1. 1Introduction

Normal renal function is very important for homeostasis, so much so, that situations in which renal functions are impaired can be life threatening. Diseases of the kidneys are among the most important causes of death and disability in many countries throughout the World. (Bijlani ,2004)

The National Kidney Foundation in India states that, kidney diseases rank 3rd amongst life threatening disease, after cancer and heart disease. About 200,000 persons go in to terminal kidney failure every year. Million more suffer from lesser forms of kidney disease.(National kidney foundation ,2006)

In United States 35,000 deaths are attributed yearly to renal diseases. The rate of kidney disease mortality in the United States has increased by 52% in the past 16 years and continues to be higher in blacks than whites.(Waknine,2007) Morbidity however is by no means insignificant. Millions of persons are affected annually by non fatal kidney diseases, most notably infections of the kidney or lower urinary tract, kidney stones and urinary obstruction. Twenty percent of all women suffer from infection of the urinary tract of kidney at sometime in their lives and at least 1% of the U.S. population develops renal stones. (Charles, 2004)

The presence of chronic kidney disease confers a markedly increased risk of cardiovascular disease, and people with CKD often have other risk factors for heart disease, such as hyperlipidemia. The most common cause of death in people with CKD is therefore cardiovascular disease rather than renal failure.Aggressive treatment of hyperlipidemia is warranted.

In CRF plasma total calcium is fall (Martin A,2006).In chronic renal failure there is a steady and continued decrease in renal clearance or

glomerular filtration rate (GFR), which leads to the gathering of urea, creatinine and other chemicals in the blood(NourAlamin *et*al,2014).

Renal diseases are associated with a variety of haemopoietic changes. Anemia parallels the degree of renal impairment and its most important cause is failure of renal erythropoietin secretion. Other factors include chronic blood loss, hemolysis and bone marrow suppression by retained uremic factors. (Dodds and Nicholls, 1983)

The aim of the present study is to find out the hematological and clinical changes in chronic renal failure patients.

1.2. Literature Review

1.2.1Chronic Kidney Disease:

1.2.1.1 The Kidneys:

The kidneys are paired' bean shaped organs located retroperitoneally on either side of spinal column(Micheal,*et al*,2005). Each kidney of adult human weight about 150 gram (Guyton and Hall,2006) .

1.2.1.2 Renal anatomy:

Macroscopically a fibrous capsule of connective tissue encloses each kidney. Two regions can be clearly discerned an outer region called the cortex and an inner region called the medulla. The pelvis can also be seen. It is basin like cavity at the upper end of the ureter in to which newly formed urine passes. The bilateral ureters are thick walled canals, connecting the kidneys to the urinary bladder (Micheal ,*et al*,2005).

The nephrons are functional units of the kidney that can only be seenmicroscopically. Each kidney contains approximately 1million nephrons. Eachnephron is complex apparatus comprised of five basic parts:

1.2.1.3 The glomerulus:

A capillary surrounded by the expanded end of renal tubule known as Bowman's capsule. Each glomerulus is supplied by an afferent arteriole carrying the blood in and efferent arteriole carrying the blood out. The efferen arteriole branches in to peritubularcapil (Philip D.M, *et al*,1994).

1.2.1.4 The proximal convoluted tubules:

Located in the cortex, receive filtrate from the glomerular spaces. Convolution increases the tubular length and therefore contact between the luminal fluid and the proximal tubular cells, thus facilitating more solute reclamation than would occur if the loops were shorter(Philip D.M, *et al*,1994).

1.2.1.5 The long loop of Henle:

It comprised of thin descending limb which spans the medulla. And the ascending limb, which is located in both the medulla and the cortex, comprised of a region that is thin and thick (Micheal,*et al*,2005).

1.2.1.6 The distal convoluted tubules:

Located in the cortex, important for fine adjustment of luminal fluid, lie near the afferent arterioles with the juxta glomerular apparatus between them. The production of renin by the latter is modified by flow in these blood vessels (Philip , *et al*, 1994).

1.2.1.7 Collecting ducts:

Formed by two or more distal convoluted tubules as they pass back down through the cortex and the medulla to collect the urine that drains from each nephron.

Collecting ducts eventually merge and empty their contents in to renal pelvis (Micheal ,*et al*,2005).

1.2.1.8 Renal failure:

Failure of renal function may occur rapidly , producing the syndrome of Acute Renal Failure (ARF), or develops insidiously, often over many years producing Chronic Renal Failure (CRF) (William and Stephen ,2004)

1.2.1.9 Chronic Renal Failure (CRF):

Is progressive loss in renal function over a period of months or years. It is differentiated from acute kidney disease in that the reduction in kidney function must be present for over 3 months.

Chronic kidney disease is identified by a blood test for creatinine. Higher levels of creatinine indicate a lower glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products (National Kidney Foundation,2002)

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1.2.1.10 Signs and symptoms

CRFis initially without specific symptoms and is generally only detected as an increase in serum creatinine or protein in the urine. As the kidney function decreases:

Hypertension , urea accumulation,hyperkalemia ,erythropoietin synthesis is decreased, fluid volume over load –symptoms may range from mild edema to life therating pulmonary edema ,hyper phosphatemia(Hruska, *et al*.,2008),hypocalcemia, metabolic acidosis, iron deficiency anemia,atherosclerosis and more likely to develop cardiovascular disease than the genral population and sex dysfunction(Vecchio ,*et al,* 2010)

1.2.1.11 Dagnosis:

Abdominal ultrasound, in which the size of the kidneys is measured, is commonly performed. Another diagnostic clue that helps differentiate CKD from ARF is a gradual rise in serum creatinine (over several months or years) as opposed to a sudden increase in the serum creatinine Additional tests may include nuclear medicine MAG3 scan to confirm blood flows and establish the differential function between the two kidneys. DMSA scans are also used in renal imaging; with both MAG3 and DMSA being used chelated with the radioactive element Technetium-99.

1.2.1.12 Stages:

All individuals with a glomerular filtration rate (GFR) <60 mL/min/1.73 m2 for 3 months are classified as having chronic kidney disease, irrespective of the presence or absence of kidney damage. The rationale for including these individuals is that reduction in kidney function to this level or lower represents loss of half or more of the adult level of normal kidney function, which may be associated with a number of complications(National Kidney Foundation ,2002) All individuals with kidney damage are classified as having chronic kidney disease, irrespective of the level of GFR. The rationale for including individuals with GFR > 60 mL/min/1.73 m2 is that GFR may be sustained at normal or increased levels despite substantial kidney damage and that patients with kidney damage are at increased risk of the two major outcomes of chronic kidney disease:

loss of kidney function and development of cardiovascular disease.

(National Kidney Foundation ,2002) .

The loss of protein in the urine is regarded as an independent marker for worsening of renal function and cardiovascular disease. Hence, British guidelines append the letter "P" to the stage of chronic kidney disease if there is significant protein loss

Stage 1

Slightly diminished function; kidney damage with normal or relatively high GFR (≥90 mL/min/1.73 m2). Kidney damage is defined as pathological abnormalities or markers of damage, including abnormalities in blood or urine test or imaging studies. (National Kidney Foundation ,2002) .

Stage 2

Mild reduction in GFR (60–89 mL/min/1.73 m2) with kidney damage. Kidney
damage is defined as pathological abnormalities or markers of damage, including
abnormalities in blood or urine test or imaging studies.

Stage 3

Moderate reduction in GFR (30–59 mL/min/1.73 m2). British guidelines
distinguish between stage 3A (GFR 45–59) and stage 3B (GFR 30–44) for purposes of screening and referral.

Stage 4

Severe reduction in GFR (15–29 mL/min/1.73 m2) Preparation for renal replacement therapy.

Stage 5

Established kidney failure (GFR <15 mL/min/1.73 m2, permanent renal replacement therapy (RRT), or end stage renal disease (ESRD) (National Kidney Foundation ,2002)

1.2.1.13 Treatment:

The goal of therapy is to slow down or halt the progression of CKD to stage 5. Control of blood pressure and treatment of the original disease, whenever feasible.

Generally, angiotensin converting enzyme inhibitors (ACEIs) or angiotensin II receptor antagonists (ARBs) are used, as they have been found to slow the progression of CKD to stage 5(Ruggenenti , *et al*,1999). Currently, several compounds are in development for CKD. These include, to, bardoxolone methyl, olmesartanmedoxomil, sulodexide, and avosentan.

Replacement of erythropoietin and calcitriol, two hormones processed by the kidney, is often necessary in people with advanced disease. Guidelines recommend treatment with parenteral iron prior to treatment with erythropoietin. A target hemoglobin level of 9–12 g/dL is recommended . (Clement, *et al.,* 2009) Phosphate binders are also used to control the serum phosphate levels, which are usually elevated in advanced chronic kidney disease.

When one reaches stage 5 CKD, renal replacement therapy is usually required, in the form of either dialysis or a transplant.

1.2.1.14Prognosis:

The prognosis of patients with chronic kidney disease is guarded as epidemiological data has shown that all cause mortality increases as kidney function decreases. The leading cause of death in patients with chronic kidney disease is cardiovascular disease,(Perazella and Khan ,2006).

While renal replacement therapies can maintain patients indefinitely and prolong life, the quality of life is severely affected(Francisco and Piñera ,2006).

Increases the survival of patients with stage 5 CKD significantly when compared to other therapeutic options; however, it is associated with an increased short-term mortality due to complications of the surgery. Transplantation aside, high intensity home hemodialysis appears to be associated with improved survival and a greater quality of life, when compared to the conventional three times a week hemodialysis and peritoneal dialysis(Pierratos ,*et al*,2005).

1.2.1.15 Cancer risk:

Patients with end-stage renal disease are at increased overall risk for cancer. This risk is particularly high in younger patients and gradually diminishes with age. (Maisonneuve,*et al*,1999).

1.2.2Blood

1.2.2.1 Blood Function

Blood is a fundamental component of life. Within the adult body approximately 4 to 5 liters of blood circulates continuously through vessels. As its moves from lungs and heart feed the cells by nutrients include oxygen which is most basic elements necessary for survival. Also blood picks up

wastes such as carbon dioxide, that ultimately be removed from the body as the blood back to lungs (Rogers, 2011).

1.2.2.2Blood composition:

Blood composed of cells carried in plasma these cells are erythrocytes which transport oxygen, leukocytes act as immune defense and platelets for blood clotting. Plasma contain protein and water. Plasma Contains 3 types of proteins: albumin, globulins and fibrinogen. Use in maintain osmotic pressure, immune defense and blood clotting respectively (Fox, 2006).

All blood cells are divided into three lineages:

Erythroid cells are the oxygen carrying red blood cells. Both reticulocytes and erythrocytes are functional and are released into the blood. In fact, a reticulocyte count estimates the rate of erythropoiesis. Lymphocytes are the cornerstone of the adaptive immune system. Myelocytes, which include granulocytes, megakaryocytes and macrophages and are derived from common myeloid progenitors, are involved in such diverse roles as innate immunity, adaptive immunity, and blood clotting.(Fischbach and Dunning, 2009).

The proliferation and self renewal of these cell depend on growth factors. One of the key players in self-renewal and development of haematopoietics cells is stem cell factor (SCF). Absence of this factor is lethal but there are other important glycoprotein growth factors, which regulate the proliferation and maturation, such as IL-2, IL-3, IL-6, IL-7. Other factors, termed colony stimulating factors (CSFs), specifically stimulate the production of committed cells. Three CSFs are granulocyte-macrophage CSF (GM-CSF), granulocyte CSF (G-CSF) and macrophage

CSF (M-CSF). These stimulate granulocyte formation and are active on either progenitor cells or end product cells (Edward *et.al*,2005).

1.2.2.3A Complete Blood Count will normally include:

1.2.2.3.1Red cells:-

Total red blood cells . The number of red cells is given as an absolute number per litre.

1.2.2.3.2Hemoglobin:

The amount of hemoglobin in the blood, expressed in grams per decilitre. (Lowhemoglobin is called anemia.)

1.2.2.3.3Hematocrit or packed cell volume (PCV):

This is the fraction of whole blood volume that consists of red blood cells.(Bijlani ,2004)

1.2.2.3.4Red blood cell indices:

1.2.2.3.4.1MCV:

The mean red cell volume (MCV) provides information on red cell size. It is measured in femtolitres(fl) and is determined from the PCV and electronically obtained RBC count. It can be calculated as follow:

$$MCV = \frac{PCV}{RBC \times 10\text{-}12\ L/L}$$

A femtolitre (fl) is 10–15 litre.

Interpretation of MCV values:

There is some variation in reference ranges for MCV depending on the method used by manufacturers of blood cell analyzers to obtain the MCV value and how an instrument has been calibrated. (Cheesbrough, 2006).

1.2.2.3.4.2MCH:

The MCH gives the amount of haemoglobin in picograms (pg) in an average red cell It is calculated from the haemoglobin and electronically obtained RBC count:

$$MCH = \frac{Hb\ g/L}{RBCs \times 10\text{-}12\ /L}$$

A picogram (pg) is 10–12 of a gram(Cheesbrough , 2006).

1.2.2.3.4.3 MCHC:

gives the concentration of haemoglobin in g/l in 1 litre of packed red cells. It is calculated from the haemoglobin (Hb) and PCV MCHC = $\frac{\text{Hb g/L}}{\text{HCT L\L}}$

If using g/dl divide the g/l figure by 10. (Cheesbrough, 2006).

1.2.2.3.5White cells:

Are formed in the bone marrow from common precursor cells.it consist many types (neutrophils, basophil, esinophil,monocyte and lymphocyte).

Main functions is protect body from infection mainly it increase with infection and cancers also it decrese some infections and bone marrow failure. (Lewis et.al,2001)

1.2.2.3.6Platelets

1.2.2.3.6.1 Platelet production

Platelets are produced in the bone marrow by fragmentation of the cytoplasm of megakaryocytes, main function is formation of mechanical plague as response to injury. Mainly it increase with infections and haemorrage and decrese with cancers and some drugs.(Hoff brand ,2006)

1.2.3Urea:

Urea(H_2N-CO-NH_2) is the main end product of protein of (amino acids) metabolism**.(**Bishop2005). Liver is the only site for urea formation , then urea is transported in the blood to the kidney to be excreted in urine.(Oraby,2005)

Most of the urea in the glomerular filtration is excreted in the urine, although up to 40% is reabsorbed by passive diffusion during passage of the

filtrate through the renal tubular, the amount reabsorbed depends on urine flow rate and degree of hydrate, small amount of urea(less than10% of the total) are excreted through the gastrointestinal(GI) tract and skin, the concentration of urea in the plasma is determined by renal function and perfusion, the protein catabolism**.(**Bishop,2005)

1.2.3.1 Pathophysiology:

An elevated concentration of urea in the blood is called azotemia ,very high plasma urea concentration accompanied by renal failure is called uremia, or the uremic syndrome , this condition is eventually fatal if not treated by dialysis or transplantation, conditions causing increased plasma urea are classified according to cause into three main categories: Prerenal, renal, postrenal.(Bishop ,2005)

1.2.4Creatinine:-

Creatinine is synthesized primarily in the liver from arginine glycine'and methionine 'it then transported to other tissue such as muscle.
Whenever it is converted to phosphocreatine which serves as high energy source cratine phosphate loses phosphoric acid and creatine leses water to form creatinine which passes into plasma.Creatinineis released into the circulation at relatively constant rate that has been shown to be proportional to individually muscle mass.It is removed from circulation by glomerular filtration and execreted by proximal tubule. Small amounts may also be reabsorbed by the renal tubules.(Bishop,2005)

1.2.4.1Causes of high serum creatinine :-

Serum creatinine tend to be higher in subject with large muscle mass as renal causes, reduce blood flow to the kidney ,blockage of urinary tract .(Kamal and Salam ,2007)

1.2.5Calcium

1.2.5.1CalciumPhysiology:

The investigators were able to show that blood-ionized Ca2 was closely regulated and had a mean concentration in humans of about 1.18 mmol/L. Because decreased ionized Ca2 im pairs myocardial function, it is important to maintain ionized Ca2 at a near normal concentration during sur gery and in critically ill patients. Decreased ionized Ca2 concentrations in blood can cause neuromuscular irritability, which may become clinically apparent as irregular muscle spasms, called **tetany.** **(**Bishop,2005)

1.2.5.2Regulation

Three hormones, PTH, vitamin D, and calcitonin, are known to regulate serum Ca2 by altering their secretion rate in response to changes in ionized Ca2

1.2.5.3Distribution:-

About 99% of Ca2 in the body is part of bone. The re maining 1% is mostly in the blood and other ECF**.(**Bishop,2005)

1. 2.6The Rationale

Relatively little is known about the development and progression of anemia in patients with renal failure. Consequently,one can not determine precisely the optimal frequency at which Hb levels should be monitored. Isuggest the renal failure decrease the level of erythropoietin and effect on erythropoiesis ,and lead to anaemia. Also kidney is affected by dialysis so we measured its function such as urea creatinine and calcium.

1.2.7 Objectives

1.2.7.1General objective:

To determine The effect of renal failure in complete blood count, urea creatinine and calcium among CRF patients before haemodialysis.

1.2.7.2Specific objectives:

1.To determine Hb concentration, Hct, RBCs count, WBCs count and platelet count in CRF patients and controls.

2.To determine the effect of duration of renal failure in Hb concentration and RBCs count in CRF patients.

3.To determine of the affect of renal failure in urea ,creatinine and calcium in CRF patients and controls.

4.To investigate the distribution of CRF patients according to age .

5-Toinvestigate the distribution of CRF patients according to gender .

6. .To investigate the distribution of CRF patients according to cause of disease .

2. Material and Method

2.1Study design:-

This study designed to investigate the effect of renal failure on Complete blood count.,urea ,creatinine and calcium in 50 renal failure patients and 50 control in Atbara town.

2.2 Study area:-

The study was done in AtbaraTeatching Hospital which is located in the north of sudan and north of the capital Khartoum through the period from may to October;2018.

2.3Study population:-

The study population comparises tow groups of patient and control samples . 50 patientsdiagnosised with CRF 50 healthy samples used as control in Atbara town.

2.4 Inclusion criteria:-

All patients diagnosis with chronic renal faliure.

2.5 Exclusion criteria:-

Patients suffering from any disease other than CRF that could affect CBC urea creatinine and calcium such as acute or chronic infalamation ,dehydration.

2.6 Blood Sampling:-

5 ml of venous blood was taken from each participant 2.5mL transferred into an EDTA For CBCand 2.5ml was added to lithumheparinecontainer for plasma analysis . The sample was then sent to the lab as early as possible (maximum 3 to 6 hours). For hematological

parameters were done by the automated Analyzer(Mindary bc3000).also urea ,creatinine ,calcium were done by chemical analyzer (A15).

2.7 Data collection tools:

The data will be collected by using questionnaire and medical files.

2.8 Ethical approval:

Consent of selected individuals to the study was taken after being informed with all detailed objectives of the study.

2.9 Principle of method used:

2.9.1 Complete blood count (CBC):

Principle of Mindary:

The Mindaryis hematology automated analyzer used to quickly perform full blood counts and it made by Mindary Corporation Principles of measurement .

Diluted blood is pass through a tube which thin enough that can pass cells by one at a time, Characteristic about the cell are measured using lasers or electrical impedance.

2.9.2 Urea ,creatinine, and calcium:-

A15:-

Random access analyzer ,clinical dedicated ,through put of 150 test in hour, 4independent position for samples and reagent racks,24 samples for rack, 10 reagent for rack, 20 and 50 reagent ml reagent bottles,primary tubes or paediateric cups as sample containers, unlimited STAT capabilities.run at any time, programming of 5 kinds of samples (serum , plasma , urine ,CSF,and whole blood), up to 15 minutes reading time, reusable methacrylate rotor , minimum reading volume of 200 uL, measurement

range from -0.05A to 2.5A, spectral range 340 nm to 900 nm and filter configuration 340,405,505,535,560,600,635,670 nm.

2.10 Statistical analysis:

The data entry and analysis was done on computer package SPSS (Statistical Packages of Social Sciences) version 15.0 use independent sample T test. The results were given in the text as mean and 95% confidence intervals of hematological and clinical values.

3.Results

3.1Demographic data:

The age percentage in CRF show highest percentage (60%) was recorded by the age group 41-60 years old then (26%) Then (14%) was recorded by 21-40 and more than60 years old .

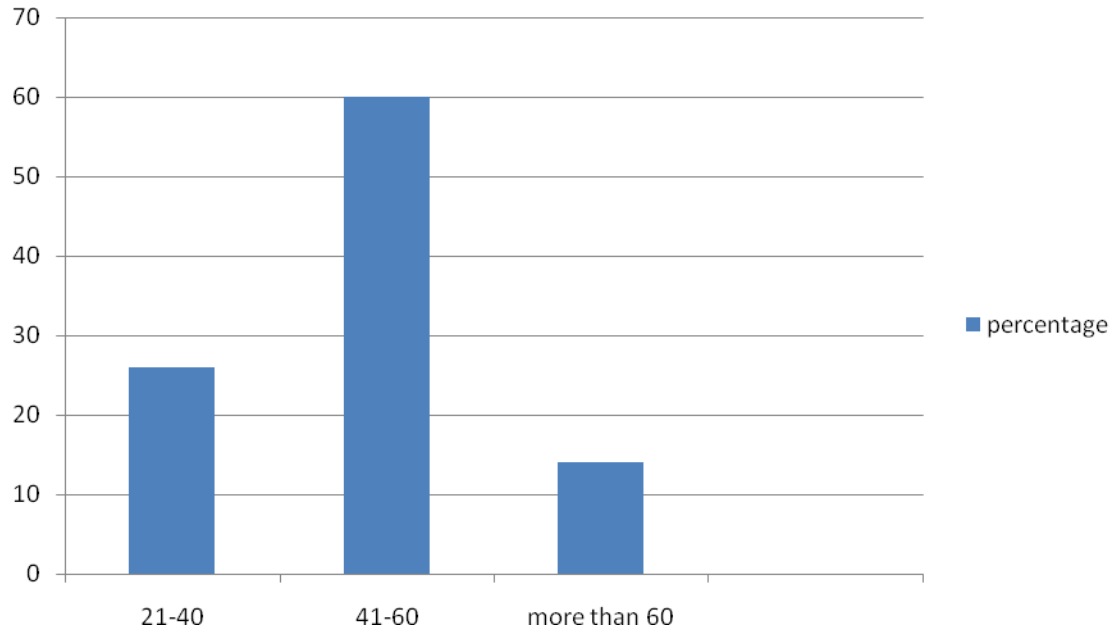


Figure (3-1) the distribution ofchronic renal faliure patients according to their age.

The gender percentage in CRF Among the study population 32(64%) patients were male and 18 (36%) were female .

The result conclude that hypertension is the primary causes of CRF(64%) then Diabetes (20%) and other cause (16%)

3.2Haematological Data:

The mean of Hb in renal failure patients were (8.3 g/dl) compared to (12.2 g/dl) in control group .Hb concentration decreased significantly (p.value 0.000).

The mean values of RBCsin renal failure patients were (3.4 m/mm3) compared to (4.7m/mm3) in control group .RBCs decreased significantly (p.valu ≤0.000) inCRF.

The mean values of Hct in renal failure patients were (28.1) compared to (41) in control group .Hct decreased significantly (p.value≤ 0.000) in patient with CRF.

The mean values of plt in renal failure patients were (211C/mm3) compared to (263C/mm3) in control group .plt decreased significantly (p.value ≤0.03).

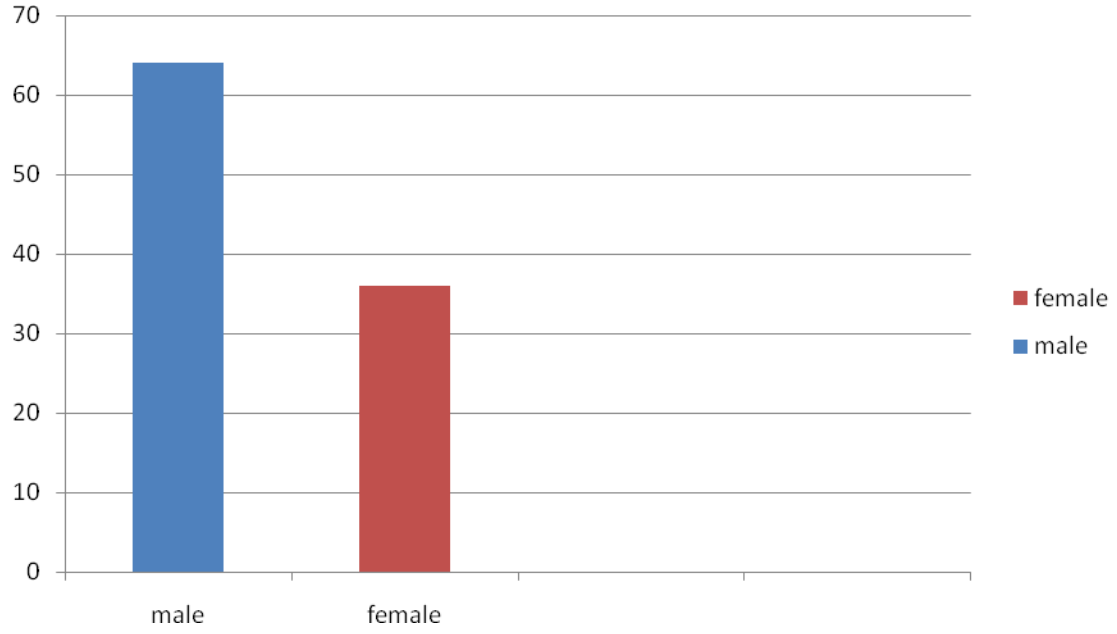


Figure (3-2) distribution of chronic renal faliure patients according to their gender.

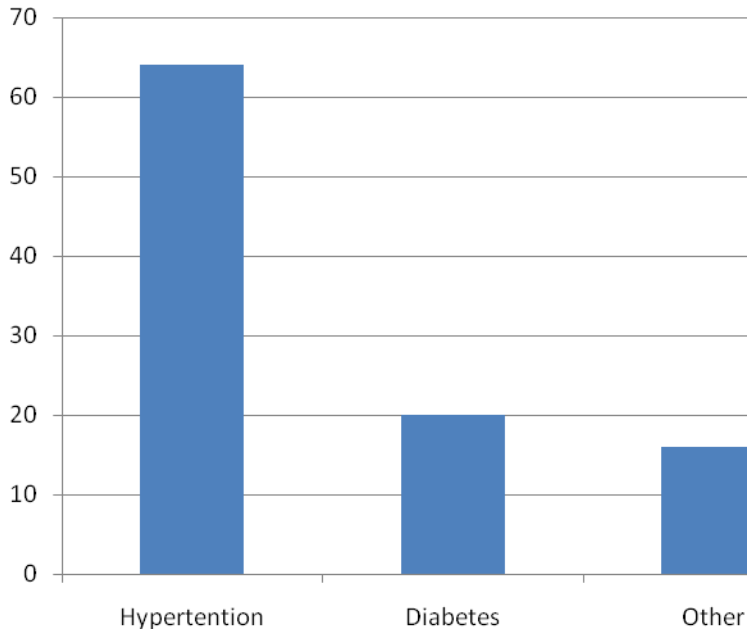
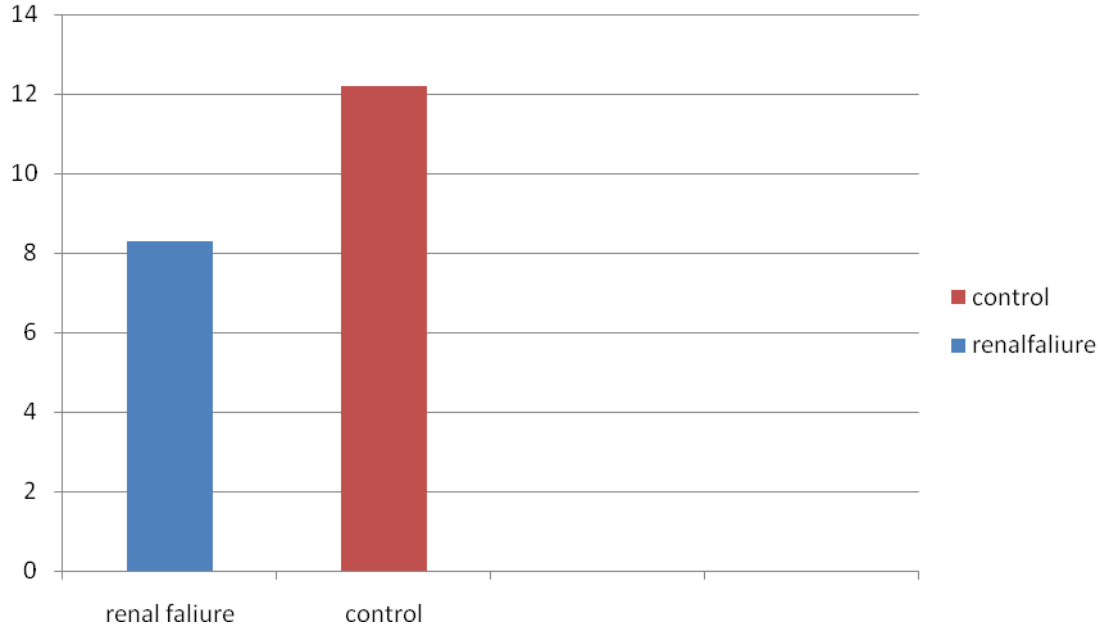
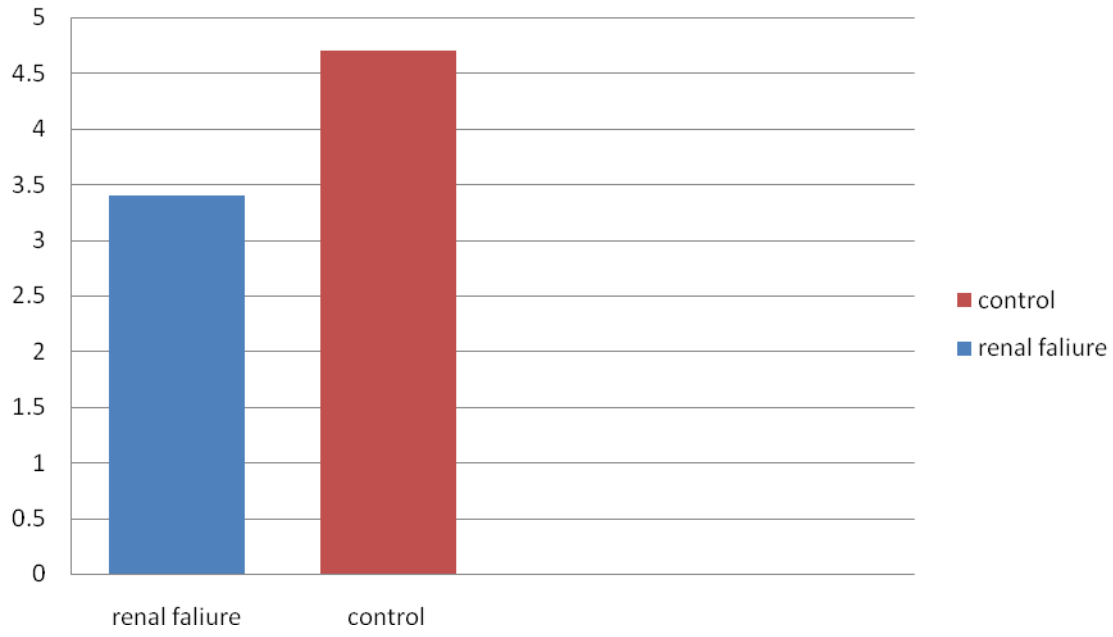


Figure (3-3): The distribuation of CRF patients according to causes.



Figure(3-4): Show the mean of Hb concentration in renal failure and control:



Figure(3-5): Show mean of RBCs count in renal failure and control:

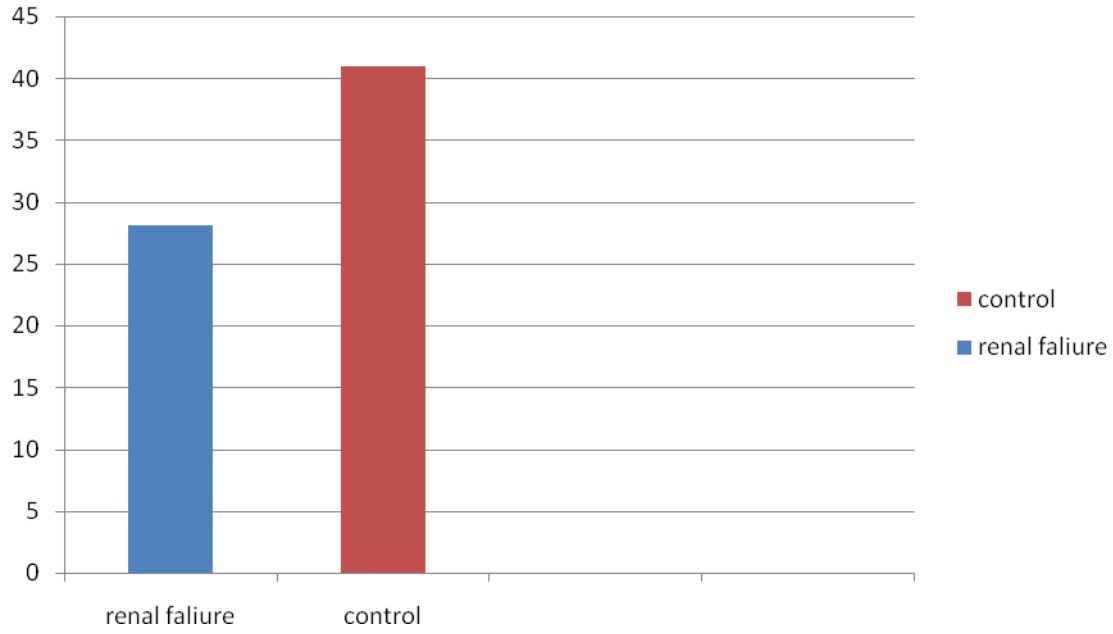
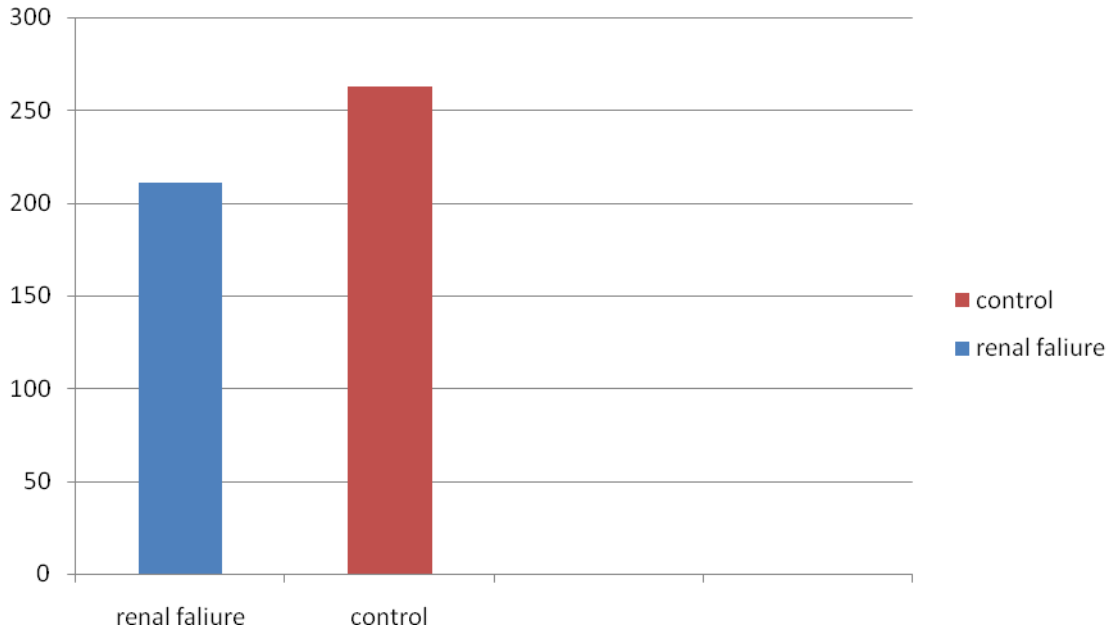


Figure (3-6) Show the mean of Hct in renal Faliure patient and control:

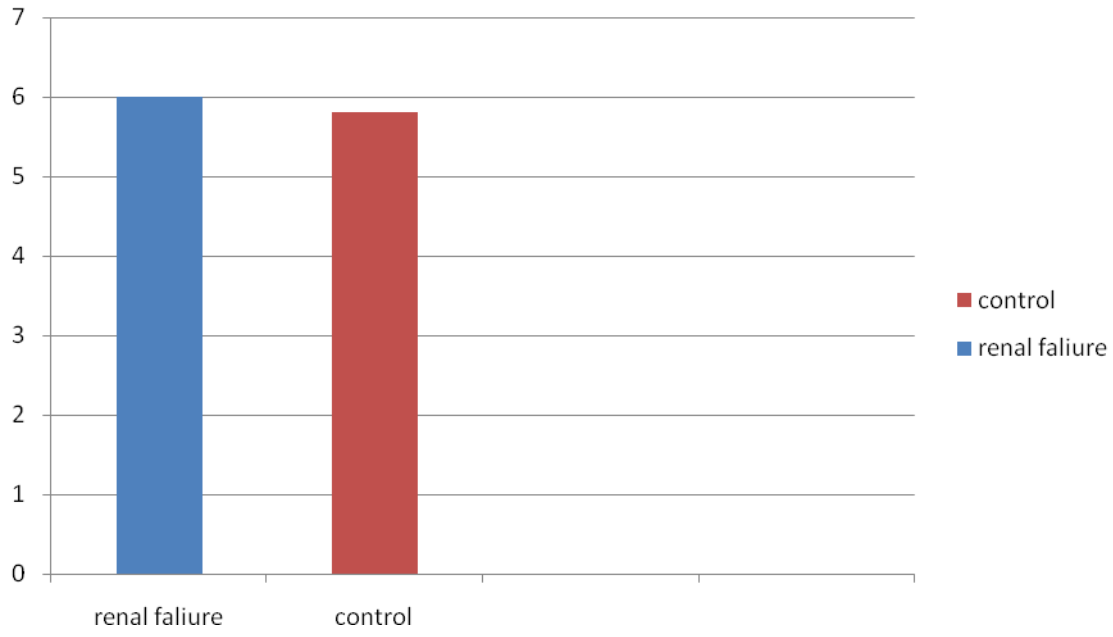


Figure(3-7) Show the mean of platelet count in renal failure patient and control

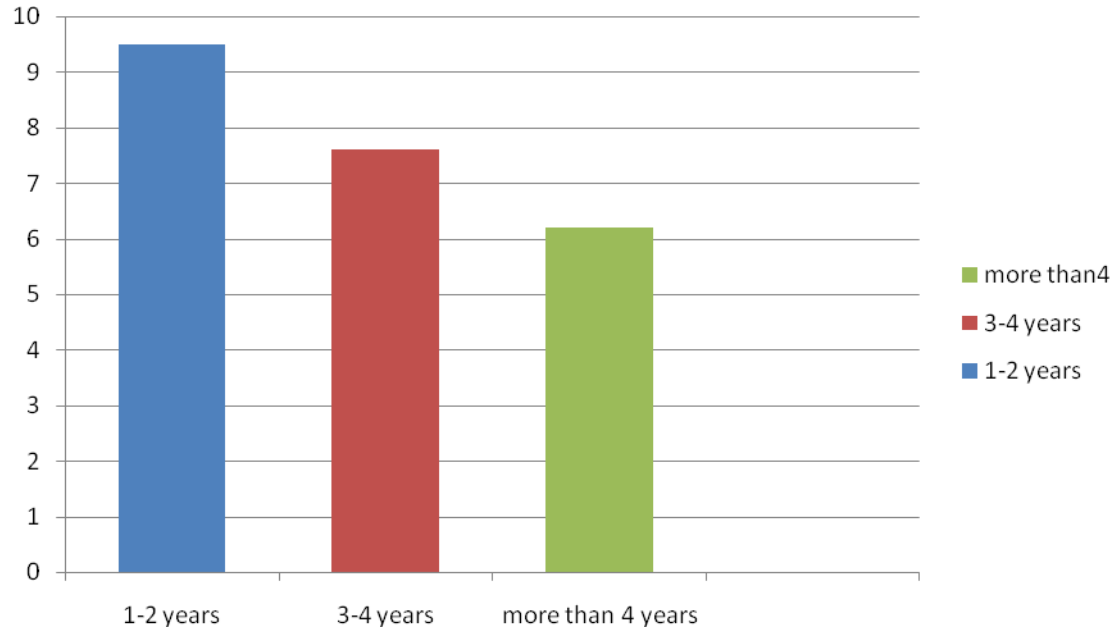
The mean values of TWBCs (figure 3-8) , show the mean of TWBCs in renal failure patients were (6m/mm3) compared to (5.8m/mm3) in control group .TWBCS is insignificantly (p.value 0.6).

The effects of duration of CRF on Hb concentration were show in(figure 3-9) .Patients with 1-2 years duration recorded significantly (p.value 0.000) higher Hb values (9.5g/dl) comparing with 3-4 years (7.6 gldl) and more than 4 years duration (6.2 g/dl).

The effects of duration of CRF on RBCs were show in(figure 4-10) .Patients with 1-2 years duration recorded significantly (p.value 0.000) higher RBcs values (3.9m/mm3) comparing with 3-4 years (2.9m/mm3) and more than 4 years duration (2.6m/mm3).



Figure(3-8) Show the mean value of TWBCs count in renal failure patient and control



Figure(3-9) Show effect of duration of renal failure in mean Hb concentration

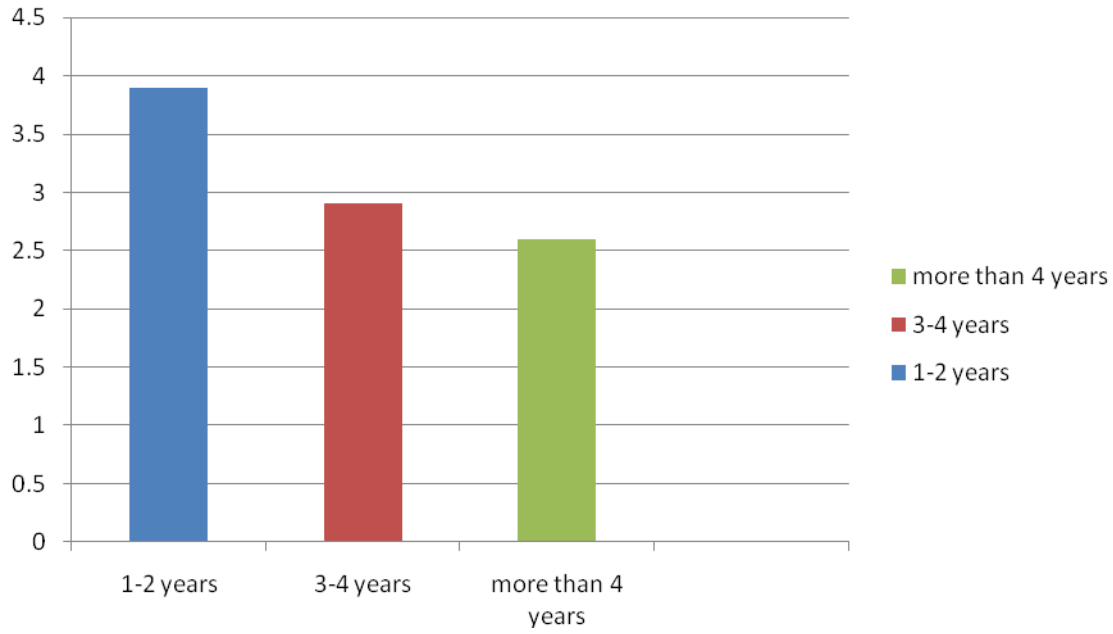


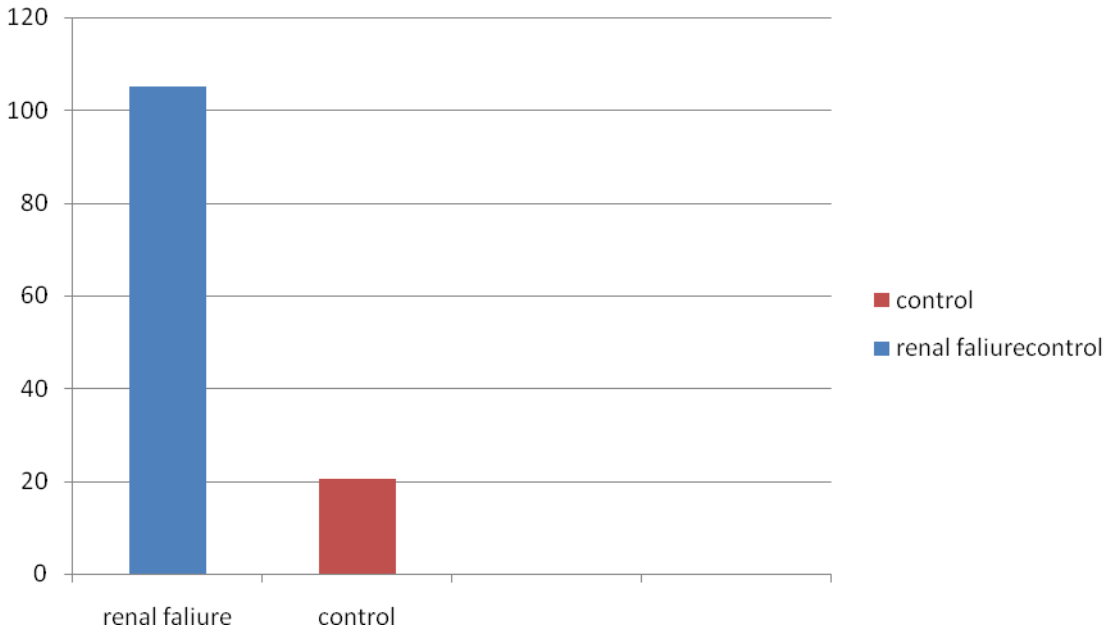
Figure (3-10) Show effect of duration of renal failure in mean RBCs count

3.3 Biochemical parameters:

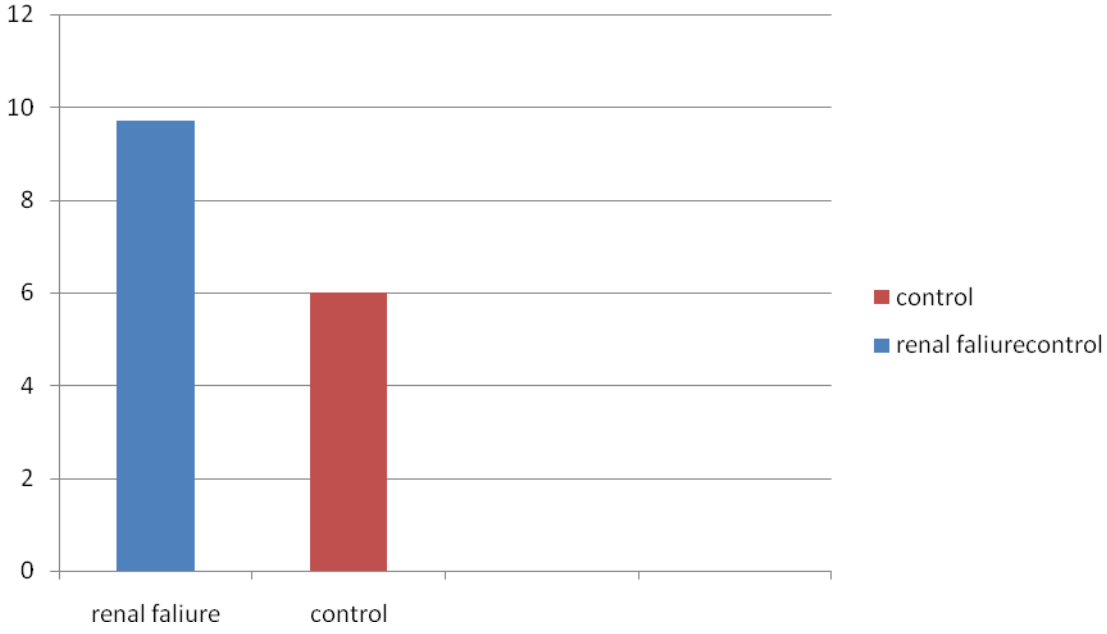
The mean values of Urea (figure 3-11) , show the mean values of Urea in renal failure patients were (105.2 mg /dl) compared to (20.5 mg/dl) in control group .Urea is decreased significantly (p.value≤ 0.000) inCRF.

The mean values ofcreatinine (figure 3-12) , show the mean ofUrea in renal failure patients were (9.7 mg /dl) compared to (0.6mg/dl) in control group .creatinine is decreased significantly (p.value 0.000).

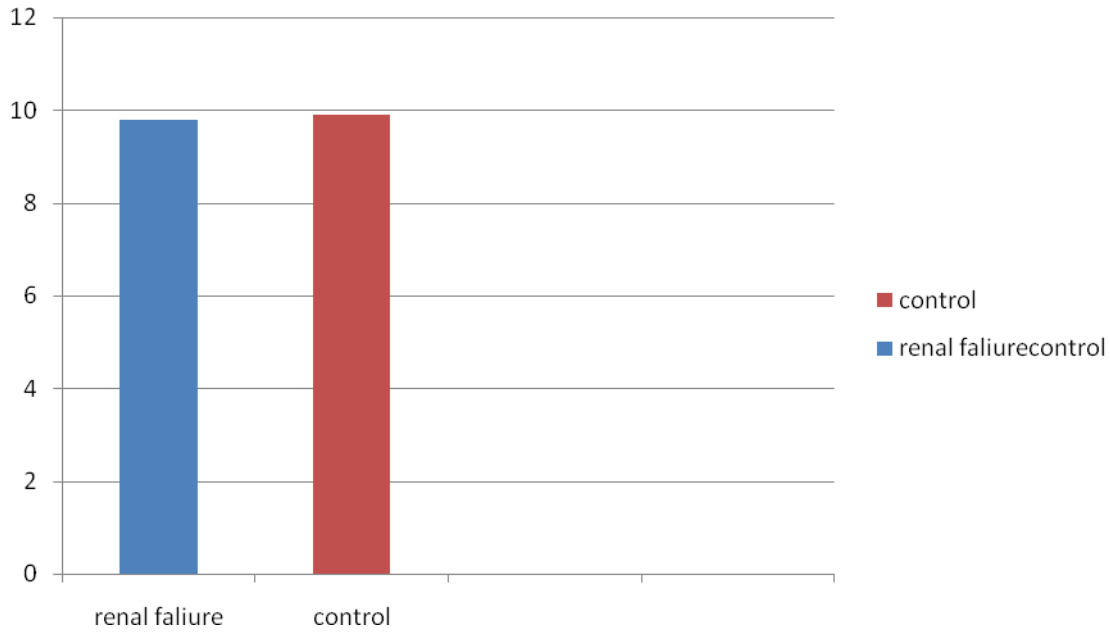
The mean values of calcium (figure 3-13) , show the mean of calcium in renal failure patients were (9.8) compared to (9.9) in control group .calcium is insignificantly (p.value 0.173).



Figure(3-11) Show the mean of urea renal failure patient and control :



Figure(3-12) Show the mean of creatinine renal failure patient and control :



Figure(3-13) Show the mean of calcium renal failure patient and control :

4.1Discussion

Kidney diseases are attributed to alterations in haematological parameters leads to anaemia.Faliure in erythropiotein secretion is the main cause of anaemia in CRF patient in addition to chronic blood loss,hemolysis and bone marrow suppression, in this study, the effects of CRF on physiological parameters have been assessed.The results indicate that Hb concentration ,Hct and RBcs were decreased significantly in patient with CRF (Levin *etal*,2007;Islam *etal*;2015).The result full agreement with (Ferdinand *etal*;2018;Hahib *etal* ;2017 and Suresh *etal*: 2012)

In the present study , total leukocyte count were within normal ranges in CRF patients . WBCs is slightly raised in CRF patients and also process of dialysis further increases WBC count this may be due to up regulation and present of cytokines such as Alpha tumor necrosis factor and interleukin-6(Tbahriti *etal*;2013)This result is agree with(Ferdinand *etal*:2018)

In this study the plt decreased compare to control .Erythropoietin potentiates effect of megakaryocyte colony stimulating factors.In CRF ,impaired erythropiotein secretion lead to decrese in platelet count .The result full agreement with(Suresh *etal;*2012 and Hahib *etal* ;2017).

The proplem in renal failure effect in erythropiotein secretion lead to decreased Hb and RBcs count with increased duration of disease .The effect of duration of renal faliure in mean of Hb concentration ,in duration1-2 years the Hb was (9.5g/dl), in duration 3-4 years the Hb concentration was(7.6g/dl) and in duration more than 4 years the Hb concentration was(6.2g/dl),the effect of duration of renal faliure in mean of RBCs count ,in duration1-2 years the RBCs count was (3.9m/mm3), in duration 3-4 years the RBCs count was(2.9m/mm3) and in duration more than 4 years the RBCs count was(2.6m/mm3),when compared between these results by SPSS the P value is(0.000),which is less than 0.05 that is mean there are significant different in RBCs count.(Bersab and Levin;2000).

The mean of urea (cases) which was (105.2mg/dl) and the mean of control which was(20.5mg/dl), when compared between these results by SPSS the P value is(0.000),which is less than 0.05 that is mean there are significant different in urea.CRF associated with decrease in renal clearance and GFR which lead to accumulation of urea.This result is agree with(NourAlamin.M etal2014) which include A. Effect of Dialysis on Serum Urea LevelIn CRF patients, pre-dialysis serum urea level was significantly higher than normal range (20-40 mg/dl). Most the patients (53 %) had serum urea level between 200-300 mg/dl .

The mean of creatinine (cases) which was (9.7) and the mean of control which was(0.6), when compared between these results by SPSS the P value is(0.000),which is less than 0.05 that is mean there are significant different in creatinine.this result is agree with(NourAlamin.M *etal*2014) which includeEffect of Dialysis on plasma Creatinine Level

Serum creatinine level was higher than normal range (up to 1.4 mg/dl) in CKD patients . Factors like age, sex and physical status of person also effect serum creatinine level, CRf associated with decrease in renal clearance and GFR which leads to accumulation of creatinine (NourAlamin *etal*2014).

The mean of plasma calcium (cases) which was (9.8) and the mean of control which was(9.9), when compared between these results by SPSS the P value is(0.173),which is more than 0.05 that is mean there are insignificant different in calcium .this result is agree with(Nahid and AbdElkarim 2013) which includemean of calcium (cases) which was (9.5) and the mean of control which was(9.7), when compared between these results by SPSS the P value is(0.07),which is more than 0.05 that is mean there are insignificant, The CRF made Ca low but in this study the patients take calcium tablet.

The distribution of Age in CRF inage 41-60 after that 21-40 lastly more than 60,.Among the study 32 (64%)patients were male and 18 (36%)were female

In this study the common cause is Hypertention after that Diabetes lastly other cause this agreement with(Hahib *etal* ;2017)

4.2 Conclusions

From the present study it can be concluded that Patients with renal failure show abnormal haematological and biochemical parameters.

The mean of hemoglobin is 8.3 g/dl, RBCs is $3.4x10^{12}$/L, PCV is 28.1, TWBCs is $6x10^{9}$/L and platelet is $211x10^{9}$/L.

- some mean of these haematological parameter are higher than the mean of control(TWBCs)and some are lower than mean of control in(Hb,RBCscount,PCV, platelet count).
- The increase duration of disease effect in Hb concentration and RBCs count by decrease.
- CRF patient higher serum urea and creatinine and normal calcium level leading to various other dangerous diseases.
- In this study most of CRF were male(64%).
- In this study age(41-60) years old is the most susceptible to disease(60%).
- In this study Hypertention is the common cause of CRF(64%).

4.3Recommendation

Renal failure is one of the health problem through out the world can cause anaemia, and renal function abnormality according to results in my study .Irecommended by integrate the official and social efforts in promote the attention about danger of the disease ,also efforts should be geared toward early identification of anaemia in patients with renal failure,evaluating and correcting persistent failure to reach or maintain intended hemoglobin concentration and red cell transfusion if need.In addition, there is need for further studies to establish the benefits or otherwise, of iron supplementation and erythropoietin therapy .Prevention of the infectious diseases such as malaria ,worm infestations andtyphoid fever that may deplete red cells and hemoglobin should be a public health priority.

Also renal function tests should be done monthly.

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Appendix (A1)

Table(3-1): show Haematological parmeters in renal failure patients and control

Parameters	Mean 0f renal Faliure	Mean of control	P.value
RBcs(million/mm3)	3.4	4.7	0.000
Hb(g/dl)	8.3	12.2	0.000
Hct	28.1	41	0.000
WBCs(million/mm3)	6	5.8	0.000
Plt(million/mm3)	211	263	0.03
Urea(mg/dl)	105.2	20.5	0.000
Creatinine(mg/dl)	9.7	0.6	0.000
Calcium(mg/dl)	9.8	9.9	0.173
Total of patient	50	50	

Table (3-2) Show effect of duration of renal failure in mean RBCs(million/mm3) count:

Duration	Frequency	Percentage	Mean	P.value
1-2 years	31	52.5	3.9	0.000
3-4 years	10	25	2.9	
More than 4 years	9	22.5	2.6	
Total	40	100%		

P.value is 0.000 that mean the result is significant.

Table (3-3) Show effect of duration of renal failure in mean Hb(g/dl)concentration:

Duration	Frequency	Percentage	Mean	P.value
1-2 years	31	52.5	9.5	0.000
3-4 years	10	25	7.6	
More than 4 years	9	22.5	6.2	
Total	50	100%		

P.value is 0.000 that mean the result is significant.

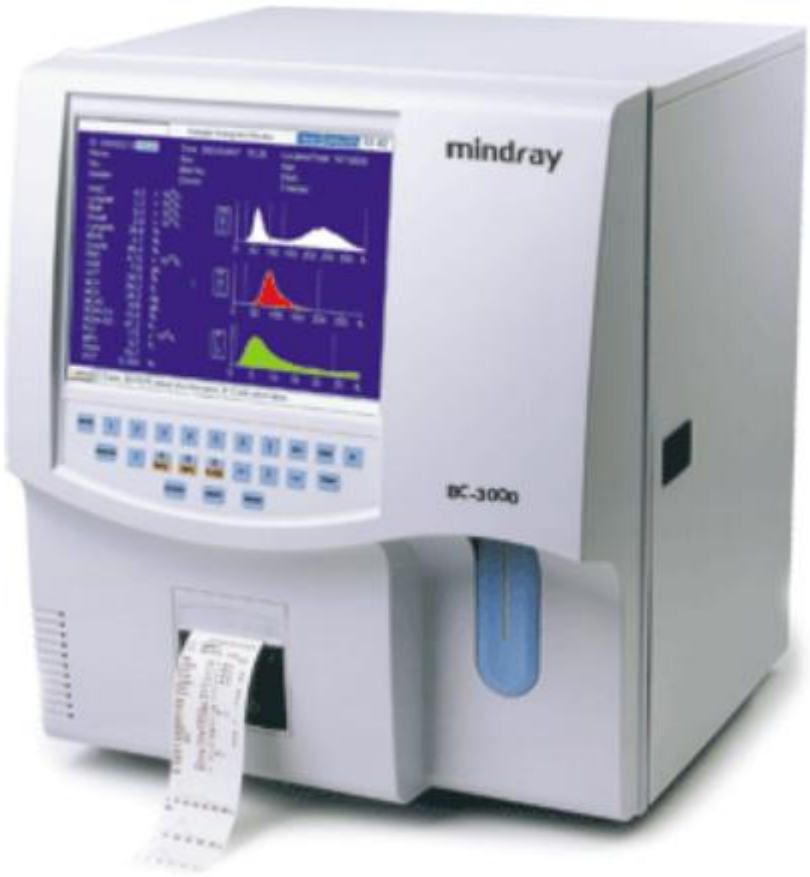
Table (3-4) show Age and gender distribution of renal faliure patients.

Age	Male	Female	Total	percentage
21-40	8	5	13	26
41-60	20	10	30	60
More than 60	4	3	7	14

Table (3-5): Show primary cause of renal failure :

Primary cause	Number of patients	percentage
Hypertention	32	64
Diabetes	10	20
other	8	16

Appendix (A2) Mindary BC 3000



Appendix (A3) : A15

Appendix (A4)

Questionnaire

جامعة السودان للعلوم والتكنولوجيا

إستبيان لمرضى الفشل الكلوي

1-العمر ؟

أ- 20-40 ب-41-60 ج-أكثر من 60

2-الجنس؟

أ-ذكر ب- أنثى

3- مكان السكن؟

4-منذ متى وأنت تعاني من الفشل الكلوي؟

أ-1 -2 سنة ب- 3-4 سنة ج- أكثر من4 سنة

5- كم عدد الغسلات في الأسبوع؟

أ- غسلة واحدة ب- غسلتين

6-هل عانيت من جلطة؟

أ- نعم ب- لا

7- هل تعاني من أمراض أخرى؟

أ- سكري ب- ضغط ج-أخرى

8- هل تعاني من فقدان الدم بعد عملية الغسيل؟

أ- نعم ب- لا

9-هل تدخن؟

أ- نعم ب- لا

خاص بالباحث:-

10- مستوى؟

Urea …….. creatinine……..ca…….

11-CBC?

TWBCs…….….

RBCS…….. Hb…….. Hct……

Platelet……..…….