DISSERTATION

ON

A STUDY ON CLINICAL PROFILE OF

CHRONIC RENAL FAILURE (CKD STAGE 3, 4&5)

SUBMITTED FOR
M.D. BRANCH I
(GENERAL MEDICINE)

THANJAVUR MEDICAL COLLEGE
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THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY
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CERTIFICATE

This is to certify that dissertation entitled **' A study on clinical profile of Chronic renal failure (CKD stage 3, 4&5)** submitted by **Dr.K.Venkataramanan** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in the partial fulfillment of the requirement of M.D Degree - Branch I (General Medicine) is a bonafide research work carried out by him under my direct supervision and guidance.

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CONTENTS

INTRODUCTION

The kidney is one of the most highly differentiated organs in the body.. Endocrine functions, the regulation of blood pressure and intraglomerular hemodynamics, solute and water transport, acid-base balance, and removal of fuel or drug metabolites are all accomplished by intricate mechanisms of renal response. Renal failure is classified into acute and chronic renal failure.

Acute renal failure (ARF) is characterized by a rapid decline in glomerular filtration rate (GFR) over hours to days. . Retention of nitrogenous waste products, oliguria (urine output <400 mL/d contributing to extra cellular fluid overload), and electrolyte and acid-base abnormalities are frequent clinical features. [1]

Chronic kidney disease (CKD) is an important, chronic, non communicable disease epidemic that affects the world, in. Because of the absence of a renal registry in India, the true magnitude of CKD/end-stage renal disease (ESRD) is unknown, eventhough it seems to be high. CKD is a worldwide public health problem and is now recognized as a common condition that is associated with an increased risk of cardiovascular disease and chronic renal failure (CRF).

The Kidney Disease Outcomes Quality Initiative (K/DOQI) of the National Kidney Foundation (NKF) defines chronic kidney disease as either kidney damage or a decreased kidney glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for 3 or more months. Whatever the underlying etiology, the destruction of renal mass with irreversible sclerosis and loss of nephrons leads to a progressive decline in GFR [(2).]

K/DOQI published a classification of the stages of chronic kidney disease,

as follows:[(5)]

- Stage 1: Kidney damage with normal or increased GFR (>90 mL/min/1.73 m^2)
- Stage 2: Mild reduction in GFR (60-89 mL/min/1.73 m^2)
- Stage 3: Moderate reduction in GFR (30-59 mL/min/1.73 m^2)
- Stage 4: Severe reduction in GFR (15-29 mL/min/1.73 m^2)
- Stage 5: Kidney failure (GFR <15 mL/min/1.73 m^2 or dialysis)

In stage 1 and stage 2 chronic kidney disease, GFR alone does not clinch the diagnosis. Other markers of kidney damage, including abnormalities in the composition of blood or urine or abnormalities in imaging tests, should also be present in establishing a diagnosis of stage 1 and stage 2 chronic kidney disease

The severity of signs and symptoms of uremia vary from patient to patient depending at least in part on the magnitude of the reduction in glomerular filtration rate and the rapidity with renal function is lost [3]

The term *chronic renal failure* applies to the process of continuing significant irreversible reduction in nephron number, and typically corresponds to CKD stages 3–5. [4]

The most important initial diagnostic step in the evaluation of a patient presenting with elevated serum creatinine is to distinguish newly diagnosed CKD from acute or sub acute renal failure because the latter two conditions may respond to therapy specific to the disease. Previous measurements of plasma creatinine concentration are particularly helpful in this regard. Normal values from recent months or even years suggest more acute, and hence reversible. Elevated plasma creatinine concentration in the past suggests that the renal disease represents the progression of a chronic process. Even if there is evidence of chronicity, there is the possibility of a superimposed acute process. Some of the laboratory tests and imaging studies can be helpful. Evidence of metabolic bone disease with hyperphosphatemia, hypocalcaemia, and elevated PTH and bone alkaline phosphatase levels suggests chronicity. Normo chromic, normocytic anemia suggests that the process has been ongoing for some time. The finding of

bilaterally reduced kidney size (<8.5 cm) favors CKD. Renal osteodystrophy strongly suggests the chronicity of renal dysfunction.

The term *end-stage renal disease (ESRD)* represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the *uremia syndrome*. This syndrome leads to death unless the toxins are removed by renal replacement therapy, using dialysis or kidney transplantation. *End-stage renal disease* will be supplanted by the term *stage 5 CKD.* In the absence of a clinical diagnosis, renal biopsy may be the only recourse to establish an etiology in early-stage CKD. Once the CKD is advanced and the kidneys are small and scarred, there is little utility and significant risk in attempting to arrive at a specific diagnosis.

Since, most of the patients of chronic kidney disease present only as CRF, the advanced of stage CKD(Stage 3,4 &5), and also present with various clinical features, this study on CRF is taken focusing the various clinical features, etiology and correlation of duration of symptoms with severity of renal failure.

AIM OF THE STUDY

1. To study the various clinical presentations of CRF.

2. To study the etiology of CRF and also their incidence.

3. To study the correlation between duration of symptoms and the severity of CRF (stages of CRF).

4. To study the prevalence of CRF in relation to age and sex.

REVIEW OF LITERATURE

The Kidney Disease Outcomes Quality Initiative (K/DOQI) of the National Kidney Foundation (NKF) defines chronic kidney disease as either kidney damage or a decreased kidney glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m^2 for 3 or more months. Whatever the underlying etiology, the destruction of renal mass with irreversible sclerosis and loss of nephrons leads to a progressive decline in GFR [(2)]

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Stage 5: Kidney failure (GFR <15 mL/min/1.73 m^2 or dialysis). [(5)]

The term *chronic renal failure* applies to the process of continuing significant irreversible reduction in nephron number, and typically corresponds to CKD stages 3–5. The term *end-stage renal disease* represents a stage of CKD where the accumulation of toxins, fluid, and electrolytes normally excreted by the kidneys results in the *uremic syndrome*. . *End-stage renal disease* will be supplanted term *stage 5 CKD*

Once the CKD is advanced and the kidneys are small and scarred, there is little utility and significant risk in attempting to arrive at a specific diagnosis The severity of signs and symptoms of uremia vary from patient to patient depending at least in part on the magnitude of the reduction in glomerular filtration rate and the rapidity with renal function is lost. [(2)]

When the GFR is 20% to 50% of normal. Azotemia appears, usually associated with anemia and hypertension. Polyuria and nocturia occur as a result of decreased concentrating ability. Sudden stress (e.g., with nephrotoxins) may precipitate uremia.

When the GFR is less than 20% to 25% of normal. The kidneys cannot regulate volume and solute composition, and patients develop edema, metabolic acidosis, and hypocalcemia. Overt uremia may ensue, with neurologic, gastrointestinal, and cardiovascular complications. When the GFR is less than 15% of normall, End-

Stage Renal Disease.where the patient cannot prolong his life without renal replacement

GLOMERULAR FILTRATON RATE

In order to stage CKD, it is necessary to estimate the GFR. Two equations commonly used to estimate GFR and incorporate the measured plasma creatinine concentration, age, sex, and ethnic origin.

1.Equation from the Modification of Diet in Renal Disease study [6]

Estimated GFR (mL/min per 1.73 m^2) = 1.86 x $(P_{Cr})^{-1.154}$ x $(age)^{-0.203}$

Multiply by 0.742 for women

Multiply by 1.21 for African Americans

2. Cockcroft-Gault equation

Estimated creatinine clearance (mL/min) =

(140-age) *body weight (kg)

72*Pcr (mg/dL)

Multiply by 0.85 for women

Clinical Manifestations of Chronic Kidney Disease [7]

Uremia leads to disturbances in the function of virtually every organ system.

Various clinical and laboratory manifestation of CKD is as follows,

Fluid and electrolyte disturbances	**Neuromuscular disturbances**	**Dermatologic disturbances**
Volume expansion	Fatigue	Pallor
Hyponatremia	Sleep disorders	Hyperpigmentation
Hyperkalemia	Headache	Pruritus
Hyperphosphatemia	Impaired mentation	Ecchymoses
Endocrine-metabolic disturbances	Lethargy	**Gastrointestinal disturbances**
Secondary hyperparathyroidism	Asterixis	Anorexia
Adynamic bone	Muscular irritability	Nausea and vomiting
Vitamin D–deficient osteomalacia	Peripheral neuropathy	Gastroenteritis
Carbohydrate resistance	Restless legs syndrome	Peptic ulcer
Hyperuricemia	Myoclonus	Gastrointestinal bleeding
Hypertriglyceridemia	Seizures	**Hematologic and immunologic disturbances**
Increased Lp(a) level	Coma	Anemia
Decreased high-density lipoprotein level	Muscle cramps	Lymphocytopenia
	Myopathy	Bleeding diathesis
	Cardiovascular and pulmonary disturbances	Increased susceptibility to infection
	Arterial hypertension	

Protein-energy malnutrition Impaired growth and development Infertility and sexual dysfunction Amenorrhea α_2-Microglobulin associated amyloidosis	Congestive heart failure or pulmonary edema Pericarditis Hypertrophic or dilated cardiomyopathy Uremic lung	

The clinical characteristics, various factors and the disturbances of various vital organs of the CKD patients, will be considered.

CARDIOVASCULAR

Cardiovascular events are the leading cause of death in CRF patients and they occur at greater frequencies,compared to the normal population .

Syndromes presented by uraemic patient in CVS [8]

Congestive heart failure/hypervolaemia

Ischaemic heart disease (±congestive heart failure)

Left ventricular hypertrophy, concentric or eccentric

Acquired valvular heart disease

Arrhythmia

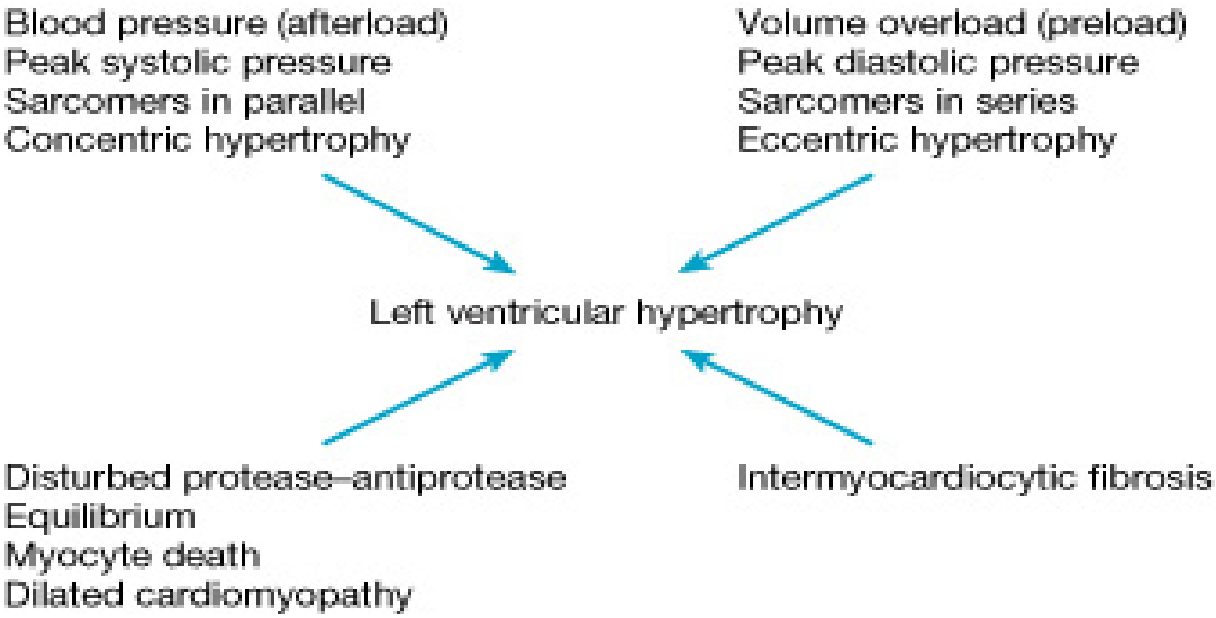
Risk factors:

Hypertension, anemia, Diabetes mellitus, sympathetic over activity, arteriovenous fistula, hypervolaemia and secondary hyperparathyroidism.

Left ventricular hypertrophy

LV hypertrophy is more frequent in early stages of chronic kidney disease and increases progressively, so that it is found in approximately 70 per cent of patients starting renal replacement therapy [9]

Factors involved in LV hypertrophy. [10]

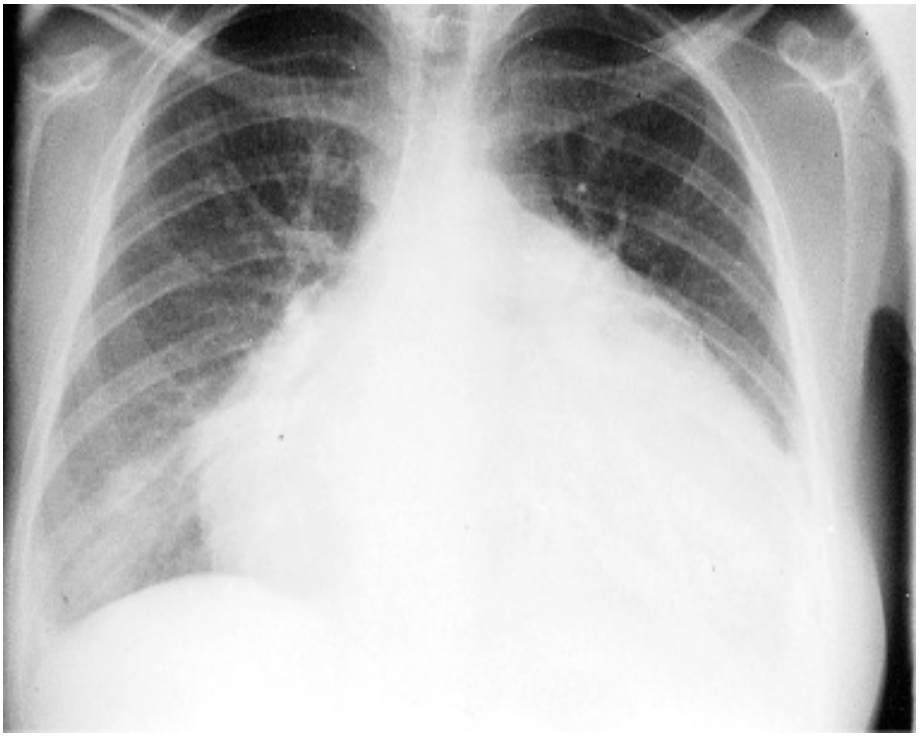


LV hypertrophy reduces actuarial survival in dialysis patients. According to echocardiographic LV hypertrophy defined as LV mitral inflow greater than 125 g/m 2.

Pericardial effusion:

Pericarditis was discovered by Richard bright in 1936 and is categorized into ESRD and dialysis associated pericarditis [(11)].Pericarditis usually presents with chest pain (70%), malaise, weight loss, fever(90%) and a pericardial rub (90%).cardiac arrythymias [(12)] can occur in 20-25% of uremic patients. Volume overload may also contribute to pericardial effusion in patients with ESRD.

X-ray chest PA view of renal failure patient shows pericardial effusion



Hypertension:

Hypertension occurs in 80% to 90% of patients with renal insufficiency. Several factors contribute:

a) Expansion of extracellular fluid volume; this may arise because of reduced ability of the kidney to excrete ingested sodium.
b) Increased activity of the renin-angiotensin system is common; many patients with advanced renal failure have renin levels that are not completely suppressed by the elevated blood pressure.
c) Dysfunction of the autonomic nervous system occurs with insensitive baroreceptor sensitive and with increased sympathetic tone. [13, 14].
d) Possible diminished presence of vasodilators: there may be decreased renal generation of prostaglandins or of factors in the kallikrein-kinin system.

Primary renal disease is the most common etiology of secondary hypertension. Conversely, hypertension is a risk factor for renal injury and ESRD. The increased

risk associated with high blood pressure is graded, continuous, and present throughout the entire distribution of blood pressure above optimal. Renal risk appears to be more closely related to systolic than to diastolic blood pressure.

The atherosclerotic, hypertension-related vascular lesions in the kidney primarily affect the preglomerular arterioles, resulting in ischemic changes in the glomeruli and postglomerular structures. Glomerular injury may also be a consequence of direct damage to the glomerular capillaries due to glomerular hyperperfusion. Glomerular pathology progresses to glomerulosclerosis, and eventually the renal tubules may also become ischemic and gradually atrophic. The renal lesion associated with malignant hypertension consists of fibrinoid necrosis of the afferent arterioles, sometimes extending into the glomerulus, and may result in focal necrosis of the glomerular tuft.

In CKD patients with diabetes or proteinuria > 1 g per 24 h, blood pressure should be reduced to 125/75, if achievable without prohibitive adverse effects. Salt restriction and diuretics should be the first line of therapy. When volume management alone is not sufficient, the choice of antihypertensive agent is to be considered.[15]

HEMOPOIETIC SYSTEM:

Anemia:

Patients with CKD almost uniformly develop a normocytic, normochromic anemia that tends to worsen in parallel with advancing azotemia. Anemia can develop as early as stage 3 CKD (eGFR, 30 to 60 ml/min. Several factors contribute:

a) Erythropoiesis is markedly depressed, mainly due to reduced erythropoietin production; in addition, there may be reduced end-organ response to erythropoietin with reduced heme synthesis.
b) Red cell survival is shortened with a mild to moderate decrease in red cell life span, possible due to a "uremic" toxin.
c) Blood loss is common in uremic patients, possibly secondary to abnormal coagulation due to decreased platelet function.
d) Marrow space fibrosis occurs with osteitis fibrosa of secondary hyperparathyroidism resulting in decreased erythropoiesis.

There was a higher incidence of cardiovascular events when achieve a target hemoglobin level of 13.5 g/dl than when it was used to achieve a hemoglobin level of 11.3 g/dl. [16]

Correction of anemia with epoetin alfa in chronic kidney disease[17] Current practice is to target a hemoglobin concentration of 110 to 120 g/L.

Peripheral blood smear may demonstrate the characteristic burr cells. [18]

Usually the T lymphocyte population is normal, but there is a reduction in the B lymphocyte population .Both cellular and humoral immunity are impaired in renal failure.

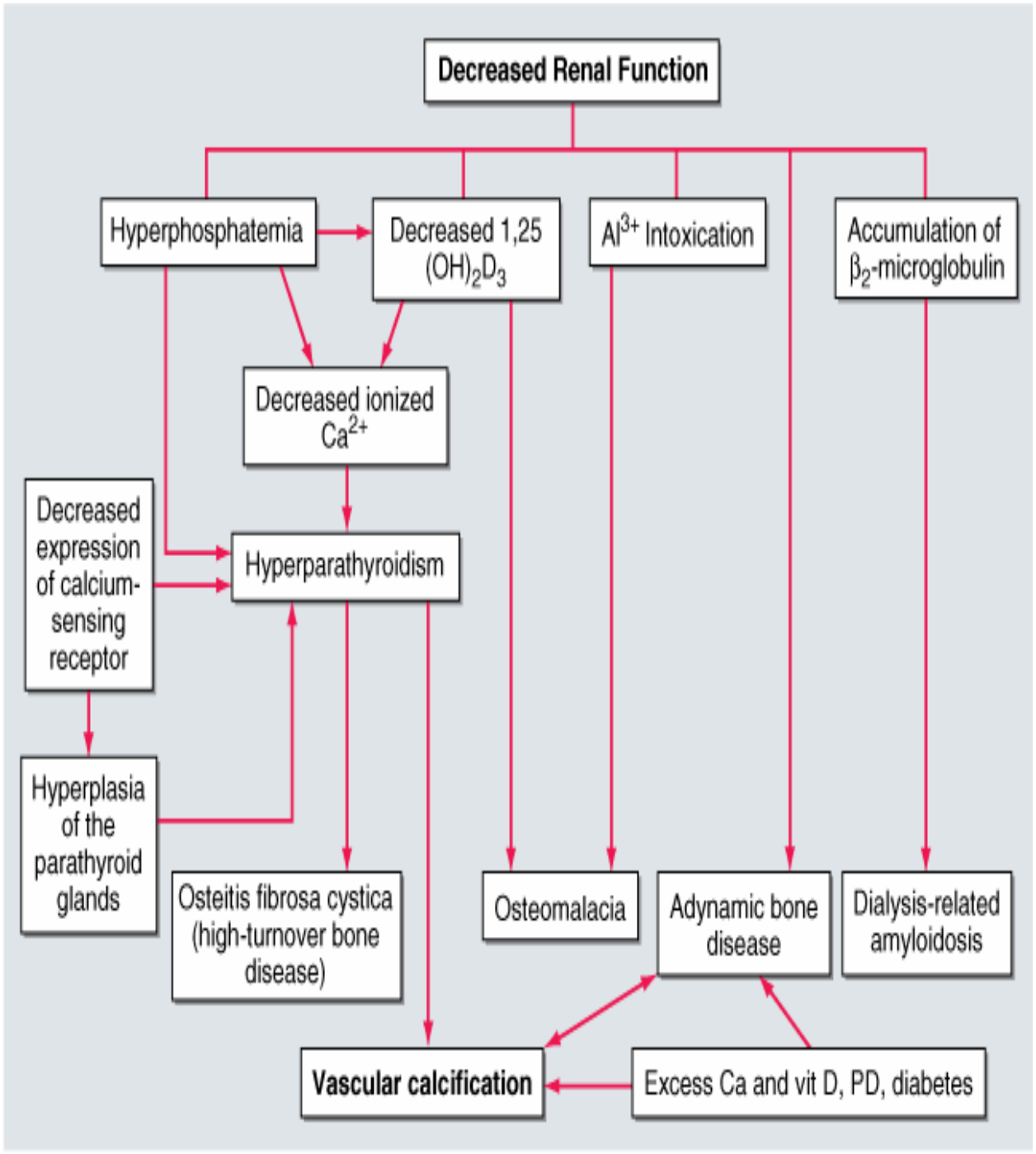
Abnormal Hemostasis

Patients with later stages of CKD may have a prolonged bleeding time, decreased activity of platelet factor III, abnormal platelet aggregation and adhesiveness, and impaired prothrombin consumption.[19] Clinical manifestations include an increased tendency to bleeding and bruising, prolonged bleeding from surgical incisions, menorrhagia, and spontaneous GI bleeding.[20]

Altered Calcium and Phosphorus Metabolism (Renal Osteodystrophy):

a. As GFR decreases there is a slight retention of phosphorus; this phosphorus retention can lead to hypocalcemia, which stimulates PTH. The latter causes phosphaturia, with restoration of serum phosphorus and calcium toward normal. However, this occurs only at the expense of elevated serum PTH levels. This cycle repeats itself in progressive renal failure with PTH

levels increasing progressively. Ultimately, the renal tubule can no longer respond to higher levels of PTH with a further decrease in phosphorus reabsorption. When this occurs, hyperphosphatemia develops, hypocalcemia may become prominent and PTH level can increase to very high levels. High PTH levels cause bone disease with severe osteitis fibrosa.



Source: Fauci AS, Kasper DL, Braunwald E, Hauser SL, Longo DL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine*, 17th Edition: http://www.accessmedicine.com

b) Altered vitamin D metabolism occurs secondary to decreased renal mass or to phosphate retention, with decreased synthesis of 1,25 (OH)2 D3. This deficiency leads to: 1. Diminished intestinal absorption of calcium, 2. decreased calcemic response of the skeleton to PTH, 3. impaired suppression of PTH secretion for any increase in serum calcium level, and 4. altered collagen synthesis. With advanced renal failure, these events can lead to secondary hyperparathyroidism and osteomalacia. Secondary hyperparathyroidism [21] is seen in almost all patients with CRF.

c) Skeletal resistance to the calcemic action of PTH develops; thus an increased PTH is required to maintain serum calcium at any level.

d) Finally, accumulation of aluminum from aluminum binding antacids may contribute to the bone disease.

Target Levels for PTH and Phosphate in Chronic Kidney Disease (22)

Disease Stage	PTH (pg/mL)	Phosphate (mg/dL [mmol/L])
3	35–70	2.7–4.6 (0.87–1.49)
4	70–110	2.7–4.6 (0.87–1.49)
5	150–300	3.5–5.5 (1.13–1.78)

PTH = parathyroid hormone.

Rocaltrol(or a calcitriol analog) 0.25 μg po once/day or 1 to 4 μg 2 times/wk. PTH levels are not corrected to normal because doing so risks precipitating adynamic bone disease.

REPRODUCTION:

Sexual function is profoundly affected in adults of both sexes in end stage renal disease. Upto 78% of males report impotence in severe CRF while the incidence in females is lower. Autonomic neuropathy and uremic polyneuropathy plays a important role in the erectile impotence of many uremic patients [(23)]. In men,the manifestations are impotence decreased libido,oligo or azoospermia. In women,they are dysfunctional uterine bleeding, cystic ovaries, amenorrhoea, anovulation and delayed puberty . These disorders are often reversed by renal transplantation, but dialysis fails to significantly improve them (24)

DERMATOLOGICAL MANIFESTATION

Abnormalities of the skin are prevalent in progressive CKD. Anemic patients may be pale, and those with defective hemostasis may show multiple ecchymoses. Pruritus is quite common. YOUNG reported an incidence of 31 out of 46 to have pruritus (25).The causes are a) Altered sebaceous secretion b) defective sweating c) Altered calcium,phosphorus and parathormone levels. In advanced CKD,

patients may become more pigmented, and this is felt to reflect the deposition of retained pigmented metabolites, or *urochromes*

A skin condition called *nephrogenic fibrosing dermopathy* has recently been reported in which progressive subcutaneous induration, especially on the arms and legs (26). Recent reports suggest that exposure to the magnetic resonance contrast agent, gadolinium, may precipitate this syndrome.

The so-called 'half-and-half nail' is typical of uraemia.The distal portion of each nail bed is red, pink, or brown, occupies 20–60 per cent of the total nail length, and always sharply demarcated; the proximal portion has a dull whitish ground-glass appearance (27)

GASTROINTESTINAL SYSTEM:

The commonest symptoms are anorexia, nausea, vomiting, pain abdomen, distension of abdomen and hiccups.

Hemorrhagic gastritis was found in 52% of uremic patients. Reflux gastritis was found in 52% of uremic patients. [28] Reflux oesophagitis is also a common manifestation.

Upper G.I bleeding [29] in renal failure could be due to hemorrhagic gastritis, duodenitis or peptic ulcer.

Ulcerations of the submucosa of small intestine is also common in CRF [30, 31, 32]. Two cases with RF had small bowel intussusception [33] secondary to intramural hematoma that acted as the lead point.

Absorption of iron is impaired accounting as an additional cause of anemia in RF. Several cases of isolated colonic ulcers have been reported in CRF [34].

Colonic obstruction usually occurs secondary to fecal impaction which is related to the use of aluminium hydroxide. Incidence is 1.7% of CRF patients.

Hypertriglyceridemia, along with hypocalcemia may predispose pancreatitis in CRF patients.

A hepatic abnormality in CRF patients varies from 4.4 to 60%. [35] Hepatomegalies, Hepatic rub, viral hepatitis especially due to hepatitis B virus are the commonest abnormalities noted.

Ascites may occur in patients with CRF, the overall incidence being about 5%. Another series demonstrated a 26% incidence of ascites which also includes that, due to hemodialysis. [36, 37].

Ascites is a consequence of overhydration and relative fluid overload even in the absence of clinically evident congestive cardiac failure and peripheral edema.

Increased capillary permeability in the peritoneal membrane in RF may also account for the development of ascites. The association between RF and ascites was first described in 1970. [38]

LUNGS:

Infection is the most adverse pulmonary complication in renal failure, with a very high mortality and morbidiy. [39]

Uremic lung, a radiological finding showing a butterfly distribution in chest x-ray is common in renal failure (R-40). Pulmonary edema occurs as a result of low intravascular oncotic pressure due to hypoproteinemia.

Uremic pleuritis, occurs in 20% of CRF patients, may demonstrate pleural rub, with bilateral pleural effusions. Patients may experience a typical pleural or catchy pain over the affected area.

JOINTS AND SOFT TISSUE:

A wide variety of rheumatic diseases can affect the patients with CRF and specific management of renal failure can create their own problems (R-41). They are bone and joint infections, crystal induced arthropathy, soft tissue calcification, bursitis, tendintis and osteonecrosis. One study in CRF reported an incidence of 70% of joint complaints including simple arthralgia.

Hyperuricemia in CRF patients can produce periarticular calcifications seen to the extent of 50%. Tumoral calcinosis tendon ruptures, tendonitis, bursitis have also been reported in CRF patients on maintenance hemodialysis. [42].

Neuro Muscular:

Peripheral neuropathy is the most common complication in CRF [43, 44, 45]. It presents as a classical sensory motor polyneuropathy with symmetrical involvement of both upper and lower extremities in a glove and stocking distribution. The lower limbs are more involved than the upper limbs. The earliest clinical symptoms are termed the restless leg syndrome [46] and the restlessness is usually worse at nights, which is usually relieved by walking about, or taking a hot bath.

Muscle cramps; usually involve the gastrocnemius muscles and intrinsic muscles under the arch of the feet. These cramps usually disturb the sleep at night.

Burning feet syndrome, also is reported, where the soles of feet feel hot and painfull with a sensation of swelling [47, 48]. This syndrome is produced by abnormal functioning of the peripheral nerves and is not a true burning sensation [49]. Loss of position and vibration sense and also loss of ankle jerks and the knee jerks are the most probable and earliest signs of uremic neuropathy [50].

As the neuropathy worsens muscle weakness and atrophy may become evident.

Hyperparathyroidism muscle weakness is a common complication in CRF patients due to altered calcium metabolism. Hyperparathyroidism can also manifest as psychiatric or neurologic illness [51].

Vicale C. T [52] reported weakness or fatigability as a presenting symptom in 70% of CRF patients.

The clinical syndrome of hyperparathyroidism include weakness, easy fatigability, symmetric proximal weakness, lower extremities more affected, atrophy and hyper reflexia in the absence of clonus, corticospinal tract signs, bulbar weakness and fasciculations.

Uremic encephalopathy, may mimic a true psychiatric disorder [53, 54] like paranoid delusions, depression etc.

Seizures occur as a late event in CRF. The etiology is usually hypertension with diastolic BP>140mm of Hg with a cerebrovascular accident.

Optic nerve abnormalities like homonymous hemianopia, amaurosis, and visual agnosia have been reported.

Retinal abnormalities are often seen on fundus examinations. They are hemorrhages, exudates, perimacular hemorrhages & arterio – venous nippings and rarely papilledema. Hearing loss may occur in over 75% of cases [55].

OCULAR:

Intra retinal leakage of lipophilic material in lipid containing macrophages produce the hard exudates,which are seen in hypertention[56].Cataracts are reported to occur prematurely due to disturbances in calcium metabolism.

OBSTRUCTIVE NEPHROPATHY:

Since obstructive nephropathy is common, and reversible,obstruction of the urinary tract should should be considered in every uremic patient,whether acute or chronic.

Some common causes of obstruction are

Within the lumen : calculus,blood clot, tumors.

Within the wall :ureteric stricture,conjenital bladder neck obstruction, Neurogenic bladder congenital urethral valve, Pin-hole meatus.

PRESSURE from outside :pelvi ureteric compression due to aberrant vessels, Retro peritoneal fibrosis,prostatic obstruction ,Phimosis.

DRUG INDUCED NEPROPATHY

Drug Induced nephropathy is common and often under diagnosed.This may take the form of a glomerulopathy, or a renal vasculitis, or an interstitial nephritis.Analgesic nephropathy usually takes the form of a tubulo interstitial disease.

Common drugs responsible for Interstitial nephritis:-
ANTIBIOTICS: (-lactams, sulfonamides, quinolones, vancomycin, erythromycin, minocycline, rifampin, ethambutol, acyclovir)
NONSTEROIDAL ANTI-INFLAMMATORY DRUGS, CYCLOOXYGENASE 2 INHIBITORS
DIURETICS:(thiazides, furosemide, triamterene)
ANTICONVULSANTS: (phenytoin, phenobarbital, carbamazepine, valproic acid)

MISCELLANEOUS: (captopril, H_2 receptor blockers, proton pump inhibitors, mesalazine, indinavir, allopurinol)

MATERIALS AND METHODS

STUDY DESIGN:

This study is a prospective study conducted in Thanjavur medical college, department of Internal medicine and department of Nephrology.

In this study patients admitted for renal failure in medical ward and Nephrology ward were included.

SELECTION CRITERIA:

1. Creatinine clearance less than 60ml/min/1.73sqm body surface area.
2. documented reduction in GFR (or) elevated renal parameters for more than 3 months (or) Kidney size less than 8.5 cm(or) increased echogenisity (or) history suggestive of chronicity of renal failure

   Both sexes were included in this study.

EXCLUSION CRITERIA:

1. Creatinine clearance greater than 60ml/min/1.73sqm body surface area.
2. Normal renal parameters within 3 months.

STUDY PERIOD:

This study was conducted between January 2007 and October 2008

STUDY POPULATIONS:

The study included 100 patients of chronic renal failure (chronic kidney disease stage3, 4&5).All satisfied the inclusion and exclusion criteria.

STUDY PROTOCOL AND LABORATORY INVESTIGATIONS:

A detailed history regarding the nature and duration of presenting symptoms was obtained from each patient. List of symptoms regarding urinary tract, fluid overload, Cardio-Respiratory, Gastro-intestinal, bleeding, skin, skeletal, neuro-muscular and reproductive systems were enquired.

A thorough physical examination of the patients were performed with particular attention towards detecting the presence of the following (Anemia, Fluid overload, Hypertension, Acidosis, Pericarditis, Neurological deficits Retinopathy, skeletal Abnormalities, Genital (or) pelvic abnormalities causing obstruction) and they were also evaluated with available investigations regarding chronic kidney disease.

Urine was examined for albumin, deposits and cast especially broad cast and GFR was estimated using creatinine clearance. Serum.creatinine, blood sugar and urea, serum electrolytes, blood calcium and phosphorus were done .X-ray chest PA view and ECG were taken. Ultra sound abdomen was performed for evaluating the size of kidneys, echo texture and other obstructive pathologies. Specific investigations were performed for deserving patients. Renal biopsy was not performed since, once the CKD is advanced and the kidneys are small and scarred, there is little utility and significant risk in attempting to arrive at a specific diagnosis. Detailed history, examination and investigations are given in proforma given below:

Patients were classified, according to the definitions given below;

Based on the creatinine clearance patients, K/DOQI categorized patients of CKD in to 5 stages as follows:

- Stage 1: Kidney damage with normal or increased GFR (>90 mL/min/1.73 m^2)
- Stage 2: Mild reduction in GFR (60-89 mL/min/1.73 m^2)
- Stage 3: Moderate reduction in GFR (30-59 mL/min/1.73 m^2)
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- Stage 5: Kidney failure (GFR <15 mL/min/1.73 m^2 or dialysis)

Source: National Kidney Foundation: Am J Kidney Dis 39(2 Suppl 1):S1, 2002.

Hypertension:

Based on the JNC 7 guidelines (2003), the blood pressure were classified as follows:

Blood pressure measurement	**Systolic BP, in mmHg**	**Diastolic BP,in mmHg**
Normal	<120	*and* <80
Prehypertension	120–139	*Or* 80–89
Stage 1 hypertension	140–159	*Or* 90–99
Stage 2 hypertension	>160	*Or*>100
Isolated systolic hypertension	140	*and* <90

Duration of Symptoms:

Based on the duration of symptoms, patients CRF grouped as

Group I - < 6 months

Group II - 6months – 2 years

Group III - >2yrs

Contracted Kidney:

Kidney size < 8.5 cm in length

Nephrotic syndrome:

24 hr urinary protein more than 3.5 gm/1.73sq mt body surface area.

Hyperkalemia:

Serum potassium > 5 meq/L

Hyponatremia:

Serum sodium < 3.5meq/L

Hyperlipidemia:

Total cholesterol > 200mg/Dl

Fluid overload:

Presence of Facial Puffiness, Pedal edema or Anasarca

Ischemic heart disease

- ECG shows in 2 concordant leads

  - Q waves width > .04s or amplitude > 1/4th of the R wave

and ECHO shows hypokinesia or akinesia of ventricular free wall

RESULTS AND OBSERVATION:

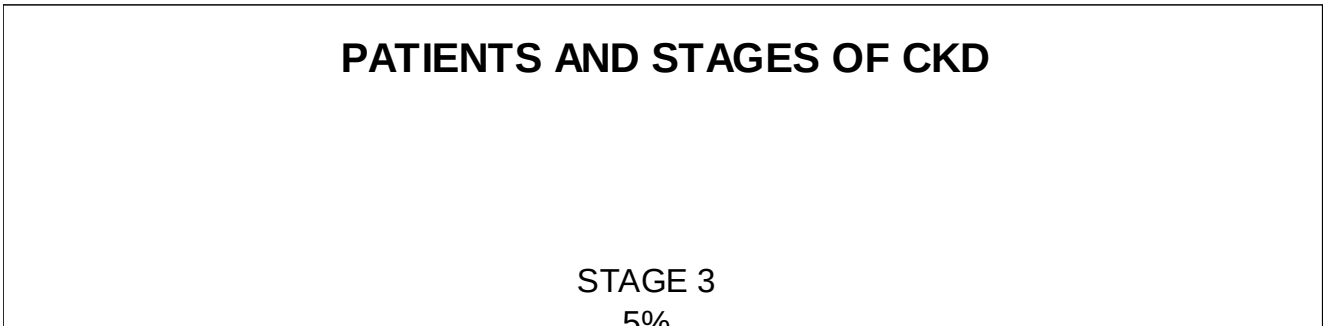
The total number of CKD patients in this study was 100, which included

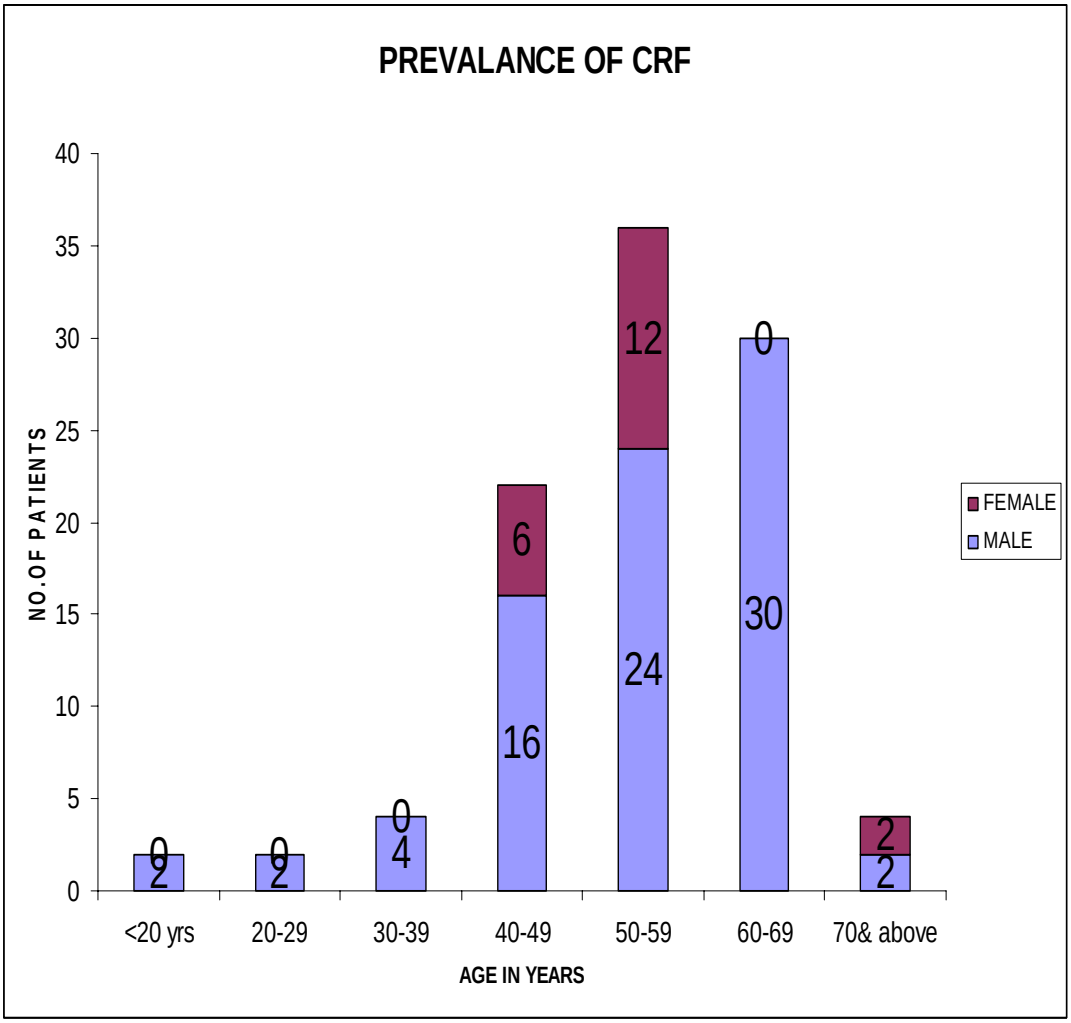
STAGE 3(GFR 30-59ML/MIN) - 5 patients

STAGE 4(GFR 15-29ML/MIN) - 61 patients

STAGE 5(GFR <15ML/MIN) - 34 patients

THIS PIE CHART SHOWS PERCENTAGE OF CRF PATIENT IN VARIOUS STAGES





This chart shows prevalence of CRF related to age and sex..

TABLE-1 AGE DISTRIBUTION

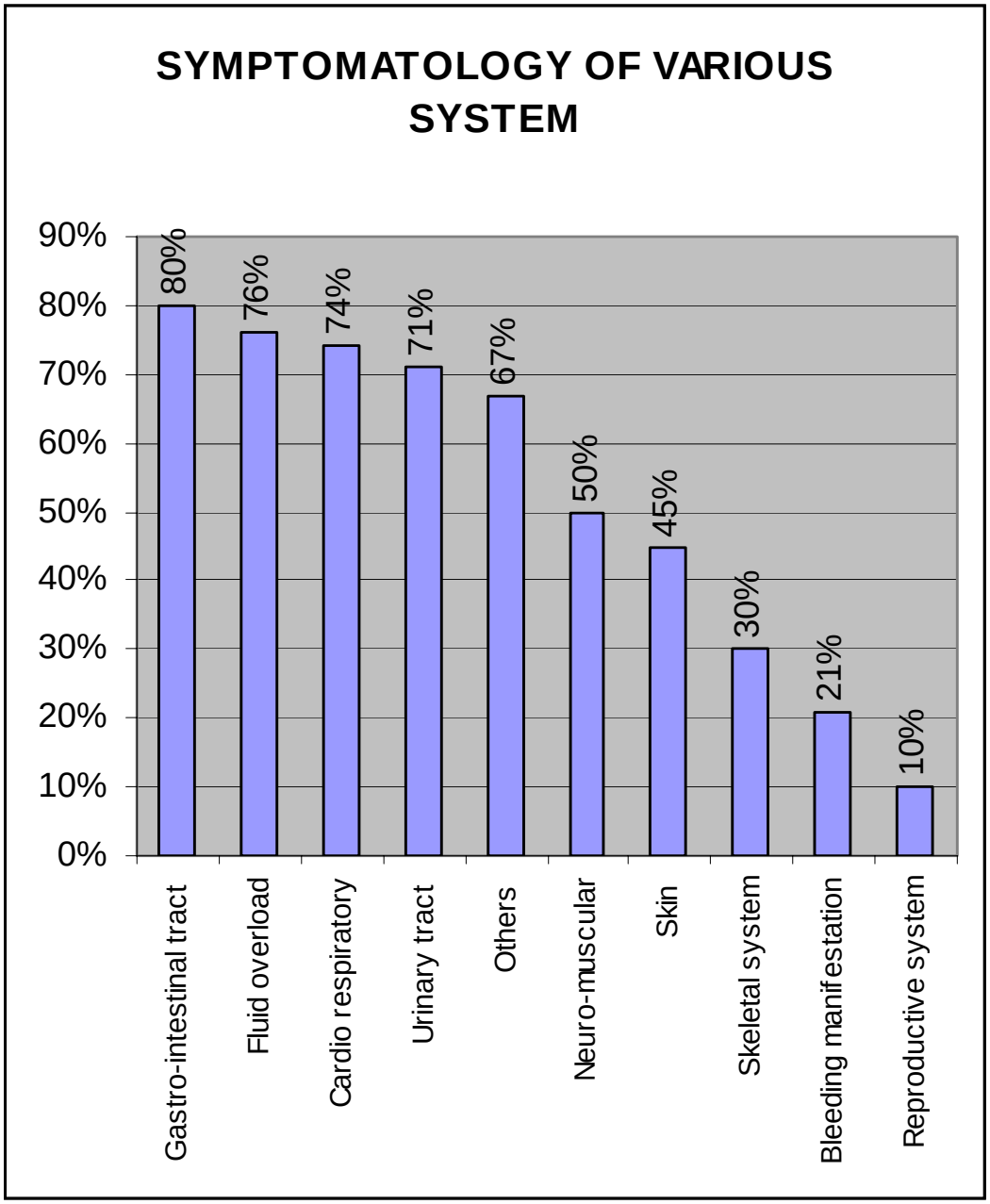
AGE	TOTAL	%
<20 yrs	2	2
20-29	2	2
30-39	4	4
40-49	22	22
50-59	36	36
60-69	30	30
70& above	4	4

From this table, the maximum no. of CRF patients is in the age group of 50-59yrs

TABLE-2 SEX DISTRIBUTION:

AGE	MALE	FEMALE
<20 yrs	2	0
20-29	2	0
30-39	4	0
40-49	16	6
50-59	24	12
60-69	30	0
70& above	2	2

From this table, the prevalence of CKD is more common in males in the 6^{th} decade and more common in females in the 5^{th} decade.



AN

ALYSIS OF SYMPTOMS:

In this study presenting symptoms were observed in the following order.

Gastro-intestinal tract symptoms `	(80%)
Symptoms of volume overload	(76%)
Cardio respiratory symptoms	(74%)
Urinary tract symptoms	(71%)
Others like (easy fatigability, lassitude, Fever, blurring of vision)	(67%)
Neurological symptoms	(50%)
Dermatological symptoms	(45%)
Musculo Skeletal symptoms	(30%)
Haematological Manifestations	(21%)
Reproductive Tract symptoms	(10%)

Presenting symptoms pertaining to each system were observed as follows:

I. Gastro intestinal symptoms:

Anorexia was present in 66%

Nausea and vomiting was present in 50%

Hiccups was present in 18%

Abdominal distention was present in 14%

Abdominal pain was present in 8%

Diarrhea was present in 4%

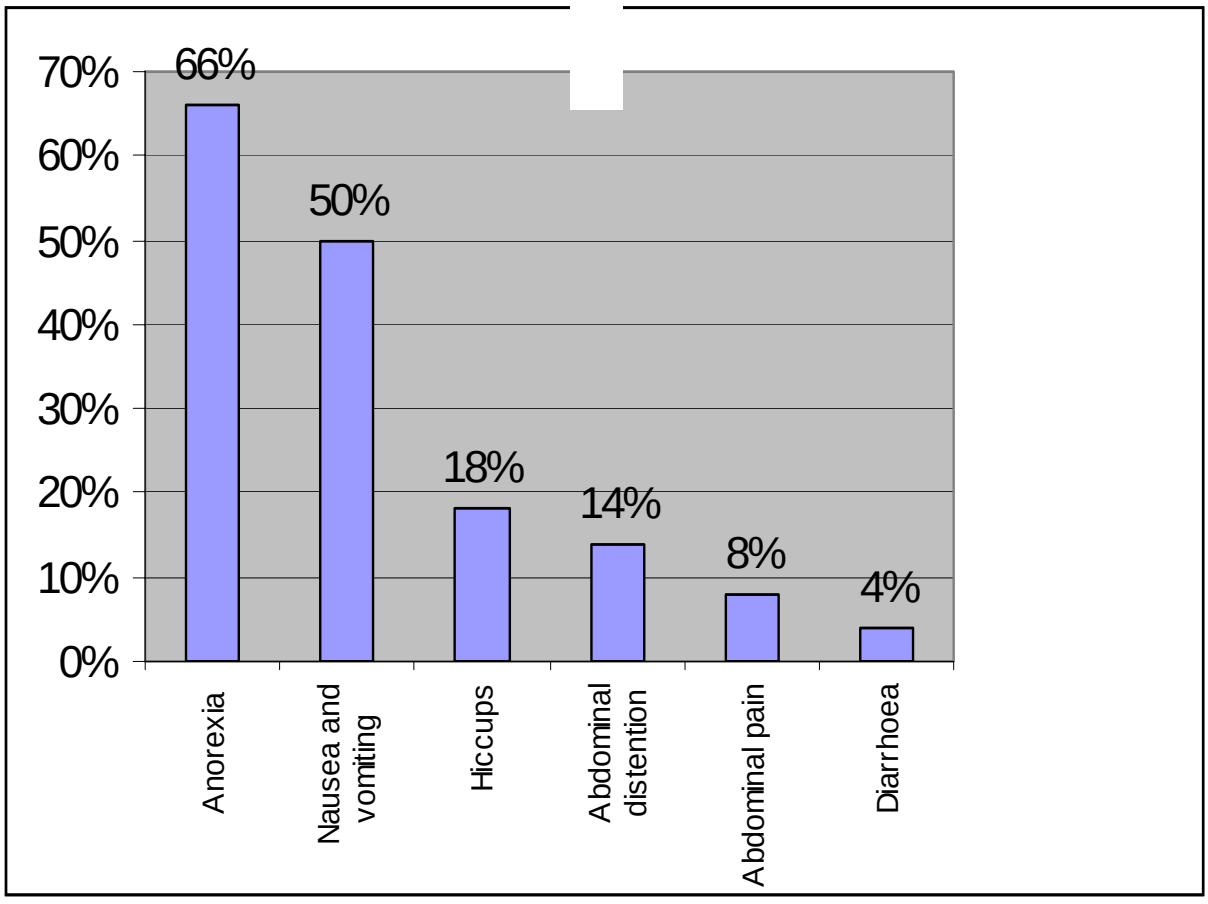


CHART SHOWS INCIDENCE OF VARIOUS SYMPTOMS OF GASTROINTESTINAL TRACT.

II. Symptoms of volume overload (76%)

Facial puffiness was present in 60%

Pedal edema was present in 55%

III cardio-respiratory symptoms (74%)

Breathlessness was present in 60%

Chest pain was present in 32%

Cough with expectoration was present in 10%

Heamoptysis was present in 4%

IV. Urinary tract symptoms (71%)

Oliguria was present in 55%

Polyuria was present in 14%

Nocturia was present in 6%

Hematuria was present in 2%

Dysuria was present in 20%

Urgency was present in 10%

Hesitancy was present in 6%

Frequency was present in 4%

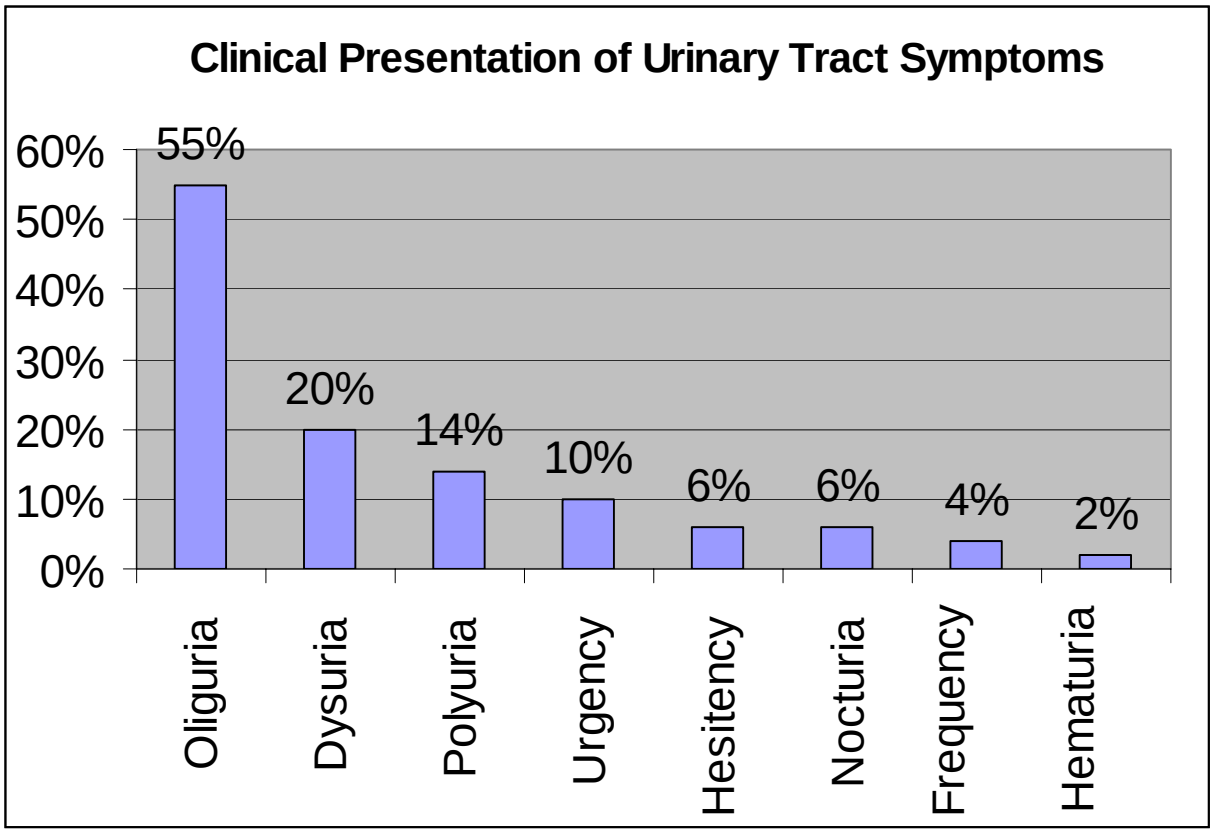


CHART SHOWS INCIDENCE OF VARIOUS SYMPTOMS OF URINARY TRACT

V Unclassified symptoms (67%),

Easy fatigability was present in 60%

Lassitude was present in 12%

Fever was present in 6%

Blurring of vision was present in 14%

VI Neurological Symptoms (50%)

Headache was present in 32%

Sleep disturbances was present in 8%

Altered sensorium was present in 8%

Motor weakness was present in2 %

Seizures was present in 6%

VII. Dermatological symptoms (45%);

Pruritus was present in 30 %

Rashes was present in 3%

Skin darkening was present in 8%

VIII Musculo skeletal symptoms (30%)

Bone and joint pain was present in 30%

IX Hematological manifestations (21%)

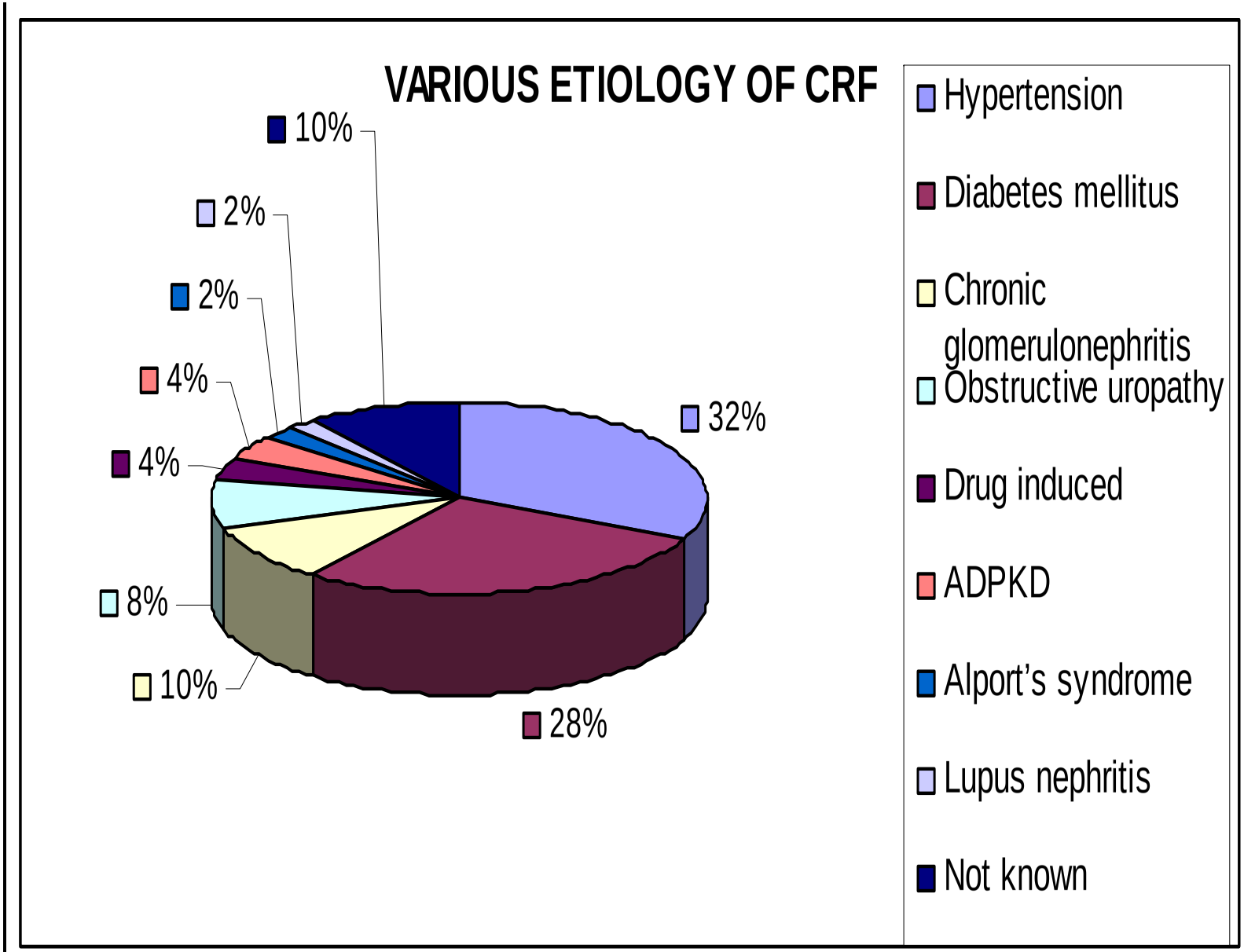
Malena was present in 16%

Epistaxis was present in 7%

X. Reproductive tract symptoms (10%)

Impotence was present in 6%

Amenorrhea was present in 4%


VARIOUS ETIOLOGY OF CRF
10%
2%
2%
4%
4%
8%
10%
28%
32%
Hypertension
Diabetes mellitus
Chronic glomerulonephritis
Obstructive uropathy
Drug induced
ADPKD
Alport's syndrome
Lupus nephritis
Not known

ETIOLOGY OF CRF PATIENTS

TABLE III:

S. NO.	ETIOLOGY	TOTAL
1	Diabetes mellitus	28
2	Hypertension	32
3	Chronic glomerulonephritis	10
4	Obstructive uropathy	8
5	Drug induced	4
6	ADPKD	4
7	Alport's syndrome	2
8	Lupus nephritis	2
9	Not known	10

This table shows various etiologies of CRF patients in this study

TABLE IV:

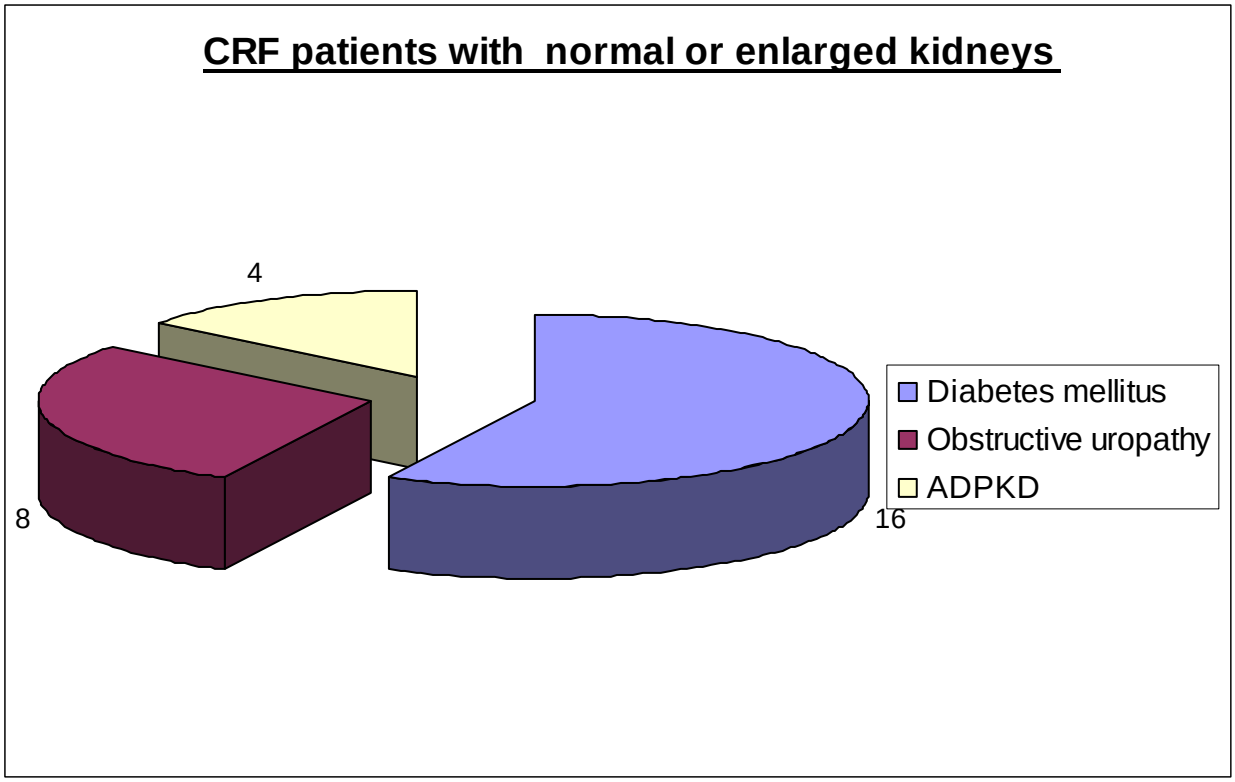
This table shows distribution of various etiologies in among different stages of CKD

SL. NO.	ETIOLOGY	STAGE 3	STAGE 4	STAGE 5
1	Diabetes mellitus	2	14	12
2	Hypertension	-	18	14
3	Chronic glomerulonephritis	1	9	-
4	Obstructive uropathy	-	4	4
5	Drug induced	-	4	-
6	ADPKD	1	3	-
7	Alport’s syndrome	-	2	-
8	Lupus nephritis	1	1	--
9	Not known	-	6	4

TABLE V:

Regarding Kidney size, 72 CRF patients had contracted kidney (<8.5cm) and the remaining 28 patients had normal or large sized kidneys in Ultrasound abdomen. Among them they are Diabetes, obstructive uropathy and ADPKD.

SL.NO.	Etiology	No. of patients
1	Diabetes mellitus	16
2	Obstructive uropathy	8
3	ADPKD	4
	Total	28



DURATION OF SYMPTOMS

Duration of symptoms in relation to stages of CKD

TABLE VI:-

Stage of CKD	< 6 months	6-24 months	> 24 months
Stage 3 (5)	4(80%)	1(20%)	-
Stage 4(61)	10(16%)	29(47%)	22(36%)
Stage 5(34)	4(11%)	10(30%)	20(59%)
Total(100)	18	40	42

In stage 3, four patients (80%) had duration of symptoms <6 months in stage 4, 29 patients (47%) had duration of symptoms 6-24months, in stage 5, 20 patients (59%) had duration of symptoms >24months

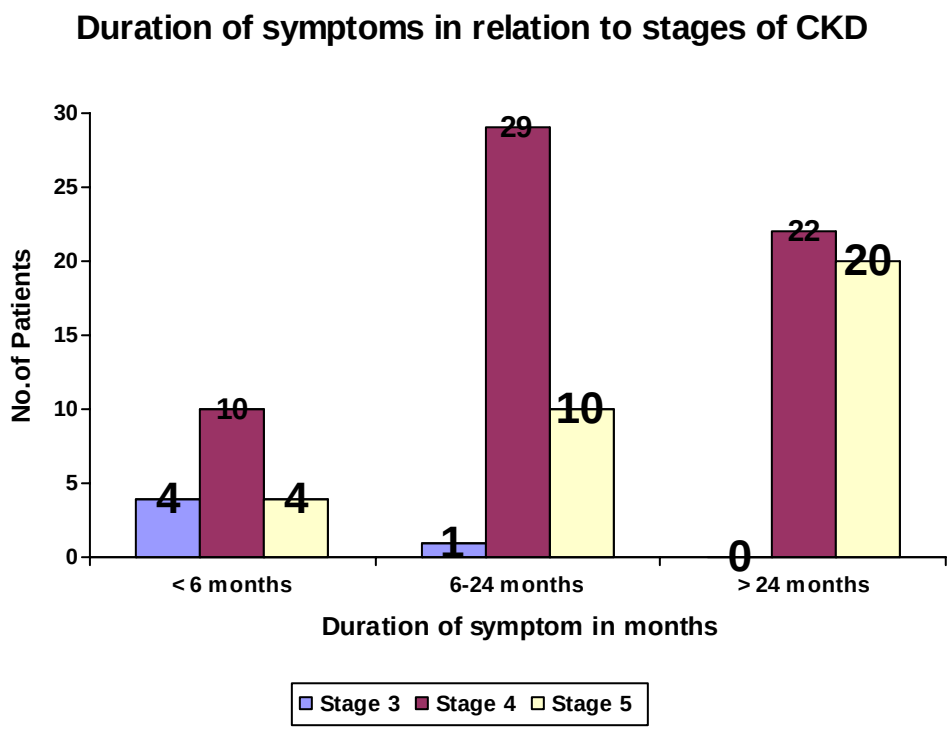


TABLE V:

Prevalence of Hypertension in CRF patients, based on systolic blood pressure

Blood pressure measurement	Systolic, mmHg	No. of patients
Normal	<120	6
Prehypertension	120–139	6
Stage 1 hypertension	140–159	34
Stage 2 hypertension	>160	50
Isolated systolic hypertension	140	4

This table shows, 6 patients were normotensive and 50% patients were in stage 2 hypertension.

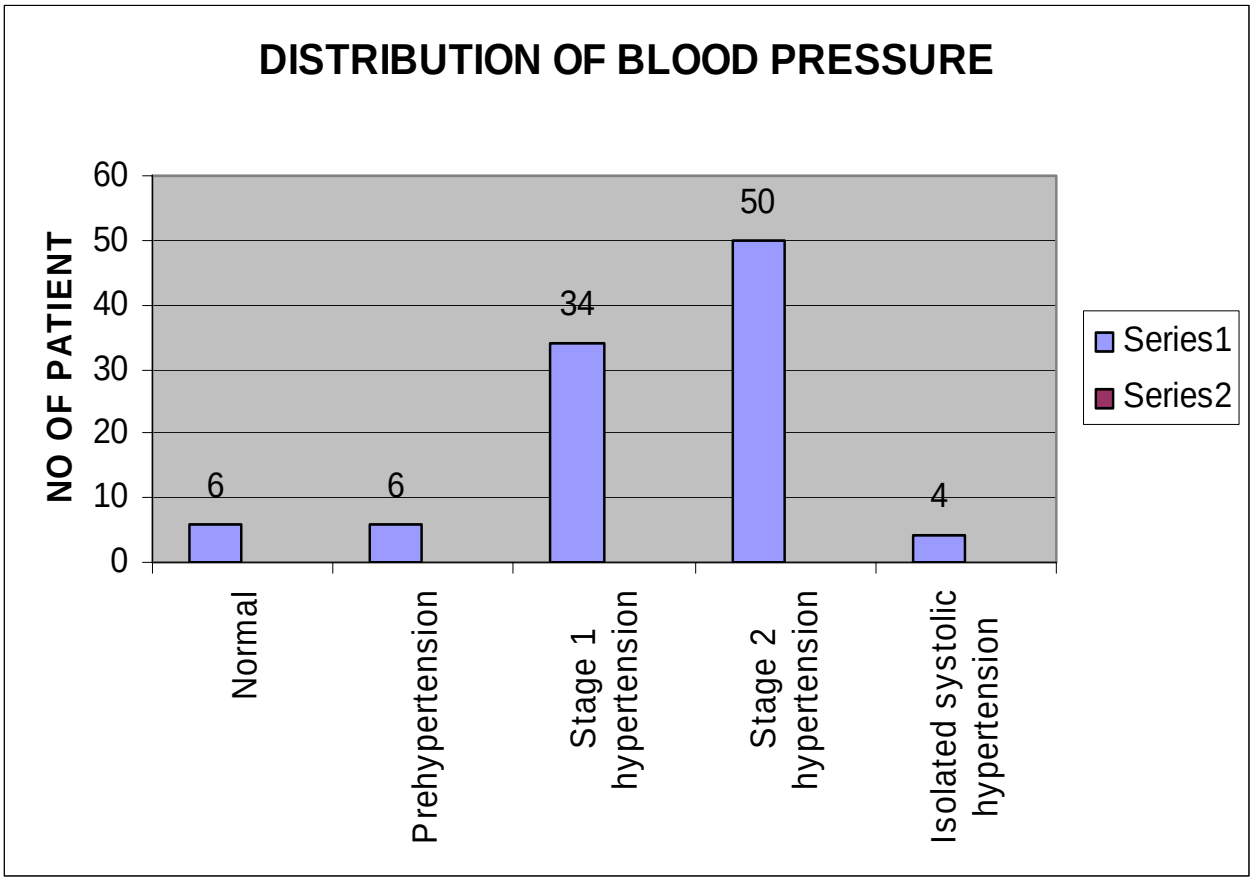


TABLE VII:-

Incidence of Retinopathy and Neuropathy

Stage of CKD	Retinopathy	Peripheral Neuropathy
Stage 3	1	-
Stage 4	10	6
Stage 5	14	4
Total	25	10

This table shows 25% of patients had Retinopathy and 10% patient had Peripheral Neuropathy, both are more common in diabetes patient..

TABLE VIII:-

Cardiac involvement in Chronic Kidney Disease

Stage of CKD	Congestive cardiac Failure	Ischemic heart disease	Pericarditis
Stage 3	1	-	1
Stage 4	13	6	3
Stage 5	16	10	8
Total	30	16	12

This table shows various incidences of CCF, CAHD and Pericarditis in CRF patients and distribution of different stages.

TABLE IX:-

Prevalence of Hyponatremia in CRF patients.

Stage of CKD	Hyponatremia
Stage 3(5)	4(80%)
Stage 4(61)	10(16%)
Stage 5(34)	14(41%)
Total(100)	28

This tables shows the prevalence of hyponatremia (serum sodium<135 mEq/dl) in various stages of CKD

TABLE X:-

Prevalence of hyperkalemia in CRF patients:

Stage of CKD	Hyperkalemia
Stage 3(5)	1(20%)
Stage 4(61)	25(41%)
Stage 5(34)	32(94%)
Total(100)	58

This tables shows the prevalence of Hyperkelemia (serum potassium>5 mEq/dl) in various stages of CKD .I n stage 5, 32 out of 34 patients (94%) had Hyperkalemia.

TABLE X:-

Prevalence of dyslipidemia in CKD

Stage of CKD	Total cholesterol >200 mg/dl	Total cholesterol <200 mg/dl
Stage 3 (5)	4	1
Stage IV(61)	22	39
Stage V(34)	14	20
Total(100)	40	60

This table shows 40% of CRF patients had hyperlipidemia.

DISCUSSION

PREVALANCE 0F CRF

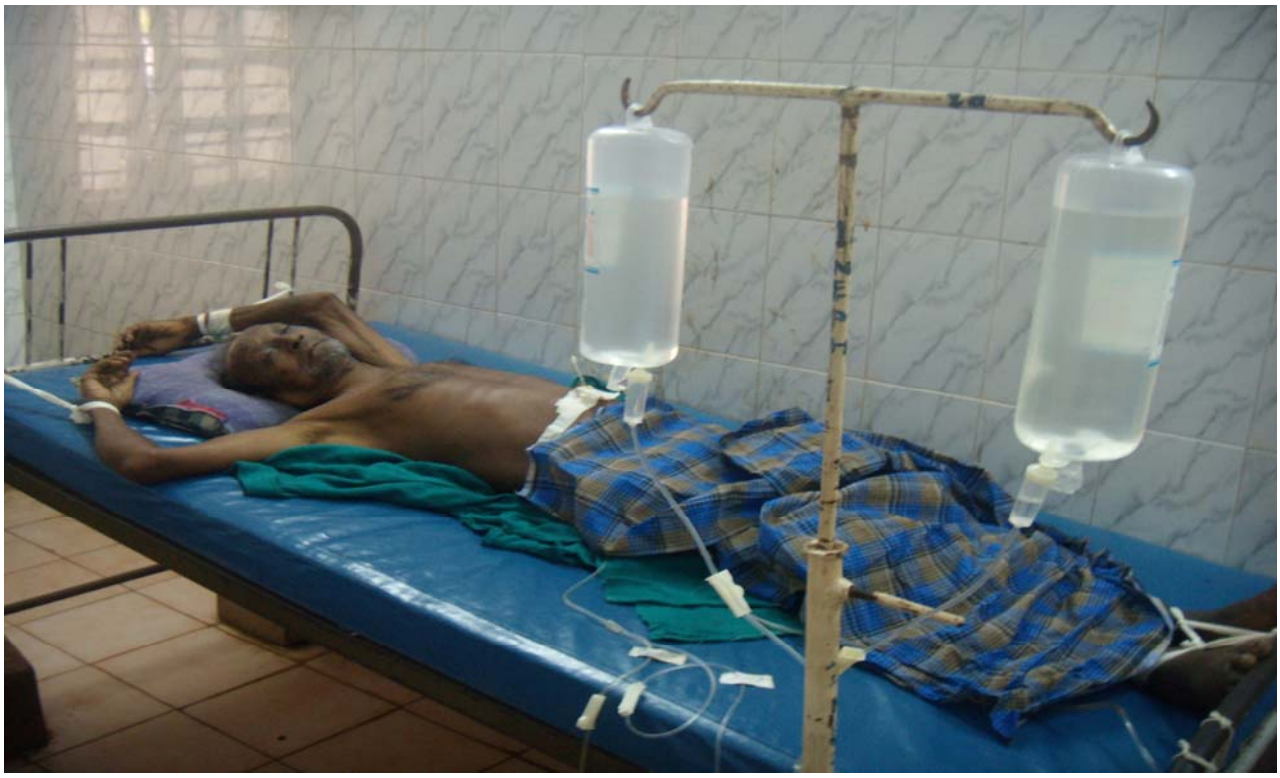
In this study, 80% were found to be males and 20% were females. ESRD is more common in males (USRDS 2001; K/DOQI 2002). The incident rate in the United States in 1999 was 380 pmp/year in males compared to 266 pmp/year in females (USRDS 2001). In this study, the maximum no. of CRF patients is in the age group of 50-59yrs (36%). The prevalence of CKD is more common in the 6th decade in males and more common in the 5th decade in females. The number of CRF patient increase with age. Eighteen major studies suggested a faster rate of decline in GFR in males and the incidence increases as age advances. (K/DOQI 2002).

ETIOLOGY OF CRF

In this study, Hypertension (32%) And Diabetes (28%) was the common causes for CRF. Other causes are chronic glomerulonephritis(10%),obstructive uropathy(8%),chronic interstitial nephritis(4%),ADPKD(2%), Alport's syndrome (2%),Lupus nephritis(2%),unknown etiology(10%).

In this study, Diabetes, Hypertension and obstructive uropathy accounting for 58% of CRF patient with compare to (USRDS 2001; UK Registry 2002), the

Incidence of hypertension (including Reno vascular hypertension), diabetes (type 2), and obstructive uropathy accounting for 40–60 per cent of patients with CKD



A CRF PATIENT UNDERGOING PERITONEAL DIALYSIS

A CRF PATIENT UNDERGOING HEMO DIALYSIS

HYPERTENTION AND DIABETES:

In this study, Hypertension (32%) and Diabetes (28%) were the causes for 60%of CRF.

Maria Eugenia Fernandes Canziani observed, main causes of CKD were:

Hypertension (33%)& Diabetes (27%). [57]

In the UK, Siegenthaler observed diabetic nephropathy constitutes 19%, & hypertension 15%.In the US diabetic nephropathy is the cause of chronic renal failure even in 45%, hypertension in 27%.In Japan diabetic nephropathy 30%, hypertension 10%, and accounting for causes. (58)

CHRONIC GLOMERULONEPHRITIS (CGN):

In this study, CGN accounting for 10% of patient. Whereas, in Japan chronic glomerulonephritis is the main reason (47%), Siegenthaler observed chronic glomerulonephrits 10%, *Maria Eugenia et al* observed glomerulonephritis (8%)

In the U S, chronic glomerulonephritis was found in 11 %.(58)

UNKNOWN:

Among the 10 patient, 2 patients were admitted for snake bite, which are asymptomatic before and found to have CRF (contracted kidney and increased echogenicity) at the time of admission. In the remaining there is no specific history or clinical manifestation to accounting for any etiology. *Maria Eugenia Fernandes Canziani.*observed 14% of patients had no etiology.

In this study, 72% patients had contracted kidneys (size <8.5 cm) and 28 % patients had normal (or) large sized kidneys. Among them 28 patients, 16 patients were Diabetes, 8 were obstructive uropathy, and 4 were ADPKD. Considering 28 patient of Diabetes, 12 patients had contracted kidneys (43%); remaining 16 patients had normal (or) large sized kidneys (57%).

CLINICAL PROFILE:

In this study, the most common presentation was, gastrointestinal tract symptoms (80%), among them anorexia was present in (66%) followed by nausea and vomiting (50%), hiccups (18%), abdominal distention (14%), abdominal pain (8%) and diarrhea (4%). Anorexia, nausea, vomiting and hiccups were resistant to medications, but improve with dialysis. Abdominal distention is mainly due to free fluid, but in 2% due to ureamic ileus.

Abdominal pain (8%) is due to gastritis, peptic ulcer and pancreatitis.

Volume overload:

Facial puffiness was present in 60% and pedal edema 55%. In all the patients, facial puffiness is more in the early morning and it is followed by pedal edema during the course of time.

Hypertension is one of the signs of volume overload. In this study, hypertension was found in 88% of patients, 6% were normotensive, and 6% were in pre hypertension. Among 88%, 34% were in stage I and 50% were in stage II.

Parfrey et al (1990) observed 7 %[59]

Wadi N. Suki et al (80%) [60] Hanne douc et al (1993) 85 %[61]

N. Mohandas et al (1999) 82 %[62]

Regarding symptoms of Cardio respiratory system (74%), Breathlessness was presented in 60%. Most of the times it is due to acidosis followed by LV dysfunction, pulmonary edema, pericardial effusion, pleural effusion, respiratory tract infection. Chest pain was present in 32% it is mainly due to CAHD, pericarditis and pleuritis.

In this study, 30 patients have CCF, 16 patients have ischemic heart disease, and 12 patients have pericarditis. Among the 16 patients with ischemic heart disease,8 patients are diabetic(50%).The main cause of death in patients with CKD is cardiovascular catastrophe and the risk to die is in ESRD patients even 10-20 times higher compared with general population [63,64].

When kidney disease progresses CKD patients become hypertensive, have acquired combined hyperlipidemia and hyperhomocysteinemia, increased oxidative stress, and decreased physical activity and psychosocial stress. If patients choose to smoke, the additive risk is profound. Diabetes mellitus is a major risk factor for both cardiovascular disease and CKD progression [65]. Finally, renal patients have a dramatic tendency for vascular and cardiac calcification that is related with hyperphosphatemia and secondary hyperparathyroidism.

History of Urinary tract symptoms were presented in 71%.In this oliguria was presented in 55%'more common in ESRD patients. Polyuria(14%) was more common symptom in diabetes.Dysuria(20%),urgency (10%) , Hesitancy(6%) were commonly presents in patients with Obstructive uropathy and urinary tract infections.

Among the Neurological symptoms (50%), Headache was the common symptom (32%).It usually relieves with adequate control of Blood pressure. Other symptoms like sleep disturbances (8%) and altered sensorium (8%) improved with dialysis. Among the 6 patient presented with seizure, 2 patients who were resistant to anti-Epileptic drugs responded with Dialysis.

With regarding to hematological manifestations (21%), History of Malena was present in 16%. Boyle. J.M et al, in 1983 has reported an incidence of 20% of Upper G I bleed in their study as evident by UGI scopy[66]

In this study, 98 patients have Hb <12 gm%, among them 76 patients have Hb between 5-10 gm%, 8 patients have Hb <5 gm%. All the 98 patients are having normocytic normochromic anemia. But among the 8 patient with Hb<5 gm%, 5 pt were having associated iron deficiency anemia (microcytic hypo chromic anemia and

S Ferritin less than normal).2 Patients with Hb >12 gm% were ADPKD. Oxford renal unit was identified, a 100% incidence of anemia of normocytic normocromic type in patients with CRF [67]

Anemia is an early and common complication of chronic kidney disease. Once end-stage kidney failure occurs, all patients are eventually affected. In the NHANES III (only 1% of participants with a glomerular filtration rate (GFR) > 60 ml/min were found to suffer from anaemia . However, in a cohort of patients with CKD, 25% of patients with a GFR >50 ml/min had Hb <12 g/dl [68].

From the available trial evidence, in CKD patients with cardiovascular disease, the benefits associated with higher Hb targets (reduced seizures) are outweighed by the harms (increased risk of hypertension and death). [69] The EBPG recommend Hb values >11 g/dl, while the K/DOQI clinical practice guidelines and clinical practice recommendations suggest Hb levels between 11 and 13 g/dl. Recent trials recommend a target Hb level between 11 and 12 g/dl in CKD patients. [70, 71] .History of both Malena and Epistaxis were present in 2 patients.

Regarding Dermatological symptoms [72], 30 out of 100 patients (30%) have pruritus.Scott Moses M.D in 2003, have reported 25% pt have pruritus in CRF. [73]

Balaskar et.al in 1993, have reported 60% incidence of pruritus in CRF patients with or without dialysis. [79]

8 out of 100 patients had hyper pigmentation due to uremia. This is felt to reflect the deposition of retained pigmented metabolites, or *urochromes*

.

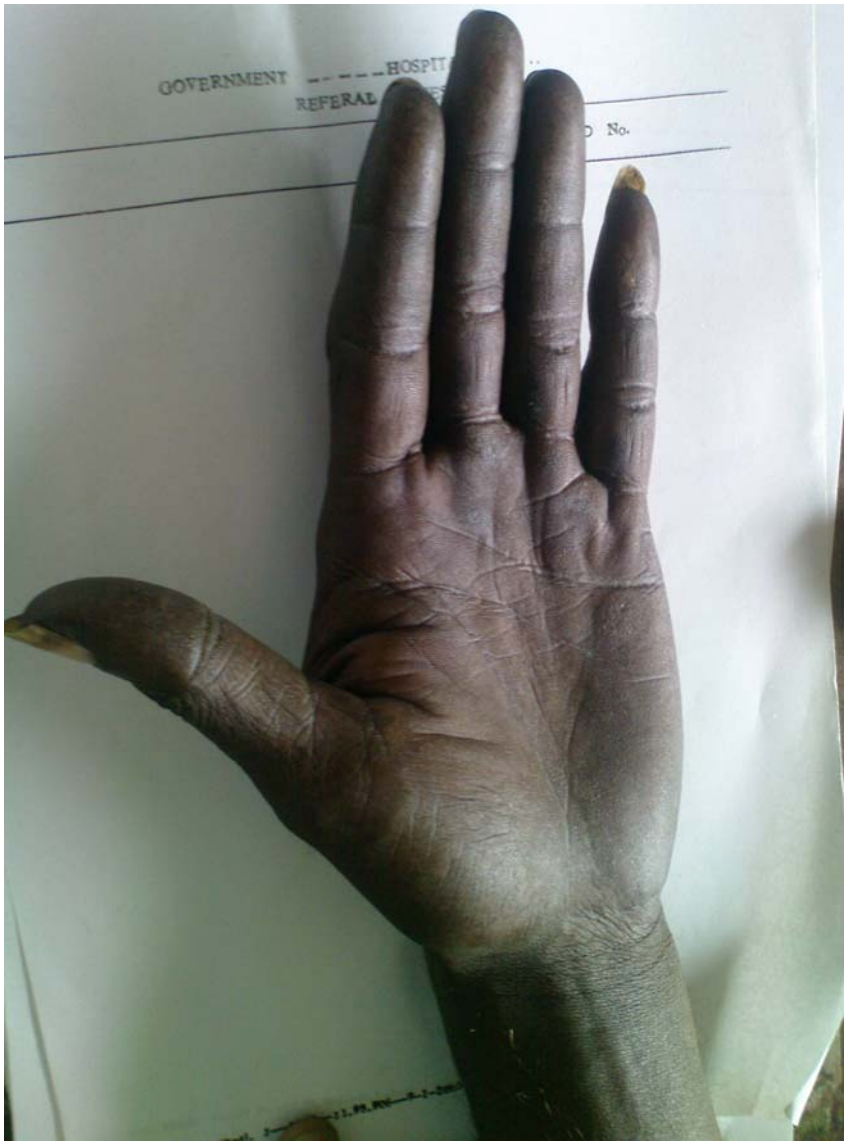


Figure shows hyper pigmentation of palm of a CRF patient

In this study, 29 out of 100 patients (29%) had Half and Half nails.

Figure shows half and half nail of a CRF patient

Agarwal S k et.al 2005 had reported an incidence of 20-50 %.[74]

Lubach et al 1982, has reported incidence of 15-50% in CRF patients with or without dialysis. [75]

Among the symptoms of skeletal system (30%), Bone and joint pain was present in 30%.Renal Osteodystrophy (ROD) was demonstrated in 8 persons.

Baurqub MA, has reported an prevalence of 55.3%in patients on maintenance Dialysis. [76]

U C odenisho showed that renal osteodystrophy, demonstrable by radiography only 3 subjects (3.35%). [77]

Adel Afifi has reported a prevalence of renal osteodystrophy ranging from 24.4% to 63 %. [78]

Regarding the duration of symptoms:

In stage 3, four patients (80%) had duration of symptoms <6 months

One patient (20%) had duration of symptoms 6-24months

No patient had duration of symptoms >24months

In stage 4,

10 patients (16%) had duration of symptoms <6 months

29 patients (47%) had duration of symptoms 6-24months

22 patients (36%) had duration of symptoms >24months

In stage 5,

4 patients (11%) had duration of symptoms <6 months

10 patients (30%) had duration of symptoms 6-24months

20 patients (59%) had duration of symptoms >24months

From this information, it is known that if the patients had more duration of symptoms, they are in more advanced stages of renal failure and vice versa.

CONCLUSION

This study on CRF shows the following,

1. The prevalence of CRF is more common in males (80%),compared to females (20%)
2. CRF occurs even in the second decade (2%) and as the age advances the incidence is also increasing and reaches peak in the fifth decade (36.6%)
3. The initial presentation of CRF is more common in stage 4 of CKD (61%) compare to stage 5 – ESRD (34%). Stage 3 was the least (5%)
4. The most common clinical presentation was Gastro intestinal symptoms (80%) compared to symptoms of volume overload (76%)
5. Among the various symptoms of CRF, anorexia was the leading one(66%)
6. Normal Blood Pressure was found in 6% of cases
7. Seizures was the only initial presentation in few cases(2%)
8. Rarely CRF is even asymptomatic (2%)
9. Anemia was observed in 98%, CCF in 30%, Pericarditis in 12% and dyslipidemia in 40% of patients.
10. Hypertension and DM were the common etiology (32%) & (28%) respectively. The remaining causes were Chronic Glomerulonephritis (10%) as well as CRF of Unknown Etiology (10%). Obstructive uropathy is found to be 8%. Drug induced CRF was 4%.
11. USG abdomen showed normal or enlarged sized kidney in 28% of cases

12. Renal scan study showed, normal or enlarged kidney in more than 50% of cases of Diabetes with CRF.
13. Duration of symptoms directly correlates with the severity of Renal failure (stage 3 to 5)
14. The best way to prevent the emergence of CRF is by strict control of the Blood Pressure in Hypertensives and Blood sugar in Diabetics.

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PROFORMA

THANJAVUR MEDICAL COLLEGE

A STUDY OF CHRONIC KIDNEY DISEASE (stage3, 4&5)

NAME	AGE	SEX
ADDRESS	OCCUPATION	INCOME
	I.P NO:	DATE:

PRESENTING COMPLAINTS **DURATION**

URINARY TRACT SYMPTOMS:

(a)Oliguria

(b)polyuria

(c)nocturia

(d)hematuria

(e)dysuria

(f)Urgency

(g)Hesitency

(h)Frequency

SYMPTOMS OF VOLUME OVERLOAD:

(a)Pedal Edema

(b)Facial Puffiness

CARDIO-RESPIRATORY SYMPTOMS:

(a)Breathlessness

(b)Chest pain

(c)Cough with expectoration.

(d)Haemoptysis.

GASTRO-INTESTINAL SYMPTOMS:

(a)Anorexia

(b)Nausea and vomiting

(c)Diarrhoea

(d)Abdominal pain.

(e)Hiccups

(f)Abdominal distention.

HAEMATOLOGICAL SYMPTOMS:

(a)Epistaxis

(b)Malena

DERMATOLOGICAL SYMPTOMS:

(a)Pruritus

(b)Rashes

(c)Skin darkening.

MUSCULO-SKELETAL SYMPTOMS:

(a)Bone and joint pain

(b)Deformities

(c)Fractures

NEUROLOGICAL SYMPTOMS:

(a)Headache

(b)Sleep disturbances

(c)Altered sensorium

(d)Paralysis

(e)Seizures

REPRODUCTIVE TRACT SYMPTOMS:

(a)Impotence

(b)Infertility

(c)Amenorrhoea

OTHERS

(a)Easy fatigability

(b)Lassitude

(c)Fever

(d)Blurring of vision

PAST HISTORY

Hypertension

Diabetes mellitus

Tuberculosis

U.T.I

Jaundice

Proteinuria

Hematuria

CAD

Drugs/chemicals

Calculi

PERSONAL HISTORY

Tobacco

Alcohol

Smoker

Analgesics

Indigenous medicines

FAMILY HISTORY

Diabetes mellitus

Hypertension

Deafness

Hematuria

GENERAL EXAMINATION

Anemia

Build

Lymph nodes

Jaundice

Edema

Skin

VITALS

Temp

Pulse

Blood pressure

JVP

Respiration

CARDIOVASCULAR SYSTEM

Heart sounds

Apex beat

Pericardial rub

Murmurs

RESPIRATORY SYSTEM

Breath sounds

Added sounds

ABDOMEN

Free fluid

Organomegaly

Renal angle tenderness

Bruit

Genitalia

NERVOUS SYSTEM

Higher mental functions

Cranial nerves

Spinal motor system

Sensory system

Reflexes

Fundus

INVESTIGATIONS

URINE ANALYSIS

Urine albumin

Urine sugar

Urine deposits (casts

24hr urinary protein

Urine c/s

CREATININE CLEARANCE (ml/min)

BIOCHEMICAL ANALYSIS

Blood urea

Serum creatinine

Serum electrolytes: Na+ k+

Blood sugar: F PP

Plasma acetone

Serum calcium

Serum cholesterol

Serum phosphorus

HAEMATOLOGICAL INVESTIGATIONS

Hb: gm% RBC'S

TC: DC:

PLATELET

PERIPHERAL SMEAR

X-RAY CHEST PA VIEW

USG ABDOMEN

ECG

ECHO

OTHER INVESTIGATIONS: