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Chronic Kidney Diseases Clinical Profile of People Living In Rural Areas

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HUMAN

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ABSTRACT

Chronic kidney disease is an irreversible condition that progresses relentlessly leading sooner or later to the end stage renal failure. The diagnosis of this stage can be achieved by eliciting the history carefully, discovering co-morbid factors, utilizing imaging techniques, interpreting histological material and placing this in the context of probability derived from epidemiological data. Screening of high-risk individuals- those with hypertension, diabetes mellitus, cardiovascular and other risk factors, lifestyle modification, physical exercise, abstinence from smoking will retard the progression to ESRD. This will help in bringing down the huge burden due to the mismatch between demand and availability of resources for renal replacement therapy in developing countries like India, especially for patients belonging to lower socioeconomic group. The assessment of the clinical profile of these patients showed the most common etiology as diabetes mellitus (36.9%). Hypertension being a cause and a complication of CKD was present in 64.6% of patients. Early detection and effective management of these illnesses can delay the onset, progression of CKD and subsequent morbidity and the requirement of renal replacement therapy, if any. Another manageable condition obstructive uropathy found in 10% of these patients, if treated at an early stage prevents progression to irreversible kidney damage. Cardiovascular disease in chronic kidney disease is more common in the presence of Diabetes mellitus, Hypertension, hypernatremia, increased kidney size, hypo albuminuria in patients with anemia, cardiovascular disease was more common when hemoglobin levels were less than 5g/dl. Cardiovascular diseases as a morbidity were identified in 28.5 % patients. Being the leading cause of mortality in CKD it would be imperative to monitor patients for this morbidity. Cardiac structural, as well as functional abnormalities, are common in patients with ESRD, more so in those with hypertension and anemia. LVH is the commonest cardiac abnormality in ESRD patients, followed by diastolic dysfunction. Both conditions are more marked in hypertensive patients and anemic patients. LVH has got prognostic implications, because this group of ESRD patients have a propensity of diastolic dysfunction or sudden cardiac death.

INTRODUCTION

Kidney disease is an important problem worldwide kidney disease is defined as the health of an individual, which can occur abruptly, and either resolve or become chronic. CKD is a general term for heterogeneous disorders affecting kidney structure and function with variable clinical presentation, in part related to cause, severity and the rate of progression.1,2 The concept of CKD evolved after the recognition of the contribution of disordered kidney structure and function on the health of individuals across a wide range of severity. Earlier stages of kidney disease are often asymptomatic, are detected during the evaluation of comorbid conditions, and may be reversible.[4,5,6]. Rapidly progressive diseases may lead to kidney failure withinmonths but most diseases evolve over decades, and some patients do not progress during many years of follow-up. Data from the US population studies like the Third National Health and Nutrition Examination Survey (NHANES) and the Kidney Early Evaluation Program (KEEP) show that US adult population of 11-15.6% has CKD.[9,11]

Based on data from major tertiary care centers, the presumptive estimates of the incidence of CKD in India are 100 per million population (p.m.p). CKD is emerging as a world public health problem. The World Health Report 2002 and Global Burden of Disease Project reports show that disease of the kidney and urinary tract contributes to the Global Burden of disease – with approximately 8, 50,000 deaths every year and 1,50,10,167 disability-adjusted life years.[2,16,17]

Globally, they represent the 12th cause of death and the 17th causeof disability. Individuals with even the earliest signs of CKD are at increased risk of cardiovascular disease and may die long before they reach End-Stage Renal Disease (ESRD) [20,21]. More and accurately but less poetically, human kidneys serve to convert more than 1700 liters of blood per day into about 1 liter of a highly specialized concentrated fluid called urine. In so doing the kidney excretes the waste products of metabolism, precisely regulates the body's concentration of water and salt, maintains the appropriate acid balance of plasma, and serves as an endocrine organ, secreting such hormones as erythropoietin, rennin, and prostaglandins.[13,14] The physiologic mechanisms that the kidney has evolved to carry out these functions, the kidney a high degree of structural complexity. There are multiple causes of kidney injury that lead to the final common pathway of ESRD, and this syndrome is characterized by hypertension, anemia, renal bone disease, nutritional impairment, neuropathy, impaired qualityof life, and reduced life expectancy. Increasing evidence acquired in the past decades

indicates that the adverse outcomes of

CKD such as renal failure, cardiovascular disease, and premature death can be prevented or delayed by early detection of CKD.[17,19,22] Earlier stages of CKD can be detected through laboratory testing only. Treatment of earlier stages of chronic kidney disease, as well as the initiation of treatment of cardiovascular risk factors at early stages of CKD, should be effective in reducing the rate of progression of CKD to ESRD.[34,35,36]. CKD is classified based on cause, GFR category, and albuminuria category. The cause of CKD is assigned based on the presence or absence of underlying systemic diseases and the location of known or presumed pathologic abnormalities (glomerular, tubule-interstitial, vascular or cystic and congenital diseases).[15,16,17]

GFR category is assigned as G1 (>90ml/min/1.73m^2), G2 (60- 89ml/min/1.73m^2), G3a (45-59ml/min/1.73m^2), G3b (30- 44ml/min/1.73m^2), G4 (15-29ml/min/1.73m^2) and G5 (<15 ml/min/1.73m^2). Albuminuria categories are A1 (<30mg/24hr), A2 (30- 300mg/24hr) and A3 (>300mg/24hr). [18,19,20]

The clinical course is typically a progressive loss of nephron function ultimately leading to end-stage renal disease (ESRD) characterized by hypertension, anemia, renal bone disease, nutritional impairment, neuropathy, impaired quality of life and reduced life expectancy ultimately needing some form of renal replacement therapy. This puts a substantial burden on global health resources since all modalities of treatment are expensive. In a developing country like India, only 3% to 5% of all patients with ESRD get some form of renal replacement therapy [33,34,35.]

STAGES OF KIDNEY DISEASE

Glomerular filtration rate (GFR) is accepted as the best index of overall kidney function in health and disease. Several stages of CKD, defined as structural abnormalities of the kidney that can lead to decreased GFR, are recognized [34,35.]

KIDNEY DAMAGE [38,39]

This stage is defined as the presence of structural or functional abnormalities of the kidney, initially without decreased GFR, which over time can lead to decreased GFR.

Mild reduction in GFR (60 to 89 mL/min/1.73 m^2)/Early CRF [40,41]

At this stage, patients usually have hypertension and may have laboratory abnormalities indicative of dysfunction in other organ systems, but most are asymptomatic. If the serum creatinine level is elevated, it maybe no more than borderline and of equivocal significance.

A moderate reduction in GFR (30 to 59 mL/min/1.73 m^2)/Moderate $_{CRF}$ 43,44

This stage is characterized primarily by the presence of azotemia,defined as the accumulation of the end products of nitrogen metabolism inthe blood and expressed by an elevation in serum creatinine and serum urea nitrogen (SUN) concentrations. Erythropoietin production decreases and laboratory abnormalities reflecting dysfunction in other organ systems are usually present. Although patients may have symptoms, they often remain remarkably asymptomatic even though their kidney function maybe reduced by as much as 70%.Severe reduction in GFR (15 to 29mL/min/1.73 m^2)/Pre-ESRD

In this extremely tenuous stage of CKD, the worsening of azotemia, anemia, and other laboratory abnormalities reflect dysfunction in several organ systems. However, patients usually have mildsymptoms. [24,25]

Kidney failure (GFR, < 15 ml/min/1.73 m^2)/ESRD

In most cases, this level of kidney function is accompanied by a constellation of symptoms and laboratory abnormalities in several organ systems, which are collectively referred to as uremia, initiation of kidney replacement therapy (dialysis or transplantation) is typically required for ltreatment of comorbid conditions or complications of decreased GFR, which would otherwise increase the risk of morbidity and mortality. [32,33]

PATHOPHYSIOLOGY OF CHRONIC RENAL FAILURE [40,45]

Renal disease is often attributed to classic antibody-mediated or cell-mediated immunologic renal injury. However, renal injury complicating such common disorders as diabetes and hypertension has no apparent immunologic basis. Therefore, the pathogenesis of injury in these conditions must occur by way of nontraditional (nonimmune)pathways,

Several nonimmune mechanisms of renal injury have recently been elucidated including alterations in circulating lipids, abnormal systemic and internal hemodynamics and disordered

regulation of endogenous renal cell function[21].

An important clinical observation in patients with nonimmunologic renal disease is the invariable progression to ESRD once the baseline serum creatinine is more than 1.5 to 2.0 mg/dL, even in the absence of the original inciting event.[13,14,15]

This observation has advanced the hypothesis that nephron loss serves to promote further nephron loss, although the mechanisms responsible for this inexorable course remain incompletely understood. It is thought that adaptive changes occur in the remaining functional nephrons and promote progressive renal scarring. Studies in rats show that experimental ablation of renal mass promotes progressive loss of renal function, and the relative reduction in total renal mass correlates with the rate of progressive renal injury. [16,17,18]

Adaptive changes associated with nephron ablation have been the subject of intense investigation over the past decade. Among these "adaptations," changes in intraglomerular hemodynamics have been investigated most intensively[22]. Remnant nephrons undergo marked sustained increase in single nephron plasma flow (hyperperfusion), single nephron glomerular filtration rate (hyperfiltration), and glomerular hydraulic pressure (glomerular hypertension) in response to ablation of renal mass.

Glomerular hypertension, characterized by increased glomerular capillary hydrostatic pressure, appears to be of considerable importance. In fact, agents that attenuate glomerular hydraulic pressure, such as angiotensin-converting enzyme (ACE) inhibitors and protein-restricted diets, can protect against progressive renal scarring[22,23]. These observations form the basis of studies evaluating the clinical effects of sustained use of ACE inhibitors in patients with chronic renal failure. While hemodynamic factors have been extensively studied, the specific cellular and biochemical pathways that link altered glomerular hemodynamics with progressive renal scarring are still poorly understood. Moreover, recent studies (3-5) have suggested that a variety of perhaps complementary mechanisms contribute to progressive renal scarring, including proteinuria and alterations in levels of circulating lipids, hormones, and electrolytes. Collectively, these factors may contribute to progressive renal injury by changing the function of various resident renal cells, such as macrophages and mesangial cells[53].

Two other important concepts in understanding the progression of CRF are the intact-nephron hypothesis and the trade-off hypothesis [42]. The first states that in general, adapted

nephrons behave like normal nephrons. Some of the failure to regulate sodium and water relates to increased solute excretion per nephron-in effect, an osmotic diuresis of the remaining nephrons that impairs sodium and water conservation, especially in states of extracellular fluid volume depletion. Thus renal concentrating ability is lost, as well as the ability of the remaining nephrons to adjust to low and high intakes of sodium, water, potassium, and other dietary solutes because these nephrons are functioning at maximum capacity even with normal intake of thesesubstances.[35,36,37]

PATHOPHYSIOLOGY OF UREMIA [42,43]

Although the pathogenesis of the different uremic symptoms is not completely understood, three major mechanisms are involved: diminished excretion of electrolytes and water reduced excretion oforganic solutes and decreased hormone production[24].

Diminished excretion of electrolytes and water [78,79]

An important function of the healthy kidney is to excrete the electrolytes and water generated from dietary intake in order to maintain a steady state in which intake and urinary excretion are roughly equal. The conditions that cause loss of kidney function induce adaptive mechanisms that attempt to preserve the homeostatic state of electrolyte and water balance. If, for example, three-quarters of nephrons have been lost, then each remaining nephron must excrete four times the amount ofelectrolytes and water to maintain the excretion at the same level of dietary intake [46].

However, all these compensatory mechanisms eventually fail, atwhich stage the continual loss of function results in the kidney's inability tomaintain balance. At this point, patients are said to have end-stage renal disease (ESRD). The number of functioning nephrons at this time is so small that urinary excretion cannot achieve a level equal to intake. Clinical manifestations include edema and hypertension (caused by sodium retention), hyponatremia (resulting from free water retention) hyperkalemia,metabolic acidosis, hyperuricemia, and hyperphosphatemia.[47,48]

Reduced excretion of organic solutes

The kidneys excrete a variety of organic solutes, the most commonly measured ones being urea and creatinine. Unlike the excretion of electrolytes and water, the excretion of urea and creatinineis not actively regulated. Thus the plasma level of these solutes begins torise with

the initial decline in GFR and increases progressively as kidney function deteriorates. Once the GFR falls below 15 mL/min/1.73m^2, patients begin to complain of many of the manifestations. It is thought that many of these symptoms are mediated by an accumulation of uremic toxins. However, it is not yet possible to identify the toxins responsible for most uremic symptoms[49]. Although it has been postulated that urea may be an important toxin, symptoms ofuremia correlate only inconsistently with the level of urea.

Decreased hormone production

The kidneys normally produce several hormones, including erythropoietin and calcitriol (1,25-dihydroxycholecalciferol), the active form of vitamin D. The decreased production of these two hormones plays an important role in the development of anemia and bone disease, respectively. [51,52]

CLINICAL MANIFESTATIONS OF CHRONIC RENALINSUFFICIENCY[53]

1. **Electrolytes**: Edema, hyponatremia, hyperkalemia, hyperphosphatemia, metabolic acidosis, hyperuricemia, hypocalcemia.

2. **Gastrointestinal:** Anorexia, nausea, vomiting, malnutrition.

3. **Cardiovascular:** Accelerated atherosclerosis, systemic hypertension, pericarditis.

4. **Hematological:** Platelet dysfunction, anemia, immune dysfunction.

5. **Musculoskeletal:** Renal osteodystrophy, muscle weakness, growth retardation in children, amyloidarthropathy caused by beta$_2$- microglobulin deposition.

6. **Neurological:** Encephalopathy, seizures, peripheral neuropathy

7. **Endocrine:** Hyperlipidemia, glucose intolerance caused by insulin resistance, amenorrhea, and infertility in women, impotence.

8. **Skin:** Pruritus, dry skin.

SYSTEMIC COMPLICATIONS [52,53]

The uremic syndrome consists of an array of complex symptoms and signs that occur when

advanced kidney failure prompts the malfunction ofvirtually every organ system. However, the onset of uremia is slow and insidious, beginning with rather nonspecific symptoms such as malaise, weakness, insomnia and a general feeling of being unwell. Patients may lose their appetite and complain of morning nausea and vomiting. Eventually, signs and symptoms of multisystem failure are evident.

Pathogenic factors in renal osteodystrophy[72]-

- Hypocalcemia
- Phosphorus retention
- Impaired calcaemic response to PTH
- Altered degradation of PTH by the kidney
- Disordered regulation of PTH and calcitonin
- Altered Vitamin D metabolism

Dyslipidemia

Abnormalities of lipid metabolism appear to occur early in the course of kidney failure and are another treatable complication of CRI. Lipoprotein lipase activity falls in patients with glomerular filtration rates (GFRs) of 50 mL/min or less, and triglyceride values rise with GFRs in the range of 15 to 30 mL/min[73,74]. As kidney failure progresses, a generalized disorder of lipid metabolism becomes apparent, irrespective of the underlying cause of the kidney disease. Abnormalities include hypercholesterolemia, elevated ratio of low-to-high-density lipoproteins, the elevation of lipoprotein(a), and elevated chylomicron remnant level[75].

Hyperlipidemia is among the many contributors to atherosclerosis in patients with ESRD. Treatment during the CRI stage may help prevent such complications, although specific data on the benefits of such intervention are not currently available. Standard regimens for the management of dyslipidemia can be employed in CRI, with appropriate modification of drug dosages based on the level of kidney function.

Thyroid disease in ESRD

ESRD is a non-thyroidal illness, which affects thyroid hormone metabolism. Multiple other comorbid conditions that affect thyroid hormone metabolism (diabetes mellitus, malnutrition, frequent infections) are also common in ESRD. Certain thyroid diseases occur in increasing frequency in these patients and they include goiter, thyroid nodules, and thyroid cancer. [59,60,61]

Chronic renal failure and skin

The cutaneous manifestations of chronic renal failure include pruritus, dry flaky skin, as well as darkening and yellow pigmentation of the skin, made more obvious by the pallor of anemia. Bullous lesions in sun-exposed areas (pseudoporphyria cutanea) are occasionally seen in patients on dialysis. Proximal skin necrosis (thighs and trunk) is a consequence of ischemia resulting from calcification and occlusion of arterioles. Trophic nail changes are frequent, including brown nail arcs (half-and-half nails).[59,60,61]

Smoking

Smoking, besides increasing the risk of cardiovascular events, is an independent risk factor for the development of end-stage renal disease in men with kidney disease. Smoking cessation alone may reduce the risk of disease progression by 30% in patients with type 2 diabetes.

Medical care: Medical care of patients with CRF should focus on the following: 23,[24,25]

- Delaying or halting the progression of CRF
- Treatment of the underlying condition if possible
- Aggressive blood pressure control to the target value
- Use of ACE inhibitors as tolerated, with close monitoring for renal deterioration and for hyperkalemia (avoid in advanced renal failure, bilateral renal artery stenosis [RAS], RAS in the solitary kidney).
- Aggressive glycemic control in patients with diabetes.
- Protein restriction – Controversial

- Treatment of hyperlipidemia
- Avoidance of nephrotoxins – IV radiocontrast, nonsteroidal anti-inflammatory agents, aminoglycosides.
- Treating pathologic manifestations of CRF [56,57,58]
- Anaemia with erythropoietin.
- Hyperphosphatemia with dietary phosphate binders and dietary phosphate restriction.
- Hypocalcemia with calcium supplements +/- calcitriol.
- Hyperparathyroidism with calcitriol or vitamin D analogs
- Volume overload with loop diuretics or ultrafiltration
- Metabolic acidosis with oral alkali supplementation

Uremic manifestations with chronic renal replacement therapy (hemodialysis, peritoneal dialysis, or renal transplantation): indications include severe metabolic acidosis, hyperkalemia, pericarditis, encephalopathy, intractable volume overload, failure to thrive and malnutrition, peripheral neuropathy, intractable gastrointestinal symptoms, and GFR less than 10 ccs/min.

Renal transplantation is the best option available to patients with ESRD. However, cultural and social factors have hampered the increase in renal transplantation in certain parts of the world, including India and other Asian countries. In fact, in Japan, most renal transplantation procedures have been performed with donations from a living relative, but the number never exceeded 100 per year despite having a large number of patients with ESRD.

A marked shortage of donor kidneys, the lack of good cadaver program, and large-scale poverty have led to the trafficking of organs. It is estimated that 50% to 50% of kidneys transplanted in India come from living unrelated donors.

MATERIALS AND METHODS

The present Descriptive study was done in the Department of General Medicine in collaboration with the nephrology department at Karpaga Vinayaga Institution of medical

sciences. The study was carried out during the period of October -2017 to June -2019 (20 months)

Sample size and sampling: 100

Inclusion criteria:

1) Patients with chronic kidney disease with stage I –V disease.

2) Patients with End-stage renal failure on renal replacement therapy in the form Of Hemodialysis and peritoneal dialysis.

3) GFR <60 ml/min/1.73 m^2 on the basis of estimated GFR using the Modification of Diet in Renal Disease (MDRD) formula (CKD stages 3 to 5).

Exclusion criteria:

1) Patients with other systemic illness without renal failure

2) Pregnancy

3) Aplastic anemia

4) Known hematological malignancy causing secondary renal failure

5) Patients with end-stage renal disease treated with renal replacement therapy in the form of renal transplantation.

6) History of blood transfusion during the last three months

During this period, all newly diagnosed cases of chronic kidney disease based on the National Kidney foundation definition were included in this study.[1] All the patients were evaluated based on detailedhistory taking, clinical examination, and laboratory investigations after informed consent was obtained from them. Staging of CKD was done based on the National Kidney Foundation (NKF/KDOQI) staging system. GFR was estimated using the abbreviated MDRD (modification of diet in renal disease) formula.

The case history was recorded on a Pro-forma. Data on age, sex, education, occupation, and lifestyle factors, tobacco usage (chewed), smoking and alcohol consumption were collected

from all subjects. Detailed medical histories were obtained regarding present complaints. All subjects were also interviewed regarding the past history of diabetes, hypertension and other co-morbid conditions.

Chronic Glomerulonephritis was diagnosed in patients with a history of edema, hypertension and documented the nephritic range of proteinuria. Hypertensive nephropathy was diagnosed in patients with a long history of hypertension and other target organ damage. Diabetic Nephropathy was diagnosed in patients with a long history of diabetes, presence of diabetic retinopathy and proteinuria more than 500mg in 24 hours. Chronic Pyelonephritis was diagnosed on ultra-sonogram when there is the presence of small kidneys with irregular borders. Obstructive Uropathy, Autosomal dominant polycystic kidney disease, and Obstructive nephropathy were diagnosed by ultra-sonogram.

Data collection/Analysis:

Investigations for assessment of renal failure

A. Blood urea, Serum creatinine, serum electrolytes.

B. Abdominal ultrasound with KUB

C. Cardiovascular parameters (Blood pressure & Echocardiogram).

STATISTICAL ANALYSIS

1. Data was collected by using pre-tested proforma to meet the objectives of the study.

2. Mean, median and standard deviation for descriptive statistics.

3. Chi-square tests for inferential statistics.

4. Statistical significance if $P < 0.05$ and 95% confidence limit will be used.

5. Fisher's exact test when appropriate was performed to analyze the univariate relations between possible prognostic factors.

6. As it is likely that different prognostic factors are mutually related, the independent effects of prognostic factors were additionally analyzed with multivariate logistic regression.

7. Analysis of variance (ANOVA), Student`s `t` test were applied and *p*-value of less than 0.05 was considered significant.

RESULTS

The present Descriptive study was done in the Department of General Medicine in collaboration with the nephrology department at Karpaga Vinayaga Institution of medical sciences. The study was carried out during the period of October -2017 to June -2019 (20 months) Totally 100 patients were included in the study who diagnosed CKD andSTAGING.

Table: 1 Age Group Distribution

S. NO	AGE GROUP	FREQUENCY	PERCENT
1	20-30 Years	15	15.0
2	31-40 Years	16	16.0
3	41-50 Years	13	13.0
4	51-60 Years	20	20.0
5	61-70 Years	15	15.0
6	Above 70 Years	21	21.0
7	Total	100	100.0

In the table the age group distribution. 20 years – 70 years above the patients are included in the study. Age group: 20-30 Years 15 patients (15%) 31-40 Years 16 patients(16%) 41-50 Years 13 patients (13.%) 51-60 Years 20 patients (20%)61-70 Years 15 patients (15%) Above 70 Years 21 patients (21%).

Table: 2 Gender Distribution

Gender	Frequency	Percent
Male	62	62.0
Female	38	38.0
Total	100	100.0

In the table shows the gender distribution Among 100 cases of CKD, there were 62 (62%) male patients and 38 (38%) female patients. The ratio of male to female was 2.63 i.e. males are 2.63 times more susceptible to CKD when compared to females.

Table: 3 Differential Diagnosis

S. No	Diagnosis	Frequency	Percent
1	Diabetic nephropathy	31	31.0
2	Interstitial	19	19.0
3	Polycystic kidney disease	17	17.0
4	Kidney stones	10	10.0
5	Glomerulonephritis	12	12.0
6	Hypertension nephropathy	11	11.0
7	Total	100	100.0

In 100 cases the differential diagnosis were analyzed Diabetic nephropathy was in 31(31%) Interstitial kidney disease was in 19(19%) Polycystic kidney disease was diagnosed in 17 patients which were (17%) Kidney stones in different types seen in 10 patients (10%) Glomerulonephritis in 12 patients (12%) Hypertension nephropathy in 11(11%). Diabetic nephropathy was more common in the study which was statistically more significant of p-value <0.005>.

Table: 4 Hemoglobin Level

HB	Frequency	Percent
<5	4	4.0
5-10	39	39.0
>10	57	57.0
Total	100	100.0

The above table reveals that 39% of the patients have their hemoglobin level in the range of 5-10 gm%. Only 4% of the patients have a value below 5mg%, but 57% of the patient's exhibit that their hemoglobin level is more than 10 mg%.

TABLE: 5 SERUM POTASSIUM

Serum Potassium	Frequency	Percent
<3.5	12	12.0
3.5-5.0	60	60.0
>5.0	28	28.0
Total	100	100.0

In Serum Potassium 28% of patients have Hyperkalemia. 60% had the value within normal limits (3.5-5 meq/1). Only 12% had a value of less than 3.5 meq/1.

Table: 6 Serum Sodium Level

SODIUM	Frequency	Percent
<130	29	29.0
130-143	71	71.0
Total	100	100.0

It appears that hyponatremia (Serum sodium level < 130 meq/1) is present in 29% of patients. Further, in 71% of cases, this value lies between the normal limits (130-143 meq/1).

Table: 7 Serum Albumin Level

Serum Albumin	Frequency	Percent
<3.5	68	68.0
3.5-5.0	32	32.0
Total	100	100.0

Hypoalbuminemia (Serum Albumin < 3.5g/dl) can be seen in 68% of cases. 32% of cases have this value within normal limits (3.5 - 5 g/dl).

Table 8: Kidney Size

Kidney Size	Frequency	Percent
Small (<8.5 cm)	61	61.0
Normal	29	29.0
Increased >12 cm	10	10.0
Total	100	100.0

In 61% of the cases seem to have decreased kidney size and 29% appear to have an increased kidney size. Whereas 10% of the patients have increased normal size.

TABLE: 9 Echo Findings

Echo	Frequency	Percent
Not done	21	21.0
LVH	41	41.0
Ischemic dilated cardiomyopathy Hypokinesiaof wall or septum	12	12.0
Diastolic dysfunction	8	8.0
Normal	14	14.0
Pericardial effusion	4	4.0
Total	100	100.0

In Echo Cardiogram Was Not done in21patients (21%) LVH was present in 41 cases(41%). Ischaemic dilated cardiomyopathy, Hypokinesia of wall or septum is seen in 12 patients (12%).Diastolic dysfunction.8 patients (8%).Normal findings in echocardiography were in14 patients (14%) Pericardial effusion was seen in 4 patients (4%) LVH was more common among CRF patients as it reflects on cardiac efficiency.

Table: 10 USG Parenchymal Changes

USG Parenchymal Changes	Frequency	Percent
Grade II parenchymal changes	38	38.0
Grade III parenchymal changes	12	12.0
b\I shurken Kidneys	18	18.0
Grade I parenchymal changes	18	18.0
Normal	14	14.0
Total	100	100.0

Table &Graph: 10 Grade II parenchymal changes were seen in 38 patients (38%).Grade III parenchymal changes in 12 patients (12%).b\I shurken Kidneys in 18(18%).Grade I parenchymal changes were seen in 18 patients (18%).Normal parenchymal seen in 14 patients (14%).

Table: 11 Comparison of Sex Distribution In Diabetic And Non-Diabetic Nephropathy Patients

			DM		Total
			Non Diabetic nephropathy	Diabetic nephropathy	
		Count			
	Male				
		% within DM	45	17	62
Sex			65.2%	54.8%	62.0%
		Count	24	14	38
	Female		34.8%	45.2%	38.0%
		% within DM	69	31	100
		Count	100.0%	100.0%	100.0%
	Total				
		% within DM			

In 100 patients 62 patients were male and 38 females.Male non-diabetic patients were 45(65.2%) male diabetic nephropathy patients were 17 (54.8%) female non-diabetic patients were 24(34.8%) female diabetic nephropathy patients were 14 (45.2. Male diabetic nephropathy patients were more in our study which correlates with increased duration of diabetes. %) Pearson Chi-Square=0.978p=0.323 were statically more significant.

Table: 12 Shows Comparison Of Diabetic Nephropathy With Age,Creatinine,Creatinine Clearance

Group Statistics							
	DM	N	Mean	Std. Deviation	Std. Error Mean	t value	P value
Age	Non Diabetic Nephropathy	69	51.5362	17.82726	2.14615	0.914	0.363
	Diabetic Nephropathy	31	55.0323	17.36372	3.11862		
creatinine	Non Diabetic Nephropathy	69	9.2478	3.41471	.41108	2.480*	0.015
	Diabetic Nephropathy	31	7.3326	3.90280	.70096		
CCL	Non Diabetic Nephropathy	69	13.7570	5.13656	.61837	0.233	0.816
	Diabetic Nephropathy	31	14.0313	6.09128	1.09403		

In non-diabetic nephropathy 69(13.75) when compared to diabetic nephropathy 31(14.03) The patient in whom diabetic nephropathy was the cause of chronic kidney disease were of an older age group and were having better creatine clearance compared to non diabetic etiology.off value (0.816)

SUMMARY

In our study, while 84.68% had hypertension, only in 8.41% was CKD due to hypertensive nephrosclerosis. In the CKD registry of India report, 71% had hypertension, but only in 19.8% the cause of CKD was hypertensive nephrosclerosis.[30,31] Cigarette smoking was prevalent in 32.73%, alcohol consumption in 6.91%, use of nephrotoxic agents like NSAIDs in 5.1% and herbominerals in 4.5% which might have contributed to the faster progression of the disease in these patients. This is consistent with the CKD registry of India report, where cigarette smoking was prevalent in 32%, alcohol consumption in 6.4% and NSAID use in 2%.[6] Family history of diabetes (8.71% (hypertension(12.91%) and CKD (1.2%) were present

only in a small number of patients.

The most common symptoms in our patients were pedal oedema (79%), oliguria (73%), breathlessness (70%), vomiting (44%) and anorexia(32%). CNS symptoms like convulsion and altered sensorium were found in 6% and 10% of patients respectively. Decrease GFR in NHANES III patients was associated with impaired walking andlifting ability. [67,69,74]

Cardiovascular disease was present in 167 (50.15%) patients, of which left ventricular hypertrophy was the commonest (141; 83.93%), while in the CKD Registry of India Report77, ischemic heart disease (44.2%) was the commonest with left ventricular hypertrophy in 31.6%. Cardiovascular disease was more common when the etiological diagnosis was diabetic nephropathy (65.8%) or hypertensive nephrosclerosis (85.7%) and the correlation was statistically significant. The correlation of cardiovascular disease and the presence or absence of DM as well as of hypertension was statistically significant. This is consistent with the findings in CKD. To execute a change in the management of patients with CKD, medical students, healthcare professionals, and established physicians, need to be educated about the prevalence and consequences of CKD. The concept that CKD is a risk factor for cardiovascular disease, and needs to be managed should be emphasized. Screening of the high-risk individuals (those with hypertension, diabetes mellitus, cardiovascular disease and first-degree relatives of patients with hypertension, diabetes mellitus or renal disease) will maximize the detection of CKD andbenefit a large population of patients.[71,72,80].

CONCLUSION

1. Chronic kidney disease is defined as abnormalities of kidney structure or function, present for>3 months, with implications for health. Kidney damage refers to a broad range of abnormalities observed during clinical assessment, which may be insensitive and non-specific.

2. The present Descriptive study was done in the Department of General Medicine in collaboration with the nephrology department at Karpaga Vinayaka institution of medical sciences. The study was carried out during the period of October -2017 to June -2019 (20 months) . Totally 100 patients were included in the study who diagonalized CKD and staging.

3. The age group distribution was 20years – 70 years above the patients are included in the study. Age group : 20-30 Years 15 patients (15%) 31-40 Years 16 patients(16%) 41-50 Years

13 patients (13.%) 51-60 Years 20 patients (20%)61-70 Years 15 patients (15%) Above 70 Years 21 patients (21%).

4. Gender distribution Among 100 cases of CKD, there were62 (62%) male patients and 38 (38%) female patients. The ratio of male to female was 2.63 i.e. males are 2.63 times more susceptible to CKD when compared to females.

5. In 100 cases the differential diagnosis was analysed Diabetic nephropathy was in 31(31%)Interstitial kidney disease was in 19(19%) Polycystic kidney disease diagnosed in 17 patients which were (17%) Kidney stones in different types seen in 10 patients (10%) Glomerulonephritis in 12 patients (12%) Hypertension nephropathy in 11(11%). Diabetic nephropathy was more common in the study which was statistically more significant of p-value <0.005>.

6. The presence of various symptoms observed in 100 patients are presented in the above table. We see that 79%of the cases had pedal edema followed by the most common urinary symptom Oliguria that is 73%. The Gastrointestinal symptom namely anorexia is found in 33 cases. 28% had general weakness and 44% were having vomiting as a symptom. The numbers of cases having facial edema were 25 and 70% of the cases exhibited breathlessness as a symptom. Puffiness of the face 62 (77.50%), swelling over feet 58 (72.50%) and breathlessness 56 (70.00%) were the next predominant symptom. Nausea and vomiting were present in 54 (67.50%) and tingling and numbness of extremities were complained by 40 (50.00%) patients. Joint pain 24 (30.00%), was the other less common presenting symptom in this study.

7. Blood pressure was recorded by standard method .systolic blood pressure was <120 in 1patients which was (1%) systolic blood pressure between 120-139 in16 patients which was(16%) Above 140-159 were observed in 20 patients were (20%) Above 160 were observed in 63 patients (63%) most of the patient were hypertensive in CKD due to decreased renin release which was statistically more significant of p-value <0.005>

8. Diastolic blood pressure was <80 seen in 6 patients(6%) diastolic blood pressure between 80-89 observed in 26 patients (26%) Above 90-99 was in 43patients(43%)were observed in 43 patients Above 100 were observed in 25 patients (25%) most of the patient were hypertensive in CKD due to decreased cardiovascular stability which was statistically more

significant of p-value <0.005>

9. 39% of the patients have their hemoglobin level in the range of 5-10 gm%. Only 4% of the patients have a value below 5mg%, but 57% of the patients exhibit that their hemoglobin level more than 10 mg%.

10. 28% of patients have Hyperkalemia. 60% had the value within normal limits (3.5-5 meq/1). Only 12% had a value of less than 3.5 meq/1.

11. hyponatremia (Serum sodium level < 130 meq/1) is present in 29% of patients. Further, in 71% of cases, this value lies between the normal limits (130-143 meq/1).

12. Hypoalbuminemia (Serum Albumin < 3.5g/dl) can be seen in 68% of cases. 32% of cases have this value withinnormal limits (3.5 - 5 g/dl).

13. 61% of the cases seem to have decreased kidney size and 29% appears to have an increased kidney size. Whereas 10% of the patients have increased normal size.

14. Echo Cardiogram Was Not done in21patients (21%) LVH was present in 41 cases(41%).Ischaemic dilated cardiomyopathy, Hypokinesia of wall or septum is seen in

12 patients (12%).Diastolic dysfunction.8 patients (8%). Normal findings in echocardiography were in14 patients (14%) Pericardial effusion was seen in 4patients (4%) LVH was more common among CRF patients as it reflects on cardiac efficiency

15. Grade II parenchymal changes were seen in 38 patients (38%).Grade III parenchymal changes in 12 patients (12%).b\I shurken Kidneys in18(18%).Grade I parenchymal changes seen in 18 patients (18%).Normal parenchymal seen in 14 patients (14%)

16. In nondiabetic nephropathy 69(13.75) when compared to diabetic nephropathy 31(14.03) The patient in whom diabetic nephropathy was the cause of chronic kidneydisease were of an older age group and were having bettercreatine clearance compared to non diabetic etiology. offp value (0.816).

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