

**A STUDY ON OUTCOMES OF HOSPITALIZATION IN  
PATIENTS ON MAINTENANCE HEMODIALYSIS IN A  
TERTIARY CARE CENTRE IN CHENNAI.**

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*In partial fulfillment of the regulations for the award of the degree of*

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**INSTITUTE OF INTERNAL MEDICINE**

**MADRAS MEDICAL COLLEGE**

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**MAY 2023**

## CERTIFICATE

This is to certify that the dissertation titled "A STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE CENTRE IN CHENNAI" submitted by **Dr. L. ARUN KHUMAR**, registration number **200120100506** appearing for **M.D. GENERAL MEDICINE** degree examination in May 2023, is a bonafide record of work done by him, under my guidance and supervision in partial fulfillment of requirements of The Tamilnadu Dr. M.G.R Medical University, Chennai. I forward this to The Tamilnadu Dr. M.G.R Medical University, Chennai.

### GUIDE



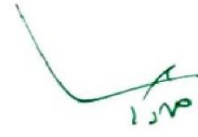
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This is to certify that the dissertation titled, "**A STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE CENTRE**" is a bonafide work done by **Dr. L. ARUN KHUMAR**, registration number : 200120100506 at Madras Medical College, Chennai in partial fulfilment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch-I) under my guidance and supervision during the academic year 2020-2023.



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## DECLARATION

I, **Dr. L. ARUN KHUMAR**, with registration number **200120100506** declare that, I carried out this work on, “ **A STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE CENTRE IN CHENNAI**” at the Institute of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai during the period of April 2022 to September 2022. I also declare that this bonafide work or any part of this work was not submitted by me or any others for any award, degree or diploma to any other University, Board either in India or abroad.

This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D degree examination in General Medicine.

Date : 14 / 12 / 2022

Place : Chennai

  
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# **ABSTRACT**

## **Introduction**

CKD is defined by the presence of kidney damage or decreased kidney function for three or more months, irrespective of the cause. These changes manifest in different ways depending on the underlying causes and the severity of the disease.

Hospitalizations is common for hemodialysis (HD) patients in view of the various comorbidities suffered by HD patients. By assessing the various etiology of hospitalisation in patients on maintenance hemodialysis and preventing or correcting those cause would result in greater reduction in hospitalisation , which has a greater impact on improving the quality of life of the patients.

## **Aim and objectives**

1. To study on the Etiology, Frequency, and outcome of Hospital admissions in patients on maintenance hemodialysis in End stage Renal Disease.
2. To analyse the various factors contributing to Hospitalisation and their correlation with outcomes of Hospitalisation in patients on Maintenance hemodialysis.

## **Material and methods**

In our study, 130 patients will be selected after obtaining informed consent as per the inclusion and exclusion criteria already mentioned. A detailed clinical history will be recorded and the patients will analysed on the basis of their demographic details, duration of illness frequency and duration of maintenance hemodialysis, presence of comorbidities, and their etiology, frequency ,duration of hospital stay and their outcome of hospitalization.



## **Results**

- Age and Cardiac abnormalities are the two major factors which increases the rate of hospitalisation among Chronic Kidney Disease patients on Maintenance Hemodialysis
- Hypoalbuminemia and Anaemia are the two majority factors which increases the duration of hospitalisation among CKD patients.
- Major etiologies for hospitalisation among patients on Maintenance Hemodialysis are Acute Pulmonary Edema and Anasarca.
- Mortality during the study period of 6 months is 6.15% , with causes being
  - 1.Acute pulmonary edema
  - 2.Acute Hemorrhagic stroke
  - 3.Uremic encephalopathy
  - 4.Sepsis.

## **Conclusion**

From our study, we conclude that regular cardiac evaluation and adequate correction of anaemia and Hypoalbuminemia in the patients of chronic kidney disease on maintenance hemodialysis, the frequency of hospitalisation and duration of stay can be very much reduced, which can very well improve the quality of life among these patients.

## INTRODUCTION

According to the Kidney Disease: Improving Global Outcomes (KDIGO) and Kidney Disease Outcomes Quality Initiative (KDOQI), CKD is a heterogeneous group of disorders marked by changes in kidney structure and function. These changes manifest in different ways depending on the underlying cause or causes and the severity of the disease. Genetic or sociodemographic predisposition, as well as the presence of illnesses that might start and spread renal disease, are risk factors for CKD. Kidney failure, the final stage of CKD, is indicated by significantly diminished kidney function or by the need for dialysis. Dialysis or kidney transplantation are typically used to treat chronic kidney failure, which is referred to as "end-stage kidney disease" (ESKD). Acute kidney injury (AKI) could make CKD more difficult and speed up its progression.

CKD is usually asymptomatic in its early stages. Symptoms appear in later stages in association with complications. In addition to commonly recognised hormonal and metabolic complications such as anaemia and hyperparathyroidism, CKD complications include increased risks for systemic drug toxicity, cardiovascular disease, infection, cognitive impairment, and impaired physical function.

Complications may also arise from the adverse effects of interventions used to prevent or treat the disease.

Hospitalizations is common for hemodialysis (HD) patients in view of the various comorbidities suffered by HD patients. By assessing the various etiology of hospitalisation in patients on maintenance hemodialysis and preventing or correcting those cause would result in greater reduction in hospitalisation , which has a greater impact on improving the quality of life of the patients.

## **AIMS AND OBJECTIVES OF THE STUDY**

1. To study on the Etiology, Frequency, and outcome of Hospital admissions in patients on maintenance hemodialysis in End stage Renal Disease.
2. To analyse the various factors contributing to Hospitalisation and their correlation with outcomes of Hospitalisation in patients on Maintenance hemodialysis.

## REVIEW OF LITERATURE

**Definition of CKD** — We agree with the KDOQI and KDIGO guidelines that CKD is defined by the presence of kidney damage or decreased kidney function for three or more months, irrespective of the cause.<sup>(1)</sup> The persistence of the damage or decreased function for at least three months is necessary to distinguish CKD from acute kidney disease. Kidney damage refers to pathologic abnormalities, whether established via kidney biopsy or imaging studies, or inferred from markers such as urinary sediment abnormalities or increased rates of urinary albumin excretion. Decreased kidney function refers to a decreased glomerular filtration rate (GFR), which is usually estimated (eGFR) using serum creatinine and one of several available equations.

**Kidney damage** — One of the following clinical signs is often present to indicate kidney damage:

**Albuminuria** — The most often used indicator of kidney impairment is albuminuria. Increased glomerular permeability to macromolecules is shown by albuminuria.<sup>(2)</sup> Albuminuria can be a sign of either primary renal disease or systemic illness affecting the kidneys. Albuminuria in particular could be a sign of extensive endothelial dysfunction, like that which is present in conditions like obesity, smoking, hypertension, diabetes, and hypercholesterolemia.

Regardless of the reason, albuminuria beyond this level should be included in the classification of CKD. Even when eGFR is normal, those with urine ACR >30 mg/g

(or similar) have a considerably higher risk of cardiovascular death, all-cause mortality, ESKD, AKI, and CKD development.

- **Urinary sediment abnormalities** – Urinary sediment abnormalities such as red or white blood cell casts may indicate the presence of glomerular injury or tubular inflammation.

- **Imaging abnormalities** – Kidney damage may be detected by the presence of imaging abnormalities such as polycystic kidneys, hydronephrosis, and small and echogenic kidneys.

- **Pathologic abnormalities** – A kidney biopsy may reveal evidence for glomerular, vascular, or tubulointerstitial disease.

**Decreased GFR** : Glomerular filtration rate (GFR) is the best measure of kidney function overall, and a falling GFR is a sign of renal disease that is progressing.<sup>(3)</sup> Age and sex, dietary protein consumption, and perhaps other variables affect measured GFR in healthy persons. The generally accepted threshold defining a decreased GFR is less than 60 mL/min per 1.73 m<sup>2</sup>, and kidney failure is defined as a GFR of less than 15 mL/min per 1.73 m<sup>2</sup> or treatment with dialysis. These definitions are based on clearance measurements in healthy individuals and individuals with kidney disease.

**GFR estimation** - Commonly utilised GFR estimation equations include the Modification of Diet in Renal Disease (MDRD) Study equation and the 2009 Chronic

Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.<sup>(4-6)</sup> Both formulae incorporate serum creatinine as well as age, sex, and race. The formulae have been altered to account for various geographic areas and racial/ethnic groupings.

The 2021 CKD-EPI creatinine equation, which was later constructed using the same data as the 2009 equation but without a race-specific component, is currently the suggested equation for calculating GFR. <sup>(7)</sup>

Any one of the following formulae may be used to calculate the 24 hour creatinine clearance or the serum creatinine to determine the GFR.

**1. The MDRD formula (Modification of Diet in Renal Disease)**

$$eGFR = 186 \times (Pcr)^{-1.154} \times (\text{Age in years})^{-0.203}$$

Multiply by 0.742 for women.

Multiply by 1.21 for Blacks.

Pcr– Plasma Creatinine in mg/dl

**2 . Cockcroft – Gault formula**

$$\text{Estimated Creatinine Clearance} = (140 - \text{Age}) \times \text{Wt in Kg} \times 72 \times \text{Serum Creatinine}$$

Multiply it by 0.85 for females

**3. Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation**

$$eGFR = 141 \times \min(\text{SCr}/k, 1)^\alpha \times \max(\text{SCr}/k, 1)^{-1.209} \times 0.993^{\text{Age}}$$

• Multiply it by 1.018 for females

• Multiply it by 1.159 for blacks

• SCr - serum creatinine (mg/dL)

- $k$  is 0.7 for females and 0.9 for males
- $\alpha$  is -0.329 for females and -0.411 for males
- $\min$  denotes the minimum of  $SCr/k$  or 1
- $\max$  denotes the maximum of  $SCr/k$  or 1

Cystatin C is a different endogenous filtration marker from creatinine that may be more advantageous for estimating GFR since its non-GFR determinants are more indicative of future cardiovascular disease and death. Creatinine and cystatin C combined allow for more precise GFR calculations and risk assessments.<sup>(8,9)</sup>

## **Staging of CKD :-**

The goal of CKD staging is to direct care, including classification of risk for CKD complications and progression. The amount of monitoring, patient education, and suitable therapies are all influenced by risk classification. The following factors are taken into consideration for staging CKD in individuals who meet the aforementioned criteria for diagnosis:

- Cause of illness
- Six GFR (G stages) categories
- Three categories of albuminuria (A stages)

It is more accurate to stage CKD patients in relation to their aetiology, GFR, and albuminuria in order to reduce their chance of developing the serious consequences.

Compared to excessive albuminuria, low GFR is a better predictor of CKD consequences.

**Cause of disease** — Identifying the cause of kidney disease (eg, diabetes, drug toxicity, auto-immune diseases, urinary tract obstruction, kidney transplantation, etc.) enables specific therapy directed at preventing further injury. In addition, the cause of kidney disease has implications for the rate of progression and the risk of complications.

It can be difficult to ascertain the cause of kidney disease. In clinical practice, CKD is most often discovered as decreased eGFR during the evaluation and management of other medical conditions.

**GFR** — The glomerular filtration rate (GFR; G-stages) follow the original CKD classification scheme :

- G1 – GFR >90 mL/min per 1.73 m<sup>2</sup>
- G2 – GFR 60 to 89 mL/min per 1.73 m<sup>2</sup>
- G3a – GFR 45 to 59 mL/min per 1.73 m<sup>2</sup>
- G3b – GFR 30 to 44 mL/min per 1.73 m<sup>2</sup>
- G4 – GFR 15 to 29 mL/min per 1.73 m<sup>2</sup>
- G5 – GFR <15 mL/min per 1.73 m<sup>2</sup> or treatment by dialysis

Since the original KDOQI classification was published, stage 3 CKD (a GFR of 30 to 59 mL/min per 1.73 m<sup>2</sup>) has been subdivided into GFR stages 3a and 3b to more accurately reflect the continuous association between lower GFR and risk for mortality and adverse kidney outcomes. Patients receiving treatment with dialysis are sub-classified as GFR stage 5D to highlight the specialised care that they require.



**Albuminuria** — The three albuminuria stages follow familiar definitions of normal, moderately increased (formerly called "microalbuminuria"), and severely increased (formerly called "macroalbuminuria" and nephrotic range) albuminuria :

- A1 – ACR <30 mg/g (<3.4 mg/mmol)
- A2 – ACR 30 to 299 mg/g (3.4 to 34.0 mg/mmol)
- A3 – ACR ≥300 mg/g (>34.0 mg/mmol)

Since the initial KDOQI classification method was released, albuminuria staging has been added to GFR staging. Due to the gradual rise in mortality risk, CKD and ESKD development, and ESKD progression with greater levels of albuminuria, irrespective of eGFR, without an obvious threshold value, albuminuria staging has been added.

Even when GFR is >60 mL/min per 1.73 m<sup>2</sup>, the risk increases significantly for urine ACR values 30 mg/g, which is in line with the current threshold value for albuminuria (30 mg/g) as a symptom of kidney injury. Urine ACR values between 10 and 29 mg/g ("high normal" albuminuria) are similarly associated with an elevated risk, indicating that levels below 30 mg/g may also require more attention.

				<b>Albuminuria</b> stages, description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<3 mg/mmol	3-29 mg/mmol	≥30 mg/mmol
<b>GFR</b> stages, descriptions and range (ml/min per 1.73m <sup>2</sup> )	Stage 1 (G1)	Normal or high	≥90			
	Stage 2 (G2)	Mildly decreased	60-90			
	Stage 3 (G3a)	Mildly to moderately decreased	45-59			
	Stage 3 (G3b)	Moderately to severely decreased	30-44			
	Stage 4 (G4)	Severely decreased	15-29			
	Stage 5 (G5)	Kidney failure	<15			

CKD categorization to determine a person's prognosis — The staging approach will assist doctors in choosing the best course of treatment and level of care for patients with established CKD. The use of risk-prediction tools can result in a patient's risk being predicted more accurately. The aetiology of kidney illness as well as additional variables (such as age, cholesterol levels, smoking status, and others) should be taken into account when evaluating prognosis in addition to eGFR and albuminuria.

# **EPIDEMIOLOGY**

## **WORLDWIDE IMPACT OF CKD**

The Global Burden of Disease (GBD) study found that there were 698 million instances of chronic kidney disease (CKD) in 2017, with a 9 percent global prevalence rate for adults (8.5 to 9.8).<sup>(9)</sup> Of this, CKD G1 to G2 made up 5%, G3 accounted for 3.9%, G4 for 0.16 %, and G5 for 0.07 % (excluding patients receiving dialysis or kidney transplant recipients). Dialysis made up 0.04 % and kidney transplantation made up 0.01 %. The global incidence of CKD, however, may be as high as 13.4% (11.7 to 15.1), according to a meta-analysis of 100 studies involving 6,980,440 individuals, with a prevalence of CKD stages 3 to 5 of 10.6% (9.2 to 12.2). In the GBD study conducted in 2017, the age-standardised prevalence of CKD in females (9.5 percent [8.8 to 10.2]) was 1.29 (1.28 to 1.30) times higher than in males (7.3 percent [6.8 to 7.9]). However, males (13.7 percent [12.6 to 14.9]) had an age-standardized incidence of dialysis and transplantation that was 1.47 (1.46 to 1.48) times higher than that of females (8.6 percent [7.9 to 9.3]). Males are more likely to begin kidney replacement therapy (KRT), in part because CKD progresses more quickly in men.

## **CKD IN INDIA**

- The incidence of ESRD is 785 per million people in Delhi, where the prevalence of chronic kidney disease is roughly 7852 per million people (or 10% of all CKD).
- Hypertension (14–22%) and Diabetes Mellitus (30–40%) are the two main causes.
- In South India, the leading causes are chronic glomerulonephritis (17.4%), chronic interstitial nephritis (20.4%), and hypertension nephropathy (11%).

## **PATHOGENESIS OF CKD<sup>(10)</sup> :-**

The pathophysiology of CKD is complex and is dependent on the primary cause.

After an acute or chronic insult, numerous common pathways are activated to perpetuate glomerular and tubulointestinal injury. There are two types of injuries. They are

1. Hemodynamic injury
2. Non-hemodynamic injury

### **HEMODYNAMIC INJURY**

The process progresses linearly as kidney mass decreases, resulting in an increase in renal plasma flow and hyperfiltration of the remaining nephrons. As a result of proteinuria and increasing glomerular damage brought on by RAAS-mediated glomerular hypertension and systemic hypertension, the afferent arteriolar tone is lower than the efferent tone. The increase in intraglomerular pressure and filtration pressure caused by the net efferent vasoconstriction hastens hyperfiltration damage. Renin, which is released from the Juxta Glomerular Apparatus with the loss of functional nephrons, transforms angiotensinogen to angiotensin I, which is then converted into angiotensin II by the Angiotensin Converting Enzyme. The main cause of glomerular hemodynamic maladaptation is angiotensin II. The potent vasoconstrictor in the post glomerular arterioles is angiotensin II. Through the use of aldosterone, it indirectly enhances distal tubular sodium absorption while directly increasing proximal tubular sodium absorption. It also stimulates the posterior pituitary, which releases antidiuretic hormone. When perfusion is reduced, these mechanisms from the auto-regulation process keep the GFR constant. Clinical proteinuria and glomerular damage result from the rise in glomerular hypertension,

which also increases the filtration fraction and the radius of the holes in the glomerular basement membrane.

### **NON-HEMODYNAMIC INJURY**

This maladaptive pathways lead to inflammation and fibrosis of kidney. Angiotensin II level is increased in almost every compartment of the kidney such as mesangial cell, podocytes, endothelial cells, the urinary space (Bowman's capsule) and the tubulointerstitium. As a result of increased Angiotensin II , several growth factors and their receptors like Connective Tissue Growth Factor, Epidermal Growth Factor, Vascular Endothelial Growth Factor(VEGF), Insulin Like Growth Factor-1, PDGF, Transforming Growth Factor- $\beta$  and Monocyte Chemotactic Protein -1 are up-regulated. This leads to over production of extracellular matrix by upregulating other factors such as Fibronectin and Type1 procollagen, PAI-1. In addition , excess adhesion molecules like Integrins or Vascular Cell Adhesion Molecule 1 result in cell proliferation, adhesion of these cells, extra cellular matrix accumulation, and functional changes ultimately resulting in fibrosis.

Nearly all types of renal disease progress in part due to inflammation, which is partially mediated by RAAS. By activating Endothelin-1, Angiotensin II recruits macrophages and T cells and boosts the activity of activated B cells' Nuclear Factor  $\kappa$  light chain enhancer. They will then release cytokines, which will increase the inflammation. An additional harm caused by free radical oxygen species leads to further inflammation and fibrosis. Primary RAAS stimulation causes a cascade of events that start with inflammation, are sped up by cell and matrix buildup, and are then made worse by their adherence, leading to tubulointerstitial necrosis and

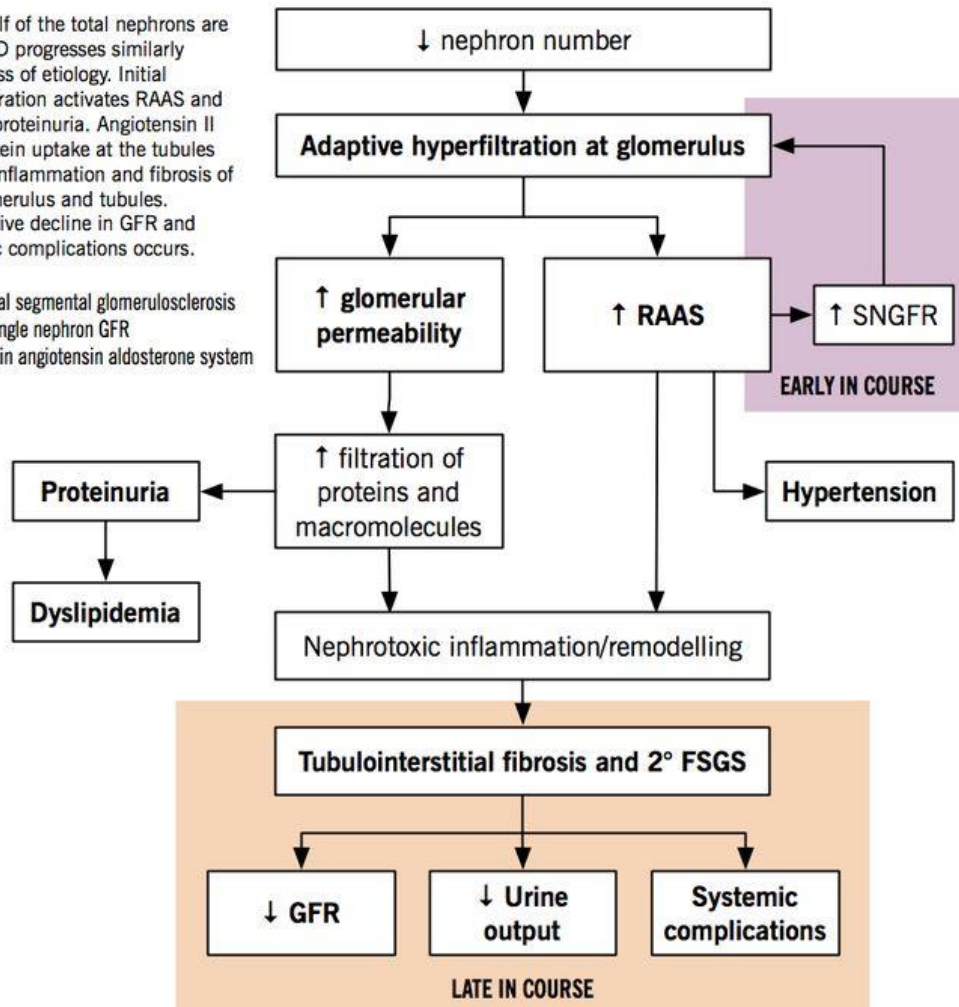
glomerulosclerosis. This creates a vicious loop of ongoing RAAS activation, proteinuria, and a worsening course of CKD.

## Pathogenesis of chronic kidney disease

Eric Wong

Once half of the total nephrons are lost, CKD progresses similarly regardless of etiology. Initial hyperfiltration activates RAAS and causes proteinuria. Angiotensin II and protein uptake at the tubules causes inflammation and fibrosis of the glomerulus and tubules. Progressive decline in GFR and systemic complications occurs.

**FSGS** Focal segmental glomerulosclerosis  
**SNGFR** Single nephron GFR  
**RAAS** Renin angiotensin aldosterone system



## PROGRESSION OF CHRONIC KIDNEY DISEASE<sup>(11)</sup>

The progression of CKD is strongly associated with progressive sclerosis of glomeruli irrespective of the nature of the underlying nephropathy. Both extra and intra glomerular cells contribute to the glomerulosclerosis.

## **INTRA GLOMERULAR CELLS**

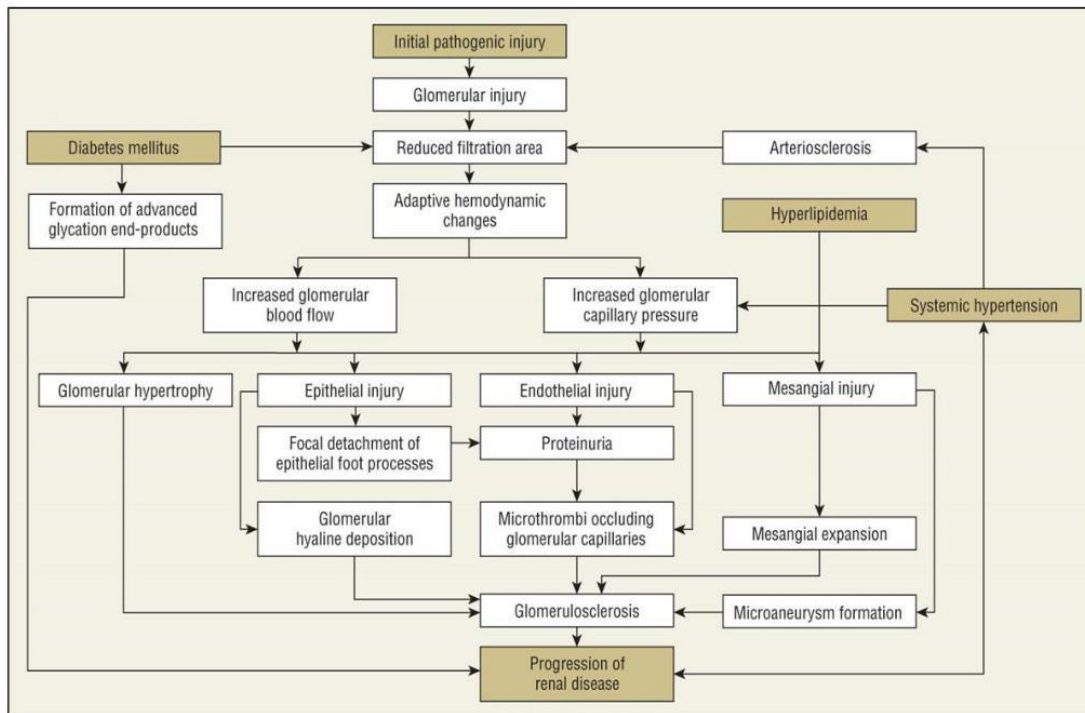
### **ROLE OF GLOMERULAR ENDOTHELIAL CELLS:**

Glomerular endothelium plays a vital role in preserving the integrity of the vascular beds of glomeruli. They are the ones initially exposed to the damage caused by hemodynamic injury, metabolic injury, immunologic injury. This endothelial injury is related with the loss of their anticoagulant and anti-inflammatory characteristics and gain of procoagulant and inflammatory properties attributing to attraction and activation of platelets and microthrombus formation. It is then further associated with the initiation of glomerular micro inflammation and infiltration of glomerular tufts by monocytes. Platelets and monocytes interact with mesangial cells resulting in the production of extra cellular matrix(ECM).

### **ROLE OF MESANGIUM :**

Following a series of endothelial injuries, infiltrating monocytes engage with mesangial cells and stimulate them either directly through cell-to-cell contact or by releasing mitogens such platelet-derived growth factor. Mesangial cells are activated and multiply as a result of the transcription factor kappa B (NF-B), as well as a number of kinases, including Mitogen Activated Protein Kinase (MAPK), Jun N-terminal Kinase, and stress activated protein kinase.

### Proposed mechanisms for progression of renal disease



Under the influence of fibrogenic growth factors like TGF-1, activated mesangial cells have the capacity to transform back into myofibroblasts that express markers like smooth muscle actin and produce Type III interstitial collagen, which is not a typical component of the glomerular extracellular matrix. The balance between the increased extracellular matrix (ECM) and its breakdown by glomerular collagenases and metalloproteinases determines whether glomerular and mesangial sclerosis will resolve.

### ROLE OF GLOMERULAR EPITHELIAL CELLS

Stretching along the glomerular basement membrane may result from podocytes' inability to proliferate after injury. This reveals the glomerular basement membrane denuded patches. Segmental glomerulosclerosis and capsular adhesions are created as



a result of the interaction between the denuded glomerular basement membrane and parietal epithelial cells.

### **EXTRA GLOMERULAR CELLS:**

#### **PLATELETS:**

The activation of platelets and their byproducts will stimulate the mesangial cell and aid in its sclerosis by initiating the coagulation cascade.

Thrombin activates TGF-1, which advances the production of mesangial ECM.

Upregulation of plasminogen activator inhibitor-1 in glomeruli with injury can result in a buildup of extracellular matrix and glomerulosclerosis.

The balance between thrombotic-antiproteolytic and anticoagulant-proteolytic activities determines the severity of glomerulosclerosis.

### **BONE MARROW – DERIVED CELLS**

Hematopoietic stem cells are involved in normal turnover of the glomerular cell and response of glomeruli to injury.

### **MONOCYTE, LYMPHOCYTES, AND MACROPHAGES**

The release of Growth Factors, Cytokines and Procoagulant factors by the lymphocytes, monocytes and macrophages is likely to contribute to the progression glomerulosclerosis.

## **TUBULO INTERSTITIAL SCARRING:**

1. Inflammation of the tubulointerstitium.
2. Proliferation of the interstitial fibroblasts.
3. Excessive deposition of the interstitial extracellular matrix

The pathophysiology of tubulointerstitial fibrosis depends on renal tubular cells.

Growth factors, angiotensin II, and other hormones that leak from damaged glomeruli may stimulate tubular cells.

When tubular cells are stimulated, they release chemotactic substances that can draw monocytes and other inflammatory cells to the interstitium and tubules, activating renal fibroblasts in the process. Renal fibroblasts that have been stimulated develop myofibroblast properties and multiply in the peritubular and periglomerular areas. The activation of plasmin and matrix metalloproteinases provides the foundation for the resolution of extracellular matrix deposition. Scarring of the tubulointerstitial tissue will ensue from inhibiting these two proteolytic enzymes.

## **VASCULAR SCLEROSIS**

The process of renal scarring includes vascular sclerosis. The progression of CKD is associated with renal arteriolar hyaline sclerosis, which can occur at an early stage of the disease even in the absence of severe hypertension. While hyaline sclerosis of the post glomerular arterioles may increase interstitial ischemia and lead to fibrosis, afferent arteriole hyaline sclerosis may be associated with the aetiology of glomerulosclerosis.

Tubular cells and fibroblasts are stimulated to create ECM components and to decrease their collagenolytic activity as a result of ischemia and the resulting hypoxia.

## CLINICAL PRESENTATION

Patients with CKD may exhibit symptoms and signs like edoema or hypertension that are directly related to their impaired kidney function. Although many may not exhibit any clinical symptoms, renal disease is frequently found in these people when an abnormal urinalysis, higher serum creatinine, or reduced estimated glomerular filtration rate (eGFR) are found by accident (when such tests are obtained as part of routine evaluation or for a possibly unrelated disorder). Furthermore, radiographic abnormalities (such as tiny and echogenic [by ultrasound] kidneys showing chronic damage, numerous bilateral renal cysts with enlarged kidneys suggesting polycystic kidney disease) may be seen on imaging carried out for some other purpose.

Patients may also exhibit signs and/or symptoms of protracted kidney failure, such as fatigue and easily becoming tired, anorexia, vomiting, pruritus, and, in very severe stages, encephalopathy or seizures, depending on the length and severity of CKD.

Rarely seen with CKD alone, abnormally low urine output (i.e., oliguria or anuria) always reflects at least some aspect of acute renal damage (AKI). Patients with AKI superimposed on CKD may exhibit oliguria or anuria, as can be seen in a patient with chronic blockage who experiences acute urine retention.

Similar to this, patients with underlying CKD may experience anuria due to severe or protracted shock, bilateral urethral blockage, or bilateral renal arterial occlusion. The most frequent test results in CKD patients are elevated blood urea nitrogen and serum creatinine levels. Proteinuria and/or aberrant red or white blood cells might be seen on urine microscopy as a result of urine tests. Anemia, hyperphosphatemia, hyperkalemia, metabolic acidosis, hypocalcemia, and increased

parathyroid hormone are additional typical laboratory abnormalities that may be present in the clinical presentation (PTH). The degree of abnormalities depends on how severe the CKD is. In CKD patients with an eGFR greater than 45 mL/min/1.73 m<sup>2</sup>, hyperphosphatemia is infrequent. Contrarily, PTH can be slightly high even with a slight decline in eGFR (i.e., 50 to 60 mL/min/1.73 m<sup>2</sup>).

## **Etiology of CKD<sup>(12)</sup>**

### **Diseases - Percentage**

Diabetes mellitus	-	31.2%
Hypertension	-	14.1%
Glomerulonephritis(GN)	-	14.4%
Tubulo-interstitial Nephritis.	-	7%
Hereditary or cystic diseases.	-	2.1%
Miscellaneous	-	15.9%
Unknown	-	15.3%

## **MANAGEMENT OF CHRONIC KIDNEY DISEASE:-**

### **GENERAL MANAGEMENT OF CHRONIC KIDNEY DISEASE<sup>(13)</sup>**

The general management of the patient with CKD involves the following issues :

- Treatment of reversible causes of kidney failure
- Preventing or slowing the progression of kidney disease
- Treatment of the complications of kidney failure
- Adjusting drug doses when appropriate for the level of estimated

glomerular filtration rate (eGFR)

- Identification and adequate preparation of the patient in whom kidney

replacement therapy will be required

**Reversible causes of kidney failure** — In addition to exacerbation of their original kidney disease, patients with CKD with a recent decrease in kidney function may be suffering from an underlying reversible process, which, if identified and corrected, may result in the recovery of function.

**Decreased renal perfusion** — Hypovolemia (such as vomiting, diarrhea, diuretic use, bleeding), hypotension (due to myocardial dysfunction or pericardial disease), infection (such as sepsis), and the administration of drugs which lower the eGFR (such as nonsteroidal antiinflammatory drugs [NSAIDs] and angiotensin-converting enzyme [ACE] inhibitors or angiotensin receptor blockers [ARBs]) are common causes of potentially reversible declines in kidney function.

In patient with CKD, hypovolemia should be diagnosed by history and physical examination rather than the urine sodium or fractional excretion of sodium. The normal response to renal hypoperfusion is to lower the urine sodium concentration (<25 mEq/L) and the fractional excretion of sodium (<1 percent in patients with advanced kidney failure) to very low levels. However, the superimposition of a prerenal process among patients with CKD may not result in the expected low values, since the tubules in the diseased kidney are unable to reabsorb sodium so efficiently. If hypovolemia is suspected, a judicious trial of fluid repletion may result in the return of kidney function to the previous baseline.

**Administration of nephrotoxic drugs** — The administration of drugs or diagnostic agents that adversely affect kidney function is a frequent cause of worsening kidney function. Among patients with CKD, common offenders include aminoglycoside antibiotics (particularly with unadjusted doses), NSAIDs, and radiographic contrast material.

**Urinary tract obstruction** — Urinary tract obstruction should always be considered in the patient with unexplained worsening kidney function, although, in the absence of prostatic disease, it is much less common than decreased renal perfusion. Renal ultrasonography is often performed to exclude urinary tract obstruction in patients with an unexplained elevation in the serum creatinine.

**Blood pressure control** — • More intensive blood pressure lowering may reduce mortality in patients with CKD (whether they have proteinuria or not), even though there is no benefit on kidney endpoints among patients without proteinuria. More intensive blood pressure lowering may reduce mortality in patients with CKD

(whether they have proteinuria or not), even though there is no benefit on kidney endpoints among patients without proteinuria<sup>(14)</sup>.

**Slowing the rate of progression** — It's possible that secondary factors unrelated to the activity of the primary disease are at least partially to blame for the advancement of CKD. The primary causes of the adaptive hyperfiltration, which results in glomerular scarring, are believed to be intraglomerular hypertension and glomerular hypertrophy (glomerulosclerosis). Systemic hypertension, hyperlipidemia, metabolic acidosis, and tubulointerstitial illness may also be contributing factors.

Secondary focal segmental glomerulosclerosis is the main histologic sign of hemodynamically driven renal damage<sup>(15)</sup>. Thus, even in primary tubulointerstitial disorders such as reflux nephropathy, proteinuria frequently coexists with CKD in patients.

**Additional therapies in proteinuric patients** — Treatment with an ACE inhibitor or ARB and achieving the target blood pressure are the main goals of therapy to limit the rate of progression in proteinuric individuals with CKD, regardless of the treatment of the underlying disease. These people may also benefit from SGLT2 inhibitor therapy. <sup>(16,17)</sup>

### **Treatment of the complications of kidney failure :-**

A wide range of disorders may develop as a consequence of the loss of kidney function. These include disorders of fluid and electrolyte balance, such as volume overload, hyperkalemia, metabolic acidosis, and hyperphosphatemia, as well as abnormalities related to hormonal or systemic dysfunction, such as anorexia, nausea,

vomiting, fatigue, hypertension, anemia, malnutrition, hyperlipidemia, and bone disease.

**Volume overload** — Homeostatic mechanisms typically keep sodium and intravascular volume balance until the eGFR drops below 10 to 15 mL/min/1.73 m<sup>2</sup>. Despite being in relative volume balance, the patient with mild to severe CKD is less able to react to a quick intake of salt and is consequently more susceptible to fluid overload.

The combination of dietary salt restriction and diuretic medication, typically with a loop diuretic administered daily, works well for patients with CKD and volume overload. Limiting sodium consumption, according to some researchers, may also slow the progression of CKD by reducing intraglomerular pressure<sup>(19)</sup>. We concur with the 2012 Kidney Disease: Improving Global Outcomes (KDIGO) recommendations that sodium consumption in persons with CKD should be limited to <2 g /day, unless contraindicated

**Hyperkalemia**<sup>(20,21)</sup> — In patients with kidney disease, potassium excretion may typically be kept at levels close to normal as long as aldosterone secretion and distal flow are kept constant. Therefore, oliguric patients or those with other issues like a high-potassium diet, accelerated tissue breakdown, or hypoaldosteronism are more likely to develop hyperkalemia (due in some cases to the administration of an ACE inhibitor or ARB). In advanced CKD, impaired potassium uptake by cells may also play a role in the development of hyperkalemia. In individuals with CKD, a number of interventions may help prevent hyperkalemia. These include following a low-potassium diet (e.g., 40 to 70 mEq/day) and, if at all feasible, refraining from using medications like NSAIDs that increase the serum potassium content.



**Metabolic acidosis**<sup>(22,23)</sup> — Patients with CKD have a rising propensity to retain hydrogen ions. With the serum bicarbonate concentration often stabilising between 12 and 20 mEq/L and hardly ever going below 10 mEq/L, this can result in a progressive metabolic acidosis. Supplemental bicarbonate may be used to treat metabolic acidosis. Because sodium is added when bicarbonate is delivered, careful volume status monitoring is necessary during bicarbonate supplementation.

**Mineral and bone disorders (MBD)** — Hyperphosphatemia is a frequent CKD side effect. Because of the decreased phosphate load filtered, phosphate retention tends to start early in renal illness. Although hyperphosphatemia is a very late event and this problem starts out modest, phosphate retention is closely linked to the common development of secondary hyperparathyroidism. The oversecretion of parathyroid hormone (PTH), which can rectify both hyperphosphatemia and hypocalcemia, is initially appropriate from the perspective of calcium and phosphate balance. As a result, patients with an eGFR of >30 mL/min/1.73 m<sup>2</sup> typically maintain phosphate balance and a normal serum phosphate content<sup>(24)</sup>. Secondary hyperparathyroidism and the onset of renal osteodystrophy are the price to be paid. In patients with CKD, dietary phosphate restriction and oral phosphate binders may prevent the onset of secondary hyperparathyroidism.

With increasing CKD, changes in bone structure are virtually always observed. Osteitis fibrosa, osteomalacia, and adynamic bone disease are the three main kinds of renal bone disease<sup>(25,26)</sup>.

Since hormonal anomalies are one of the earliest indicators of aberrant mineral and bone metabolism with increasing CKD, PTH levels should be evaluated in such individuals. In patients with predialysis CKD, dietary phosphate restriction, oral

phosphate binders, and the administration of calcitriol (or vitamin D analogues) to directly decrease PTH secretion are the main methods for the prevention and/or treatment of osteitis fibrosis.

The kidney is primarily responsible for producing circulating calcitriol (1,25-dihydroxyvitamin D), which is vitamin D's most active metabolite. Circulating calcitriol levels are often significantly decreased in patients with ESKD and start to decline when the eGFR is 40 mL/min/1.73 m<sup>2</sup>. Phosphate retention also lowers calcitriol synthesis in addition to the decrease of functional renal mass.

Calcimimetics are substances that allosterically boost the parathyroid gland's calcium-sensing receptor's sensitivity to calcium. The main factor controlling PTH release and hyperplasia from the parathyroid gland is the calcium-sensing receptor. By means of mechanisms that are complimentary to and possibly synergistic with those used by vitamin D analogues, which target the vitamin D receptor, the distinct target has the potential to reduce PTH secretion.

Although not approved for patients with CKD not yet on dialysis, cinacalcet, the only currently available calcimimetic, is an emerging option in the treatment of secondary hyperparathyroidism in predialysis patients with CKD.

**Anemia**<sup>(27)</sup> — Most CKD patients have normocytic and normochromic anaemia, which is primarily caused by a decrease in erythropoietin production by the kidney (perhaps reflecting a decrease in functional renal mass) and a drop in red blood cell survival. Anemia is a frequent condition in many CKD patients who do not yet require dialysis, becoming more prevalent as eGFRs fall below 60 mL/min/1.73 m<sup>2</sup> [36,37], especially in diabetics.

The 2012 KDIGO guidelines suggest that, among patients who do not have anemia, the Hb concentration should be checked when it is clinically indicated and at least yearly among all patients with stage 3 CKD (ie, eGFR 30 to 59 mL/min/1.73 m<sup>2</sup>), at least every six months among patients with stage 4 to 5 CKD (ie, eGFR  $\leq$ 29 mL/min/1.73 m<sup>2</sup>), and at least every three months among patients who are on dialysis. Although primarily used in patients with ESKD, ESAs such as erythropoietin and darbepoetin alfa also correct the anemia in those with CKD who do not yet require dialysis.

**Dyslipidemia**<sup>(28)</sup> — Abnormal lipid metabolism is common in patients with kidney disease. The primary finding in CKD is hypertriglyceridemia, with the total cholesterol concentration usually being normal (perhaps due in part to malnutrition in some patients). In the patient with hypercholesterolemia, a statin with or without ezetimibe can effectively and safely lower the plasma cholesterol concentration to or near acceptable levels.

**Sexual dysfunction**<sup>(29)</sup> — In patients with severe renal disease, significant abnormalities in sexual and reproductive function are typically seen. For instance, more than 50% of uremic men report symptoms such as erectile dysfunction, decreased libido, and noticeably decreased frequency of sex; in addition, women with CKD frequently experience menstrual and fertility disturbances, which typically result in amenorrhea by the time the patient reaches ESKD.

Pregnancy that is carried to term is unusual in women with a plasma creatinine content of less than 3 mg/dL (265 micromol/L), which is a significant clinical implication of these abnormalities.

## **Treatment of complications of end-stage kidney disease :-**

Once the patient has reached the stage of near ESKD (eGFR <15 mL/min/1.73 m<sup>2</sup>), signs and symptoms related to uremia begin to occur, such as malnutrition, anorexia, nausea, vomiting, fatigue, sexual dysfunction, platelet dysfunction, pericarditis, and neuropathy.

**Malnutrition**<sup>(30,31)</sup> — Due to decreased food intake (mostly because of anorexia), impaired intestinal absorption and digestion, and metabolic acidosis, malnutrition is a prevalent problem in individuals with severe CKD. Malnutrition may be indicated by a decreased albumin content in the plasma. Serum albumin concentration and body weight should be measured serially in order to determine nutritional status. For people with eGFRs below 20 mL/min/1.73 m<sup>2</sup>, these measurements should be made roughly every one to three months, and more frequently if necessary, for people with eGFRs below 15 mL/min/1.73 m<sup>2</sup>. Overall, the diet of the majority of CKD patients should provide between 30 and 35 kcal/kg per day.

**Recommended Dietary Intake for Chronic Kidney and End-Stage Renal Disease Patients**

Substance	Chronic kidney disease	Maintenance hemodialysis
Protein	0.8 to 1.0 g/kg/day of high biological value protein	>1.2 to 1.3 g/kg/day
Energy	≥ 35 kcal/kg/day; if the body weight is greater than 120% of normal or the patient is greater than 60 years of age a lower amount may be prescribed	
Fat, percent of total energy intake	30 to 40	30 to 40
Polyunsaturated-to-saturated ratio (fatty acid ratio)	1.0:1.0	1.0:1.0
Carbohydrate	Balance of nonprotein calories	
Total fiber, g/day	20 to 25	20 to 25
Sodium, mg/day	< 2,000	< 2,000
Potassium, meq/day	40 to 70	40 to 70
Phosphorus, mg/day	600 to 800	600 to 800
Calcium, mg/day	1,400 to 1,600	1,400 to 1,600
Magnesium, mg/day	200 to 300	200 to 300
Iron, mg/day	≥ 10 to 18	≥ 10 to 18
Zinc, mg/day	15	15
Water, mL/day	Up to 3,000 as tolerated	Usually 750 to 1500

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 Ikizler IA. Nutrition and kidney disease. In: *Primer on Kidney Diseases*, Greenberg A (Ed). Elsevier, Philadelphia, 2005, p.496.

**Uremic bleeding** — Patients with CKD have a greater propensity to bleed. This seems to be most directly related to the lengthening of the bleeding period, which is predominantly caused by reduced platelet function. Asymptomatic patients don't need any special treatment. However, patients who are actively bleeding or who are about to have a surgical or invasive procedure are preferable to address the platelet deficiency (such as a kidney biopsy). Desmopressin (DDAVP), cryoprecipitate, oestrogen, the treatment of anaemia, and the start of dialysis are just a few of the various modalities that can be applied in this situation.

**Pericarditis** — The three main symptoms of uremic pericarditis are fever, pleuritic chest discomfort, and pericardial friction rub. The ECG typically does not reveal the normal generalised ST and T wave elevation in uremic pericarditis, likely because this is a metabolic pericarditis and epicardial damage is infrequent.

Dialysis should be started when pericarditis develops in a patient with advanced kidney failure that is otherwise undiagnosed (providing there is no circulatory compromise or evidence of impending tamponade). Most patients with uremic pericarditis react to dialysis quickly, with the chest pain going away and the pericardial effusion shrinking in size.

**Uremic neuropathy** — Important consequences of ESKD include dysfunction of the central and peripheral neurological systems, including encephalopathy (impaired mental status advancing, without treatment, to seizures and coma), polyneuropathy, and mononeuropathy. They are now far less common as a result of the inclination to start dialysis sooner.

Uremic neuropathy frequently manifests as sensory dysfunctions, such as the restless leg or burning feet syndromes. These issues are typically clear signals that dialysis should begin. The degree and scope of dysfunction before to the start of dialysis is directly correlated with the degree of recovery from uremic neuropathy.

**Thyroid dysfunction** — The kidney normally plays an important role in the metabolism, degradation, and excretion of several thyroid hormones. The disturbances that can occur include low serum-free and total T3 concentrations and normal reverse T3 and free T4 concentrations. The serum thyrotropin (TSH) concentration is normal, and most patients are euthyroid.

**Infection and vaccination**<sup>(32,33)&</sup> — Patients with CKD are at increased risk for infection. The risk of bacterial infection (particularly pulmonary and genitourinary) increases with the decline in kidney function.

Careful attention should be paid to preventive measures such as influenza and pneumococcal immunization. The following 2012 are KDIGO guidelines :

- Adults with all stages of CKD should be offered annual vaccination with influenza virus unless contraindicated.
- Adults with stage 4 and 5 CKD who are at high risk of progression of CKD should be immunized against hepatitis B and the response confirmed by immunologic testing.
- Adults with CKD stages 4 and 5 should be vaccinated with polyvalent pneumococcal vaccine unless contraindicated. Patients who have received pneumococcal vaccination should be offered revaccination within five years.

## **PREPARATION FOR AND INITIATION OF KIDNEY**

### **REPLACEMENT THERAPY**

Patients who may eventually need kidney replacement therapy should be identified because proper planning can reduce morbidity and possibly mortality. Early detection allows for the most effective start of dialysis with a functioning chronic access. It may also allow for the identification and assessment of family members in order to place a renal allograft before the need for dialysis. Additionally, if insufficient time has passed between the diagnosis of end-stage kidney disease (ESKD) and the start of dialysis, the patient's capacity to psychologically accept the need for lifelong kidney replacement therapy is frequently compromised.

**Choice of kidney replacement therapy**<sup>(35)</sup> — Once it is determined that kidney replacement therapy will eventually be medically indicated, the patient should be counselled to consider the advantages and disadvantages of hemodialysis (in-center or at home), peritoneal dialysis (continuous or intermittent modalities), and kidney

transplantation (living or deceased donor). The option of conservative management should also be discussed among patients who are unwilling or unable to undergo kidney replacement therapy. The 2015 Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines recommend that patients with an estimated glomerular filtration rate (eGFR)  $<30$  mL/min/1.73 m<sup>2</sup> should be educated concerning these issues.

The preferred course of treatment for ESKD is a kidney transplant. When compared to maintenance dialysis, a successful kidney transplant enhances the quality of life and lowers the mortality risk for the majority of patients. If accessible, living-donor transplants also have the benefit of being performed quickly, allowing for preventative transplantation (transplantation prior to dialysis). Comparing such individuals to those who have a term of dialysis prior to transplantation, it appears that the graft survival of the former group is better.

**Preparation for hemodialysis**<sup>(36)</sup> — For hemodialysis to be effective, the bloodstream must be accessible consistently. Due to the higher risk of infection and more severe effects of arterial steal syndrome with lower extremity grafts, the access should typically be put in the non-dominant upper extremity. Therefore, to protect the veins in the other arm, venipuncture should be limited to the arm that was not selected for future access insertion.

Primary arteriovenous (AV) fistulas, AV grafts, and tunnelled hemodialysis catheters are the three basic methods of vascular access for maintenance hemodialysis. Refer the patient to an access surgeon if they have late stage 4 CKD, which is indicated by an eGFR of 20 to 25 mL/min/1.73 m<sup>2</sup>, to help with the placement of a permanent vascular access.



**Preparation for peritoneal dialysis<sup>(37)</sup>** — The abdominal cavity-based peritoneal dialysis catheters can be utilised right away after insertion. However, it is better to wait at least 10 to 14 days before starting dialysis in order to reduce the chance of fluid leak. Small volume exchanges done while lying flat can be done with little risk of leak if dialysis is necessary within 10 days of catheter insertion.

**Indications for kidney replacement therapy<sup>(38,39)</sup>** — There are a number of clinical indications to initiate dialysis in patients with CKD. These include ,

- Pericarditis or pleuritis (urgent indication).
- Progressive uremic encephalopathy or neuropathy, with signs such as confusion, asterixis, myoclonus, wrist or foot drop, or, in severe, cases, seizures (urgent indication).
- A clinically significant bleeding diathesis attributable to uremia (urgent indication).
- Fluid overload refractory to diuretics.
- Hypertension poorly responsive to antihypertensive medications.
- Persistent metabolic disturbances that are refractory to medical therapy. These include hyperkalemia, hyponatremia, metabolic acidosis, hypercalcemia, hypocalcemia, and hyperphosphatemia.
- Persistent nausea and vomiting.
- Evidence of malnutrition.

Relative indications for the initiation of dialysis include decreased attentiveness and cognitive tasking, depression, persistent pruritus, or the restless leg syndrome.

There is no defined threshold eGFR level for the start of dialysis, and the timing of dialysis commencement is questionable among asymptomatic individuals with progressing CKD. Dialysis should be started in the asymptomatic patient with an exceptionally low eGFR, such as an eGFR of roughly 8 to 10 mL/min/1.73 m<sup>2</sup>, to help prevent the onset of potential life-threatening consequences of uremia.

**Conservative kidney management**<sup>(40)</sup> – All patients who opt against receiving kidney replacement therapy should have the option of managing their ESKD through conservative means. The provision of proper palliative care is a component of conservative care, along with the control of symptoms and advance care planning.

## **PATIENT SURVIVAL IN MAINTENANCE HEMODIALYSIS:-**

### **OVERVIEW**

The mortality rate among patients with end-stage kidney disease (ESKD) is still significant even though regular dialysis avoids uremia-related death.

Comparing ESKD patients to the general population, they have a worse overall survival rate. The death rate for ESKD patients is, however, steadily declining. The adjusted relative decrease in mortality throughout this time was 28% for hemodialysis (HD) patients, 43% for peritoneal dialysis (PD), and 40% for kidney transplant recipients. When compared to other Medicare beneficiaries in the same age group who have cancer, diabetes, or cardiovascular disease, dialysis patients over 65 continue to have a much higher mortality rate.

It is well known that insufficient dialysis contributes to a decreased overall survival rate. This has significant ramifications since more extensive dialysis, especially above a particular threshold value, may increase survival, as in the case of nocturnal HD.

Additional factors associated with the dialysis procedure may also correlate with decreased survival. As examples:

- Dialysis vintage is associated with an enhanced risk of death, with each additional year of dialysis treatment associated with an increase in the risk of death of approximately 6 percent.<sup>(41)</sup>
- Potassium levels  $<4.0$  or  $>5.6$  mEq/L are associated with increased mortality in HD patients compared with serum levels between 4.6 and 5.3 mEq/L .
- The size of the dialysis facility may affect mortality. One study of 385,074 dialysis patients suggested that a higher mortality was observed among patients dialyzed at facilities with  $<15$  stations

## **CAUSES OF DEATH<sup>(42,43)</sup>**

It is helpful to briefly examine the three main causes of death in this patient population—cardiovascular disease, infection, and withdrawal from dialysis—before reviewing the various factors affecting mortality in dialysis patients.

- 1) Most deaths are caused by cardiovascular disease, which accounts for about 50% of deaths. While there has been a decrease in cardiovascular fatalities in the general community, dialysis patients have not seen a similar trend.

- 2) The second most prevalent cause of death is infection, which is typically brought on by common microorganisms (such *Staphylococcus aureus*) and frequently involves the hemodialysis (HD) vascular access.
- 3) 15 to 25% of patient fatalities are related to dialysis withdrawal.

## **RISK FACTORS NOT RELATED TO DIALYSIS**

A large number of risk factors that are unrelated to the dialysis procedure have been associated with decreased survival among dialysis patients.

**Comorbid conditions**<sup>(43)</sup> — Comorbid illness is a condition that is becoming more prominent, affecting new patients starting dialysis much more frequently than in the past. Diabetes mellitus was the most common cause of end-stage kidney disease (ESKD) in people starting hemodialysis (HD), according to the 2007 United States Renal Data System (USRDS) Annual Report (44 percent). Individuals with diabetes appear to have a worse chance of extended survival at 10 years than patients without diabetes (4 versus 11 to 14 percent).

Additionally prevalent in the dialysis group is heart disease. In the Hemodialysis (HEMO) Study, it was shown that about 40% of patients had ischemic heart disease, accounting for around 80% of all heart disease cases. Additionally, it has been predicted that 19% of patients who are about to start a dialysis regimen already have severe left ventricular hypertrophy, while only 27% of those patients have an echocardiography that is normal. Additionally, at least one coronary artery has a 50% constriction in at least 75% of people with ESKD overall. Chronic kidney illness alone is regarded as an analogous risk factor for coronary heart disease, and even

patients with mild to moderate chronic renal failure are much more at risk for cardiovascular disease. Additional characteristics that are frequent among dialysis patients, in addition to preexisting coronary artery disease, may encourage the development of coronary disease and increased cardiovascular mortality:

- Hypertension, which is present in approximately 80 percent of patients at the onset of dialysis and, with effective fluid control, approximately 25 to 30 percent at the end of the first year.
- Metabolic abnormalities, particularly hyperphosphatemia, an elevated calcium phosphorous product, and increased parathyroid hormone (PTH) levels. These extremely common abnormalities are increasingly recognized as possibly important unique risk factors for dialysis patients.
- Left ventricular hypertrophy, due in part to hypertension, chronic anaemia, and perhaps decreased kidney function.
- Hyperlipidemia.
- Diabetes mellitus, which is a major risk factor for coronary artery disease and is common among patients requiring dialysis.

The concurrent presence of other life-threatening conditions will also affect overall survival.

**Underlying kidney disease** — Five-year survival among dialysis patients is best with chronic glomerular diseases and polycystic kidney disease (PKD), intermediate with hypertension-induced kidney disease, and worst with diabetic nephropathy; five-year survival of the patient with diabetic nephropathy is only 20 percent.

**Age<sup>(44)</sup>** — Survival declines with increasing age, with patients <45 years of age doing best. Older patients with renal vascular disease appear to have the worst prognosis, with such patients having 5- and 10-year survival rates of only approximately 15 and 5 percent, respectively.

**Country<sup>(45)</sup>** — In comparison to Europe and Japan, the US has much poorer dialysis patient survival rates. According to some data, this is partly a result of regional variations in death rates (particularly those linked to cardiovascular disease) among the general population. Approximately 25% of the difference in death among dialysis patients between the northern and southern regions of Europe could be ascribed to variations in mortality in these locations, according to a European study that took age, sex, and diabetes into account.

**Race** — African Americans and Asian Americans as a whole experience less mortality than White patients. In one study, the five-year survival rates for individuals who were Black, White, and of other races were 35, 25, and 32 percent, respectively. Black patients 50 years of age experienced higher mortality compared to White patients, especially when transplantation was taken into account as a competing risk. Black patients >50 years of age continued to have a survival advantage.

**Psychosocial factors** — Independent of the existence of any concomitant illnesses, psychosocial factors also seem to have a major impact on mortality. A lower chance of passing away is linked to increased social support, improved behavioural compliance, and favourable perceptions of the impacts of sickness. They appear to have a similar impact on mortality as medical risk factors.

**Location of residence** — It may alter survival. Residence in areas with higher median household income has been associated with improved survival.

**Nutrition**<sup>(46)</sup> — Patients who are undernourished, especially those who have hypoalbuminemia, are at higher medical risk and die more frequently. The existence of malnutrition prior to the start of dialysis is substantially predictive of an increase in mortality following the start of dialysis, even though these observations have mostly been observed in patients on maintenance HD and are connected to the dialysis dose.

**Salt intake**<sup>(47)</sup> — A post-hoc analysis of the HEMO Study including 1770 HD patients has suggested that higher reported dietary sodium intake and the ratio of sodium to calorie or potassium intake are associated with increased all-cause mortality and a slightly increased ultrafiltration requirement.

**Residual kidney function**<sup>(48)</sup> — The survival of dialysis patients is improved by residual kidney function. Numerous studies have demonstrated the positive impact of sustained urine production on survival among peritoneal dialysis (PD) patients.

Remaining renal function may increase the likelihood of cardiovascular survival by better controlling the fluid and electrolyte balance. Patients with maintained urine production may require less fluid removal during dialysis because they may retain less fluid between treatments. Another explanation is that residual renal function permits higher clearance of uremic toxins that are attached to proteins, some of which may have negative cardiovascular effects.

**Others** —

- **Sleep disorder**<sup>(49)</sup> — The presence of a sleep disorder enhances the risk of death in patients without renal failure

- Predialysis care – An increasing number of studies, although all observational and retrospective, suggest that patients referred late to a nephrologist for predialysis medical care, compared with those referred early in the disease course, have an enhanced mortality risk once dialysis is initiated.
- Hemoglobin levels – A low hemoglobin levels in association with mortality among patients with kidney disease
- Frailty – Frailty is defined by the presence of three or more of the following: weight loss, muscle weakness, fatigue or exhaustion, low physical activity, and slow gait. Independent of age, increased mortality is noted among frail dialysis patients.
- Noncompliance – Noncompliance, which can be defined in part by regularly skipping HD sessions and poor adherence to dietary restrictions, is associated with increased mortality.
- Physical activity – Habitual physical activity has been associated with decreased mortality among patients undergoing maintenance HD .

### **ADEQUACY OF DIALYSIS<sup>(50)</sup>**

It was believed that the greater death rate was caused, at least in part, by insufficient dialysis. When compared to patients dialyzed four or more hours three times per week, patients dialyzed for less than 3.5 hours three times per week have roughly a double the mortality risk.

The majority of those patients were made normotensive and required no antihypertensive drugs, and those patients who underwent very intensive dialysis (Kt/V of 1.67) also had a high frequency of complete recovery. Patients having



nocturnal hemodialysis have seen similar advantages with longer periods of treatment (HD).

### **HEMODIALYSIS SESSION LENGTH<sup>(51)</sup>**

The effect of hemodialysis (HD) session length on mortality independent of conventional markers of dialysis adequacy is unclear among patients undergoing standard, three times per week dialysis therapy. The treatment time that was >240 minutes per session was significantly associated with decreased risk of mortality (relative risk [RR] 0.81) .

### **CONTROL OF FLUID BALANCE AND HYPERTENSION<sup>(52,53)</sup>**

Establishing a precise dry weight and achieving this objective weight are crucial. Increased mortality is linked to chronic fluid excess. A strong correlation between baseline and one-year cumulative fluid overload and elevated mortality among outpatient incident hemodialysis (HD) patients was found in a large observational study. Systolic blood pressure was utilised to categorise patients into groups (130, 130 to 160, and >160 mmHg), and bioimpedance spectroscopy was employed to determine fluid status. Within three months of beginning HD, the first measurement of fluid overload—defined as a fluid excess of 15% for males and 13% for women—was taken. In all blood pressure ranges, higher mortality was predicted by both fluid overload at the start and one-year cumulative fluid overload. Cumulative fluid overload had the larger effect on mortality (for patients with systolic blood pressure <130 mmHg, hazard ratio [HR] 1.94; for patients with systolic blood pressure 130 to 160 mmHg, HR 1.51; for patients with systolic BP >160 mmHg, HR 1.62). The effect

of fluid overload was similar in all groups as stratified by age, sex, body mass index (BMI), and comorbidities including diabetes and heart disease.

### **INTERDIALYTIC INTERVAL<sup>(53)</sup>**

When patients are dialyzed three times each week, long interdialytic breaks over the weekend are likely to increase mortality and morbidity. For patients on a three times weekly dialysis programme, the two-day interval has a higher risk of cardiovascular and non-cardiovascular death and morbidity. Fluid buildup, hemodynamic alterations, and electrolyte abnormalities or changes were possible causes of the elevated risk, at least in relation to cardiovascular events.

### **MALNUTRITION<sup>(54)</sup>**

With hypoalbuminemia, lower plasma levels of urea nitrogen and creatinine than expected given the intensity of dialysis, and undernourished and small dialysis patients (as indicated by a low BMI), there is a greater medical risk and increased mortality. As an illustration, the single laboratory finding most closely linked to a higher risk of death is a plasma albumin concentration 4 g/dL (40 g/L). The risk rises gradually between 3.5 and 3.9 g/dL (35 and 39 g/L) of plasma albumin, but is much higher between values below 3.0 g/dL (30 g/L). Malnutrition alone has been linked to an increase in mortality, but malnutrition paired with the presence of a comorbid disease was linked to a high death rate. On the other hand, indicators of healthy nutrition, like a high BMI and normal or high levels of muscle mass, are linked to a higher rate of survival.

Lean body mass and plasma albumin concentration significantly correlated in the group as a whole, however there was significant variation between patients. Low body weight to height ratios are another indicator of malnutrition that indicates mortality in HD patients.

Numerous chronic inflammatory disorders can lower the blood albumin content in dialysis patients, however inadequate diet is a prevalent cause of hypoalbuminemia. Interleukin-1 and tumour necrosis factor-alpha, which inhibit hepatic albumin synthesis, are thought to have a role in this impact. Thus, the presence of an untreated, underlying inflammatory process may contribute to the decreased survival observed among chronic HD patients with hypoalbuminemia.

## **CHRONIC HEMODIALYSIS ACCESS**

Arteriovenous fistulas have advantages over arteriovenous grafts and central venous catheters, with some evidence suggesting a survival benefit with fistulas.

## **MATERIALS AND METHODS**

### **STUDY POPULATION:**

This prospective Observational study has been conducted among 130 patients who are undergoing their maintenance hemodialysis for End-Stage Renal Disease in Rajiv Gandhi Government General Hospital during the study period from April 2022 to September 2022 after obtaining informed consent as per the inclusion and exclusion criteria already mentioned.

A detailed clinical history will be recorded and the patients will analyzed on the basis of their demographic details, duration of illness, frequency and duration of maintenance hemodialysis, presence of comorbidities, and their etiology, frequency ,duration of hospital stay and their outcome of hospitalization.

### **INCLUSION CRITERIA**

- Age > 18 years of age
- Patients undergoing Maintenance hemodialysis in Government Rajiv Gandhi Government Hospital & Madras Medical College

### **EXCLUSION CRITERIA**

- Patients not willing to participate in the study.
- Patients less than 18 years of age.
- Patients less than 3months of initiating regular hemodialysis.

## **ANTICIPATED OUTCOME:**

Hospitalisation and mortality among patients on Maintenance Hemodialysis are more among patients who are Elderly, Anaemic , Hypoalbuminemia and have multiple comorbidities like Diabetes Mellitus, Hypertension and so on.

## **DATA COLLECTION:**

- After confirmation of diagnosis and explaining the purpose & procedure of study, written informed consent in Tamil will be obtained
- A previously designed profoma will be used to collect the demographic and clinical details of the patients.A detailed history will be taken and a clinical examination will be performed.
- The following information will be collected for each patient: age, gender, etiology of Chronic Kidney Disease(CKD) and duration of CKD and MHD, Dialysis frequency, Vascular access, Other comorbidities like Diabetes Mellitus, Systemic Hypertension, coronary artery disease, Hypothyroidism etc., Baseline Hemoglobin, Urea, Creatinine, calculated eGFR using CKD- EPI creatinine 2021 formula, serum sodium, potassium, calcium, phosphorus, Albumin, Echocardiography, Drug and fluid compliance.
- During each Hospitalisation, Diagnosis for current hospitalisation, Duration of stay and outcome in the form of Discharge or Death were collected.
- With the available above parameters, various factors responsible for hospitalisation among patients on maintenance hemodialysis were analysed.

**DESIGN OF STUDY:** Prospective Observational study.

**PERIOD OF STUDY:** April 2022 to September 2022

**COLLABORATING DEPARTMENTS:**

Institute of Nephrology

Department of Biochemistry

Institute of Cardiology

**ETHICAL CLEARANCE:** Approved

**CONSENT:** Individual written and informed

**ANALYSIS: STATISTICAL METHODS:**

The data collected during the study was formulated into a master chart in Microsoft office excel and statistical analysis was done with help of computer using statistical software package SPSS V.17 for windows. Using this software, frequencies, range, mean, standard deviation and 'p' were calculated through student 't' test, one way ANOVA, pearson correlation and chi square test .

P value of  $< 0.05$  was taken as significant.

**CONFLICT OF INTEREST:** NIL

**FINANCIAL SUPPORT: NIL**

**PARTICIPANTS:** Patients of age >18yrs, who are undergoing Maintenance Hemodialysis for End- Stage Renal Disease in Rajiv Gandhi Government General Hospital, Chennai.

**RESULTS**

**Table 1 . Age distribution in our study:**

Age group	Count	%
<20	4	3.1%
21-30	16	12.3%
31-40	29	22.3%
41-50	42	32.3%
51-60	19	14.6%
>61	20	15.4%

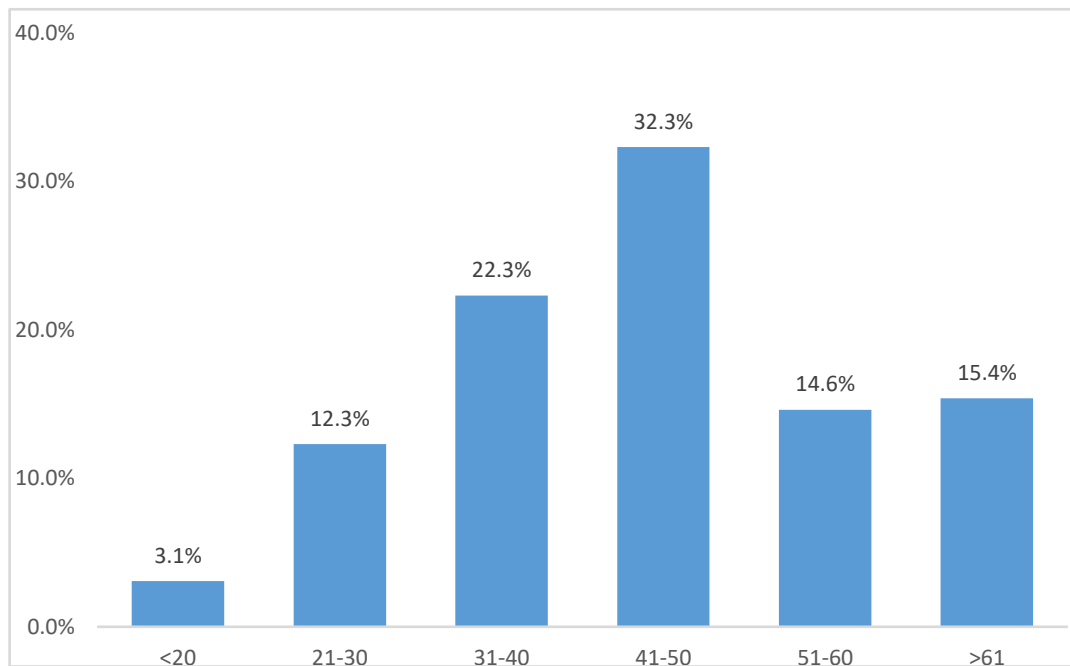


Fig. 1. In our study, most of the patients were between 41-50 years of age. The youngest patient in our study was 18 years of age and the oldest was 76 years of age, with a median age of 41 years of age



**Table 2. Gender Distribution in our study:**

Gender	Count	%
Female	43	33.1%
Male	87	66.9%

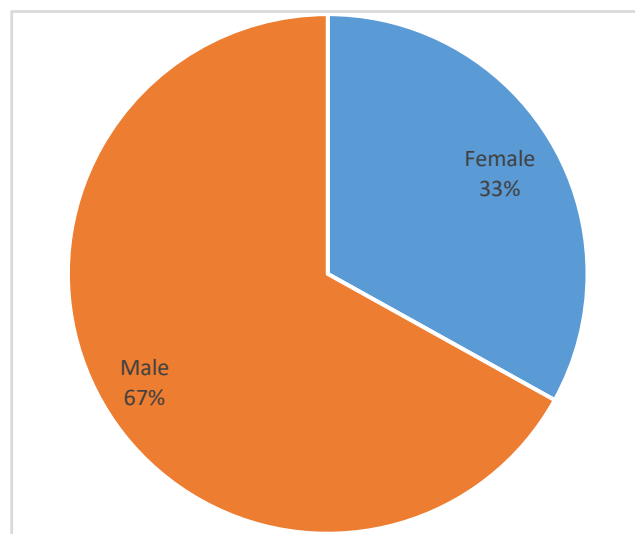


Fig 2. In our study, males contributed to the majority of cases of around 67%, whereas females contributed to only 33% of the cases.

**Table 3. Comorbidities distribution in our study :-**

Comorbidities	Count	%
CAD	27	20.8%
DM	37	28.5%
HTN	114	87.7%
THYROID	12	9.2%
SEIZURE	3	2.3%
OLD PTB	14	10.8%
SLE	5	3.8%

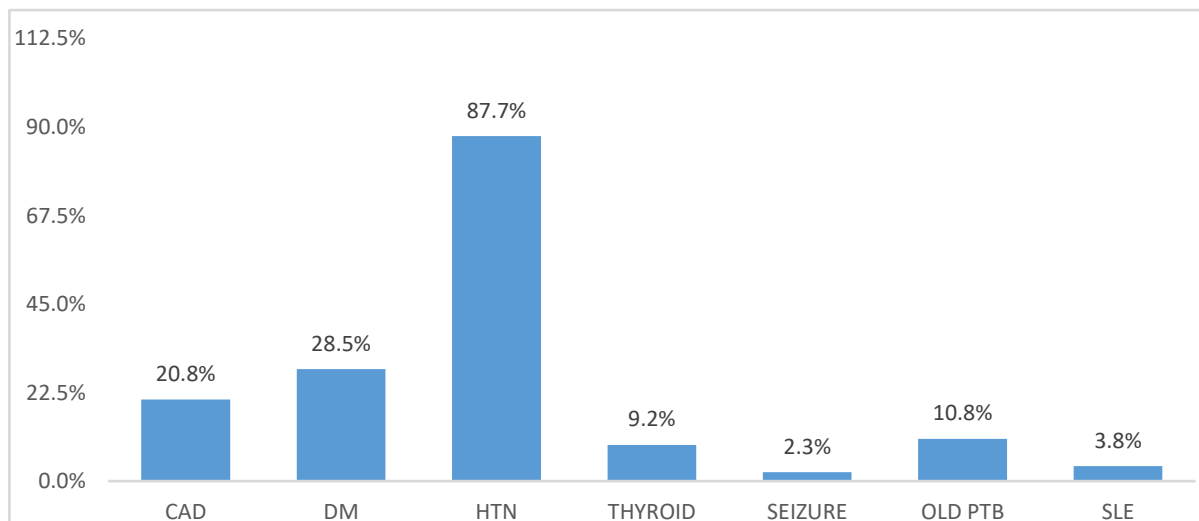


Fig 3. In our study, majority of patients are Hypertensive (87.7%), about 28.5% patients were Diabetic and about 20.8% has known Coronary Artery Disease.

**Table 4. Hemoglobin distribution in our study :-**

Hb	Count	%
<8	50	38.5%
>8	80	61.5%

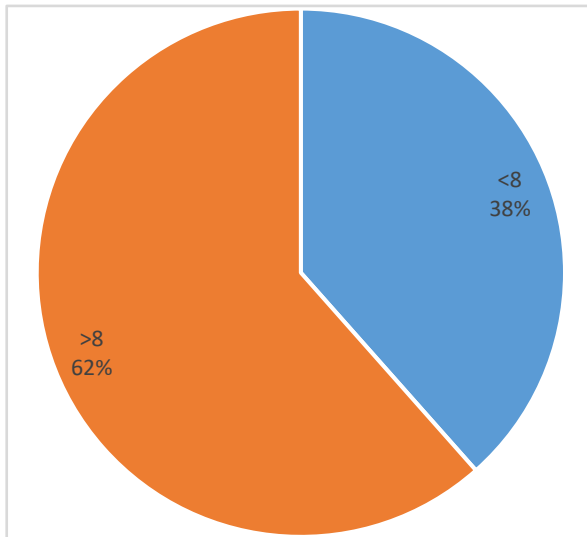


Fig 4. In our study, 61.5% of patients had Hemoglobin above 8gm/ dl, whereas 38.5% of patients had Hemoglobin less than 8gm/ dl.

**Table 5. Serum Albumin distribution in our study :-**

S. Albumin	Count	%
<3.4	39	30.0%
>3.4	91	70.0%

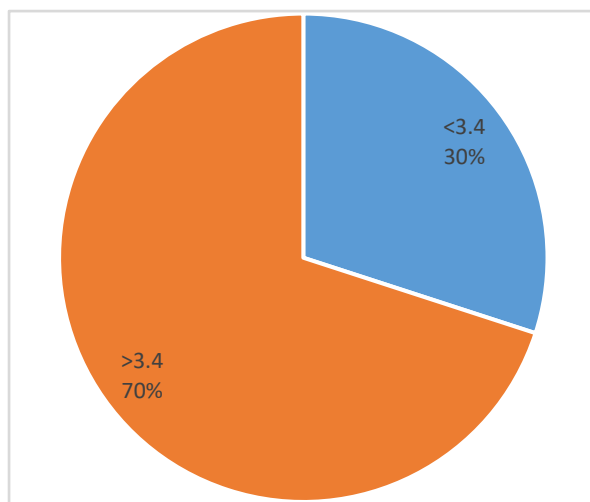


Fig 5. In our study, about 70% of patients had serum albumin more than 3.4gm/dl, whereas 30% of patients had serum albumin less than 3.4gm/ dl.

**Table 6. Outcome of Hospitalisation among our study population :-**

Outcome of Hospitalisation	Count	%
Death	8	11%
Discharge	70	89%

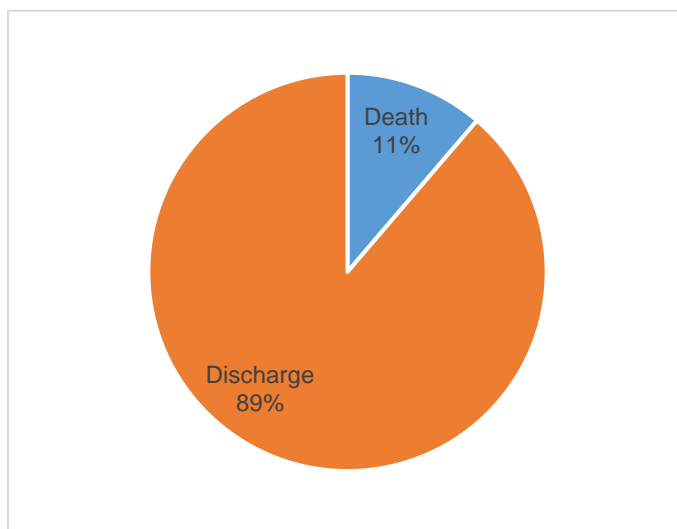


Fig 6.

**Table 7. Readmission among our study population :-**

Readmission	Count	%
No	115	88.5%
Yes	15	11.5%

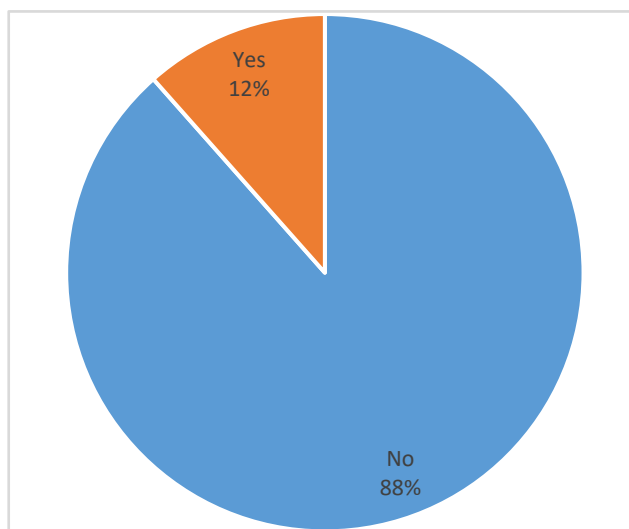


Fig 7. Comment:

In our study among 130 patients, there are 78 hospitalisation out of which 8 patients have expired and 70 patients have been discharged and about 15 patients have 2nd hospitalisation during the study period of 6 months.

**Table 8. Cause for Hospitalisation among patients on Maintenance****Hemodialysis:-**

<b>Diagnosis</b>	<b>Count</b>	<b>%</b>
Accelerated HTN	3	4.3%
Active PTB	1	1.4%
Acute diarrhoeal disease	1	1.4%
Acute febrile illness	5	7.1%
Acute hemorrhagic stroke	2	2.9%
Acute MI	1	1.4%
Acute pulmonary edema	22	31.4%
Cardiac failure	1	1.4%
Anasarca	8	11.4%
Appendicitis	1	1.4%
Ascites	1	1.4%
Cardiogenic shock	1	1.4%
Epistaxis - hypertensive	1	1.4%
Fistula failure	2	2.9%
For ovarian mass	1	1.4%
Hernia surgery	1	1.4%
Hypoglycemia	2	2.9%
Pleural effusion	2	2.9%
Pneumonia	4	5.7%
Sepsis / septic shock	1	1.4%
Severe metabolic acidosis	4	5.7%
Tubercular frontal lobe abscess	1	1.4%
Uremic encephalopathy	2	2.9%
Uremic gastritis	6	8.6%
Urinary tract infection	1	1.4%
UTI	3	4.3%

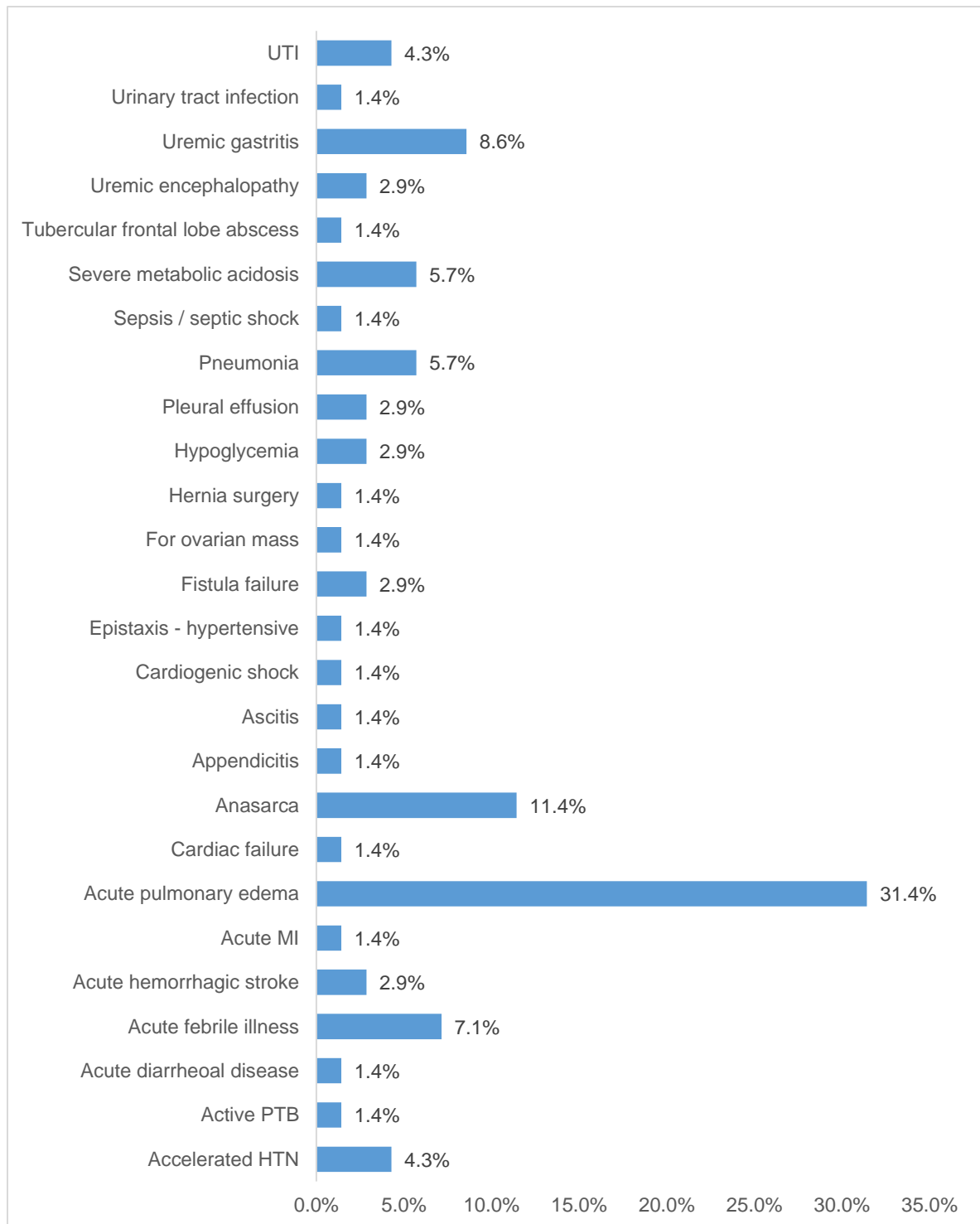


Fig 8. In our study population, Acute Pulmonary edema is the highest etiology for hospitalisation contributing about 31.4% of hospitalisation followed by Anasarca and uremic gastritis.



**Table 9. Distribution of Duration of Renal disease in our study:-**

Duration of disease	Frequency	Percent
<1	8	6.2%
1.1-5	54	41.5%
5.1-10	60	46.2%
>10.1	8	6.2%
Total	130	100.0%

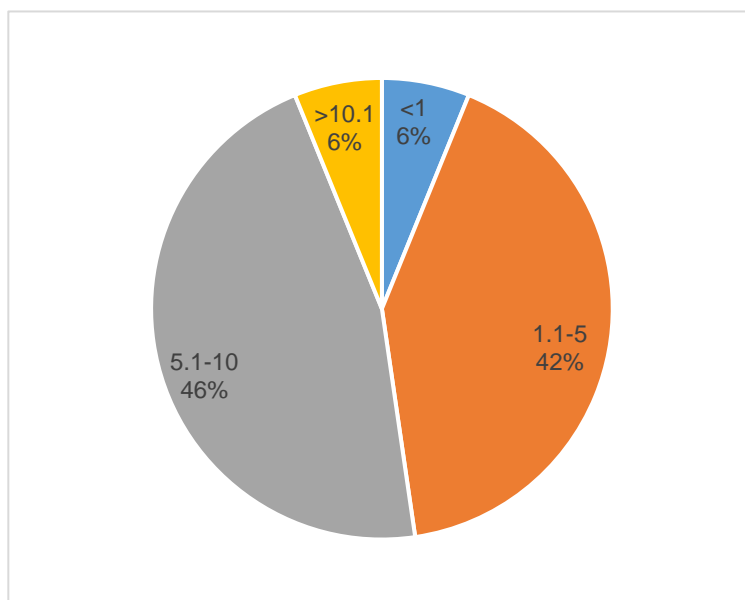


Fig 9. Majority of patients in our study population have renal disease for duration of 5-10 years contributing about 46.2% followed by 1-5 years contributing about 41.5%

**Table 10. Distribution of Duration of MHD in our study:-**

<b>Duration of MHD</b>	<b>Frequency</b>	<b>Percent</b>
<1	11	8.5%
1.1-5	64	49.2%
5.1-10	50	38.5%
>10.1	5	3.8%
<b>Total</b>	<b>130</b>	<b>100.0%</b>

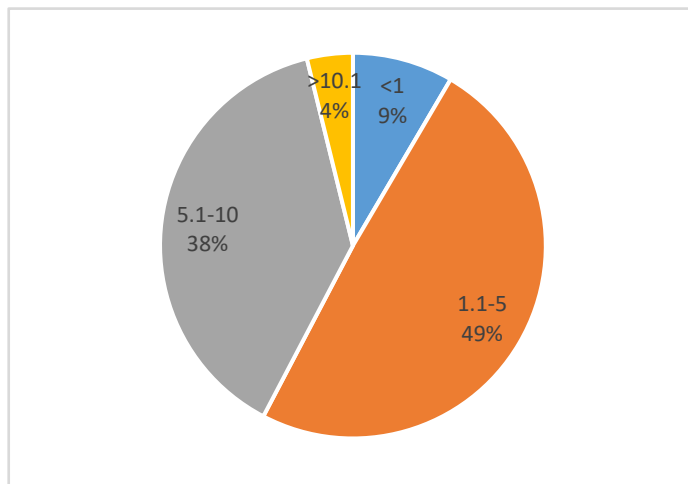


Fig 10. Majority of patients in our study are under MHD for about 1-5 years which constitutes about 49.2% followed by 5-10 years contributing about 38.5% , about 8.5% of patients are under MHD less than 1 years and about 3.8% of patients are under MHD for more than 10years.

**Table 11. Distribution of Frequency of MHD in our study:-**

Frequency of MHD	Frequency	Percent
1	1	0.8%
2	89	68.5%
3	40	30.8%
Total	130	100.0%

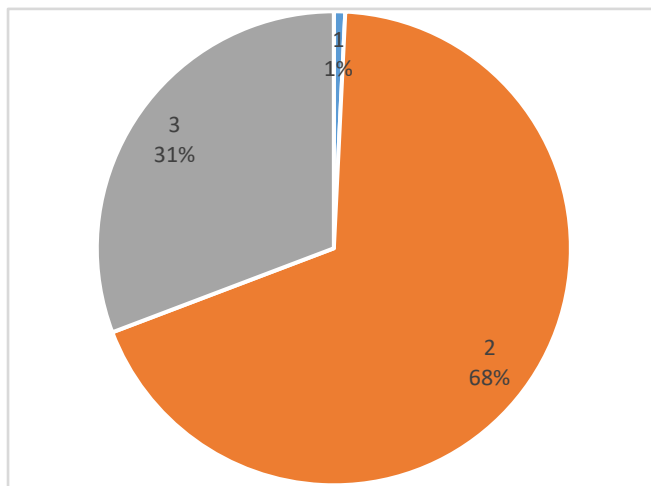


Fig 11. In our centre, majority of the patients are under 2/7 Dialysis which constitute about 68.5% and about 30.8% patients are under 3/7 maintenance hemodialysis.

**Table 12. Correlation of Age with Hospitalisation in our study population:-**

			Hospitalization		Total	P value
			Yes	No		
Age group	<60	Count	56	54	110	0.047
		% within Hospitalization	78.9%	91.5%	84.6%	
	>60	Count	15	5	20	
		% within Hospitalization	21.1%	8.5%	15.4%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

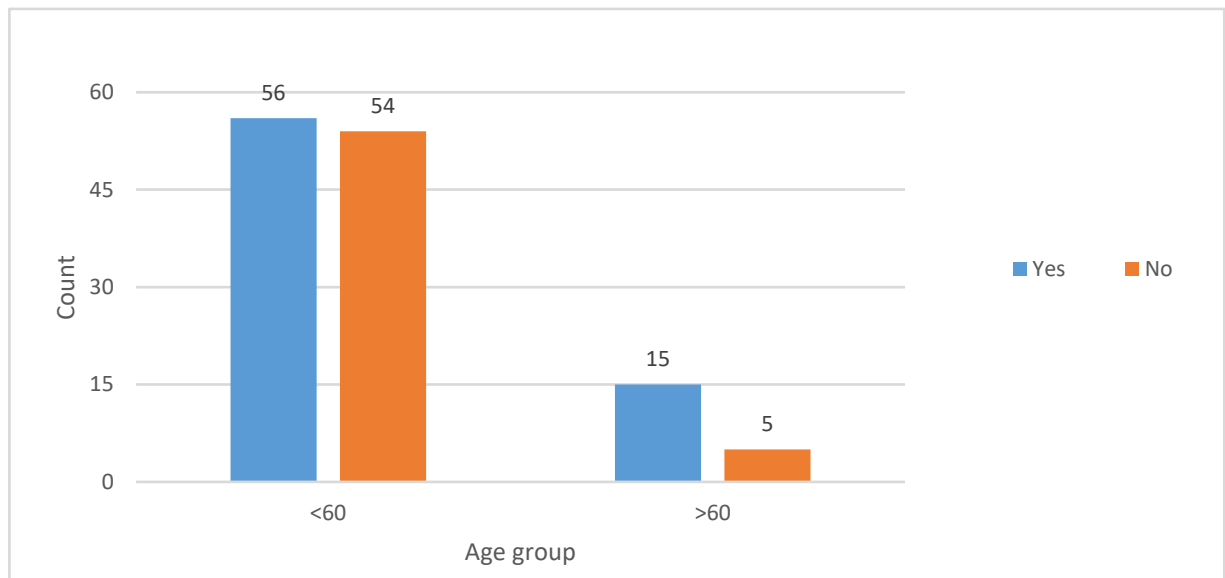


Fig 12. Patients of Age > 60 years has increased rate of Hospitalisation with a significant p-value of 0.047.

**Table 13. Correlation of Hemoglobin with Hospitalisation in our study**

**population:-**

			Hospitalization		Total	P value
			Yes	No		
Hemoglobin	<8	Count	32	18	50	0.089
		% within Hospitalization	45.1%	30.5%	38.5%	
	>8	Count	39	41	80	
		% within Hospitalization	54.9%	69.5%	61.5%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

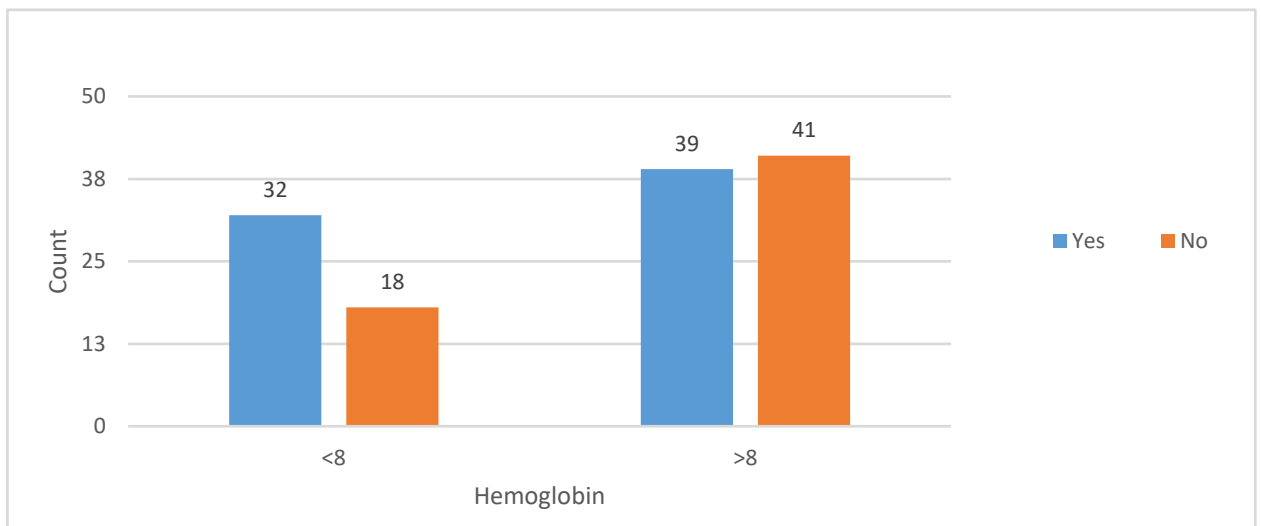
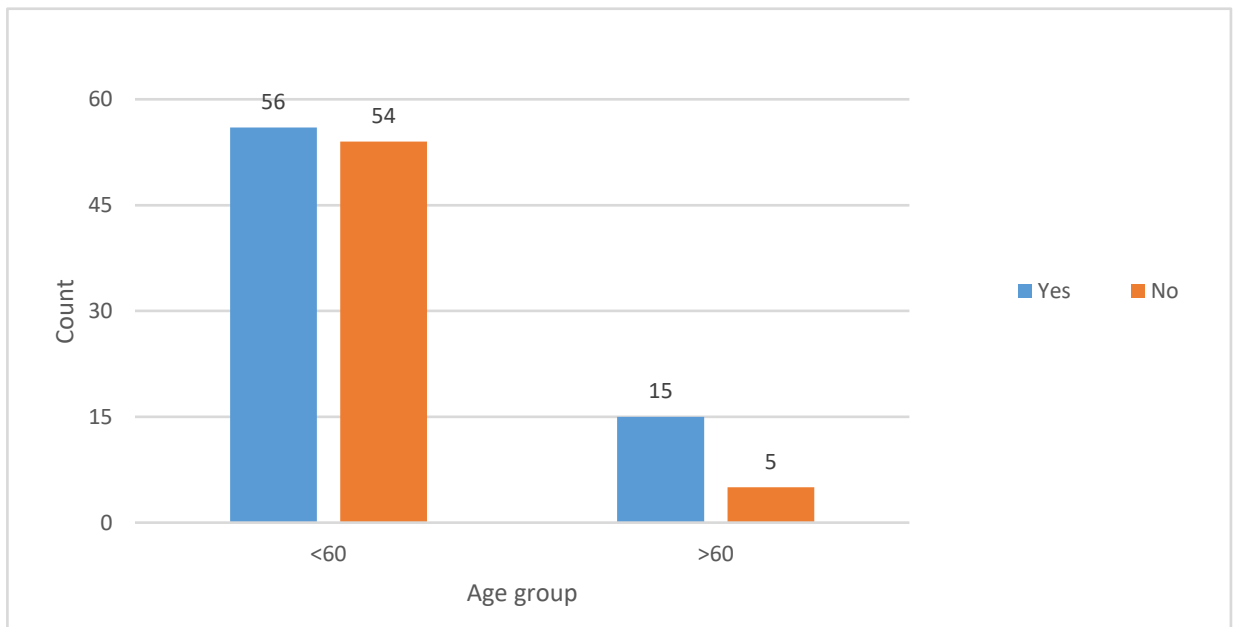


Fig 13. Though Hemoglobin less than 8gm/dl had higher admissions when compared to those above 8gm/dl, it fails to show a statistically significant rate of hospitalisation.

**Table 14. Correlation of serum Albumin with Hospitalisation in our study**

**population :-**

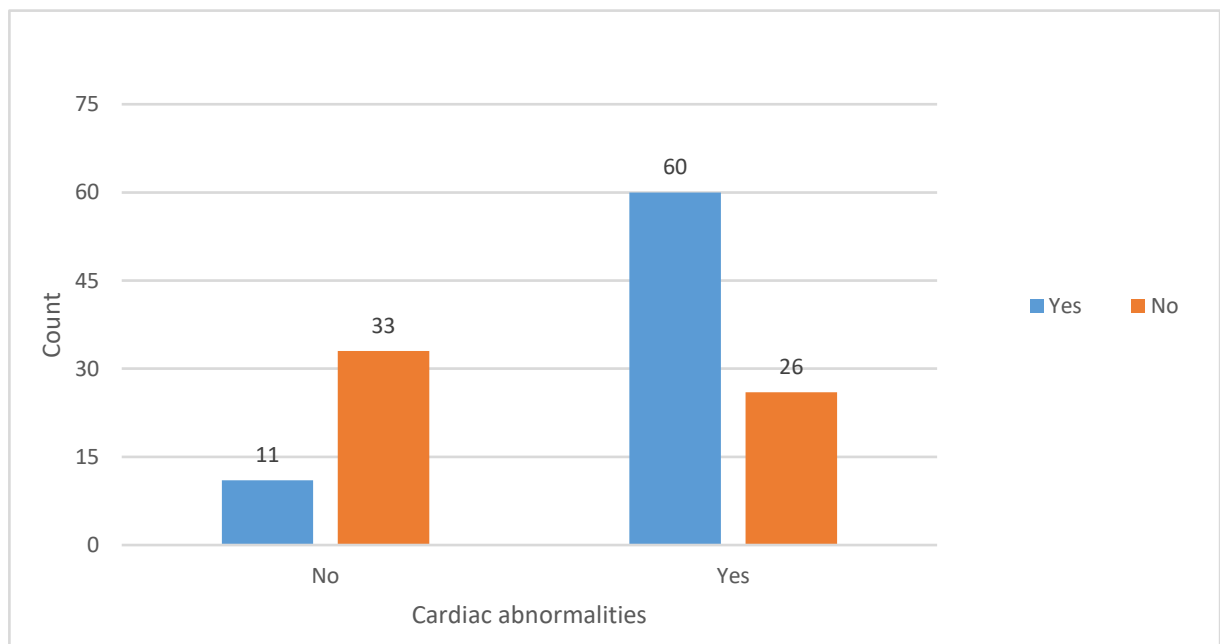
			Hospitalization		Total	P value
			Yes	No		
S. Albumin	<3.4	Count	23	16	39	0.513
		% within Hospitalization	32.4%	27.1%	30.0%	
	>3.4	Count	48	43	91	
		% within Hospitalization	67.6%	72.9%	70.0%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	



**Fig 14.** In our study, serum albumin less than 3.4gm/dl doesn't show any significant increase in hospitalisation.

**Table 15. Correlation of cardiac abnormalities with Hospitalisation in our study population:-**

			Hospitalization		Total	P value
			Yes	No		
Cardiac abnormalities	No	Count	11	33	44	<0.0001
		% within Hospitalization	15.5%	55.9%	33.8%	
	Yes	Count	60	26	86	
		% within Hospitalization	84.5%	44.1%	66.2%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	



**Fig 15. Patients with cardiac abnormalities have much higher rate of hospitalisation than those who have stable cardiac status with a very significant p-value of < 0.0001.**

**Table 16. Correlation of Duration of Renal disease with Hospitalisation in our study population:-**

			Hospitalization		Total	P value
			Yes	No		
Duration of disease	<5	Count	28	34	62	0.39
		% within Hospitalization	39.4%	57.6%	47.7%	
	>5	Count	43	25	68	
		% within Hospitalization	60.6%	42.4%	52.3%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

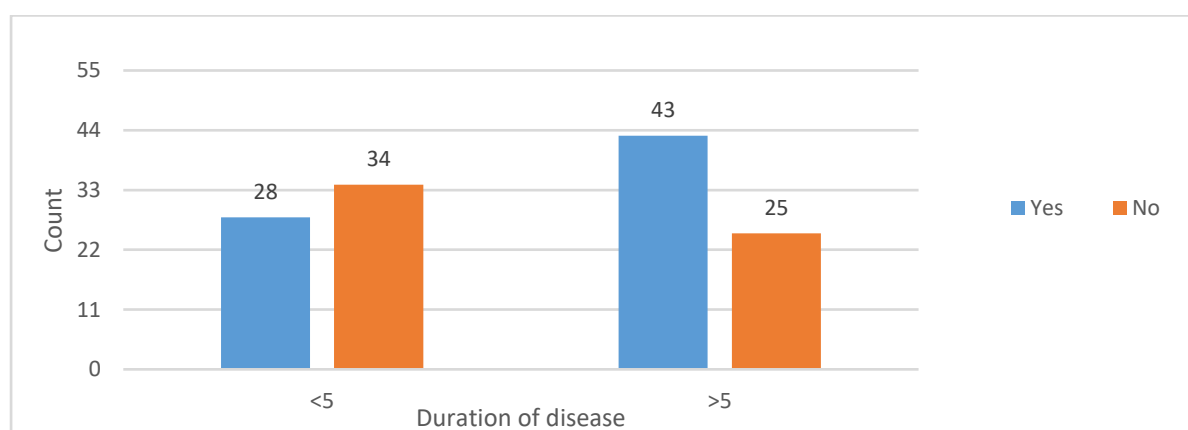


Fig 16. In our study, most of the patients even with duration of renal failure for more than 5 years , due to their excellent compliance to fluids, salt, drugs, dialysis and regular follow up, able to lead reasonably good quality of life. Our study was not able to demonstrate correlation between duration of disease and frequency of hospitalisation.



**Table 17. Correlation of Duration of MHD with Hospitalisation in our study**

**population:-**

			Hospitalization		Total	P value
			Yes	No		
Duration of MHD	<5	Count	37	38	75	0.159
		% within Hospitalization	52.1%	64.4%	57.7%	
	>5	Count	34	21	55	
		% within Hospitalization	47.9%	35.6%	42.3%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

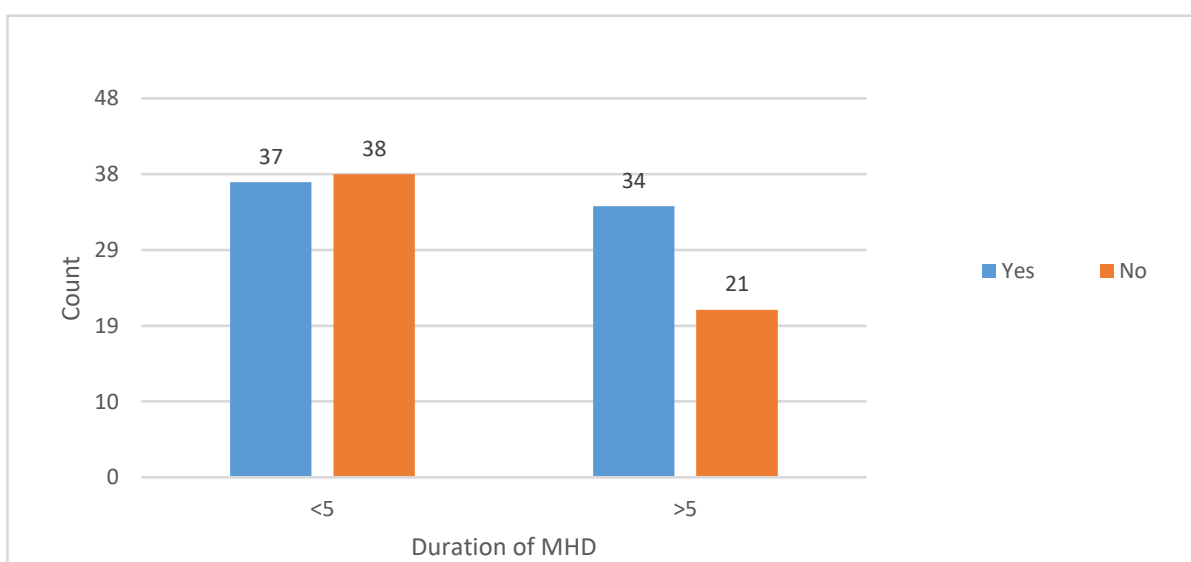


Fig 17. In our study, Duration of Maintenance Hemodialysis more than 5years doesn't show significant increase in the rate of hospitalisation.

**Table 18. Correlation of Frequency of MHD with Hospitalisation in our study**

population :-

			Hospitalization		Total	P value
			Yes	No		
Frequency of MHD	1	Count	1	0	1	0.004
		% within Hospitalization	1.4%	0.0%	0.8%	
	2	Count	40	49	89	
		% within Hospitalization	56.3%	83.1%	68.5%	
	3	Count	30	10	40	
		% within Hospitalization	42.3%	16.9%	30.8%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

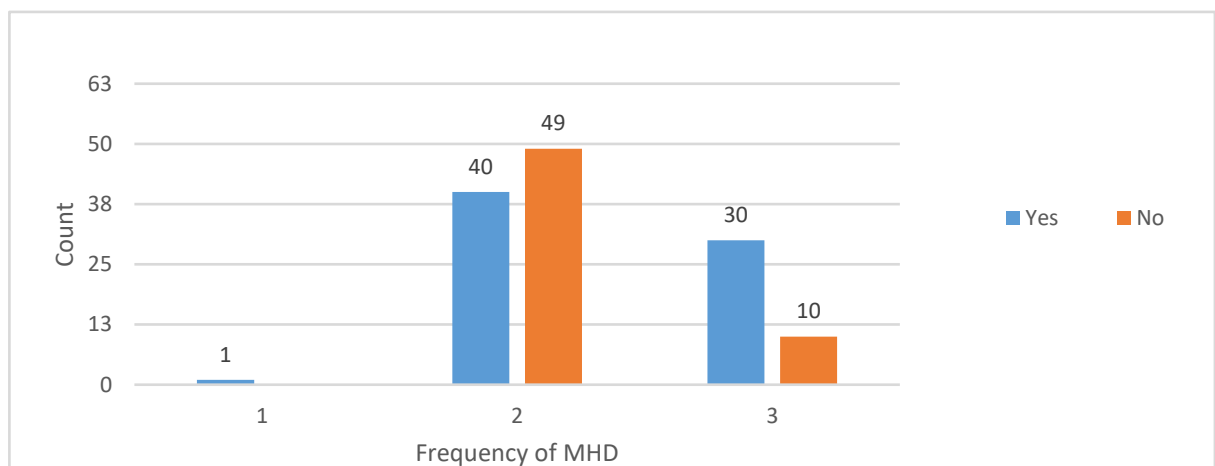


Fig 18. In our centre, Most of the patients who are under 2/7 MHD when not tolerated in the form of development of Acute pulmonary edema or uremic symptoms are changed to 3/7 MHD and hence most of the patients under 3/7 have longer duration of disease and also has multiple comorbidities and hence higher rate of hospitalisation were noted among patients on 3/7 MHD.

**Table 19. Correlation of Diabetes Mellitus with Hospitalisation in our study**

**population:-**

			Hospitalization		Total	P value
			Yes	No		
DM	NO	Count	47	46	93	0.139
		% within Hospitalization	66.2%	78.0%	71.5%	
	YES	Count	24	13	37	
		% within Hospitalization	33.8%	22.0%	28.5%	
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	

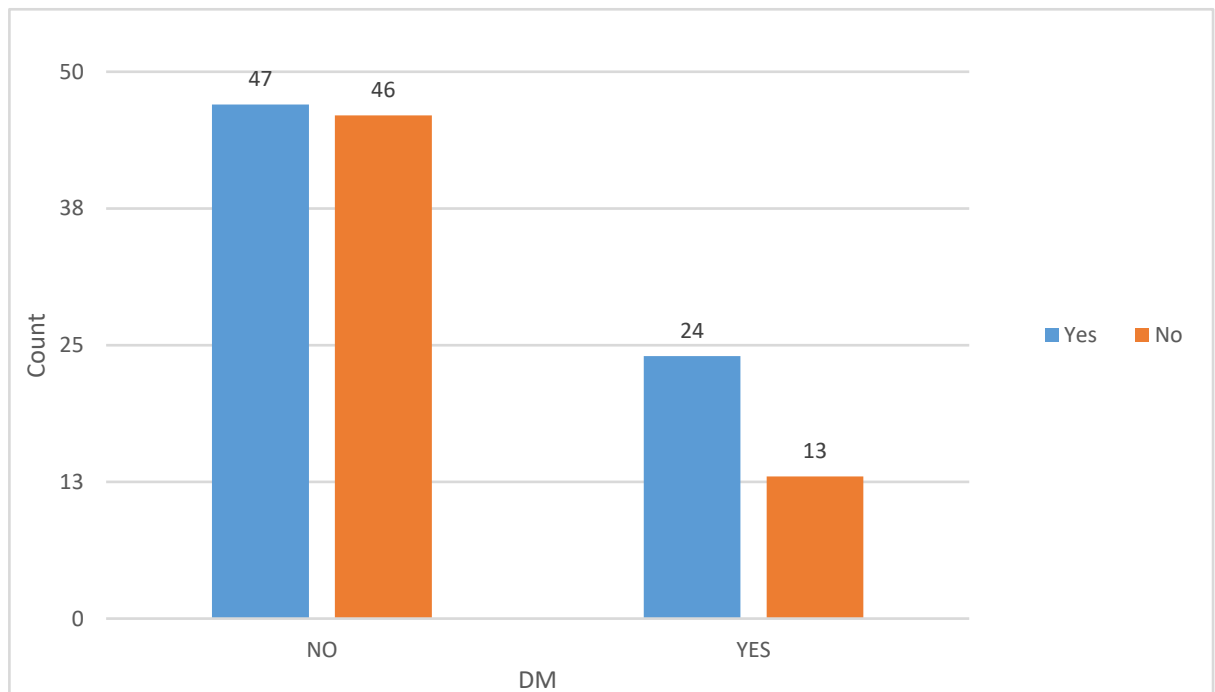


Fig 19. Though patients with Diabetes Mellitus have higher rate of hospitalisation than those who doesn't, but our study fails to prove statistically significant rate of hospitalisation among Diabetes patients.

**Table 20. Correlation of Duration of Stay in Hospital with various factors:-**

		Duration of stay	
		Mean	Standard Deviation
Hemoglobin	<8	7.12	6.47
	>8	5.27	3.44
S. Albumin	<3.4	7.43	6.97
	>3.4	5.49	3.86
Frequency of MHD	1	3.00	-
	2	5.62	4.04
	3	6.84	6.25
DM	NO	7.04	5.77
	YES	4.13	2.13
Cardiac abnormalities	No	6.08	3.86
	Yes	6.10	5.32

- Patients with Hemoglobin < 8gm% has higher duration of hospitalisation compared to patients with Hemoglobin >8gm%
- Patients with serum Albumin < 3.4gm /dl has higher duration of hospitalisation compared to patients with Serum Albumin > 3.4gm/ dl.
- Variation in Frequency of Maintenance Hemodialysis, Cardiac abnormalities and presence of Diabetes Mellitus has no correlation with Duration of hospital stay among the study population.

**Table 21. Mortality in our study population:-**

	Frequency	Percent
Acute hemorrhagic stroke	2	25.0%
Acute pulmonary edema	3	37.5%
Sepsis / septic shock	1	12.5%
Uremic encephalopathy	2	25.0%

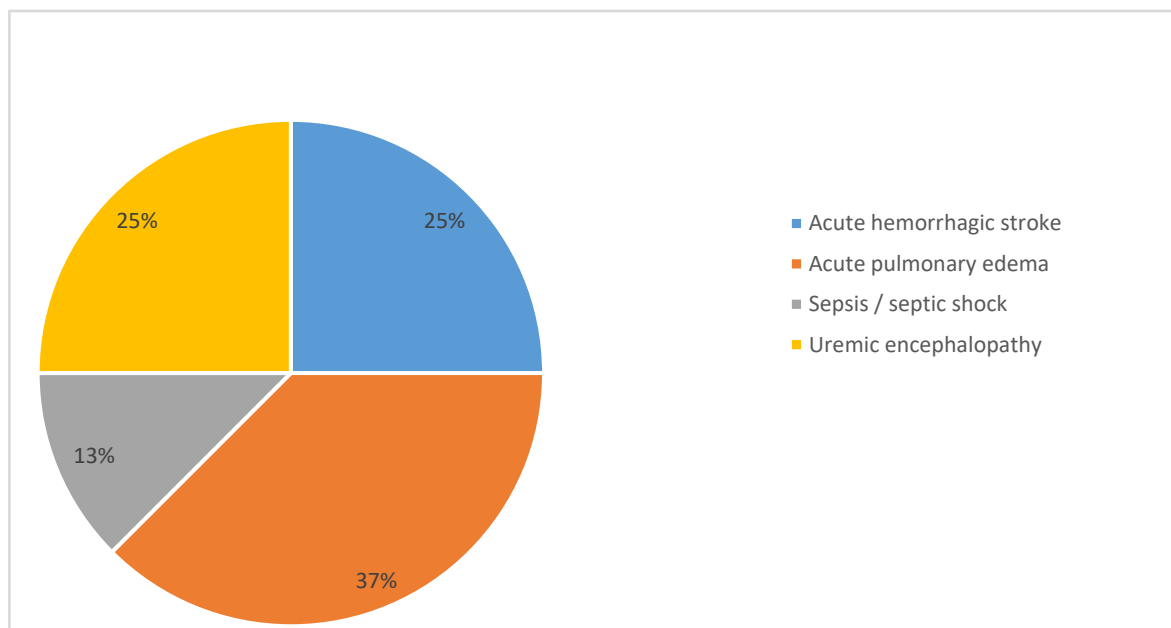


Fig 20. Among 130 patients under study, about 8 patients had expired during the study period of 6 months. Among those expired, about 3 patients have expired due to Acute Pulmonary edema and 2 patients have expired due to Acute Hemorrhagic stroke and uremic encephalopathy.

## **DISCUSSION**

This study was conducted in Rajiv Gandhi Government General Hospital from April 2022 to September 2022. A total of 130 cases were studied. The clinical and diagnostic findings of this study are compared with our studies in literature here.

### **DISTRIBUTION OF AGE GROUPS IN OUR STUDY:**

In our study, most of the patients were between 41-50 years of age. The youngest patient in our study was 18 years of age and the oldest was 76 years of age, with a median age of 41 years of age.

### **GENDER DISTRIBUTION OF THE PATIENTS:**

In our study also males contributed to the majority of cases as similar to major studies done on CKD which contributes around 67%, whereas females contributed to only 33% of the cases.

### **DISTRIBUTION OF COMORBIDITIES IN OUR STUDY:**

In our study population, 20.8% of patients had Coronary Artery Disease, 28.5% of patients had Diabetes Mellitus, 87.7% of patients had Hypertension, 9.2% of patients had Hypothyroidism, 10.8% of patients had Old Pulmonary Tuberculosis, 2.3% of patients of patients had Seizure disorder and 3.8% of patients had SLE.

#### DISTRIBUTION OF HEMOGLOBIN IN OUR STUDY:

About 38.5% of study population had Hemoglobin < 8gm/dl, and 61.5% of study population had Hemoglobin > 8gm/dl. Majority of our study population are under treatment with Erythropoietin and Iron supplements, which could be the possible cause for less percentage of CKD patients with severe anaemia.

#### DISTRIBUTION OF SERUM ALBUMIN IN OUR STUDY:

About 30% of study population had Serum Albumin < 3.4 gm/ dl, and 70% of study population had Serum Albumin > 3.4 mhd/ dl.

#### OUTCOME OF HOSPITALISATION AMONG OUR STUDY POPULATION:

In our study among 130 patients, there are 78 Hospitalisation out of which 8 patients have expired and 70 patients have been discharged and about 15 patients have 2nd hospitalisation during the study period of 6 months.

#### CAUSES FOR HOSPITALISATION AMONG PATIENTS ON MHD:

Majority of hospitalisation among our study population is due to Acute Pulmonary edema which constitutes about 31.4% of hospitalisation, Anasarca about 11.4% of hospitalisation, Uremic Gastritis about 8.6% of hospitalisation followed by febrile illness, Pneumonia, severe metabolic acidosis and so on.

#### DISTRIBUTION OF DURATION OF RENAL DISEASE IN OUR STUDY:

Majority of patients in our study population have renal disease for duration of 5-10 years contributing about 46.2% followed by 1-5 years contributing about 41.5% , and 8% of patients have each less than 1 years and more than 10 years duration of Renal failure.

#### DISTRIBUTION OF FREQUENCY OF MHD IN OUR STUDY:

Majority of patients are under 2/7 dialysis which constitutes about 68.5% and about 30.8% patients are under 3/7 maintenance hemodialysis.

#### CORRELATION OF AGE WITH HOSPITALISATION IN OUR STUDY

##### POPULATION:

About 20 patients in our study are above 60 years of age and out of which about 15 patients had history of hospitalisation during 6 months follow up, whereas only 56 patients out of 110 patients under 60 years had history of hospitalisation. Thus Age more than 60 years is one of the important factor responsible for hospitalisation which had significant p value of 0.047 which is very much similar to the study by Jishu Deb Nath et al.



## CORRELATION OF HEMOGLOBIN WITH HOSPITALISATION IN OUR STUDY:

Though anaemia is thought to be one of factor with good correlation with Hospitalisation, in our study patients with Hemoglobin < 8gm /dl was compared with those who have higher. There were no significance in rate of hospitalisation among both the groups.

## CORRELATION OF SERUM ALBUMIN WITH HOSPITALISATION IN OUR STUDY:

Hypoalbuminemia is an another factor that is thought to have correlation with hospitalisation, thus patients with serum albumin less than 3.4gm/dl was compared with those who have higher levels. There was no significant higher risk of hospitalisation among both groups.

## CORRELATION OF CARDIAC ABNORMALITIES WITH HOSPITALISATION:

In our study population, about 86 patients have cardiac abnormalities in echocardiography, out of which about 60 patients had hospitalisation during the study period, whereas 44 had normal echocardiography, out of which only 11 patients had hospitalisation. Thus cardiac abnormalities is one of the important factor which increases the risk of hospitalisation. Our study also proves that with a significant p value of < 0.0001. This finding is in consistent with previous studies done by Joachim Jankowski et al that patients with CKD have high cardiovascular risk, which

contributes to significant morbidity and mortality among patients on End Stage Renal Disease.

#### CORRELATION OF DURATION OF RENAL DISEASE WITH HOSPITALISATION:

Though higher the duration of Renal disease, higher the rate of hospitalisation was expected initially, but it was found that most of the patients even with duration of renal failure for more than 5 years , due to their excellent compliance to fluids, salt, drugs, dialysis and regular follow up, able to lead reasonably good quality of life. Our study was not able to demonstrate correlation between duration of disease and frequency of hospitalisation.

#### CORRELATION OF FREQUENCY OF MHD WITH HOSPITALISATION:

In our centre, Most of the patients who are under 2/7 MHD when not tolerated in the form of development of Acute pulmonary edema or uremic symptoms are changed to 3/7 MHD and hence most of the patients under 3/7 have longer duration of disease and also has multiple comorbidities and hence higher rate of hospitalisation were noted among patients on 3/7 MHD.

#### CORRELATION OF DIABETES MELLITUS WITH HOSPITALISATION:

Though patients with Diabetes Mellitus have higher rate of hospitalisation than those who doesn't, but our study fails to prove statistically significant rate of hospitalisation among Diabetes patients.

## CORRELATION OF DURATION OF HOSPITAL STAY WITH VARIOUS

### FACTORS:

Patients with Hemoglobin  $< 8\text{gm}\%$  has higher duration of hospitalisation compared to patients with Hemoglobin  $>8\text{gm}\%$

Patients with serum Albumin  $< 3.4\text{gm}/\text{dl}$  has higher duration of hospitalisation compared to patients with Serum Albumin  $> 3.4\text{gm}/\text{dl}$ .

Variation in Frequency of Maintenance Hemodialysis, Cardiac abnormalities and presence of Diabetes Mellitus has no correlation with Duration of hospital stay among the study population.

### MORTALITY IN OUR STUDY POPULATION:

Among 130 patients under study, about 8 patients had expired during the study period of 6 months. Among those expired, about 3 patients have expired due to Acute Pulmonary edema, 2 patients have expired due to Acute Hemorrhagic stroke, 2 patients due to uremic encephalopathy and 1 patient due to sepsis and septic shock.

This can be related with the study on cause of death in patients on renal failure by Stephanie Thompson et al.

## CONCLUSION

- Age and Cardiac abnormalities are the two major factors which increases the rate of hospitalisation among Chronic Kidney Disease patients on Maintenance Hemodialysis
- Hypoalbuminemia and Anaemia are the two majority factors which increases the duration of hospitalisation among CKD patients.
- Anaemia, Hypoalbuminemia, duration of Renal failure, Duration of Maintenance Hemodialysis, Frequency of Maintenance Hemodialysis, Diabetes Mellitus doesn't significantly increase Hospitalisation among Chronic kidney disease patients on Maintenance Hemodialysis.
- Major etiologies for hospitalisation among patients on Maintenance Hemodialysis are in the order of increased frequency as follows,
  1. Acute Pulmonary Edema
  2. Anasarca
  3. Uremic Gastritis
  4. Acute febrile illness
  5. Pneumonia
  6. Severe metabolic acidosis
- Mortality during the study period of 6 months is 6.15% , with causes being
  - ◆ Acute pulmonary edema

- ◆ Acute Hemorrhagic stroke
- ◆ Uremic encephalopathy
- ◆ Sepsis.

From our study, we conclude that regular cardiac evaluation and adequate correction of anaemia and Hypoalbuminemia in the patients of chronic kidney disease on maintenance hemodialysis, the frequency of hospitalisation and duration of stay can be very much reduced, which can very well improve the quality of life among these patients.

**Limitations of the study:**

- The study was based at a single academic center
- Sample size is small.
- Long term follow up is not done.
- Fluid and Drug compliance - one of the prime factor for hospitalisation could not be accurately elicited.

## ABBREVIATIONS:

1. CKD - Chronic Kidney disease
2. AKI - Acute kidney injury
3. GFR - Glomerular filtration rate
4. ACR - Albumin creatinine ratio
5. RAAS - Renin angiotensin aldosterone system
6. ACE - Angiotensin converting enzyme
7. ARB - Angiotensin Receptor blocker
8. SGLT - Sodium Glucose Co-Transporter
9. ESRD - End stage Renal Disease
10. MHD - Maintenance Hemodialysis
11. CAD - Coronary Artery Disease
12. DM - Diabetes Mellitus
13. SLE - Systemic lupus erythematosus
14. PTB - Pulmonary Tuberculosis
15. UTI - Urinary Tract Infection

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# **PROFORMA**

## **A PROSPECTIVE STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE CENTRE IN CHENNAI**

PROFORMA NO.

Name :

Age :

Sex :

Occupation:

Address:

Contact number:

Diagnosis:

Duration of Renal disease :

Duration and frequency of maintenance hemodialysis:

Vascular assess :

### **COMORBIDITIES**

1. Diabetes Mellitus- yes / no - duration
2. Systemic Hypertension- yes / no - duration
3. Coronary artery disease- yes / no - duration
4. Others

## HOSPITALISATION

### HISTORY

History of presenting illness

History of drug therapy, dose and compliance

Fluid compliance

### GENERAL EXAMINATION

Conscious, Oriented to time, place and person.

Pallor, icterus, cyanosis, clubbing, pedal edema, lymphadenopathy

VITALS - Blood pressure, Pulse rate, Respiratory rate, Temperature, saturation.

### SYSTEMIC EXAMINATION

CVS -

RS-

CNS-

Abdomen -

### INVESTIGATIONS:

TC-

Hb-

Plt-

RBS-

Urea-

Creatinine-

Serum Electrolytes :- Na<sup>+</sup> -

K<sup>+</sup> -

Serum Calcium -

Phosphorus -

Total bilirubin-

Direct-

Indirect-

Total protein-

Sr. Albumin-

Sr.Globulin-

SGOT-

SGPT-

ABG -

Urine routine -

Electrocardiography-

Chest X ray -

Echocardiography-

DIAGNOSIS OF CURRENT ADMISSION -

Outcome of this hospitalisation :-

- ✓ Duration of stay -
- ✓ Discharge/ Death.



## INFORMATION SHEET

We are conducting a study on **“A PROSPECTIVE STUDY ON OUTCOMES OF HOSPITALISATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE HOSPITAL IN CHENNAI”** among patients attending Rajiv Gandhi Government General Hospital, Chennai.

The purpose of this study is to analyze the etiology, frequency and outcomes of hospitalization in patients on maintenance hemodialysis.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator

Signature of Participant

Date:

Place:

## PATIENT CONSENT FORM

Study Detail : **“A PROSPECTIVE STUDY ON OUTCOMES OF HOSPITALISATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE HOSPITAL IN CHENNAI”**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's  
Name :

Patient's Age :

Identification  
Number :

Patient may check (√) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

I hereby consent to participate in this study.

I hereby give permission to undergo detailed clinical examination and blood investigations as required.

Signature of investigator Signature/Thumb impression of participant

**Patient name and address**

## நோயாளி ஒப்புதல் படிவம்

ஆய்வு விவரம் : ஹீமோடையாலிசிஸ் நோயாளிகளில்  
மருத்துவமனையில் சேர்க்கப்படுவதற்கான காரணவியல்  
பற்றிய ஆய்வு.

ஆய்வு மையம்: ராஜீவ் காந்தி அரசு பொது மருத்துவமனை,  
சென்னை.

நோயாளியின் பெயர்:

நோயாளியின் வயது:

அடையாள எண் :

நோயாளி இந்த பெட்டிகளை (✓) செய்யலாம்:-

- மேற்கண்ட ஆய்விற்கான நடைமுறையின் நோக்கத்தை நான் புரிந்து கொண்டேன் என்பதை உறுதிப்படுத்துகிறேன். கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளது, எனது முழு திருப்திக்கும் எனது எல்லா கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளது.
- ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும், எனது சட்ட உரிமைகள் பாதிக்கப்படாமல், காரணமின்றி எந்த நேரத்திலும் திரும்பப் பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்துகொள்கிறேன்.
- மருத்துவ ஆய்வின் ஸ்பான்சர், ஸ்பான்சர் சார்பாக பணிபுரியும் மற்றவர்கள், நெறிமுறைக் குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதார பதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்துகொள்கிறேன், தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள எந்தவொரு ஆராய்ச்சியையும் பொறுத்தவரை இது தொடர்பாக, நான் ஆய்வில் இருந்து விலகினாலும் இந்த அணுகலை ஒப்புக்கொள்கிறேன். எவ்வாறாயினும், சட்டத்தின் கீழ் தேவைப்படாவிட்டால், மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிடப்பட்ட எந்தவொரு தகவலிலும் எனது அடையாளம் வெளிப்படுத்தப்படாது என்பதை நான் புரிந்துகொள்கிறேன். இந்த ஆய்வில் எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டை கட்டுப்படுத்த வேண்டாம் என்று நான் ஒப்புக்கொள்கிறேன்.
- மேற்கண்ட ஆய்வில் பங்கேற்கவும், ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும்,

ஆய்வுக் குழுவுடன் உண்மையுடன் ஒத்துழைக்கவும், எனது உடல்நலம் அல்லது நல்வாழ்வில் ஏதேனும் சரிவு ஏற்பட்டால் அல்லது எதிர்பாராத அல்லது ஏதேனும் ஏற்பட்டால் உடனடியாக ஆய்வு ஊழியர்களுக்கு அறிவிக்கவும் ஒப்புக்கொள்கிறேன். அசாதாரண அறிகுறிகள்.

- இந்த ஆய்வில் பங்கேற்க நான் இதன்மூலம் ஒப்புக்கொள்கிறேன்.
- தேவைக்கேற்ப விரிவான மருத்துவ பரிசோதனை மற்றும் இரத்த விசாரணைகளை மேற்கொள்ள நான் இதன்மூலம் அனுமதி அளிக்கிறேன்.

புலனாய்வாளரின்  
கையொப்பம்

பங்கேற்பாளரின் கையொப்பம் / கட்டைவிரல் எண்ணம்

நோயாளியின் பெயர் மற்றும் முகவரி

# INSTITUTE ETHICAL COMMITTEE APPROVAL

**INSTITUTIONAL ETHICS COMMITTEE  
MADRAS MEDICAL COLLEGE, CHENNAI 600 003**

EC Reg. No(CDSCO).ECR/270/Inst./TN/2013/RR-20  
EC Reg. No(DHR).EC/NEW/INST/2021/1618  
Telephone No.044 25305301  
Fax: 011 25363970

**CERTIFICATE OF APPROVAL**

To  
**Dr. L.ARUN KHUMAR,**  
MD Internal Medicine Post Graduate student,  
Institute of Internal Medicine,  
Madras Medical College,  
Chennai-600 003.

Dear Dr. L.ARUN KHUMAR,

The Institutional Ethics Committee has considered your request and approved your study titled "**A STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS**"- NO.29052022. The following members of Ethics Committee were present in the meeting held on **18.05.2022** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar,MS Orth.,D.Orth.,M.Ch Orth (Liverpool) :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Meena Suresh, MD.,DGO.,Prof.of Obst & Gynaec, IOG,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.








Member Secretary - Ethics Committee

**MEMBER SECRETARY  
INSTITUTIONAL ETHICS COMMITTEE  
MADRAS MEDICAL COLLEGE  
CHENNAI-600 003.**

### Document Information

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## PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled of the **A PROSPECTIVE STUDY ON OUTCOMES OF HOSPITALIZATION IN PATIENTS ON MAINTENANCE HEMODIALYSIS IN A TERTIARY CARE CENTRE IN CHENNAI** by the candidate **Dr. L. ARUN KHUMAR** with registration Number **200120100506** for the award of **DOCTOR OF MEDICINE** in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **6%** percentage of plagiarism in the dissertation.



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