Dissertation

CHRONIC GLomerulonephritis in Adult Sudanese Patients with Chronic Renal Failure

Presented by:
Dr. Neila Mohamed Hassan Abdalla
MB, B.S.
University of Khartoum

Submitted for Partial Fulfillment of the Degree of Master of Medicine

Department of Medicine
Faculty of Medicine
University of Khartoum

July, 1990
Lovingly DEDICATED to the memory of my FATHER.
DECLARATION

I would like to declare that all the research work was done by me and I consulted all the literature included in this study.

The thesis has not been submitted to any other university and none of the information in this thesis has been published elsewhere.
ACKNOWLEDGEMENT

I would like to present my highest considerations and deepest gratitude to my supervisor, Dr. Amer I. Abboud for his valuable comments, suggestions and useful advice which made the completion of this work possible.

I acknowledge with thanks the kind assistance of the dental unit staff and record officers, in Saba University Hospital, in providing necessary records for this research. I am extremely grateful to them all.

Also I wish to thank the statistician who helped me a lot with the statistical work. Finally I appreciate the good effort made by the typist for typing, photocopying and binding this thesis.
ABSTRACT

78 patients with chronic renal failure (CRF) admitted to Soha University Hospital, Khartoum - Sudan, were studied. Chronic glomerulonephritis (CGN) was the commonest cause of CRF (65.71%) while other renal diseases (RO) causing CRF were reported as follows: pyelonephritis 5.71%, urinary calculus disease 14.29%, obstructive uropathy 1.43%, multigland disease 10.71%, hereditary disease 1.43% and uncertain aetiology 11.43%.

Males predominated in this study and the mean age of the patients was 33.01 ± 11.66. Patients with CGN were younger (21 – 35 years) than those with other RO (more than 50 years). Most of the patients studied were coming from Central and Northern Sudan possibly due to easy transport. Analysis of clinical presentation showed that anaemia was the commonest but there was no significant difference between patients with CGN and those with other RO. Urinalysis showed that proteinuria was the commonest abnormality seen but there was no difference between the CGN group and the other RO group. Most of the patients studied in the two groups had low haemoglobin (less than 10 g/dl), high erythrocyte sedimentation rate (more than 60 mm/hr), hypotenaemia (less than 135 mmHg) and normal potassium level. High blood urea and serum creatinine were found in all patients; however, biochemically as well as haematologically there was no significant difference between the two groups.

Ultrasound was found to be the best in comparison to intravenous urography in diagnosis of obstructive uropathy and parenchymal RO. Renal biopsy (RB) was done in 4 patients early in the course of their RO before going into the end-stage but none of the rest had RB when presented with end-stage renal disease (ESRD). The results of RB were as follows: 2 had membranous GN and the other 2 had membranoproliferative GN. Most of the
patients in the two groups were kept on chronic dialysis because of difficulty in doing transplant operation; however, 10% of the studied patients had renal transplant done successfully either in Sudan or abroad. The mortality rate was high in both patients with EGN (45.65%) and in those with other RD (20%).
الخلاصة

دراسة في الشايبر الكبخي والكلي المزمز بين الموظفين والممرضين

الموظفين والممرضين

استخدمت هذه الدراسة ناحية 40 موظفًا مصابين بالضيوع الكلي المزمز من مصلحة الرعاية الصحية في طنطا، والذين أجريت الدراسة على أن الباكتيريا المزمنة للعظام والكليين يشكل السبب الرئيسي للضيوع الكلي المزمز والكلوي المزمن

(25 %) مما تبين نتائج نسبة أجريت للفحص الكلي المزمز على النحو التالي: الكحولات الكبانية والكلي (41.3 %) حالات الجهاز البولي (39 %) أجريت للأمراض الإسقاطي (24.5 %) أجريت للأمراض الصدرية (20 %)

/report.pdf
كان معدل المضاحين طبيعياً، ورغبة من المرضى أيضاً أن يدعي المضاحين من أربع الجوانب الدموية والمكملات، إلا أنه لا يقدر أن هناك ترقباً دي. بلإن المجموعات سواء من المرضى الكيميائية أو الموضعة،

ولقد أثبتت هذه الدراسة أن المرضى دفع الحساب أفضل من تصوير الخلايا البولية، حيث قابلة على تطبيقات استخدام الجهاز البولي وأمراض الكبد. الرشحية، ولد أظهرت الهوية الكيميائية على أربعة من المرضى في المرحلة الأولية من داء الكلسي في أن يرى مهم إلى الكرومو الكرومي، وكانت نتيجة الخريطة الكيميائية، على النحو التالي: أحد المرضى بعلاج سرطان من الهدام الكبد والكلاسي،

فهناك، بينما يعاني المرضى الآخرين من السرطان الكلي والكلاسي، الخصائص الطبية، وتنص على علاج معظم المرضى في المجموعتين في إجراء الخصائص الجينية للعمليات التكبير الكلي على أنه قد تؤدي إجراء عمليات غرس الكلاسي في المرضى، وتحتاج السرطان على سرطان من هذين المرضى تتطلب نحو 50% حتى أن معدل المرضى

كان مرتين في كل المجموعتين من المرضى المحتملين بالهجمات الكبد والكلاسي المزمن (0.5 0).
# TABLE OF CONTENTS

**Declaration**

**Acknowledgement**

**Abstract:**
- English
- Arabic

**List of Tables and Figures**

**Chapter one:**
- Introduction
- Review of Literature

**Chapter two:**
- Patients and Methods

**Chapter three:**
- 3.1 Patients Particulars
  - 3.1.1 Age
  - 3.1.2 Sex
  - 3.1.3 Residence
- 3.2 Clinical Presentation
- 3.3 Urinalysis and Haematological and Biochemical Investigations
  - 3.3.1 urinalysis
  - 3.3.2 Hemoglobin
  - 3.3.3 Erythrocyte Sedimentation Rate
  - 3.3.4 Serum Creatinine
- 3.3.5 Blood urea
- 3.3.6 Serum Sodium
- 3.3.7 Serum Potassium
- 3.4 The Role of Chronic Glomerulonephritis in the Production of Chronic Renal Failure


Chapter four:

4.1 Patients Particulars
4.1.1 Age
4.1.2 Sex
4.1.3 Residence
4.2 Clinical Presentation
4.3 Urinalysis and Haematological and Biochemical Investigations
4.3.1 Urinalysis
4.3.2 Haemoglobin
4.3.3 Erythrocyte Sedimentation Rate
4.3.4 Serum Creatinine
4.3.5 Blood Urea
4.3.6 Serum Sodium
4.3.7 Serum Potassium
4.4 The Role of Chronic Glomerulonephritis in the Production of Chronic Renal Failure
4.5.1 Intravenous urography and Ultrasonography
4.5.2 Renal Biopsy
4.6 Management and Mortality Rate
4.7 Conclusion
4.8 Recommendations
References
Abbreviations
### LIST OF TABLES

<table>
<thead>
<tr>
<th>Table No.</th>
<th>Description</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>The age pattern of patients with CRF and a comparison between those with CDN and other RD</td>
<td>18</td>
</tr>
<tr>
<td>(2)</td>
<td>Sex pattern of patients with CRF, CDN and other RD</td>
<td>18</td>
</tr>
<tr>
<td>(3)</td>
<td>Geographical distribution of patients with CRF, CDN and other RD</td>
<td>19</td>
</tr>
<tr>
<td>(4)</td>
<td>Clinical presentation of patients with CRF and a comparison between CDN and other RD</td>
<td>21</td>
</tr>
<tr>
<td>(5)</td>
<td>Urinalysis in patients with CRF and a comparison between those with underlying CDN and other RD</td>
<td>23</td>
</tr>
<tr>
<td>(6)</td>
<td>Haemoglobin concentration in patients with CRF and in those with CDN and other RD</td>
<td>24</td>
</tr>
<tr>
<td>(7)</td>
<td>Pattern of erythrocyte sedimentation rate in patients with CRF and in those with CDN and other RD</td>
<td>25</td>
</tr>
<tr>
<td>(8)</td>
<td>Serum creatinine in patients with CRF and in those with underlying CDN and other RD</td>
<td>26</td>
</tr>
<tr>
<td>(9)</td>
<td>Blood urea level in patients with CRF and a comparison between those with CDN and other RD</td>
<td>27</td>
</tr>
<tr>
<td>(10)</td>
<td>Serum sodium concentration in patients with CRF and in those with underlying CDN and other RD</td>
<td>28</td>
</tr>
<tr>
<td>(11)</td>
<td>Serum potassium concentration in patients with CRF and in those with underlying CDN and other RD</td>
<td>29</td>
</tr>
<tr>
<td>(12)</td>
<td>Autopsy of CRF</td>
<td>31</td>
</tr>
<tr>
<td>(13)</td>
<td>Type of therapy in patients with CRF and a comparison between those with underlying CDN and other RD</td>
<td>34</td>
</tr>
</tbody>
</table>

### LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure No.</th>
<th>Description</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1)</td>
<td>Age pattern of patients with CRF, CDN and other RD</td>
<td>19</td>
</tr>
</tbody>
</table>
CHAPTER ONE

- INTRODUCTION

- REVIEW OF LITERATURE
Renal disease (RD) is a common cause of morbidity and mortality throughout the world. Patients presenting with the disease in Sudan seem to be increasing in number probably because the population is becoming more aware of the problem and tends to seek medical advice more than before. Improvement of diagnostic facilities also play a role in early detection of renal disorders.

The etiology of chronic renal disease varies widely. Some of the causes of renal failure (RF) may be reversible and non-progressive if detected early and treated promptly e.g. pyelonephritica (PN), obstructive uropathy or analgesic nephropathy. A leading cause of chronic renal failure (CRF) is chronic glomerulonephritis (GN) which accounts for one-third of patients with end-stage renal disease (ESRD) requiring dialysis or transplant (Gwyn Williams and Peters, 1981).

The syndrome of GN is characterized chiefly by persistent urinary abnormalities and by slowly progressive impairment of renal function ending into hypertension, contracted granular kidneys and ESRD (Glassock and Brenner, 1980).

The detection of (GN) usually occurs in one of several ways:

1- Incidental finding of urinary abnormalities, impaired renal function or hypertension during screening of asymptomatic population.
2- Insidious onset of progressive symptoms or signs of advanced renal disease especially hypertension and anemia.
3- Exacerbation of GN usually during the course of a non-specific viral or bacterial illness (Glassock and Brenner, 1990).
In advanced stages of the syndrome, the clinical differentiation of GN and other causes of R.F. is difficult. However, there are certain changes which might suggest the diagnosis of GN. These changes are:

1- Symmetrically contracted kidneys.
2- Moderate to heavy proteinuria.
3- Abnormal urinary sediment especially red cell casts.
4- X-ray evidence of normal pelvicalyceal systems.

Once the patient has developed CRF, the etiology has to be determined as this might help in planning future therapy e.g. if the patient has got HD which tends to recur on transplanted kidney this might contraindicate this form of therapy.

The facilities for dialysis and transplant in Sudan are few but rewarding. The main peritoneal dialysis (PD) centres are in Soba University Hospital (S.U.H.), Khartoum Teaching Hospital and Jan Sine Hospital while most of the haemodialysis (HD) is done in Khartoum Dialysis and Transplant Centre. A lot of patients had renal transplant done in S.U.H. with good outcome and have been followed regularly. Some of the patients had their transplant done abroad but have been followed up in Sudan regularly with good outcome.

Many studies were carried out about GN as an aetiological factor of CRF but few were done regarding the clinical picture, biochemical and haematological changes occurring during the course of the disease. Thus, I set out to study this important renal disease with the following objectives:

1- Type of patients involved with emphasis on their age, sex and residence.
2- The clinical picture at presentation to hospital.

3- Urinary abnormalities, haematological and biochemical changes accompanying the illness and their use in differentiating ECDN from other causes of ESRD.

4- The role of ECDN in the production of ESRD.

5- The usefulness of diagnostic tools in the diagnosis of ECDN e.g. intravenous urography (IVU), ultrasonography (US) and renal biopsy (RB).

6- Difference in management between patients with ESRD due to ECDN and those with other causes.

7- The mortality rate among patients with ESRD in relation to ECDN.
REVIEW OF LITERATURE

There are about 1.3 million glomeruli in the cortex of the human kidney. The glomerulus consists of endothelial, glomerular basement and epithelial elements and mesangial cells. Each of these has different structure and function which determine the final composition of the urine (Short and Matijev, 1986).

The nature of the glomerular barrier is both physical and electrostatic. In the diseased state, loss of charge and/or size selectivity will result in increasingly heavy proteinuria and complete loss of glomerular capillary flow or transmembrane flux will result in progressive azotemia and eventually oliguria.

The glomerulus acts as a filter and responds to injury in a limited number of ways:

1- It can leak so that abnormal amounts of normal blood constituents appear in the urine.

2- It can fail to filter effectively and cause retention of metabolites which are normally excreted (Short and Matijev, 1986).

Glomerulonephritis (GN) is a major cause of morbidity and mortality from kidney disease. Its etiology is usually unknown; although accumulation of immune complex in renal tissues appears to be responsible in most conditions. Morphological classification is limited because the glomerulus can respond to injury in only a relatively small number of ways. Experimental studies in animals showed that the same pathogenic mechanism can produce glomerular disease with a wide range of functional and histological abnormalities depending on factors which include the
duration and rate of deposition of complexes in the kidney (Drew Williams and Peters, 1983).

The incidence of CGN as a cause of CRF is increasing and it constitutes a major aetiological factor. In Sudan, CGN causes CRF in 38% (Abdou et al, 1989) whereas in European countries it affects 28.3% (Morrise, 1988) and in Ethiopia 53% (Habte and Teklu, 1980).

Normal plasma urea concentration is 15 - 50 mg/dl and that of creatinine is 0.15 - 1.4 mg/dl. If the clearance of these substances parallels the rate of glomerular filtration it is clear that when the filtration rate falls their plasma concentration will rise. Unfortunately the relation between glomerular filtration and plasma concentration is such that these concentrations are only of limited usefulness in this respect (de Vernejoul, 1973a).

The plasma concentrations of urea and creatinine depend on their rate of production and elimination. If their rate of elimination is via the glomerular filtrate and their daily production is relatively constant, then a fall in glomerular filtration rate (GFR) will cause a rise in their plasma concentration until a new equilibrium is reached. Conversely, if GFR remains constant and the rate of urea and creatinine production increases their plasma concentrations will also increase. By studying the relationship between the blood urea concentration and GFR, it can be seen that as the GFR diminishes there is at first only a small absolute rise in blood urea. Further reductions in GFR however produce large absolute changes. The implications behind this relationship are first plasma concentration of urea and creatinine show little absolute change until functionally the patient has lost one kidney and second when the GFR is low a small additional reduction in it will produce large changes in plasma...
concentration (de Wardener, 1972a).

The plasma concentration of urea is affected by dietary protein intake and endogenous protein catabolism; with low protein intake the level of blood urea may remain within the normal range though there is a substantial fall in GFR. Conversely, a high protein diet will raise the blood urea to pathological levels though the GFR is normal or unchanged (de Wardener, 1972a).

The rate of creatinine production is mainly a function of the size of the muscle mass. When patients with small muscle mass develop renal failure they have plasma creatinine which are misleadingly low (de Wardener, 1972b). However, recently it has been shown that a meal containing 300 g of meat will produce 70 - 80% rise in serum creatinine (Gabriel, 1986).

The deterioration of renal function may be assessed by measurement of GFR. The interpretation of renal function of subjects of different age and sex is difficult as there is some sex difference and decrease in renal function with age. Estimation of GFR by measurement of creatinine clearance is the method most used in clinical practice. However, it needs a precise timed urine collection and in addition urine creatinine tends to have a substantial day-to-day fluctuation because of variations in protein intake. So creatinine clearance is an inaccurate and imprecise measure of GFR in clinical practice (Elsaviers et al, 1987).

In most patients with stable GFR modest increases in total body sodium and water content can be documented although objective signs of extracellular fluid volume expansion may not be apparent clinically. With ingestion of excessive amounts of salt and water however, control of excess volume becomes an important clinical and therapeutic consideration. Excessive salt ingestion contributes to congestive heart failure, hypertension,
ascites and oedema formation. On the other hand, hypokalaemia and weight gain are the typical consequence of excessive water ingestion. Hypokalaemia is encountered relatively infrequently in CRF (Brenner and Lazarus, 1980).

Derangements in potassium balance are occasionally documented by laboratory analysis in patients with CRF and are rarely responsible for clinical symptoms unless CRF falls below 5 ml/min or an exogenous or endogenous potassium load is encountered. With advancing renal failure most patients maintain their potassium balance until final stages of uremia and this is due to the adaptive mechanisms in the renal distal tubules and colon where aldosterone and other factors enhance potassium secretion. On the other hand, oliguria and disturbance of the adaptive mechanisms can lead to hyperkalaemia and its clinical sequelae. Hypokalaemia due to diminished renal ability to conserve potassium is uncommon in CRF. The occurrence of hypokalaemia in such patients should raise the possibilities of poor dietary potassium intake, excessive use of diuretic therapy or gastrointestinal losses. Hypokalaemia may occur as a result of primary potassium wasting in urine and may represent a single renal reabsorptive defect or more commonly associated with other solute transport abnormalities like sodium in salt losing nephropathy due to interstitial nephritis or obstructive uropathy (Brenner and Michael Lazarus, 1980).

The clinical picture in CRF is variable. Patients may present with symptoms not referring to the urinary system but referring to other systems. These latter changes are due to metabolic and biochemical changes accompanying renal function deterioration.

Many types of CRF present clinically as nephrotic syndrome [AS]. The
structural and functional events which determine the appearance of excess protein in the urine are still not understood. (Cameron, 1981). However, animal studies showed that in renal-ablation model there is progressive injury to remnant glomeruli and this is reflected morphologically and by increasing proteinuria. As the GFR declines, protein excretion per nephron increases greatly. Studies with macromolecular tracers revealed that the proteinuria is due to defects in both the charge-sensitive and size-selective properties of the glomerular capillary wall (Brenner et al., 1982).

In its overt form, NS is characterized by albuminuria, hypoalbuminemia, hyperlipidemia and edema. These abnormalities are direct or indirect consequences of excessive glomerular leakage of plasma proteins into the urine. Heavy albuminuria is the hallmark of NS and urinary protein excretion in excess of 3.5 g/day is considered in the nephrotic range (Glassock and Brenner, 1980).

Patients presenting with asymptomatic urinary abnormalities are identified by the finding of proteinuria in the non-nephrotic range and/or hematuria unaccompanied by edema, reduced GFR or hypertension. These abnormalities are discovered incidentally and may be persistent or intermittent. It may represent a phase in the natural history of other glomerulopathic syndrome especially NS or GN (Glassock and Brenner, 1983).

Hypertension is observed frequently in ESRD. Fluid overload and renin-angiotensin system are the major causes of hypertension in uraemia and dialysis usually restore the blood pressure to normal levels. However, some patients despite fluid and salt restriction and ultrafiltration remain hypertensive and this is due to hyperreninemia. This group usually responds to antihypertensive drugs especially angiotensin converting enzyme.
inhibitors. However, some do not and such group require bilateral nephrectomy and their response is dramatic (Brenner and Lazarus, 1980).

Raised blood pressure contributes to nephrosclerosis and accelerated atherosclerosis with morbidity and death from cardiovascular disease. Renal function may improve after treatment of malignant hypertension but not as often after the treatment of lesser degree of raised pressure. On the other hand, very rapid reduction of the blood pressure may result in impairment of renal perfusion and temporary decline in renal function (Oliver, 1983).

Early detection of hypertension and thorough application of drugs for its control reduces the number of patients who would otherwise gradually develop progressive arteriosclerotic nephrosclerosis and thus reduce the number of deaths due to ESRD (Ning, 1977).

The anaemia of CRF is normochromic normocytic and hypoproliferative and it is not correctable with dialysis but correctable with transplant. Its main cause is lack of erythropoietin but there are several factors which contribute: blood loss from the gut and during dialysis, reduced survival of red cells and the inhibitory effect of uremic toxins on proliferating red cell progenitors (Mary Cotes, 1988).

The stimulus for erythropoietin production is renal ischaemia and the rate of its production depends on the presence of a certain quantity of renal parenchyma regardless of its secretory efficiency (de Roosamer, 1976).

Increased haemolysis in patients with CRF is a minor cause of anaemia and is due to retained uremic toxins and appears when blood urea exceeds 180 mg/dl. It has been shown that the erythrocytes are not abnormal in patients with CRF and if such cells are transfused into healthy recipients the cells appear to have a normal life span. In most dialysis patients, normal erythroid marrow can compensate for this haemolysis which causes
erythrocyte life span to range from one-third of normal to normal. In
general, increased red cell destruction appears related to the retention
of protein metabolism products and not to renal damage (Paganini, 1989).

Polyamines are organic cations and play various roles in normal cel-
lular proliferation and differentiation. They accumulate in plasma and
body fluids of patients suffering from ESRD and have been reported to
reduce the proliferation and maturation of erythroid cells. Improvement
in the anemia of CRF has been reported with the use of continuous ambu-
latory peritoneal dialysis. This suggests the existence of dialyzable
substances present in the sera of uremic patients that act as inhibitors
of erythropoiesis. These substances could be polyamines.

Several workers had demonstrated the presence of elevated levels of
polyamines in the sera of uremic patients as well as a correlation betwee
polyamine levels with the degree of uremia and anemia. By amino
acids analysis technique, elevated levels of spermidine, spermine, putre-
scine and cadaverine were found in the sera of patients with renal failu-
re. Polyamines exert an erythroid-specific inhibitory effect (Kushner
et al., 1989).

Abnormal hematostasis is a common hematological defect in CRF and is
categorized by a tendency to abnormal bleeding and bruising. Bleeding
into gastrointestinal tract, pericardial sac and intracranial vault is of
greatest concern. There are many factors which contribute to the clotting
defects in uremia and these are: decreased platelets factor III activity,
abnormal platelet aggregation and adhesiveness and impaired prothrombin
consumption (Greener and Lazarus, 1980).

There are many changes in leukocyte function and formation in uremia.
leading to increased susceptibility to infection. Lymphocytopenia and atrophy of lymphoid tissue occur in CRF whereas neutrophil production is relatively unimpaired. Chemotaxis is impaired in uraemic leukocytes resulting in impaired acute inflammatory response and decreased delayed hypersensitivity. There is a tendency for uraemic patients to have less fever in response to infection and thus infections may be more difficult to recognize (Brenner and Lazarus, 1980).

Infections frequently complicate other parenchymal renal diseases. Animal experiments have shown that the intravenous administration of large numbers of Gram-negative organisms only cause acute pyelonephritis if the ureters are temporarily occluded or the kidneys are already scarred from previous infections (de Wachter, 1975c).

The main value of an intravenous urogram (IVU) is that it demonstrates the size and configuration of the pelvis and calyces, it determines the size, shape and position of the kidneys and it suggests the possibility of parenchymal disease. Patients suffering from renal failure must not be dehydrated for this will make little difference to the concentration of the urine and it will often aggravate the renal failure. IVU might help in determining the etiology of CRF beside other investigations. ECG as a cause of CRF might be suggested from the IVU by symmetrical small kidneys and normal pelvis-calyceal system. However, a lot of renal parenchymal disorders might produce small contracted kidneys and differentiation is impossible without the help of other investigations (de Wachter, 1975d).

The use of ultrasonography (US) is firmly established as a simple non-invasive way of examining the kidney and upper urinary tract. US findings are independent of renal function and therefore can be used to study patients with renal failure. It measures with considerable accuracy the shape,
depth from the surface and internal architecture of the kidneys and upper urinary tract. It detects reliably the dilated pelvis, calyces and ureter of the obstructed kidney and displays the normal or increased size and cortical thickness of other diseases causing acute renal failure. It can differentiate those from the small kidneys with decreased cortical thickness of most chronic renal diseases thus determining whether or not renal biopsy (RB) is indicated in the diagnosis of unexplained renal failure. Also US is helpful in identifying the cause of abnormalities detected on IVD (Morrison et al., 1983).

RB is the only method of making an exact histological diagnosis during life. The information obtained from RB is of greatest value in throwing light upon the natural course of renal disease and in identifying an exact histological diagnosis (de Wardener, 1973a). RB is not without risk especially in patients with ESRD who might develop severe bleeding necessitating transfusion as they have defective coagulation. Also the kidney is small and fibrotic and thus difficult to biopsy and to interpret the histological specimen.

CCN appears to be the most important cause of CRF and this has been confirmed by RB in relatively few cases. Among those biopsied, in 22.5% of the European and 28.7% of the British cases the histological diagnosis was advanced sclerosing GN which implies that about one-third of patients had presented for diagnosis at a stage of the illness where there was little opportunity for therapy. The problem of how to detect early progressive disease in asymptomatic patients ensures that it will be difficult to reduce this large number of patients needing renal replacement therapy (Kings, 1977).
CKD is a progressive disease but any effort should be made to detect and correct any of the numerous potentially reversible aetiological factors and acute complications that may arise in such a condition. The complications include active infection, uncontrolled hypertension, volume depletion, urinary tract obstruction and/or stone formation, hyperuricaemia and the use of nephrotoxic drugs.

Conservative management aims at fluid and protein restriction. Patients with CKD are either oliguric or polyuric. The thirst mechanism in the majority of patients with ESRD is intact and so there is no need to restrict fluid intake. However, in patients with oliguria fluid restriction is essential. The amount of fluid needed per day is calculated as the urine output in the previous 24 hours plus 500 - 1500 ml which represents the daily insensible fluid loss. This minimizes the risk of fluid overload.

Protein restriction to about 40 g per day, with supplements of carbohydrates and vitamins has been used as a means for maintaining nitrogen balance in patients with other than severe uraemia. This protein restriction also reduces slightly the level of blood urea and the rate of accumulation of metabolic toxins (Brenner and Lazarus, 1980). Severe protein restriction on the other hand, may result in malnutrition. However, several semi-synthetic diets are produced and they provide free amino-acids and ketoacids. The problem with such diet is the imitable taste and that it is expensive.

When conservative measures can no longer sustain a reasonable quality of life and when CKD had progressed to the stage of oliguria, hyperkalaemia, advanced neuropathy, intractable hypertension, congestive heart failure and pericarditis, dialysis and/or transplantation becomes mandatory. Renal transplant is the treatment of choice of ESRD. However, when this cannot be done for a reason or another, chronic dialysis is an alternative therapy.
for maintaining adequate quality of life. Chronic dialysis is generally preferred in elderly patients and those with malignancies or certain systemic diseases in whom immunosuppression and chronic corticosteroid therapy may add serious risks. Successful transplantation offers the greatest likelihood of total rehabilitation and recovery for patients with ESRD since virtually all the complications and metabolic consequences of uraemia tend to be reversed completely. Rehabilitation tends not to be complete with chronic dialysis which itself is costly, time-consuming and demanding (Brenner and Lazarus, 1980).
CHAPTER TWO

- PATIENTS AND METHODS
2.1 PATIENTS AND METHODS:

100 consecutive patients admitted with renal failure to Soha University Hospital (SUH), Khartoum - Sudan, were studied. SUH which is a 140 bed general hospital is one of the two main teaching hospitals in Sudan and contains one of the three renal units in the country. The hospital receives patients with CRF referred from all other hospitals throughout Sudan. Because of difficulty in transport, most patients received in SUH are coming from Central and Northern parts of the country. So, the sample obtained for the study represents patients from all parts of Sudan with CRF possibly with some preponderance of patients from Central and Northern Sudan.

The study was carried out from September 1988 to October 1989.

The criteria for inclusion in the study were elevated blood urea (more than 100 mg/dl) and/or serum creatinine (more than 4 mg/dl) due to CRF. Patients with acute intrinsic renal or pre-renal failure were excluded from the study. The data collection included the following:

1- Patient particulars: age, sex and residence.
2- Clinical presentation to hospital e.g oliguria, hypertension, anaemia, nephrotic syndrome and asymptomatic urinary abnormalities.
3- The presence of associated diseases e.g diabetes mellitus, heredofamilial disease.
4- Urinalysis, haemoglobin, erythrocyte sedimentation rate (ESR), urea and electrolytes and serum creatinine were done in all patients.
5- IUW, US and RB when indicated.
6- Type of management each patient had including conservative
(protein and fluid restriction), dialysis and/or transplant. The mortality rates were studied also.

Of the patients admitted, 70 fulfilled the criteria. The consent of the majority of the patients was obtained but in few of them it was not possible. The study included review of patient's notes and their follow up during the study period.

Statistical analyses were used and included t - student test and \( \chi^2 \) test.
CHAPTER THREE

- RESULTS
3.3 PATIENTS PARTICULARS

3.1.1. AGE

The age of the studied patients ranged between 16 and 74 years with a mean of 35.01 ± 13.66.

Table (1) and Figure 1 shows the age pattern of patients with CHF and those due to CDA compared with other RD. It is seen that the majority of the patients with CHF were in the age group 21 - 30 years followed by those more than 60 years old.

When analyzing the age pattern of patients with CDA, the majority lie in the age group 21 - 30 years while those with other RD lie in the age group more than 40 years. For patients in the age group 16 - 30 years and 31 - 40 years, there is no difference in the pattern between patients with CDA and those with other RD.

Using x² test at probability of 5 %, there is no statistical significant difference in the age pattern of patients with CDA and those with other RD (P = 1.971).

3.1.2. SEX

The majority of the patients studied were males constituting 45 (64.29 %) out of 70. In both groups of CDA and other RD, males outnumber females (Table 2). However, when comparing both groups of CDA and other RD with regard to sex, there is no statistical significant difference between the two groups using t - student test at probability of 5 % (P = 0.72).

3.1.3. RESIDENCY

Most of the patients were coming from Central and Northern Sudan constituting 94 patients (72.4 %) out of 70. The next group was of
those coming from Western Sudan (12.06%) while patients coming from Eastern Sudan were (8.57%). Only one patient (1.43%) came from the South.

From Table (1), it is apparent that CDN was seen in all geographical areas of the country but it was more common in Central and Northern Sudan. In Eastern Sudan, other RD causing CHF were seen more than CDN. The single patient who was from Southern Sudan had CDN.

Table (1)

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>CHF (%)</th>
<th>CDN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16 - 20</td>
<td>6 (8.37)</td>
<td>4 (6.67)</td>
<td>2 (8.33)</td>
</tr>
<tr>
<td>21 - 30</td>
<td>25 (35.22)</td>
<td>18 (29.13)</td>
<td>7 (29.17)</td>
</tr>
<tr>
<td>31 - 40</td>
<td>18 (25.71)</td>
<td>12 (18.09)</td>
<td>6 (25)</td>
</tr>
<tr>
<td>&gt; 40</td>
<td>21 (30)</td>
<td>12 (18.09)</td>
<td>9 (37.5)</td>
</tr>
</tbody>
</table>

Table (2)

<table>
<thead>
<tr>
<th>Sex</th>
<th>CHF (%)</th>
<th>CDN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>45 (64.29)</td>
<td>29 (64.04)</td>
<td>16 (66.67)</td>
</tr>
<tr>
<td>Female</td>
<td>25 (35.71)</td>
<td>17 (35.96)</td>
<td>8 (33.33)</td>
</tr>
</tbody>
</table>
Table (1)

Geographical distribution of patients with CRF, CDN and other RD

<table>
<thead>
<tr>
<th>Residence</th>
<th>CRF (%)</th>
<th>CDN</th>
<th>Other RD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central and Northern</td>
<td>54 (77.14)</td>
<td>55</td>
<td>19</td>
</tr>
<tr>
<td>Western</td>
<td>9 (12.86)</td>
<td>8</td>
<td>1</td>
</tr>
<tr>
<td>Eastern</td>
<td>6 (8.57)</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Southern</td>
<td>1 (1.44)</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Figure (1)

Age pattern of patients with CRF, CDN and other RD

<table>
<thead>
<tr>
<th>Age group in years</th>
<th>CRF</th>
<th>CDN</th>
<th>Other RD</th>
</tr>
</thead>
<tbody>
<tr>
<td>16 - 20</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>21 - 30</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31 - 40</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 40</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3.2. CLINICAL PRESENTATION:

Among the clinical presenting symptoms, pallor due to anaemia was the commonest and was found in 80% of all patients with CRF (Table 4). Among the 46 patients with underlying GN, anaemia was seen in 37 patients (80.43%) while in the 24 patients with other RD it was seen in 19 patients (79.17%). It is apparent that anaemia is seen equally in both groups.

Oliguria was seen in 41 patients (61.43%) with CRF. In those with GN, it was seen in 63.04% whereas in those with other RD it was seen in 59.13%. There is no statistical significant difference between those with GN and those with other RD by using t-student test at probability of 5% (P 0.772).

Hypertension was seen in 40 patients (57.14%) with CRF. Among those with GN it occurred in 56.52% compared with 56.33% in those with other RD. Thus hypertension seems to occur equally in patients with GN and those with other RD.

Acute nephritic syndrome (ANS) was seen in 6 patients (11.42%) with CRF. 6 of these (13.04%) were in the GN group and 2 were in the other RD group (0.33%). There is no statistical significant difference between the 2 groups (P 0.160).

Asymptomatic urinary abnormalities in the form of asymptomatic proteinuria were seen in 2 patients (2.85%) with CRF. One had underlying hyperuricemia which possibly caused proteinuria due to interstitial nephritis and the other had no evidence of GN and his renal function deteriorated rapidly but none of them had RD.
Among the other clinical manifestations were: encephalopathy seen in 6 patients with CRF (8.57 %); 4 of them had CGN (8.69 %) and 2 had other RD (8.33 %) and bleeding tendency seen in 5 patients (7.14 %) with CRF; 4 of them had CGN (8.69 %) and one had other RD (4.17 %). Hypotension was seen in one patient with CRF who had underlying PN.

Table (4)

<table>
<thead>
<tr>
<th>Clinical Presentation</th>
<th>CRF (%)</th>
<th>CGN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anemia</td>
<td>56(80.6)</td>
<td>27(80.6)</td>
<td>19(79.1)</td>
</tr>
<tr>
<td>Glomeruria</td>
<td>44(61.4)</td>
<td>29(63.0)</td>
<td>14(58.3)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>40(57.1)</td>
<td>26(56.5)</td>
<td>14(58.3)</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>8(11.4)</td>
<td>6(12.0)</td>
<td>2(8.3)</td>
</tr>
<tr>
<td>Asymptomatic proteinuria</td>
<td>2(2.8)</td>
<td>0</td>
<td>2(8.3)</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>6(8.7)</td>
<td>4(8.0)</td>
<td>2(8.3)</td>
</tr>
<tr>
<td>Bleeding tendency</td>
<td>5(7.1)</td>
<td>4(8.0)</td>
<td>2(8.3)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>1(1.4)</td>
<td>0</td>
<td>1(4.1)</td>
</tr>
</tbody>
</table>
3.3. URINALYSIS AND HEMATOLOGICAL & BIOCHEMICAL INVESTIGATIONS

3.3.1. URINALYSIS

Among the 70 studied patients 2 had total anuria (2.86%) when presented to hospital and no urine sample could be sent for analysis. In these 2 patients the underlying cause of CRF was EGN.

Normal urinalysis was observed in 12 patients (17.14%) with CRF; those with underlying EGN were 8 (17.39%) while those with other RD were 4 (16.67%). Thus normal urinalysis was seen equally in both groups of patients (Table 3).

Proteinuria was the commonest urinary abnormality and it was seen in 46 patients (65.57%) with CRF. In patients with underlying EGN, it was seen in 31 patients (67.39%) while in those with other RD in 15 patients (75.83%) but there is no statistical significant difference between the two groups of EGN and other RD.

Pyuria was the next common urinary abnormality noticed and was seen in 37 patients (52.86%) with CRF. In patients with EGN it was seen in 26 patients (56.92%) and in those with other RD in 11 patients (45.83%). The presence of pyuria is not a feature of EGN but it is known that whatever the cause of damaged kidney, they are more prone to infection than normal ones.

Haematuria was seen in 16 patients (22.86%) with CRF. In those with underlying EGN, it was reported in 8 patients (17.39%) while in those with other RD in 8 patients also (33.33%) but there was no significant difference.
Urinary casts were found in 16 patients (22.86%) with CRF. 10 patients (21.74%) had underlying CCN and the remaining 6 had other RA (24%) but there was no statistically significant difference.

All urinary abnormalities seen together were found in 5 patients with CRF (7.14%). CCN was the cause of CRF in 3 patients (6.22%) while other RA was the cause in 2 patients (3.33%).

Table (5)

<table>
<thead>
<tr>
<th>Urinalysis</th>
<th>CRF (%)</th>
<th>CCN (%)</th>
<th>Other RA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>(2) (17.14)</td>
<td>(8) (17.29)</td>
<td>(4) (16.67)</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>(68) (68.57)</td>
<td>(51) (62.39)</td>
<td>(17) (70.83)</td>
</tr>
<tr>
<td>Pyuria</td>
<td>(22) (32.86)</td>
<td>(26) (32.32)</td>
<td>(11) (45.83)</td>
</tr>
<tr>
<td>Hematuria</td>
<td>(16) (22.86)</td>
<td>(6) (17.29)</td>
<td>(4) (25.33)</td>
</tr>
</tbody>
</table>

All urinary abnormalities 5 (7.14%) 2 (6.52%) 2 (8.33%)
3.3.2. HAEMOGLOBIN (Hb)

Estimation of Hb was done in all patients. 33 patients (47.14%) with CRF had a Hb less than 7 g/dl, 21 of these had CCG (65.63%) while 12 had other RD (34.37%). A Hb ranging between 7 and 10 g/dl was seen in 32 patients (43.71%) with CRF. Of these, 23 patients (36%) had underlying CCN and 9 patients (37.5%) had other RD. A Hb in excess of 10 g/dl was seen in only 5 patients (7.14%) with CRF and 2 of these had CCG (40%) and 3 had other RD (62.5%) (Table 6).

From these data it is apparent that 50% of the patients with CCG had a Hb ranging between 7 and 10 g/dl while 50% of those with other RD had a Hb of less than 7 g/dl.

By using χ² test at probability of 5%, there is no statistical significant difference between patients with CCG and those with other RD (P 0.107).

Table 6

| Haemoglobin concentration in patients with CRF and in those with CCN and other RD |
|--------------------------------|----------------|----------------|----------------|
| CRF (%) | CCG (%) | Other RD (%) |
|< 7 | 33 (47.14) | 21 (65.63) | 12 (34.37) |
| 7-10 | 32 (45.71) | 23 (50) | 9 (37.5) |
| > 10 | 5 (7.14) | 2 (4.35) | 3 (12.5) |

* χ² test at probability of 5% P ≤ 0.107
3.3.3. Erythrocyte Sedimentation Rate (ESR):  

18 patients (54.28%) with CRF had an ESR of more than 100 mm/hr. 26 patients of these had underlying CGN (56.52%) and 12 patients had other RD (50%) (Table 7).

24 patients with CRF (39.29%) had an ESR ranging between 60 and 100 mm/hr. 15 patients had underlying CGN (32.61%) whereas 9 patients had other RD (37.5%). Patients with an ESR of less than 60 mm/hr were 8 with CRF (11.43%); 5 of these had CGN (10.07%) and 3 had other RD (12.5%).

From these data it is apparent that in most patients with CRF as well as in those with CGN or other RD, the ESR is commonly above 100 mm/hr.

By using the $x^2$ test, there is no statistically significant difference between patients with CGN and those with other RD at probability of 5% ($P = 0.284$).

Table 7: Pattern of erythrocyte sedimentation rate (ESR) in patients with CRF and in those with CGN and other RD

<table>
<thead>
<tr>
<th>ESR (mm/hr.)</th>
<th>CRF (%)</th>
<th>CGN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60</td>
<td>6 (11.43)</td>
<td>5 (10.82)</td>
<td>3 (12.3)</td>
</tr>
<tr>
<td>60 - 100</td>
<td>24 (34.29)</td>
<td>15 (32.61)</td>
<td>9 (37.5)</td>
</tr>
<tr>
<td>&gt; 100</td>
<td>38 (54.28)</td>
<td>26 (56.52)</td>
<td>12 (50)</td>
</tr>
</tbody>
</table>

* $x^2$ test at probability of 5% $P = 0.284$
3.3.4. SERUM CREATININE:

33 patients with CRF (47.14%) had serum creatinine ranging between 11 and 20 mg/dl. 24 patients of these had underlying CCN (52.17%) and 9 patients had other RD (37.5%) (Table 8).

Serum creatinine ranging between 4 and 10 mg/dl was found in 24 patients with CRF (34.29%) and of these, 15 had underlying CCN (32.61%) and 9 had other RD (37.5%).

Serum creatinine in excess of 20 mg/dl was seen in 13 patients with CRF (18.57%). 7 of these had underlying CCN (15.22%) and 6 had other RD (12%).

From these data, it is evident that half of the patients with CCN had a serum creatinine ranging between 10 and 20 mg/dl whereas those with other RD had a level in excess of 20 mg/dl. The change in serum creatinine reflects the degree of severity of CRF and has no relation to the etiology of CRF.

Table (8)

<table>
<thead>
<tr>
<th>Serum Creatinine (mg/dl)</th>
<th>CRF (%)</th>
<th>CCN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 - 10</td>
<td>24 (24.29)</td>
<td>15 (32.61)</td>
<td>9 (37.5)</td>
</tr>
<tr>
<td>11 - 20</td>
<td>33 (47.16)</td>
<td>24 (52.17)</td>
<td>9 (37.5)</td>
</tr>
<tr>
<td>&gt; 20</td>
<td>13 (18.57)</td>
<td>7 (15.22)</td>
<td>6 (29)</td>
</tr>
</tbody>
</table>
3.1.5. Blood Urea:

35 patients (50%) with CRF had blood urea ranging between 100 and 250 mg/dl. 25 patients of these had underlying CDN (71.43%) while 10 had other RD (41.67%) (Table 9).

A blood urea ranging between 251 and 500 mg/dl was seen in 27 patients with CRF (46.57%) and of these, 17 patients had CDN (62.96%) and 10 had other RD (41.67%).

Only 8 patients with CRF (11.43%) had a blood urea in excess of 500 mg/dl. These with underlying CDN were 4 (8.69%) and the rest had other RD (16.67%).

It is apparent that more than 50% of the patients with CDN had a blood urea ranging between 100 and 250 mg/dl. However, change in blood urea, like serum creatinine, reflects the degree of severity of CRF rather than its aetiology.

Table (9)

| Blood urea level in patients with CRF and a comparison between those with CDN and other RD |
|---|---|---|---|
| Urea (mg/dl) | CRF (%) | CDN (%) | Other RD (%) |
| 100 - 250 | 35 (50) | 25 (54.35) | 10 (41.67) |
| 251 - 500 | 27 (38.57) | 17 (36.96) | 10 (41.67) |
| > 500 | 8 (11.43) | 4 (8.69) | 4 (16.67) |
3.3.6. SERUM SODIUM:

51 patients of the studied (72.86%) had a sodium level less than 135 mEq/l. 43% of these had underlying CCN (73.91%) while 17 patients had other RD (70.43%) (Table 10).

Serum sodium in the normal range i.e 135 - 145 mEq/l, was seen in 18 patients with CRF (25.71%) and in those with underlying CCN in 11 patients (23.91%) and in those with other RD in 7 patients (29.17%).

Only one patient had a sodium level more than 145 mEq/l and he had underlying CCN.

From these results it is clear that nearly three-quarter of the patients with CRF as well as those with underlying CCN or other RD tend to have low sodium level and this is probably secondary to water retention.

Table (10)

<table>
<thead>
<tr>
<th>Serum sodium (mEq/l)</th>
<th>CRF (%)</th>
<th>CCN (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 135</td>
<td>34 (72.86)</td>
<td>32 (73.91)</td>
<td>17 (70.43)</td>
</tr>
<tr>
<td>135 - 145</td>
<td>18 (25.71)</td>
<td>11 (23.91)</td>
<td>7 (29.17)</td>
</tr>
<tr>
<td>&gt; 145</td>
<td>1 (1.45)</td>
<td>1 (2.17)</td>
<td>0</td>
</tr>
</tbody>
</table>
3.3.7. SEUM POTASSIUM:

Serum potassium levels within normal range i.e. 3.5 - 5.5 mEq/l, was seen in 47 patients (67.14%) with CRF. 33 patients of these had underlying ECON (71.74%) while 14 patients had other RD (38.33%) (Table 11).

In patients with CRF, hyperkalaemia occurred in 17 patients (24.29%); 10 patients of these had underlying ECON (21.74%) whereas 7 patients had other RD (29.17%). On the other hand, hyperkalaemia was seen in 6 patients with CRF (8.57%). 1 patients of these had ECON (6.52%) and the rest had other RD (12.50%).

From these results, it is clear that most of the patients with ECON as well as those with other RD had a normal potassium level.

Table (11)

<table>
<thead>
<tr>
<th>Serum potassium (mEq/l)</th>
<th>CRF (%)</th>
<th>ECON (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3.5</td>
<td>17 (24.29)</td>
<td>10 (21.74)</td>
<td>7 (29.17)</td>
</tr>
<tr>
<td>3.5 - 5.5</td>
<td>47 (67.14)</td>
<td>33 (71.74)</td>
<td>14 (58.33)</td>
</tr>
<tr>
<td>&gt; 5.5</td>
<td>6 (8.57)</td>
<td>3 (6.52)</td>
<td>3 (12.50)</td>
</tr>
</tbody>
</table>
3.4. THE ROLE OF CHRONIC GLOMERULONEPHRITIS (CGN) IN THE PRODUCTION OF CHRONIC RENAL FAILURE (CRF):

Of the 70 patients studied 46 (65.71 %) had CGN. The criteria for diagnosing CGN were finding small smooth contracted kidneys in US or IVU, urinary abnormalities as RBC casts and gross albuminuria and by exclusion of other illness which might affect the kidneys.

Pyelonephritis (PN) was seen in 4 patients (5.71 %) and the diagnosis depended on finding pyuria and small scarred kidneys (Table 12).

Urinary calculus disease was seen in 3 patients (4.29 %) while obstructive uropathy due to prostatic enlargement in 1 patient (1.43 %). The diagnosis was based on radiological and ultrasonographic finding of hydronephrosis, hydronephrosis or stones and on clinical finding of prostatic enlargement.

7 patients (10 %) had systemic disease, 5 patients of these 10 had diabetes mellitus, one had rheumatoid arthritis and one had gout. The diagnosis of diabetic nephropathy was based on the duration of the disease, the presence of significant proteinuria and of diabetic retinopathy which was found in 3 patients as reported on their records. That patient with rheumatoid arthritis was diagnosed clinically and serologically with positive rheumatoid factor early on and she presented latter with ESRD.

Of the hereditary cause of CRF one patient (1.43 %) had Alport syndrome which manifested clinically with deafness and nephropathy and with a positive family history.

In 8 patients (11.43 %) the etiology of CRF could not be determined because the general condition of the patients deteriorated rapidly and they died before being fully investigated.
Table (12)

Aetiology of Chronic Renal Failure

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>No of patient (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glomerulonephritis</td>
<td>46 (65.71)</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>8 (5.71)</td>
</tr>
<tr>
<td>Urinary calculus disease</td>
<td>3 (4.29)</td>
</tr>
<tr>
<td>Obstructive uropathy</td>
<td>1 (1.43)</td>
</tr>
<tr>
<td>Multisystem disease</td>
<td>7 (10)</td>
</tr>
<tr>
<td>Hereditary disease</td>
<td>1 (1.43)</td>
</tr>
<tr>
<td>Uncertain</td>
<td>8 (11.43)</td>
</tr>
</tbody>
</table>
3.2.1. INTRAVENOUS UROGRAPHY (I.V.U) AND ULTRASONOGRAPHY (U.S.):

I.V.U was done in 19 patients only (27.14%). In the remainder it was not done because either enough information was gained from the U.S or special contraindications like fluid overload, high blood pressure or allergy to radiocontrast material were there or because the general condition of the patients deteriorated rapidly and they died before being fully investigated.

Of those who had an I.V.U done, 17 patients had small kidneys with poor function and of these, one had small kidneys with irregular outline indicating pyelonephritis while the other 16 patients had small smooth kidneys indicating glomerulonephritis. The other 2 patients had obstructive uropathy and one of these had renal stones.

U.S. of the kidneys was done in 62 patients out of 70 (88.57%) and in the rest it was not done because information was gained from I.V.U or because the general condition of the patients deteriorated rapidly and they died.

Of the 62 patients, 4 (6.45%) had obstructive uropathy and 3 of these 4 patients had renal calculi. The remaining 58 patients (93.55%) showed features of parenchymal renal disease and small-sized kidneys which could be due to GN, PN or other parenchymal disease secondary to systemic illness.

Both I.V.U and U.S were carried out in 14 patients and the findings were the same in both procedures.

3.2.2. RENAL BICPSY (RB):

Only 6 patients out of 70 had RB and this was done in the early course of the disease when these patients presented with nephritic
syndrome and normal renal function. 2 of these patients had membranous GN and the other 2 had membranoproliferative GN. The renal function of these patients deteriorated with time and later they presented with ESRD when they were included in this study.

None of the 70 patients had RB during this study because it was not without hazard e.g. bleeding or it would not affect the prognosis or management.
3.6. MANAGEMENT AND MORTALITY RATE:

Of the 70 patients with CRF, 9 (12.86%) were kept on conservative management, during the study period, in the form of fluid and protein restriction. They could manage with this form of therapy and their renal function remained within reasonable limit. When observing the effect of conservative therapy with regard to the aetiology, it was not possible to do so as this form of therapy is dependant on the degree of severity of CRF rather than the underlying aetiology.

In more than half of the patients with CRF (52.85%), the decision of dialysis had been made early and it was either peritoneal or haemodialysis. In patients with underlying CCB, 27 out of 46 (45.65%) were kept on dialysis as compared with 16 out of 24 (66.67%) patients with other underlying RD. However, there is no statistical significant difference between the two groups (Table 13).

Of the 37 patients with CRF who were on dialysis 16 died and 11 of them had underlying CCB.

17 patients (24.29%) of all studied, died before any decision on their therapy could be made and 11 of these had CGB. Thus the overall deaths were 11 patients (15.71%) while in patients with CCB was 21 (45.65%) as compared with 12 patients (50%) with other RD. However, there is no statistical significant difference between the two groups (P 0.284).

Renal transplant was done in 7 patients (10%) with CRF. Of these, 6 had underlying CCB and this constituted 17.04% of all patients with CCB as compared with 4.17% in those with other RD.
## Table (15)

Type of therapy in patients with CRF and a comparison between those with underlying CDG and other RD

<table>
<thead>
<tr>
<th>Type of therapy</th>
<th>CRF (%)</th>
<th>CDG (%)</th>
<th>Other RD (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conservative</td>
<td>9 (12.86)</td>
<td>9 (17.39)</td>
<td>1 (4.17)</td>
</tr>
<tr>
<td>Dialysis</td>
<td>37 (52.05)</td>
<td>21 (45.62)</td>
<td>16 (66.67)</td>
</tr>
<tr>
<td>Transplant</td>
<td>7 (10)</td>
<td>6 (13.04)</td>
<td>1 (4.17)</td>
</tr>
</tbody>
</table>
CHAPTER FOUR
- DISCUSSION
- CONCLUSION
- RECOMMENDATIONS
4.1. PATIENTS PARTICULARS:

4.1.1 AGE:

In this study, the age of the patients ranged between 16 and 75 years with a mean of 35.01 ± 13.66. When comparing this result with other countries, in a study of CRF in Ethiopia the average age of the patients was 42.7 years (Mahle and Teklu, 1980) while in Nigeria approximately 70% of the patients were aged under 40 years (Onyeirim and Aliekugbe, 1970).

From the results, it is clear that most of the patients with CDV lie in the age group 21 - 30 years while those with other HD were older (more than 40 years old). However, when analysing this result, there is no statistical significant difference between the two groups.

4.1.2 SEX:

In this study males outnumber females patients in both CDV and other HD group. This is probably due to the delay and difficulty in seeking medical advice in females. In Ethiopia, a study of the clinical picture of CRF was carried out in 45 patients and the number of females were 12 (Mahle and Teklu, 1980). Another study done in Nigeria, showed the same pattern of less females (48) compared with males (79) (Onyeirim and Akinkugbe, 1970).

From these data, the small size of female patients compared to males might represent the social background in the third world where females spend most of their day looking after household jobs and looking after the children neglecting their health and only seek medical advice when they are seriously ill.
4.1.3. RESIDENCE:

Most of the patients studied were coming from Central and Northern Sudan (77.14%). This is similar to what had been reported before in Sudan by Abboud et al. (1989).

CDV is seen in all geographical areas of the country but is more common in Central and Northern Sudan. However, other RD are seen more in Eastern Sudan.

There was only one patient coming from Southern area of the country and he had underlying CDV. This is probably due to difficulty in transport resulting in delayed arrival of the patient to specialized hospital.
4.2. CLINICAL PRESENTATION:

Pallor due to anaemia at presentation was the commonest clinical sign seen being found in 80% of patients with CRF. It was seen equally in the CGN group (80.43%) and in the other HD group (79.17%). When comparing this result with other studies, in Ethiopians anaemia is reported in 89% (Habtu and Teklu, 1980) which is similar to this study. In Nigerian 62 out of 127 patients (48.81%) had anaemia and this is lower than reported in this study (Oyediran and Akinkugbe, 1970).

Oliguria was seen in 61.41% of those with CRF but there was no significant difference between the two groups of CGN and other HD. In Ethiopians with CRF, oliguria was reported in 66% which is more or less similar to this study (Habtu and Teklu, 1980).

Hypertension was seen equally in the two groups of patients in this study. Comparing the figure (57.14%) reported in the patients with CRF in this study with other studies, in Nigerians hypertension was seen in 62% which is approaching this study (Oyediran and Akinkugbe, 1970). In Ethiopians 101 out of 127 patients with CRF (79.53%) had hypertension and this is higher than this study (Habtu and Teklu, 1980).

Nephrotic syndrome (NS) was reported in 6 patients with CGN. 4 of these had NS with normal renal function in their early life and 8B was done in all of them and showed evidence of primary GN. The other 2 presented with NS and impaired renal function from the start and progressed to ESRD before RBS could be done. In many children with the NS in West Africa CGN has been diagnosed following RBS. Oyediran and Akinkugbe (1970) had shown that the undue high incidence of NS in children and young adults appeared to correlate with the high percentage of CGN obtained from autopsy results carried out in 84 patients dying with CRF; of these
84 patients 52 had CGN.

Asymptomatic proteinuria was reported in 2 patients with other RD. One had underlying hyperuricaemia which possibly caused proteinuria due to interstitial nephritis and the other had no evidence of CGN. Persistent non-nephrotic proteinuria, but in excess of 2 g per day, demonstrable in both orthostatic and recumbent positions generally indicates the presence of some significant and readily demonstrable glomerular pathology. Some of the patients might prove to have a clinically unsuspected disease e.g. amyloidosis or diabetes mellitus. In the others the lesions are usually trivial and non-specific and the long-term significance is quite uncertain. In the case of primary glomerular disease as long as the proteinuria remained modest, the prognosis would be excellent and the deterioration in renal function will be uncommon. Biopsy is commonly done in patients with persistent and isolated proteinuria as a means of accurate estimation of long-term prognosis (Glassock and Brenner, 1985).

Other clinical presentation were seen with less frequency in the two groups of CGN and other RD and there is no statistical significant difference between them. The pathogenesis of nephropathy was variable: high blood pressure, high blood urea or electrolyte and calcium imbalance. Bleeding tendency in the form of epistaxis, rectal bleeding or others was possibly due to platelets defects or anticoagulation with haemodialysis.
4.3. URINALYSIS AND HISTOLOGICAL AND BIOCHEMICAL INVESTIGATIONS:

2.3.1. URINALYSIS:

Proteinuria is the commonest urinary abnormality seen in patients with CRF but there is no difference between those with EGN and other RD. The events which determine the appearance of excess protein in the urine are still under speculation; however, animal studies showed that in renal-ablation model, there is progressive injury to remnant glomeruli and this is reflected morphologically and by increasing proteinuria. As the GFR declines, protein excretion per nephron increases greatly. These changes result from defects in charge-selective and size-selective properties of the glomerular capillary wall (Brenner et al., 1989).

Pyuria is seen in more than half of the patients with EGN but pyuria is not a feature of CRF. It is known that damaged kidneys due to any cause are more prone to infections than normal ones.

Hematuria is reported equally in both patients with EGN and other RD and the presence of red cells in the urine is not specific for EGN as it is seen in other diseases e.g. malignant hypertension and polycystic kidney disease. However, you can tell whether hematuria is of glomerular origin or arising from other renal tissues by using phase-contrast microscopy but this is not available in Sudan. Many granular and hyaline casts are present in renal disease with predominant glomerular involvement. The finding of broad casts in the urinary sediment is specific for CRF; the wide diameter of such casts reflects the compensatory dilatation and hypertrophy of surviving nephrons (Doe and Brenner, 1980).

Normal urinalysis was reported in 12 patients (17.14%) with CRF and 8 of these had underlying EGN. Urinalysis may show little or no abnormalities though nephron destruction has progressed to the stage of
CRF; but this is seen commonly in chronic obstructive uropathy, polycystic and medullary cystic diseases, analgesic nephropathy and the inactive end stage of any chronic tubulointerstitial nephropathy (Coe and Brenner, 1980).

4.4.3. **HAEMOGLOBIN (Hb):**

50% of the patients with CRF had a Hb level ranging between 7 and 10 g/dl. The anaemia is usually due to deficient erythropoietin. Some patients with CRF may have a reasonable Hb concentration and those have been form of the disease and might have normal or high level of erythropoietin due to associated polycystic kidneys or hydronephrosis.

A low Hb concentration is well tolerated by the patients of CRF because of the right shift of the oxygen dissociation curve related to the high concentrations of 2,3-diphosphoglycerate and phosphates in the red cells.

4.4.3. **ERYTHROCYTE SEDIMENTATION RATE (ESR):**

The majority of the patients with CRF as well as those with underlying CNV and other HD tend to have an ESR which is more than 50 mm/hr. This high ESR is possibly due to multiple factors; anaemia, presence of intercurrent infection or fluid retention causing haemodilution.

4.4.4. **SERUM CREATININE:**

Patients with CRF tend to have a serum creatinine ranging between 11 and 20 mg/dl while those with other HD have a level ranging between 8 and 20 mg/dl. However, the changes in serum creatinine is related to the degree of severity of CRF rather than its etiology.
The plasma concentration of creatinine depends on the rate of production and elimination. The rate of production is mainly a function of the size of the muscle mass (de Wardener, 1973a) but it is also affected by high intake of dietary protein as it was shown that a diet containing 400 g of meat will raise the serum creatinine by 70 - 80 % (Gabriel, 1986). The elimination of creatinine depends on glomerular filtration rate (GFR) and as this declines there is at first a small absolute rise in serum creatinine concentration but further reduction produces absolute change. Thus plasma concentration of creatinine shows little change until functionally the patient has lost more than one kidney (de Wardener, 1973a).

4.5.5. Blood urea:

The majority of the patients with CRF have a blood urea ranging between 100 - 250 mg/dl while most of those with other RD have a level ranging between 100 - 400 mg/dl; however the change in blood urea concentration is related to the degree of severity of CRF rather than its etiology.

The plasma concentration of urea is affected by dietary protein intake and endogenous protein catabolism. The protein intake affects directly the level of blood urea; thus a high protein diet will raise the blood urea to pathological levels though the GFR is normal or unchanged (de Wardener, 1970a).

4.4.6. Serum sodium:

The majority of patients with CRF as well as those with underlying CGN and other RD, have a sodium level which is less than 135 mEq/l and this is possibly due to fluid retention and accommodation. Hypernutrionism is seen only in one patient who has CGN.
It has been suggested that a reticulo-endothelial hormone plays a role in sodium chloride transport across the mammalian renal tubule. The obligatory high rate of solute excretion per surviving nephron, i.e. osmotic diuresis due to urea and other retained solutes, may contribute to enhancing fractional sodium chloride excretion (Brenner and Hostetter, 1980).

9.3.7. SERUM POTASSIUM:

Most of the studied patients have a serum potassium level ranging between 3.5 – 5.5 mEq/l.

Potassium imbalance is rarely responsible for clinical symptoms unless GFR falls below 5 ml/min or an exogenous or endogenous potassium load is encountered. With advancing renal failure, potassium balance is maintained until final stages of uraemia; this is due to the adaptive mechanisms in the renal distal tubules and colon where aldosterone and other factors enhance potassium excretion. Hyperkalaemia, on the other hand, occurs with disturbance of the adaptive mechanism and with oliguria (Brenner and Michael Lazarus, 1980).
4.4. THE ROLE OF CHRONIC GLomerulonephritis (CGN) IN THE
PRODUCTION OF CHRONIC RENAL FAILURE (CRF):

This study has shown that CGN is the commonest cause of CRF in
Sudan (61.71%). In a previous Sudanese study carried by Aboua et al
(1989) CGN constituted 48%. Comparing the figure quoted in this study
with others, in Europeans CGN is the commonest cause of CRF and is re-
ported in 28.3% (Morris, 1988). In a study done in Ibadan, Nigeria (1964 -
1966), 52 patients out of 84 (62.3%) had CRF due to GN (Uvediran and
Akinkugbe, 1970) and this figure approximates that quoted in this study.
In Ethiopia, GN as a cause of CRF was reported in 53.3% (Habto and
Ieklu, 1980).

From these data it is apparent that in Sudan, the number of patients
with CRF due to CGN is increasing.

Multisystem diseases as a cause of CRF came as the second commonest
cause and constituted 10%. 50% of these patients were diabetics and
from this result diabetes mellitus as a cause of CRF was seen in 2%-
compared with 9% in a previous Sudanese study (Aboua et al, 1989), 10.2% in
Europeans (Morris, 1988) and 11.5% in Ethiopians (Habto and Ieklu, 1980).

PN came as the third common cause of CRF in this study (9.71%) and
this is lower than a figure reported before in Sudan (7%) (Aboua et al,
1989). Comparing the result of this study with other countries, PN was
seen in 17.1% of Europeans (Morris, 1988), in 28.37% of Nigerians (Uvedi-
iran and Akinkugbe, 1970) and in 6.6% of Ethiopians (Habto and Ieklu,
1980).

Urinary calculi was seen in 4.28% compared with 12% and obstructive
urethrapathy not due to calculi in 1.93% compared with 2% in previous
Sudanese study (Abboud et al, 1989). From these data, it seems that the percentage of patients with CRF due to urinary calculi is decreasing while that due to obstructive uropathy was more or less the same.

Hereditary renal diseases were reported in 7.42% compared with 2.3% in Europeans (Morris, 1988).

The percentage of patients with ESRD due to uncertain etiology was 41.42% compared with 20% as a previous Sudanese figure (Abboud et al, 1989). This drop in the percentage in Sudan is probably due to the increasing availability of the diagnostic aids which helped in determining the etiology of ESRD.
4.5.1. INTRAVENOUS UROGRAPHY AND ULTRASONOGRAPHY:

Kidney imaging in renal failure aims to show renal size since small kidneys indicate irreversible CRF and to demonstrate or exclude pelvic-ureteral system dilatation which commonly indicates obstruction. For the past 15 years the high-dose urogram has been the investigation of choice but recent studies indicate that US and plain films of the renal tract may be used safely instead (Webb and Britton, 1988).

In the studied patients, 19 had IVU. Of these, 16 patients had radiological features of GN, one of PK and 2 of obstructive uropathy.

US was done in 62 patients and 56 (91.93%) of these had features of parenchymal renal disease but it was not possible to classify the aetiology of CRF from the US. In case of obstructive uropathy the US was more accurate as it showed dilated pelvocalyceal system in the 3 patients whose CRF was due to obstructive uropathy.

In renal failure, US is a sensitive detector of pelvocalyceal dilatation even when it is minor. In all cases at least a plain film should be obtained. Minor degree of collecting system dilatation may occur in severe renal failure. Since false positive scans also may show minor collecting system dilatation, all such patients should be further investigated by high-dose urography or computed tomography (CT) (Webb and Britton, 1988).

4.5.2. RENAL BIOPSY (RB):

When analysing the results of RB in this study, it was unsatisfactory as RB had been done in only 4 patients out of the 70 (5.71%) and it was done early in the course of the disease when patients had normal renal function. None of the studied group had RB done when they presented with CRF because it was either not without a hazard or the patient might bleed
or it did not alter the treatment or the prognosis of the kidneys were small, fibrotic and difficult to biopsy.

The histological findings in the 5 RBs showed that 2 patients had membranoproliferative GN and the other 2 had membranous GN. Comparing this result with previous Sudanese study (Abbud et al., 1989), 13 patients (34.21%) out of 39 with GN had RB done. The histological findings were: 6 had diffuse proliferative GN, 2 had membranoproliferative GN, 2 membranous GN, 1 total segmental sclerosis and 2 were unclassifiable because of advanced sclerosis.

In a study carried out in Nigeria, autopsy was performed in 81 cases died with CRF and the renal histological diagnoses showed that 52 patients had GN, 24 patients had PN, 4 patients had nephrosclerosis and 4 patients had other findings. The kidneys of patients with CRI were almost always small and granular. On microscopy, the glomerular involvement varied widely. Tubular damage, interstitial fibrosis and lymphocyte infiltration were often present with the occasional fibrinoid change in the afferent arterioles and glomeruli (Iyediran and Akinkugbe, 1972). 

Wing (1977) had shown that GN appeared to be the most important cause of ESRD. However, this had been confirmed histologically in relatively few cases.

From these data it is evident that RB contributed little to the diagnosis of CRI as a cause of CRF.
4.6. MANAGEMENT AND MORTALITY RATE:

Of the 70 patients with CRF, 9 were kept on fluid and protein restriction during the study period and their renal function remained within stable range. 8 of these had underlying CDK. Wing and Hutt (1977) showed that the staple diet of tropical countries consisted of vegetable foods and contained little protein. This had some implications in the clinical management of patients with renal failure. Firstly, because of low-protein intake, a rise in blood urea was a late indication of advanced renal disease. Secondly, the palliative treatment of CRF with a low-protein diet instituted later and it had less burden but one should ensure high-caloric intake. Thirdly, these tropical diets produced less acid than a meat-containing diet and the urine was frequently alkaline in patients in the lower socioeconomic groups. This might have some relation to the apparent absence of azotemia, malnutrition despite the large number of patients with chronic renal disease.

Severe protein restriction might result in malnutrition. This had been overcome by producing a semi-synthetic diet rich in free amino acids and ketone bodies. The advantages of such diet that it minimizes the uremic toxicity and improves nutrition and may even offer an alternative to some patients who would otherwise need dialysis. The disadvantage is that it is imitable and expensive (Wing, 1977).

In the studied patients, more than half were kept on dialysis either peritoneal or haemodialysis. Of these, 21 patients had CDK while 16 had other RD. Patients who were on peritoneal dialysis had indwelling catheters and the problems in such patients were the development of peritonitis and catheter blockage. Patients on haemodialysis had either arterio-venous shunt or
Fistula and they did regular dialysis in Khartoum haemodialysis and transplant centre. The problems in such patients were bleeding from or blockage of the shunt or fistula. Also they tended to lose blood in the artificial kidney and this might lead to anaemia and hypovolaemia and the patients might not tolerate the dialysis.

Renal transplant was successfully done in 7 patients with CRF and 6 of these had CCN. These patients were kept initially on dialysis for some years before the operation and this delay was due to the shortage of facilities for regular renal transplant operation in Sudan. Most of the patients had renal transplant done in Kuwait according to treatment protocol between the two countries.

17 patients with CRF died before any decision on their therapy could be made. Of the 37 patients who were kept on dialysis, 16 died. Thus the overall deaths were 33 patients (47.14%) while in those with CCN was 21 patients (46.85%) as compared with 12 patients (30%) with other RD.
4.7. CONCLUSION:

CDN is the commonest cause of CRF and most of the patients with CDN are within the age group 21 - 30 years unlike those with other RD who are older (more than 60 years). Males are affected more than females. The majority of the patients studied are coming from Central and Northern Sudan possibly due to the availability of transport and early arrival to specialized hospitals.

Clinical assessment shows no difference in the frequency of symptoms or signs in those with CDN and those with other RD and there is no specific symptom or sign which point to the possible etiology of CRF.

Urinalysis shows that proteinuria is the commonest abnormality, but there is no significant difference between the CDN group and the other RD group.

Haemoglobin estimation shows that the majority of the patients have a haemoglobin level which is less than 10 g/dl and the erythrocyte sedimentation rate tends to be high (more than 60 mm/hr.).

Blood urea ranging between 100 - 250 mg/dl is seen in most of the patients with CDN while higher levels are seen more in those with other RD. The serum creatinine is elevated in all patients and the changes in serum creatinine as well as in blood urea reflect the degree of severity of CRF rather than its etiology.

Hypokalaemia and normal potassium level are the most common electrolyte findings.

US is preferable to IVP as it is a non-invasive procedure with less risk to the patient and gives information about the size of the kidneys, the structural changes and the pelvicalyeal system.
High-dose infusion IVU without the availability of tomography will show the nephrogram only and thus only the kidney size will be determined.

RB is not helpful in establishing the diagnosis in ESRD as the histological changes (glomerulosclerosis) will be the same irrespective of the etiology. So it is helpful if it has been done early on in the course of the illness.

Renal transplant is the treatment of choice of ESRD but it was done only in a small group of our patients (10%) because of difficulties and lack of facilities in doing the operation in Sudan. So chronic dialysis remains the treatment of choice.

The mortality rate is high in all patients with CKD as well as in those with underlying ESRD and other BD.
4.6. RECOMMENDATIONS:

The population should be aware of the problem of renal disease and its chronic sequelae through health education and should be advised to seek medical care early on when they have symptoms referring to the urinary system.

Early detection and correction of any reversible causes of renal failure, e.g. severe hypovolemia, recurrent infections or obstructive uropathy, will arrest the progression of the disease.

Improvement and availability of diagnostic facilities will allow early and proper diagnosis. Urinalysis should be done routinely in any patient. Since some patients with ESRD have normal urinalysis, blood urea should be the second important investigation which have to be done routinely in patients with symptoms referring to the urinary system.

IVU is time consuming, costly, has some hazard to the patient and is less informative with regard to structural and pelvicalyceal changes. US, unlike IVU, is simple, non-invasive procedure which give more information regarding the size, structural changes and pelvicalyceal system. Thus, US should replace the IVU as an imaging procedure.

RG should be done in the early stage of RD when indicated, because as the disease progresses to ESRD, it will not give valuable information about the etiology.

The presence of specialized renal clinics should be started. This will allow close followup of the patients with RD and early detection of any deterioration in the renal function; thus renal transplant could be done early without exposing the patients to the risks of chronic dialysis which is costly at long-term.
REFERENCES


**ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>CGN</td>
<td>Chronic glomerulonephritis</td>
</tr>
<tr>
<td>CRF</td>
<td>Chronic renal failure</td>
</tr>
<tr>
<td>ESR</td>
<td>Erythrocyte sedimentation rate</td>
</tr>
<tr>
<td>ESRD</td>
<td>End-stage renal disease</td>
</tr>
<tr>
<td>GFR</td>
<td>Glomerular filtration rate</td>
</tr>
<tr>
<td>Hb</td>
<td>Haemoglobin</td>
</tr>
<tr>
<td>HD</td>
<td>Haemodialysis</td>
</tr>
<tr>
<td>IVU</td>
<td>Intravenous urography</td>
</tr>
<tr>
<td>NS</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>PD</td>
<td>Peritoneal dialysis</td>
</tr>
<tr>
<td>PN</td>
<td>Pyelonephritis</td>
</tr>
<tr>
<td>RB</td>
<td>Renal biopsy</td>
</tr>
<tr>
<td>RD</td>
<td>Renal disease</td>
</tr>
<tr>
<td>S.U.H</td>
<td>Saba University Hospital</td>
</tr>
<tr>
<td>US</td>
<td>Ultrasound</td>
</tr>
</tbody>
</table>