Dedication

**To my family, husband, teachers,
Colleagues and friends**

Acknowledgements

Firstly thanks to Allah for giving me strength and patience to do this work.

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LIST OF ABBRIVIATIONS

GFR	Glomerular Filtration Rate
ARF	Acute Renal Failure
CRF	Chronic Renal Failure
Na^+	Sodium
K^+	Potassium
DM	Diabetes Mellitus
HBV	Hepatitis B Virus
H.T	Hypertension
T.B	Tuber culosis
R.S	Renal stone

LIST OF TABLES

	Page

LIST OF FIGURES

ABSTRACT

In this study, 80 patients (47 men and 33 women) of different ages, with chronic renal failure on heamodialysis were selected to asses the effect of dialysis on the concentration of plasma electrolytes (sodium & potassium).

All patients were seen at Khartoum Center for Dialysis and Kidney Transplantation.

Tow samples from each patient were taken, pre and post dialysis. Then the concentration of plasma electrolytes (sodium & potassium), were estimated so as to know the impact (influence) of the renal heamodialysis on the concentration of sodium and potassium. And 20 healthy volunteer samples for comparison.

The study show that, the concentration of sodium was not affected by the dialysis, and remain in the normal range before and after heamodialysis. on the other hand the concentration of potassium was found to be more affected by heamodialysis, was remarkably high before dialysis and significantly reduced after the dialysis.

The prelimary studies showed most of patients were presented with other diseases such as hypertension, diabetes millets and hepatitis B virus, renal stones and tuber culosis.

From this study the age of chronic renal failure patients had no effect on the concentration of sodium after the heamodialysis while the potassium concentration

was affected by the age, it was found to be more reduced in age group of more than 30 years.

المستخلص

أجريت هذه الدراسة على 80 مريضا(47 من من الذكور و 33 من الاناث) ,فى أعمار مختلفة، يعانون من الفشل الكلوى ويجرون الغسيل الكلوى الدموى. وذلك لدراسة تأثيرالغسيل الدموى على تركيز الصوديوم والبوتاسيوم فى الدم بمركز الخرطوم للغسيل وزراعة الكلى.

وتم أخذ عينتين من كل مريض قبل وبعد الغسيل. ومن ثم تم قياس تركيز الصوديوم والبوتاسيوم لمعرفة تأثيرالغسيل الدموى على تركيزهما.

أيضا تم أخذ 20 عينة من الاصحاء المتبرعين وذلك للمقارنة. أوضحت الدراسة أن تركيز الصوديوم لم يتأثر بعملية الغسيل الدموى، كان فى المدى الطبيعى له قبل وبعد الغسيل، وفى المقابل وجد أن تركيز البوتاسيوم أكثر تآثرآ بالغسيل الدموى، زاد بوضوح قبل الغسيل فى حين أنه أنخفض أنخفاضآ وأضحآ بعد الغسيل الدموى.

اوضحت الفحوصات الاولية للمرض ان غالبية المرضى يعانون من امراض اخرى مصاحبة لمرض الفشل الكلوى مثل ارتفاع ضغط الدم، مرض السكرى، الاصابات بالتهاب الكبد الفيروسى ،وجود حصاوى بالكلى والسل الرئوى عند المرضى تحت الغسيل الدموى.

من هذه الدراسه أتضح أن العمر لمرضى الفشل الكلوى المزمن لا تأثير له على تركيز الصوديوم بعد الغسيل الدموى، فى حين وجد أن تركيز البوتاسيوم قد أنخفض أكثر فى المرضى الذين يزيد عمرهم عن 30 سنه.

1. Introduction and Literature Review

1.1 Introduction

Renal failure has been recognized as major health problem occurring in almost all population of the world in Sudan, It has been disclosed from Hospital records that the number of patients admitted Hospital suffering kidney disease are increasing month after and year after year.

A wide range of kidney lesions characterized by a slow, steady decline in renal function are already know A number of disease lead to renal failure. Renal failure divides into type's acute renal failure which is a sudden onset of renal disorder, and chronic renal failure which is progressive and irreversible disease, chronic a number of years. In fact, progression may be so gradual that symptoms do not occur until kidney function is less than one-tenth of normal.

Dialysis may relieve many of the symptoms of chronic renal failure and rectify abdominal fluid and electrolytes and acid- base balance. These treatment do not however, reveres, the other metabolic endocrine and hematological consequences of chronic Renal failure measurement of blood electrolytes in chronic renal failure patients under dialysis can give a good indication of osmolarity, sodium is very important cations to mention osmolarity of the body, the osmolarity of the body, the mechanism of controlling body osmolarity are under the control of hormones that act on the kidney. The major hormones involved in maintaining plasma osmolarity, and consequently water and sodium balance, are argentine vasopressin (AVP), also called antiduretichormone (ADH) and aldosterone, sodium measurements are the most important requited tests in which sodium concentration can give information about the state of hydration of the patients and may also indicate rarer condition when there is a disorder of hormonal control of water and electrolytes

balance. Changes seen in the body sodium concentration which are associated with water and electrolytes balance reflect the osmolarity of the extra cellular fluid (ECF). Abnormality of sodium concentrations fall into two categories there characterized by high sodium concentration (hypernatraemia) and those characterized, low sodium concentration (Hyponatraemia).

Potassium on the other hand, is measured in the clinical Biochemistry Laboratories in the same group of tests as sodium, forming apart of electrolyte profile. It is an important measurements as large changes in the blood potassium concentration can be life- threatening.

Potassium is the most intracellular cation with approximately 3500 mmol in the (ICF) and 55mmol in the (ECF) so potassium levels are greatly.

Influenced by the acid- base balance of the patients, in which hydrogen Ion play a major role in this balance. The presence of high concentration of potassium in the blood is called hyperkalaemia, and a low concentration is called hypkalaemia.

1.2- LITERATURE REVIEW

1.2.1 The Kidney

1.2.1.1 Structure and Anatomy:

The kidneys are paired, bean-shaped organs Located retroperitoneally on either side of spinal column. In the adults the weight of the kidney averages about 150gm. The kidneys are slightly larger in the male than in the female. The general structure of the kidney is shown in figure (A). Each kidney composed of about one million nephrons. A schematic structure is illustrated in figure (B).

The blood supply of the kidneys is relatively large, about one- quarter of the cardiac output at rest, i.e. 1300ml per minute, and subject to considerable physiological variation. The afferent arterioles, which give rise to the glomerular capillaries arise from intralobular branches of the renal artery, emerging from the glomeruli, the capillaries unit to form the afferent arterioles which supply blood to proximal and distal convoluted tubules in the cortex. The medulla is supplied by arterioles arising from glomeruli in the deeper regions of the cortex, and directly by branches from the accurate arteries. For a short distance the afferent arterioles and distal convoluted tubules are in contact, at this point the tubular cells become tall and columnar, forming the macula densa, and the wall of the arteriole is thickened by myoepithelial cells which contain large granular

Lacies cells which lie between the glomerular hilum and the macular densa, constitute the juxta glomerular apparatus (JGA) which is intimately concerned in extra cellular fluid volume regulation, blood pressure control and potassium balance. Glomerular filtration is the process when by water and solutes pass across the glomerular membrane by bulk flow or diffusion. (Berner, 1983) (Stanley et al, 1984)(Edward et al,1986).

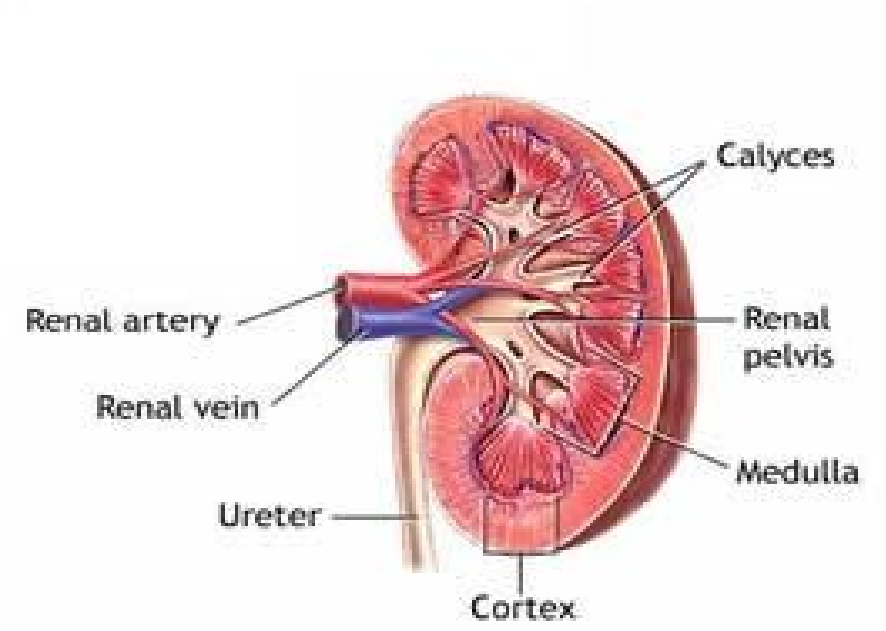


Figure (A): Structure of the kidney

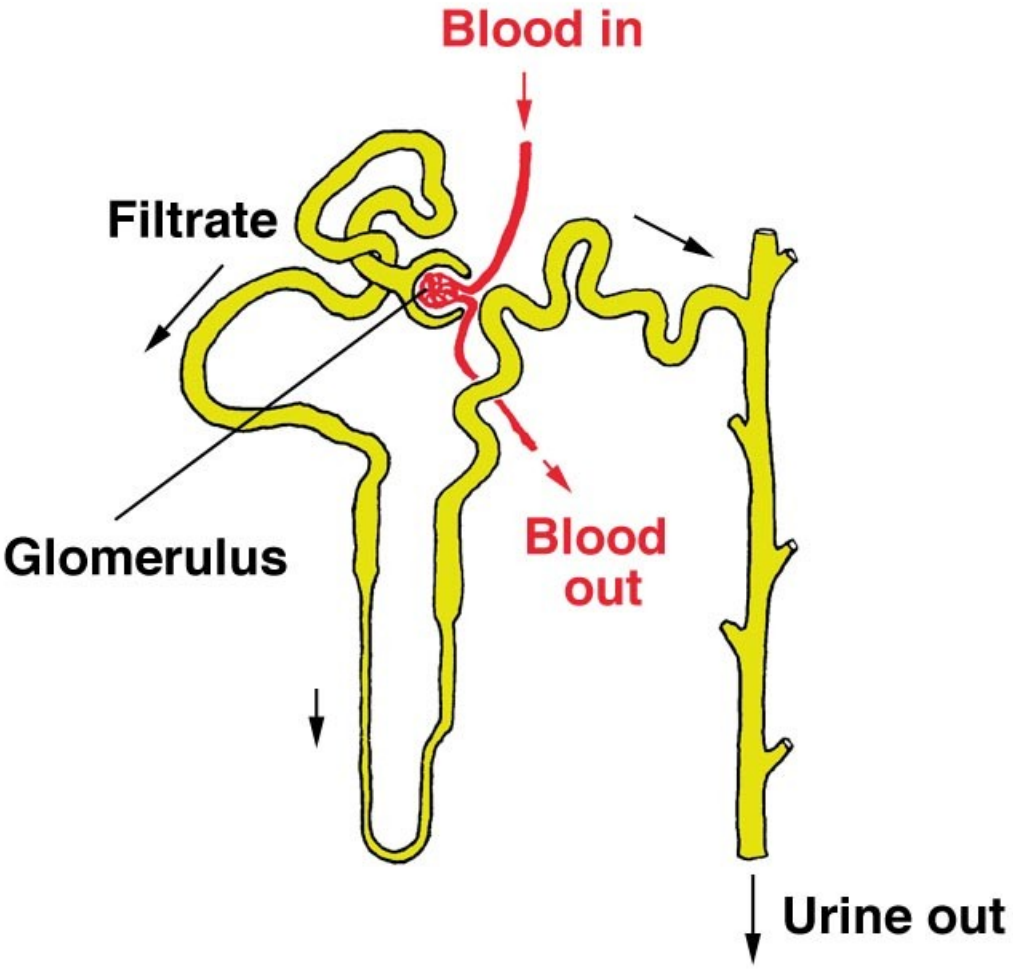


Figure (B): The Nephron

1.2.1.2 Functions of the kidneys:

The kidney are largely responsible for maintaining this states **:-**

1.2.1.2.1 Retention of substances vital to body economy:

Glucose is normally reabsorbed so completely by the proximal tubules that none can be detected in urine by clinical tests. Amino acids are reabsorbed in the proximal tubules, where several specific transport mechanisms exist.

Most filtered phosphate is reabsorbed in the proximal tubules, by processes linked to reabsorption of sodium and water, inhibited by parathyroid hormone and increased by vitamin D. In health only a small amount of protein reaches the fluid in Bowman's capsule. The volume of glomerular filtrate, however, is so great that if this small amount were not reabsorbed, more than 3g of protein would be excreted in the urine in 24 hours. In health up to 150 mg protein daily may be excreted in the urine (Gangong,1993).

1.2.1.2.2 Excretion of waste metabolic products:

The end products metabolisms, especially those of protein, are excreted in the urine. Excretion of urea depends mainly on the GFR, but a certain amount is reabsorbed by diffusion down concentration gradients produced by water reabsorption. Thus the proportion excreted increases during water diuresis. Creatinine is excreted mainly by filtration, though a small amount is secreted. Proximal tubular reabsorption of filtered uric

acid is linked to that of sodium and water, and is there for increased in extra cellular fluid volume depletion. In the distal nephron, uric acid is both secreted and reabsorbed, and certain drugs compete with it for these processes.

1.2.1.2.3 Hormonal and metabolic functions:

The JGA secretes rennin in response to number of stimuli **:-**

1. Reduced afferent arteriolar pressure.
2. Increased sympathetic activity.
3. Changes in composition of fluid reaching the macula densa.

The kidney is the main source of erythropoietin, the deficiency of erythropoietin is the main cause of the severe anemia which characteristic of advanced chronic renal failure, and hydroxylates 25-hydroxy cholecalciferol to from 1,25 dihydroxy cholecalciferol. Two vasodilator prostaglandins (PGE 2and PG12), both concerned with the control of the renal circulation, are produced by mesangial cells in the medulla. The kidney also secretes glandular kallikrein, an enzyme which generates vasodilator peptides, bradykinin, from kininogen precursors these may influence renal blood flow, sodium and water excretion, but their precise function is not yet known. (Gangong,1984)(Edward et al 1986).

Normal renal function depends on a normal filtration rate, and normal tubular function.

1.2.1.3 Glomerular function:

GFR depends on the net pressure excreted across the glomerular membrane, the physical nature of the membrane and its surface area, which reflects the number of functioning glomeruli. All these factors may be modified by disease. However, in the absence of large changes in blood pressure or gross changes in the structure of the glomerular membrane, the GFR provides an index of the number of functioning glomeruli.

- **Glomerular function test:•**

The GFR is determined by measuring the concentrations in plasma and urine of a substance which ideally fulfils the following criteria:

1. It is readily filtered from the plasma at the glomerulus.
2. It is neither reabsorbed nor secreted by the tubules.
3. Its concentration in plasma remains constant through out the period of urine collection.
4. The measurement of its concentration in plasma and urine in both and analytically convenient and reliable.

$$GFR = \frac{Us}{Ps} \cdot V \qquad \text{(formula of the clearance)}$$

If a substance (S) meets the four criteria listed above, the amount excreted in urine (Us) in unit time equals the amount cleared from the

plasma (Ps). The units for V. the rate of formation of urine, and for GFR are ml/min. (Whitby et al, 1987).

1.2.1.4 Tubular function test:

The tubular reabsorbed water and solutes from the glomerular filtrate, and secrete solutes into it, both reabsorption and secretion of substances increase with rising plasma concentration but there are maximum rates for both processes. The useful test for tubular function is water deprivation test.

1.2.1.5 Electrolytes (Sodium & potassium) and renal function:

- **Sodium reabsorption :**

There is active reabsorption of sodium (Na^+) in the proximal tubules up to about 75% of the (Na^+ in the filtrate, further reabsorption occurs in the loops of Henle only about 10% reaches the distal tubules.

In the distal tubules and collecting ducts, fine regulation of the sodium reabsorption occurs under influence of aldosterone, According to the needs of homeostasis in the absence of this hormone most of the remaining sodium is lost in the urine, when aldosterone is secreted in large amounts, almost all of sodium reabsorbed.

The kidney reabsorbs quite a large amount of sodium each day. The amount which passes in the filtrate is about 6259/day the transport of sodium in the distal tubules in an active process, which occur against concentration gradient from the tubular cell to the per tubular fluid.

This net movement of sodium creates negative electrical potential in side the tubular cell, leads to withdraw sodium into the cell from the tubular lumen.

The active sodium transport also results, in the pumping of potassium into the cell. Sodium transport however, is greater than that of potassium.

- **Potassium reabsorption:**

Potassium reabsorption occurs by two mechanisms:

1. Active reabsorption in the proximal tubule almost completely conserves potassium.
2. Exchange with sodium is stimulated by aldosterone. Hydrogen competes with potassium for this exchange.

The amount of (K^+) secreted is directly related to aldosterone secretion and sodium reabsorption.

Small increases of serum (K^+) directly stimulated the adrenal cortex to release aldosterone and, thus, (K^+) secretion enhanced, in the absence of aldsterone, (K^+) secretion stops and reabsorption occurs. This shows that (K^+) reabsorption taking place all the time but it is usually masked by the normally greater amount secreted.

(Michaed L.*et al* ,1996) (Sukkar et al.1997).

1.2.1.6 Renal diseases:-

There are six most common disease of the kidney.

- **The Nephrotic Syndrome:**

Is a condition characterized by:-

Heavy proteinuria, hypoalbuminemia, severe odema, hyper lipidemia and lipiduria.

Causes: In children minimal change nephrotic syndrome or minimal change disease is a commonest cause, where in adult membranous glomerulonephritis is most commonly found.

The nephrotic syndrome can also caused by systemic disease affecting the glomerulus's, D.M, infections, drugs, toxins and neoplastic diseases can also be etiological factors.

- **The acute Nephrotic syndrome:**

In this condition there is proteinuria – Haematuria – Granular casts hypertention – oliguria (urine volume < 400ml /day).

- **Asymptomatic proteinuria:**

It can result from the same disease which causes the nephrotic syndrome, the main different being that the protein uria is of in sufficient degree to cause hyper albuminemia and odema.

- **Pain less haematuria:**

In this conditions presence of red blood cell in urine in abnormal (amount).

Causes: Benign and malignant disease of the bladder – kidney and other parts of urinary tract.

- **Hypertension:**

Is usually idiopathic or essential in type but in around 10% of cases is due to renal disease.

- **Renal failure:**

Is divided into two types acute renal failure and chronic renal failure.

1.2.1.7 Acute renal failure:

The renal failure is characterized by an acute and usually reversible deterioration of renal function, which develops over a period of days or weeks and results in uraemia. A marked reduction in urine volume is usual, but not in variable clinical features are determined by the underlying condition and by rapidly developing uraemia, if the patients survives, renal function usually returns to normal or near normal. (Macsween et al, 1995).

1.2.1.7.1 Pathophysiology:

Acute renal failure (ARF) may occur in three clinical settings, including:

A: As an adaptive response to severe volume depletion and hypotension, with structurally and functionally intact nephrons.

B: In response to cytotoxic insults to the kidney, with structural and functional damage.

C: With obstruction to the passage of urine therefore, ARF may be classified as pre renal, intrinsic, and post renal. While these classifications are useful in establishing a differential diagnosis, many pathophysiologic features are shared among the different categories.

The intrinsic form of the syndrome may be accompanied by a well-defined sequence of events. The first is an initiation phase, characterized by daily increases in serum creatinine and reduced urinary volume. The second is a maintenance phase, during which the glomerular filtration rate (GFR) is relatively stable and urine volume may be increased; and the third is a recovery phase, in which serum creatinine levels fall and tubule function is restored. This sequence of events is not always apparent, and oliguria may not be present. The reason for this lack of a uniform clinical presentation is a reflection of the variable nature of the injury.

Classifying (ARF) as oliguric or non oliguric based on daily urine excretion may be useful. Oliguria is defined as a daily urine volume of less than 400ml/d. Anuria is defined as urine output of less than 50ml/d and, if abrupt in onset, is suggestive of obstruction. Stratification of renal failure along these lines helps in decision- making (eg, timing of dialysis)

and seems to be an important criterion for patient response of therapy. (Mahendra, 2002).

1.2.1.7.2 Causes:

The causes of Acute renal failure may be divided into there main categories: Prerenal, intrarenal, and postrenal.

In prerenal ARF, perfusion of kidneys is compromised by the following.

Hypotension may be causing compromised renal perfusion.

Hypovolemia from either renal loss (e.g., due to Addison disease or diabetic ketoacidosis).or extrarenal loss (e.g., due to vomiting, diarrhea, pancreatitis, burns, or sweating) may by present.

Patients may have intense vasoconstriction due to hypercalcemia, prostaglandin inhibition. Causes of intrarenal ARF can be grouped into vascular interstitial, and glomerular factors, as follows.

Vascular causes include vasculitis involving small vessels, scleroderma, atheroembolic renal disease, malignant hypertension, and thrombotic angiopathy. More frequently they cause ischemic tubular necrosis.

Glomerular factors may suggest glomerulonephritis. ARF secondary to glomerulonephritis is observed in severe forms of crescentic glomerulonephritis , post infective glomerulonephrities Lupus nephritis, hepatitis and several other vasculitis- associated glomerulorephrities,

The most common cause of postrenal failure is secondary to bladder outlet obstruction due to prostatic hypertrophy. (Mahendra, 2002).

1.2.1.8 Chronic renal failure:

Chronic renal failure (CRF) is a gradual and progressive loss of the ability of the kidney to excrete wastes, concentrate urine, and conserve electrolytes.

In CRF, the kidneys normally do not heal. It can range from mild dysfunction to severe kidney failure. Progression may continue to end-stage renal disease.

1.2.1.8.1 Pathophysiology:

Approximately 1 million nephrons are present in each kidney, each contributing to the total GFR. Regard less of the etiology of renal injury, the progressive destruction of nephrons, the kidney has an innate ability to maintain GFR by hyper filtration and compensatory hypertrophy of the remaining healthy nephrons. This nephron adaptability allows for continued normal clearance of plasma solutes such that substances such as urea and creatinine start to show significant increase in plasma levels only after total GFR has decreased to 50% when the renal reserve has been exhausted. The plasma creatinine value will double with 50%

reduction in GFR. Arise in plasma creatinine from a base line value of 0.6mg/dl to 1.2mg/dl in a patient, although still within the reference range, actually represents a loss of 50% of functioning nephron mass.

The residual nephron hyper filtration and hypertrophy, although beneficial for the reasons noted, has been hypothesized to represent a major cause of progressive renal dysfunction.

This is believed to occur because of increased glomerular capillary pressure, which damages the capillaries and leads initially to focal and segmental glomerulosclerosis and eventually to global glomerulosclerosis. This hypothesis has been based on studies of five-sixths nephrectomized rates, which develop these lesions that are identical to those observed in humans with CRF (Verrelli, 2004).

1. 2.1.8.2 Aetiology:

Chronic renal failure may be caused by any condition which destroys the normal structure and function of the kidney.

The a etiology of chronic renal failure are:

1. Glomerulonephritis.
2. Diabetic glomerulosclerosis
3. Chronic pyelonephritis.
4. Obstructive uropathy.
5. Hypertensive nephrosclerosis.
6. Polycystic kidneys.
7. Drugs and toxins.

(Edward et al,1986).

1.2.1.8.3 Clinical Features:

The predominant symptom of chronic renal failure is tiredness, due mainly to the accompanying anaemia. Urinary symptoms may include

polyuria resulting from the osmotic diuresis. This in turn may cause polydipsia and thirst. Dyspnoea is common. Particularly in the older patient, due again to the anaemia and often also to heart failure. Tachypnoea may also be due to the acidosis, in which case it will be unaccompanied by the visible distress of dyspnoea, this type of sighing breathing carries the eponym of kussmaul's respiration. Hypertension may be a primary cause of chronic renal failure but much more often it is a sequel to the renal disease. Anoxia, nausea, pruritus and vomiting are features of more advanced ureamia while neuromuscular feature include myopathy, peripheral neuropathy, and encephalopathy with convulsions and intellectual deterioration in the long- term patient.

1. Anaemia:

Anaemia is common and to some extent reflects the severity of uraemia. Several factors contribute, including:

i. a reduced dietary intake of iron and other haematinics due to anorexia and dietary restrictions.

ii. Impaired intestinal absorption of iron.

iii. Diminished erythropoiesis due to toxic effects of uraemia on marrow precursor cells.

iv. Reduced red cell survival.

v. Increased blood loss due to capillary fragility and poor platelet function.

In patients with poly cystic kidneys, anemia is less severs. Possibly because the large kidneys produce more erythropoietin.

2. Endocrine function:

A number of hormonal abnormalities may be present in the female. Amenorrhea is common. In both sexes there is loss of libido presumably due to the associated hyper prolactinaemia. Thyroid function is diminished, although clinical hypeothroidism is uncommon. Uraemia is frequently mistaken for hypothyroidism.

3. Cardiovascular disorders:

Hypertension develops in approximately 80% of patients with chronic renal failure. It must be controlled, as it causes further vascular damage, thus increasing renal failure. Atherosclerosis is common due to abnormalities of lipid and carbohydrate metabolism, and may be accelerated by hypertension.

4. Infection:

Both cellular and humoral immunity are impaired, and thus there is increased susceptibility to infection. Urinary tract infections are very common and must be treated promptly, as they lead to further destruction of functioning renal tissue. (Edward et al, 1986).

1.2.1.9 End stage renal disease:

End –stage renal disease (ESRD) is loss of renal functions requiring treatment with any of chronic dialysis or transplantion.

1.2.2 DIALYSIS

Dialysis is a method of removing toxic substance (impurities or wastes) from the blood when the kidneys are unable to do so.

Dialysis is most frequently used for patients who have kidney failure, but may also be used to quickly remove drugs or poisons in acute situations. This technique can be life saving in people with acute or chronic kidney failure.

Dialysis Is done by using a special fluid called dialysate, a mixture of pure water and chemicals, is carefully controlled out of blood without removing substances of the body needs.

Dialysis acts as an artificial kidney, there are two types of dialysis haemodialysis and peritoneal dialysis. (Jeremy et al,2001) (Mayne,1994).

1.2.2.1 Types of dialysis:

1.2.2.1.1 Peritoneal dialysis:

It has been used for treating acute and chronic renal failure since 1923.In peritoneal dialysis a fluid is put into patient abdomen. The fluid called dialysate, captures the waste products from patient blood. The fresh bag of dialysate is dripped into abdomen. (Figure C).

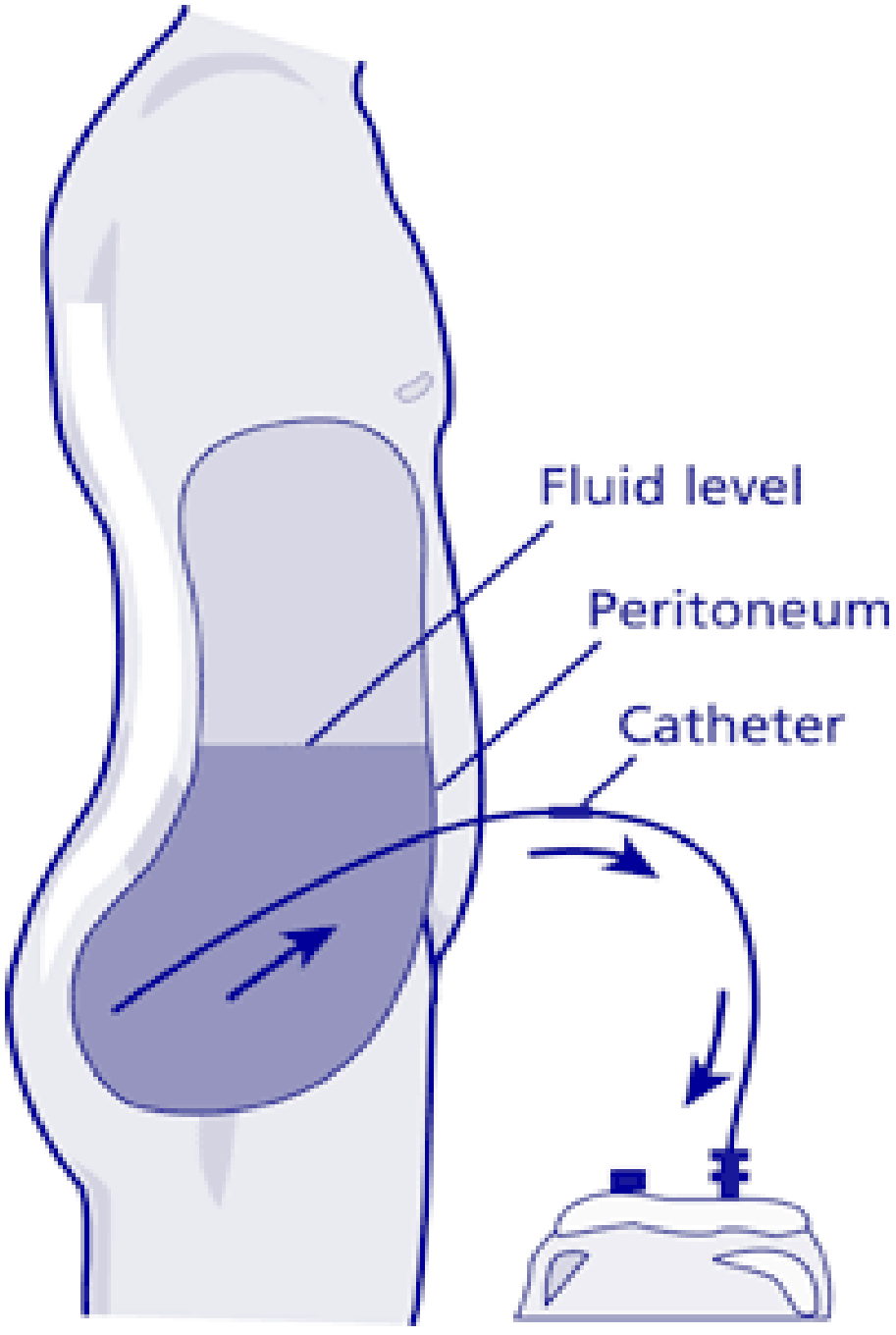


Figure (C): Peritoneal dialysis

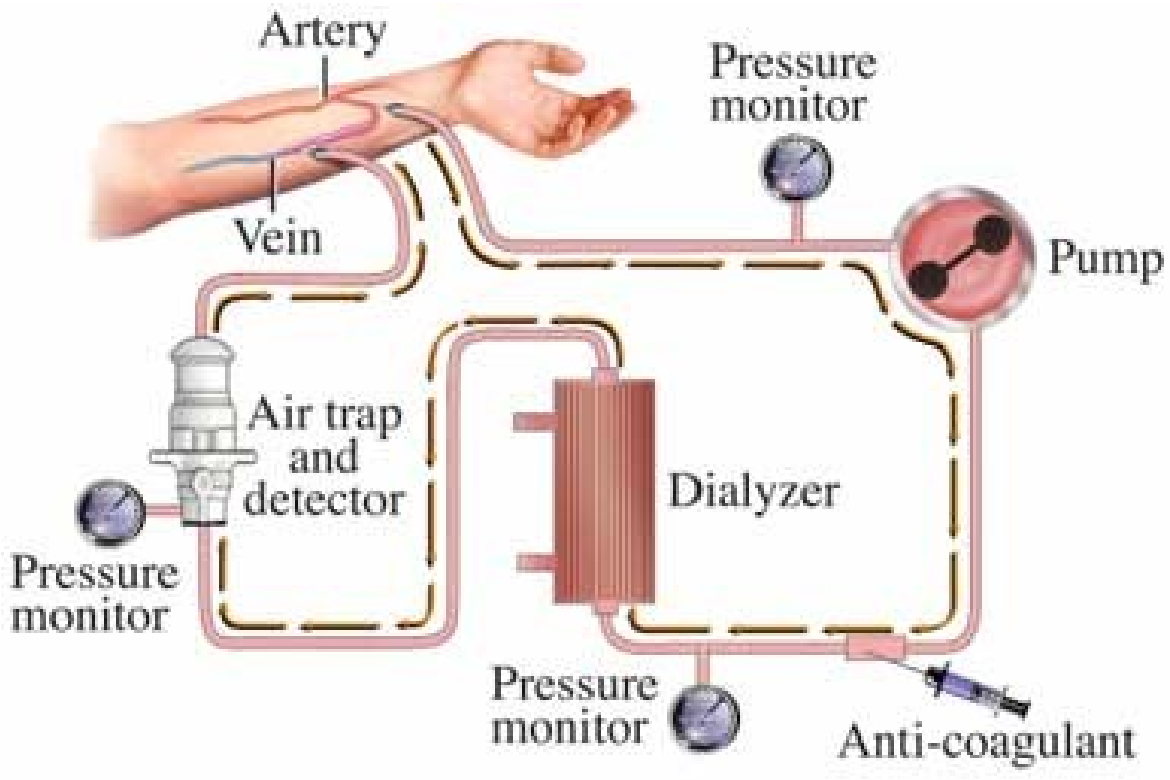
- **Types of Peritoneal Dialysis:**

1. Manual peritoneal dialysis.
2. Intermittent peritoneal dialysis.
3. Continuous ambulatory peritoneal dialysis.
4. Continuous cyclic peritoneal dialysis.
5. Nocturnal intermittent peritoneal dialysis.

1.2.2.1.2 Haemodialysis:

About 90% of dialysis patients receive in which the blood is circulated out side the body and cleaned in side a machine before returning to the patient. (Figure D).

In haemodialysis a man-made membrane or dialyzer, partly does the work of the kidneys to filter waste and remove extra fluid. Blood circulates through the dialyzer for several hours during a treatment, with a machine controlling the speed and several safety factors. Haemodialysis is most often done three times per week for three to four hours at a dialysis center to prevent blood clotting in the system, the patients receives heparin during treatment there are also two less common methods of haemodialysis Nocturnal haemodialysis, typically performed in the home. Involves extended treatment three or more rights a week while patient sleep. Daily home haemodialysis involves two or three-hour sessions five or six times a week.



Figure(D):Haemodialysis

1.2.2.2.1 Principles of haemodialysis:

Haemodialysis mainly works through two principles:

- **Diffusion**: which is the movement of particles a cross a membrane with tiny holes in it (semi permeable) until the particles concentration on both sides of the membrane is equal. The rate of diffusion is greatest when the concentration gradient is highest. And can occur in either direction across a membrane.
- **Ultra-filtration:** Is the convection flow of water and dissolved solutes down a pressure gradient. The pressure gradient can be caused by hydrostatic or osmotic forces. In haemodialysis it usually occurs as a result of the negative pressure generated in the dialysate compartment by the dialysate effluent pump.

1.2.2.2.2 Nutritional requirement:

People undergoing haemodialysis need special diet. Daily consumption of sodium and potassium is even more restricted. Food high in phosphorus also may have to be limited, daily fluid intake is limited for people who have a persistently low or a decreasing sodium concentration in the blood daily. Weighting is important to monitor weight gain. Excessive weight gain between haemodialysis treatments indicates that the person is consuming excusive fluid. In haemodialysis formal dietary Potassium restriction is usually required but the level of restriction is partly dependent on residual renal function. (Jeremy et al, 2001).

1.2.3. ELECTROLYTES

Electrolytes are ions capable of carrying an electric charge. They are classified as anions or cations based on the type of charge they carry. Anions have a negative charge and move toward the anode; cations have a positive charge and migrate toward the cathode. Electrolytes enter the body through the diet and leave the body primarily through the kidney, roles are also important such as sweat and the gastro intestinal tract hormones, secreted by endocrine gland, and control the absorbance of electrolyte by the renal tubules and thus play a vital role in maintaining homeostasis. One of the most important functions of electrolysis to maintain the acid-base balance of the body, such electrolysis as sodium, potassium, chloride, phosphate, bicarbonate and carbonic acid are in same way related to this process. Osmotic balance is maintained by sodium potassium and chloride in addition potassium play role in active transport material against the concentration gradient. Some of electrolytes also play important constituent of the body structure and also play important role in imbalance in the body is fluid loss through diarrhea, vomiting and sweating.

Several methods are applied in analysis of electrolytes titrimetry, photometry and electrometer.

Electrolyte concentrate are expressed either as mill equivalent per liter (MEq/L) or standard international (SI) units is mmoles per liter (mmol/L) because 1mEg/L in equal 1mmol /L for monovalent ions.

1.2.3.1 Sodium (Na^+):

Is the major positive ion in the fluids out side of cells. The concentration of sodium in side cells is only about 5mEq/L compared with 140 mEq/L outside. The sodium content of the blood is a result of a balance between the amount in the food and beverages body consume, and the amount of kidneys excrete. (In addition, a small percent is lost through the stool and sweat).

In health plasma sodium level (135-145 mmol/L) it's primary functions in the body are to maintain osmotic pressure and acid-base balance chemically and transmit nerve impulses. The body has a strong tendency to maintain a total base content and only slight changes are found even under pathologic conditions. Mechanisms for maintaining a constant sodium level in the plasma and extra- cellular fluid include renal blood flow, carbonic anhydrase enzyme activity. aldosterone a cation of other steroids whose plasma level is controlled by the anterior pituitary gland, rennin enzyme secretion, Anti-diuretic hormone (ADH) and vasopress in secretion.

Sodium activity pumped out of the cells by the Na^+, K^+ AT-pase pump. The ATP is the one of the major energy source in the body. ATP is responsible for the exchange of Sodium and Potassium ions across the cell membrane. (Juhap et al,1987), (Bishop et al, 2000).

1.2.3,1.1 Reference value: (Kaplan et al,1999), (Fischbach ,2000), (William et al, 2004)

Adults	136 – 145 m eq./L (136 – 145 mmol/L)
Children (1-16 years)	136 – 145 m eq./L (136 – 145 mmol/L)
Full term infants	133 – 142 m eq./L (133 – 142 mmol/L)
Pre-mature infants	132 – 140 m eq./L (132 – 140 mmol/L)

2.3.1.2 Hyponatrimia: (Lower than normal sodium level) Classified according to total body water:

- A decrease in total body water (hypovolemic hyponatremia indicates dehydration, over diuresis, ketonuria (excression of ketone bodies in urine), vomiting or diarrhea.
- Near normal total body water (normovolemic hyponatremia) indicates syndrome of inappropriate anti-diuretic hormone secretion (SIADH), hypothyroidism, or Addison disease.

- An increase in total body water (hypervolemic hyponatremia indicates kidney failure, congestive heart failure, nephrotic syndrome, or cirrhosis of the liver.

1.2.3.1.3 Hypernatremia: (Greater than normal sodium level)

Classified according to the amount of fluid that is out side cells (extra-cellular volume).

- In extra-cellular volume is low, water lost due to burns, excessive sweating, and diarrhea or osmotic/loop diuretics (usually not thiazides).
- If extra- cellular volume is normal, this may indicate diabetes If extra-cellular volume is high, this may indicate hyper-aldosteronism, Cushing's Syndrome, or salt or sodium bicarbonate ingestion.(Zilva et al, 1993), (Maxwell et al,1987).

1.2.3.2 Potassium (K^+):

Is the major positive ion within cells. The total body potassium of a 70 – Kg body man accounts about 3500 mmol, 98% of this in intra-cellular since most intra-cellular potassium is within muscles cells.

In health plasma potassium level (3-5 mmol/l). Potassium is one of the most important electrolytes, since it is needed for neuron scular activity and myocardial contractility. Therefore both sever hypokalemia and hyperkalemia are dangerous.

Potassium is important in muscles for glycogen synthesis and maintains the intra-cellular content of muscles, and properties of cell membrane.

Potassium in association with sodium plays a primary role in maintenance of intracellular fluid volume and pressure.

Potassium acts as activator for the enzyme sodium-potassium activated adenosine triphosphate (Na^+,K^+-AT pase) which is present in large amount.

In active tissues such as nerve, kidney and epithelial potassium levels are mainly controlled by the steroid hormone aldosterone. Aldosterone is secreted from the adrenal gland when level of potassium increase. Aldosterone, in turn, cause the body to get rid itself of the excess potassium. (Juhap et al, 1986), (Bishop et al, 2000).

1.2.3.2.1 Reference value: (Kaplan et al, 1999), (Fischbach ,2000), (William et al, 2004)

Adults	3.5 – 5.2 m eq./L (3.5 – 5.2 mmol/L)
Children (1-18 years)	3.4 – 4.7 m eq./L (3.4 – 4.7 mmol/L)
Infants (7 days -1 years)	4.1 – 5.3 m eq./L (4.1 – 5.3 mmol/L)
Neonates (0 – 7 days)	3.7 – 5.9 m eq./L (3.7 – 5.9 mmol/L)

1.2.3.2.2 Hypokalemia: (Lower than normal levels of potassium)

- The person is not getting enough potassium intake in the diet.
- Excessive potassium loss because of a gastrointestinal disorder- e.g. chronic diarrhea or use of laxatives.
- Vomiting.
- Diuretic use.
- Renal artery stenosis (narrowing of the major blood vessels to the kidney).
- Hyper aldostronism.
- Hypokalemic periodic paralysis.
- Cushing's syndrome (rare)

1.2.3.2.3 Hyperkalemia: (Greater than normal levels of potassium)

- Crushed tissue from an injury.
- Kidney failure.
- Red blood cell destruction.
- Metabolic or respiratory acidosis.
- Transfusion of haemolyzed blood.
- Hyperkalemic periodic paralysis (potassium is elevated during episodes of paralysis).
- Addison's disease (rare)
- Hypo aldosteronism (very rare).

(Zilva et al, 1993), (Maxwell et al,1987).

OBJECTIVES

The aim of this study was to:

1. Evaluate the effect of heamodialysis on the concentration of electrolytes. (Sodium and potassium)
2. To study the correlation (If any) between the levels of these electrolytes.

2. MATERIALS AND METHODS

2.1 Materials:

2.1.1 Patient's:

The chronic renal failure patient's were (80), including (47) males and (33) females were selected to participate in this study, in which the concentration of plasma electrolytes (sodium and potassium) were evaluated pre and post dialysis.

All patients were seen at Khartoum Center for Dialysis and Kidney Transplantation. CRF patients were under haemodialysis two times per weak. All clinical investigation and assessments were done by medical physician.

Other (20) apparently healthy individuals were also participated in this study to serve as control.

2.1.2 Samples:

5 ml of blood sample were taken from each CRF patient's as well for control in heparinized bottles, after centrifugation for 5 minutes (3000 r pm),the clear plasma was obtained and refrigerated at (-4 C°) until used.

2.1.3 Chemicals and Reagents:

All chemical and reagents used in this study were purchased from the local market.

2.1.3.1 Equipments:

The measurement of sodium and potassium is done by:

- Flame photometer that is designed and manufactured in UK by Jenway LTD. Felsted, Dunmow. Essex CM63LB.
- Centrifuge model MSE BA 6350. Serial no BA, made in England.
- 10 ml plastic pipettes.
- Automatic micro pipettes 100 U/L.
- Eppindorf tube 2ml.
- Lithium heparin container.
- Plastic container.
- 5ml disposable syringes.

2.1.3.2 Reagents:

- Standard (sodium, potassium).
- Control - N (Human serum normal). &
- Control - P (Human serum pathogen).

Deionized water.

2.2 Methods:

Methods Estimation of Sodium and Potassium:

- The method used is Emission flame photometer.

2.2.1 Principles of flame photometer:

Using compressed air, diluted serum or plasma is sprayed as fine mist droplets (neublised) into a known luminous gas flame which become colored by the characteristic emission of sodium or potassium metallic ions in the sample. Light of a wave length corresponding to the metal being measured is selected a light filter and allow to fall on photosensitive detector system the amount of light emitted depends on the concentration of metallic ions present in the sample.

2.2.2 Procedure:

Samples were diluted 1: 200 by manual dilution.

1. 0.1ml of (sample / standard / control) were added to 19.9ml deionized water in container.
2. The device pre washed by deionized water for 5min.
3. Fuel knob opened and flame ignited.
4. Zero is adjusted by blank knob with deionized water.
5. The standard inserted and adjusted at (140mmol/L) for Na+ and (5.0 mml/L) for K+
6. Repeat step (4) and (5) till deionized water give zero and standard gives 140mmol for Sodium&5.0mmol for Potassium.
7. Result of the sample was obtained directly from the display in mmol/L.
8. Post washing has been done by putting of the flame and allow the device to work by deionized water only for 5min.

9. The gas supply was cut off.

10. The electric current was discontinue

- The control serum from Spinreact Company was used for normal and pathological.

Measurement	**Normal**	**Pathological**
Na^+	(128-158) mmol/L	(144-180) mmol/L
K^+	(38-5.0) mmol/L	(5.8-7.8) mmol/L

3. RESULTS

Plasma electrolyte (sodium and potassium) was estimated in 80 patients with chronic renal failure (CRF) and 20 volunteers used as control, to assess the adverse effect of haemodialysis on (Sodium and potassium) concentration.

Table (1) and figure (1) represented the disease history in CRF patients. From table and fig it is clear that 33.8% of patient's under haemodialysis presented with hypertension, 8.8% presented with HBV, 8.8% presented with renal stone, 2.5% presented with T.B, 2.5% presented with heart, 1.3% presented with nephropathy and 35.0% free of disease.

Table (2) and figure (2) represented the mean age of CRF patients and control a significant difference was seen, being 50.7$\pm$ 3.2 and 37.55 $\pm$ 2.5. mmol/L.

Table (3) indicated no significance difference between the mean sodium and potassium concentration in control and CRF patient before haemodialysis. Which in figure (3-a) the mean sodium concentration in control and CRF patient before haemodialyas and in figure (3-b) the mean potassium concentration in control and CRF patient before haemodialyas. Significant difference was seen being 4.09$\pm$ 0.82 and 5.49 $\pm$ 0.28. mmol/l, at P> 0.05.

Table (4) and figure (4) illustrated the average sodium concentration in CRF patient pre and post dialysis. A significance difference was seen. Being 135.035 ± 18.2 and 129.36 ± 22 mmol/L.

Table (5) and figure (5) presented the mean potassium concentration in CRF patient's pre and post dialysis. A significance difference between the two values was seen, being 5.498 ± 0.32 and 3.38 ± 0.042mmol/L.

Table (6) figure (6) shows that no significant difference between the mean sodium concentration in CRF patients according to sex pre and post dialysis.

Table (7) and figure (7) indicated the concentration of potassium according to sex in CRF patient's pre and post dialysis. A significance difference was seen. In males being 4.874 ± 0.82 and 3.38 ± 0.31 mmol/L and in female being 4.969 ± 0.77 and 3.40 ± 0.28 mmol/L.

Table (8) and figure (8) represented the effect of age on sodium levels in CRF patient. No significance changes in sodium level pre and post dialysis at the age less than 30 yeas or more than 30 years.

Table (9) and figure (9) presented the effect of age on potassium levels in CRF patient. A significance difference was seen at the age than 30 years, being 4.64 ± 0.42 and 3.581 ± 0.3 mmol/L and in the age more than 30 years, being 4.98 ± 0.49 and 3.34 ± 0.01mmol/L.

Table (10) and figure (10) shows no significance difference in the concentration of sodium in CRF patients according to duration of disease at haemodialysis.

Table (11) and figure (11) indicated the concentration of potassium in CRF patients according to duration of disease at haemodialysis.

A significance difference was seen, at duration less than 3 years, being 4.760 $\pm$ 0.41 and 3.302 $\pm$ 0.32 and at duration more than 3 years, being 5.144 $\pm$ 0.5 mmol/L and 3.518 $\pm$ 0.2 mmol/L.

Table (1): Disease history in CRF patients

Disease	Number	Percent
hypertension	27	33.75
HBV	7	8.75
Renal Stone	7	8.75
Diabetes Mellitus	6	7.5
T.B.	2	2.5
Heart	2	2.5
Nephropathy	1	1.25
Free of Disease	28	35
Total	**80**	**100**

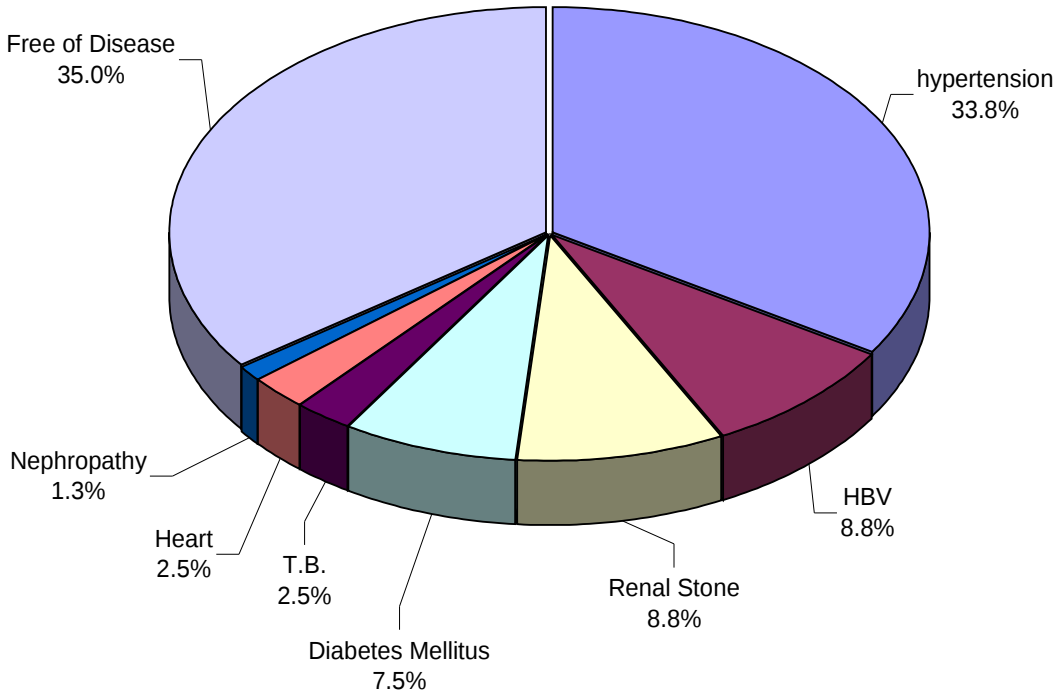


Figure (1): Disease history in CRF patients

Table (2): The mean age of CRF patients and control

Age (year)	Control	CRF patients	P. value
Mean age	37.55 ± 2.5 (27-48)	50.7 ± 3.2 (16-76)	0.002*

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

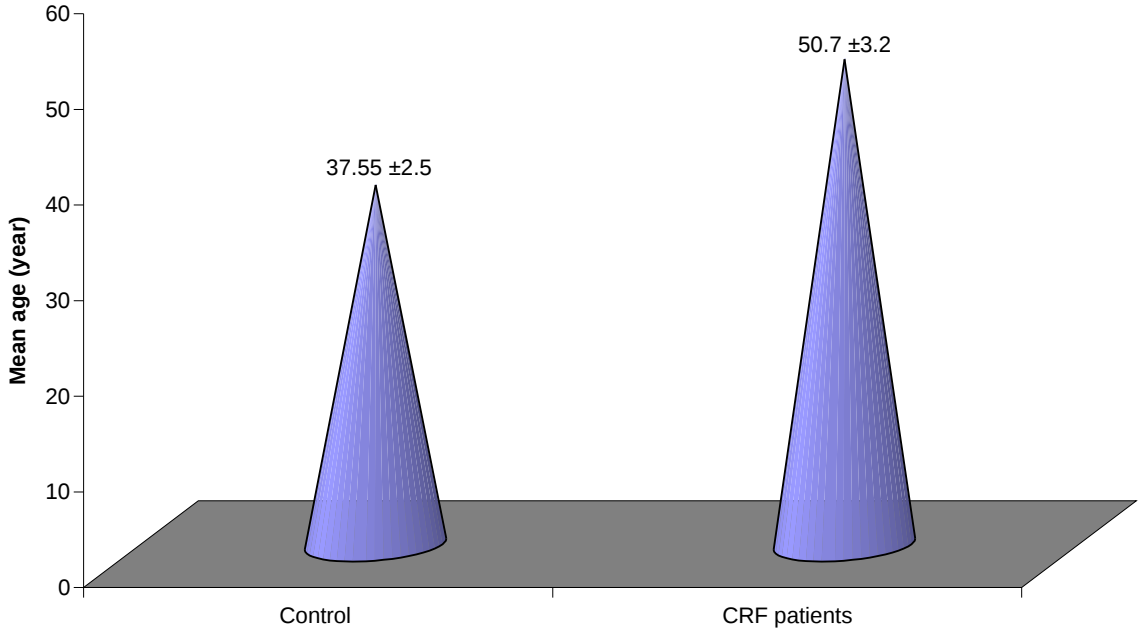


Figure (2): The mean age of CRF patients and control

Table (3): The mean sodium concentration in control and CRF patient before haemodialysis

Parameters	Control	CRF patients	P. value
Sodium (mmol/L)	138.05 ± 17.2 (134-142)	135.04 ± 22.2 (117-146)	0.825*
Potassium (mmol/L)	4.09 ± 0.82 (3.5-4.5)	5.498 ± 0.28 (2.1-7.5)	0.003**

Result expressed in mean and ± SD
Range between brackets
* Not significantly differnet (P. vlaue >0.05)
** significant difference at P. value <0.05

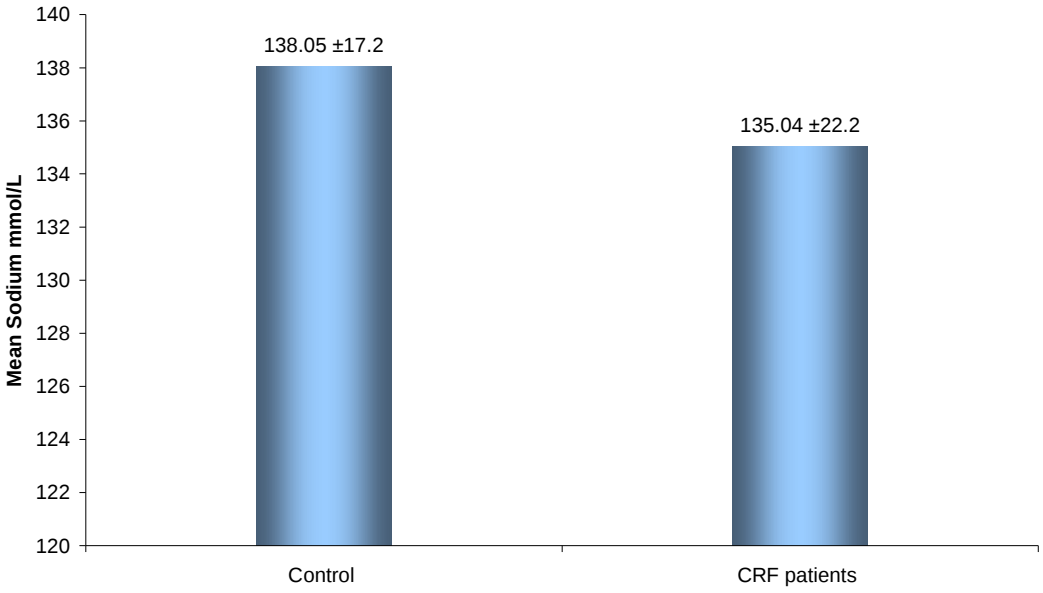


Figure (3-a): The mean sodium concentration in control and CRF patient before haemodialysis

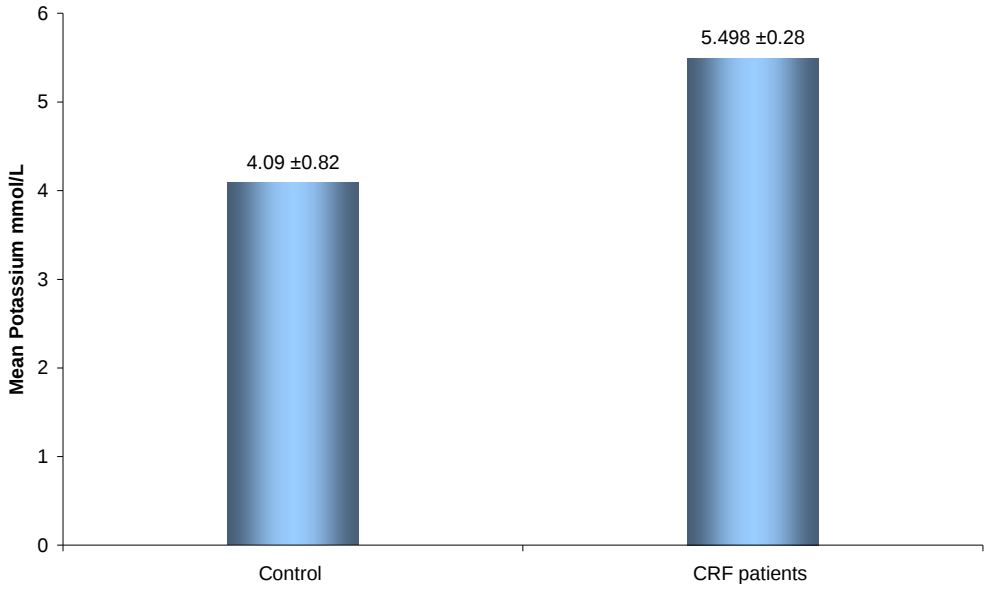


Figure (3-b): The mean potassium concentration in control and CRF patient before haemodialysis

Table (4): The mean sodium concentration in CRF patient pre and post dialysis

Measurement	Number	Pre-dialysis	Post-dialysis	P. vlaue
Sodium Con. (mmol/L)	80	135.035 ± 18.2 (122-146)	129.36 ±22.0 (117-142)	0.005

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

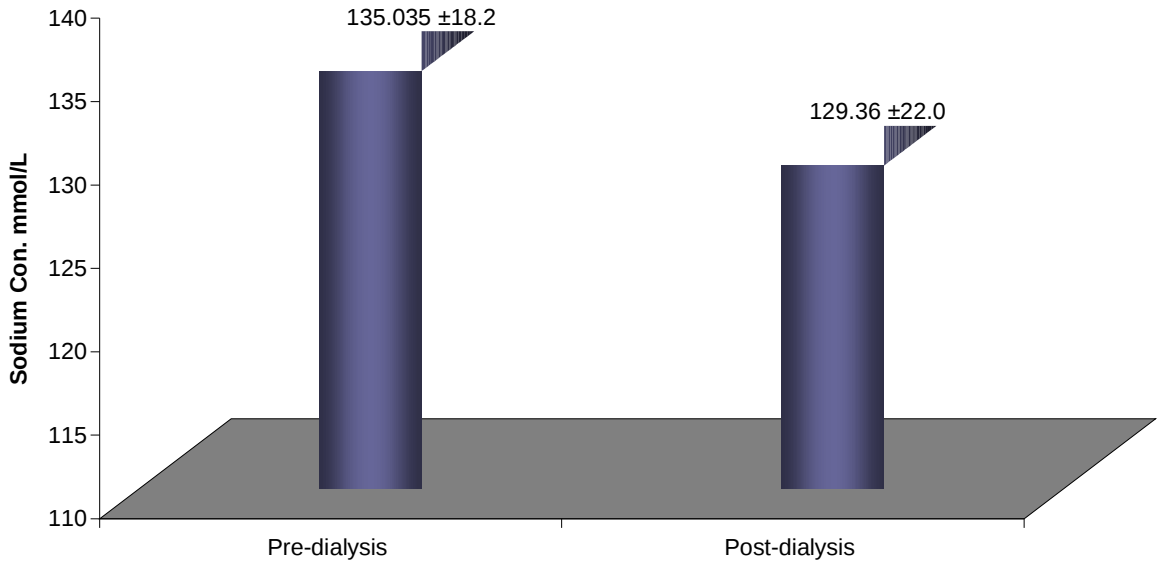


Figure (4): The mean sodium concentration in CRF patient pre and post dialysis

Table (5): The mean sodium concentration in CRF patient pre and post dialysis

Measurement	Number	Pre-dialysis	Post-dialysis	P. vlaue
Potassium Con. (mmol/L)	80	5.498 ±0.32 (2.1-7.0)	3.38 ±0.042 (1.8-4.9)	0.003

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

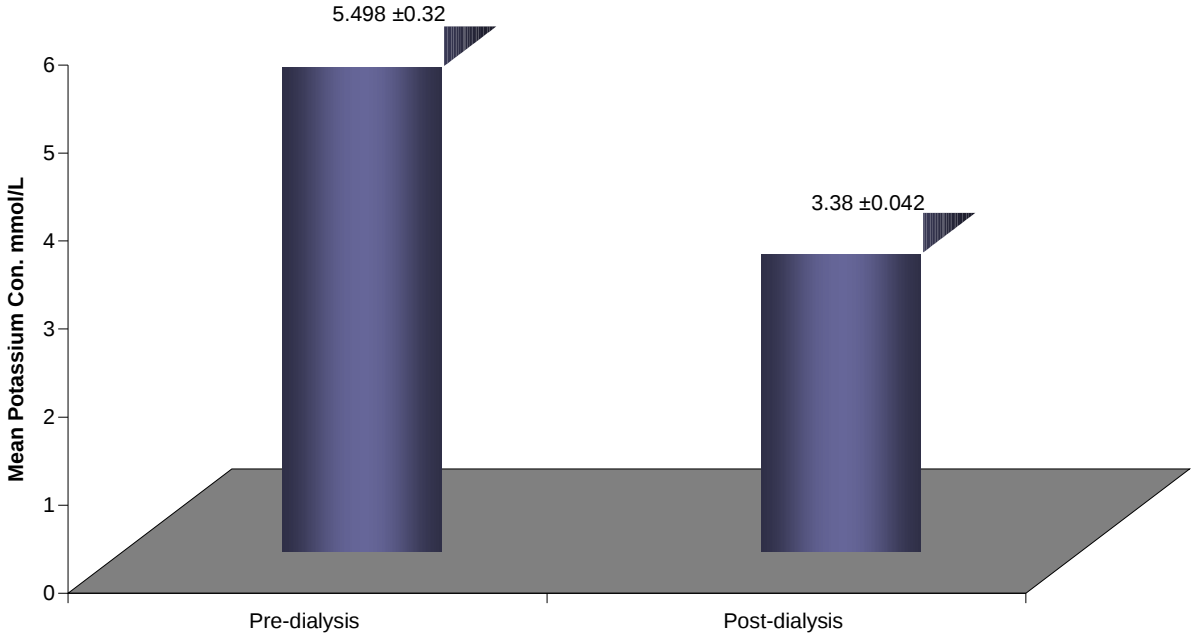
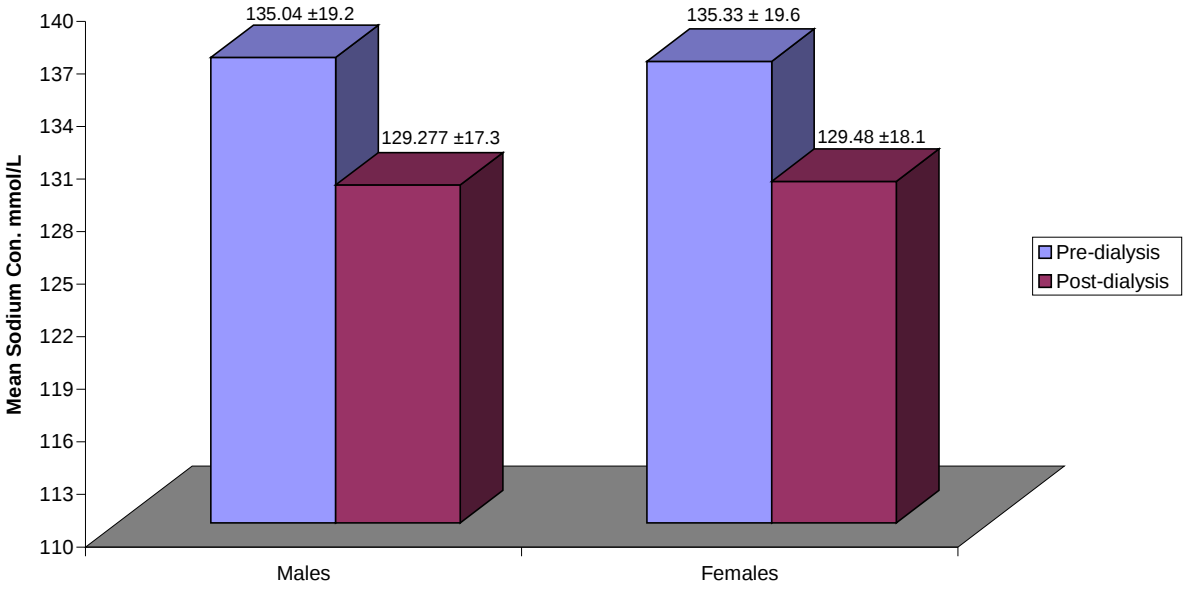


Figure (5): The mean potassium concentration in CRF patients pre and post dialysis

Table (6): The mean sodium concentration in CRF patients according to sex pre and post dialysis

Gender	Number	Sodium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
Males	47	135.042 ±19.2 (122-145)	129.277 ±17.3 (1.8-4.9)	0.320*
Females	33	135.33 ±19.6 128-150)	129.48 ±18.1 (120-142)	0.820*

Result given in mean and ± SD
Ranges between brackets
* Not significant difference at P. value <0.05



Figure(6): The mean sodium concentration in CRF patients according to sex pre and post dialysis

Table (7): The mean potassium concentration in CRF patients according to sex pre and post dialysis

Gender	Number	Potassium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
Males	47	4.874 ±0.82 (2.1-6.7)	3.38 ±0.31 (1.4-4.9)	0.001
Females	33	4.969 ±0.77 (2.7-7.0)	3.40 ±0.28 (2.1-4.40)	0.008

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

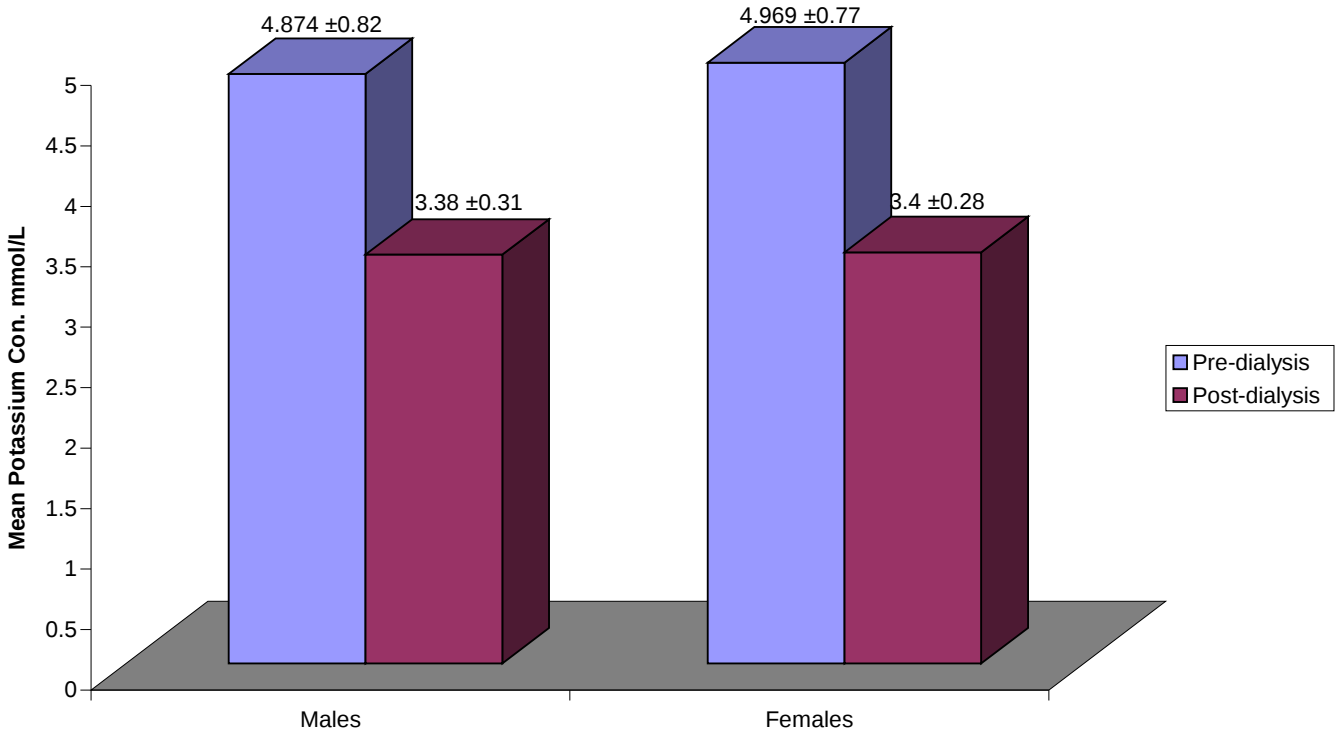


Figure (7): The concentration of potassium according to sex in CRF patients pre and post dialysis.

Table (8): The effect of age on sodium levels in CRF patient

Age (year)	Number	Sodium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
< 30 yrs	16	136.38 ±15.7 (124-145)	131.38 ±13.2 (121-138)	0.321*
≥ 30 yrs	64	134.86 ±11.2 (122-150)	128.84 ±11.2 (120-142)	0.225*

Result given in mean and ± SD
Ranges between brackets
* Not significant difference at P. value <0.05

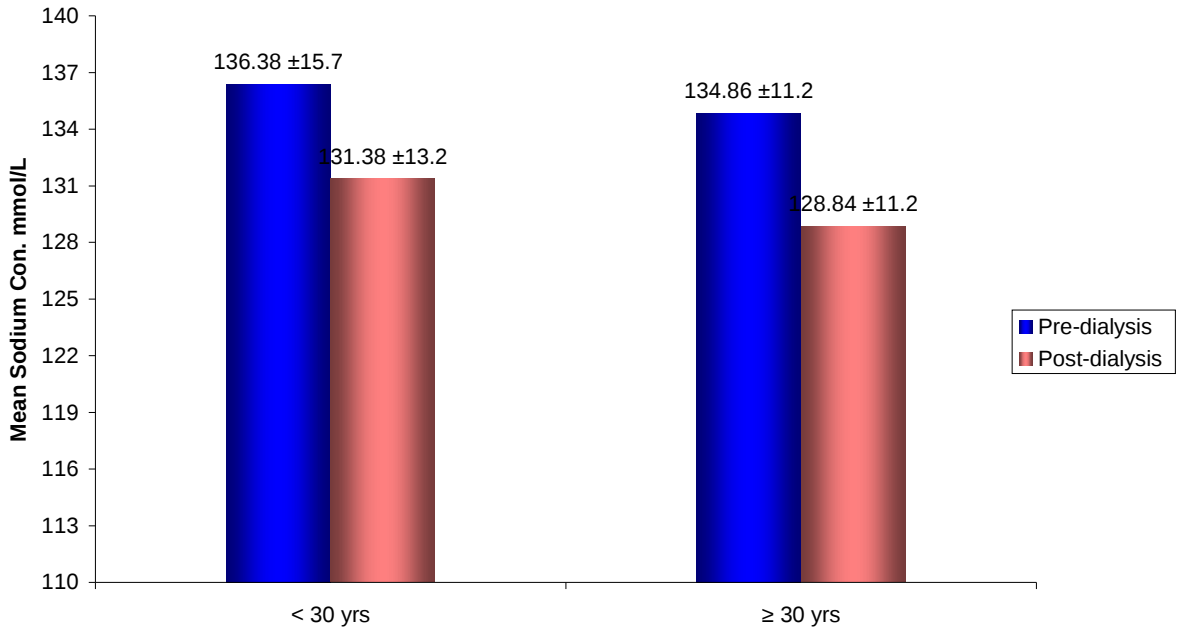


Figure (8): The effect of age on sodium levels in CRF patient

Table (9): The effect of age on potassium levels in CRF patient

Age (year)	Number	Potassium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
< 30 yrs.	16	4.64 ±0.42 (3.7-5.7)	3.58 ±0.3 (2.3-4.4)	0.001*
≥ 30 yrs.	64	4.98 ±0.49 (2.1-7.0)	3.34 ±0.01 (1.8-4.9)	0.001*

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

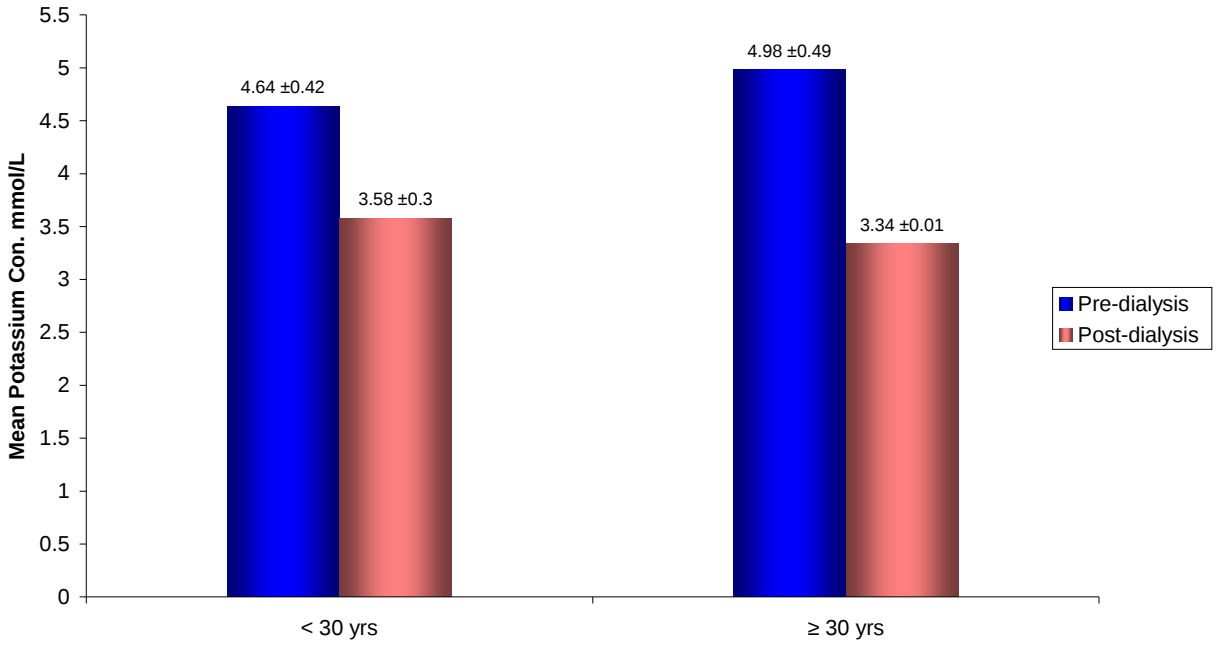


Figure (9): The effect of age on potassium levels in CRF patient

Table (10): The concentration of sodium in CRF patients according to duration of disease at haemodialysis

Duration	Number	Sodium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
< 3 yrs (14-36 months)	48	135.48 ±16.6 (122-145)	129.35 ±20.2 (120-139)	0.250*
≥ 3 yrs (37-84 months)	32	134.69 ±18.8 (125-150)	129.38 ±18.2 (117-142)	0.121*

Result given in mean and ± SD
Ranges between brackets
* Not significant difference at P. value <0.05

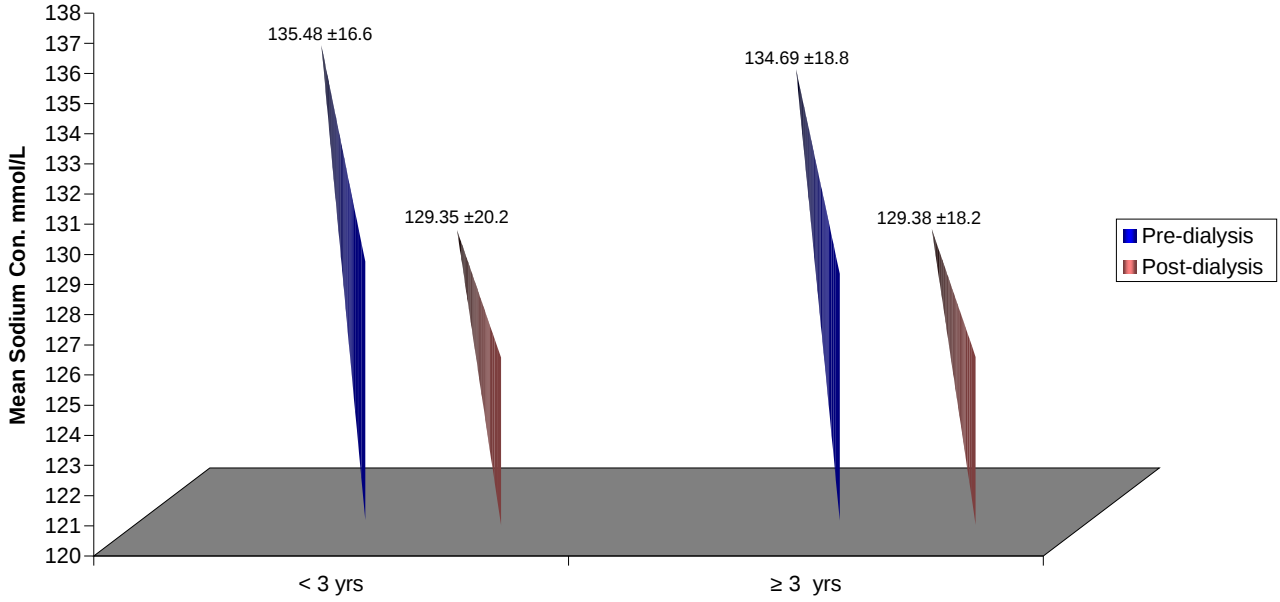


Figure (10): The concentration of sodium in CRF patients according to duration of disease at haemodialysis

Table (11): The concentration of Potassium in CRF patients according to duration of disease at haemodialysis

Duration	Number	Potassium Con. mmol/L		P. vlaue
		Pre-dialysis	Post-dialysis	
< 3 yrs (14-36 months)	48	4.76 ±0.41 (2.1-7.0)	3.302 ±0.32 (1.8-4.9)	0.002*
≥ 3 yrs (37-84 months)	32	5.144 ±0.5 (3.8-7.0)	3.518 ±0.2 (2.0-4.5)	0.001

Result given in mean and ± SD
Ranges between brackets
* Significant difference at P. value <0.05

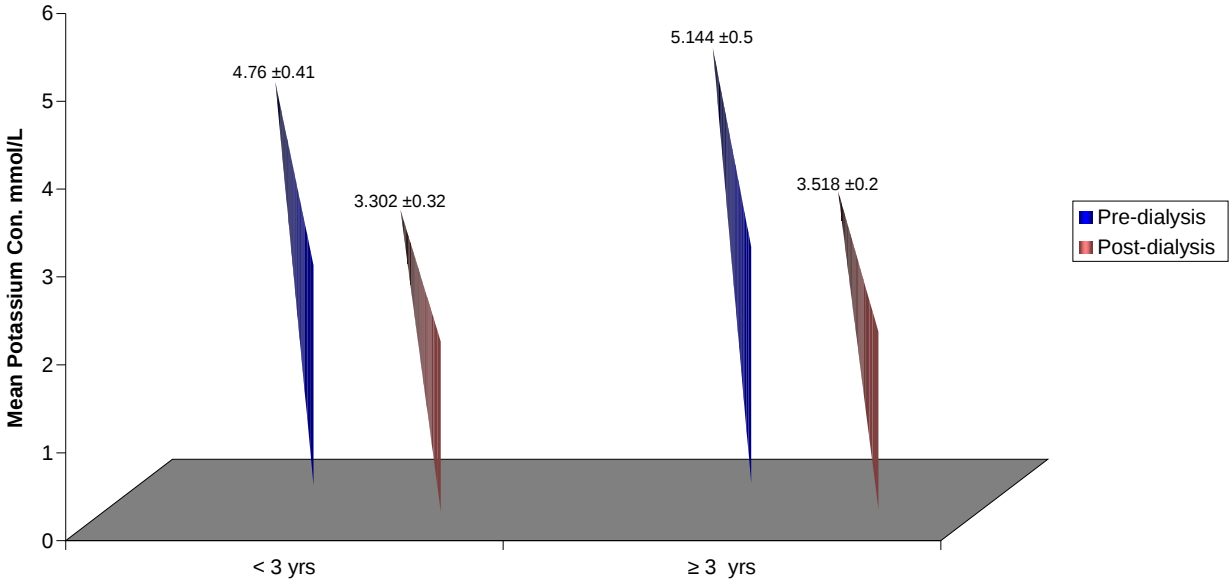


Figure (11): The concentration of Potassium in CRF patients according to duration of disease at haemodialysis

4. DISCUSSION

Renal Failure has been recognized as a major health problem occurring in almost all population of the world at a variable prevalence.

In Sudan it has been estimated from hospital records that the number of chronic renal failure patients is increasing in all socioeconomic classes and about 60% of all Sudanese renal failure- patients attending a renal dialysis centers in central states.

Renal dialysis, as a mean of sustain patient's life, was considered to have physiological impact on whole body metabolism, and major changes in metabolites and elements were reported to be occurred. In this study, we investigated the changes that may to the elements, sodium and potassium, during the course of renal dialysis, since these elements have a major role in central water balance in the body.

The preliminary investigations obtained from this study revealed that the chronic renal failure patient's participated in this study, men are more affected than women in regards with renal disease, this remarks are also reported by (lowie et al, 1989), who postulated that women with renal disease progress to end – stage renal disease more slowly than men, similarly (Mogensium and Christense,1984).Claimed that men with renal disease may hyper filtration more quickly than women with similar renal

disease and this might cause existent renal disease to progress more quickly in men relative to women.

In this study, preliminary investigation also showed that the patients with chronic renal failure participated in this study was presented with other disease; diabetes mellitus was 7.5% in patients under haemodialysis. It has been postulated that chronic renal failure has been observed to be the most serious complications of diabetes and about 7.5% of diabetic patient's are believed to end with chronic renal failure (CRF), (Raine, 1995). showed that about 36% of incidence of end- stage renal disease cases in 192 were diabetic, This study showed that about 34% of chronic chronic renal failure patient's in haemodialysis treatment were hypertensive, this remarks are in agreement with the findings of (laker,1966),who reported that cardiovascular system (CVS) is greatly affected in patients suffering from chronic renal disease in which hypertension is believed to develop in about 80% of (CRF) patient's under go renal dialysis, and atherosclerosis is common. On other hand, (Danish,1997), reported that hypertension is the major complications of recombinant human erythropoietin (rhuepo) therapy arising in at least of third of the haemodialysis population treated with the hormone.

The explanation of increased blood pressures in (CRF) patients was postulated by (Laker, 1966), who reported that increases blood pressures

is due to increase in cardiac out put as a result of increase in the peripheral flow resistance.

The most interesting observation obtained from this study is the prevalence of hepatitis (B) virus (HBV) in about 8.8% of (CRF) patient's under haemodialysis treatment. This result are in agreement with findings of (Suleiman et al, 1995) who reported that (HBV) infection has been found high among haemodialysis patient's without a history of blood transfusion.

Hepatitis B virus (HBV) infection among haemodialysis patients remains a major problem globally, and prevalence of (HBV) antibody sensitivity was reported to be high in haemodialysis population in Sudan (Michael et al, 1986).

The virus is mainly transmitted parent rally e.g. by blood transfusion (Timbury, 1986). Infection can be spread by the use of communal or inadequate sterilized syringes or needles (Timbury, 1986).

In renal dialysis units, hepatitis B virus has been a particular problem. Infection is introduced by blood transfusion required by the patients and spread from one patient's to other patient's or stuff. HBV also is said to be readily spread by sexual intercourse’s and intravenous drug abusers by sharing of needles and syringes (Collier, and Oxford, 1993).

Another causes of (HBV) presence in (CRF) patient's is the possibility of blood transfusion to same patients participated in this study due to anemia.

It is well documented that anemia is mostly represents the symptomatic clinical feature in (CRF) patient's. (Kumar, 1999), reported that in chronic renal failure, anemia is frequently present and arise chiefly as a result of an inappropriate low erythropoietin (EPO) production. The concentration of the electrolysis, Sodium and potassium in 80 chronic renal failure patient's under go haemodialysis, was presented on table (3) and figure (3a) and (3b), it appeared that sodium concentration in (CRF) patient's was not much affected even before dialysis, no significant differences were observed between the level pre and post dialysis, the pattern in potassium concentration is not the same, significant elevation in potassium concentration.

As far as potassium is concerned table (3) and figure (3b)pointed that potassium concentration in CRF patients predialysis was slightly higher than in control subjects, but after haemodialysis, a remarkable reduction in potassium concentration was observed compared with control, a reduction of about 62% was obtained. These results also reported by (Frazer, 1971). Who claimed that haemodialysis may reduce the level of potassium in CRF patients?

It is also pointed out that potassium levels in CRF male patients pre dialysis was the same as in CRF female patients, no significant difference were observed between the two levels at ($P<0.05$).

In this study age on the other hand, was found to have a profound effect on the concentration of potassium in CRF patients after dialysis, a reduction of about 30% and about 43% was found in CRF patients at an age group of less than 30 years and in an age group more than 30 year respectively (table-9 and figure -9).

The study also showed that potassium concentration in CRF patients having the disease for less than 3 years is nearly the same as in CRF patients having the disease for more than 3 years, moreover, the reduction in potassium levels after dialysis in the first age group (less than 3 year) was about 44% from the initial value (and about 41% in the second age group (more than 3 years). This results may suggest that disease duration has no effect on the level of potassium in CRF patients undergo renal dialysis.

Although a remarkable in potassium was observed in CRF patients after hamodialysis, state of hypokalemia was not noticed or reached, hypokalaemia occurs when the level of potassium depleted to less than 3mmol, Jones, 1970. Hypokalaemia and potassium depletion are not synonymous because low plasma potassium concentration can occur owing to redistribution from the extra cellular fluid, rather than as a result

of loss, in this study, potassium concentration was decreased following haemodialysis to about 3.338 mmol/l.

The causes of potassium depletion and hypokalaemia were reported by (laker, 1996). As results from several factors including internal redistribution in alkalotic state in which the potassium movement from the extra cellular fluid (ECF) to intracellular fluid (ICF) is governed by the H^+ ions, that H^+ ions move out of cells in exchange for extra cellular potassium. Insulin infusion or increased insulin secretion following a glucose load was found to increase potassium cellular uptake, mainly by the liver and muscle, by increasing sodium- potassium AT pase activity. Renal losses are well recognized to be one of the causes of hypokalaemia specially when taken diuretic therapy, especially in hypertension patients and elderly.

Gastrointestinal losses is also considered to be gastrointestinal one of the cause of hypokalaemia, in which the secretion of gastrointestinal tract contain a relatively large proportion of (ECF) potassium and therefore vomiting and aspiration of gut secretions can cause hypokalaemia. Gastrointestinal secretions also reported to contain sodium and if this is not replaced adequately hyper aldosteronism will develop, this will have the effect of increasing potassium depletion, Aldosteron is well recognized as a major regulatory potassium excretion and intracellular urinary loss (Morgan, 1984).

The study also showed that sodium concentration in both sexes was nearly the same in pre- dialysis as in post-dialysis, but it significantly reduced to about 4.5% of control value after dialysis.

The ages of CRF patients seemed to have no effect on the concentration of sodium after dialysis in which no statistical difference at ($P<0.05$) were obtained between the two age groups, similarly the duration of the renal disease was also showed no effects on the levels of Sodium during renal dialysis, as the level of Na+ in patients suffering the disease for less than 3 years is the same as in CRF patients having the disease more than 3 years.

Potassium, on the other hand showed that before renal dialysis, their slight increase in CRF over control value, but after dialysis, remarkable significant reduction was observed from control value, a reduction of about 62% was obtained, these results were also reported by (Frazer. 1971). In that the haemodialysis ways reduce the level of potassium is CRF patients. It is also pointed out that the K^+ level in males CRF patients before dialysis was the same as in females CRF patients before dialysis, the levels in both sexes were statistically reduced after dialysis.

5. CONCLUSION AND RECOMMENDATION

5.1.Conclusion:

According to the results obtained from this study, we can conclude that men were more affected than women with CRF in regards with renal disease. The study also revealed that patients may presented with other diseases such as hypertension, DM and HBV.

The study also showed that sodium concentration in CRF patients was not much affected even before dialysis, no significant differences were observed between the level pre and post dialysis. But after dialysis slight reduction occurred.

Potassium on the other hand, slight increase in CRF patients, over control value pre dialysis but after dialysis remarkable significant reduction was observed compared with control value.

The study also showed that sodium concentration in both sexes was nearly the same in pre – dialysis as in post –dialysis. While, potassium concentration in males patients before dialysis was the same as CRF females patients. The ages of CRF patients seemed to have no effect on the concentration of sodium after dialysis, age on the other hand, was found to have a profound effect on the concentration of potassium in CRF patients after dialysis.

5.2. Recommendation

1. HBV was found among CRF patients under this study, this might be due to contamination of the dialysis machine. Care must be taken to avoid this contamination.
2. CRF patients still complain that, they can not afford the high cost of having the continuous dialysis. Intervention of the government must take place to reduce this cost.
3. Considerable number of CRF patients claimed that they acquired the CRF disease from another disease, such as D.M, hypertension so attention must paid by people suffering these disease and periodical medical check up must be considered.

REFERENCES

1-Berner L.M , The kidney physiology , 1983 ; p. 928.

2-Bishop ML, Duben EJ, Fody EP. Clinical Chemistry Principle, Procedures and Correlation, 4th ed, Washington: Lipincott Williams and Wilkins; 2000.

3-Collier I, Oxford J; Human virology for students of medicine. Dentistry and micro, New York, Tokyo, Oxford Univ, Press 1993.

4-Danish MI : Renal disease “Short Textbook of Medical Diagnosis and Management “, 2nd ed, Karachi, Danish Publications, 1997, p. 217-29.

5-Edward C.R, Bouchier I.A, Haslett C, Chilver E; Disease of the kidney and geneto-urinary system, Davidson's principle Practice of medicine 17th ed , London ELBS/ 1986; p. 52

6-Fischbach F. A manual of Laboratory and Diagnostic Test, 6th ed, Philadelphia. Baltimore. New York: Lippincott; 2000. P. 348 – 355.

7-Frzer, S.C,C,The interpretation and biochemical management of abnormal plasma levels of electrolytes, particularly Sodium and

Potassium. Annals of clinical biochemistry. 1971. P.8, 37 – 42.

8- Gangong, W. F, Medical physiology, 16th ed. Appleton a Lange pp 635-39 (1993).

9-Jeremy Leavy, J.M., Edwin B. Oxford handbook of dialysis, Oxford University press (2001.

10 -Kaplan A, Jack R, Ophein KE, Toivola B, Lyon AW. Clinical Chemistry Interpretation and Techniques, 4th ed, Philadelphia: Lippincott Williams and Wilkins; 1999.

11- Kumar P, Clark, M; Clinical medicine Textbook for medical students and doctors, 3rd ed, 1999.

12-Laker, M.F. Clinical Biochemistry for Medical studeSaunders Company LTd, 1996.

13-Lowie, C.C; porl, F.K; WALF;R.A; Disparities incidence of diabetic end-stage renal disease according to race and t diabetes; England J of Med. 1989 : p. 321,1074. A; Acute Renal Failure, Emedicine, July 12. 2002.

14-Macsween RM, Whraly K: The kidney and urinary tract, “Muir,s Text book of Pathology” , 13 the ed., London ELBS, 1995, P. 892-933.

15-Maxwell, M.H, Kleeman, C.R, Narions, R.G. Clinical Disorder of Fluid and Electrolyte Metabolism, 4th ed, New York MC Coraw Hill; 1987.

16-Mayne P. D. Clinical chemistery in diagnosis and treatment 6th ed, London Edward Arnold, 1994; 225, 233, 4.

17-Mogensiu-m, A. S; Christensen, S. A; Kidney and urinary tract. Muirs textbook of pathology; 13 ed, London, Elbs, (1984).

18-Mickael, C; M; EL Tigani, A, Khalid, I.O; Hyams, K; Heptitis B and C in Juba, Southern Sudan. Results of a serosurvey trans. Roral Soc. Trop. Med., and Hyg. 1994: 88, 534-536.

19-Morgan, D.B ; Electrolyte disorders. Clinics in endocrinology and metabolism. 1984: P.13, 231 – 434.

20-Raine, A.E; the rising tide of diabetic nephropathy, the warning before the food, Nephrol Dial Transport 1995: 10,460-461.

21-Stanley L Rdins ; Ramzi S.Cotran ;V inay Kumar pathologic basis of disease; 3rd edition,1984.

22-Suliman, S.M; Fessaha, S; ELsading, M; ELhadi, M.B, Lombert, S; present of hepatitis C virus infection in haemodialysis patients in Sudan, Saudi J. kidney Dis-Transplant, 1995. P. 6,154-156.

23-Sukkar M.Y,Munshid H.A.EL, Ardawi M.S.M, Concise Human physiology, 3rd edition, 1997.

24- Timbury, M;C; Notes on medical virology , 8th ed. Churchill Livingstone ,Edinbura, London 1986 .

25-Juhap kokko and Richard L. Tannen. Fluids and Electrolytes, W.B Saunders Philadelphia. 1986; P: 16-17, 164-165.

26-Verrelli: M, Chronic renal failure, Emedicine, Jan 20, 2004.

27-Whiteby L G, Percy I w, Smith A F. Lecture notes on clinical chemistry, 3th ed. London: Black well scientific publications 1987.

28-William JM, Stephen KB. Clinical Chemistry, 5th ed, China: Mosby; 2004.

29-Zilva JF, Peter R, Pannall RP. Clinical Chemistry Diagnosis and Treatment, 6th ed, London: Lloyd-Luke; 1993. P. 26, 37-39, 61 . 65.

APPENDIX 1

Questionnaire:

A-General information:

1- Name ……

2- Age ……

3-Sex ……

4- Occupation ……..

B-Present history of disease:

1-Hypertension

2-Diabetes mellitus

3- Hepatitis B virus infection

4-Renal stone

5-Tuberculosis

C- Investigations:

1-Plasma Sodium pre and post haemodialysis.

2-Plasma potassium pre and post haemodialysis .

APPENDIX 2

Row Data of Chronic Renal Failure

sn	gender	age	duration	Na+ predialysis	Na+ postdialysis	K+ predialysis	K+ postdialysis	history
1	1	49	18	139	126	6.0	3.1	-
2	2	63	36	135	131	6.1	3.1	T.B
3	1	40	14	128	122	5.2	2.8	H.T
4	2	39	19	137	121	5.0	2.6	-
5	1	71	73	134	120	5.8	2.7	HBV
6	2	42	84	138	135	4.2	3.6	-
7	2	32	28	136	132	2.7	2.5	-
8	1	63	29	143	139	4.3	3.2	D.M + H.T
9	2	38	41	130	126	4.5	3.5	D.M + H.T
10	2	50	33	134	126	4.4	4.0	H.T
11	1	23	36	142	136	4.1	3.5	-
12	1	40	24	145	139	4.3	3.7	-
13	1	25	24	140	133	4.6	4.0	-
14	1	29	24	136	123	5.0	4.2	H.T
15	1	53	39	134	128	4.8	4.3	-
16	1	40	84	135	117	4.0	2.0	H.T
17	1	63	24	145	142	3.8	3.5	-
18	1	46	36	122	120	3.6	3.0	R.S
19	2	55	36	130	120	3.2	2.2	H.T
20	1	25	49	130	123	5.7	3.6	H.T
21	2	42	84	146	138	4.5	3.2	-
22	2	65	19	128	120	4.8	3.0	H.T + R.S
23	1	60	37	146	141	6.1	3.5	HBV
24	1	61	24	139	133	4.3	3.9	D.M
25	2	33	24	141	134	4.5	3.9	D.M
26	2	76	28	130	128	5.7	2.8	-
27	1	47	21	132	124	5.3	3.0	-
28	1	73	72	131	126	6.0	3.2	H.T
29	1	72	37	135	121	4.1	2.5	-
30	1	39	18	134	125	4.8	2.8	-
31	1	63	22	138	129	5.3	3.0	-
32	2	42	19	133	126	7.0	3.2	HBV
33	1	34	47	131	126	4.6	2.7	-
34	1	63	32	135	127	6.0	2.8	H.T
35	2	37	16	130	129	4.3	2.4	H.T
36	2	48	28	132	125	4.4	2.5	-
37	2	35	31	134	125	6.2	2.7	H.T + R.S
38	2	60	34	133	125	5.4	2.4	H.T
39	2	37	40	128	124	4.0	2.1	H.T + R.S
40	1	27	18	130	121	5.1	2.6	H.T
41	1	29	17	124	124	5.2	2.9	HBV
42	2	31	72	142	138	5.0	4.1	H.T
43	2	27	27	140	136	4.8	4.4	-
44	1	50	72	125	120	4.7	4.1	H.T
45	1	23	48	138	132	5.0	3.9	-
46	1	28	72	139	136	4.8	4.2	-

47	1	59	19	138	133	4.4	3.8	H.T+Nephropathy
48	2	32	35	140	134	4.0	3.2	-
49	2	31	72	142	138	7.0	4.5	H.T
50	2	27	27	140	136	4.5	3.8	-
51	1	50	73	125	120	5.6	2.8	-
52	1	23	48	138	132	4.0	3.4	-
53	1	28	72	139	136	3.8	3.0	-
54	1	60	24	141	136	4.6	4.0	H.T
55	1	16	36	145	138	4.6	3.9	-
56	2	60	72	150	142	4.5	3.8	H.T
57	2	60	21	140	133	7.0	3.9	-
58	1	20	24	130	126	4.5	3.5	-
59	1	60	24	144	136	2.1	1.8	D.M+ Heart
60	1	27	36	140	133	3.7	2.3	-
61	1	51	38	136	122	5.5	3.7	HBV
62	1	39	61	135	132	4.6	3.4	H.T
63	1	47	73	132	129	6.7	2.9	HBV + T.B
64	1	49	23	136	133	4.7	3.7	-
65	1	33	31	133	129	5.0	3.9	R.S
66	1	18	21	140	138	4.8	4.1	-
67	1	36	32	136	130	5.5	4.9	-
68	2	62	42	128	136	4.8	4.4	-
69	2	34	36	134	120	5.4	4.3	H.T+ D.M+Heart
70	1	65	53	126	130	6.0	4.9	H.T
71	2	67	70	137	120	5.4	4.3	H.T
72	2	54	61	130	129	5.4	4.2	-
73	1	74	72	135	131	5.7	4.2	-
74	1	31	38	137	131	6.4	3.6	HBV
75	2	55	32	130	123	4.4	4.1	H.T + R.S
76	2	51	18	138	131	4.2	3.8	-
77	1	73	68	130	128	5.4	2.4	H.T
78	2	68	37	139	133	6.0	3.9	-
79	2	31	29	140	133	4.5	2.9	-
80	2	54	36	128	126	6.2	2.9	R.S

Row Data of Control

sn	gender	age	Na+	K+
1	1	42	136	3.7
2	2	35	138	3.5
3	1	37	138	4.3
4	1	40	138	4.2
5	2	35	137	4.4
6	1	30	137	3.9
7	1	28	134	4.3
8	2	34	138	3.5
9	2	43	140	3.5
10	2	38	139	4.2
11	2	31	140	4.0
12	1	29	140	4.2
13	2	39	136	4.5
14	1	42	138	4.3
15	1	48	138	4.5
16	1	42	138	4.5
17	1	49	136	4.3
18	1	38	138	4.0
19	2	27	140	3.8
20	1	44	142	4.3