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

Dissertation submitted to

## THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU.

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

## M.D. BRANCH - I (GENERAL MEDICINE)

## **REGISTRATION NUMBER - 200120100506**



## **INSTITUTE OF INTERNAL MEDICINE**

MADRAS MEDICAL COLLEGE

**CHENNAI - 600 003** 

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

Deceden

Professor of Medicine, Institute of Internal Medicine, Professor of Medicine, Prof. Of Medicine Reg. No. 55530

MMC & RGGGH,

Chennai - 600 003.

HOD

Prof. Dr. C. HARIHARAN, M.D.,

Professor and Director, Dr. C. HARIHARAN, M.D., Institute of Internal Menoi No. 45626 Director & Fronssor of Medicine Director & Fronssor of Medicine MMC & RG Raily Gandhi Govt. General Hospital, MMC & RG Raily Gandhi Govt. General - 600 000.

Chennai - 600 003.

PROF. Dr. E. THERANIRAJAN M.D., D.C.H., DEAN The Dean MADRAS MEDICAL COLLEGE

Madras Medical College &

Rajiv Gandhi Government General Hospital

Chennai- 600 003

# CERTIFICATE OF GUIDE

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.

Queden

Prof. Dr. T.B. UMADEVI M.D., Professor of Medicine, Institute of Internal Medicine, Madras Medical College, RGGGH, Chennai – 600 003.

#### 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

4

Place : Chennai

LAS Signature of the candidate.

Dr. L. ARUN KHUMAR

Reg. no. 200120100506

#### ACKNOWLEDGEMENTS

At the outset, I wish to thank our Dean **DR. E. THERANIRAJAN, M.D., DCH., MRCPCH(UK).,FRCPCH(UK).,** for permitting me to use the facilities of Rajiv Gandhi Government General Hospital to conduct this study. My beloved Director of the Institute of Internal Medicine, **Prof. Dr. C. HARIHARAN M.D.,** has always guided me with his valuable words of advice and has encouraged innovative thinking and original research work done by post graduates.

I shall remain eternally grateful to my unit chief **Prof. Dr. T. B. UMADEVI , M.D.**, who has given me her moral support and encouragement throughout the conduct of the study.

I am extremely grateful to the Director and Professor of Institute of Nephrology, Rajiv Gandhi Government General Hospital, **Prof. Dr. N. GOPALAKRISHNAN. M.D DM**, without whose constant support, guidance, cooperation and encouragement this study would not have been possible.

I would also to express my special thanks to the Assistant Professor Institute of Nephrology, **Prof. Dr. T. DINESH KUMAR, M.D DM** for his valuable guidance throughout the study. I offer my heartfelt thanks to my unit Assistant Professors Dr. Jayaraj M.D.,

**Dr. Umamaheshwari M.D., Dr. D. Venkatesan M.D.,** for their constant encouragement and critical suggestions throughout the study.

My patients, who form the most integral part of the work, were always kind and cooperative. I pray God to give them courage and strength to endure their illness.

I thank my friends and family who have stood by me during my times of need. Their help and support have always been invaluable to me. And last but not the least I would like to thank the Lord Almighty for His grace and blessings without which nothing would have been possible.

## **TABLE OF CONTENTS**

S.NO	TOPIC	PAGE NUMBER
1	INTRODUCTION	9
2	AIMS AND OBJECTIVE	10
3	REVIEW OF LITERATURE	11
4	METHODS AND MATERIALS	51
5	RESULTS	55
6	DISCUSSION	77
7	CONCLUSION	83
	ANNEXURES	
	≻ BIBLIOGRAPHY	
	> PROFOMA	
8	➢ INFORMATION SHEET	86
	► ETHICAL CLEARANCE	
	➢ PLAGIARISM CERTIFICATE	
	➤ MASTER CHART	

## 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.
- 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 m2, and kidney failure is defined as a GFR of less than 15 mL/min per 1.73 m2 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 x (Pcr)-1.154 x (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

Estimated Creatinine Clearance = (140-Age) x Wt in Kg 72x Serum Creatinine

Multiply it by 0.85 for females

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

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

- 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 m2
- G2–GFR60to89mL/min per 1.73m2
- G3a-GFR45to59mL/min per 1.73m2
- G3b-GFR30to44mL/min per 1.73m2
- G4–GFR15to29mL/min per 1.73m2
- G5 GFR <15 mL/min per 1.73 m2 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 m2) 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 m2, 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.

			Γ	Albuminuria stages, description and range			
			Ī	A1	A2	A3	
				Normal to mildly increased	Moderately increased 3-29 mg/mmol	Severely increased ≥30 mg/mmol	
			Ĩ	<3 mg/mmol			
GFR 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 agestandardized 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 hyperfilteration 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 upregulated. 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 k 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.



# **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 Nterminal 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 membrance 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 protolytic enzymes.

## VASCULAR SCLEROSIS

The process of renal scarring includes vascular sclerosis. The progression of CKD is associated with renal arteriolar hyalinosis, which can occur at an early stage of the disease even in the absence of severe hypertension. While hyalinosis of the post glomerular arterioles may increase interstitial ischemia and lead to fibrosis, afferent arteriole hyalinosis 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 m2, 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 m2).

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

Diseases - Per	centage
Diabetes mellitus - 2	31.2%
Hypertension - 1	4.1%
Glomerulonephritis(GN) -	14.4%
Tubulo-interstitial Nephritis	- 7%
Hereditary or cystic diseases.	- 2.1%
Miscellaneous - 15	5.9%
Unknown - 15.39	%

## **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 angiotensinconverting 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

patients.

## Additional therapies in proteinuric patients — Treatment with an ACE

disorders such reflux nephropathy, proteinuria frequently coexists with CKD in

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 m2. 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 m2 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 (1,25dihydroxyvitamin 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 m2. 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 calciumsensing 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 m2 [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 m2), at least every six months among patients with stage 4 to 5 CKD (ie, eGFR  $\leq$ 29 mL/min/1.73 m2), 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 m2), 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 m2, 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 m2. 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)	ad ratio 1.0:1.0 1.0:1.0				
Carbohydrate	Balance of nonprotein calories	1			
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			

Ahmed K, Kopple J. Nutritional management of renal disease. In: Primer on Kidney Diseases, Greenberg A (Ed). Academic Press, San Diego, CA, 1994, p.289.

**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.

Readenic Press, dan Diego, CA, 1994, p.205.
Ikizler IA. Nutrition and kidney disease. In: Primer on Kidney Diseases, Greenberg A (Ed). Elsevier, Philadelphia, 2005, p.496.

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** $^{(32,33)\&}$  — 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 :

38

• 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 m2 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 m2, 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 m2, 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.

 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.

45

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.

46

**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

48

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

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%



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:-

Diagnosis	Count	%
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.	Distribut	ion of	Duration	of MHD	in our	study:-
							•/

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



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		Tetel	Р	
		Yes	No	lotal	value	
		Count	56	54	110	
Age group >60	<00	% within Hospitalization	78.9%	91.5%	84.6%	
		Count	15	5	20	
	>00	% within Hospitalization	21.1%	8.5%	15.4%	0.047
	Count	71	59	130		
Total		% 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		T- 4-1	Р	
		Yes	No	Total	value	
	~0	Count	32	18	50	
Hemoglobin	<8	% within Hospitalization	45.1%	30.5%	38.5%	
	>8	Count	39	41	80	
		% within Hospitalization	54.9%	69.5%	61.5%	0.089
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	





# Table 14. Correlation of serum Albumin with Hospitalisation in our study population :

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

		Hospitalization		Tatal	D 1		
		Yes	No	Total	r value		
	No	Count	11	33	44		
Cardiac abnormalities		% within Hospitalization	15.5%	55.9%	33.8%		
	Yes	Count	60	26	86	<0.0001	
		% 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 o	<b>Duration</b>	of Renal d	lisease with	Hospitalisation	in our
-------------------------	-----------------	------------	--------------	-----------------	--------

#### study population:-

			Hospitalization	Total	P value	
			Yes No			Total
		Count	28	34	62	
Ouration of disease<5>5Total	<5	% within Hospitalization	39.4%	57.6%	47.7%	
	>5	Count	43	25	68	
		% within Hospitalization	60.6%	42.4%	52.3%	0.39
		Count	71	59	130	
	Total % 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			Dyrahua
			Yes	No	Total	P value
Duration	<5	Count	37	38	75	
of MHD		% within Hospitalization	52.1%	64.4%	57.7%	
	>5	Count	34	21	55	
		% within Hospitalization	47.9%	35.6%	42.3%	0.159
Total		Count	71	59	130	
		% within Hospitalization	100.0%	100.0%	100.0%	





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

#### population :-

			Hospita	lization	Tatal	Р	
			Yes	No	Total	value	
	1	Count	1	0	1		
	1	% within Hospitalization	1.4%	1.4% 0.0% 0.8			
Frequency	2	Count	40	40 49 89			
of MHD	Z	% within Hospitalization	56.3%	83.1%	68.5%		
	2	Count	30	10	40	0.004	
	3	% within Hospitalization	42.3%	16.9%	30.8%		
		Count	71	59	130		
Total		% 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:-

			Hospitalizat	tion	Total	Dyalua
			Yes	No	Total	P value
		Count	47	46	93	
DM	NO	% within Hospitalization	66.2%	78.0%	71.5%	
DIVI		Count	24	13	37	
	YES	% within Hospitalization	33.8%	22.0%	28.5%	0.139
		Count	71	59	130	
Total		% 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
Hamaalahin	<8	7.12	6.47
Hemoglobin	>8	5.27	3.44
C. Alburgin	<3.4	7.43	6.97
S. Albumin	>3.4	5.49	3.86
	1	3.00	-
Frequency of MHD	2	5.62	4.04
	3	6.84	6.25
DM	NO	7.04	5.77
DIVI	YES	4.13	2.13
Condias shuarmalities	No	6.08	3.86
Cardiac abnormalities	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 < 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.

#### 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



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 erythematosis
- 14. PTB Pulmonary Tuberculosis
- 15. UTI Urinary Tract Infection

### **BIBLIOGRAPHY**:

Chapter 1: Definition and classification of CKD. Kidney Int Suppl (2011) 2013;
 3:19.

2) Remuzzi G, Benigni A, Remuzzi A. Mechanisms of progression and regression of renal lesions of chronic nephropathies and diabetes. J Clin Invest 2006; 116:288.

3) Hostetter TH, Olson JL, Rennke HG, et al. Hyperfiltration in remnant nephrons: a potentially adverse response to renal ablation. Am J Physiol 1981; 241:F85.

 4) Earley A, Miskulin D, Lamb EJ, et al. Estimating equations for glomerular filtration rate in the era of creatinine standardization: a systematic review. Ann Intern Med 2012; 156:785.

5) Levey AS, Stevens LA, Schmid CH, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med 2009; 150:604.

6) Stevens LA, Schmid CH, Greene T, et al. Comparative performance of the CKD Epidemiology Collaboration (CKD-EPI) and the Modification of Diet in Renal Disease (MDRD) Study equations for estimating GFR levels above 60 mL/min/1.73 m2. Am J Kidney Dis 2010; 56:486.

7) Delgado C, Baweja M, Crews DC, et al. A Unifying Approach for GFR Estimation: Recommendations of the NKF-ASN Task Force on Reassessing the Inclusion of Race in Diagnosing Kidney Disease. J Am Soc Nephrol 2021. 8) Stevens LA, Coresh J, Schmid CH, et al. Estimating GFR using serum cystatin C alone and in combination with serum creatinine: a pooled analysis of 3,418 individuals with CKD. Am J Kidney Dis 2008; 51:395.

9) GBD Chronic Kidney Disease Collaboration. Global, regional, and national burden of chronic kidney disease, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet 2020; 395:709.

10) Barry M.Brenner. Brenner & Rector's The Kidney. 8th edition. Philadelphia:Saunders Elsevier; 2008.p.1728-1737

11) Mohsen El Kossi and Meguid El Nahas, Epidemiology and Pathophysiology of Chronic Kidney Disease: Natural History, Risk Factors, and Management & Iain C. Macdougall and Kai-Uwe Eckardt, Anemia in Chronic Kidney Disease. In: John Feehally, Jurgen Floege and Richard J Johnson, editors. Comprehensive Clinical Nephrology. 3rd edition. Philadelphia: Mosby Elsevier; 2007.p.813-821, 853-860.

12) Arend, Armitage, Clemmons, Drazens, Griggs, LaRusso .Cecil Medicine 23rd Edition Volume I Section I to XV p.921. Elsevier

13) Schieppati A, Pisoni R, Remuzzi G. Pathophysiology and management of c hronic kidney disease. In: Primer on Kidney Diseases, Greenberg A (Ed), El sevier Saunders, Philadelphia 2005. p.444.

14) Stefanski A, Schmidt KG, Waldherr R, Ritz E. Early increase in blood pressure and diastolic left ventricular malfunction in patients with glomerulonephritis. Kidney Int 1996; 50:1321. 15) Rennke HG, Anderson S, Brenner BM. Structural and functional correlations in the progression of renal disease. In: Renal Pathology, Tisher CC, Brenner BM (Eds), Lippincott, Philadelphia 1989. p.43.

16) Heerspink HJ, Perkins BA, Fitchett DH, et al. Sodium Glucose Cotransporter 2Inhibitors in the Treatment of Diabetes Mellitus: Cardiovascular and Kidney Effects,Potential Mechanisms, and Clinical Applications. Circulation 2016; 134:752.

17) Orth SR. Effects of smoking on systemic and intrarenal hemodynamics: influence on renal function. J Am Soc Nephrol 2004; 15 Suppl 1:S58.

18) Orth SR, Hallan SI. Smoking: a risk factor for progression of chronic kidney disease and for cardiovascular morbidity and mortality in renal patients-- absence of evidence or evidence of absence? Clin J Am Soc Nephrol 2008; 3:226.

19) Weir MR, Fink JC. Salt intake and progression of chronic kidney disease: an overlooked modifiable exposure? A commentary. Am J Kidney Dis 2005; 45:176.

20) Gonick HC, Kleeman CR, Rubini ME, Maxwell MH. Functional impairment in chronic renal disease.3. Studies of potassium excretion. Am J Med Sci 1971;261:281.

21) Hsu CY, Chertow GM. Elevations of serum phosphorus and potassium in mild to moderate chronic renal insufficiency. Nephrol Dial Transplant 2002; 17:1419.

22) Warnock DG. Uremic acidosis. Kidney Int 1988; 34:278.

23) Widmer B, Gerhardt RE, Harrington JT, Cohen JJ. Serum electrolyte and acid base composition. The influence of graded degrees of chronic renal failure. Arch Intern Med 1979; 139:1099. 24) Delmez JA, Slatopolsky E. Hyperphosphatemia: its consequences and treatment in patients with chronic renal disease. Am J Kidney Dis 1992; 19:303.

25) National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002;39:S1.

26) Muntner P, Jones TM, Hyre AD, et al. Association of serum intact parathyroid hormone with lower estimated glomerular filtration rate. Clin J Am Soc Nephrol 2009; 4:186.

27) Eschbach JW. Erythropoietin 1991--an overview. Am J Kidney Dis 1991; 18:3.

28) Appel G. Lipid abnormalities in renal disease. Kidney Int 1991; 39:169.

29) Procci WR, Goldstein DA, Adelstein J, Massry SG. Sexual dysfunction in the male patient with uremia: a reappraisal. Kidney Int 1981; 19:317.

30) Kopple JD, Greene T, Chumlea WC, et al. Relationship between nutritional status and the glomerular filtration rate: results from the MDRD study. Kidney Int 2000; 57:1688.

31) Garg AX, Blake PG, Clark WF, et al. Association between renal insufficiency and malnutrition in older adults: results from the NHANES III. Kidney Int 2001; 60:1867.

32) Dalrymple LS, Katz R, Kestenbaum B, et al. The risk of infection-related hospitalization with decreased kidney function. Am J Kidney Dis 2012; 59:356.

33) Wu MY, Hsu YH, Su CL, et al. Risk of herpes zoster in CKD: a matched- cohort study based on administrative data. Am J Kidney Dis 2012; 60:548.

34) Berlyne GM, Shaw AB. Red eyes in renal failure. Lancet 1967; 1:4.

35) Gómez CG, Valido P, Celadilla O, et al. Validity of a standard information protocol provided to end-stage renal disease patients and its effect on treatment selection. Perit Dial Int 1999; 19:471.

36) Mange KC, Joffe MM, Feldman HI. Effect of the use or nonuse of long-term dialysis on the subsequent survival of renal transplants from living donors. N Engl J Med 2001; 344:726.

37) Sidawy AN, Spergel LM, Besarab A, et al. The Society for Vascular Surgery: clinical practice guidelines for the surgical placement and maintenance of arteriovenous hemodialysis access. J Vasc Surg 2008; 48:2S.

38) Fink JC. Do you need to stay in school to get a kidney transplant? Am J Kidney Dis 2008; 51:717.

39) KDIGO. Chapter 1: Definition and classification of CKD. Kidney Int Suppl 20
13; 3:19. http://www.kdigo.org/clinical\_practice\_guidelines/pdf/CKD/KDIG
O\_2012\_CKD\_GL.pdf (Accessed on March 04, 2013).

40) Levinsky NG. The organization of medical care. Lessons from the Medicare end stage renal disease program. N Engl J Med 1993; 329:1395.

41) Chertow GM, Johansen KL, Lew N, et al. Vintage, nutritional status, and survival in hemodialysis patients. Kidney Int 2000; 57:1176.

42) Bloembergen WE, Port FK, Mauger EA, Wolfe RA. Causes of death in dialysis patients: racial and gender differences. J Am Soc Nephrol 1994; 5:1231.

43) Collins AJ, Hanson G, Umen A, et al. Changing risk factor demographics in endstage renal disease patients entering hemodialysis and the impact on long-term mortality. Am J Kidney Dis 1990; 15:422. 44) Mailloux LU, Bellucci AG, Napolitano B, et al. Survival estimates for 683 patients starting dialysis from 1970 through 1989: identification of risk factors for survival. Clin Nephrol 1994; 42:127.

45) Yoshino M, Kuhlmann MK, Kotanko P, et al. International differences in dialysis mortality reflect background general population atherosclerotic cardiovascular mortality. J Am Soc Nephrol 2006; 17:3510.

46) Chung SH, Lindholm B, Lee HB. Influence of initial nutritional status on continuous ambulatory peritoneal dialysis patient survival. Perit Dial Int 2000; 20:19.

47) Mc Causland FR, Waikar SS, Brunelli SM. Increased dietary sodium is independently associated with greater mortality among prevalent hemodialysis patients. Kidney Int 2012; 82:204.

48) Termorshuizen F, Dekker FW, van Manen JG, et al. Relative contribution of residual renal function and different measures of adequacy to survival in hemodialysis patients: an analysis of the Netherlands Cooperative Study on the Adequacy of Dialysis (NECOSAD)-2. J Am Soc Nephrol 2004; 15:1061.

49) Benz RL, Pressman MR, Hovick ET, Peterson DD. Potential novel predictors of mortality in end-stage renal disease patients with sleep disorders. Am J Kidney Dis 2000; 35:1052.

50) Held PJ, Levin NW, Bovbjerg RR, et al. Mortality and duration of hemodialysis treatment. JAMA 1991; 265:871.

51) Saran R, Bragg-Gresham JL, Levin NW, et al. Longer treatment time and slower ultrafiltration in hemodialysis: associations with reduced mortality in the DOPPS. Kidney Int 2006; 69:1222.

52) Zoccali C, Moissl U, Chazot C, et al. Chronic Fluid Overload and Mortality in ESRD. J Am Soc Nephrol 2017; 28:2491.

53) Foley RN, Gilbertson DT, Murray T, Collins AJ. Long interdialytic interval and mortality among patients receiving hemodialysis. N Engl J Med 2011; 365:1099.

54) Lowrie EG, Lew NL. Death risk in hemodialysis patients: the predictive value of commonly measured variables and an evaluation of death rate differences between facilities. Am J Kidney Dis 1990; 15:458

# **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 Electrolyt	es :- Na <sup>+</sup> -		K <sup>+</sup> -						
Serum Calcium -		Phosph	10rus -						
Total bilirubin-		Direct-		Indirect-					
Total protein-		Sr. Albur	nin-	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 ( $\sqrt{}$ ) these boxes

o 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.

o 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.

o 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.

o 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.

o I hereby consent to participate in this study.

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

Signature of investigatorSignature/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 0 புரிந்து கொண்டேன் நான் என்பகை உறுதிப்படுத்துகிறேன். கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளது. திருப்திக்கும் எல்லா ഞച്ച (முழு ഞച്ച கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளது.
- ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும், எனது சட்ட உரிமைகள் பாதிக்கப்படாமல், காரணமின்றி எந்த நேரத்திலும் திரும்பப் பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்துகொள்கிறேன்.
- ஆய்வின் ஸ்பான்சர், ் மருத்துவ ஸ்பான்சர் சார்பாக பணிபுரியும் மற்றவர்கள், நெறிமுறைக் குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதார பதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்துகொள்கிறேன், தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள ஆராய்ச்சியையும் எந்தவொரு பொறுத்தவரை இது தொடர்பாக, நான் ஆய்வில் இருந்து ஒப்புக்கொள்கிறேன். விலகினாலும் இந்த அணுகலை எவ்வாறாயினும், சட்டத்தின் கீழ் தேவைப்படாவிட்டால், தரப்பினருக்கு வெளியிடப்பட்ட மூன்றாம் 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

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,

To

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

5 Decimies and a second	Chennai : Member
5. Prof. Meena Suresh, MD., DGO., Prof. of Obst & Gynaec, IC	OG,Chennai : Member
o. 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	·Lawver
9. Thiru K.Ranjith, Ch- 91	· Lov 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.

: Lay Person

# Ouriginal

#### **Document Information**

Analyzed document	A STUDY ON OUTCOMES OF HOSPITALISATION ON PATIENTS ON MAINTENANCE HEMODIALYSIS .docx (D152512763)
Submitted	12/7/2022 4:57:00 PM
Submitted by	L Arun Khumar
Submitter email	arunkhumar1179@gmail.com
Similarity	6%
Analysis address	arunkhumar1179.tnmg@analysis.urkund.com

1

#### Sources included in the report

W	URL: https://www.slideshare.net/deeevardone/chronic-kidney-disease-ckd-nephrology/116 Fetched: 10/31/2021 4:10:02 PM	88	2
SA	<b>1 (1).pdf</b> Document 1 (1).pdf (D31151133)	88	2
SA	<b>Anti PLA.docx</b> Document Anti PLA.docx (D30996432)		15
W	URL: https://olfu.instructure.com/courses/67120/files/7312974/download?download_frd=1 Fetched: 12/23/2021 6:05:29 PM		4
SA	<b>Satpal Thesis 171022.pdf</b> Document Satpal Thesis 171022.pdf (D146638464)	88	1
SA	CONTENT.docx Document CONTENT.docx (D138465871)	88	1
SA	Section 2 - Main Thesis Report - Complete.pdf Document Section 2 - Main Thesis Report - Complete.pdf (D121006596)		1

# 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.

Queden

Guide & Supervisor sign with Seal.

Dr. T. B. Umadevi MD Prof. Of Medicine Reg. No. 55530

# **MASTER CHART**

SNO	Submitted 1 Name 2 Age	3.Gen	ter 4 Nobile 15. Occupati 6. Addres	s 7. Native k 8. Du	iaior9.Dua	for 10 Frec	oue 11. Dans of 12. Shih	tol B. Vacuk 14. Dur	atio 15. Pastao 16. Ser	olog 17. Comort 18. He	nce 19. Bloc	d L 20. Sen	n (21 cila	lai 22. Seru	m 121. Seru	m 24. Seru	m 125. Serur	n 126 Tota	1 P 27. S. A	bu 28.ECG 29.CVR or 90.Echocar31.Dru	eco 32. Flui	lo 31 Diano 34 Resor 35 Oxim 45 Dire of 37 Dire of 88 Duratio 39 Hospits 40 Diano 44 Resor 42 Oxim 43 Dire of 44 Direto 44 Hospits 47, Farv
_	1 71-10-2022 Malarvithi 44	Femal	9905990Hove with Triccell	urNotkoowr5	ţ	3/7	Vinday   2nd	leface 5	Ni Ni	SHILL (197	2	55	9	139	46	79	3)	64	37	Tinesia Normal - MV mild Ves	) NK	Satifis No assue Infeined 2007-11-24/07-11-218 Infeined No assue
	2 31-10-2022 Zohara bar 49	Femal	9677741Ahouse wife Karnadas	aNothowr2	ĺ	217	Vonday   2nd	Left BCF 2	No Arswer Nil	SHTN   CAI8	112	8	8	134	59	84	1	66	41	Nomal Nomal DOVef32 Ves	Yes	Acite oli Noarsier Dischare Noarsier Noarsier 1899 - Acite oli Noarsier Dischare Noarsier Noarsier 1893 - 1893 - 1893 - 1893
	3 31-10-2022 Deenariha AD	Ndla	ARXIGTRA Plint workin Konstan	Nich rech 19	ł	ন্- মা	Montay   2nd	ReltRTE 5	left RCE fa Nil	DM   SHTM10.8	172	111	ţ	116	6	11	11	67	11	Normal Normal News	Vec.	III lin zener Tichtere Vin Jenner Vin Jenner J. 1997 Vin Jenner Vi
	1 (1.11.707) Ramendra 15	Nde 1	RAUTTANInt which have	dilationari	f	41 2/1	Norday   2nd	lefaCE 1	No Answer Nil	SHTN 57	00	85	ĺ	1/4	11	11	a	65	15	luhraten Carlinnes luhef 64 eVes	Nec.	Renzi ef Tinzaner Tichare Vin Januer II. Januer J. 1997 – Janier in Vinzener Tichare Vin Januer Vin Januer V. 1994 – Vin Januer
	5 31-11 2022 Galia - 64	Fand	AUGRASS Grown of Nerkond	alinkeel 11	÷	11 11	Montav   Onl	Ridit RTF 1	Nn Losver Nil	Knothun 75	и К	78	6	10	4	113	5	67	20	Normal Normal FFRI Inh Nor	Vac	Latine TRA (In secure) Technology, Annance (In Leave T) ACCER. Violine on Violence Conference (In Leave T) ACCER. Violine on Violence (In Leave T) ACCER. Violence
	C21.10.2023011 30	Mole	1005.ECNetwelicholi	Net lanua C	÷	47 11	Montav   Del	Gideore c	No Jecuci Nil	CUTN OF	15	60	11	122	11	1	0	70	15	Normal Normal Normal Nac	Ver	paner no na
	1 31 10 1031 Jackers of D	NOC	MUSECHICKUM ADD	Notineur 1	ť	41 10	Notay   210	lufare a	No Lerver Vil	nu icumian	110	0.5	1	10	11	01	11	13	10	Normal Normal Named No.	lis Ver	Nexus in preside using in marchineness. Income in none in and in marchinene in marchinene in marchinene in marchinene in marchinene in the second s
	0.21 10 2022 Ali Urube 3 00	Net	01111011114 webie Vesser	Notineer D	,	41 17	Muluay   210 Mandar   2ni		No forwer/Nil	UNI (31111442 CUTNI 70	10	1) 1)(	1	102	N.	62	24 20	w Cl	-	Normal Normal Normal Nor	lin Ver	in a second second in a second se In second seco
	a or so out the and the	NOC Travel	02220410100.000011000	NULNIUM 2	,	41 10	ייר ארו ארו ארו ארו ארו ארו איז	LEUNUT Z	NUMBREINI	ann 10 ann 10-10	10	110	•	141	49	10	20 17	11	10	Number Number Collisies	10	In A de la valet in A de la Adre in A de la valet in A de la La valet la valet in A de l
	10110200000000000000000000000000000000	rend	<ul> <li>SUPERING NUMBER</li> <li>SUPERING NUMBER</li> </ul>	KINULKIUWI /	-	3// 1/2	NUTURY   210	LELOUT D	LEURO AJNI	yun ulu Ciringa	09	00	0	11	03	13	ι) V	13	30 17	NUTHA NUTHA GIZNOU, NS	10	Appelou invariante lucitatige invariante invariante da la constructiona da la constructiona da la constructiona En 1613 Marcan Radam Martana Martana da la constructiona da la constructiona da la constructiona da la construct
	U SELEZUZ VEMIA ZI	rena M.L	994LDAUBRINGEWORKENDTRUT MUTHATING	(NEIBBOL)		41	DCL VEDIN	Permican U4	LETTACE AJNI	SEQUE DISS	00	19	1	11	44	00	10 - 1	1	10 12	NUTER NUTER NO.	16	Hotoan in advised using the name in a control of the international of the international sector in a size of the
	11 31-11-2022 ABAI(49738 35	186	SUAUSUSZERLOUNEYSEI IINDIIBIG	BINOCKNOWIS	+	41	NODBY   200	KØTBU D	NO ATSWEINI	SHIN TO	1	1.A	9	H) (1)	44	1	)4 17	64	42	Noma Noma Noma NS	16	NOARSIE IN ZISHEI NAAISHEI NAAISHEI NAARSHEI NAARS
	12 SE-10-2022 Alex pandi 25	Male	994077333/Security Palarara	mNocknown2	1	\$ I	Monday   3rd	LETTBUH 15	Lettkü: Mi	CE NIRC	88	13	1	138	46	/.b	40	64	4J	Nomal Nomal Nomal Yes	165	Geotos Ilioanske usoarge Ilioanske No Anske No Anske Ilioanske Ilioanske Usoarge Ilioanske Ilioanske bij Haadah Ilioanske
	13 31-10-2022 Kamaleshv 54	Nale	88.577.890Not workin Valasraki	kaDiab reph 1	1	20	Monday   3rd	Right RCF 1	No ArswerNil	DN   SHINS	95	59	11	137	5	59	34	65	38	Nomal Nomal Nomal Nes	Yes	No Arswe No answer No No Answer No
	14 31-10-2022 Sartha kris 30	Male	637455473:Tea shop i Sivagangi	ai Notknowr 3	1	3/7	Monday   3rd	Lett BCF 1	LeftRCF Nil	SHTN 8.8	89	67	11	125	45	82	66	12	39	Nomal Nomal Nomal Nes	Yes	UT Noarsne Osdage. No Arsner No Arsner 10 RGGEN No Arsne Noarsner No Arsner No Arsne No Arsne No Arsner No
	15 31:10:2022 Amudha 48	Fenal	8531572349Not workir Ambattur	r Notknowr1	1	ų)	Monday   3rd	Right BCF 1	No ArswerNil	DW   SHTN81	84	55	9	137	51	91	75	58	3,4	Nomal Nomal Corc.Lvh, Ves	Yes	No Arone
	16/31-10-2022 Prathu 35	Male	994258355Driver Tondiarp	eiNothowr 11	11	3/7	Monday   3rd	Left BCF 0.5	Right nó an Nil	SHTN   CA(122	84	7.9	8	112	42	82	52	73	12	luh Normal Corclin Ves	Yes	Acceptin No answer Oscharger. No Answer No: Answe
	17 31-10-2022 Raj Kumar 22	Nale	84440/885Not workir Vyeserpe	dilupus nepi 7	2	3/1	Monday   3rd	Left BCF 2	No Arswer Nil	SHTN   Sle 7.7	124	68	10	138	48	1.1	19	7.0	41	No Arswe No Arswe Corchit, elles	No	Gabitis Noarone Dischage NoAroner(NoAroner4 R663H Acute publicoaronerDischage NoAroner(NoArone 3 R663H NoAroner
	1831-10-2022 Gopal 25	Nale	783475389Not workir Gurudip	oNotknown6	6	3/1	Norday   3rd	Right RCF 6	leftRF Nil	SHTN 108	90	85	9	135	48	7.9	ų	78	43	No Arswe No Arswe Severe Lus Yes	Yes	Aute púr Noarsner Discharge: Noarsner Noarsner 3 1966691 Ali Noarsner Discharge: Noarsner Noarsner Noarsner 3 1966694 Noarsner
	19 01-11-2022 Nagarajan 58	Nale	96252978XVot vorkir Thiruali	urNotknown6	ļ	3/1	Tuesday   2nd	leftBCF 5	No Arswer Nil	DM   SHT17.6	115	39	12	134	43	7.9	43	5,4	11	No Arswe No Arswe Dilated Iv, Yes	No	Acte pů Slápády Stech – Na Answella Answella Answella Answella Answella Answella Answella Answella Answella Answe
	20 (0-11-2022 Munirathin 70	Nale	955655594Not workir Red hills	Notknown5	5	2/1	Tuesday   2nd	leftBCF 5	No Arswei HCV	DN   SHT164	135	9	1	126	58	68	50	53	21	No Answe No Answe Severe Lus No	No	Acte på Porfåd Deth – No Anne No Anne 2 – RGGH – No Anne
	21 (01-11-2022 Devabalari 34	Nale	852897899:Security Thirvier	naNotiknown8	6	2/1	Tuesday   2nd	Left RCF 6	No Arswer Nil	SHTN 7.6	152	85	8	134	48	7,6	50	62	35	No Arswe No Arswe Corchit Mes	Yes	Acté her Notsze Dezh No Anseello Anseell RGGBH No Ansee No Anseello Anseello Anseello Anseello Anseello Anseello Ansee
	22 (01-11-2022 Rahamathi 33	Nale	81648942)Not workin Kancheep	p.Notknown6	6	3/1	Nonday   3rd	Right BCF 5	No Arswer Nil	SHTN 6.7	152	68	9	136	51	82	18	61	32	No Arswe No Arswe No Arswer Yes	No	Acet pub Stip datas Cestin No Answer No Answer 1 RECEN No Answer No
	23 (0:11:2022 Yesu 46	Nale	8023659889Not workin Chernai	Notknown2	2	2/7	Tuesday   2nd	Left BCF 2	No Arsver Nil	DW   SHTN 10.6	129	13	9	133	61	92	59	7.6	41	No Arswe No Arswe Normal - Yes	Yes	No Arsue
	24 (0-11-2022 Munusam) 31	Nale	85800349 Not workir Aradhi	Notknown 2	2	2/1	Tuesday   2nd	Right RCF 2	No Arswei Nil	SHTN 83	91	105	1	10	44	7.9	5]	6	38	No Arswe No Arswe Gobal hyp Yes	Yes	Acelerate No Anne Discharge No Anner No Anne 2 193334 Acute pult Prov Fluid Discharge No Anner No Anne 2 193334 No Anner
	25 (01-11-2022 Komethi 31	Fenal	98789280 Not workin Ranipet	No Answei 4	2	2/1	Tuesday   2nd	Left RCF 2	No Arswer Nil	SHTN   Hyd72	5	66	11	134	52	87	39	64	40	No Arswe No Arswe Normal 145	Yes	No Arswel No
	26 01-11-2022 Perumal 60	Nale	8528998569Not workin Nerkundi	aNotinowr3	3	3/7	Tuesday   2nd	Left RCF 3	No Arswer Nil	SHTN   CAUR7	58	64	13	137	40	11	36	69	36	No Arswe No Arswe Gobal hro Yes	Yes	Arazara Poorfuid Discharee No AnswerNo AnswerA RGGGH No AnswerNo
	27 (0-11-2022 Ravichands 47	Nale	9500790427Not workir Thirusall	urNotknowr3	3	217	Tuesdav   2nd	Reht BCF 1	LeftBCF Nil	SHTN   old 10.1	1%	69	10	139	44	82	19	80	40	No Arswe No Arswe Corclin, r Yes	Yes	No Assue
	78 (11-11-2022 Selva Kum: 27	Nale	FROTASTNot workin Mandaue	lilel rechtifi	í.	3/1	Tuesday   2nd	Refrace 3	leftRCF Nil	970 69	80	119	8	137	46	8	"	13	36	No Arswe No Arswe Corc. Ivb. (Ves	NR.	linzanta latore. Dictaze Na Assaella Assae
	74/01-11-2022Swetha 21	Femal	ASTORSION Internetic Trinican	e Notionar)	,	11	Tiestay   Ind	leface (	No Answer Nil	SHTN 119	 Ø	90	1	179	13	11	51	64	1)	No Arsue No Arsue Normal Nes	Nec.	Lante nie Parefinit Ticheze. Na Lanae Na
	3) (01-11-2022 Thanzama 73	Nole	AND	niel renhr 7	-	41 2/1	Tiestay   2nd	leface 6	No Answer Nil	9111 41	119	71	1	1/1	ي 11	80	1	79	u	No Arsue No Arsue Normal Nes	Nec.	No brave
	21 (11-11-2022) Kanimerki 22	Fand	GATAGER With writin Londina	cVirthowr7	;	11 11	Tuesday   2nd	lefate s	leftRCF Mil	SHU 1487	 K	68	11	135	50	61	u.	60	20	In Jose In Jose Antal In Vis	Vac	interniciintore Tichere Viningerile Jean-J. 2007. Un Jean-Viningerile Jean-Viningerile Jean-Viningerile Jean-Vin
	20 (11.11.000) Marilani 20	Fond	<ul> <li>GOTELETER WORK FORGER</li> <li>GOTELETER WORK FORGER</li> </ul>	iu Drimory hy 11	÷	या भा	Tiector   Ind	LLING 5	No Jecupi Nil	uni Giri	10	28	1)	100	50	16	R.	20	1)	No brown Wo brown Core Lin - Noc	Vac	para particular, balangi, annancin manci in manci in mana chunancin manci annanci annanci annanci annanci annan Tritamiz liri raz. Ticrizza (la Izaazila Izaazili) 2002) (la Izaazilia Izaazilia Izaazilia Izaazilia Izaazilia Izaazilia Izaazilia
	20 (N 11 0001Guelesi ) (7	Cord	DISIONETANI	Nisharah 1)	÷	41 217	Tunchu   Jad		Increased in the second		0	10	11	10	17	00	17	10	22	No Lorus No Lorus Colei kue Ver	Ner In	In term to be the second se
	20101112022001081 41	ICID Mela	COMPLETATION AND A STATE	National D	+	2/1 2/1	Tuesday   210	LEILOUT D	LEILING INI Na Jerum UCV	011 111 110	00 00	70		100	10	11	u u	72	20 1	IN A CHICH WA CHIC COULD HIP ICS	lt) Ver	in a sector sector in a sector a sector a sector in a sector in a sector a sector a sector a sector a sector a Un termita termi
	37 (0) 49 10010kurseen 17	NOC Travel		Incorporate de la constante de	÷	9/ 17	Tranks   210	LEUNUT D	NUMBREITUT	000 00	W D	10	) 11	10	10	70	19	12	10	In terre lite terre Critical alter	10	in a die in van ein van ein van ein van ein van ein van ein na die in van ein va
	20 (01-11-20/2/01/01/01/2012) 20 (01-44-20/2/2012)	relidi Tami	20202000000000000000000000000000000000	uyus ieyi o Nationard	-	9// 1/7	Tranks   20	LEUNUT 0	NUHISNEITU Na Jacuari IIC/	0.000	Я	93 01	1/	111	9 17	70	20	70	11	NUHENCHUHENCULINU, CE	10	UU, RUU WADIE USUdeen WADIE IN ANDREI IN ADDREI IN ADDREI USUdeen WADIE IN ADDREI WADIE IN ADDREI ADDREI ADDREI In fahr fahr Wadie usudeen Wadie In andrei II. In andrei Verlagen Wadie II. In andrei Verlagen Wadie II. In andr
	20 01-11-2022 (BTINSEN) 10	rend		NULKIOWI 4	-	41	Tuesday   200		NUHISNEITLY	0111 04	10 N	64	3	13	40	13	ນ 	1.0	4	NURSHEINURISHEINUTTA TE	10	
	SUDETERNYSOUS R	988	SHESHANDENDER STREET	BINOCKNOWI 4		\$   >17	100503Y   200	LETIBLE 4	NO ATSWEIHLY	SHIN LUA	9	2	y 	B/	20	1.3	43	bð ca	41	NOAISNE NOAISNE NOTTAL 195	16	ACCERENTICESIE IN DATSHEIN DATSHEIN ANSHEI / HCCCF IN DATSHEIN DATSHE
	3K (0-11-2022 KB) 55	Male	894625946:Not workin Veliote	Adoko /		41	luesday   2nd	Kightpern 5	MutplealHLV	NO ATSWEI'S	j]	4	1	B	38	18	11	61	3	NO ATSWEI NO ATSWEI LOTCHIN, gifts	16	VOLNE O POSTILIO USDažge. No Alsine No Alsine IX. Alsine No
	41 (1-11-2022) Narendra 1-34	Male	9/16/5/3/Not worker Chrompe	t (NSADad 5	3	4	luesday   3rd	RgtHU-5	No Arswei Nil	SHIN 15	35	112	9	3/	45	80	41	/1	4	No Arswe No Arswe Normal 1985	165	No Arswe
	40(01-11-2022Karthik 40	Male	9566091322Carperter Perambu	r Nephrotic 4	1	3/1	Tuesday   3rd	Letrof 4	No ArswerNil	SHIN 12.1	88	91	10	136	47	7.9	11	6.6	41	No Answe No Answe Conc. Lvh, vies	Yes	Aade M. No Arowe Oscharge. No Arower No Arower 6. 196559. Acute puir Provindui Obscharge. No Arower No Arower 3. 196559. No Arower
	41 01-11-2022 Nagarmal 47	Fenal	e 99411091 Notworkir Thandian	peNotknown8	1	3/7	Tuesday   3rd	Right BCF 6	leftBCF Nil	SHTN 88	113	11	10	138	68	84	67	68	41	No Arswe No Arswe Normal Yes	Yes	No Arswe
	42 (01-11-2022 Arun 50	Nale	9994979614Dailylabou Ayanavar	alfenaliston 5	5	2/1	Tuesday   3rd	Right RCF 5	No Arswer Nil	DM   SHT192	58	83	10	137	38	85	19	69	45	No Arswe No Arswe Lusdef 44, Yes	Yes	No Arswe
	49 (01-11-2022 Rajakumar 65	Fenal	7010735463Not workir Triplican	e Pyeloneph3	1	ųt	Tuesday   3rd	leftBCF 1	No ArswerNil	SHTN 84	84	63	10	135	54	7,4	30	65	12	No Arswe No Arswe Corc. Lvh., Yes	Yes	Aceteputrilo Anove Discharge No Anoverilo Anoverilo RGGGA Catheters No Anove Discharge No Anoverilo Anove 9 RGGGA No Anove
	44 (01-11-2022 Nandha ku 21	Nale	9092797144Bcom 1st y Kodungai	iyNotknowr4	ł	3/1	Tuesday   3rd	Left RCF 4	No <i>Ar</i> sverNil	SHTN 9.9	107	94	9	133	41	94	36	69	Q	No Arswe No Arswe Gobal hyp Yes	Yes	Geotis, ulio Anone Distarge. No Anone No Anone 3. ROGON. No Anone No
	45 (01-11-2022 Saravanan 46	Nale	733865865/Electrician Myrnaga	r Uretericca 1	1	2/1	Tuesday   3rd	leftRCF 1	No Arswer Nil	SHTN 65	90	96	10	136	61	7.6	Q.	65	41	No Arswe No Arswe Corc. Lvh, Hes	Yes	Acte pir hor fluid Dischage. Vio Answellio
	46 (0-11-2022 Anala 31	Fenal	962291819Not workir Choolai	Notknowr3	3	2/1	Tuesday   2nd	Mulóplea.4	No ArswerHCV	SHTN   CAI99	93	44	12	133	6	7.5	51	79	35	No Arswe No Arswe Severe Lus Yes	Yes	Gridgen No Anave Dischage No Anave No
	47 (07-11-2022 Lakshmana 45	Male	9551885910Not workir Nungand	taNotkown8	ł	3/1	Monday   1st	Left RCF 8	No <i>Ar</i> sverNil	SHTN 132	12)	84	9	15	51	64	32	1	13	No Arswe No Arswe Corc LVH , Ves	Yes	No Assue
	48(07-11-2022/Kishore 27	Male	950067149Not workir Thirustr	iyDiab neph 6	i	3/1	Monday   1st	Left RCF 6	No Arswer Nil	DW   SHT102	85	98	9	128	47	7.3	12	12	ų	No Arswe No Arswe Corc LVH, vies	Yes	No Assie
	49 (07-11-2022 Rajeshwari 45	Fenal	9940741069Notworkin Anadi	Notknown8	í	2/7	Monday   1st	Left BCF 3	leftRCF Nil	CAD   dld F7.3	90	18	10	134	49	7.3	46	7.1	3.7	No Arswe No Arswe Gobal hyp Yes	Yes	Fernariz Na Aswe Disbage. Na Aswe

	90 07-11-2022 Ayesta 54	Fer	nale (86	456668NetworkinPerambur Notknown4	3	2/1	Norday   1	st left RG	3	No <i>Ans</i> ve Nil	DW   947785	136	69	12	16	45	1	5	52	Ļ	No Answei No Answei Global hyp Yes	No	Acteput Porfluid Dictorge Nakone Nakone 5 RGGH Urenicen Notsne Dictorge Nakone Nakone 5 RGGH Nakone
	51 07-11-2022 Rajah 173	Na	le 19	41824ENot workin Purasivalik Not known?	3	2 1	Norday   1	st left (C	3	No Ansve Ni	DV   94777.9	70	86	1)	141	46	1	32	12	39	Na Ansvei Na Ansvei Carc. Uni v Tes	Yes	Naksie
	52 (0+11-2022 Stirtinasan 52	Na	le  97	88001415Auto drive Pallavaran Not known?	1	2/1	Norday   1	st left BCP	2	leftRF Ni	SHTN B	102	103	8	19	55	82	55	66	Ļ	No Ansviei No Ansviei Concl.VH (* 115	Yes	Na Arane Na
	5107-11-2021 Karitha - 56	Fen	nale 19	4041557Net workin Peorebarn Lupus nepl <sup>1</sup> 3	3	3/1	Norday   1	st left BCP	3	No Arsve Ni	DM   SE 7.2	82	68	11	16	48	84	44	61	38	Na Arsie Na Arsie Gobal hy Yes	Yes	lidureo Victore Dictorge Nakone Nakone 3 — RGGAL Nakone Nakone Nakone Nakone Nakone Nakone Nakone Nakone Nakone
	5407-11-2022Lalshni 4	Fen	nale 19	409425NetworkirThirumulaNetknowr10	6	3/1	Norday   1	st left RO	6	No Ansve Nil	SHTN   CN7.9	62	36	12	137	46	19	35	11	41	Na Arsie Na Arsie Gobal hy Yes	Yes	Anteputritature Doctarge Navione Navione IS — RGGBH - Navione Navione Navione Navione Navione Navione Navione Na
	55107112022Sjedali ( <sup>3</sup> 4	Na	e W	894783NetworkirThirovetriyNetknowr 15	11	2/1	Norday   1	st left RG	11	No Ansve IVI	SHTN 6	94	101	}	145	53	18	54	59	36	No Answei No Answei Conc LVH ( Yes	Yes	Uenicge Votsve NoAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAswelloAsw
	55 (P 11 2022 Aron Kuma33	Na	le 90	REERRENot workin Pulianthop Not known 1	1	2/1	Norday   1	st left RG	ł	No Ansve Ni	SHTN 7.8	94	153	ł	N	6	87	84	5]	36	No Answei No Answei Conc LVH (His	Yes	Accelerate Northere Northere Northere Northere Parameter Northere
	57 (0-11-2022 Thangson 45	Na	le 68	RAGSENotworkinPalaaran 2	2	2/1	Tuesday   1	st left RO	1	No Ansve Nil	SHTN 9.4	82	83	9	14	4	83	3	62	Ļ	No Answei No Answei Global hup Yes	Yes	katepárliotsve Distage NoAssiel 10 Assiel 2 - REGET NoAssiel NoAssiel NoAssiel NoAssiel NoAssiel NoAssiel NoAssiel
	98 07-11-2022 Arun — 98	Na	le 🕅	535640Vet workin Reyacuran Net known?	6	3/7	Norday   1	st left80	5	leftRF HCV	SHIN 61	Q	61	1)	134	50	11	42	61	25	No Answei No Answei Conc LVH ("Its	Yes	Herizary No Arave Discharge No Arave No Arave 10 RCCCH No Arave No Arave No Arave No Arave No Arave No Arave No Arave No Arave No Arav
	99 07-11-2022 Dhashan <sup>1</sup> 13	Na	e 19	4563451Net workin Thiowarm Net known?	1	2/1	Norday   12	st left BCF	1	NoArsveHCV	No Answei 95	122	81	1)	14)	56	11	43	59	33	No Ansie No Ansie Norral - 165	Yes	No Assie 110 Assie
	60 07-11-2022 Vetrise Inar/28	Na	e 10	669959Notworkir Thania ur Notworkir 4	ł	3/7	Norday   2	nd Left RG	I	NoArsveHCV	SHTN 62	114	81	9	134	50	81	45	62	32	No Answe No AnswerConcl.VH . Yes	Yes	To Assie
	61 07-11-3022 Sathra 40	Fer	nale 19	966/551VetworkirThinethani Netkoown7	6	2/1	Norday   1	st left BCP	5	leftRG HCV	SHTN 61	104	53	9	15	41	81	44	68	40	No Answer No Answer Conc LIVH # Ves	Yes	No Assie
	67 (0-11-007)Yan 18	Ma	e 97	GG4660Networkin/holineausNetknown <sup>1</sup> 2	17	3/7	Norday   1	st left RG	1	NoArsieHCV	SHTN   dd61	105	8)	1	15	45	11	11	 SJ	36	No Answer No Answer Conc 11H e Ves	Ves	kate fely lintene. Tactare lin kane lin kane 10. Anne 10. Anne lin kane lin kane lin kane lin kane lin kane lin
	8 (7-11-107) (himson 4)	Ma	e qu	55094FNetworkin Knuthats Not knowr 3	)	3/1	Norday   1	st left RCP	;	NoArsie HV	970 (dd 83	101	68	10	16	51	83	50	4	76	No Answer No Answer Conc 11 H e Ves	les.	Premori Notore Notore Natione Natione V. ASSER Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/Natione/N
-	64 (17-11-107) Vacariha kuG	i Na	6 N	erren GERRANstundiskali Notlemurk	1	11	Norday   1	d left(7	6	No Ansver Nil	DV I SHINKE	 %	56	1)	18	5)	81	46	 68	29	lo Ansie lo Ansie (mr.1187)es	Vec.	fattiis. Virture Victore Victore Victore I Arcent Arcent Victore Victore Victore Victore Victore Victore Victore
-	A (1.11.100) Infia (im SL		rale No	n Answerlin Answerlin Answerlin Eishetirne F	i	17	Monday   1	d left (F	6	No Anguje Nil	nv i simpi	00	68	ì	111	46	41	51	#*	0	lin Answer lin Answer Girka hur Ves	Vec.	taria ni Parfini Notze Intene Intene 5 RTTH Untene Intene Intene Intene Intene Intene Intene Intene Intene
-	66 (12.11.100) Romani - 33	Fan	nale 70	USUSARIIn Unsuerlin Unsuerlin Unsuerk	6	117	Montay   1	d leftig	6	No Loove Nil	ALL IN THE REAL	100	61	Í	10	46	41	11	44 65	10	laterelisterelierd is	Vec	nan ya na mana ya na mana ya na
-	A 10.10.000 bilmers A	Fan	nala No	n Lecuardin Lecuardin Lecuardin Lecuard	1	47 2/1	Mentar   1	e Geira	, ,	No brova Ni	UN COLLEG	100	67	í	10	56	81	11 [[	ω Ω	10	Na Leona Na Leona Grégi har Vec	Vac	laine a Daviai Anala anna anna anna anna anna anna ann
-	R In 11 2021 Honora	10	la No	n lecuerlin Incuerlin Jecuerlin Incuerli	2	1/7	Meetau   1	a ingitua et lations	1	No Irova Ni	מחווק אט מחו גם	102	ці ((	11	11	.u	01	и (1	Ω Ω	0	National Interventional Vice	Ver	The set of the second provided in the set of
-	00 01 11 2022 Notified 140	lid Day	ec IIU eda Na	n leven lin leven lin leven lin leven (	3	41 117	Muluay   1 Manton   1	a lakoro	1	Nu Hono Mi	כס הוונ זירודט אח	101	1J [1	1	100	μ D	0.) 70	11	U0 CC	12	NUMBRICHUMBRICHUMB	lt) Wr	numere numere Internet Gardie Yorizza de Internet ( 2000). De terrette terrette terrette terrette terrette terrette terrette
-	10 01 11 10 10 10 10 10 10 10 10 10 10 1	. ICI 114	lac ilu In ila	n locurillo locurillo locurillo locuri)	) )	41 117	Muluay   1 Manton   1	a renon	1	Nu Hono Mi	באוווגן אינ ניתו גע	1/0	U1	2	1)(	01	01	10	10 []	10	NUMBIC NUMBIC QUALITY IS	lt) Wr	Ruccyu Sylaspisologi nivnon nivnon v nivon nivnon nivnon nivnon nivnon nivnon nivnon nivnon nivnon nivnon nivnon Telena listemalistemalistemalistemalistemalistemalistemalistemalistemalistemalistemalistemalistemalistemalistem
-	10 UF112U2008809 41	. Nd	nt IV anta Ita	n henrelin henrelin henrelin henrelin 1 henrelin henrelin henreliner erstil	) )	41 117	Nuluay   2 Needer   3	a iteruo A	1	NUMBRE NI	LQ NINC	100	11	n	100	40	01	40	12 [1	1) 11	NUMBRE NUMBRE NUMBRE No.	IC) Ver	Turkais whole whol
-	71 07 117 117 117 117 117 117 117 117 11	rei La	nde nu mela na	o herrer Nie herrer Nie herrer Nie herrer 7	) 7	41 117	Nuluay   2 Needer   3	a iteruo A	) (	NUMBRICINI Na Jeografii	NUHIDIIELO NU LOIDINI	112	43 U	11	10	4/ 10	11	е) И	12 (1	10	NATION NATION AND A STATEMENT	IC) Ver	To Able to Able The Able to Able
-	00001300jA2202212023	rei Fai	ide nu 	n hanei nu hisinei nu hisinei nu hisinei /		41	Noticay   1 Notica   1	a Lann	0 r	NUHISIKI NI	ענותנן אין אין הייק	30 401	24 14	11	10	23	11 111	41	12 [1	90 17	NATIONE NATIONE WILLIA IN	10	Na Asie Naise Wakie Nakie Wakie Wa
	CONSTANT AND A CONSTA	i iti M	TBE NU	) ANNIELNO ANNIELNO ANNIELNOUELLINE	ð ,	41	Norday   2	si letno 4 latarr	0 r	NDADNENI Na lava ki	נא וחנן אע ניא וחט	11/2	24	1	10	UC 10	LLI nr	4/	ររ (1	20	No Alexandra Levandra Marcada - Marc	185	AULE JULINUSZE U SZARE NARONE NARONE 14. KOSZT TO ADMENIATORE NARONE NARONE NARONE NARONE NARONE NARONE NARONE V koszt 14.
-	AU-LLULKOTED D	18	e no	DANSWEIND ANSWEIND ANSWEI IBA NEDITIED	)	41	NOODAY   1	ST LETHU	)	NOARSVEINI	or an	9/	40 17	lj n	19	48	ររ 	14 11	0 11	59 20	NDAISHENDAISHEINDITTä HS	1ES	NCASIE INAISIE INAISIE Inaisie inaisie
	IS UPIDAUZIYANDIG ISI	. Hen	nale No	DARSWEINO ARSWEINO ARSWEILUGUS REPT8	1	41	Morday   12	st Letku	4	NOArsve NI	NE 1.5	8	4)	ß	18	4/	92 A	4/	រវ 	19	Noarskei Noarskei Norra i les	165	NO ASIRE
	NU-11-ULLNUTB) 49	Hen .	nale No	) ANSWER IND ANSWER IND ANSWER NOT KNOWN IS	8	41	Norday   12	st letku		NO ANSWEI NI	DM (SHINES	9/	11	0	lù 	4/	91 	19 	61 	48	No Arsivei No Arsivei Corc LVH ( les	185	NO ASNE ID ASNE
	// 0+11-2022/Safth 52	. Hen	nale No	DARSWERING ARSWERING ARSWERINGT KROWT 11	9	41	Norday   2	st Letiko Letiko	6	lettili ini	DM (SHIV)9	103	1	0	18	42	3 	42	У.	31	No Answei No Answei Global hyp Hes	165	Acte pur lotsve. Dostage. No Arsne No Arsne V. Arsne V. Arsne No Arsne V.
	78 07-11-2022 Astolan 62	Na	le No	o Answerliko Answerliko Answerliko Answer12	11	2/1	Norday   12	st LetRO	1	No Arsve Ni	DV   SHN7.1	103	53	1)	136	46	93	45	65	41	No Arswei No Arswei Godal hyp Yes	No	Vencen Portud Osstage No Arsne No Arsne 7 - 196591 - No Arsne No
	79 (7-11-2022 Naveen Ku 41	Na	le No	o Answerllio Answerllio Answerllio Answer5	5	2/1	Norday   12	st letRG	5	NoAnsvelNi	SHIN 102	96	51	1)	16	46	91	42	65	39	No Ansire No Ansire Normal 195	Yes	Na Asne Na Asne Na Asne Na Asne
	80 07-11-2022 Uchaya mc53	Na	le No	o AnswerNo AnswerNo AnswerDiabeticne8	5	2/1	Norday   1	st leftRO	5	No Ansve Ni	DM   SHTV102	95	51	11	134	50	92	48	59	35	No Answei No Answei Conc LVH elles	Yes	Hypelyce Notice – Dicitizer Noticine Noticine) 3 – RGGEH – Noticine Notici
	81 07-11-2022 Anitha - 38	Fen	nale No	o Answerliko Answerliko Answerliko Answer5	5	2 1	Tuesday   1	st leftRO	4	No <i>kra</i> ve Ni	SAN Hyst	98	49	1)	19	46	92	46	64	41	No Answei No Answei Normal - Yes	Yes	Na Arsne Na
	82.07-11-2022.Kalyaram 72	Na	le No	o Answerllio Answerllio Answerllio Answerl	8	2/1	Tuesday   1	st leftRG	8	No <i>Ans</i> ve Ni	DM   9117.5	102	51	1)	19	41	101	54	51	29	No Answei No Answei Global hyp Yes	Yes	UN lotsve Distarge Nakove lokove 12. RSSBI. Nakove
	8 (7:11:002 Rangarajar 59	Na	le No	o Answerliko Answerliko Answerliko Answerß	6	2/1	Norday   1	st left RO	6	No <i>krave</i> Ni	DM   SHT19.8	95	[]	1)	139	46	85	42	65	42	No Ansire No Ansire Conc LVH e Tes	Yes	Naksie laksie
	84 07-11-2022 Neelakand 75	Na	le No	o Answerliko Answerliko Answerliko Answeri?	1	2/1	Tuesday   1	st left RO	6	No <i>krave</i> Ni	DW   \$47100.1	102	5,4	}	19	46	91	43	65	40	No Arsie No Arsie Goba hy Yes	Yes	Acteput Notice: Dictage: Notice Notice: 3 RCCCH. Notice:Notice:Notice:Notice:Notice:Notice:Notice:Notice:Notice
	85 07-11-2022 Rajalakstm38	Fen	rale No	) Answer No Answer No Answer No Answer A	4	2 1	Tuesday   1	st left RG	ł	No <i>Ans</i> ve Ni	SHTN 105	112	53	}	18	45	93	46	65	40	No Arsve No Arsve Norral 145	Yes	Na Arsne Na
	85 07-11-2022 Karitha - 38	Fer	nale No	o Arswerllio Arswerllio Arswerllio Arswer3	}	2/1	Tuesday   1	st left RG	}	No Ansve Ni	SHTN 112	98	45	11	135	46	93	41	62	4O	No Ansver No Ansver Normal - 145	Yes	Naksie Kaksie
	87 07-11-0021 Sanachara 45	Na	le No	o Answerllio Answerllio Answerllio Answerli	6	3/1	Tuesday   1	st left BCP	6	No <i>kr</i> sve Ni	DM   SHTV103	105	46	11	16	46	93	45	62	32	No Answei No Answei Conc L'IH e No	Yes	kate CVA Porching Dezith . Na Arsne Na Arsne 10 Arsne
	88 07-11-2022 Rampa - 27	Fer	nale No	o Answer No Answer No Answer Lupus nepi 7	5	2 1	Tuesday   1	st left RG	5	No <i>Arsve</i> Ni	SE 67	89	43	1)	15	43	92	41	52	38	Nakisie Nakisie Norral I II:s	Yes	Na Asne
	89 07-11-2022 Murugesar55	Na	le No	) Answer No Answer No Answer Not known?	1	3/1	Tuesday   1	st leftRG	1	No Ansve IVI	DW   947795	99	51	1)	16	46	93	45	59	38	Nakisie Nakisie Gota hip Yes	Yes	Hypgiya Notice Dictorge Notione Notione3 RGGBH Notione No
	90 07-11-2022 Tarrikehi 42	Fer	nale No	o Answer No Answer No Answer No Answer G	6	2/1	Tuesday   1	st leftRG	6	No Ansve IVI	911) hp1	106	41	11	N	38	92	41	65	40	No Ansie No Ansie Norral - Yes	Yes	No Assie
	91 07-11-2022 Dowlath be 76	Fen	rale No	) Answer No Answer No Answer No Answer8	8	1/1	Tuesday   1	st left80	8	No Ansve Ni	DM   SHINB3	104	52	11	143	50	93	43	9	38	No Answei No Answei Global hyp Yes	Yes	kate pur Nature Distage Nakane Nakane 3 AGGA Nakane Nakane Nakane Nakane Nakane Nakane Nakane Nakane Nakane Nak
	92 (7+11-2022 Mari 66	Na	le No	) Answerliko Answerliko Answerlikot known 10	1)	2/1	Tuesday   1	st left BCF	8	No <i>Arsve</i> Ni	DW   94769	102	58	}	143	48	86	46	58	38	No Answei No Answei Global hyp Yes	Yes	lidure o Vokove Distarje. Nakove Vokove 5
	93 (7-11-1022 Paramasine 13	Na	le No	o Answerllio Answerllio Answerlliot known 8	8	2/1	wed   Satu 1	st Right R	FS	No <i>Arsve</i> Ni	DW   97791	102	49	12	10	48	86	39	58	39	No Arsvei No Arsvei Global hyp Yes	Yes	Na Assie
	94 (7-11-2022 Rabu — 62	Na	le No	o Answer No Answer No Answer Not known 4	4	2/1	wed   Situ 1	st left BCP	4	No Ansve Nil	SHTN   CA191	101	1)	12	146	45	79	41	53	38	Na Arsiei Na Arsiei Global hyp Yes	Yes	Naksie
	95 07-11-2022 Mohamme 42	Na	le No	o Answer No Answer No Answer Not known 6	6	2/1	wed   Satu 1	st Right Bl	FA	No Ansve Ni	SHTN 93	95	49	13	10	48	83	39	56	30	No Ansirei No Ansirei Concli/H ( 165	Yes	Nakase
	95 (0-11-2022 Venkatesh 42	Na	le No	o Answer Nio Answer Nio Answer Nio Answer 3	}	2 1	ved   Satu 1	st left80	3	No <i>kre</i> ve Ni	SHTN DO3	96	51	12	10	35	83	<b>[</b> ]	59	31	NoArsie NoArsie Norral 14s	Yes	Nakase
	97 07-11-2022 Alamelum;62	Fer	nale No	o Answerliko Answerliko Answerliko Answer5	5	2/1	wed   Satu 1	st leftRCP	1	No Ansve Ni	SHTN   Hig10.2	108	53	1)	18	46	86	43	58	32	No Answei No Answei Global hyp Yes	Yes	Premoti listare Distage Nokove
	98 07-11-2022 Rangaiyan 51	Na	le No	o Answerlio Answerlio Answerliot intown5	5	2/1	ved   Satu 1	st left80	5	No Ansve Ni	SHTN   0095	104	52	11	145	48	92	<b>[</b> ]	5]	31	No Answei No Answei Conc LVH e Ves	Yes	No Arsie
	99 07-11-2022 Ismail sher 44	Na	le No	o Answerllio Answerllio Answerllio Answerläs	35	2/1	ved   Satu 1	st leftBCF	35	No Ansve Nil	SHTN 10.2	101	52	1)	18	46	92	45	58	36	No Answei No Answei Normal - Ves	١ <u></u>	PERALE Notice: Dictory: Notice: Notice: Notice: S RCCCH. Notice: Notice
			11	and the second sec	122	14			1.1			1.11	1	11	1.1	1.1	1	1.1	1.1	1.1			

1011723001 3	Female	lohsielohsielohsielohsielohsie	1	1	ned (stolist	letnö 2	loksvelli	STN   M111	11	3	ł	1	[]	92	Į)	9	19	lo Arana lo Arane Corci, Helias	6	lakae laka
1104/20ath 5	Nate	lohsielohsielohsielohsielohsie	6	1	linty 13	letiti 5	lokoreHBkg	SAN 105	9	18	1	18	51	92	(1	3	19	lo Arana lo Arana Corci Helts	6	ktéárikkaelősze kkaelakaes – 1939 kaselakaelakaelakaelakaelakaelakaelakae
1001100 km prô	Vale	lohsielohsielohsielohsielohsie	8	1	lody 19	let OF	lo <i>ks</i> ieliisię	91 B	13	3	ļ	115	19	96	lj	5	1	llo Arane llo Arane Gobalhip Ves	6	kiepintaselisise laaelakse5 – 1639 lakselakselakselakselakselakselakselakse
1804.02 Mil 9	Nate	lohorelohorelohorelohorelohore	IJ	1	linty 13	letni s	loksvelikkę	91 MR	11	1	1)	18	51	96	Į)	]	1	lo Araie lo Araielloma 🛛 Ies	6	lakae laka
11011201kippa1	Vale	lohsieloksieloksieloksieloksie	8	11	lodaj 13	let OF 5	lokoveliBkg	911 (9	1	1	1)	1	51	95	<u>}</u>	49	18	lo Araie lo Araie Gotalhp Ves	6	lebekitse Tidag lokaelakael – 1669 lokaelakaelakaelakaelakaelakaelakae
150-1725 korv (	Vale	lohsieloksieloksieloksieloksie	}	11	lodaj 13	let OF 2	lokovelikkę	lo <i>h</i> sieß1	5	Ŋ	1	1	33	82	3	48	)6	lo Asiello Asiellona (Is	6	lotaelotaelotaelotaelotaelotaelotaelotae
167472kią 9	Female	lohsielohsielohsielohsielohsie	1	1	linty 13	let 10	lokoreHBkg	Hpathyo 86	10	3	ł	18	Ŋ	86	3	58	19	lo Araie lo Araielloma 🛛 Ies	6	lakae laka
110-110214997788	Vale	lohsieloksieloksieloksieloksie	1	1	isti 11	let OF 6	lokovelikkę	STI (ARI	16	Į	1	1	39	85	(1	5]	1	lo Araie lo Araie Gotalhp Ves	6	Azərə Partid Tidəşə İlahərellakane) — 1939 - İlahərellakanellakanellakanellakanellakanellakane
100402161 0	Nate	lohorellohorellohorellohorellohore	5	1	iestų lit	let KF 5	loksvelikkę	SM 55	9	18	1	1	36	91	45	49	]j	llo Arane llo Arane Gobalh p Ves	6	Seerelukae Didage Tukae Tukae Tukaae Tukaae Tukae
1904).201km (	Vale	lohsieloksieloksieloksielohsie	1	11	ista 19	let NF 6	lokovelikkę	d#B 12	10	Į	1	1	1)	91	Ą	51	1	lo Asie lo Asie llona 🗄 Es	6	lotaelotaelotaelotaelotaelotaelotaelotae
10117011814	Vale	lohsieloksieloksie lohsie	6	11	isti 11	let OF 6	lokovelikkę	91 11	10	Ŋ	1	18	35	91	<b>(</b> 5	<u>[</u> ]	1	llo Arsne llo Arsne (62) hóbi (165	6	lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae laka
111.71%	Fenale	lohsielohsielohsielohsielohsie		1	nel (stolst	let KF 5	loksvelikkę	di#B H9	1	[]	1)	1	ļ]	90	Ą	S	]j	llo Arane llo Arane Gobalh p Ves	6	keen lakaelidee lakaelakaesi 1669 lakaelakaelakaelakaelakaelakaelakaelaka
110-11-120 Jaeshol	Female	lohsieloksieloksie lohsie	l	11	ved (statist	let 10	lokovelikkę	00 §	11	[]	1	3	IJ	81	12	9	1	llo Arane llo Arane Gobalhip Ves	6	latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae
13091-021 kęctor S	Vale	lohsieloksieloksie lohsie	6	1	ista 19	Right BEF 6	lo <i>kovel</i> (i	911 1)4	10	96	8	1	ļ]	13	5]	68	Į	lo Arane lo Arane Corcil Heles	6	kietalitse Tidag Iokoelokoel - 1669 Tokoelokoelokoelokoelokoelokoelokoelokoe
1.81.00 mas.	Nate	lohsielohsielohsielohsielohsie	}	1	iestų lit	litili 4	loksvelli	91 B	14	11)	1	1	54	9]	18	68	ļ	lo Araie lo Araielloma 🛛 Ies	6	lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae laka
1599-2010are?	Vale	lohsielohsielohsie Hjetes 7	}	11	ista 19	letiti 3	lo <i>kovel</i> (i	911 B	16	1	8	1	5]	58	64	14	l)	lo Asie lo Asie llona 🗄 Es	6	latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae
16091420Sopti 4	Nate	lohorellohorellohore Dizette2	1	1	īssbį (11	letitő 2	lokovelli	DN   \$775	Ŵ	19	1	3	63	84	]ļ	12	ļ	lo Asiello Asielocci Heles	6	ğisis litse Tistey İskaelakaelis — 1669 Takaelakaelakaelakaelakaelakaelakae
1000kj 6	Vale	lohselokselokse Dåreph	ļ	11	ista 19	Right BCF 4	lo <i>kovel</i> (i	DN  STAGA	13	9]	8	3	58	80	}]	61	]]	llo Arane llo Arane Gobalhip Ves	6	latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae
1881-200hadi 9	Nate	lohsieloksieloksie lohsie	ļ	11	ista) (št	let 07 4	No Answer Ni	lohsie 48	"		1)	]	3	64	]]	9	18	lo Asiello Asiellona (Is	6	lokaelokaelokaelokaelokaelokaelokaelokae
1991-Widen S	Nate	lohsielohsielohsie lohsie3	}	17	īedaļ 11	letiti 3	lokovelli	DN   \$7113	N	1	1)	3	59	1	9	66	IJ	lo Asie lo Asie Gdahp As	6	kiepihitse Tidsg lokaelokae3 - 1669 lokaelokaelokaelokaelokaelokaelokaelokae
1181-121ata 5	Nate	lohsielohsielohsielCTééc3	}	1	iestų lit	letni 3	loksvelli	SH 6	9	11	8	18	50	19	6	6]	39	lo Araie lo Araielloma 🛛 Ies	6	lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae lakae laka
1181-112 haqaa (	Nate	lohsieloksieloksie lohsie	}	11	isti (13	letiti 3	No Answer Ni	SA N	1	8	1)	14	IJ	83	]]	62	Ņ	lo Asie lo Asie Gdahp Vis	6	lokaelokaelokaelokaelokaelokaelokaelokae
1191-11Selma	Vale	lohsielohsielohsielohsielohsie3	}	11	ista 19	letitő 3	lo <i>kovel</i> (i	SFN   dd 123	Q	11	8	1	54	86	5]	12	Ņ	lo Asiello Asiellona (Is	W	kiepinkurlid Ditage lakanellakane3        K669   kiepinkurlid Ditage lakanelakane4       K669   kiepinkurlid Ditage lakanelakane4        K669   kiepinkurlid
18041-120stip 8	Female	lohsieloksieloksie lohsie	ļ	1	ista 19	letiti 3	lo <i>kovel</i> (i	91 15	13	1	1)	1	1)	15	50	]]	Į)	llo Arane llo Arane Gobalhip Ves	6	latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae latae
148142Ande 5	Nate	lohselloksielioksie HAU-1	1	1	īstaļ (11	letiti 1	lo <i>h</i> svell/	91 B	g	ļ	1	]	50	13	61	9	1	lo Asiello Asiellona (Is	6	Remilitise Tidag Iokaelakael - 1669 Takaelakaelakaelakaelakaelakaelakae
15041400Add A	Nate	lohsielohsielohsie lohsie3	}	1	īssbį (11	Ryth (F 3	lo <i>h</i> rsveli()	lo <i>losie</i> /1	5	82	ļ	1	1)	11	63	11	Į	lo Asiello Asiellona llis	6	Fabrálitae Dateg Iokaelokaef - 1669 Tokaelokaelokaelokaelokaelokaelokae
1881-120 Sostiu S	Nate	lohsieloksieloksielohsielohsiel	8	1	ista) (št	RytkF 1)	No AnswerHBAg	K971 (1	9	62	ļ	1	ļ	]ļ	9	]	1	lo Asiello AsieGdahp15	6	lokaelokaelokaelokaelokaelokaelokaelokae
1281:120kt/km	Nate	lohsieloksieloksieloksielohsiel	1	11	ista) (št	Right ACF 5	No AnswerHCV	911 N	5	6	ļ	]	50	18	ļ	9	1	lo Asie lo Asie Gdahp Vis	6	kieśdnitse Ditsę lokaelokael – lokaelokaelokaelokaelokaelokaelokaelokae
1881-128tml 8	Nate	lohsiellohsiellohsie lohsie5	Ş	\$1	īssbį (11	letiti 5	lohoveNi	DN   \$1192	1	61	1	3	1)	65	9	1	j)	lokorelokoreĉoci.Hele	б	ktelliktor beti lokaelokael 1689 lokaelokaelokaelokaelokaelokaelokae
1981-128til 5	Nate	lohsieloksieloksie lohsie	1	1	lloday (19	let05 6	lohoveNi	DN   \$FT\$4	1	62	1	3	1	ļļ	56	64	35	lo korello koreGoalhpYs	6	kiepinktse bzil lokaelokael 1669 lokaelokaelokaelokaelokaelokaelokae
31341-00 km (	Vale	lokaelokaelokaelokaelokae	6	11	Testa Dd	Røttelf 5	lo <i>h</i> svel\1	DN  STAGA	10	]	}	3	51	81	64	59	1	lo Asiello AsieGotahptes	8	lerenkise leit (okaelokae) – KKP lokaelokaelokaelokaelokaelokaelokaelokae