"A STUDY ON PREVALENCE AND RISK FACTORS OF DIABETIC NEPHROPATHY IN NEWLY DETECTED TYPE 2 DIABETES MELLITUS PATIENTS"

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BRANCH-1

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CHENGALPATTU-603001.

MAY-2020

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON PREVALENCE AND RISK FACTORS OF DIABETIC NEPHROPATHY IN NEWLY DETECTED TYPE 2 DIABETES MELLITUS PATIENTS"** is the bonafide original work of **Dr. SIDDHARTHAN.R** in partial fulfilment of the requirements for **M.D. Branch-1 (General Medicine)** Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in May 2020. The period of study was from June 2018 to May 2019.

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This is to certify that dissertation **"A STUDY ON PREVALENCE AND RISK FACTORS OF DIABETIC NEPHROPATHY IN NEWLY DETECTED TYPE 2 DIABETES MELLITUS PATIENTS"** is a bonafide work performed by **Dr. SIDDHARTHAN.R** , postgraduate student of General medicine, Chengalpattu medical college, Chengalpattu, under my guidance and supervision in fulfilment of regulations of The Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree during the Academic period 2017-2020.

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DECLARATION

I, Dr. SIDDHATHAN.R, solemnly declare that dissertation titled **"A STUDY ON PREVALENCE AND RISK FACTORS OF DIABETIC NEPHROPATHY IN NEWLY DETECTED TYPE 2 DIABETES MELLITUS PATIENTS"** is a bonafide record of work done by me in the Department of Internal Medicine, Government Chengalpattu Medical College and Hospital during June 2018 to May 2019 under the guidance of **Prof. Dr. B. PRABAKAR, M.D.,** Professor of General Medicine, Government Chengalpattu Medical College and Hospital, Chengalpattu. This dissertation is submitted to Tamil Nadu Dr. M.G.R. Medical University, in partial fulfilment of the University regulations for the award of M.D. Degree (Branch 1) General Medicine- May 2020.

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INSTITUTIONAL ETHICAL COMMITTEE

CHENGALPATTU MEDICAL COLLEGE, CHENGALPATTU

Title of Work	:	A study on prevalence and risk factors of Diabetic Nephropathy in newly detected type 2 Diabetic patients.
Principal Investigator	:	Dr.Siddharthan.R
Designation	:	1st yr PG
Co-Investigators	:	Dr.B.Prabakar,M.D., Associate Professor, Department of General Medicine
Department	:	General Medicine

The request for an approval From the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 27.03.2018 at the Medical Education Unit, Government Chengalpattu Medical College, Chengalpattu at 11.00 AM.

The Members of the committee, the Secretary and the Chairman are pleased to inform you that your proposed project mentioned above is approved.

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CONTENTS

INTRODUCTION

Diabetes mellitus is a major health problem and causes considerable morbidity and mortality, primarily due to its microvascular and macro vascular complications. It is a metabolic cum vascular disorder. India is predicted to be capital of diabetes. There are about estimated 70 million patients with diabetes in India and this number is projected to explode beyond 100 million by 2030 [1].

Diabetes has become the most common cause of chronic Kidney Disease (CKD) in most countries including countries like India [1]. 20 to 30% of all patients with diabetes will have evidence of diabetic kidney disease after a period of 15 to 20 years with diabetes [5]. There are many interventions available to prevent the progression of diabetic nephropathy but these interventions must be initiated as early as possible for it to be effective.

After 20 years of diagnosis in patients with type 2 diabetes mellitus, the cumulative incidence of nephropathy is about 25% [5]. Among them 5-10% of diabetic nephropathy will present at the time of diagnosis that is when they are newly detected with type 2 diabetes mellitus [5]. This is because these patients remain symptom free for long periods before they are diagnosed clinically.

Nephropathy is a major cause of morbidity and mortality in patients with diabetes mellitus. Persistent albuminuria is the hallmark of diabetic nephropathy. According to Mogensen staging system, diabetic nephropathy consists of five stages

which include microalbuminuria as stage 3 known as incipient nephropathy [18]. Control of Microalbuminuria in patients with Type 2 DM is an important indicator for renal and cardiovascular risk reduction [11].

The most important risk factor for development of diabetic nephropathy is poor glycaemic control. Inpatients with poor glycaemic control, uncontrolled Hypertension may predispose them further as was noted by studies that showed those with HbA1C >12 % and uncontrolled hypertension were at higher risk for developing nephropathy when followed up for 20 years [3]. Obesity, smoking, age, gender, dyslipidemia, degree of proteinuria at diagnosis, family history of diabetes and kidney diseases have also been suggested as possible contributing factors. Individuals who develop type 2 DM after the age of 50 years are considered more prone for nephropathy, so are family history of hypertension and cardiovascular events in first degree relatives.

Primary prevention of diabetic nephropathy is possible with vigorous glucose and blood pressure control. Screening for diabetic nephropathy falls within scope of secondary prevention. Standard recommendation such as by ADA is available to screen for microalbuminuria in Type 1 Diabetes mellitus who are having the disease for more than five years and with Type 2 Diabetes mellitus at the stage of diagnosis itself [6].

Diabetes is preventable and so are its complications. It is predicted that End Stage Renal Disease (ESRD) occurs in 20% of type 2 Diabetes patients during their lifetime [15]. Detecting the patients in the early stage of nephropathy and thereby timely intervention prevents as well as retards the progression towards End Stage Renal Disease.

AIM

- This Study aims to determine the Prevalence of Diabetic Nephropathy in Newly detected Type 2 Diabetic patients above 30 years of age in a tertiary care Centre.

- This Study aims to analyze the risk factors associated with the development of Diabetic Nephropathy and their significance.

REVIEW OF LITERATURE

Diabetes is the most common single disease causing end stage renal disease (ESRD) in both Type 1 and Type 2 DM [1]. About 20 – 30% of patients with Type 1 and Type 2 diabetes develop evidence of Nephropathy [5], but in Type 2 diabetes, a considerably smaller fraction of them progress to ESRD. Native Americans, Hispanics and Afro-Americans have much risk of developing ESRD than non-Hispanic whites with Type 2 Diabetes [19]. The Pima Indians have the highest prevalence of Diabetic Nephropathy in the World [19]. In India the prevalence of microalbuminuria varies from 19.7 to 28.5 of unselected Type 2 Diabetes whereas the prevalence of diabetic Nephropathy in Type 2 Diabetes is reported to be 5 to 9% from various Indian studies [2,11].

ETIOLOGY

Factors which have shown to have etiologically importance in pathogenesis of diabetic nephropathy are

1. Biological factors

2. Immunological factors

3. Hormonal factors

4. Rheological factors

The biochemical factors implicated include hyperglycaemia and glycosylated proteins in blood and basement membranes of kidneys, both endogenous and exogenous insulins, anti-insulin antibody complexes mediated immunological factors contribute to basement membrane thickening in diabetics .The rheological factors responsible are loss of deformability of RBCs due to glycosylation and fibrin deposition resulting from altered permeability and hypercoagulability. Though the basement membrane is thickened in diabetic nephropathy due to various factors mentioned above, it is important to note that it functions as a "poor filter".

RISK FACTORS

1. FAMILIAL/GENETIC FACTORS

Familial clustering of diabetic nephropathy has been reported, which postulates that inherited factors may play a role in determining the susceptibility of diabetic nephropathy. Familial predisposition of raised arterial blood pressure is a contributory factor in some patients. Genetically determined sodium –lithium counter transport in red cells play a crucial role in renal resorption of sodium and thus in regulation of blood pressure. ACE polymorphisms are risk factors for initiation of diabetic nephropathy [10].

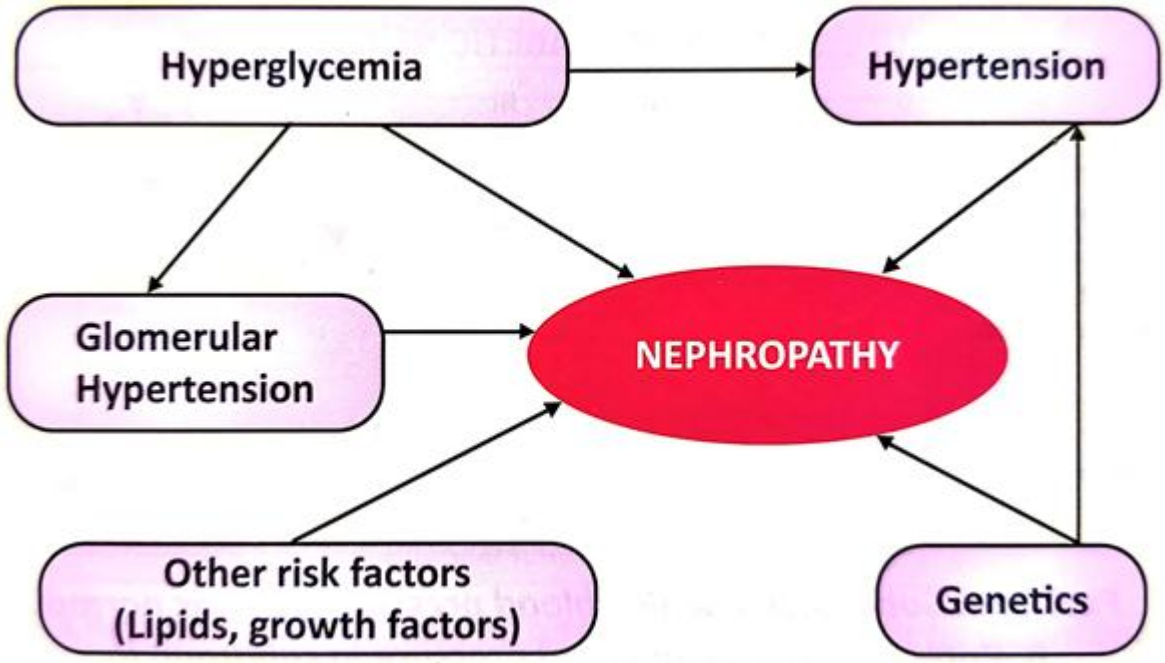

Fig -1: Interplay of Genetics and other Risk factors

2. DURATION OF DIABETES

The peak of incidence is seen in fifteenth year and it is rare during initial five years and after 25 years [8]. Patient diagnosed as diabetic after the age of 50 years have higher prevalence and degree of microalbuminuria than those diagnosed before the age of 40.

3. HYPERTENSION

The progression of early nephropathy is related to blood pressure. Hypertension is a definitive risk factor for development of diabetic nephropathy [1,8]. With the progression of renal disease the incidence of hypertension increases and by the time of overt nephropathy hypertension is usually always present.

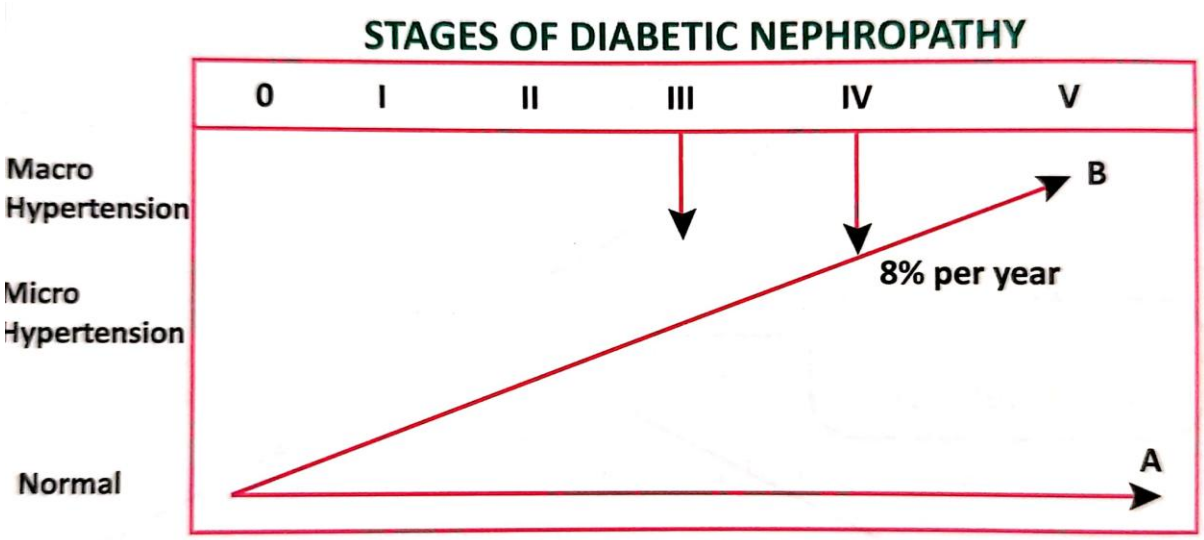

Fig – 2: A. Subgroup in which blood pressure remains normal, B. Subgroup in which Hypertension develops.

4. RENAL HYPERTROPHY AND HYPERFILTRATION

When the hyperfiltration is pronounced (GFR >150 ml/min /1.73 m2 body surface) for many years there is increased risk for diabetic nephropathy. The GFR is higher than normal in stage of glomerular hypertrophy and hyperfiltration which progressively declines in further stages to a rate of 1.2 ml per minute per month culminating in ESRD in a matter of months to years when left untreated .In established nephropathy increased renal size persists despite decrease in GFR .High protein diet induces some degree of hyperfiltration in normal man .Hence high protein diet of more than 0.8 mg/kg ideal body weight is a risk factor for diabetic nephropathy.

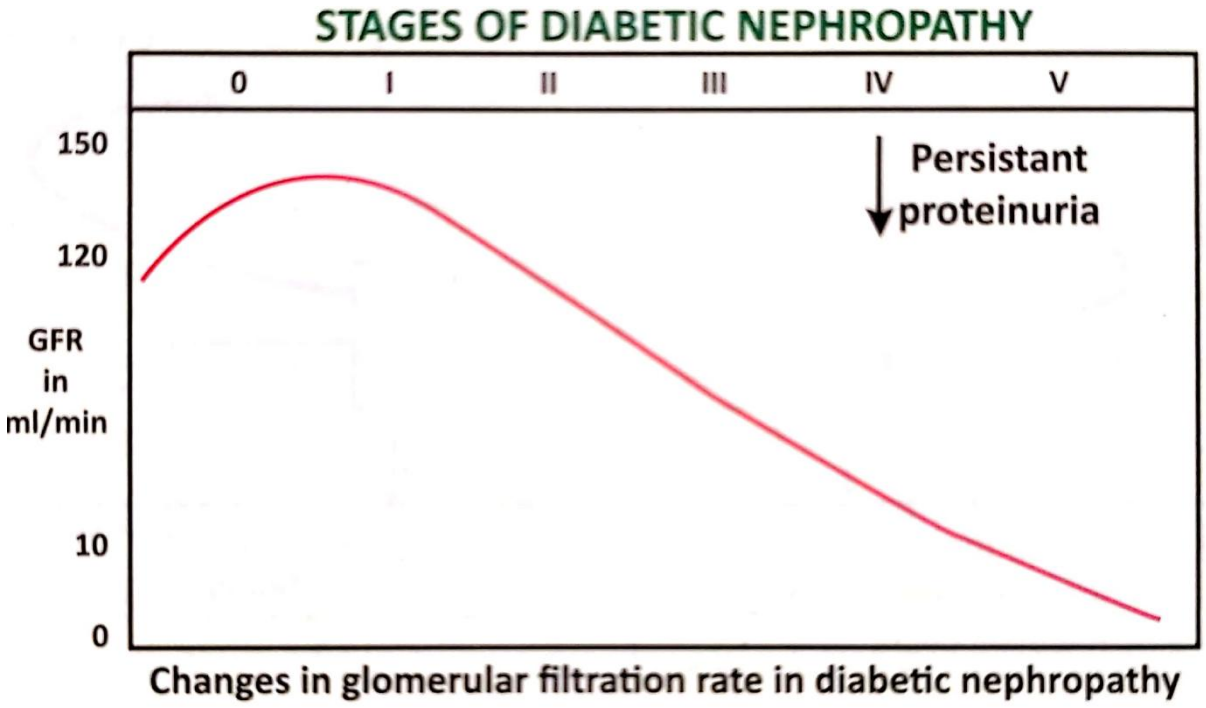

Fig – 3 Changes in GFR in Diabetic Nephropathy

5. HYPERGLYCEMIA

GFR is positively correlated to HbA1c .Patients with HbA1c <7 % are at lower risk of nephropathy. DCCT [3] and UKPDS [4] have clearly demonstrated that glucose control has resulted in reduction of diabetic nephropathy. The rate of progression of nephropathy is correlated with metabolic control [4].

6. SMOKING

Smoking causes vasoconstriction, platelet dysfunction and coagulation abnormalities which can accelerate the vascular damage .Smoking is also an independent risk factor for essential hypertension [14]

PATHOGENESIS

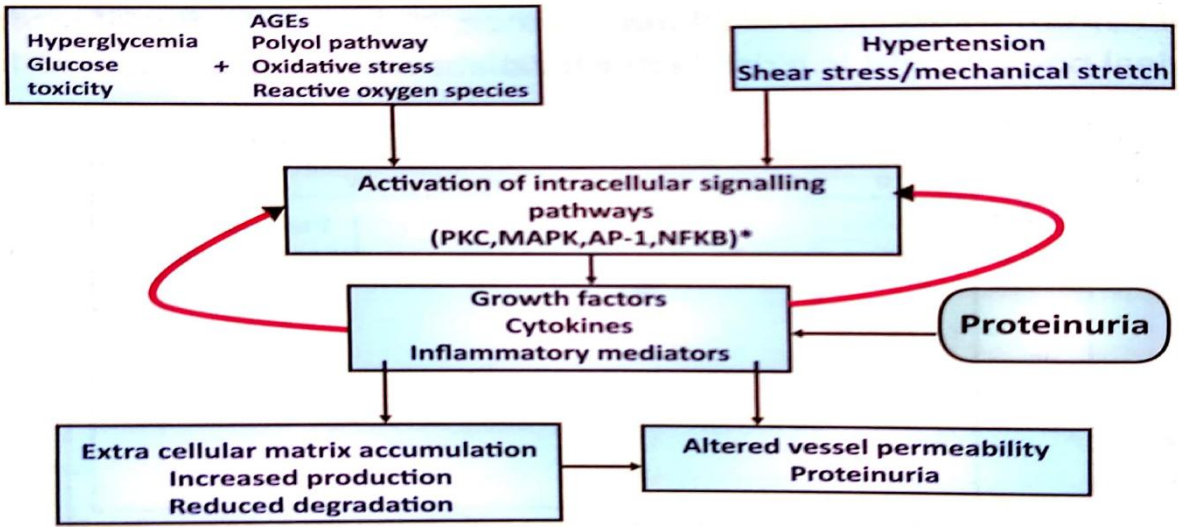

Fig – 4 Mechanisms in Diabetic Nephropathy [8]

PKC (Protein Kinase C), MAPK (Mitogen Activated Protein Kinase), AP (Activator Protein-1 transcription factor), NFKB (Nuclear Factor KB)

The main pathophysiological mechanisms causing diabetic nephropathy are 1.metabolic pathway 2.hemodynamic 3.hormonal pathway. There is interplay of all these pathways in development of diabetic nephropathy.

1. METABOLIC PATHWAY

The biochemical factors implicated are as follows

A) HYPERGLYCEMIA

The DCCT [3] and UKPDS [4] trials have shown that a tight metabolic control may prevent diabetes related renal damage. There is a positive relationship between the microvascular complications and hyperglycemic milieu of diabetics.

B) NONENZYMATIC GLCOSYLATION

Glucotoxicity in diabetics leads to reaction with tissue protein producing Amadori products .The rate of formation of these products is directly proportional to glucotoxicity. These products are slowly converted into advanced glycated end products which parallels the degree of renal insufficiency.

C) POLYOL PATHWAY

Aldose reductase present in glomerular epithelial cells , distal tubular cells of kidney play a role in generation pf sorbitol in response to high salinity in medullary interstitium. Increased sorbitol interferes with inositol signalling, depletes NADPH stores causes oxidative injury.

D) BIOCHEMICAL ABNORMALITIES OF EXTRACELLUAR MATRIX

The rate synthesis of matrix and glomerular basement membrane are increased in diabetic nephropathy. Biosynthesis of collagen is increased. The activity of lysyl hydroxylase an enzyme involved in collagen biosynthesis is increased. Further abnormalities in ECM is depicted in flowchart below

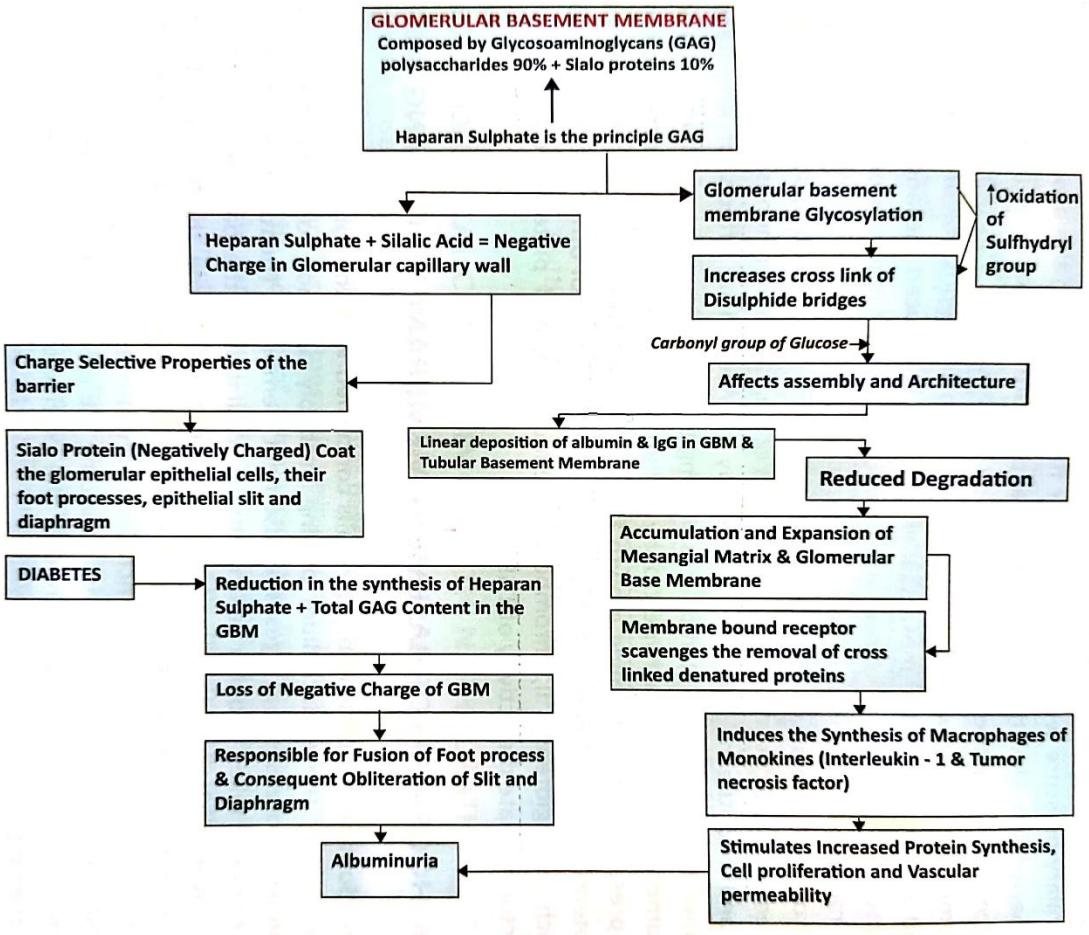

Fig – 5 Biochemical abnormalities of Extracellular matrix [8]

1. HEMODYNAMIC AND HORMONAL EVENTS

There are a) elevated GFR b) increased renal plasma flow and c) glomerular capillary pressure which mediate hyperfiltration even in the absence of systemic hypertension. Elevation in glomerular pressure leads to physical stress and shear forces damaging endothelial and epithelial surfaces.as this normal glomerular barrier, which leads to accumulation and deposition of plasma and lipoproteins in mesangial area. There is reduced mesangial clearance of proteins which act as a local stimulus for mesangial matrix production, leading to mesangial expansion. There is suppression of matrix degradation leading to histological alterations pathognomonic of diabetic glomerulopathy. These haemodynamic events are due to actions of vasoactive hormones like angiotensin II and endothelin. Renal hypertrophy in diabetes is correlated with increased renal expression of transforming growth factor (TGF) which has strong fibro genetic potential.

PATHOGENESIS OF BASEMENT MEMBRANE THICKENING

The turnover of basement membrane takes about a year normally. In diabetics the basement membrane turnover is slowed down and basement membrane thickening is thought to arise from augmented synthesis of epithelium and diminished removal of mesangium. The mesangium plays a key role in the pathogenesis of basement membrane thickening.

MORPHOLOGICAL CHANGES IN DIABETIC NEPHROPATHY

With the onset of diabetes, biochemical changes occur within the glomerulus. In two years widening of glomerular basement membrane occurs. It takes 5 years for the mesangial expansion to take place .The mesangial expansion signals the advent of microangiopathy in the diabetics. Diabetics in whom mesangial expansion remains non-progressive or slowly progressive do not develop clinical diabetic nephropathy. Rapidly progressive mesangial expansion is responsible for development of clinical diabetic nephropathy. Rapidly progressive mesangial expansion together with hyalinization of the glomerulus is the cause for progressive decline in renal function culminating in end stage renal disease

NODULAR GLOMERULOSCLEROSIS

The glomerular lesions which are ovoid, spherical, mostly laminated nodules of matrix which are PAS positive and are situated in the periphery of glomerulus. This is called as kimmelstiel Wilson disease or inter capillary glomerulosclerosis. As the disease is progressing, the nodules will enlarge and may eventually compress and engulf capillaries, which causes obliteration of glomerular tuft. The nodular lesion is highly but not completely specific for diabetes. Approximately 15-30% of patients with long term diabetes develop glomerulosclerosis which is mostly associated with renal failure [12].

FUNCTIONAL STAGING OF DIABETIC NEPHROPATHY

Three cardinal functional changes characterise the natural history of diabetic nephropathy .They are

1. Changes in GFR
2. Proteinuria and albuminuria
3. Changes in arterial pressure

MOGENSEN'S STAGING OF DIABETIC NEPHROPATHY [1,8,18]

Stage	Designation	GFR	Urinary Excretion	Blood Pressure	Main Structural Changes
I	Hyperfunction/ Hypertrophy	May be increased	May be increased	Usually normal	Hypertrophy increased kidney volume
II	Normoalbuminuria	Normal/increased	Normal	Normal	Increasing basement membrane thickness and Mesangial expansion
III	Incipient Nephropathy	Normal/increased	Micro albuminuria (20-200µg/min)	Rise of 3mm Hg/year	
IV	Overt diabetic Nephropathy	Decreasing	Macro albuminuria (More than 200µg/min)	Usually Hypertensive	Increasing GFR reduction, severe mesangial expansion
V	End Stage	< 20 ml/min	Macro albuminuria	Frank Hypertension	

Table – 1 Stages of Diabetic Nephropathy [8]

Mogensen explained the natural history of diabetic nephropathy in Type 1 Diabetes mellitus patients. But the classification holds good for all diabetes patients including Type 2 Diabetes mellitus patients also. Urinary albumin excretion rate of 20-200 µg/min is defined as microalbuminuria .As the proteinuria increases in different stages of disease, blood pressure tends to increase as shown in various studies. It is an indication of worsening of nephropathy to further stages thereby culminating in end stage renal disease. Microalbuminuria is a independent marker for development of cardiovascular disease even in nondiabetic patients [11].

STAGE I: STAGE OF HYPERPERFUSION AND HYPERFILTRATION

Hyperperfusion and hyperfiltration is the first stage in development of diabetic nephropathy. Hyperfiltation is seen in 90-95% of Type 1 diabetes mellitus patients which shows increase in GFR. Similar finding is seen in 41% of type 2 diabetes mellitus patients. There is an increased renal perfusion in this this stage, plasma oncotic pressure also seems to be low. There is an increase in glomerular capillary hydrostatic pressure in patients with diabetes mellitus.

STAGE II: SILENT STAGE

GBM thickening becomes to manifest in these patients in about 2 years in stage 2. Further there is an increase in mesangial volume and interstitial expansion in patients of diabetic nephropathy in stage 2. The difference between type 1 and type 2 diabetes mellitus patients in development of nephropathy occurs in histological changes in

kidney. Typical Kimmelsteil-Wilson lesions are not seen in type 2 diabetes mellitus patients with nephropathy during early stages of disease. In advance stages when UAE is between 15 and 20 microgram/min, typical kimmelsteil Wilson lesions will be present in most of the patients. GFR is still high in stage 2 of the disease. The blood pressure is usually normal or may be slightly elevated. In patients with Type 2 Diabetes mellitus with microalbuminuria seems to have an increase in glomerular volume which indicates to us that microalbuminuria is predictive and prognostic marker for diabetic nephropathy regardless of GFR [11].

STAGE III: MICROALBUMINURIA STAGE

When Urinary albumin excretion is between 20-200 mg/min or 30-300 mg/24 hr, it is termed as stage of microalbuminuria. They show a negative urinary protein dipstick test as they have proteinuria less than 300mg. This stage of nephropathy is thus termed as stage of incipient nephropathy. GFR is high in this stage too. Hypertension further begins to increase, so is the renal disease. There is an increase in cardiovascular risk also in these diabetic patients with nephropathy. Prevention to further stages is possible if better metabolic control is achieved. Further the proteinuria must be treated by angiotensin converting enzyme inhibitors like enalapril which is highly beneficial in prevention of renal and cardiovascular risk

STAGE IV: OVERT DIABETIC NEPHROPATHY

Urinary albumin excretion of more than 300 mg/ day or urinary protein excretion of more than 500 mg/ day signifies stage of overt diabetic nephropathy. Patients with type 2 diabetes mellitus take about 10 years to reach this stage of nephropathy .Urinary dipstick test will be positive. Diagnosis of this stage is by clinical findings. In renal biopsy, most patients will have kimmelsteil Wilson lesions, though it is not advocated in all cases. GFR begins to decline rapidly in this stage of disease and the hypertension worsens.

STAGE V: END-STAGE RENAL FAILURE

As the GFR declines down to very low renal failure is imminent. The patient presents with volume overload features like swelling of legs, pleural effusion and ascites with elevated renal parameters. Many metabolic abnormalities like hyperkalemia is usually coexistent. These patients are prone for many infections like urinary tract infections with gram negative organisms and pneumonia which might be community acquired or due to tuberculosis. These patients will have diffuse glomerular sclerosis, uncontrolled hypertension as the renal failure progresses further.

PATHOGENESIS OF DIABETIC PROTEINURIA [7]

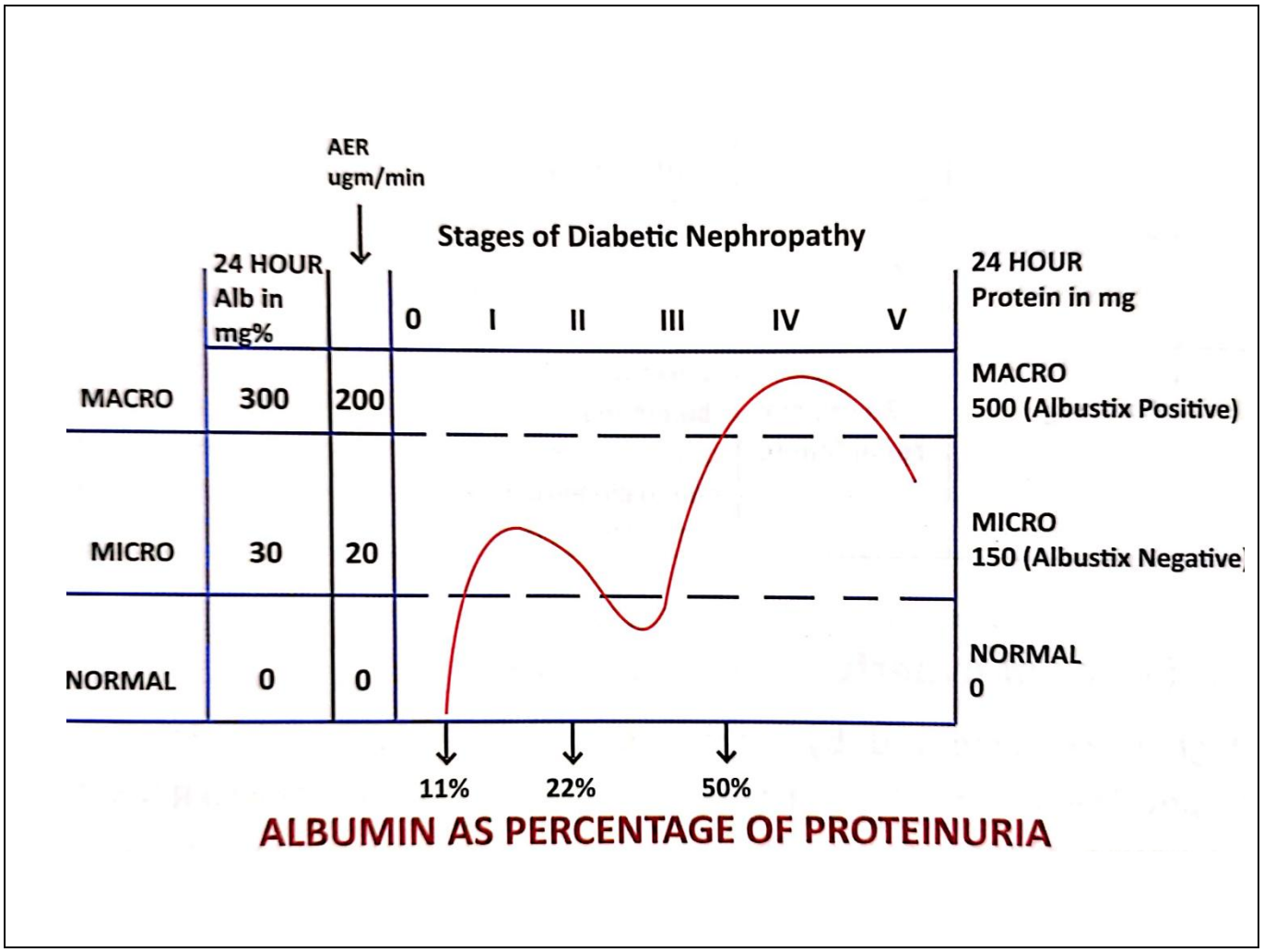

Fig – 6 Abnormalities in Albumin excretion

The barrier between glomerular capillary and urinary space of Bowman's capsule may be considered as a membrane perforated by pores of average size of 5.5 nm coated with a negative charge that is attributed to heparin sulphate, sialic acid and other proteoglycans. The size and charge of the molecules determine the passage across the membrane in addition to haemodynamic forces that controls the filtration. In early microalbuminuria, the clearance of albumin and IgG both are increased. As microalbuminuria increases there is a dispropionate increase in albumin clearance and results in fall in the ratio of the clearance of IgG to albumin. This is due to loss of

electronegative glycoproteins and proteoglycans with further haemodynamic abnormalities. In due course of time the effective pore size increases, microalbuminuria further progresses to macroalbuminuria and GFR begins to decline. Finally with advancing renal failure, proteinuria becomes mixed tubular and glomerular in origin.

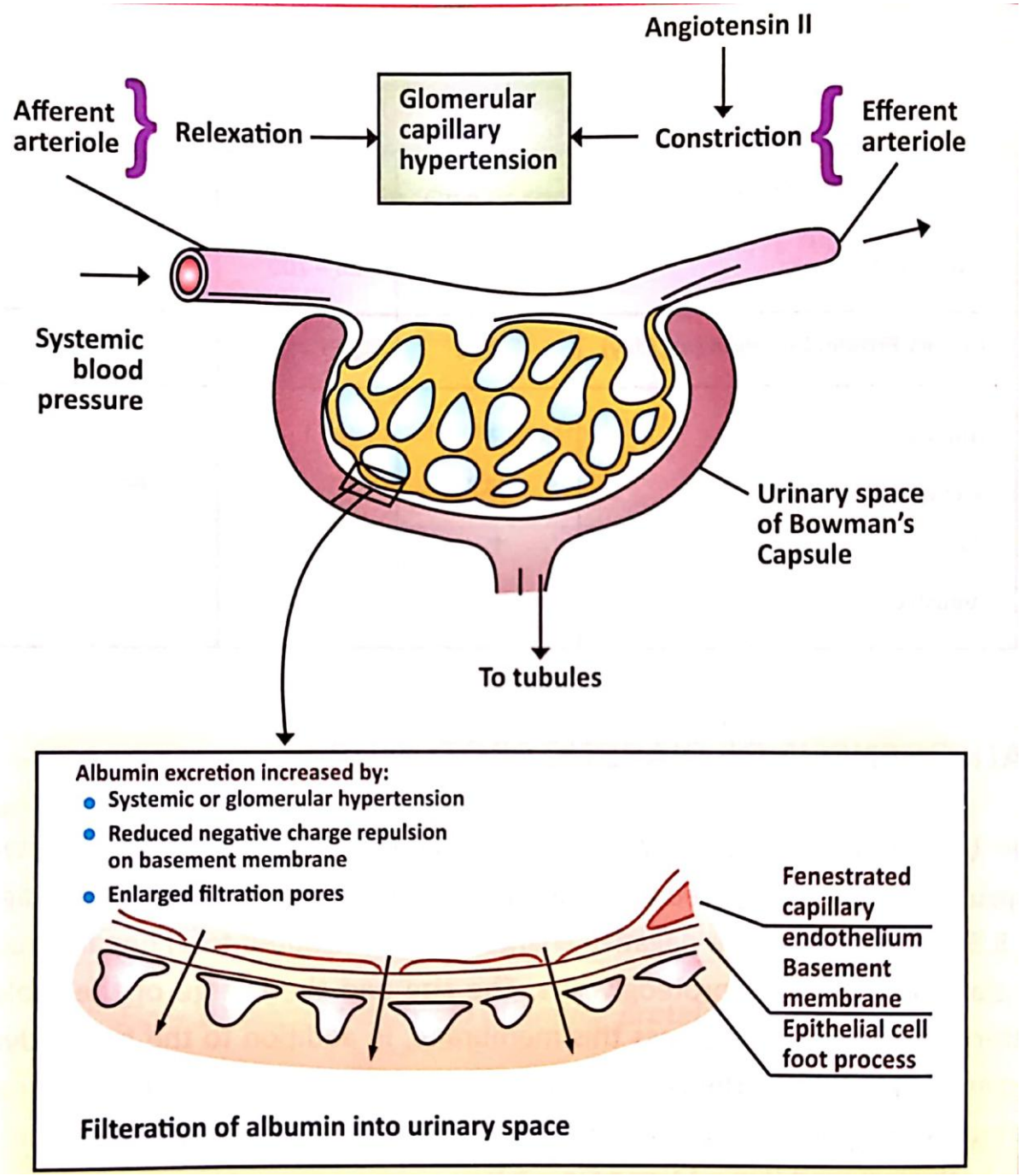

Fig – 7 Pathogenesis of Diabetic proteinuria

CLINICAL COURSE

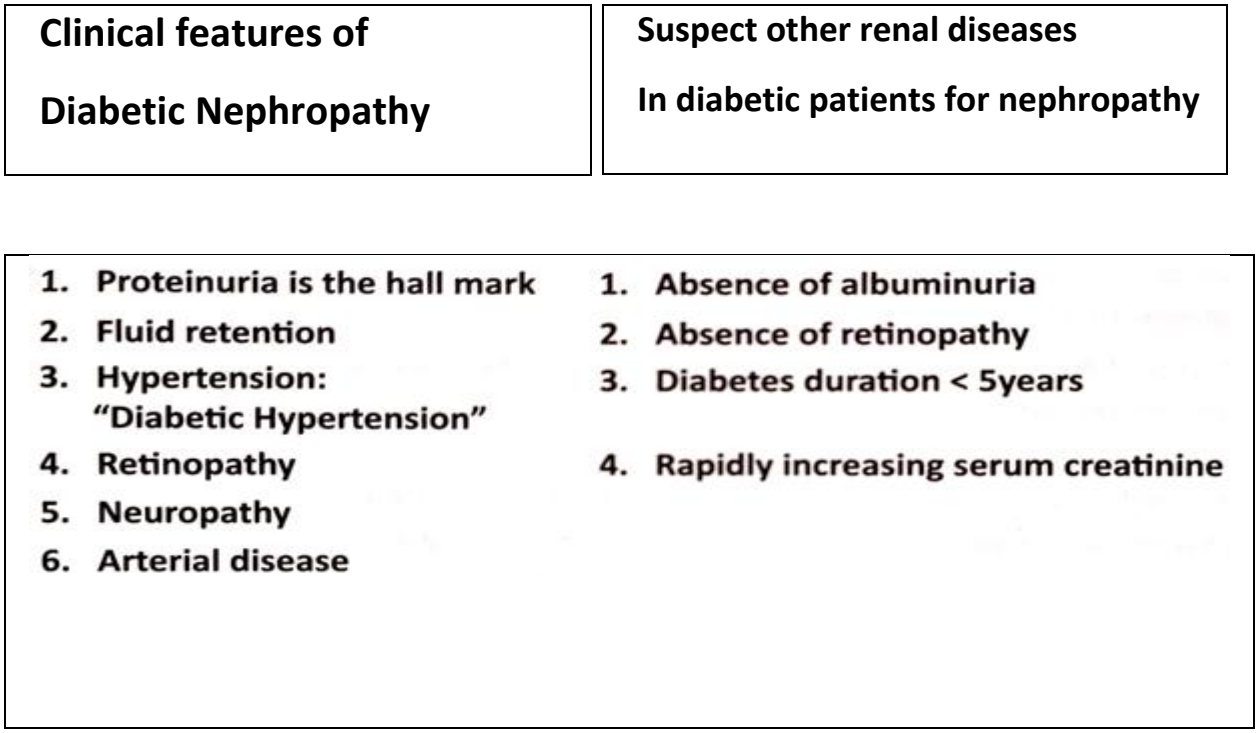

Fig – 8 Clinical features of Diabetic Nephropathy

EARLY PHASE

Important abnormalities of renal function and structure takes place during this stage. In 25% of patients GFR is exceeding upper limit of normal. Renal plasma flow is elevated. It is accompanied by increase in renal size by 20%. In the incipient stage, when the albumin excretion is between 20 -200 microgram/minute there is no clinical evidence of proteinuria, but special screening tests like micral test could detect microalbuminuria. The term persistent proteinuria or macroalbminuria is used when the Albumin excretion rate is more than 200 microgram/min manifesting as overt nephropathy [11].

The phase of microalbuminuria or moderately increased albuminuria is stage of incipient nephropathy. It is a sign of early disease but not a marker of susceptibility to nephropathy. There is a good correlation between AER and albumin/creatinine ratio particularly in first morning sample. Even in random spot collection of ACR generally gives accurate information and is therefore preferred .AER> 30 microgram /min to ACR of >2.5. Semi quantitative dipstick technique such as Micral test is also useful.

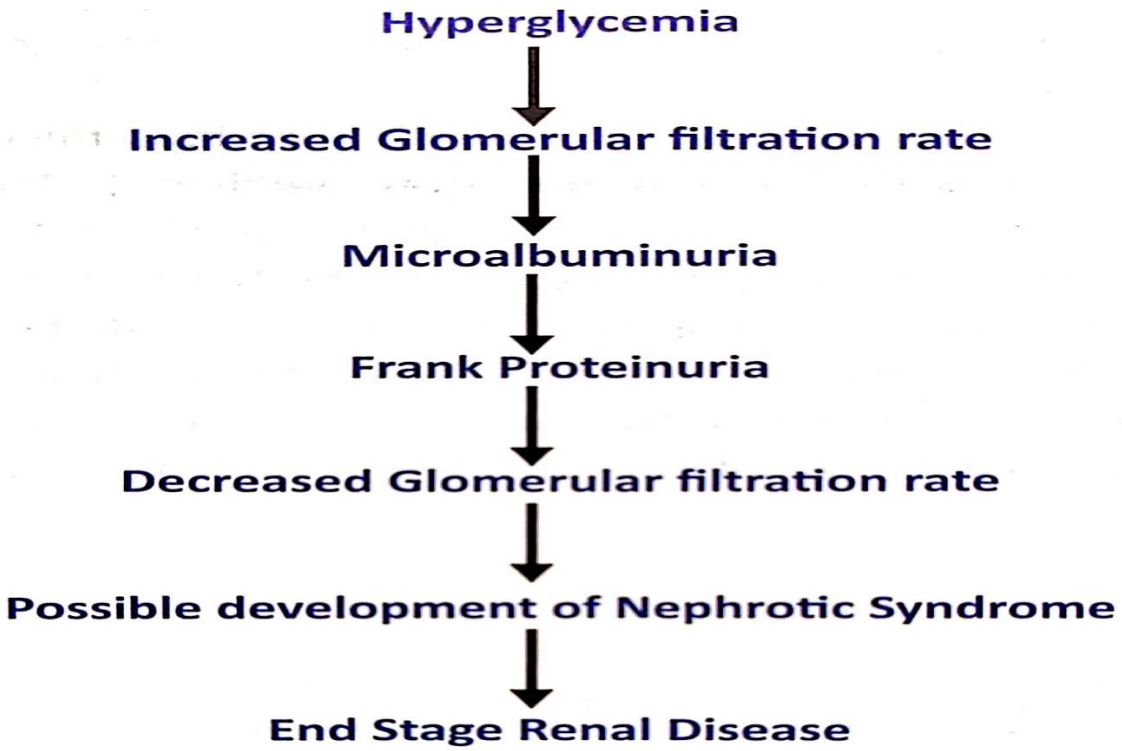

Fig – 9 Natural history of Diabetic Nephropathy

LATE PHASE

This phase of clinical nephropathy corresponds to total protein excretion of more than 0.5 g/day which corresponds to AER of 200 microgram/min or 300mg/day [11]. With

persistent proteinuria, there is a decline in GFR to end stage renal failure. Diastolic BP correlates with progression of established nephropathy. Serum creatinine does not rise until more than 50% of GFR has been lost. The use of creatinine clearance tends to overestimate GFR because of enhanced tubular secretion of creatinine in advanced renal failure. The degree of proteinuria has been related directly to the renal outcome as it is related to severity of glomerular lesions and histological damage.

A diabetic can have primary or any secondary hypertension resulting from diabetic nephropathy termed as " diabetic hypertension" which is volume dependant, low renin, low aldosterone hypertension that responds well to diuretics and fluid restriction.

Over 90% of diabetics with renal damage have retinopathy [7]. Neuropathy is common in uremic diabetic. Autonomic neuropathy of the urinary bladder is an important co-existent complication that contraindicates renal transplantation.

SCREENING OF DIABETIC NEPHROPATHY

Vigorous glucose and blood pressure control are modalities of primary prevention of diabetic nephropathy. Screening for diabetic nephropathy within the scope of secondary prevention.

RECOMMENDATION FOR SCREENING

The American Diabetic Association in concert with National Kidney foundation recommended screening for microalbuminuria for patients with type1 diabetes longer

than 5 years and starting at diagnosis for type 2 diabetes patients. Exercise, stress, diurnal fluctuation of urine albumin excretion is well known and importantly during a episode of urinary tract infection, it is suggested to has at least 2 or 3 samples tested in the course of 6 months.

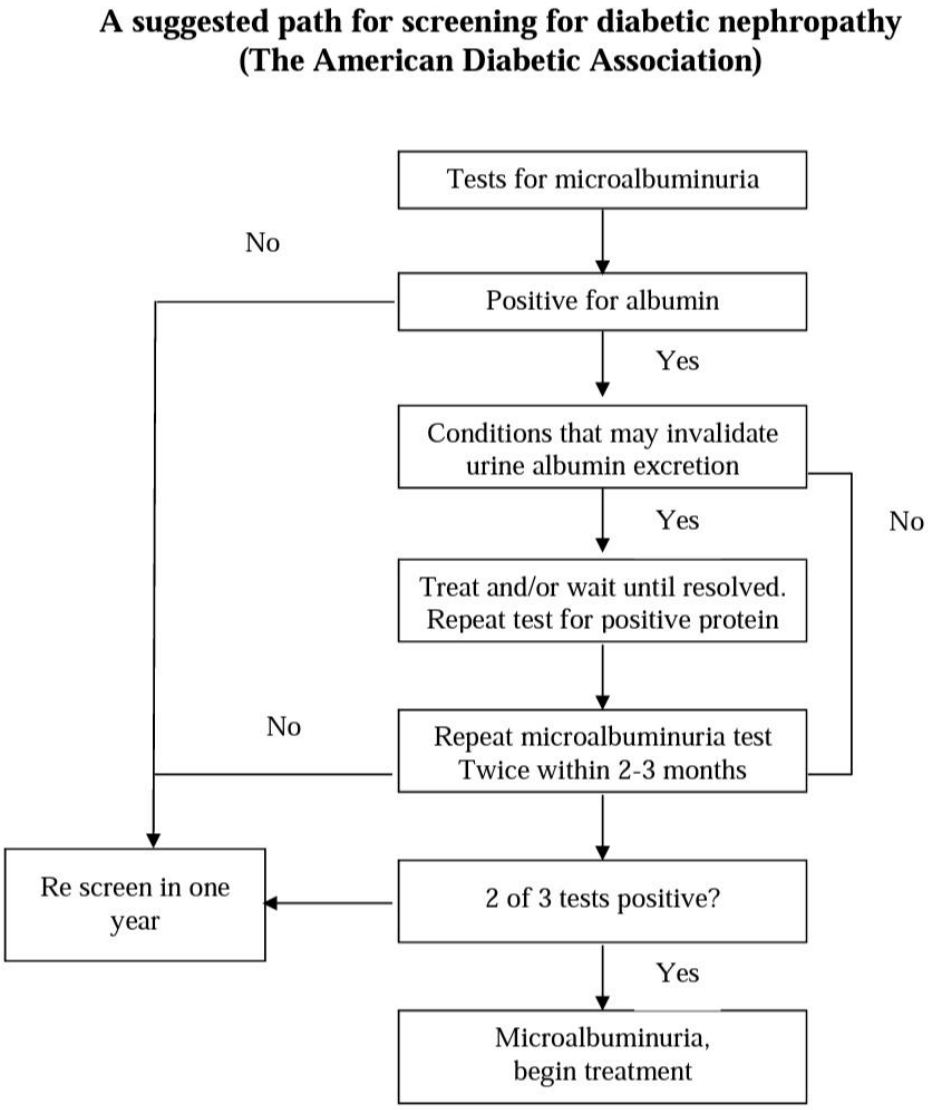

Fig – 10 Flow chart for screening of Diabetic Nephropathy [11]

CHOICE OF SCREENING TEST

1. GFR

GFR can be normal in diabetic nephropathy even in the stage of overt nephropathy. It is to note that when GFR is declined to abnormal range much of course of the disease is over and intervention to reverse the same becomes minimal. It can be estimated by MDRD (Modification of Diet in Renal Disease) or Cockrauft-Gault formula. Decline in GFR is thus late index of kidney damage in diabetic renal disease and not a good early marker for screening.

MDRD Equation:

GFR (mL/min/1.73 m^2) = 186.3 x Pcr (e-1.154) x age (e-0.203) x (0.742 if female) x (1.21 if black)

ALBUMINURIA

Urinary albumin excretion rate (UAER) is very vital in early screening of diabetic nephropathy as it can detect the disease in very early stages .For designating a patient has microalbuminuria ,at least 2 or 3 samples within 6 months should be positive.

NORMO	NORMAL	MICRO	MACRO (CLINICAL)
Albumin Excretion Rate(AER)			
mg/day	<30	30 – 300	>300
µ g/ min	<20	20 – 200	>200
Urinary Protein Excretion (mg/day)	<150	150 – 500	>500
Urinary Albumin /Protein%	11%	22%	50%
Radioimmunoassay	+	+	+
Albustix	-	-	-

Fig – 11 Proteinuria and Albuminuria Differentiation

DIAGNOSIS OF DIABETIC NEPHROPATHY

URINARY ALBUMIN EXCRETION RATE

Direct measurement of urinary albumin is more accurate than total urinary protein excretion. Persistent microalbuminuria is the earliest reliable predictor of diabetic nephropathy. Several methods such as Radioimmunoassay, Enzyme Linked Immunosorbent Assay (ELISA), semi quantitative dipstick tests are available

Condition	24-h urinary albumin excretion rate	Urinary albumin exeretion rate	Albumin Creatinine ratio
Macroalbuminuria (overt nephropathy)	> 300mg/day	> 200 µg/min	> 25 mg/mmol
Microalbuminuria	30-300 mg/day	20-200 µg/min	2.5-25mg/mmol (for men) 3.5-25mg/mmol (for women)
Normoalbuminuria	< 30 mg/day	< 20 µg/min	< 2.5 mg/mmol (for men) < 3.5 mg/mmol (for women)

Fig – 12 Albumin excretion and Albumin/Creatinine ratio

DIPSTICK TEST OR MICRAL TEST [11]

Micral–test is an immunochemically based urinary dipstick for semi quantitative determination of microalbuminuria .When compared with Radioimmunoassay micral test result of more than 20 mg/L had a sensitivity of 92.2%, specificity of 92.3% in predicting AER more than 20 microgram /min

SERUM CREATININE

It is a simple test but it is a late marker in diabetic nephropathy. It is usually not elevated up to the stage of overt nephropathy. An elevated serum creatinine in diabetic nephropathy is the cause of renal failure when it is associated with significant proteinuria.

RENAL BIOPSY

It is not done usually to diagnose diabetic nephropathy. The classical lesion of Kimmelsteil-Wilson lesion or inter capillary glomerulosclerosis is seen in stage 4 or 5 diabetic nephropathy. Though it is not pathognomonic, it is an important specific pathological lesion in diabetic kidney disease. Fibrin caps and capsular drops along with hyaline lesions are seen in afferent and efferent arteries of patients of Diabetic Kidney Disease. Immunofluorescence studies show no immune deposits or scanty deposits of IgG in capillary loops.

Renal biopsy is considered if atypical features of diabetic kidney disease are present such as

1. Minimal proteinuria
2. RBC casts in urinary sediment
3. Short duration of diabetes
4. Absence of Diabetic Retinopathy
5. Decline of GFR or rise in Albumin Excretion Rate falls outside established norms or clinical/laboratory findings suggestive of non-diabetic kidney disease
6. When multisystem system disease such as Systemic Lupus Erythematosus is suspected

MARKERS OTHER THAN MICROALBUMINURIA

There is intensive research to identify earlier biochemical/clinical or genotypic marker that will be predicting diabetic nephropathy with high specificity and sensitivity before the actual development of disease. Of these genes which are encoding for renin angiotensin system, very notably the genes that for Angiotensin Converting Enzyme (ACE) is a field of interest .3 genotypes such as II /ID/DD have been found in population. In Japanese population there is higher incidence of DD genotype in Type 2 DM patients with declining renal functions [16].

PROGNOSIS

A) CLINICAL

Amount of proteinuria is a guide to prognosis. Ten year survival rate has been estimated to be 19-23%. In those nephrotic syndrome, two year survival rate has been found to be less than 50% [13]

B) RENAL FUNCTION

With the onset of clinical proteinuria, GFR falls at the rate of 1.2 ml per minute per month. Serum creatinine levels more than 2.3 mg% indicates poor prognosis[8]. Estimation of serum creatinine and then plotting the inverse creatinine against months in a graphic form generally shows a linear decline which if extrapolated may predict when ESRD likely to occur.

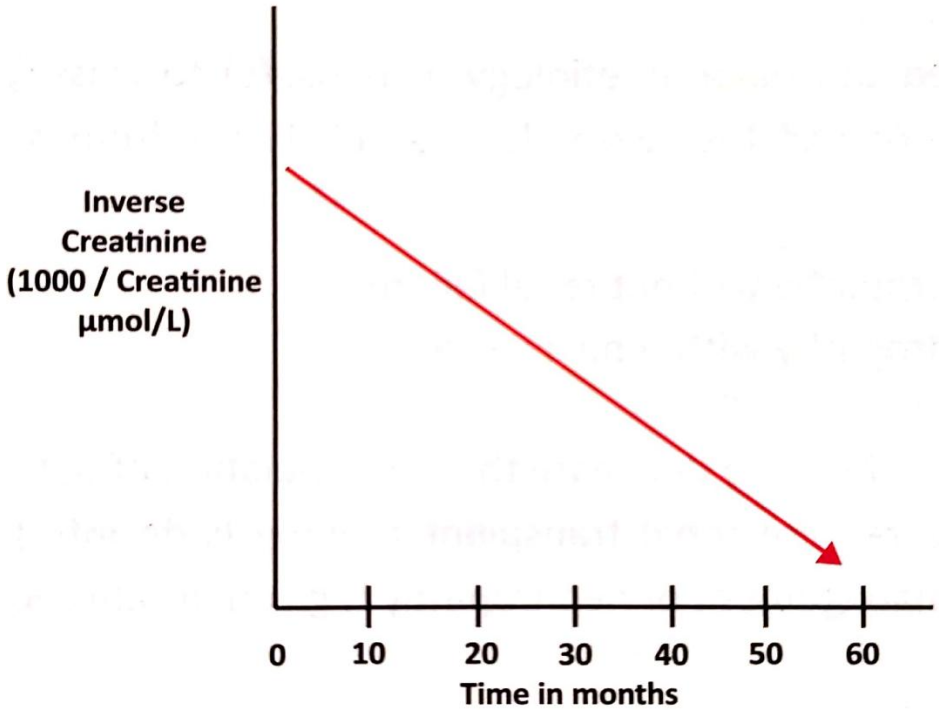

Fig – 13 Showing Inverse Serum Creatinine against time to determine prognosis in Diabetic Nephropathy

C) BIOPSY

The presence of kimmelsteil Wilson nodules on biopsy suggests a poor prognosis. Mean survival rate after the kimmelsteil Wilson nodules have been noted as 1.3 years and 3 –year survival rate as low as 10% [13].

Thus heavy proteinuria, fluid retention and serum creatinine of >2.3 mg% are poor prognostic indices in a diabetic with nephropathy.

FACTORS AFFECTING THE PROGNOSIS

Tight glycemic control may not be mandatory as it is not going to affect the prognosis in uraemic diabetic patients significantly. Hypertension and infection control are the two most important factors that adversely affect the prognosis and hence are required to be controlled adequately at every stage of disease. Glycemic control assumes importance in post-transplant uraemic diabetic.

MANAGEMENT OF DIABETIC NEPHROPATHY [1,7,8]

The first step in the management of a diabetic with renal involvement is to determine whether the renal involvement is diabetes induced or not .for the renal involvement due to non-diabetic cause the prognosis is superior. The poor prognosis in diabetic is due to multi system involvement.

Having established the diabetic etiology, it is useful to classify a diabetic with nephropathy into one of the two following subclasses for practical stand point

1. Diabetic nephropathy without renal failure
2. Diabetic nephropathy with renal failure

Stage	**Creatinine Clearance**	**Strategy**
Silent	Normal to supranormal	Maintain Euglycemia and control hypertension. Do eye screening
Early Proteinuria	Normal – 30 ml/min	Exclude other causes of Proteinuria and patient education
Nephrotic Proteinuria	20 – 30 ml/min	Consider Dialysis access and inventory of Kidney donors. Review eye and Cardiac status
Azotemia	5 – 25 ml/min	Review uremia therapies and eye status create dialysis access emphasize transplantation
Renal Death	5 ml/min	Uremia-therapy

Table – 2 The Stages and Strategies in management of Nephropathy [8]

1. CONTROL OF HYPERGLYCEMIA

Metabolic control is related to nephropathy in normoalbuminuric subjects. GFR is positively correlated to HbA1c [3]. High GFR predicts future nephropathy probably via hyperfiltration.HbA1c is also related to increased urine albumin excretion. When the strict metabolic control is enforced with intensive insulin therapy, there was a significant reduction in GFR propionate to reduction in HbA1c and mean blood glucose levels. Glycemic control retards the progression of overt nephropathy.

Biguanides are contraindicated for fear for lactic acidosis. Sulphonylurea drugs that are excreted unchanged by kidneys such as gilbenclamide are not recommended .Drugs such as gliclazide and glipizide which are metabolized in liver are preferable. Though insulin is chosen good, it must be remembered that the requirement may decrease since one third of insulin is degraded by kidneys, which does not happen due to nephropathy.

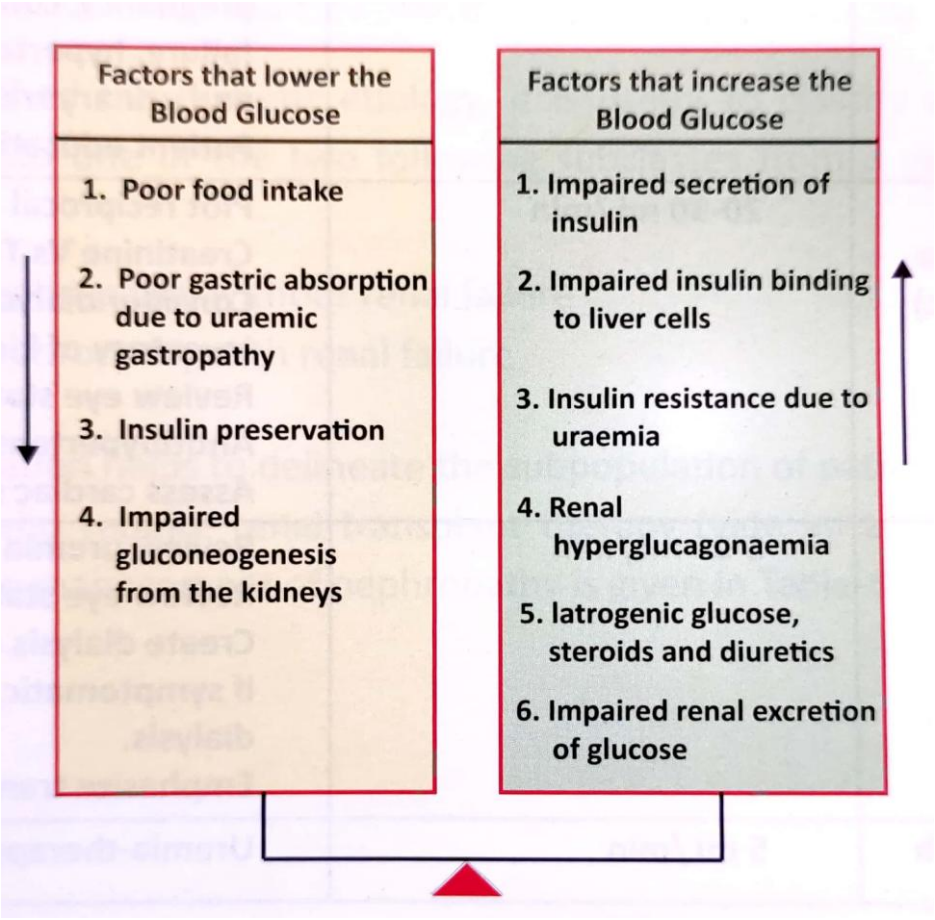

Fig – 14 Factors affecting Glucose homeostasis in Chronic Renal failure

S.No	Indices	Good control
1	**PLASMA GLUCOSE (mg/dl)**	
	i) Fasting/Pre prandial	< 100
	ii) 2 hour Post prandial	< 140
2	**SERUM LIPIDS (mg/dl)**	
	a) Total Cholesterol	Upto 200 (< 170)
	b) B)Triglycerides	Upto 150
	c) HDL Cholesterol	> 50
	d) LDL Cholesterol	Upto 100
	e) Total Cholesterol/HDL Cholesterol	< 4.5
3	**GLYCOSYLATED HEMOGLOBIN**	
	HbA1c (%)	< 6.5
4	**FRUCTOSAMINE (mmol/l)**	Upto 2.8

Table – 3 Monitoring of Diabetes Mellitus [7]

2. DIETARY PROTEIN RESTRICTION

Dietary protein restriction has shown to be having definite beneficial effect on the declining renal function in uraemic diabetics. Isocaloric low protein diet reduces the UAE in microalbuminuria patients. Low protein diet (40 g/day) in overt nephropathy retards the progression of renal disease.

3. CONTROL OF HYPERTENSION

Normalizing the blood pressure at every stage of disease is stressed as a important component of therapeutic program. Blood pressure should be reduced

to a standing BP of 120 /70 to 130/80 mmHg. Indeed the control of hypertension in a uraemic diabetic is more than control of hyperglycemia itself. In normoalbuminuric patients hyperfiltration is observed by use of ACE inhibitors, which causes relaxation of afferent arteriole and have been associated with reduction in protein leakage and renal protection in patients both with and without diabetes. ACE inhibitors are more beneficial in stage of incipient nephropathy [14]. Intra renal hypertension is the important causative factor in the progression of renal disease, effective normalization by ACE inhibitors not only reduces the proteinuria but also ESRD.

Other antihypertensive drugs that are effective are non-dihydropyridine, calcium channel blockers and angiotensin II receptor blockers. The recent trend is to give combination of ACE inhibitors and ARBs. ARBs have also got renal protection effect.

4. CONTROL OF INTRAGLOMERULAR HYPERTENSION

ACE inhibitors such as captopril lowers intra glomerular hypertension and thus reduce hyperfiltration of diabetic nephropathy[14]. It is therefore recommended that in diabetics with normal blood pressure, ACE inhibitors to be given to lower the intra glomerular pressure.

5. URINARY INFECTIONS

Urinary tract infection is one of the principal cause of worsening of renal function in a otherwise normal uraemic diabetic and require to be sought for and energetically treated with appropriate antibiotics. Drugs having renal toxicity

such as aminoglycosides and contrast agents are to be avoided in such patients with a creatinine clearance of less than 25 ml/min

6. DIURETICS

As the renal reserve declines to about 25% of normal value, chlorthiazide and hydrochlorthiazide becomes ineffective and must be replaced by a loop diuretic such as furosemide. As the creatinine clearance falls to 10 -20 ml/min as high as 480 mg of furosemide daily may be required to effect diuresis.

LIMITATIONS OF CONSERVATIVE THERAPY

Neither the patient nor the physician should be surprised by the ultimate need for dialysis or transplantation in a diabetic who has undergone years of declining renal reserve. With the reduction in GFR to below 10 ml/min, anemia, acidosis, lethargy and uncontrollable hypertension dictate the end of conservative care and signal the need for dialysis or renal transplantation.

MANAGEMENT OF URAEMIC DIABETIC PATIENTS

Diabetic nephropathy is a tragic and devastating illness that is attended by enormous medical, economic and social problems. Yet there is a life after the onset of renal failure today

OPTIONS IN URAEMIC THERAPY

1. Maintenance of hemodialysis
2. Peritoneal dialysis a)Intermittent peritoneal dialysis b)continuous ambulatory peritoneal dialysis c)continuous cyclic peritoneal dialysis
3. Hemofiltration
4. Renal transplantation a)living – Related/unrelated donor b)Cadaver donor

When the graft and patient survival, quality of life, rehabilitation, cost and availability are considered together, living related donor renal transplantation is now considered gold standard treatment of end stage renal failure. However for many maintenance hemodialysis remains the main usually the sole treatment for duration of their post-uraemic lives.

PRIMARY PREVENTION OF DIABETIC NEPHROPATHY [8]

Primary prevention involves intervention before stage 1 that is before hyperfiltration and hyperperfusion develops

1. Early diagnosis of diabetes mellitus and strict control of blood glucose from very beginning
2. Control of hypertension
3. Lipid control
4. Dietary protein of acceptable quantity

5. Identification of high risk group as those with a) Family history of hypertension b)Red cell marker i.e. sodium lithium counter transport exchange system activity

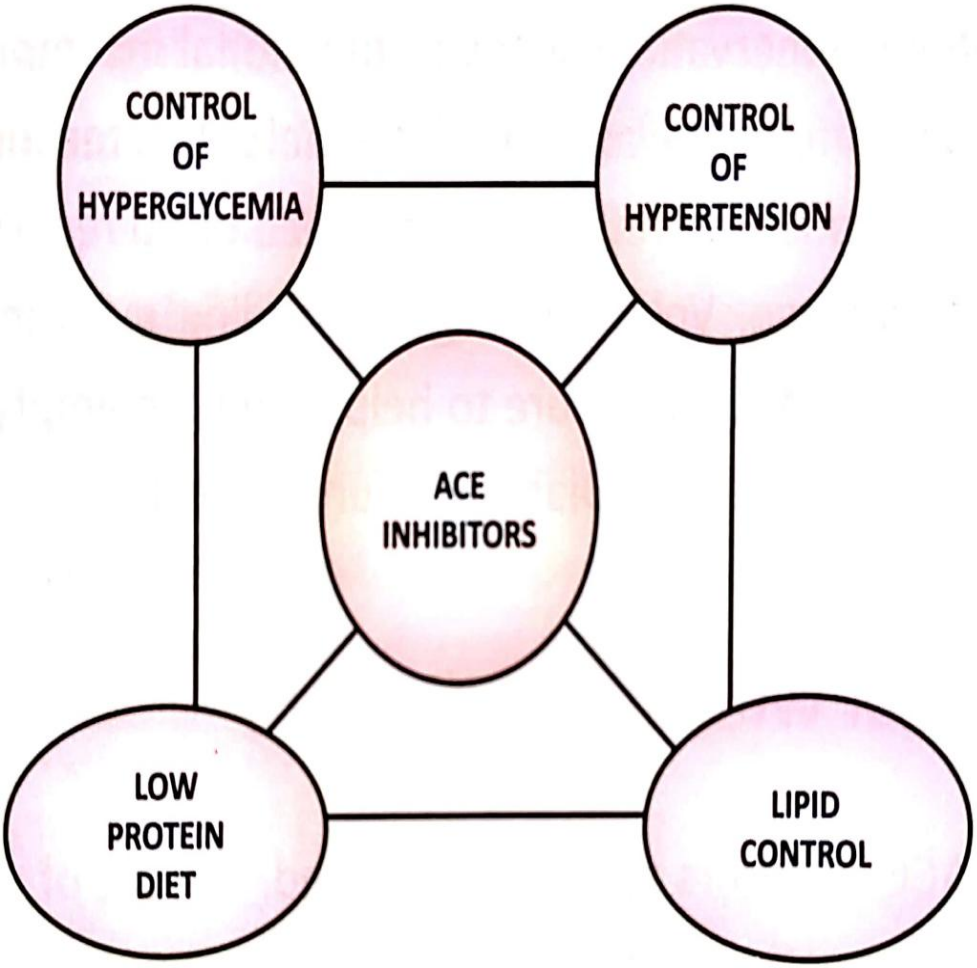

Fig – 15 Prevention of Diabetic Nephropathy

MATERIALS AND METHODS

1. STUDY POPULATION :

Patients who are attending General Medicine/Diabetic Outpatient department in Chengalpattu Government medical college hospital and are newly detected as Diabetes Mellitus who are above 30 years of age were included in this study

2. STUDY DURATION :

This study was conducted for ONE year period between June2018 to May2019

3. STUDY DESIGN:

We followed Cross sectional design for this study.

4. STUDY PLACE :

Government Chengalpattu medical college, Department of General Medicine

5. SAMPLE SIZE AND SAMPLING METHOD :

100 newly detected Diabetes Mellitus patients were selected by convenient sampling method

6. INCLUSION CRITERIA :

A) Newly detected Type 2 Diabetes Mellitus subjects who are above 30 years of age were included in study

7. EXCLUSION CRITERIA:

A) Patients who are not willing to participate in the study

B) Patients with poor glycemic control and drug compliance

C) Patients with uncontrolled hypertension

D) Patients with urinary tract infection by urine culture and sensitivity

E) Patients suspected to have non diabetic kidney disease

F) Patients with multiple comorbidities

8. DATA COLLECTION METHOD:

By using Questionnaire, comprehensive clinical examination, biochemical investigations and appropriate imaging as prescribed in proforma, explained as follows

A. HISTORY TAKING

Age, History of previous illness of hypertension, family history of diabetes and kidney diseases, personal history of smoking were Obtained.

B.ANTHROPOMETRY

Height, weight was recorded. Body Mass Index was calculated using Quetelet index

BMI = WEIGHT (in kilogram) / HEIGHT in (m) ^{2}

C.BLOOD PRESSURE MEASUREMENT

Right upper arm blood pressure measured in supine position using sphygmomanometer under appropriate condition.

D. FASTING LIPID PROFILE

Triglyceride (TGL), Low Density Lipoprotein (LDL) and High Density Lipoprotein (HDL) were estimated and calculated using standard methods in our biochemical department.

E.RENAL FUNCTION TEST

Blood samples are collected for blood urea and serum creatinine in biochemical laboratory of Chengalpattu medical college hospital. The blood Urea was estimated by Diacteyl monoxime method and Modified Jaffe's method for estimation of serum creatinine.

F.GFR CALCULATION

GFR is calculated by Modified Diet in Renal Disease (MDRD) equation by using Medcal calculator

GFR (mL/min/1.73 m^2) = 186.3 x Pcr (e-1.154) x age (e-0.203) x (0.742 if female) x (1.21 if black)

G.URINE ANALYSIS

Urine sample was obtained for analysis of sugar and albumin, Culture and sensitivity using appropriate methods.

H. URINE SPOT PCR

Urine sample is collected to estimated spot PCR by sulphosalicyclic precipitation method for estimation of protein.

I. MICRAL TEST

MICRAL dipstick test strips were used to detect the presence of microalbuminuria in early morning sample.

J. DIABETIC RETINOPATHY SCREENING

Fundus examination is done for all subjects with direct ophthalmoscope

K. ULTRASOUND KUB

Ultrasound KUB was done to find out renal size and to rule out other causes of nephropathy

DEFINITIONS

Diabetes Mellitus

WHO recommends Oral Glucose Tolerance Test (OGTT) for diagnosis or exclusion of diabetes mellitus .It was done with 75 gram of glucose in 250 ml of water after an overnight fasting of 10-16 hours following at least three days of unrestricted diet

	Plasma Glucose Venous Sample	
DIABETES MELLITUS:	mmol/l	Mg/dl
Fasting value	≥ 7.0	(≥ 126)
2hr. after 75gm glucose load	≥ 11.1	(≥ 200)
IMPAIRED GLUCOSE TOLERANCE (IGT):		
2 hr. after 75 gm glucose load	7.8 – 11.0	(140 – 199)
IMPAIRED FASTING GLUCOSE (IFG):		
Fasting value	5.6 – 6.9	100 – 125
2hr. after 75 gm glucose load	< 7.8	< 140

Table – 4 Diagnostic Values for OGTT [7]

Microalbuminuria

It is defined as urinary albumin excretion greater than 30 mg/ 24 hours or 20 microgram/min and less than or equal to 300 mg in 24 hours or 200 microgram /min [11].At least 2 out of 3 samples collected within 6 months period irrespective of how the urine is collected should be positive to be considered as microalbuminuria

MICRAL test showing positivity more than 20 mg/L is considered Microalbiminuria [11]. PCR value of more than 0.03 to 0.3 is considered as excretion of about 30 mg to 300 mg in 24 hours. Both showed one/one correlation in our study

Macroalbuminuria

Persistent albuminuria greater than 300mg/24 hours or albumin excretion rate greater than 200 microgram/ min is considered as macroalbimunuria [11]

Systemic Hypertension

Systolic Blood pressure more than or equal to 140 mmHg and /or diastolic pressure more than or equal to 90 mmHg were considered to have hypertension in our study. JNC VIII recommends good blood pressure control of less than 140/90 in diabetic patients with hypertension [14].

ACE inhibitors and non dihydropyridine calcium channel blockers were not used for control of blood pressure as they had modifying effect on proteinuria.

DYSLIPIDEMIA

National Cholesterol Education programme – Adult treatment panel (NCEP ATP III) updated in 2004 and ACC/AHA recent 2017 guidelines were considered and following guidelines were taken for our study

TYPE	CUT OFF VALUES (mg/dl)
HDL Dyslipidemia	< 40
LDL Dyslipidemia	>100
TGL Dyslipidemia	>150

Table – 5 Dyslipidemia cut-off values

BMI CLASSIFICATION

The WHO classification for classification of weight status and obesity is followed for our study

BMI GROUP	BMI (kg/m2)
Underweight	<18.5
Normal BMI	18.5-24.9
Overweight	25- 29.9
Obesity	≥30

Table – 6 BMI Classification

DATA ANALYSIS

The following statistical methods were used for analysis

1) Chi –square test
2) Two sample t test

   All data were entered using MS – Excel. All Analysis was done by using Windows –based SPSS version 16. Both Descriptive and Inferential analysis was done.

RESULTS

Total No. of subjects in the study – 100

No. of Males – 36 (36 %)

No. of Females –64 (64 %)

Age distribution in the study population

Table - 7

Age group (Years)	No. of subject	Percentage
31-40	17	17%
41-50	39	39%
51-60	32	32%
Above 60	12	12%
Total	100	100%

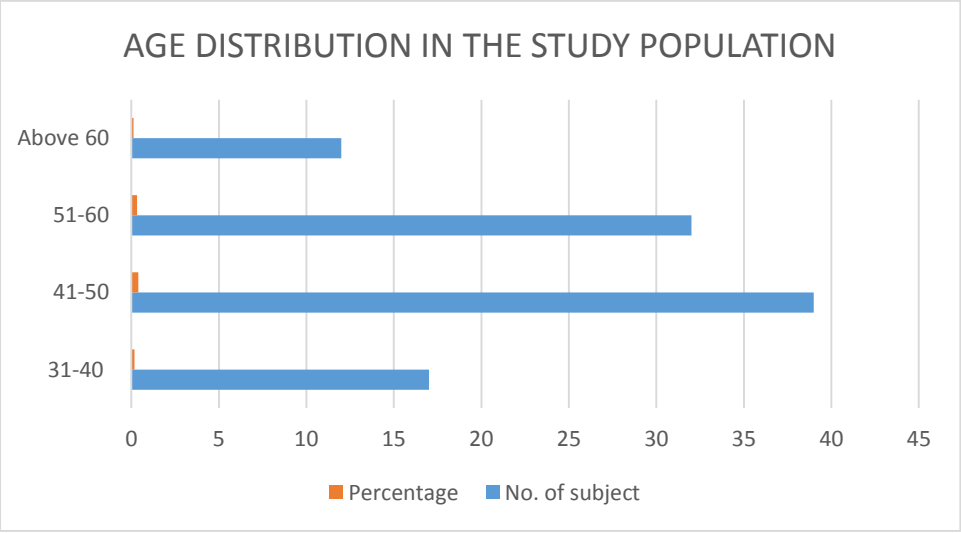

Chart – 1

Age distribution in the Diabetic Nephropathy Group

Table – 8

Age group (Years)	No. of subject	Percentage
31-40	2	11.8%
41-50	5	29.4%
51-60	7	41.2%
Above 60	3	17.6%
Total	17	100%

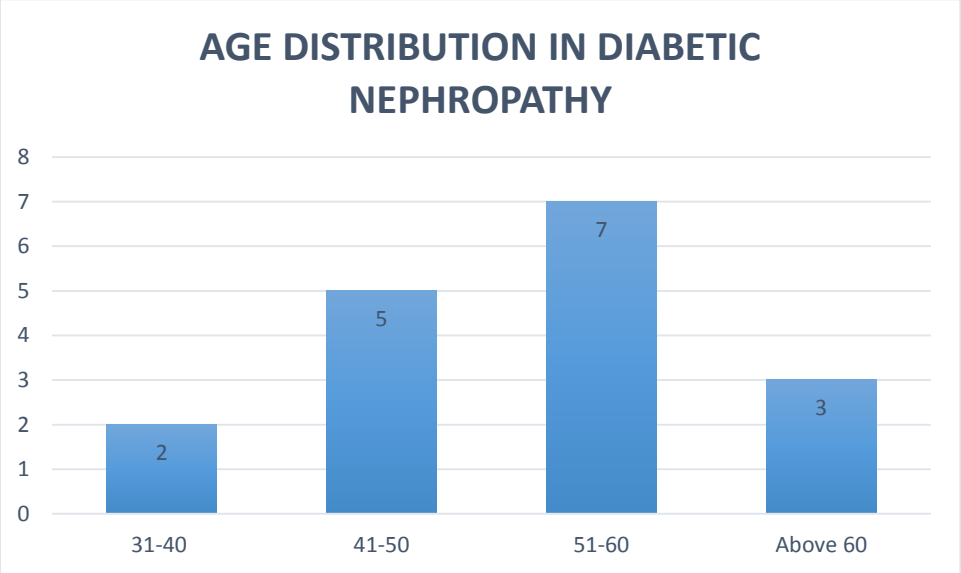

Chart - 2

Age and Diabetic Nephropathy

Table - 9

Nephropathy	**Subject**	**Age Mean**	**Standard Deviation**	**Standard Error**
Yes	17	50.47	9.59	2.32
No	83	47.41	9.12	1.00

P Value = 0.2144 **Not Significant**

The Average age in our study is 48 ± 9 years. The average age among Nephropathy group is 50 years and Non Nephropathy group is 47 years. There was no significance between age and Diabetic Nephropathy in our study.

Distribution of Subjects according to B.M.I

Table - 10

BMI Group (Kg/m^2)	No. of subject	Percentage
< 18.5	5	5%
18.5-24.9	33	33%
25-29.9	49	49%
≥ 30	13	13%
Total	100	100%

- In our study 33 % of the subjects had normal BMI, 49 % were overweight and 13 % of our subjects were obese.

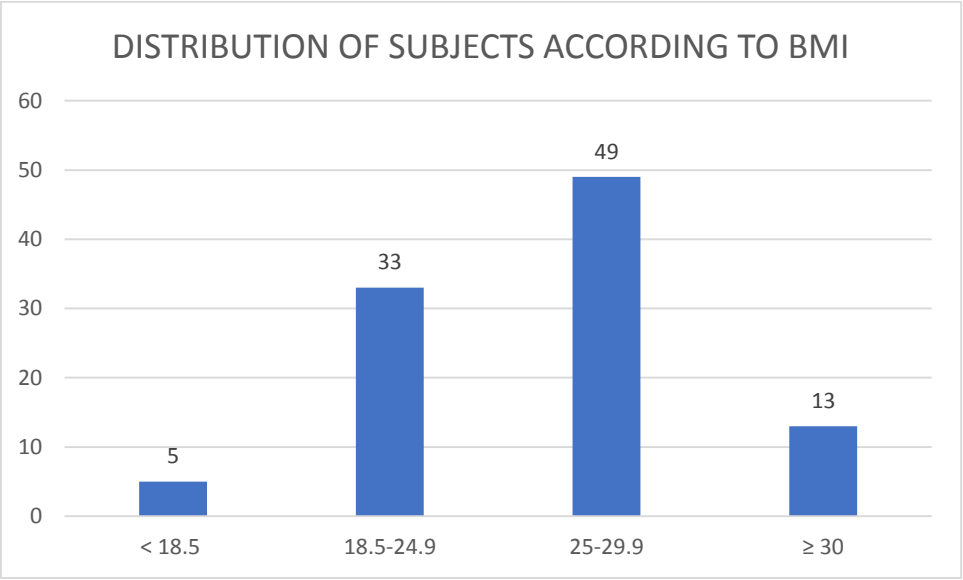

Chart – 3

BMI Distribution in Nephropathy Group

Table - 11

BMI Group (Kg/m^2)	No. of subject	Percentage
< 18.5	0	0%
18.5-24.9	4	23.6%
25-29.9	10	58.8%
≥ 30	3	17.6%
Total	17	17%

- In our study among Diabetic Nephropathy subjects 58.8% and 17.6% were overweight and obese respectively.

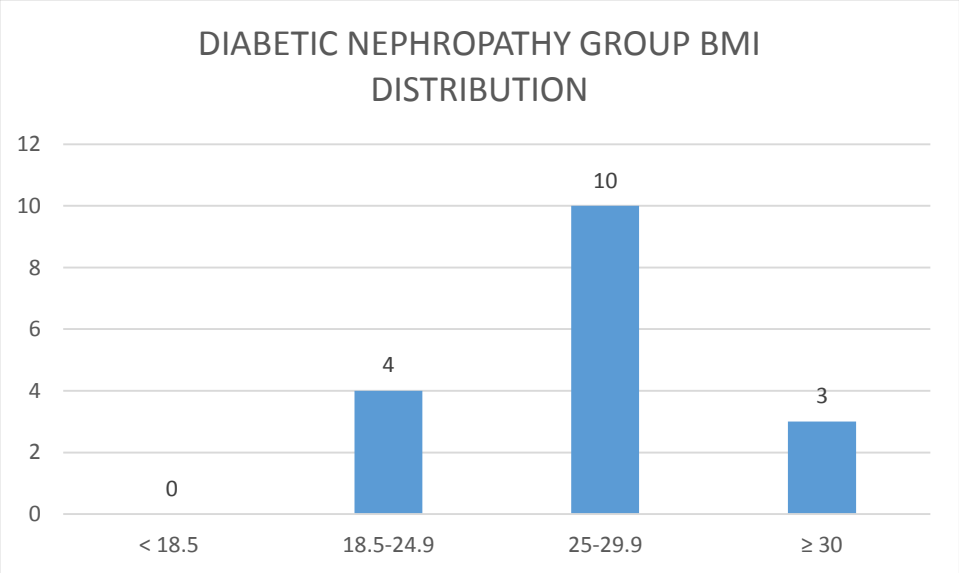

Chart – 4

BMI and Diabetic Nephropathy

- The mean BMI among the Nephropathy group is 27.46% and among the Non Nephropathy group is 24.38%
- There was a significance between body mass index and Diabetic Nephropathy in our study.

Table - 12

Nephropathy	**Subject**	**BMI Mean**	**Standard Deviation**	**Standard Error**
Yes	17	27.46	2.87	0.69
No	83	24.38	3.92	0.92

P Value = 0.0028 **Significant**

BMI and Diabetic Nephropathy

Table - 13

BMI Group (Kg/m^2)		**Nephropathy**		**Total**
		Yes	**No**	
< 18.5	Count	0	5	5
	% Within Nephropathy and Non Nephropathy	0	6%	5%
18.5-24.9	Count	4	29	33
	% Within Nephropathy and Non Nephropathy	23.5%	34.9%	33%
25-29.9	Count	10	39	49
	% Within Nephropathy and Non Nephropathy	58.8%	46.9%	49%
30 and above	Count	3	10	13
	% Within Nephropathy and Non Nephropathy	17.7%	12.2%	13%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

Gender and Diabetic Nephropathy

Gender		Nephropathy		Total
		Yes	No	
Male	Count	5	31	36
	% Within Nephropathy and Non Nephropathy	29.4%	37.3%	36%
Female	Count	12	52	64
	% Within Nephropathy and Non Nephropathy	70.6%	62.7%	64%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 0.386 **Not Significant**

P = 0.9836

There was no significance found between Gender and Diabetic Nephropathy in our study.

Diabetic Nephropathy in our study

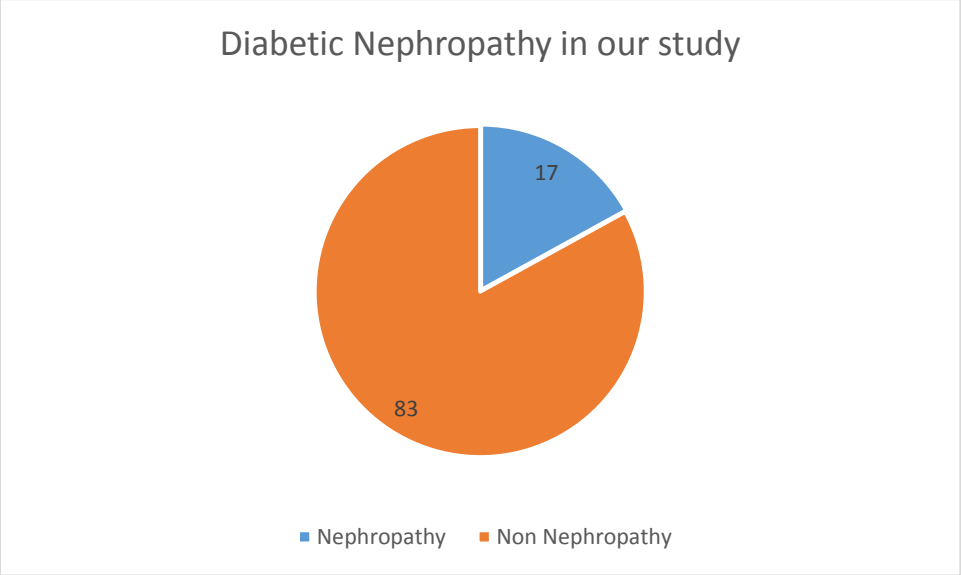

Chart – 5

- In our study, 17 subjects were found to have Diabetic Nephropathy. Among them 16 subjects had Microalbuminuria or moderately severe proteinuria, 1 subject had Macroalbuminuria or severe proteinuria.

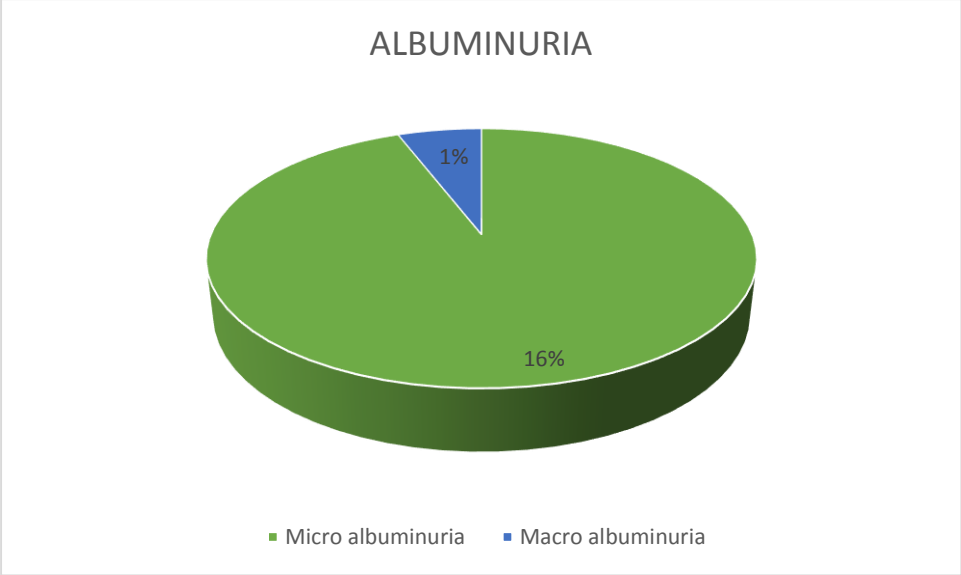

Chart - 6

Family History of Diabetes and Diabetic Nephropathy

- In our study, 41 subjects were having family history of Diabetes. Among the Nephropathy group 58.8 % had family history of Diabetes and among Non-Nephropathy 37.3 % had Family History of Diabetes.

Table - 14

Family History of Diabetes		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	10	31	41
	% Within Nephropathy and Non Nephropathy	58.8%	37.3%	41%
No	Count	7	52	59
	% Within Nephropathy and Non Nephropathy	41.2%	62.7%	59%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 2.690 **Not Significant**

P = 0.6110

Family History of Kidney diseases and Diabetic Nephropathy

- In our study, 2 of the subjects among Nephropathy group had family history of Kidney Diseases while no such history was found among the Non Nephropathy group
- There was significance between family history of Kidney diseases and Diabetic Nephropathy in our Study

Table - 15

Family History of Kidney Diseases		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	2	0	2
	% Within Nephropathy and Non Nephropathy	11.8%	0%	2%
No	Count	15	83	98
	% Within Nephropathy and Non Nephropathy	88.2%	100%	98%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X2 = 9.964

P = 0.0410 **Significant**

Smoking and Diabetic Nephropathy

- Among the Nephropathy group, one subject was a smoker. In Non Nephropathy group, five of the subjects were smokers.
- There was no significance between smoking and Diabetic Nephropathy in our study

Table - 16

Smoking		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	1	5	6
	% Within Nephropathy and Non Nephropathy	5.9%	6%	6%
No	Count	16	78	94
	% Within Nephropathy and Non Nephropathy	94.1%	94.0%	94%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X2 = 0.001 **Not Significant**

P = 1.0000

DYSLIPIDEMIA IN OUR STUDY

TGL Dyslipidemia

- In our study 51% of subjects had Hypertriglyceridemia.
- It was 64.7% among Nephropathy group

Table - 17

TGL Dyslipidemia		Nephropathy		Total
		Yes	No	
Yes	Count	11	40	51
	% Within Nephropathy and Non Nephropathy	64.7%	48.2%	51%
No	Count	6	43	49
	% Within Nephropathy and Non Nephropathy	35.3%	51.8%	49%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 1.540 **Not Significant**

P = 0.8196

LDL Dyslipidemia

- 92% of the subjects had LDL Cholesterol Dyslipidemia in our study.
- It was 88.2% among Nephropathy group.

Table - 18

LDL Dyslipidemia		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	15	77	92
	% Within Nephropathy and Non Nephropathy	88.2%	92.8%	92%
No	Count	2	6	8
	% Within Nephropathy and Non Nephropathy	11.8%	7.2%	8%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 0.394 **Not Significant**

P = 0.9829

HDL Dyslipidemia

- 59% of subjects had Dyslipidemia in our study
- It was 64.7% among Nephropathy group

Table - 19

HDL Dyslipidemia		Nephropathy		Total
		Yes	No	
Yes	Count	11	48	59
	% Within Nephropathy and Non Nephropathy	64.7%	57.8%	59%
No	Count	6	35	41
	% Within Nephropathy and Non Nephropathy	35.3%	42.2%	41%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 0.276 **Not Significant**

P = 0.9913

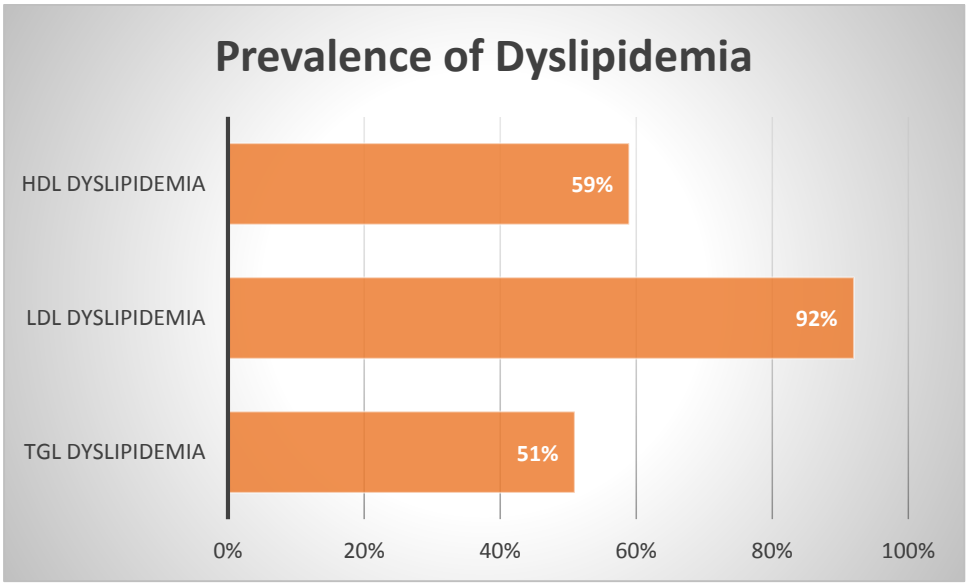

Chart – 7

Hypertension and Diabetic Nephropathy

- 10% of the subjects were found to be Hypertensive in our study.
- 41.2% of subjects among Nephropathy and 3.6% among Non-Nephropathy were Hypertensive.
- There was Significance between Hypertension and Diabetic nephropathy in our study**.**

Table - 20

Hypertension		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	7	3	10
	% Within Nephropathy and Non Nephropathy	41.2%	3.6%	10%
No	Count	10	80	90
	% Within Nephropathy and Non Nephropathy	58.8%	96.4%	90%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 22.120 **Significant**

P = 0.0002

GFR and Diabetic Nephropathy

- 24% of subjects in the study group had hyperfitration and 7% had Low GFR.
- Among the Nephropathy group, 52.9% had normal GFR, 5.9% had Hyperfiltration and 41.2% had low GFR.
- There was a significance between GFR and Diabetic Nephropathy in our study.

Table - 21

GFR Group (mL/min./ 1.73^2)		**Nephropathy**		**Total**
		Yes	**No**	
Low < 90	Count	7	0	7
	% Within Nephropathy and Non Nephropathy	41.2%	0%	7%
Normal 90 – 125	Count	9	60	69
	% Within Nephropathy and Non Nephropathy	52.9%	72.3%	69%
Hyper filtration >125	Count	1	23	24
	% Within Nephropathy and Non Nephropathy	5.9%	27.7%	24%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

P = 0.000 (< 0.001) **Significant**

Serum Creatinine and Diabetic Nephropathy

- The mean Serum Creatinine among Nephropathy group is 0.80 mg/dl and among the Non-Nephropathy group is 0.75 mg/dl

Table - 22

Nephropathy	Subject	Serum Creatinine Mean	Standard Deviation	Standard Error
Yes	**17**	**0.80**	**0.08**	**0.02**
No	**83**	**0.75**	**0.59**	**0.06**

P Value = 0.7290 **Not Significant**

Retinopathy and Diabetic Nephropathy

- Four of the subjects found to have Diabetic Retinopathy were among the Nephropathy group.
- There was a significance between Retinopathy and Diabetic Nephropathy in our study.

Table - 23

Retinopathy		**Nephropathy**		**Total**
		Yes	**No**	
Yes	Count	4	0	4
	% Within Nephropathy and Non Nephropathy	23.5%	0%	4%
No	Count	13	83	96
	% Within Nephropathy and Non Nephropathy	76.5%	100.0%	96%
Total	Count	17	83	100
	% Within Nephropathy and Non Nephropathy	100.0%	100.0%	100.0%

X^2 = 20.343 **Significant**

P = 0.0004 (< 0.001)

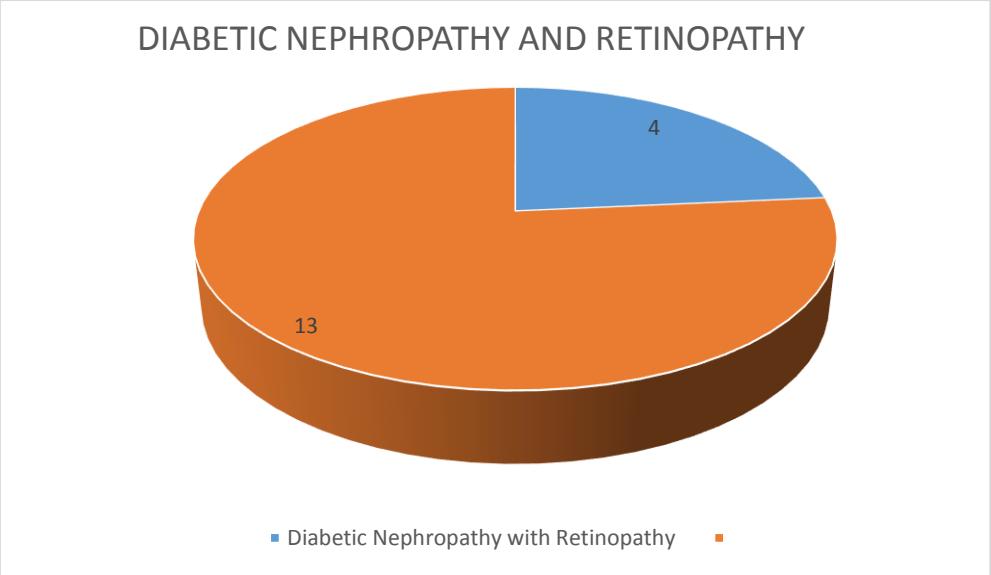

Chart - 8

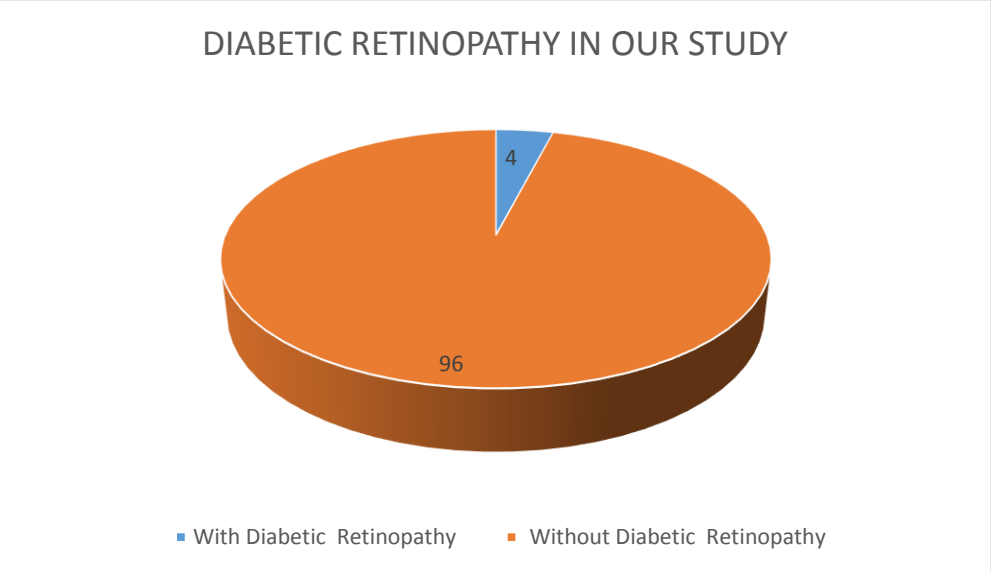

Chart – 9

DISCUSSION

The Prevalence of Diabetic Nephropathy in our study was compared with several protocol studies done previously in different places. A study called as CURES [2] Study was done in Chennai Urban Region in the year 2004 by Unnikrishnan et al. CURES[2] study showed 23.9% Prevalence of Microalbuminuria and 2.2% of Macroalbuminuria. In our study conducted in Chengalpattu Medical College, 16% had Microalbuminuria and 1% had Macroalbuminuria.

The lesser Prevalence in our study may be due to small sample size and exclusion of uncontrolled hypertensive subjects in our study. Further highly sensitive methods like Immunometric assays were used in Protocol studies.

Table - 24

Study	**Place and Year**	**Micro albuminuria (Stage 3 nephropathy)**	**Macro albuminuria (Stage 4 nephropathy)**	**Nephropathy (Total)**
Wirta et al [22]	Finland, 1995	29.00%	4%	33.00%
Collins et al [23]	Western Samoa, 1995	22%	3.90%	25.90%
Unnikrishnan et al [2]	Chennai, India, 2004	26.90%	2.20%	29.10%
This study	CMCH, Chengalpattu, 2019	16%	1%	17.00%

CORRELATION WITH VARIOUS RISK FACTORS

The correlation between various risk factors studied and Diabetic Nephropathy were analysed. The obtained results were compared with available data of Chennai Urban Rural Epidemiology study-CURES 45 [2] done by Unnikrishnan et al.

Correlation between age and diabetic nephropathy

Study	**Mean Age**
CURES [2] Study	51 ± 11
This study	50 ± 9

- The Mean age in our study group is 50 ± 9 years which is comparable with CURES study
- There was no correlation between age and Diabetic Nephropathy in our study
- CURES study showed has the Age advances, the risk of Diabetic Nephropathy increases.

Table - 25

AGE MEAN			
Study	**Nephropathy (Years)**	**Non Nephropathy (Years)**	**P value**
Unnikrishnan et al [2]	52 ± 11	50 ± 11	< 0.0001
This study	50 ± 9	47 ± 9	0.0002

Correlation between Gender and Diabetic Nephropathy

- In our study there was no significance correlation between Gender and Diabetic Nephropathy.
- The P value for Gender and Diabetic Nephropathy in our study is 0.9836 which is more than 0.05
- This is in comparison to Unnikrishnan et al study [2] which also showed no correlation between Gender and Diabetic Nephropathy.

Correlation between Family History of Diabetes and Diabetic Nephropathy

Table - 26

Family History of Diabetes		
Nephropathy	**Non Nephropathy**	**P value**
10	**31**	**0.6110**

- There was no significant correlation between family history of Diabetes and development of Diabetic Nephropathy in our study.

Correlation between Family History of Kidney Diseases and Diabetic Nephropathy

- There is a significant correlation between family history of Kidney diseases and development of Diabetic Nephropathy in our study.

Table - 27

Family History of Kidney Diseases		
Nephropathy	**Non Nephropathy**	**P value**
2	**0**	**0.0410**

Correlation between Smoking and Diabetic Nephropathy

- There is no significant correlation between Smoking and Diabetic Nephropathy in our study
- In CURES [2] Study there was a significant correlation found
- This could be due to lesser number of subjects who had the history of Smoking in our study.

Table - 28

Smoking		
Nephropathy	**Non Nephropathy**	**P value**
1	**5**	**1.0000**

Correlation between BMI and Diabetic Nephropathy

- There was a significance between BMI and Diabetic Nephropathy in our study
- Subjects who were overweight and obese showed significant development of Nephropathy

Table - 29

BMI Mean		
Nephropathy	**Non Nephropathy**	**P value**
27.46 ± 3	**24.38 ± 4**	**0.0028**

Correlation between Hypertension and Diabetic Nephropathy

- There is a highly significant correlation existing between hypertension and Diabetic Nephropathy in our study.
- This is comparable with results of CURES [2] Study
- In our study, uncontrolled hypertensive subjects were excluded so the bias is also less
- Our study and CURES [2] Study signifies the importance of screening early detection and effective control of blood pressure in Diabetes patients for the prevention of development of Nephropathy.

Table - 30

Hypertension			
Study	**Nephropathy**	**Non Nephropathy**	**P value**
Unnikrishnan et al [2]	59.70%	40.80%	< 0.001
This study	70 %	30%	0.0002

Correlation between Dyslipidemia and Diabetic Nephropathy

Table - 31

TGL Dyslipidemia		
Nephropathy	**Non Nephropathy**	**P value**
11	**40**	**0.8196**

Table - 32

LDL Dyslipidemia		
Nephropathy	**Non Nephropathy**	**P value**
11	**48**	**0.9913**

Table - 33

HDL Dyslipidemia		
Nephropathy	**Non Nephropathy**	**P value**
15	**77**	**0.9829**

- The Prevalence in total subjects who are newly detected Diabetes Mellitus for TGL, LDL and HDL Dyslipidemia are 51%, 92% and 59% respectively.
- The subjects presented with Nephropathy in our study had TGL, LDL and HDL Dyslipidemia with Prevalence of 64.7%, 88.2% and 64.7% respectively.
- Our study shows high Prevalence of Dyslipidemia in both newly detected Diabetes Mellitus as well as Diabetic Nephropathy Group

- There is no statistical significance however between Dyslipidemia and Diabetic Nephropathy in our study
- Various other studies showed significant correlation between Dyslipidemia and Nephropathy.
- This could be due to higher Prevalence of Dyslipidemia among the subjects in our study

Correlation between GFR and Diabetic Nephropathy

Table - 34

Subjects	**Hyperfiltration**	**Normal GFR**	**Low GFR**	**P value**
Nephropathy	**5.9%**	**52.9%**	**41.2%**	**0.000 (<0.01)**
Non-Nephropathy	**27.7%**	**72.3%**	**0%**	

- There is a highly significant correlation between GFR and Diabetic Nephropathy in our Study
- Among the Nephropathy group, 52.9% has a normal GFR. 5.9% had Hyperfiltration and 41.2% subjects among Nephropathy group had a Low GFR.
- Among the Non-Nephropathy group 27.7% subjects had Hyperfiltration and none had Low GFR.

Correlation between Retinopathy and Diabetic Nephropathy

Table - 35

STUDY	**Prevalence of retinopathy**
Mohan Rema et al [21]	**5.10%**
This study	**4%**

- There was a highly significant correlation between Retinopathy and Nephropathy in our study. All four patients who had Retinopathy were in Nephropathy group.
- The results of our study are comparable with Previous Protocol study in this regard**.**

CONCLUSION

1. Based on our study there is a significant prevalence of Diabetic Nephropathy in newly detected Type 2 Diabetes Mellitus patients. Hence, the results of our study signifies the importance of not only screening and treatment of Diabetes Mellitus but also for early screening of all new Type 2 Diabetic patients for Diabetic Nephropathy.
2. Statistical analysis in our study showed significant correlation between development of Nephropathy and risk factors like family history of Kidney disease, Body Mass Index especially being overweight or obese, systemic hypertension, Glomerular filtration Rate and Retinopathy.
3. Our study signifies the importance of normalizing the blood pressure at every stage of Diabetes Kidney Disease. Indeed, the control of Hypertension is more important in a Uremic Diabetic than the control of Hyperglycemia
4. There was no significant correlation between diabetic nephropathy and risk factors like age, gender, family history of diabetes, smoking and dyslipidemia in our study.
5. Urinary protein creatinine ratio and urinary dipstick test Micral test for microalbuminuria could be potent tools for screening for detection of diabetic nephropathy in newly detected diabetes patients. This is not only cost effective but also gives reliable results which is most suitable for developing countries like India.
6. Our study signifies the importance for screening for risk factors and early detection of diabetic nephropathy so that it prevents the diabetic population to

progress towards end stage renal disease. This is key to quality of life in Diabetic population, by reducing morbidity and economic burden of self and the country.

7. The presence of a microvascular complication like diabetic nephropathy in newly detected type 2 diabetes mellitus patients shows the importance of early detection of diabetic mellitus as well as screen for its complications, to have a tight glycemic control as well as blood pressure to reduce morbidity and mortality in diabetes mellitus and also to have a good quality of life

SUMMARY

Our study was aimed to detect the Prevalence of Diabetic Nephropathy in newly detected Type 2 Diabetes Mellitus patients and also to evaluate the correlation between various risk factors associated with development of Diabetic Nephropathy. The Prevalence found in our study was 17% for Diabetic Nephropathy at the time of diagnosis itself. Among these, 16% had Microalbuminuria, 1% had Macroalbuminuria.

The various risk factors that were found to have significant association in our study are Family History of Kidney Diseases, being overweight or Obese, Systemic Hypertension, Glomerular Filtration Rate and presence of Retinopathy.

The active screening, early Detection and treatment of not only Type 2 Diabetes Mellitus but also its complications is highly necessary. We have to take measures to create awareness among the Diabetes patients, to provide health Education and appropriate treatment for a quality life.

The effective Glycemic control, treatment of systemic Hypertension, prevention of other risk factors as suggested will prevent the development of Nephropathy and halts its progression.

ABBREVIATIONS

ACE	–	Angiotensin Converting Enzyme
ADA	_	American Diabetes Association
AER	–	Albumin Excretion Rate
AHA	–	American Heart Association
ARB	–	Angiotensin Receptor Blockers
BMI	–	Body Mass Index
CKD	–	Chronic Kidney Disease
DM	–	Diabetes Mellitus
DCCT	–	Diabetes Control and Complications Trial
ESRD	–	End Stage Renal Disease
GBM	–	Glomerular Basement Membrane
GFR	–	Glomerular Filtration Rate
HDL	–	High Density Lipoprotein
JNC	–	Joint National Committee
LDL	–	Low Density Lipoprotein
MDRD	–	Modification of Diet in Renal Disease
NADPH	–	Nicotinamide Adenine Dinucleotide Phosphate

PCR	–	Protein Creatinine Ratio
RPF	–	Renal Plasma Flow
TGL	–	Triglycerides
UAER	–	Urinary Albumin Excretion
UKPDS	–	United Kingdom Prospective Diabetic study

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PROFORMA

Name of the patient :

IP/OP No :

Age :

Sex :

Address :

H/O smoking :

H/O hypertension :

Family H/O diabetes :

Family H/O kidney disease :

Height :

Weight :

BMI :

Blood pressure :

Lipid profile :

TGL :

LDL :

HDL :

RFT

Blood urea :

Serum creatinine :

GFR :

Urine routine :

Sample 1

Sample 2

Sample 3

Urine culture :

Urine PCR :

Sample 1

Sample 2

Sample 3

Dipstick (MICRAL) test :

Sample 1

Sample 2

Sample 3

USG KUB :

Fundus examination :

MASTER CHART

S.NO.	NAME	AGE	SEX	H/O SMOKING	H/O HYPERTENS	FAMILY H/O DIA	FAMILY H/O KID DISEASES	HEIGHT(cm)	WEIGHT(Kg)	BMI Kg/m²	BP (mm Hg)	LIPID PROFIL			BLOOD UREA	S CREATININE	GFR ml/min /1.73	URINE ALBUM			Urine C/S	URINE PCR			DIPSTICK MICR TEST			USG KUB	FUNDS DR
												TGL	LDL	HDL				Sample 1	Sample 2	Sample 3		Sample 1	Sample 2	Sample 3	Sample 1	Sample 2	Sample 3		
	-	-	-	-	-	-	-	-	-	-	-	mg%	mg%	mg%	mg%	mg%	-	-	-	-	Growth	-	-	-	-	-	-	-	-
1	ABIRAMI	59	F	No	No	Yes	No	175	84	27.4	130/80	190	136	42	21	0.8	N	+	Nil	Nil	No	1	0	0	+	-	-	NSK	-
2	AMMU	64	F	No	No	No	No	160	71	27.0	110/70	167	110	31	27	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
3	ARUMUGAM	44	Male	No	No	No	No	155	61.5	25.0	100/60	165	110	38	24	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
4	ARUNA	64	F	No	No	No	No	157	73	29.6	110/90	177	128	42	29	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
5	BAKYALAKSHMI	51	F	No	No	No	No	155	44	18.3	110/70	131	96	42	29	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	⊕
6	BHARADAN	54	Male	No	No	No	No	165	54.5	20.0	100/70	126	128	37	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
7	BHOOPATHY	49	Male	No	No	Yes	No	155	50	20.0	120/80	132	92	38	28	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
8	BHUVANESHWARI	64	F	No	No	No	No	160	71	27.7	150/100	138	119	50	30	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
9	CHINNAPONNU	53	F	No	No	Yes	No	157	71	28.8	150/100	222	120	32	28	0.7	N	+	+	+	No	1	1	1	+	+	+	NSK	⊕
10	CHITRA	42	F	No	No	Yes	No	162	57	21.0	130/80	176	115	44	24	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
11	CHRISTINA	46	F	No	No	No	No	175	86.5	28.2	110/70	138	110	30	21	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
12	DHANALAKSHMI	55	F	No	No	No	Yes	170	93	32.1	110/60	142	119	48	28	0.7	L	+	+	+	No	1	1	1	+	+	+	NSK	-
13	DHINESH	44	Male	Yes	No	No	No	155	44	18.3	110/60	139	130	37	29	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
14	DURAIKANNU	42	Male	Yes	No	Yes	No	155	64	26.6	100/60	158	128	28	24	0.7	N	+	Nil	Nil	No	1	0	0	+	-	-	NSK	-
15	DURAISAMY	48	Male	No	No	No	No	155	75	31.2	130/80	201	130	42	28	0.8	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	⊕
16	GOMATHI	37	F	No	No	Yes	No	167	64	22.0	130/80	136	131	46	24	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
17	GOTHANDARAMAN	53	Male	No	No	Yes	No	165	79.5	29.0	100/60	215	130	36	25	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	⊕
18	GOVINDHAMMAL	63	F	No	No	No	No	160	70	27.3	130/70	138	110	44	28	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
19	GUNA	64	Male	No	No	Yes	No	162.5	72.7	27.0	100/60	201	130	48	27	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
20	ILAVARASI	46	F	No	No	No	No	155	75	31.2	160/100	186	128	43	22	0.8	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
21	ILAVIYA	54	F	No	No	No	No	175	86.4	28.2	110/60	190	140	41	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
22	KALA	45	F	No	No	No	No	155	64	26.6	140/80	138	110	34	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
23	KALIAMMAL	39	F	No	No	Yes	No	170	82	28.0	110/70	189	118	41	28	0.8	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
24	KANDASAMY	53	Male	No	No	No	No	162	82	31.0	120/80	201	120	36	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
25	KANNAMMAL	62	F	No	No	Yes	No	160	73	28.5	160/90	212	19	52	28	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
26	KANNAN	42	Male	Yes	No	Yes	Yes	165	73	26.7	150/100	155	110	38	29	0.8	N	+	+	+	No	1	0	1	+	+	+	NSK	-
27	KANNAYI	32	F	No	No	Yes	No	155	61.5	25.0	110/70	168	106	41	26	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
28	KARUPPASAMY	52	Male	No	No	No	No	175	86.4	28.0	100/70	198	128	36	26	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
29	KASTHURI	45	F	No	No	No	No	160	75	29.0	110/80	196	112	31	28	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
30	KATHIRVEL	62	Male	No	No	No	No	165	59	21.0	120/80	138	119	42	26	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-

31	KAVITHA	61	F	No	No	No	No	170	93	32.1	170/100	190	130	44	27	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
32	KEERTHANA	55	F	No	No	No	No	155	43	18.0	110/70	140	140	42	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
33	KRISHNAMMAL	58	F	No	No	No	No	170	70.5	24.4	170/100	165	96	33	27	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
34	KRISHNAN	44	Male	No	No	No	No	163	71	26.7	110/70	175	126	36	32	0.9	N	+	+	+	No	1	1	1	+	+	+	NSK	-
35	KUMARI	55	F	No	No	No	No	155	75	31.2	130/80	176	110	31	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
36	LAKSHMI	41	F	No	No	Yes	No	160	70.5	27.0	110/70	180	115	38	24	0.7	N	+	Nil	Nil	No	1	0	0	+	-	-	NSK	-
37	LATHA	42	F	No	No	No	No	175	86	28.0	120/80	178	105	45	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
38	LEELAVATHY	47	F	No	No	No	No	155	64	26.6	120/70	141	114	31	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
39	MALARKODI	44	F	No	No	Yes	No	160	61	24.0	110/80	137	110	42	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
40	MANIKAVASAGAM	52	Male	No	No	Yes	No	165	77.5	28.0	110/80	212	128	35	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
41	MANOGARI	51	F	No	No	No	No	165	68	25.0	110/70	139	126	38	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
42	MANOHARAN	51	Male	No	No	No	No	168	82	29.0	155/70	225	126	35	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
43	MUNIYAMMAL	34	F	No	No	No	No	155	68.5	28.5	110/90	172	110	33	26	0.7	N	+	+	+	No	1	1	1	+	+	+	NSK	-
44	MURUGAN	46	Male	No	No	Yes	No	160	62	24.2	110/60	160	115	39	24	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
45	MUTHU	56	Male	No	No	Yes	No	160	68	26.0	110/60	132	110	36	27	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
46	NAGAPUSHAM	49	F	No	No	Yes	No	152	46	19.0	110/80	136	94	44	23	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
47	NENKATESHWARAN	65	Male	No	No	Yes	No	170	95.5	33.0	100/80	226	140	48	28	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
48	NILOPER	48	F	No	No	No	No	165	59	21.0	120/80	131	109	43	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
49	PADMAVATHI	58	F	No	No	Yes	No	152	52	22.5	120/60	132	115	42	23	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
50	PADUVETTAMMAN	43	Male	No	No	No	No	175	84	27.4	100/70	136	110	38	25	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
51	PARAMESHWARI	52	Male	No	No	Yes	No	165	59	21.0	155/70	130	118	36	26	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
52	PARTHIBAN	56	Male	No	No	Yes	No	162	71	26.0	120/80	201	126	38	25	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
53	PATTAMMAL	33	F	No	No	No	No	152	52.5	22.0	110/70	134	110	42	25	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
54	PATTU	46	F	No	No	No	No	152	64	27.7	100/70	184	113	38	29	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
55	PERUMAL	56	Male	No	No	No	No	155	65.5	28.5	160/100	180	110	38	23	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
56	POKKISHAM	58	F	No	No	No	No	155	64	26.6	130/80	180	128	43	21	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
57	PRAKASH	32	Male	Yes	No	Yes	No	155	44	18.3	100/60	140	96	31	29	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
58	PRIYA	42	F	No	No	No	No	165	80	29.0	110/80	186	109	44	23	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
59	RAMASAMY	54	Male	No	No	Yes	No	155	59	24.5	100/30	138	130	32	24	0.8	N	+	+	+	No	1	1	1	+	+	+	NSK	-
60	RAMAYI	63	F	No	No	Yes	No	167	89	31.9	110/70	225	121	30	32	0.6	N	++	++	++	No	2	2	2	++	++	++	NSK	-
61	RAMU	32	Male	No	No	Yes	No	1570	55Kg	22.3	120/80	140	98	41	28	0.7	H	+	+	+	No	1	1	1	+	+	+	NSK	-
62	RAMYA	43	F	No	No	No	No	165	59	21.0	110/70	136	104	31	21	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
63	RAVICHANDRAN	49	Male	No	No	Yes	No	152	71	30.7	130/70	190	121	44	23	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
64	RENUKA	54	F	No	No	No	No	165	80	29.3	110/60	186	130	34	26	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
65	REVATHI	51	F	No	No	No	No	155	68.5	28.5	110/70	132	140	31	28	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
66	RUPA	42	F	No	No	Yes	No	160	59	23	150/100	135	105	36	28	0.7	N	+	+	+	No	1	1	1	+	+	+	NSK	-
67	RUTHRAN	37	Male	No	No	No	No	152	52	22.5	120/80	138	110	31	25	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
68	SANKARAN	47	Male	No	No	No	No	153	52	22.2	110/70	138	119	42	22	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
69	SARASWATHI	37	F	No	No	Yes	No	160	52.3	20.0	110/70	201	95	37	26	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
70	SASIKALA	43	F	No	No	No	No	152	61.5	26.6	130/80	190	110	31	23	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-

71	SATHYA	47	F	No	No	No	No	157	73	29.6	155/80	192	118	46	30	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
72	SAVEETHA	56	F	No	No	No	No	160	57	22.2	120/80	133	120	41	22	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
73	SHOBANA	44	F	No	No	No	No	160	75	29.0	130/80	216	130	48	22	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
74	SIVA	31	Male	No	No	Yes	No	155	50	20.8	110/70	142	159	32	28	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
75	SIVAGAMI	51	F	No	No	Yes	No	175	96	31.3	120/70	213	108	34	26	0.8	L	+	+	+	No	1	1	1	+	+	+	NSK	-
76	SIVAN	61	Male	No	No	Yes	No	160	84	32.0	180/70	212	115	37	27	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
77	SRIVIDYA	53	F	No	No	No	No	157	52.5	21.2	155/70	140	95	36	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
78	SUBASH	52	Male	No	No	Yes	No	160	68	26.5	160/90	165	128	45	28	0.8	N	+	+	+	No	1	1	1	+	+	+	NSK	-
79	SUBBU RATHINAM	36	F	No	No	Yes	No	160	57	22.2	110/70	128	110	30	26	6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
80	SUBBULAKSHMI	43	F	No	No	Yes	No	160	80	31.0	110/70	196	115	38	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
81	SUDHA	56	F	No	No	No	No	165	68	25.0	120/70	131	120	38	24	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
82	SUGANYA	44	F	No	No	No	No	157	66	26.7	120/80	130	126	42	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
83	SUKUMAR	41	Male	No	No	Yes	No	157	66	26.0	120/80	133	120	30	25	0.8	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
84	SULOCHANA	34	F	No	No	No	No	155	50	20.8	120/70	140	108	42	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
85	SUNIL	36	Male	No	No	Yes	No	165	68	25	120/80	160	116	34	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
86	SURESH	33	Male	Yes	No	No	No	157	52.5	21.2	120/70	144	126	33	26	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
87	SYED	58	Male	No	No	No	No	160	57	22.0	110/70	128	101	46	24	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
88	TAJUNEESHA	44	F	No	No	No	No	155	45.5	18.0	130/80	136	128	35	26	0.6	H	+	Nil	Nil	No	1	0	0	+	-	-	NSK	-
89	TAMILARASI	31	F	No	No	Yes	No	165	54.5	20.0	130/80	132	116	37	27	0.7	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
90	TAMILARASI	57	F	No	No	No	No	165	59	21.0	120/60	142	128	32	23	0.6	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
91	UMA	46	F	No	No	No	No	160	59	23.0	120/70	140	113	33	26	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
92	VALARMATHI	58	F	No	No	No	No	165	81	29.6	120/70	179	120	34	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
93	VALLI	36	F	No	No	No	No	180	91	28.0	110/70	138	108	45	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
94	VANI	33	F	No	No	Yes	No	160	66	25.0	130/80	200	121	37	29	0.6	H	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
95	VEDHAKUMARI	32	F	No	No	No	No	157	71	28.8	130/60	139	129	43	26	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
96	VENNILA	43	F	No	No	No	No	170	70.5	24.4	120/80	138	108	33	22	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
97	VIJAYALAKSHMI	41	F	No	No	No	No	172	93	31.0	110/70	190	117	41	28	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
98	VIJAYALAKSHMI	41	F	No	No	No	No	160	57	22.0	120/70	139	113	42	24	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
99	VINOTH	41	Male	Yes	No	Yes	No	160	61	24	110/60	172	128	36	28	0.7	N	Nil	Nil	Nil	No	0	0	0	-	-	-	NSK	-
100	YOGALAKSHMI	59	F	No	No	Yes	No	152	61.5	26.6	120/70	146	120	28	31	0.6	N	+	+	+	No	1	1	1	+	+	+	NSK	-

KEY TO MASTER CHART

SEX

F – Female

GFR

H – Hyperfiltration

N – Normal GFR

L – Low GFR

URINE ALBUMIN

+ = Urine Albumin present

_ = Urine Albumin absent

URINE PCR

0 = Normoalbuminuria

1 = Microalbuminuria

2 = Macroalbuminuria

DIPSTICK MICRAL TEST

+ = Microalbuminuria present

_ = Microalbuminuria not present

USG Kidney Size

NSK = Normal Sized Kidneys

FUNDUS

⊕ = Diabetic Retinopathy present

_ = Diabetic Retinopathy not present