden PA, Taal MW, Yu ASL. Brenner& Rector's the Kidney. 10th ed. Philadelphia PE 2016.
29. Chen TK, Knicely DH, Grams ME. Chronic Kidney Disease Diagnosis and Management: A Review. JAMA. 2019 Oct;322(13):1294–304
30. Levey AS, Coresh J. Chronic kidney disease. Lancet. 2012;379(9811):165–80.
31. Carrero JJ. Gender differences in chronic kidney disease: underpinnings and therapeutic implications. Kidney Blood Press Res. 2010;33(5):383–92.
32. Vos T, Barber RM, Bell B, Bertozzi-Villa A, Biryukov S, Bolliger I, et al.; Global Burden of Disease Study 2013 Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet. 2015 Aug;386(9995):743–800
33. Fraser SD, Blakeman T. Chronic kidney disease: identification and management in primary care. Pragmat Obs Res. 2016 Aug 17;7:21-32.
34. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013 Jul;382(9888):260–72
35. Ketteler M, Wanner C. Chronic Kidney Disease - Update 2018. Dtsch Med Wochenschr. 2018;143(3):169–73.
36. Snyder S, Pendergraph B. Detection and evaluation of chronic kidney disease. Am Fam Physician. 2005;72(9):1723–34.
37. Jager KJ, Fraser SD. The ascending rank of chronic kidney disease in the global burden of disease study. Nephrol Dial Transplant. 2017 Apr;32

suppl_2:ii121–8

38. Tomino Y. Pathogenesis and treatment of chronic kidney disease: a review of our recent basic and clinical data. Kidney Blood Press Res. 2014;39(5):450–89
39. Ardalan MR, Sanadgol H, Nasri H, Baradaran A, Tamadon MR, Rafieian-Kopaei M. Vitamin D therapy in diabetic kidney disease; current knowledge on a public health problem. JPD. 2014;2(1):17-9.
40. Rafieian-Kopaie M, Nasri H. Silymarin and diabetic nephropathy. J Renal Inj Prev. 2012 Jan;1(1):3–5.
41. Nasri H R-KMD mellitus and renal failure: P and management. JRMS 2015;20(11):1112-20.
42. Behradmanesh S, Nasri H. Association of serum calcium with level of blood pressure in type 2 diabetic patients. J Nephropathol. 2013 Oct;2(4):254–7
43. Mulhall BP, Veerappan GR, Jackson JL. Meta-analysis: computed tomographic colonography. Ann Intern Med. 2005;142(8):635-50.
44. Freedman BI, Hicks PJ, Bostrom MA, Cunningham ME, Liu Y, Divers J, Kopp JB, Winkler CA, Nelson GW, Langefeld CD, Bowden DW. Polymorphisms in the non-muscle myosin heavy chain 9 gene (MYH9) are strongly associated with end-stage renal disease historically attributed to hypertension in African Americans. Kidney Int. 2009 Apr;75(7):736-45
45. Jones-Burton C, Mishra SI, Fink JC, Brown J, Gossa W, Bakris GL, et al. An in-depth review of the evidence linking dietary salt intake and progression of chronic kidney disease. Am J Nephrol. 2006;26(3):268–75
46. Hiß M, Kielstein JT. Chronic kidney disease (ckd). Urol a Glance. 2014;145–50.
47. Collins AJ, Foley RN, Herzog C, Chavers B, Gilbertson D, Herzog C, US

Renal Data System 2012 Annual Data Report. Am J Kidney Dis. 2013 Jan;61(1 Suppl 1):A7, e1-476

48. Ku E, Lee BJ, Wei J, Weir MR. Hypertension in CKD: Core Curriculum 2019. Am J Kidney Dis. 2019 Jul 1;74(1):120–31.

49. Nørgaard K, Feldt-Rasmussen B, Borch-Johnsen K, Saelan H, Deckert T. Prevalence of hypertension in type 1 (insulin-dependent) diabetes mellitus. Diabetologia. 1990 Jul;33(7):407–10

50. Patney V, Whaley-Connell A, Bakris G. Hypertension management in diabetic kidney disease. Diabetes Spectr. 2015;28(3):175–80.

51. Hussain S, Chand Jamali M, Habib A, Hussain MS, Akhtar M, Najmi AK. Diabetic kidney disease: An overview of prevalence, risk factors, and biomarkers. Clinical Epidemiology and Global Health. 2020:0–1.

52. Wagnew F, Eshetie S, Kibret GD, Zegeye A, Dessie G, Mulugeta H, et al. Diabetic nephropathy and hypertension in diabetes patients of sub-Saharan countries: A systematic review and meta-analysis. BMC Res Notes. 2018;11(1):1–7.

53. Pecoits-Filho R, Abensur H, Betônico CC, Machado AD, Parente EB, Queiroz M, et al. Interactions between kidney disease and diabetes: dangerous liaisons. Diabetol Metab Syndr. 2016 Jul;8(1):50.

54. Anothaisintawee T, Rattanasiri S, Ingsathit A, Attia J, Thakkinstian A. Prevalence of chronic kidney disease: A systematic review and meta-analysis. Clin Nephrol. 2009;71(3):244–54.

55. Ghaderian SB, Beladi-Mousavi SS. The role of diabetes mellitus and hypertension in chronic kidney disease. J Renal Inj Prev. 2014 Dec;3(4):109–10

56. Okada R, Yasuda Y, Tsushita K, Wakai K, Hamajima N, Matsuo S. The number of metabolic syndrome components is a good risk indicator for both early- and late-stage kidney damage. Nutr Metab Cardiovasc Dis. 2014;24(3):277–85.

57. Bakris GL. Recognition, pathogenesis, and treatment of different stages of nephropathy in patients with type 2 diabetes mellitus. Mayo Clin Proc. 2011 May;86(5):444–56

58. Verma A, Vyas S, Agarwal A, Abbas S, Agarwal DP, Kumar R. Diabetic kidney disease and hypertension: A true love story. J Clin Diagn Res. 2016;10(3):OC11–3.

59. Sathyan S, George S, Vijayan PM. M J. Clinical and epidemiological profile of chronic kidney disease patients in a tertiary care referral centre in South India. Int J Comm Med Public Heal. 2016;3(12):3487–92.

60. Joshi SR, Saboo B, Vadivale M, Dani SI, Mithal A, Kaul U, et al.; SITE Investigators. Prevalence of diagnosed and undiagnosed diabetes and hypertension in India—results from the Screening India's Twin Epidemic (SITE) study. Diabetes Technol Ther. 2012 Jan;14(1):8–15

61. Agarwal N, Sengar NS, Jain PK, Khare R. Nephropathy in newly diagnosed type 2 diabetics with special stress on the role of hypertension. J Assoc Physicians India. 2011 Mar;59:145–7

62. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis. 2002 Feb;39(2 Suppl 1):S1-266.

63. Persson F, Rossing P. Diagnosis of diabetic kidney disease: state of the art and future perspective. Kidney Int Suppl. 2018;8(1):2–7.

64. Agarwal SK, Dash SC, Irshad M, Raju S, Singh R, Pandey RM. Prevalence of chronic renal failure in adults in Delhi,India. Nephrol Dial Transplant.2005;20(8):1638-42.

65. Mani MK. Experience with a program for prevention of chronic renal failure in India. Kidney Int Suppl. 2005 Apr;67(94):S75–8

66. Idowu AA, Ajose AO, Adedeji AT, Adegoke AO, Jimoh KA. Microalbuminuria other markers of nephropathy and biochemical derangementsin type 2 diabetes mellitus: relationships and determinants. Ghana Med J.2017;51(2):56–63.

67. Alicic RZ, Rooney MT, Tuttle KR. Diabetic kidney disease: challenges, progress and possibilities. Clin J Am Soc Nephrol.2017;12(12):2032–45.

68. Ang YG, Heng BH, Saxena N, Liew ST, Chong PN. Annual all-cause mortality rate for patients with diabetic kidney disease in Singapore. J Clin Transl Endocrinol. 2016 Jan;4:1–6

69. Habib A, Najmi A. Mon-298 anemia prevalence and its impact ON health-related quality OF life IN diabetic kidney disease patients: evidence from a cross-sectional study. Kidney Int. Rep. 2019;4:S421–S422

70. Pettitt DJ, Saod MF, Bennett PH et al. F predispostition to renal disease in two generations of PI with type 2 (non insulin-dependent) diabetes mellitus. Diabetologia. 1990;33(7):438-43.

71. Engelgau MM, Narayan KM, Herman WH. Screening for type 2 diabetes. Diabetes Care. 2000 Oct;23(10):1563–80.

72. Nelson RG, Pettitt DJ, Baird HR, Charles MA, Liu QZ, Bennett PH, et al. Pre-diabetic blood pressure predicts urinary albumin excretion after the onset of type 2 (non-insulin-dependent) diabetes mellitus in Pima Indians.

Diabetologia. 1993 Oct;36(10):998–1001

73. Hallab M, Bled F, Ebran JM. Elevated serum angiotensin converting enzyme activity in type I insulin-dependent diabetic subjects with persistent microalbuminuria. Arch Mal Coeur Vaiss.1992;85(8)29:1185-8.

74. Belayneh K. Current Management of Chronic Kidney Disease: Literature Review. JOJ uro & nephron. 2018; 6(2): 555684

ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
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Ref: MDC/DOME/ 20 **Date: 24/11/2018**

To,

Dr. Abhiram N,
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME – DHKD SYNDROME – A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL AND MRC"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study tilted, **"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME - DHKD SYNDROME"** being conducted by Dr. ABHIRAM N, a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of DR.NAVEEN S ANGADI, Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME - DHKD SYNDROME"

PURPOSE OF THE STUDY:

PREVALANCE OF DIABETES HYPERTENSION KIDNEY DISEASE IN DIABETIC-HYPERTENSIVE PATIENTS BY USING ESTIMATED GLOMERULAR FILTRATION

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few blood investigations, namely **RENAL FUNCTION TESTS,URINALYSIS,USG KUB, GLUCOSE TOLERANCE TEST,**

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

By taking part in this study, prognosis and risk of development chronic kidney disease and diabetic nephropathy and effects of hyperension on kidney.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article.When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you

will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study.

It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

PRINCIPAL INVESTIGATOR: Dr. ABHIRAM N(Post Graduate Student), Department of General Medicine,Jawaharlal Nehru Medical College, Mobile – 8867817715

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, this consent form and have had all the questions answered.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression: ……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

ANNEXURE III

PROFORMA

DATE

CASE NO:

NAME:

AGE/SEX: CONTACT:

IP NO.:

ADDRESS:

OCCUPATION

COMPLAINTS AT PRESENTATION:

SPECIFIC QUESIONNAIRE

1] Diabetic since how long?

Medications?

Are your 1st degree relatives diabetic?

2] Hypertensive since how long?

medications?

3] Previously admitted for any kidney/ urine related problems?

4] Use of NSAIDS previously?

5] Are you aware of diabetic complications? Whatall?

6] History of dialysis in the past? Howmany?

7] Any habits like smoking/drinking/tobacco chewing?

PHYSICAL EXAMINATION:

PALLOR- YES/NO

LYMPHADENOPATHY-YES/NO

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

PULSE:

RESPIRATORY RATE:

BLOOD PRESSURE: STD 3 RECORDINGS

systolic bp diastolic bp

HEIGHT: CMS

WEIGHT: KG

BMI:

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

C.N.S.:

P.A.:

INVESTIGATIONS

FBS/PPBS

HbA1C

RENAL FUNCTION TESTS

creat

urea

sr electrolytes

URINALYSIS

urineprotien

urine glucose

GFR

1) MDRD

2) CKD EPI

URINE ALBUMIN TO CREATININE RATIO

MICROALBUMINURIA / MACROALBUMINURIA

USG KUB

kidney size rtlt

daibetic retinopathy - no / mild / moderate / severe

hypertensive retinopathy - yes / no

ECG –

ANNEXURE IV

KEY TO THE MASTER CHART

Age Group	40-49=3, 50-59=4, 60-69=5, 70-79=6, 80-89=7
Gender	Male=1, Female=2
GENERALISED WEAKNESS	Yes=1, No=2
PERIPHERAL EDEMA	Yes=1, No=2
DECREASED URINE OUTPUT	Yes=1, No=2
LOSS OFAPETITE	Yes=1, No=2
NAUSEA AND VOMITING	Yes=1, No=2
DYSPNOEA	Yes=1, No=2
WEIGHT LOSS	Yes=1, No=2
CHANGES IN MENTAL STATUS	Yes=1, No=2
OHA	A= Metformin, B=Sulfonylureas, C= Thiazolidinediones, D= Dpp4 inhibitors, E=Alpha glucosidase inhibitors
INSULIN	Yes=1, No=2
BOTH INSULIN AND OHA	Yes=1, No=2
ARE YOUR 1st DEGREE RELATIVES DIABETIC OR HYPERTENSION	Yes=1, No=2
ACE-I/ARBS 1	Yes=1, No=2
B BLOCKER 2	Yes=1, No=2
CCBs 3	Yes=1, No=2
NITRATES 4	Yes=1, No=2
DIURETIC 5	Yes=1, No=2
OTHERS- PRAZOSIN AND CLONIDINE 6	Yes=1, No=2
PREVIOUSLY ADMITTED FOR ANY KIDNEY/ URINE RELATED PROBLEMS	Yes=1, No=2
NSAIDS	Yes=1, No=2
HISTORY of Dialysis	Yes=1, No=2
HISTORY OF DIALYSIS present	Yes=1, No=2
NEPHROPATHY	Yes=1, No=2
RETINOPATHY	Yes=1, No=2

NEUROPATHY	Yes=1, No=2
IHD	Yes=1, No=2
PVD	Yes=1, No=2
CVA	Yes=1, No=2
SMOKING	Yes=1, No=2
ALCOHOL	Yes=1, No=2
TOBACCO CHEWER	Yes=1, No=2
PALLOR	Yes=1, No=2
EDEMA	Yes=1, No=2
MUSCLE WASTING	Yes=1, No=2
ALTERED MENTAL STATUS	Yes=1, No=2
SBP	<130=1, >=130=2
DBP	<80=1, >=80=2
BMI	<24.9=1, 25-29.9=2, >=30=3
Hemoglobin	<12.5=1, 13.5-15.5=2
FBS	<126=1, >=126=2
PPBS	<200=1, >=200=2
HBA1C	<6.49=1, 6.6-7.49=2, 7.5-8.49=3, 8.5-9.49=4, >=9.5=5
URINE PROTIEN	1+(30-70) =1, 2+ (100-200) =2, 3+ (300-600) =3, 4+ (600) =4, Traces (10-20) =5, Negative=6, No urine output=7
URINE GLUCOSE	1+(70-100) =1, 2+(150-200) =2, 3+ (300-500) =3, 4+ (>1000) =4, Traces (30-50) =5, Negative=6, No urine output=7
eGFR	>=90=1, 60-89=2, 45-59=3, 30-44=4,13-29=5, >=15=6
GFR	<=60=1, >60=2
UACR	<30=1, 31-299=2, >300=3
NORMAL, PROTENURIA	Yes=1, No=2
Micro Albuminuria	Yes=1, No=2
Macro Albuminuria	Yes=1, No=2

MILD NPDR	Yes=1, No=2
MODERATE NPDR	Yes=1, No=2
SEVERE NPDR	Yes=1, No=2
PDR	Yes=1, No=2
ECG FINDINGS Normal	Yes=1, No=2
Combined Anti Hypertensives	1= Ace inhibitors or arbs, 2= Beta blockers, 3= Calcium channel blocker, 4= Nitrates, 5= Diuretics, 6= Others
Hypertensive medications	Yes=1, No=2
Micro Albuminuria	Yes=1, No=2
Macro Albuminuria	Yes=1, No=2
DECREASED URINE OUTPUT	Yes=1, No=2
USG KUB PARENCHYMAL CHANGES	Normal=1, Grade 1 rpc=2, Grade 2 rpc=3, Grade 3 rpc=4

ANNEXURE I

ETHICAL CLEARANCE CERTIFICATE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 20 **Date: 24/11/2018**

To,

REG NO. BG0118001
PG student in Medicine,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME – DHKD SYNDROME – A ONE YEAR CROSS SECTIONAL STUDY AT KLES DR PRABHAKAR KORE HOSPITAL AND MRC"**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

INFORMED CONSENT

Dear Mr./Mrs./Dr. _____, you are kindly requested to enroll yourself in a research study tilted, **"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME - DHKD SYNDROME"** being conducted by **REG NO. BG0118001**, a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of DR._____, Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether or not to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"CLINICAL AND LABORATORY PROFILE OF DIABETES HYPERTENSION KIDNEY DISEASE SYNDROME - DHKD SYNDROME"

PURPOSE OF THE STUDY:

PREVALANCE OF DIABETES HYPERTENSION KIDNEY DISEASE IN DIABETIC-HYPERTENSIVE PATIENTS BY USING ESTIMATED GLOMERULAR FILTRATION

PROCEDURES INVOLVED:

If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few blood investigations, namely **RENAL FUNCTION TESTS,URINALYSIS,USG KUB, GLUCOSE TOLERANCE TEST,**

RISKS AND BENEFITS:

There are no potential risks involved in this study.

Benefits of taking part in this research:

By taking part in this study, prognosis and risk of development chronic kidney disease and diabetic nephropathy and effects of hyperension on kidney.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY:

Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES:

Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY:

All data collected or disclosed by you during the course of participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent.

The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

- In emergency to protect your rights AND welfare.
- If required by law.

AUTHORIZATION TO PUBLISH RESULT:

The results of the study may be used to publish an article.When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION:

No additional costs shall be incurred upon you for the purpose of this study.

It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION:

In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS:

You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

PRINCIPAL INVESTIGATOR: REG NO. BG0118001 (Post Graduate Student), Department of General Medicine,Jawaharlal Nehru Medical College,

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, this consent form and have had all the questions answered.

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name :……...

Signature / Left thumb impression: ……...

of the participant

Name of the legally authorized :……...

representative / guardian

Signature / Left thumb impression :……...

Witness' name :……...

Signature / Left thumb impression :……...

Investigator's name and signature :……...

Date:

Place:

ANNEXURE III

PROFORMA

DATE

CASE NO:

NAME:

AGE/SEX: CONTACT:

IP NO.:

ADDRESS:

OCCUPATION

COMPLAINTS AT PRESENTATION:

SPECIFIC QUESIONNAIRE

1] Diabetic since how long?

Medications?

Are your 1st degree relatives diabetic?

2] Hypertensive since how long?

medications?

3] Previously admitted for any kidney/ urine related problems?

4] Use of NSAIDS previously?

5] Are you aware of diabetic complications? Whatall?

6] History of dialysis in the past? Howmany?

7] Any habits like smoking/drinking/tobacco chewing?

PHYSICAL EXAMINATION:

PALLOR- YES/NO

LYMPHADENOPATHY-YES/NO

CYANOSIS- YES/NO

CLUBBING-YES/NO

EDEMA-YES/NO

VITALS:

PULSE:

RESPIRATORY RATE:

BLOOD PRESSURE: STD 3 RECORDINGS

systolic bp diastolic bp

HEIGHT: CMS

WEIGHT: KG

BMI:

SYSTEMIC EXAMINATION:

R. S.:

C.V.S.:

C.N.S.:

P.A.:

INVESTIGATIONS

FBS/PPBS

HbA1C

RENAL FUNCTION TESTS

creat

urea

sr electrolytes

URINALYSIS

urineprotien

urine glucose

GFR

1) MDRD

2) CKD EPI

URINE ALBUMIN TO CREATININE RATIO

MICROALBUMINURIA / MACROALBUMINURIA

USG KUB

kidney size rtlt

daibetic retinopathy - no / mild / moderate / severe

hypertensive retinopathy - yes / no

ECG –

ANNEXURE IV

KEY TO THE MASTER CHART

Age Group	40-49=3, 50-59=4, 60-69=5, 70-79=6, 80-89=7
Gender	Male=1, Female=2
GENERALISED WEAKNESS	Yes=1, No=2
PERIPHERAL EDEMA	Yes=1, No=2
DECREASED URINE OUTPUT	Yes=1, No=2
LOSS OFAPETITE	Yes=1, No=2
NAUSEA AND VOMITING	Yes=1, No=2
DYSPNOEA	Yes=1, No=2
WEIGHT LOSS	Yes=1, No=2
CHANGES IN MENTAL STATUS	Yes=1, No=2
OHA	A= Metformin, B=Sulfonylureas, C= Thiazolidinediones, D= Dpp4 inhibitors, E=Alpha glucosidase inhibitors
INSULIN	Yes=1, No=2
BOTH INSULIN AND OHA	Yes=1, No=2
ARE YOUR 1st DEGREE RELATIVES DIABETIC OR HYPERTENSION	Yes=1, No=2
ACE-I/ARBS 1	Yes=1, No=2
B BLOCKER 2	Yes=1, No=2
CCBs 3	Yes=1, No=2
NITRATES 4	Yes=1, No=2
DIURETIC 5	Yes=1, No=2
OTHERS- PRAZOSIN AND CLONIDINE 6	Yes=1, No=2
PREVIOUSLY ADMITTED FOR ANY KIDNEY/ URINE RELATED PROBLEMS	Yes=1, No=2
NSAIDS	Yes=1, No=2
HISTORY of Dialysis	Yes=1, No=2
HISTORY OF DIALYSIS present	Yes=1, No=2
NEPHROPATHY	Yes=1, No=2
RETINOPATHY	Yes=1, No=2

NEUROPATHY	Yes=1, No=2
IHD	Yes=1, No=2
PVD	Yes=1, No=2
CVA	Yes=1, No=2
SMOKING	Yes=1, No=2
ALCOHOL	Yes=1, No=2
TOBACCO CHEWER	Yes=1, No=2
PALLOR	Yes=1, No=2
EDEMA	Yes=1, No=2
MUSCLE WASTING	Yes=1, No=2
ALTERED MENTAL STATUS	Yes=1, No=2
SBP	<130=1, >=130=2
DBP	<80=1, >=80=2
BMI	<24.9=1, 25-29.9=2, >=30=3
Hemoglobin	<12.5=1, 13.5-15.5=2
FBS	<126=1, >=126=2
PPBS	<200=1, >=200=2
HBA1C	<6.49=1, 6.6-7.49=2, 7.5-8.49=3, 8.5-9.49=4, >=9.5=5
URINE PROTIEN	1+(30-70) =1, 2+ (100-200) =2, 3+ (300-600) =3, 4+ (600) =4, Traces (10-20) =5, Negative=6, No urine output=7
URINE GLUCOSE	1+(70-100) =1, 2+(150-200) =2, 3+ (300-500) =3, 4+ (>1000) =4, Traces (30-50) =5, Negative=6, No urine output=7
eGFR	>=90=1, 60-89=2, 45-59=3, 30-44=4,13-29=5, >=15=6
GFR	<=60=1, >60=2
UACR	<30=1, 31-299=2, >300=3
NORMAL, PROTENURIA	Yes=1, No=2
Micro Albuminuria	Yes=1, No=2
Macro Albuminuria	Yes=1, No=2

MILD NPDR	Yes=1, No=2
MODERATE NPDR	Yes=1, No=2
SEVERE NPDR	Yes=1, No=2
PDR	Yes=1, No=2
ECG FINDINGS Normal	Yes=1, No=2
Combined Anti Hypertensives	1= Ace inhibitors or arbs, 2= Beta blockers, 3= Calcium channel blocker, 4= Nitrates, 5= Diuretics, 6= Others
Hypertensive medications	Yes=1, No=2
Micro Albuminuria	Yes=1, No=2
Macro Albuminuria	Yes=1, No=2
DECREASED URINE OUTPUT	Yes=1, No=2
USG KUB PARENCHYMAL CHANGES	Normal=1, Grade 1 rpc=2, Grade 2 rpc=3, Grade 3 rpc=4

MASTER SHEET

Sl. No.	Age	Age Group	Gender	IP 1	Generalised weakness	Peripheral Edema	Decreased Urine Output	Loss of Appetite	Nausea and Vomiting	Dyspnoea	Weight Loss	Change in mental status	Diabetics since how long	OHA	Insulin	Boh insulin and OHA	Are your 1st degree relatives diabetic or hypertensive	Hypertensive since how long	ACEI / ARBS 1	B Blocker 2	CCBS 3	Nitrates 4	Diuretics	Others Prazosin and Clonidine 6	Combines Anti-Hypertensives	PREVIOUSLY ADMITTED FOR ANY KIDNEY URINE RELATED PROBLEMS	APROXIMATE AVERAGE URINE OUTPUT (IN ML)	USE O FNSAIDS PREVIOUSLY	HISTORY of Dialysis	Duration of dialysis	HISTORY OF DIALYSIS present
1	51	4	1	992299	1	1	1	2	2	1	2	2	15	yes A	1	1	3	3	2	2	1	2	2	1	3+6	1	600	2	2		1
2	65	5	2	991755	1	1	1	1	2	1	2	1	10	yes A+B+D	2	2	1	15	2	1	1	2	1	2	2+3+5	1	800	2	2		1
3	85	7	2	992158	1	1	1	2	1	1	2	2	22	yes D	2	2	3	20	2	2	1	2	2	2	3	1	700	2	2		2
4	60	5	1	992503	1	1	2	1	2	2	2	1	18	yes B	2	2	1	18	1	1	2	2	2	2	1+2	2	1000	2	2		2
5	63	5	1	992901	2	1	1	2	2	1	2	2	16	yes D	2	2	3	16	2	1	1	2	2	2	2+3	2	600	2	2		1
6	59	4	1	993880	1	1	2	2	2	1	1	2	14	yes A+B	2	2	1	10	2	1	1	2	2	2	2+3	1	500	2	1	2	1
7	75	6	1	987227	1	1	2	1	2	1	1	2	16	not taking n	1	2	2	10	2	2	1	2	1	2	3+5	2	500	2	2		1
8	66	5	2	994173	2	2	2	1	2	1	2	2	24	not taking n	1	2	1	20	2	2	1	1	2	2	3+4	2	450	1	2		1
9	74	6	2	995499	1	1	1	1	2	1	2	2	25	not taking n	1	2	1	25	2	2	1	2	2	2	3	1	400	2	1	3	1
10	72	6	1	994292	1	1	2	2	2	2	2	2	20	not taking n	1	2	2	16	2	1	1	2	2	2	2+3	1	400	2	2		1
11	68	5	1	996984	2	1	1	1	2	2	1	2	20	not taking n	1	2	1	20	1	2	2	2	2	2	1	1	400	2	1	3	1
12	55	4	2	994598	2	1	2	2	1	2	2	2	9	yes B	2	2	1	9	1	2	2	2	2	2	1	2	900	2	2		2
13	59	4	1	992617	1	2	1	2	2	1	1	2	15	yes A+B	1	1	3	15	2	2	2	2	2	1	6	1	500	1	2		1
14	65	5	2	996080	1	1	2	1	1	1	2	2	15	not taking n	1	2	2	25	2	1	2	2	2	1	2+6	1	600	1	2		1
15	63	5	1	995084	1	1	2	2	1	2	2	2	24	not taking n	1	2	2	20	1	2	2	2	2	2	1	2	700	2	2		1
16	62	5	2	1003382	1	2	2	2	2	2	2	2	20	not taking n	1	2	2	15	1	2	1	2	2	2	1+3	2	1200	1	2		2
17	55	4	1	1003079	1	2	2	2	2	1	2	1	10	yes D	2	2	1	10	1	2	2	2	2	2	1	2	1000	2	2		2
18	63	5	2	1003904	1	2	2	2	2	2	2	2	13	yes A+B+D	2	2	1	8	1	2	1	2	2	2	1+3	2	900	2	2		2
19	64	5	1	1001807	1	1	2	1	1	1	2	2	10	not taking n	1	2	3	10	2	2	1	2	2	1	3+6	1	400	2	2		1
20	75	6	1	1003002	1	1	2	2	2	1	2	2	10	yes B	2	2	1	7	1	1	2	2	2	2	1+2	2	900	2	2		2
21	53	4	1	1002986	1	2	2	2	2	2	2	2	4	yes A+B	2	2	1	15	1	2	2	2	2	2	1	2	1300	2	2		2
22	82	7	2	984367	1	2	2	2	2	1	2	2	8	not taking n	1	2	1	20	1	2	1	2	1	2	1+3+5	2	700	1	2		2
23	80	7	1	984626	1	1	1	1	2	1	2	1	12	yes A+D+F	2	2	2	12	2	2	1	2	2	1	3+6	2	400	2	2		1
24	57	4	1	984641	2	2	2	2	2	1	2	2	15	not taking n	1	2	1	15	2	2	1	2	2	2	3	1	850	2	2		1
25	75	6	1	984180	1	1	2	1	2	1	1	2	25	yes A+D+F	1	2	1	20	1	2	2	2	1	2	1+5	2	800	1	2		2
26	69	5	1	984848	2	1	2	2	2	1	2	2	2.5	yes A+B	2	2	1	3	2	2	2	2	2	2	NOT TAKING	2	1200	1	2		2
27	62	5	1	983162	1	2	1	2	2	2	2	2	14	yes A+B	2	2	1	4	1	2	1	2	2	2	1+3	2	650	2	2		2
28	68	5	1	985230	1	1	1	1	1	1	1	1	13	yes B	2	2	2	13	2	2	1	2	1	1	3+5+6	2	300	2	2		1
29	70	6	1	985778	1	1	1	2	2	1	2	2	8	yes A	2	2	2	8	2	2	1	2	2	2	3	2	450	2	2		1
30	73	6	1	984538	1	2	2	1	2	2	1	2	35	yes A+B+D	2	2	1	20	2	2	1	1	2	1	3+4+6	1	650	2	1	3	1
31	63	5	1	986137	1	2	1	1	2	2	2	2	20	yes B+D	1	1	2	5	1	2	2	2	1	2	1+5	2	500	1	2		2
32	68	5	1	990819	1	1	2	1	1	1	2	2	22	yes A+D	1	1	1	15	2	2	2	2	1	2	5	2	800	2	2		2
33	58	4	1	997722	1	2	1	1	2	2	1	2	14	yes D	1	1	1	14	2	1	1	2	2	1	2+3+6	1	750	1	1	4	1
34	50	4	2	998718	1	1	1	1	1	1	2	1	15	not taking n	1	2	2	15	1	2	1	2	2	2	1+3	2	200	2	2		1

35	57	4	1	998505	1	1	1	1	1	1	2	1	12	yes A+B	2	2	2	14	2	2	2	1	1	2	4+5	2	250	1	2		1
36	56	4	2	998705	1	1	2	2	2	1	2	2	9	yes A+B	1	1	2	7	2	2	1	2	2	2	3	2	1000	1	2		2
37	65	5	1	997539	1	2	2	1	2	2	2	2	25	not taking n	1	2	1	20	2	2	2	2	2	1	6	2	400	2	2		1
38	75	6	1	19	1	2	2	1	1	1	2	2	20	not taking n	1	2	3	20	2	1	2	2	2	2	2	2	800	2	2		2
39	69	5	1	100588	1	2	1	2	2	1	1	2	18	not taking n	1	2	2	18	2	2	2	2	2	1	6	2	400	1	2		1
40	85	7	2	995724	1	1	1	2	2	1	2	2	30	not taking n	1	2	2	30	2	2	1	2	2	1	6	2	300	2	2		1
41	65	5	2	1000817	2	1	1	2	1	2	2	2	15	yes A+B	2	2	2	15	1	1	2	2	1	2	1+2+5	2	800	2	2		2
42	70	6	2	1002024	2	1	1	2	2	1	1	2	8	yes A+B	1	1	3	8	1	2	2	2	1	2	1+5	2	150	1	2		1
43	61	5	1	1003303	2	2	1	1	2	1	2	2	12	not taking n	3	2	2	12	2	1	1	2	2	1	2+3+6	1	0	2	1	6	1
44	60	5	1	1002642	1	1	1	2	2	2	2	2	16	yes D	1	1	2	16	2	1	1	2	2	2	2+3	1	550	2	1	2	1
45	58	4	1	1004046	1	1	2	2	2	2	2	2	15	not taking n	1	2	2	15	1	2	2	2	1	2	1+5	2	1000	2	2		2
46	71	6	2	1004596	1	1	2	1	2	2	2	2	25	not taking n	1	2	1	20	2	2	1	2	2	2	3	2	200	2	2		1
47	66	5	1	1004077	1	2	2	2	2	1	2	2	17	yes D	1	1	2	15	2	2	2	2	2	1	6	1	600	2	1	2	1
48	59	4	1	1003960	1	1	2	2	2	1	2	2	14	yes D	2	2	1	14	2	1	1	2	2	1	2+3+6	2	700	2	1	1	1
49	70	6	1	1005729	1	1	1	1	2	1	2	2	16	not taking n	1	2	1	20	2	2	1	1	2	2	3+4	1	400	2	1	3	1
50	65	5	1	1006224	2	1	2	1	2	2	2	2	10	yes A+B	2	2	3	8	2	2	1	2	2	2	3	2	1400	2	2		2
51	89	7	1	1005542	1	2	1	1	2	1	2	2	30	not taking n	3	2	2	30	2	1	2	2	2	2	2	1	0	2	1	12	1
52	75	6	2	963137	1	2	2	2	2	2	2	2	20	yes B	1	1	2	20	2	1	1	2	2	2	2+3	2	1200	2	2		2
53	73	6	1	1008125	2	2	1	2	2	1	1	2	30	not taking n	3	2	2	30	2	1	2	2	1	2	2+5	1	0	2	1	10	1
54	63	5	2	918804	1	1	1	2	2	1	2	2	15	not taking n	1	2	1	15	2	1	2	2	2	2	2	1	600	2	1	6	1
55	64	5	1	919851	1	1	2	2	2	2	2	2	20	yes B	1	1	1	15	2	1	1	2	2	2	2+3	2	1000	2	2		2
56	64	5	2	919026	2	2	2	1	1	1	2	2	10	yes B	1	1	3	10	1	1	1	2	2	2	1+2+3	2	350	2	2		1
57	53	4	2	920038	1	1	2	1	2	2	2	2	4	not taking n	1	2	2	4	1	2	2	2	2	2	1	2	1100	2	2		2
58	82	7	1	921723	1	1	1	2	2	1	1	2	20	yes C+D	1	1	2	25	2	1	1	2	2	2	2+3	2	550	2	2		2
59	68	5	1	922164	1	1	2	1	2	1	2	1	30	not taking n	1	2	1	20	2	1	2	2	2	2	2	1	750	2	1	8	1
60	79	6	1	924076	1	1	2	2	2	1	1	2	18	not taking n	1	2	1	16	2	1	2	2	2	2	2	2	450	2	2		1
61	46	3	1	924769	1	2	2	2	2	2	2	2	7	yes A	2	2	1	7	1	2	2	2	2	2	1	2	1000	2	2		2
62	58	4	1	925210	1	1	2	1	2	2	2	2	17	yes A+B+D	2	2	1	15	2	1	2	2	2	2	2	2	900	2	2		2
63	63	5	1	925103	1	2	2	1	2	2	1	2	7	yes B	2	2	2	5	1	1	2	2	2	2	1+2	2	1000	2	2		2
64	46	3	2	925787	1	1	1	2	2	1	2	2	15	yes D	1	1	2	5	2	1	1	2	2	2	2+3	1	700	1	1	4	1
65	51	4	1	926462	1	1	2	1	2	1	2	2	12	yes B+D	2	2	3	9	2	1	2	2	1	1	2+5+6	1	890	1	1	5	1
66	60	5	2	925414	2	2	1	2	2	1	2	1	26	yes A+B	2	2	1	20	1	1	2	2	2	2	1+2	2	200	1	2		1
67	64	5	1	926933	1	1	1	1	2	1	1	2	10	not taking n	3	2	1	2	2	2	1	2	2	2	3	2	350	1	2		1
68	66	5	1	927904	1	1	1	2	2	1	2	2	19	not taking n	1	2	2	15	2	2	1	2	2	1	3+6	1	0	2	1	7	1
69	75	6	1	927456	1	1	2	2	2	1	2	1	25	yes B	1	1	1	15	2	2	1	2	2	2	3	2	800	2	2		2
70	46	3	1	927428	1	1	2	2	2	2	2	2	18	yes D	2	2	2	6	2	2	2	2	2	2	NOT TAKING	2	500	2	2		1
71	70	6	1	928808	1	2	1	1	2	2	1	2	30	not taking n	3	2	2	30	2	2	1	2	2	2	3	1	650	2	1	3	1
72	65	5	2	928582	2	1	2	2	2	1	1	2	14	yes A+B+D	2	2	1	10	2	2	1	2	2	2	3	2	400	2	2		1
73	77	6	1	927091	1	1	1	1	2	1	1	2	20	not taking n	1	2	2	20	2	1	2	2	2	2	2	2	200	2	2		1
74	54	4	1	972862	1	1	2	2	2	1	2	2	16	not taking n	3	2	2	10	1	1	2	2	1	2	1+2+5	1	650	2	1	2	1
75	73	6	1	929393	1	1	2	1	2	2	2	2	25	not taking n	1	2	1	15	2	2	1	2	2	2	3	1	600	2	1	4	1
76	40	3	1	907579	2	1	2	2	2	1	2	2	3	yes A+B+C	2	2	2	3	2	2	1	2	2	1	3+6	1	550	2	1	1	1
77	72	6	1	927321	1	1	2	1	1	1	2	2	35	not taking n	3	2	2	35	1	1	2	2	2	2	2	1	500	2	1	5	1
78	53	4	1	930048	1	1	1	2	2	1	2	2	19	yes D	1	1	1	10	2	1	2	2	2	1	2+6	1	650	2	1	3	1
79	70	6	1	910419	1	1	1	2	2	1	1	2	15	not taking n	3	2	1	5	2	1	2	2	2	2	2	1	0	2	1	7	1
80	54	4	2	929943	1	1	2	1	1	1	2	1	10	not taking n	1	2	3	6	2	1	2	2	2	2	2	2	450	2	2		1
81	58	4	2	929108	1	1	1	1	2	1	2	2	20	not taking n	3	2	2	20	2	1	2	2	1	2	2+5	2	250	2	2		1
82	70	6	1	930454	2	1	2	2	2	1	2	2	35	not taking n	1	2	1	20	2	1	1	2	2	2	2+3	1	250	2	1	5	1

83	50	4	2	931941	1	1	2	1	1	1	2	2	8	yes D	2	2	1	8	2	1	1	2	2	2	2+3	1	500	2	1	1	1
84	60	5	1	989607	1	1	1	1	2	2	2	2	10	yes A+D	1	1	1	10	2	1	1	2	2	2	2+3	2	400	2	2		1
85	47	3	1	931479	1	1	2	1	1	2	2	2	2	yes A+B+D	2	2	2	2	2	2	2	2	2	2	NOT TAKING	2	1000	1	2		2
86	60	5	1	932275	2	2	1	1	2	2	2	2	12	yes B	2	2	1	12	1	2	2	2	2	2	1	1	400	2	1	3	1
87	48	3	1	930458	1	1	1	2	2	2	2	2	14	yes D	1	1	2	4	2	2	1	2	2	2	3	1	600	2	1	1	1
88	74	6	1	965843	1	1	2	2	2	1	2	2	16	yes D	2	2	1	16	2	2	1	1	1	2	3+4+5	2	800	2	2		2
89	74	6	1	966585	2	1	1	2	2	2	1	2	20	not taking n	1	2	2	20	2	1	2	2	1	1	2+5+6	1	450	2	1	1	1
90	48	3	1	931827	2	1	1	2	2	2	2	2	8	yes A+B	1	1	2	6	2	1	2	2	2	1	2+6	2	400	2	2		1
91	70	6	1	933802	1	1	1	1	2	2	1	2	25	not taking n	1	2	1	25	1	1	2	2	2	2	1+2	1	400	2	1	3	1
92	48	3	2	908489	1	1	1	1	2	2	1	2	10	yes A+B	1	1	1	12	2	2	1	2	2	1	3+6	1	500	2	1	2	1
93	75	6	2	935194	1	1	1	1	2	1	1	2	30	yes D	1	1	1	30	2	2	1	2	2	2	3	1	350	2	1	4	1
94	40	3	1	935142	2	2	2	2	2	1	2	2	10	yes A+B+D	2	2	1	6	1	2	2	2	2	2	1	2	900	2	2		2
95	70	6	2	935950	2	1	2	1	2	1	2	2	12	not taking n	1	2	1	12	2	1	1	2	2	2	2+3	2	1100	2	2		2
96	71	6	2	932912	1	1	2	2	1	2	2	2	16	not taking n	1	2	2	10	2	1	1	2	2	2	2+3	2	550	2	2		1
97	70	6	1	936729	1	1	1	1	2	1	2	1	25	not taking n	1	2	2	25	2	2	1	2	2	2	3	2	250	2	2		1
98	69	5	1	938351	1	2	1	1	1	2	1	2	22	not taking n	3	2	1	22	2	2	1	2	2	2	3	2	300	2	2		1
99	61	5	1	938420	1	2	1	1	2	2	2	1	22	not taking n	1	2	1	15	2	2	1	2	2	2	3	2	400	2	2		1
100	54	4	1	938694	2	1	1	2	2	2	2	2	10	not taking n	1	2	1	14	2	2	1	2	2	2	3	1	650	2	1	3	1
101	57	4	2	937423	1	1	1	1	1	1	2	2	12	yes A	1	2	1	10	1	1	2	2	2	2	1+2	2	350	2	2		1
102	70	6	1	941662	1	1	2	2	2	2	2	2	20	not taking n	1	2	1	20	2	1	2	1	1	2	2+4+5	2	900	2	2		2
103	54	4	1	942365	2	2	2	1	2	2	1	2	2	yes D	1	1	1	2	1	2	2	2	2	2	1	2	1400	2	2		2
104	66	5	1	944566	2	1	1	2	2	1	1	2	15	not taking n	2	2	2	15	2	2	1	2	2	2	3	1	600	2	1	7	1
105	63	5	2	944826	1	1	1	1	2	1	2	2	22	not taking n	1	2	2	25	2	1	2	2	2	2	2	1	850	2	2		2
106	78	6	1	945658	1	2	1	1	2	2	1	2	18	not taking n	1	2	2	18	2	2	2	2	1	2	5	1	0	2	1	2	1
107	74	6	2	942334	1	1	1	1	2	2	1	2	12	yes B	2	2	1	10	2	2	1	2	1	2	3+5	1	500	2	1	1	1
108	46	3	1	946444	1	1	2	2	1	2	1	2	5	not taking n	1	2	2	5	2	1	1	2	2	2	2+3	2	350	2	2		1
109	60	5	1	946526	1	1	2	2	2	1	2	2	13	not taking n	3	2	2	10	2	1	1	2	2	1	2+3+6	2	350	2	2		1
110	87	7	1	948696	1	1	2	1	2	1	2	2	30	yes D	2	2	2	30	1	2	1	2	1	2	1+3+5	2	700	2	2		2
111	62	5	1	948415	1	2	2	2	2	2	2	2	10	not taking n	1	2	1	7	1	1	2	2	2	2	1+2	2	1000	2	2		2
112	63	5	1	949383	1	1	1	1	2	2	2	2	20	not taking n	2	2	2	17	2	1	1	2	2	2	2+3	1	550	2	1	3	1
113	48	3	1	949231	1	1	1	1	1	1	2	2	14	yes A	1	1	2	10	2	1	1	2	2	2	2+3	2	250	2	2		1
114	53	4	2	949631	1	1	2	2	2	1	2	2	14	not taking n	1	2	2	12	1	1	2	2	2	1	1+2+6	2	900	2	2		2
115	82	7	1	950448	1	2	2	1	2	2	2	2	18	not taking n	2	2	2	18	2	2	1	2	2	2	3	2	850	2	2		2
116	40	3	1	952569	1	2	1	2	2	1	2	2	4	yes A+B	2	2	2	4	1	2	2	2	2	2	1	2	200	2	2		1
117	50	4	1	955462	1	1	2	1	2	2	2	2	8	yes D	1	1	1	8	2	2	1	2	2	2	3	2	1220	2	2		2
118	46	3	1	956055	2	1	2	2	2	1	2	2	2	yes A	1	1	2	2	2	2	1	2	2	2	3	2	1300	2	2		2
119	47	3	1	958314	2	1	1	2	2	1	2	2	10	not taking n	1	2	1	10	2	2	2	2	2	1	6	1	650	2	1	1	1
120	82	7	1	958623	1	1	1	2	2	2	1	2	26	not taking n	3	2	2	26	2	2	1	2	2	2	3	1	0	2	1	8	1

Sl. No.	FREQUENCY OF DIALYSIS	NEPHROPATHY	RETINOPATHY	NEUROPATHY	IHD	PVD	CVA	SMOKING	ALCOHOL	TOBACCO CHEWER	PALLOR	EDEMA	MUSCLE WASTING	ALTERED MENTAL STATUS	SYSTOLIC BP	SBP-cat	DIASTOLIC BP	DBP cat	HEIGHT	WEIGHT	BMI	BMI cat	RESPIRATORY SYSTEM EXAMINATION	HEMOGLOBIN	Haemoglobin Male	Haemoglobin female	Haemoglobin cat	FBS	FBS cat	PPBS	PPBS cat	HBA1C	Hba1CCat
1	3/week	2	2	2	2	2	2	2	1	1	1	1	2	2	210	2	100	2	166	74	26.9	2.00	1r	11.40			1	200	2	382	2	15.4	5
2	2/week	2	2	2	1	2	2	2	2	2	1	1	2	1	200	2	100	2	156	76	31.2	3.00	b/	9.00			1	105	1	189	1	5.8	1
3	No	2	2	2	2	2	2	2	2	1	1	1	2	2	130	2	80	2	158	84	33.6	3.00	b/	10.00			1	237	2	289	2	6.3	1
4	No	2	2	2	1	2	2	2	2	1	2	1	2	1	180	2	80	2	170	76	26.3	2.00	1r	10.30			1	153	2	211	2	5.9	1
5	3/week	2	2	2	1	2	2	2	2	2	1	1	2	2	150	2	80	2	170	87	30.1	3.00	b/	8.80			1	154	2	195	1	7.2	2
6	3/week	1	1	1	1	2	2	2	1	1	1	1	1	2	130	2	80	2	176	66	21.3	1.00	b/	10.40			1	242	2	186	1	9.2	4
7	3/week	1	1	2	1	2	1	2	2	2	1	1	1	2	160	2	90	2	165	56	20.6	1.00	1r	10.70			1	417	2	390	2	13.8	5
8	3/week	1	1	1	1	2	1	2	2	2	1	1	2	2	140	2	70	1	162	82	32.0	3.00	b/	7.90			1	343	2	290	2	8.0	3
9	3/week	1	1	2	1	2	1	2	2	2	2	1	2	2	140	2	80	2	154	65	27.4	2.00	b/	12.00			1	102	1	143	1	6.2	1
10	emergency hd	1	1	1	1	1	1	2	2	2	1	1	1	2	120	1	60	1	167	63	22.3	1.00	1r	7.60			1	266	2	199	1	5.3	1
11	2/week	1	1	1	1	1	1	2	2	2	2	1	1	2	120	1	80	2	168	50	17.7	1.00	1r	11.60			1	163	2	144	1	5.8	1
12	No	1	2	2	1	2	2	2	2	2	2	1	2	2	140	2	90	2	162	60	22.9	1.00	1r	12.10			1	186	2	331	2	8.2	3
13	3/week	1	1	2	2	2	2	2	1	2	1	1	1	2	180	2	110	2	172	62	21.0	1.00	b/	9.00			1	277	2	317	2	8.6	4
14	2/week	2	2	2	1	2	2	2	2	1	1	1	1	2	140	2	80	2	156	58	23.8	1.00	b/	10.20			1	107	1	225	2	6.9	2
15	2/week	1	1	2	1	2	2	2	1	2	1	1	1	2	110	1	70	1	175	75	24.5	1.00	1r	9.00			1	127	2	196	1	7.4	2
16	No	2	2	2	1	2	1	2	2	2	2	2	2	2	130	2	80	2	165	76	27.9	2.00	1r	12.40			1	114	1	164	1	8.4	3
17	No	1	2	2	1	2	1	2	1	1	2	2	2	2	150	2	90	2	170	80	27.7	2.00	1r	13.20		1.00	2	162	2	202	2	7.6	3
18	No	2	2	2	1	2	2	2	2	2	1	2	2	2	170	2	110	2	170	86	29.9	2.00	1r	9.10			1	179	2	327	2	12.2	5
19	emergency hd	2	2	2	1	2	2	1	2	2	1	1	2	2	160	2	100	2	180	86	26.5	2.00	b/	9.00			1	193	2	252	2	6.8	2
20	No	1	2	2	1	2	1	2	2	2	2	2	1	2	170	2	110	2	172	65	22.5	1.00	b/	12.50		1.00	2	230	2	202	2	9.9	5
21	No	1	1	2	1	2	2	2	1	2	2	2	2	2	140	2	90	2	177	76	24.3	1.00	1r	14.10	1.00	1.00	2	109	1	145	1	9.1	4
22	No	2	2	2	2	2	2	2	2	2	1	1	2	2	190	2	110	2	159	88	34.8	3.00	b/	9.40			1	145	2	183	1	6.2	1
23	emergency hd	2	2	2	2	2	1	2	2	2	1	1	2	2	200	2	110	2	172	76	25.7	2.00	b/	9.70			1	147	2	195	1	6.6	2
24	3/week	1	1	1	1	2	1	2	2	2	1	1	1	2	160	2	100	2	172	68	23.0	1.00	b/	9.60			1	332	2	343	2	11.6	5
25	No	1	1	1	1	2	2	2	2	2	2	2	1	2	190	2	100	2	166	58	21.0	1.00	1r	11.70			1	259	2	186	1	6.5	2
26	No	2	2	2	1	2	2	2	2	2	2	1	1	2	130	2	80	2	165	59	21.7	1.00	1r	12.60		1.00	2	142	2	82	1	8.7	4
27	No	2	2	2	2	2	2	2	2	1	2	2	2	2	140	2	90	2	173	72	24.1	1.00	1r	12.10			1	226	2	239	2	6.7	2
28	emergency hd	2	2	2	1	2	1	2	2	2	2	1	1	1	150	2	80	2	163	52	19.6	1.00	b/	12.80		1.00	2	191	2	415	2	9.9	5
29	emergency hd	2	2	2	1	2	2	2	2	2	2	1	2	2	100	1	70	1	176	72	23.2	1.00	b/	11.80			1	242	2	201	2	7.6	3
30	3/week	1	1	2	1	2	2	2	1	2	1	1	1	2	160	2	90	2	164	48	17.8	1.00	b/	10.00			1	132	2	155	1	8.3	3
31	No	1	2	2	1	2	2	2	2	2	2	2	2	2	130	2	90	2	168	66	23.4	1.00	1r	11.10			1	322	2	371	2	10.6	5
32	No	1	2	2	1	2	2	1	1	1	1	1	2	2	150	2	90	2	176	88	28.4	2.00	b/	11.50			1	374	2	273	2	8.6	4
33	3/week	1	1	1	1	1	1	2	2	2	2	1	1	2	200	2	100	2	164	54	20.1	1.00	b/	10.50			1	176	2	346	2	10.6	5

34	emergency hd	2	2	2	2	2	2	2	2	2	1	1	2	1	170	2	90	2	158	105	42.1	3.00	b/	8.50			1	258	2	317	2	9.6	5
35	emergency hd	2	2	2	2	2	2	2	2	2	2	1	2	1	200	2	100	2	170	74	25.6	2.00	b/	13.00		1.00	2	234	2	211	2	6.3	1
36	No	2	2	2	1	2	2	2	2	2	1	1	2	2	100	1	70	1	166	66	24.0	1.00	1r	8.60			1	101	1	162	1	7.6	3
37	emergency hd	2	2	2	1	2	2	2	2	2	2	2	2	2	130	2	70	1	172	74	25.0	2.00	1r	9.60			1	274	2	202	2	6.9	2
38	No	2	2	2	1	2	2	2	2	2	2	2	2	2	160	2	80	2	170	78	27.0	2.00	b/	11.20			1	177	2	152	1	6.7	2
39	emergency hd	1	2	2	1	2	2	2	1	2	1	1	1	2	140	2	70	1	174	62	20.5	1.00	b/	10.80			1	149	2	301	2	9.6	5
40	emergency hd	2	2	2	2	2	2	2	2	2	2	1	1	2	170	2	80	2	162	58	22.1	1.00	b/	10.70			1	106	1	142	1	5.6	1
41	No	1	2	2	1	2	2	2	2	2	1	1	2	2	110	1	70	1	166	70	25.4	2.00	1r	10.80			1	173	2	142	1	6.8	2
42	emergency hd	1	2	2	1	2	2	2	2	1	1	1	2	2	200	2	100	2	166	92	33.4	3.00	b/	7.40			1	195	2	166	1	7.2	2
43	3/week	1	1	1	1	1	1	2	2	2	2	1	2	2	220	2	100	2	169	66	23.1	1.00	b/	11.00			1	103	1	96	1	5.8	1
44	3/week	1	1	2	1	2	1	2	2	2	1	1	2	2	160	2	100	2	178	86	27.1	2.00	b/	9.60			1	205	2	275	2	8.2	3
45	No	1	1	2	1	2	2	2	2	2	2	1	2	2	150	2	90	2	170	68	23.5	1.00	1r	13.20		1.00	2	211	2	186	1	7.8	3
46	emergency hd	1	1	1	1	1	1	2	2	2	1	1	2	2	140	2	90	2	162	66	25.1	2.00	1r	7.90			1	210	2	255	2	8.6	4
47	2/week	1	1	2	1	2	2	1	1	1	2	1	2	2	190	2	90	2	172	81	27.4	2.00	b/	11.40			1	193	2	257	2	7.8	3
48	3/week	1	1	2	1	2	2	1	2	1	2	1	2	2	170	2	90	2	186	92	26.2	2.00	b/	11.70			1	125	1	186	1	6.7	2
49	2/week	1	1	1	1	1	1	2	2	2	2	1	2	2	150	2	80	2	180	76	23.5	1.00	b/	12.10			1	336	2	316	2	11.0	5
50	No	2	2	2	1	2	2	2	2	1	1	1	1	2	120	1	70	1	172	66	22.3	1.00	1r	8.60			1	181	2	196	1	6.5	2
51	3/week	1	1	1	1	1	1	2	2	2	2	2	1	2	140	2	90	2	165	52	19.1	1.00	1r	11.00			1	102	1	86	1	5.6	1
52	No	2	2	2	1	2	2	2	2	2	2	2	2	2	150	2	70	1	158	72	28.8	2.00	1r	12.40			1	481	2	511	2	12.6	5
53	3/week	1	1	1	1	1	1	2	2	2	2	2	1	2	110	1	70	1	168	58	20.5	1.00	1r	10.30			1	121	1	79	1	6.2	1
54	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	120	1	70	1	162	62	23.6	1.00	1r	9.30			1	287	2	151	1	8.4	3
55	No	1	1	2	1	2	1	2	2	2	2	2	2	2	150	2	70	1	176	78	25.2	2.00	1r	13.60	1.00	1.00	2	364	2	265	2	11.0	5
56	emergency hd	1	1	1	1	2	1	2	2	2	2	2	2	2	140	2	100	2	160	80	31.2	3.00	1r	11.90			1	242	2	199	1	7.8	3
57	No	1	1	2	1	2	2	2	2	2	2	1	2	2	150	2	70	1	158	72	28.8	2.00	1r	11.40			1	320	2	282	2	9.6	5
58	No	1	1	1	1	1	1	2	2	2	1	2	1	2	130	2	70	1	156	51	21.0	1.00	1r	8.60			1	136	2	202	2	7.2	2
59	3/week	1	1	1	1	1	1	2	2	2	1	2	2	1	150	2	70	1	166	70	25.4	2.00	b/	7.70			1	116	1	174	1	6.6	2
60	emergency hd	1	1	1	1	1	1	2	2	2	1	1	1	2	110	1	60	1	166	60	21.8	1.00	b/	9.00			1	106	1	137	1	7.6	3
61	No	1	2	2	1	2	2	2	1	2	2	2	2	2	150	2	90	2	180	86	26.5	2.00	1r	13.90	1.00	1.00	2	161	2	322	2	12.0	5
62	No	2	2	2	1	2	2	2	2	2	1	2	2	2	120	1	90	2	172	82	26.5	2.00	1r	9.90			1	172	2	158	1	7.6	3
63	No	2	2	2	1	2	2	1	2	1	2	2	2	2	130	2	60	1	172	62	21.0	1.00	1r	11.00			1	194	2	272	2	8.9	4
64	2/week	1	1	2	1	1	1	2	2	2	1	1	2	2	160	2	90	2	155	62	25.8	2.00	1r	10.50			1	266	2	480	2	8.9	4
65	3/week	1	1	1	1	1	1	2	2	2	1	1	2	2	140	2	90	2	168	72	25.5	2.00	1r	8.50			1	242	2	184	1	9.3	4
66	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	2	130	2	80	2	162	76	29.0	2.00	b/	9.50			1	172	2	200	2	6.4	1
67	emergency hd	2	2	2	1	2	2	2	1	2	1	1	1	2	150	2	70	1	166	58	21.0	1.00	b/	10.10			1	84	1	103	1	6.3	1
68	3/week	1	1	2	1	2	1	1	2	2	1	1	1	2	210	2	110	2	169	76	26.6	2.00	b/	9.10			1	302	2	280	2	12.7	5
69	No	1	1	1	1	1	1	1	1	2	2	1	2	1	120	1	80	2	182	85	25.7	2.00	1r	13.20		1.00	2	77	1	195	1	7.6	3
70	3/week	2	2	2	1	2	2	2	2	2	1	1	2	2	160	2	100	2	176	73	23.6	1.00	1r	12.40			1	178	2	144	1	6.3	1
71	3/week	1	1	2	1	2	2	1	2	2	1	1	2	2	110	1	70	1	172	66	22.3	1.00	1r	11.80			1	106	1	92	1	6.2	1
72	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	2	150	2	60	1	162	56	21.3	1.00	b/	9.90			1	110	1	180	1	6.5	2
73	emergency hd	1	1	2	1	2	1	2	2	2	1	1	1	1	190	2	80	2	162	50	19.1	1.00	b/	8.90			1	186	2	250	2	7.8	3
74	3/week	1	1	1	1	1	1	2	2	2	1	1	2	2	130	2	80	2	172	92	31.1	3.00	b/	8.50			1	86	1	126	1	5.8	1
75	2/week	1	1	1	1	1	1	2	2	2	1	1	2	2	110	1	80	2	180	75	23.1	1.00	1r	10.40			1	131	2	180	1	10.0	5

76	3/week	1	1	2	1	2	2	2	2	1	1	1	1	2	150	2	70	1	172	66	22.3	1.00	b/	11.00			1	215	2	311	2	9.2	4
77	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	130	2	60	1	162	59	22.5	1.00	1r	8.90			1	106	1	142	1	7.8	3
78	3/week	1	1	1	1	1	1	1	1	2	1	1	2	2	130	2	60	1	174	78	25.8	2.00	1r	10.50			1	180	2	242	2	6.9	2
79	3/week	1	1	1	1	1	1	2	2	2	1	1	2	2	160	2	90	2	164	52	19.3	1.00	b/	9.80			1	208	2	311	2	9.8	5
80	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	1	130	2	80	2	170	66	22.8	1.00	b/	8.00			1	236	2	255	2	7.2	2
81	emergency hd	1	1	1	1	1	1	2	2	2	1	1	2	2	130	2	60	1	166	72	26.1	2.00	b/	9.20			1	70	1	126	1	5.6	1
82	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	190	2	80	2	178	69	21.8	1.00	1r	9.90			1	120	1	176	1	6.1	1
83	3/week	1	1	1	1	1	1	2	2	2	1	1	2	2	120	1	70	1	158	74	30.4	3.00	1r	10.80			1	157	2	187	1	10.5	5
84	emergency hd	1	1	2	1	2	2	2	2	2	2	1	2	2	170	2	90	2	169	65	22.8	1.00	b/	11.30			1	136	2	149	1	7.2	2
85	No	2	2	2	2	2	2	1	1	1	2	1	2	2	180	2	90	2	182	78	23.5	1.00	1r	12.40			1	461	2	441	2	11.6	5
86	3/week	1	1	1	1	1	1	2	2	2	1	2	2	2	140	2	90	2	176	80	25.8	2.00	1r	9.06			1	232	2	186	1	7.8	3
87	2/week	1	1	2	1	2	1	2	1	1	1	2	2	2	130	2	80	2	176	80	25.6	2.00	1r	8.00			1	138	2	178	1	6.8	2
88	No	1	1	1	1	1	1	2	2	2	1	1	2	2	200	2	100	2	172	74	25.0	2.00	1r	10.60			1	205	2	229	2	7.6	3
89	2/week	1	1	2	2	2	2	2	2	2	2	1	1	2	160	2	90	2	175	62	20.2	1.00	1r	11.60			1	206	2	289	2	6.9	2
90	emergency hd	1	1	2	1	2	2	2	2	2	1	2	2	2	150	2	100	2	170	90	30.1	3.00	b/	9.60			1	211	2	252	2	10.2	5
91	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	170	2	90	2	166	60	21.8	1.00	1r	9.10			1	176	2	211	2	6.2	1
92	3/week	1	1	1	1	1	1	1	1	2	2	1	1	2	190	2	100	2	166	62	22.5	1.00	1r	10.90			1	233	2	312	2	7.2	2
93	3/week	1	1	1	1	1	1	1	2	2	1	1	1	2	100	1	70	1	160	55	21.5	1.00	1r	9.90			1	132	2	176	1	5.6	1
94	No	1	1	2	1	2	2	2	2	2	2	2	2	2	170	2	90	2	180	92	28.4	2.00	b/	12.60		1.00	2	326	2	333	2	9.2	4
95	No	2	2	2	1	2	1	2	2	2	1	1	2	2	110	1	70	1	162	72	27.4	2.00	1r	8.60			1	83	1	142	1	7.6	3
96	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	2	130	2	60	1	167	80	28.7	2.00	b/	8.40			1	174	2	256	2	7.9	3
97	emergency hd	1	1	1	1	2	2	2	2	2	1	1	2	1	130	2	60	1	168	76	26.9	2.00	b/	8.40			1	120	1	159	1	5.9	1
98	emergency hd	1	1	2	1	2	2	2	2	2	1	1	2	2	140	2	90	2	176	66	21.3	1.00	b/	8.90			1	136	2	142	1	6.1	1
99	emergency hd	1	2	2	1	2	2	2	2	2	2	1	2	1	140	2	80	2	172	81	27.4	2.00	b/	13.40		1.00	2	296	2	245	2	7.9	3
100	3/week	1	1	1	1	2	1	2	2	2	1	1	1	2	180	2	100	2	162	50	19.1	1.00	1r	8.90			1	142	2	156	1	6.2	1
101	emergency hd	1	2	2	1	2	2	2	2	2	1	1	2	2	170	2	80	2	156	65	26.7	2.00	b/	9.90			1	197	2	339	2	8.6	4
102	No	1	1	2	1	2	1	2	2	2	2	2	2	2	100	1	70	1	172	68	23.0	1.00	1r	11.90			1	145	2	202	2	7.4	2
103	No	2	2	2	1	2	2	2	2	2	2	2	2	2	140	2	90	2	176	74	23.9	1.00	1r	13.90	1.00	1.00	2	183	2	166	1	12.1	5
104	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	130	2	60	1	170	60	20.8	1.00	1r	10.10			1	226	2	158	1	6.6	2
105	No	1	1	1	1	1	1	2	2	2	1	1	2	2	160	2	80	2	166	66	24.0	1.00	b/	9.50			1	257	2	211	2	9.4	4
106	3/week	1	1	1	1	1	1	2	2	2	2	1	1	2	130	2	80	2	172	61	20.6	1.00	1r	12.50		1.00	2	274	2	177	1	8.3	3
107	2/week	1	1	1	1	1	1	2	2	2	1	1	2	2	160	2	100	2	160	72	28.1	2.00	1r	10.50			1	140	2	156	1	6.8	2
108	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	2	100	1	70	1	168	55	19.5	1.00	b/	8.80			1	95	1	106	1	13.9	5
109	emergency hd	2	2	2	1	2	2	2	2	2	1	1	2	2	170	2	100	2	172	84	28.4	2.00	b/	9.80			1	108	1	127	1	10.4	5
110	No	1	1	1	1	1	1	2	2	2	1	1	1	2	170	2	100	2	168	59	20.9	1.00	1r	10.20			1	202	2	166	1	7.6	3
111	No	1	1	1	1	1	1	2	2	2	2	2	2	2	140	2	70	1	160	66	25.6	2.00	1r	10.20			1	112	1	196	1	8.2	3
112	3/week	1	1	1	1	2	2	2	2	2	2	2	2	2	110	1	80	2	174	74	24.2	1.00	1r	12.20			1	198	2	224	2	8.9	4
113	emergency hd	1	1	2	2	2	2	1	2	1	1	1	2	2	180	2	110	2	176	78	25.2	2.00	1r	9.10			1	210	2	188	1	8.1	3
114	No	2	2	2	1	2	2	2	2	1	1	1	2	2	150	2	90	2	162	71	27.1	2.00	1r	8.70			1	94	1	122	1	6.2	1
115	No	1	1	2	1	2	1	2	2	1	2	2	1	2	100	1	70	1	165	69	25.3	2.00	1r	10.80			1	91	1	112	1	5.2	1
116	emergency hd	2	2	2	1	2	2	1	1	2	2	2	2	2	160	2	100	2	160	66	25.6	2.00	1r	8.90			1	542	2	441	2	15.6	5
117	No	1	2	2	1	2	2	2	2	2	2	2	2	2	100	1	70	1	172	86	29.1	2.00	1r	11.90			1	236	2	188	1	8.2	3

118	No	2	2	2	2	2	2	1	1	1	2	1	2	2	210	2	120	2	169	71	24.9	1.00	1r	12.40			1	219	2	255	2	7.6	3
119	3/week	1	1	2	1	2	1	2	2	2	1	1	2	2	140	2	90	2	166	61	22.1	1.00	b/	8.40			1	211	2	301	2	8.9	4
120	3/week	1	1	1	1	1	1	2	2	2	1	1	1	2	170	2	80	2	162	49	18.7	1.00	1r	9.80			1	102	1	82	1	5.2	1

CREATININE	UREA	SODIUM	POTASSIUM	CALCIUM	PHOSPHOROUS	URINE PROTIEN	URINE GLUCOSE	MDRD ml min	CKDEPI ml min	GFR	eGFR cat	GFR cat	URINE ALBUMIN CREATININE RATIO	Albuminuria	Normal Proteinuria	MICROALBUMINURIA	MACROALBUMINURIA	Kidney size RIGHT	Kidney size LEFT	MILD NPDR	MODERATE NPDR	SEVERE NPDR	PDR	HYPERTENSIVE RETINOPATHY	ECG FINDINGS Normal	ECG FINDINGS Abnormal	Hypertensive medications	Microalbuminurea1	Macroalbuminuria1	Urine output	UG abdomen	USG KUB PARENCHYMAL CHANGES
12.22	140	135	4.08	7.9	8.9	3	4	4.7	4	4.35	6.00	1	2464	3	2	2	1	9*6	9.2*5	2	1	2	2	no	1		1	2	1	1	4	grade 3 rpc
3.35	104	139	5.16	8.1	10.1	2	1	14.7	14	14.35	5.00	1	1792	3	2	2	1	10*4	10.2*4	2	2	1	2	no	1		1	2	1	1	2	grade 1 rpc
4.85	169	132	4.62	7.6	9.5	3	1	9.1	8	8.55	6.00	1	2922	3	2	2	1	10.3*3	10*4.1	1	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
3.56	92	117	6.27	8.7	6.2	3	1	18.7	18	18.35	5.00	1	988	3	2	2	1	10.7*4.3	10.5*4	2	2	2	2	no	1		1	2	1	2	1	Normal
13.15	173	132	4.74	6.3	8.1	2	1	4.1	4	4.05	6.00	1	10824	3	2	2	1	9.8*4.2	9.5*4.1	1	2	2	2	no	1		1	2	1	1	3	grade 2 rpc
8.81	140	136	4.50	12.2	4.1	2	2	6.6	6	6.30	6.00	1	6489	3	2	2	1	10*3.3	9.6*3.7	2	1	2	2	no	1		1	2	1	2	3	grade 2 rpc
5.89	125	140	5.57	7.8	7.7	2	4	9.0	10	9.50	6.00	1	2540	3	2	2	1	9.3*4	9.6*4.2	2	1	2	2	no	1		1	2	1	2	3	grade 2 rpc
4.90	82	131	7.04	8.2	8.0	2	2	9.4	9	9.20	6.00	1	8642	3	2	2	1	9*4	9.6*4.5	2	2	1	2	no	1		1	2	1	2	2	grade 1 rpc
9.03	140	141	4.76	9.3	8.2	1	6	4.6	4	4.30	6.00	1	212	2	2	1	2	8.2*3.4	8.6*3	2	2	2	2	no	1		1	1	2	1	3	grade 2 rpc
5.94	106	141	5.16	6.6	5.2	2	1	10.0	9.5	9.75	6.00	1	1126	3	2	2	1	10.4*4.6	11*4.3	1	2	2	2	no	1		1	2	1	2	1	Normal
7.96	109	141	5.95	9.0	7.0	3	1	7.2	6	6.60	6.00	1	542	3	2	2	1	8.8*4.1	9.8*4	1	2	2	2	no	1		1	2	1	1	3	grade 2 rpc
2.74	66	134	4.46	8.6	3.5	2	2	19.1	19	19.05	5.00	1	254	2	2	1	2	11*4	10.6*5	1	2	2	2	no	1		1	1	2	2	1	Normal
11.84	154	134	5.34	9.4	4.4	3	4	4.1	4	4.05	6.00	1	1755	3	2	2	1	9.5*4.6	10.1*4.1	2	2	1	2	no	1		1	2	1	1	3	grade 2 rpc
5.53	94	138	5.39	7.0	7.3	4	3	8.2	8	8.10	6.00	1	5260	3	2	2	1	10.3*4	11*4	1	2	2	2	no	1		1	2	1	2	3	grade 2 rpc
5.22	90	138	5.45	7.4	2.0	1	2	11.9	11	11.45	6.00	1	372	3	2	2	1	11.2*4.7	11.7*4.8	1	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
2.20	39	140	4.58	9.9	3.0	1	5	24.0	23	23.50	5.00	1	176	2	2	1	2	12.4*4.2	12.7*4.3	2	2	2	2	no	1		1	1	2	2	1	Normal
1.70	29	135	4.26	10.5	3.7	2	6	44.7	44	44.35	3.00	1	137	2	2	1	2	11.9*4.7	11.6*4.4	2	2	2	2	no	1		1	1	2	2	1	Normal
1.31	35	128	5.10	9.0	4.0	2	4	43.6	43	43.30	4.00	1	1212	3	2	2	1	12.3*4.7	12.6*5	2	2	2	1	grade 1	1		1	2	1	2	1	Normal
6.12	126	138	4.64	9.3	2.7	1	2	9.6	9	9.30	6.00	1	3236	3	2	2	1	8.8*4.7	7.8*4	2	1	2	2	no	1		1	2	1	2	2	grade 1 rpc

1.30	37	134	3.76	8.9	4.2	2	1	53.6	53	53.30	3.00	1	5087	3	2	2	1	10*3.9	10.3*4	2	2	2	2	no	1		1	2	1	2	1	Normal
1.76	42	148	3.47	10.2	4.0	1	4	43.3	43	43.15	4.00	1	1314	3	2	2	1	10.5*3.9	11*4	2	2	2	2	no	2	LVH	1	2	1	2	1	Normal
4.29	106	127	4.83	8.0	5.1	1	2	10.8	9	9.90	6.00	1	998	3	2	2	1	9.2*3.4	9.6*4.6	2	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
4.56	115	138	5.36	8.4	4.7	1	5	13.3	11	12.15	6.00	1	10722	3	2	2	1	9*4	9.8*4.5	1	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
3.91	84	120	6.46	7.0	6.2	2	4	17.0	16	16.50	5.00	1	704	3	2	2	1	9*3.7	9.8*4.2	1	2	2	2	grade 2	2	LVH	1	2	1	2	2	grade 1 rpc
1.46	36	135	4.57	10.4	2.9	2	2	50.0	46	48.00	3.00	1	491	3	2	2	1	10*5	10.4*4.7	1	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
3.19	98	132	4.39	10.6	3.4	5	5	20.6	19	19.80	5.00	1	1591	3	2	2	1	11*4.2	10.9*4.9	2	2	2	2	no	1		2	2	1	2	1	Normal
1.70	36	136	4.20	8.9	4.1	6	5	43.6	42	42.80	4.00	1	21	1	1	2	2	11.8*4.2	12.9*4.3	2	2	2	2	no	1		1	2	2	1	1	Normal
6.20	130	132	5.58	7.6	5.2	4	1	9.6	8	8.80	6.00	1	4252	3	2	2	1	8.7*4	9.1*4	2	2	1	2	no	1		1	2	1	1	3	grade 2 rpc
5.52	112	133	4.25	8.2	4.6	1	5	10.9	10	10.45	6.00	1	533	3	2	2	1	9.2*3.9	9.1*4.1	2	2	2	2	grade 2	1		1	2	1	1	2	grade 1 rpc
6.34	138	129	4.99	8.4	5.9	1	1	9.2	8	8.60	6.00	1	825	3	2	2	1	8.9*3.4	9.4*3.9	2	1	2	2	grade 2	1		1	2	1	2	3	grade 2 rpc
3.33	44	142	3.85	8.1	3.2	3	4	20.2	19	19.60	5.00	1	31454	3	2	2	1	11.6*4.3	11.9*4	2	1	2	2	no	1		1	2	1	1	1	Normal
1.77	27	139	4.65	9.2	4.1	3	4	40.9	39	39.95	4.00	1	4056	3	2	2	1	10*4.5	9.4*4.9	1	2	2	2	no	1		1	2	1	2	1	Normal
8.90	128	132	5.68	8.8	5.0	3	3	6.5	6	6.25	6.00	1	13882	3	2	2	1	8.2*3.4	8.9*3.4	2	2	2	2	no	1		1	2	1	1	4	grade 3 rpc
9.73	120	119	5.90	7.7	3.6	3	1	4.0	4	4.00	6.00	1	9721	3	2	2	1	9.7*3.7	9.9*4.1	2	1	2	2	no	2	low voltage	1	2	1	1	3	grade 2 rpc
4.50	54	141	5.66	8.0	5.9	1	4	14.4	13	13.70	6.00	1	3413	3	2	2	1	8*3.2	8.2*2.9	1	2	2	2	no	1		1	2	1	1	4	grade 3 rpc
1.80	72	142	4.15	9.4	3.5	3	3	30.9	31	30.95	4.00	1	4236	3	2	2	1	8.4*4.3	9*4.3	2	1	2	2	no	1		1	2	1	2	2	grade 1 rpc
6.39	99	134	5.90	8.2	4.9	2	3	9.4	8	8.70	6.00	1	10803	3	2	2	1	8.6*3.4	9*3.9	2	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
3.94	98	134	4.36	9.5	3.6	2	2	15.9	14	14.95	5.00	1	3211	3	2	2	1	9.8*4.6	10.2*5	2	2	2	2	no	1		1	2	1	2	1	Normal
9.90	133	134	5.85	8.6	6.0	3	4	5.6	5	5.30	6.00	1	1290	3	2	2	1	9.1*4	9.5*4.3	2	1	2	2	no	1		1	2	1	1	3	grade 2 rpc
7.47	148	136	5.86	9.4	6.0	3	2	5.0	5.5	5.25	6.00	1	3203	3	2	2	1	8*3.2	8.5*3.5	2	2	1	2	no	1		1	2	1	1	4	grade 3 rpc
2.53	79	139	4.32	8.6	3.2	1	1	20.3	19	19.65	5.00	1	256	2	2	1	2	11.2*5	11.5*5.2	2	2	2	2	no	1		1	1	2	1	1	Normal
6.93	143	144	6.05	8.0	5.2	1	6	6.2	6	6.10	6.00	1	3408	3	2	2	1	10*6	10*5.5	1	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
7.20	112	138	5.45	8.9	7.8	7	7	8.3	7	7.65	6.00	1			3	3	3	8.5*3.2	8.9*4	2	2	2	2	no	1		1	3	3	1	4	grade 3 rpc
8.57	65	140	4.15	8.1	4.1	3	4	6.8	6	6.40	6.00	1	9241	3	2	2	1	9.3*4.6	9.9*5	2	1	2	2	no	1		1	2	1	1	2	grade 1 rpc
3.71	30	136	4.98	9.5	3.5	4	4	17.0	18	17.50	5.00	1	8325	3	2	2	1	11.2*4.5	11.6*4.9	2	2	2	2	no	1		1	2	1	2	1	Normal
5.00	48	140	5.25	8.4	2.6	2	2	9.1	8	8.55	6.00	1	1168	3	2	2	1	10.9*4.5	11*4.5	2	2	2	2	no	1		1	2	1	2	1	Normal
5.28	136	144	5.75	7.8	4.3	2	1	11.6	10	10.80	6.00	1	1422	3	2	2	1	11*4.2	11.5*4.1	1	2	2	2	no	1		1	2	1	2	1	Normal
10.51	92	133	4.21	8.2	4.2	2	2	5.4	5	5.20	6.00	1	2332	3	2	2	1	9.3*4.2	9.7*4.0	2	1	2	2	grade 1	1		1	2	1	2	3	grade 2 rpc
8.70	79	133	5.36	9.2	6.3	4	3	6.5	6	6.25	6.00	1	1241	3	2	2	1	10.4*4.9	11*4.3	1	2	2	2	no	1		1	2	1	1	2	grade1 rpc
2.55	53	142	4.25	9.5	3.5	1	1	27.1	25	26.05	5.00	1	208	2	2	1	2	11*4.6	12.1*4.5	2	2	2	2	no	1		1	1	2	2	1	Normal

7.17	138	139	4.95	8.6	6.0	7	7	7.7	6	6.85	6.00	1			3	3	3	9*5	9*4	1	2	2	2	no	1		1	3	3	1	3	grade 2 rpc
1.20	58	129	4.89	8.3	3.1	2	3	46.5	44	45.25	3.00	1	1485	3	2	2	1	11.4*4.2	11*4.3	2	2	2	2	no	1		1	2	1	2	1	Normal
7.25	106	135	4.15	8.0	5.9	7	7	7.9	7	7.45	6.00	1			3	3	3	9.4*3.6	9.5*3.4	2	1	2	2	no	1		1	3	3	1	3	grade 2 rpc
6.57	95	132	6.39	7.7	4.1	3	1	6.8	6	6.40	6.00	1	2319	3	2	2	1	9.2*4	9.5*3.7	2	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
1.81	49	133	5.05	8.6	3.2	2	4	40.6	39	39.80	4.00	1	1472	3	2	2	1	11*4.9	12.6*5.2	2	2	2	2	no	1		1	2	1	2	1	Normal
6.52	111	138	4.37	8.7	4.9	2	3	6.8	6	6.40	6.00	1	2381	3	2	2	1	10*4.4	9.8*3.6	2	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
2.70	30	136	4.25	9.6	3.5	2	2	19.6	19	19.30	5.00	1	2842	3	2	2	1	11.6*5	12*4.9	1	2	2	2	no	1		1	2	1	2	1	Normal
1.80	42	133	4.50	9.0	4.2	1	1	38.6	34	36.30	4.00	1	251	2	2	1	2	10*4	9.6*4.2	2	2	2	2	grade 2	1		1	1	2	1	2	grade 1 rpc
6.49	92	124	6.30	7.9	6.0	3	5	9.1	8	8.55	6.00	1	555	3	2	2	1	8.9*4	9.2*4.1	1	2	2	2	no	1		1	2	1	2	3	grade 2 rpc
7.88	194	136	5.68	10.1	3.0	5	6	7.1	6	6.55	6.00	1	47	2	2	1	2	10*4.2	10.8*4.4	2	1	2	2	no	1		1	1	2	2	3	grade 2 rpc
1.50	35	140	4.30	9.9	3.2	3	3	53.1	54	53.55	3.00	1	1470	3	2	2	1	11*5.2	11.6*5.3	2	2	2	2	no	1		1	2	1	2	1	Normal
2.49	62	136	3.78	9.2	2.5	3	6	28.3	27	27.65	5.00	1	9432	3	2	2	1	11.1*4.2	12*4.7	1	2	2	2	no	1		1	2	1	2	1	Normal
2.20	40	128	3.60	7.5	8.7	4	1	32.3	31	31.65	4.00	1	12853	3	2	2	1	12*4.9	11.6*4.6	2	2	2	2	no	1		1	2	1	2	1	Normal
4.70	39	136	6.70	9.1	5.4	1	2	10.6	10	10.30	6.00	1	1821	3	2	2	1	8.9*4	9*4.7	2	1	2	2	no	1		1	2	1	1	2	grade 1 rpc
16.36	156	136	5.72	7.7	8.0	2	3	3.3	3	3.15	6.00	1	1239	3	2	2	1	8.3*3.9	7.6*3.2	2	1	2	2	no	1		1	2	1	2	4	grade 3 rpc
6.13	96	144	4.93	8.6	5.3	2	6	7.4	7	7.20	6.00	1	972	3	2	2	1	10.2*4.2	11*4.6	2	1	2	2	no	1		1	2	1	1	2	grade 1 rpc
10.36	112	136	4.79	7.0	3.0	2	5	5.4	5	5.20	6.00	1	1762	3	2	2	2	9.9*4.2	9.4*4	1	2	2	2	no	1		1	2	2	1	2	grade 1 rpc
10.11	94	136	7.20	8.0	4.1	7	7	5.5	5	5.25	6.00	1			3	3	3	8.9*4	9.5*4.2	2	2	1	2	no	1		1	3	3	1	3	grade 2 rpc
1.72	37	144	4.20	9.3	3.7	2	6	42.0	39	40.50	4.00	1	172	2	2	1	2	11*4.8	12*4.9	2	2	2	2	no	1		1	1	2	2	1	Normal
7.80	86	140	4.63	8.9	4.9	2	2	8.0	7.8	7.90	6.00	1	3726	3	2	2	1	10*4.3	10.6*4.9	1	2	2	2	no	1		2	2	1	2	2	grade 1 rpc
3.20	30	137	5.20	7.8	4.6	1	6	20.5	19	19.75	5.00	1	162	2	2	1	2	8.2*4	8.5*3.6	1	2	2	2	no	1		1	1	2	1	4	grade 3 rpc
5.60	122	134	5.90	7.7	6.4	4	5	8.1	7	7.55	6.00	1	9788	3	2	2	1	9.2*4	8.7*5.4	1	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
7.10	61	136	5.80	7.9	3.5	1	1	7.5	7	7.25	6.00	1	290	2	2	1	2	8.6*3.5	8.9*4	1	2	2	2	no	1		1	1	2	1	2	grade 1 rpc
4.39	36	131	3.65	8.6	3.2	1	5	15.0	14	14.50	5.00	1	256	2	2	1	2	9.8*4.2	10.4*4.6	2	1	2	2	no	1		1	1	2	2	1	Normal
4.29	146	133	5.22	8.8	7.7	5	6	14.5	13	13.75	6.00	1	76	2	2	1	2	10*5.2	10*5.4	1	2	2	2	no	1		1	1	2	2	2	grade 1 rpc
13.10	98	135	5.90	9.2	6.6	2	1	4.4	4	4.20	6.00	1	10721	3	2	2	1	8*3	8.7*3.9	2	2	2	2	no	1		1	2	1	2	3	grade 2 rpc
9.20	110	142	5.50	8.1	5.4	1	2	6.0	5	5.50	6.00	1	662	3	2	2	1	9.2*4.5	9.5*4.2	1	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
9.20	78	139	4.90	8.4	5.9	2	3	6.4	6	6.20	6.00	1	1254	3	2	2	1	8.9*4.2	9.2*4	2	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
10.81	98	132	5.90	7.9	6.8	7	7	5.0	4	4.50	6.00	1			3	3	3	8.1*3.6	8.5*4	2	2	2	2	no	1		1	3	3	1	3	grade 2

																															rpc
6.60	142	123	4.99	7.8	2.1	2	1	7.0	7	7.00	6.00	1	4341	3	2	2	1	9.6*4	10.3*4.9	2	2	2	2	no	1		1	2	1	2	3	grade 2 rpc
4.30	99	135	4.80	8.5	4.2	6	6	11.2	11	11.10	6.00	1	270	2	2	1	2	10*5	11*5.2	2	2	1	2	grade 1	2	lvh changes	1	1	2	1	1	Normal
5.70	46	133	4.50	8.9	5.6	5	6	10.5	10	10.25	6.00	1	15	1	2	2	2	9*4.5	9.5*4.9	2	1	2	2	no	1		1	2	2	2	2	grade 1 rpc
14.50	102	134	5.23	8.5	6.5	3	1	2.8	3	2.90	6.00	1	3231	3	2	2	1	9.6*4.2	9.9*4.9	1	2	2	2	no	1		1	2	1	2	3	grade 2 rpc
13.30	209	131	4.40	7.6	6.6	3	1	4.1	4	4.05	6.00	1	1445	3	2	2	1	9.8*4.1	9.5*4.5	2	1	2	2	grade 2	2	lvh changes	1	2	1	1	2	grade 1 rpc
2.46	56	134	5.20	8.8	3.1	4	4	30.1	30	30.05	4.00	1	2081	3	2	2	2	10.4*4.5	10.9*4.2	2	2	2	2	no	1		2	2	2	2	1	Normal
9.06	89	138	4.81	8.0	3.1	3	2	6.4	6	6.20	6.00	1	1024	3	2	2	1	10.1*4.1	10.5*3.9	1	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
6.13	49	140	5.16	7.9	4.3	1	2	10.0	10.5	10.25	6.00	1	782	3	2	2	1	9.7*4.5	9.9*4.2	2	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
2.20	59	139	6.40	8.3	3.6	1	1	31.3	28	29.65	5.00	1	206	2	2	1	2	9.9*4.6	9.4*4	2	2	2	2	no	1		1	1	2	2	1	Normal
7.40	95	135	4.95	7.9	4.2	1	1	7.7	7	7.35	6.00	1	2312	3	2	2	1	8.4*3	8.1*3.5	2	2	2	1	no	1		1	2	1	1	4	grade 3 rpc
6.99	82	138	5.36	6.2	3.2	3	1	9.6	9	9.30	6.00	1	531	3	2	2	1	7*4	7.5*4.5	1	2	2	2	no	1		1	2	1	1	3	grade 2 rpc
13.20	103	140	5.50	7.9	4.4	5	1	4.0	3	3.50	6.00	1	2312	3	2	2	1	9.2*4.1	9.4*4.8	1	2	2	2	no	1		1	2	1	1	2	grade1 rpc
13.20	96	133	5.20	7.5	4.9	2	2	3.2	3	3.10	6.00	1	1012	3	2	2	1	9.8*4.2	9.5*4	2	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
5.33	48	132	4.80	8.3	4.2	3	6	11.2	10	10.60	6.00	1	3231	3	2	2	1	8.9*4.5	9.1*4.2	2	1	2	2	no	1		1	2	1	1	2	grade1 rpc
1.30	45	140	4.75	9.6	3.2	2	3	65.0	68	66.50	2.00	2	232	2	2	1	2	11.2*4.2	10.9*4	2	2	2	2	no	1		1	1	2	2	1	Normal
2.18	63	139	4.63	8.6	5.0	2	5	23.7	22	22.85	5.00	1	6827	3	2	2	1	8.5*3.7	7*4.3	2	2	2	2	no	1		1	2	1	2	2	grade1 rpc
7.90	89	123	4.95	7.9	4.9	1	2	5.3	5	5.15	6.00	1	3719	3	2	2	1	8.9*3.5	9.2*3	2	2	2	1	no	1		1	2	1	2	3	grade 2 rpc
8.76	166	139	5.51	8.4	4.4	3	5	6.4	6	6.20	6.00	1	4120	3	2	2	1	8.5*3.3	8.3*4	1	2	2	2	no	1		1	2	1	1	4	grade 3 rpc
14.60	202	135	4.00	7.6	5.8	2	5	3.6	3	3.30	6.00	1	899	3	2	2	1	7.9*4.1	8.3*4.1	2	2	1	2	no	1		1	2	1	1	4	grade 3 rpc
6.22	39	138	6.20	9.3	2.3	2	2	9.8	9	9.40	6.00	1	2662	3	2	2	1	9.2*4.3	9.3*4.1	2	2	1	2	grade 1	1		1	2	1	1	3	grade 2 rpc
4.15	103	139	3.36	7.0	4.2	1	2	16.0	15	15.50	5.00	1	3362	3	2	2	1	8.2*3.6	8.9*4	1	2	2	2	no	1		1	2	1	1	4	grade 3 rpc
5.40	56	140	4.50	7.5	4.2	1	3	8.7	8	8.35	6.00	1	92	2	2	1	2	9.5*4.1	9.2*3.9	1	2	2	2	no	1		1	1	2	1	2	grade 1 rpc
3.65	74	142	5.16	9.3	4.5	1	6	17.6	16	16.80	5.00	1	952	3	2	2	1	10.8*4	9.8*4.2	2	2	2	2	no	1		1	2	1	2	2	grade1 rpc
2.23	39	135	5.54	8.6	4.0	1	3	32.8	32	32.40	4.00	1	3417	3	2	2	1	11.2*4.2	11*4	2	2	2	2	no	1		1	2	1	2	1	Normal
8.72	190	137	4.05	8.5	4.0	3	2	6.5	6	6.25	6.00	1	1556	3	2	2	1	8*3.5	8*4.5	1	2	2	2	no	1		1	2	1	1	4	grade 3 rpc
3.82	75	142	3.90	9.9	2.1	3	3	12.7	12	12.35	6.00	1	1897	3	2	2	1	8.7*4.2	9.3*4.5	1	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
6.56	127	144	5.41	8.4	4.8	7	7	8.8	7	7.90	6.00	1			3	3	3	8*3.2	8.1*3.5	2	2	1	2	no	1		1	3	3	1	4	grade 3 rpc

5.51	142	138	4.20	8.4	4.1	2	6	8.0	7	7.50	6.00	1	244	2	2	1	2	9.2*4.1	9.4*3.5	2	1	2	2	no	1		1	1	2	1	2	grade 1 rpc
7.16	100	138	3.35	7.5	1.7	2	6	8.8	8	8.40	6.00	1	1713	3	2	2	1	9.9*4.2	9.7*4.1	1	2	2	2	no	1		1	2	1	2	2	grade 1 rpc
11.29	146	133	5.68	9.9	1.8	2	6	4.9	4	4.45	6.00	1	2714	3	2	2	1	8*3.4	8.3*3.1	2	1	2	2	no	1		1	2	1	2	4	grade 3 rpc
2.10	74	126	5.08	9.2	3.0	1	1	31.9	27	29.45	4.00	1	170	2	2	1	2	8.9*4.6	8.7*4.2	2	2	2	2	no	1		1	1	2	2	2	grade 1 rpc
4.02	29	129	3.90	10.4	4.9	7	7	16.2	15	15.60	5.00	1			3	3	3	11.3*4.9	12.1*4.7	2	2	2	2	no	1		1	3	3	2	1	Normal
6.30	45	136	4.50	7.6	5.6	3	6	9.6	9	9.30	6.00	1	221	2	2	1	2	10.6*4.2	9.1*4	2	2	2	2	no	1		1	1	2	1	3	grade 2 rpc
9.90	97	140	4.60	7.2	5.9	3	3	6.0	6	6.00	6.00	1	985	3	2	2	1	9.32*4.1	9.7*4.5	2	2	2	2	no	1		1	2	1	1	1	Normal
5.80	77	139	4.60	8.3	2.1	1	6	8.1	8	8.05	6.00	1	9413	3	2	2	1	9*4.3	9.7*3.9	2	1	2	2	no	1		1	2	1	2	2	grade 1 rpc
3.80	83	137	5.16	10.0	3.2	5	6	16.3	14	15.15	5.00	1	252	2	2	1	2	8.8*5.1	9.9*5.1	1	2	2	2	no	1		1	1	2	2	2	grade 1 rpc
8.80	114	126	6.20	8.6	3.2	3	1	7.2	7	7.10	6.00	1	422	3	2	2	1	9.5*4.2	9.4*4	2	2	2	2	no	1		1	2	1	1	2	grade 1 rpc
2.50	56	134	4.30	9.2	4.0	1	2	29.5	29	29.25	5.00	1	133	2	2	1	2	11*4	11*4.6	2	2	2	2	no	1		1	1	2	2	1	Normal
3.78	57	132	3.74	8.6	3.9	7	7	18.4	18	18.20	5.00	1			3	3	3	9.4*4.2	8.6*4.1	2	2	2	1	no	1		1	3	3	2	2	grade 1 rpc
14.75	112	132	5.60	8.1	4.9	2	2	3.8	3	3.40	6.00	1	2257	3	2	2	1	8.9*4	8.5*3.4	2	1	2	2	no	1		1	2	1	1	3	grade 2 rpc
6.40	38	135	5.43	7.8	6.3	7	7	8.9	8	8.45	6.00	1			3	3	3	8.2*3.9	8.5*4.1	2	2	1	2	grade 3	1		1	3	3	1	4	grade 3 rpc