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THE PREVALENCE OF DYSNATREMIA IN HEMODIALYSIS PATIENTS

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ABSTRACT

THE PREVALENCE OF DYSNATREMIA IN HEMODIALYSIS PATIENTS

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The objective of this study was to assess the sodium and potassium levels in patients at different stage of chronic kidney disease, and determine the correlation between sodium and potassium with GFR. The current study is a case (patient with chronic renal failure) and control (healthy persons) study Chronic renal disease patients of both genders (65 males and 44 females) aged 20 to 71 years old who visit Al-jumhuri Hospital in Kirkuk, Iraq (from Novembe 2020 to February 2021), are the target population. Each patient with chronic kidney failure underwent four hours of hemodialysis twice or three times a week. The results of the current study demonstrated that the highest age group with renal failure was 51-60 years, where it was 26.2%. Then, the age group was 61-70, as it was 22.1%, while the lowest age group of patients of renal failure was 31-40, where it was 5.4%. The results of the current study showed that the percentage of healthy people in the current study was 40 (26.8%), while the number of patients with kidney failure was 83 (55.7%), while other stages of kidney disease were reported, the third stage included 9 (6%) patients while. The fourth stage included 17 (11.4%) patients. The findings of the current study showed that patients had higher levels of urea, creatinine, and potassium than the control group ($P<0.05$). In contrast, the results of the study demonstrated a significant ($P\leq0.05$) reduction in patient sodium levels. Otherwise, the results of the current study demonstrated a positive relationship between CKD and sodium and inverse relationship with potassium.

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Keywords: Hemodialysis, Dysnatremia

ÖZET

HEMODİYALİZ HASTALARINDA DİSNATREMİ PREVALANSI

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Bu çalışmanın amacı, kronik böbrek hastalığının farklı evrelerindeki hastalarda sodyum ve potasyum düzeylerini belirlemek ve sodyum ve potasyum ile GFR arasındaki ilişkiyi belirlemektir. Bu çalışma, bir vaka (kronik böbrek yetmezliği olan hasta) ve kontrol (sağlıklı kişiler) çalışmasıdır. Al-jumhuri Hastanesini ziyaret eden 20 ila 71 yaşları arasındaki her iki cinsiyetten (65 erkek ve 44 kadın) kronik böbrek hastalığı hastaları Irak, Kerkük'te (Kasım 2020-Şubat 2021) hedef nüfus. Son dönem böbrek hastalığı olan her hasta, toplam dört saat boyunca haftada iki veya üç kez hemodiyaliz aldı. Bu çalışmanın sonuçları, böbrek yetmezliği olan en yüksek yaş grubunun %26,2 ile 51-60 yaş olduğunu göstermiştir. Daha sonra yaş grubu %22,1 ile 61-70 olurken, böbrek yetmezliği hastalarının en düşük yaş grubu %5,4 ile 31-40 oldu. Bu çalışmanın sonuçları, mevcut çalışmada sağlıklı kişilerin yüzdesinin 40 (%26,8) olduğunu, böbrek yetmezliği olan hasta sayısının 83 (%55,7) olduğunu, böbrek hastalığının diğer evrelerinin bildirildiğini gösterdi. Evre ise 9 (%6) hastayı içeriyordu. Dördüncü aşamada 17 (%11,4) hasta vardı. Bu çalışmanın sonuçları, kontrol grubuna kıyasla hastalarda üre, kreatinin ve potasyum düzeylerinde anlamlı ($P \leq 0,05$) artış olduğunu göstermiştir. Sonuçlar kontrol grubuna göre hastalarda sodyum düzeylerinde anlamlı ($P \leq 0,05$) düşüş olduğunu göstermektedir. Aksi takdirde, mevcut çalışmanın sonuçları, KBH ile sodyum arasında pozitif bir ilişki ve potasyum ile ters bir ilişki olduğunu göstermiştir.

2021, 55 sayfa

Anahtar Kelimeler: Hemodiyaliz, Dysnatremi

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CONTENTS

ABSTRACT i
ÖZET ii
PREFACE AND ACKNOWLEDGEMENTS iii
CONTENTS iv
LIST OF SYMBOLS vi
LIST OF ABBREVIATIONS vii
LIST OF FIGURES viii
LIST OF TABLES ix
1. INTRODUCTION 1
1.1 The Aim of Study 2
2. LITERATURE REVIEW 3
2.1 Chronic Kidney Disease (CKD) 4
2.2.1 Glomerular filtration rate (GFR) 5
2.2.2 Clearance 6
2.3 Haemodialysis (HD) 7
2.3.1 History 8
2.3.2 The mechanism of hemodialysis 9
2.3.3 Types of dialysis 10
2.3.4 Patient safety and technical advances 12
2.3.5 Complications of hemodialysis 13
2.4 Health Problems With Water and Sodium Regulation 13
2.4.1 Physiology 14
2.4.2 Hyponatremia 15
2.4.3 Hypernatremia 17
3. MATERIALS AND METHODS 19
3.1 Materials 19
3.2 Study Design 19
3.3 Setting of The Study 19
3.4 Target Population 20
3.5 Collection of Blood Samples and Processing 20

3.6 Estimated Glomerular Filtration Rate (eGFR) 22
3.7 Biochemical Tests 22
3.7.1 Kidney funtion parameters 22
3.7.2 Electrolytes 25
3.8 Statistical Analysis 27
4. RESULTS 28
4.1 Ages of Patients 28
4.2 Estimated Glomerular Filtration Rate (eGFR) 28
4.3 Kidney Functions 29
4.3.1 Creatinine levels 29
4.3.2 Urea levels 30
4.4 Electrolytes 31
4.4.1 Sodium level 31
4.4.2 Potassium level 32
4.5 Correlations 33
4.5.1 GFR and sodium 33
4.5.2 GFR and potassium 34
4.6 Complete Blood Counts (CBC) 35
4.6.1 Red blood cells (RBC) count 35
4.6.2 Hemoglobin (Hb) Level 36
4.6.3 Packed cell volume (PCV) 37
4.6.4 Mean corpuscular volume (MCV) 38
5. DISCUSSION 40
5.1 Kidney Function (Urea & Creatinine) 40
5.2 Sodium Concentration 41
5.3 Potassium Concentration 43
5.4 Complete Blood Count (CBC) 44
6. CONCLUSIONS AND RECOMMENDATION 46
6.1 Conclusions 46
6.2 Recommendation 46
REFERENCES 47
CURRICULUM VITAE 55

LIST OF SYMBOLS

μL	Micro-litter
ADH	Vasopressin (Antidiuretic Hormone)
Gm	Gram
L	Litter
Mg	Mille-gram
ML	Mille-litter
Mmol	Mille-mole
NaCl	Sodium chloride
$NaHCO_3$	Sodium bicarbonate

LIST OF ABBREVIATIONS

ANOVA	Analysis of Variance
CDC	Central dialysis catheter
CHF	Congestive heart failure
CKD	Chronic kidney disease
CRF	Chronic renal failure
ECF	Extracellular fluid
eGFR	Estimated glomerular filtration rate
ESRD	End stage renal disease
GFR	Glomerular filtration rate
GlDH	Glutamate dehydrogenase
HD	Haemodialysis
ICF	Intracellular fluid
SD	Standard deviation
SQ	Subcutaneously
TBW	Total body water

LIST OF FIGURES

Figure 2.1 Kidney anatomy & nephron filtration diagram 4
Figure 2.2 Diagrammatic representation of a dialyzer 10
Figure 2.3 Hemodialysis system 12
Figure 2.4 Relationship of fluid compartments to total body weight 15
Figure 3.1 EDTA (hematology tests) (CBC) 20
Figure 3.2 Gel vacutainer tube for chemistry tests 21
Figure 3.3 Centrifuge machine 800-8 21
Figure 3.4 Spectrophotometer 23
Figure 4.1 Creatinine level in studied groups 30
Figure 4.2 Urea level in studied groups 31
Figure 4.3 Sodium level in studied groups 32
Figure 4.4 Potassium level in studied groups 33
Figure 4.5 Correlation between GFR and sodium 34
Figure 4.6 Correlation between GFR and potassium 35
Figure 4.7 RBC count in studied groups 36
Figure 4.8 Hb level in studied groups 37
Figure 4.9 PCV level in studied groups 38
Figure 4.10 MCV level in studied groups 39

LIST OF TABLES

Table 3.1 A list devices and equıpments used in the present study 19
Table 3.2 Urea procedure steps 23
Table 3.3 Creatinine procedure steps 24
Table 3.4 Sodium procedure steps 25
Table 3.5 Potassium procedure steps 26
Table 4.1 Age groups of in present study 28
Table 4.2 The percentage of CKD stages in current study 29
Table 4.3 Creatinine level in studied groups 29
Table 4.4 Urea level in studied groups 30
Table 4.5 Sodium level in studied groups 31
Table 4.6 Potassium level in studied groups 32
Table 4.7 RBC count in studied groups 35
Table 4.8 Hb level in studied groups 36
Table 4.9 PCV level in studied groups 37
Table 4.10 MCV level in studied groups 38

1. INTRODUCTION

The process of eliminating wastes and extra water from the blood is known as dialysis (Hakim and Lazarus 1995). This procedure is employed in kidney failure instances to artificially replace normal kidney function. Dialysis cannot replace impaired kidney function entirely, but it does help control it to some degree by diffusion and ultrafiltration (Lee 2017). When the glomerular filtration rate slips below 15 ml/min/1.73m^2 in chronic renal failure (CRF) (Tattersall *et al.* 2011). CRF is a disorder in which kidney function is gradually lost over months or years. The serum creatinine level, a breakdown product of muscle protein, may be used to diagnose CRF. The glomerular filtration rate (GFR) is determined by creatinine levels, and its activities are increased in CRF, suggesting a lower creatinine (Patel *et al.* 2002). CRF includes five stages that depending on GFR, during the stage of 5 (GFR 15 ml/min/1.73m^2) requiring dialysis; this last stage is called end stage renal failure (ESRD) (Inker *et al.* 2014).

Dissemination over a semi-permeable membrane of molecules in solution over an electrochemical gradient is called dialysis (Depner 1991). One of the primary functions of hemodialysis is to replicate the normal water and electrolyte balance of the body, which is what the kidneys do naturally. This is performed by transferring solutes and blood urea into the dialysate, solutes and bicarbonate back into the blood from the dialysis. The solute concentration and molecular weight of the solution are the two major determinants of diffusion concentrations. However, whereas small molecules like urea are able to diffuse fast, larger molecules like phosphate, 2-microglobulin, and albumin, as well as protein-bound solutes like p-cresol, are able to move more slowly. Additionally, solutes can move through holes in the membrane by convective processes which are driven by hydrostatic or osmotic pressure gradients. This process is known as ultrafiltration, and it can be either diffusion- or convection-based (Patel *et al.* 2002, Locatelli *et al.* 2002).

Chronic kidney disease (CKD) is a term used to describe any kidney disorder that lasts more than three months and involves signs of kidney failure and/or decreased kidney function. Imaging scans may diagnose proteinuria or albuminuria, hematuria, or scarring

as signs of kidney injury (Coresh *et al.* 2003). Sodium and potassium are critical for the maintenance of cellular homeostasis and regulation of osmotic pressure. The extracellular fluid has a high concentration of sodium cations, while the intercellular fluid has a high concentration of potassium ions. Potassium and sodium ions are heavily engaged in the acid-base regulation of the kidneys (Pohl *et al.* 2013). Chronic renal illness is known for its prevalent side effect of metabolic acidosis, as shown by a low serum bicarbonate (Basile *et al.* 2016).

1.1 The Aim of Study

- The prevalence of dysnatremia in hemodialysis patient.
- The prevalence of dysnatremia in CKD non dialysis patient.
- Find a correlation of sodium and potassium with GFR.

2. LITERATURE REVIEW

Human kidneys are large, bean-shaped, crimson organs placed in the rear of the belly, just below the bladder. Because to the liver's placement, the right kidney is around 1 to 2 cm lower than the left kidney. Each kidney measures approximately 11 centimeters long, 4 to 5 centimeters high, and 2 to 3 centimeters thick (Gartner and Hiatt 2001).

A concave medial boundary, the hilum, where nerves, blood and lymph vessels join and leave, and the ureter exits, and a convex lateral surface define each kidney. The extended upper end of the ureter, known as the renal pelvis, is divided into two or three main calyces. Each main calyx has many small branches called minor calyces (Junqueira and Carneiro 2005).

The kidney is split into two parts: outer and inner. The renal cortex is the outermost part of the kidney. It has a rugged, granular appearance and is reddish-brown in colour. The renal medulla is the inner part of the renal pelvis. It has a glossy exterior with a dark purple outer region that emits rays into the cortex and a pale gray-red inner area that reaches down to the renal pelvis (Colville and Bassert 2002).

The medullary pyramids are 10 to 18 conical or pyramidal structures that make up the renal medulla in humans. Parallel clusters of tubules, known as medullary rays, enter the cortex from the base of each medullary pyramid; each kidney contains 1-4 million nephrons. The renal corpuscle is a dilated part of each nephron. The various components of the tubules system include the distal convoluted tubule, collecting ducts, the ascending and descending limbs of Henle's loop, and the proximal convoluted tubule. Scholars differ on whether collecting tubules and ducts are a part of the nephron (Junqueira and Carneiro 2005, Young *et al.* 2004). As shown in Figure 2.1.

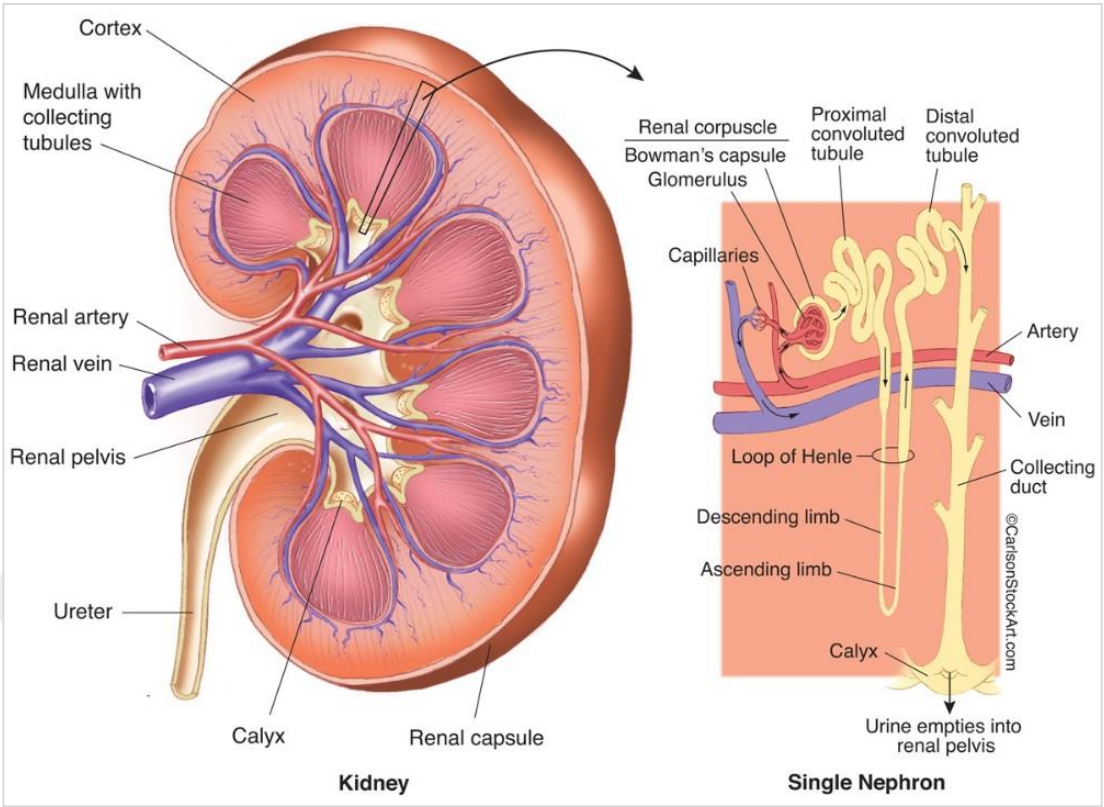

Figure 2.1 Kidney anatomy & nephron filtration diagram

2.1 Chronic Kidney Disease (CKD)

CKD, which has a glomerular filtration rate (GFR) of less than 60 ml/min/1.73 m^2 for three months or more, no matter the etiology, is defined as kidney damage. The kidneys stop working properly over time, and hence require renal replacement therapy (dialysis or transplantation). If imaging studies or a renal biopsy reveal pathologic anomalies, urinary sediment abnormalities, or high urine albumin excretion rates, kidney damage is a real possibility (Webster *et al.* 2017).

The KDIGO CKD 2012 categorization informs the cause of CKD and classifies it into six groups based on the amount of glomerular filtration (G1 to G5 with G3 split into 3a and 3b). Albuminuria staging (A1, A2, and A3) is also included, and it is split into three stages based on the urinary albumin-creatinine ratio (mg/gm or mg/mmol) in a morning "spot" urine sample. The more detailed CKD classification has made it easier to find prognostic signs including decreased kidney function and elevated albuminuria.

Classification systems, however, are accompanied by the danger of CKD overdiagnosis, especially in elderly patients (Inker *et al.* 2014).

2.2.1 Glomerular filtration rate (GFR)

The amount of primary urine purified from plasma through the glomeruli is known as the GFR, which is the most widely used indicator of kidney function (in both kidneys). In a stable person, this equates to about 180 litres per day (Aurell 2004).

Despite this, roughly 99% of the main pee is reabsorbed, so only a little percentage of it is left to be evacuated. The GFR, which is regulated by both the afferent and efferent arterioles, as well as systemic blood pressure, is determined by the net filtration pressure across the glomeruli membrane. The GFR is additionally controlled by the number of glomeruli, which means that the total filterable area is also influenced. The GFR is given in mL/min/1.73m^2 and is generally based on the size of the body (relative GFR). The absolute GFR (the individual's real filtration rate) is often defined in mL/min. The GFR is crucial for a variety of purposes, including predicting kidney activity, monitoring kidney disease development, drug dosing, and general hemodynamics. The GFR, on the other hand, is an estimate of the kidney filtering ability rather than a full assessment of kidney activity (Maisel 2012).

It is believed that “true” GFR, a conceptualized filtration rate of all nephrons in the kidneys, is a hypothesis that is unquantifiable. Because it is challenging to measure all one million nephrons filtering plasma at the same time, as well as because primary pee is subject to varying composition and volume as it progresses from the tubuli to the urinary bladder, this is why urine is usually collected via catheterization. However, if a marker is present in the plasma, its concentration can be determined, and the marker is easily filtered and not reabsorbed by the tubule, then the filtered plasma volume may be calculated. If plasma concentration is established, the volume of marker excreted in urine each unit of time is equal to the amount of plasma that passes through the glomeruli in the same period (Swedish Council on Health Technology Assessment 2013).

The glomerular filtration rate, or GFR, is the amount of plasma removed of a molecule by the nephrons in a unit of time. In young men, normal values range from 130 ml per minute per 1.73 m^2 up to 1.3 L per minute per 1.73 m^2. Young women, on the other hand, have normal values of 120 ml per minute per 1.73 m^2 up to 1.2 L per minute per 1.73 m^2 (Swedish Council on Health Technology Assessment 2013). Numerous factors, including age, ethnicity, body size, hydration, and health conditions, influence the speed of metabolism. On average, GFR decreases with age, and higher filtration levels are seen in young adulthood. This rate of decline is uncertain, however, as it is expected to start at around 10 mL/min per decade in the fourth decade. However, GFR is considered the most accurate overall index of renal activity, notwithstanding its shortcomings (Stevens *et al.* 2006, Levey *et al.* 2011).

2.2.2 Clearance

The cumulative plasma clearance is the amount of plasma that is removed from the substance per unit of time. However, total clearance is the amount of a chemical that is removed overall, not merely by the kidneys. Compared to other removal systems (e.g. liver, bile, respiratory) the amount of plasma which is directly cleaned from the kidneys is the renal plasma clearance (renal clearance). You can only detect the renal plasma clearance by finding a detectable marker/substance in the urine after the kidneys have been removed. A simple way to determine the renal plasma clearance (renal clearance) is to divide the GFR by the amount of time it takes to reach equilibrium, provided the marker is not produced or taken (Swedish Council on Health Technology Assessment 2013). While this is true, it is important to emphasize that clearance will depend on the marker utilized. This is because some markers may not be filtered or indicate tubular secretion or absorption, which will lead to plasma clearance being either overestimated or underestimated. The belief is that creatinine clearance is an overestimation of GFR, as creatinine is partially secreted by tubules (Aurell 2004). In clinical practice, this is discouraged, but it is nevertheless in use because of the mistaken belief that it estimates GFR.

However, the clearance is only valid if all of the following are true:

- The marker must be able to pass through the glomeruli without getting blocked.
- The marker must be present in the blood at a consistent quantity, and it must not be secreted or absorbed.
- GFR must not be impacted by the marker.

In addition to stability, the perfect GFR marker must be non-toxic and accurately detectable in urine and plasma. The protein binding or large electrical imbalance of the candidate marker will impair its filterability. In clinical practice, the overwhelmingly prevalent ways for determining kidney function are by endogenous creatinine or cystatin C estimations (Swedish Council on Health Technology Assessment 2013).

2.3 Haemodialysis (HD)

Haemodialysis, the most widely used dialysis method (about 80% of patients) (HD). Dialysis helps the patient avoid the pain of long needles by providing sufficient blood supply and a blood artery that may be pierced without discomfort (typically three times per week). For this reason, in preparation for dialysis, a surgical procedure is conducted to create an arteriovenous fistula (AV fistula) in the forearm. When the pressure of arterial blood is applied to a vein, it expands, boosting blood flow by up to 1,000-1,500 ml/min. It may be used as a blood access point in order to feed blood to the dialysis machine, and as a point of entry and exit of blood to and from the patient after a month. The patient will receive hemodialysis through a CDC with two ports if there is no fistula (Daugirdas *et al.* 2007).

A dialyzer is a device that has two semipermeable membranes: one for the blood and one for the dialysis liquid. They are set up to enable the fluids to flow in opposing directions and on separate sides of the membranes. Through the dialyzer membrane, dialysis fluid drains out toxic waste products through diffusion. The bicarbonate and electrolytes in the dialysis fluid and blood go through the dialyzer membrane in opposing directions, both using the same mechanism. In addition, the membrane removes any extra fluid that has collected between procedures. Some patients gain 4-5 kg during dialysis sessions as a result of their increased liquid consumption from drinking and feeding, as well as their

inability to achieve enough urine volumes due to impaired renal function. The process of extracting fluid from a dialysis pump is known as ultrafiltration.

The dialysis system can be set to only remove the fluid (ultrafiltration) and not do dialysis at the same time. The dialysis machine and a specially qualified nurse track and operate the whole dialysis operation. Dialysis may be performed at a dialysis centre or at home. Dialysers who dialyze at home are known as home dialysers, and they have learned to dialyze on their own. Self-dialysis patients are those that dialyze in the hospital but administer more or half of the procedure themselves; they only provide the usual protection of being in the hospital and the presence of experienced nurses during dialysis. For more information on this topic, see (Kotanka *et al.* 2007).

2.3.1 History

The first dialysis machine was developed by Leonard Rowntree and John Abel from Johns Hopkins Hospital in 1913, and it was initially tested on animals (Abel *et al.* 1990). Dutch scientist Willem Johan Kolff, during the Nazi occupation of the Netherlands in 1943, created the first working dialyzer. Without a funding, Kolff had to craft the first machine using sausage casings, beer containers, a washing machine, and various other items he could get his hands on. However, the findings for Kolff's machine were not as hoped for: during the following two years, the machine treated 16 patients with acute renal illness, but the results were unsatisfactory. Next, in 1945, a 67-year-old lady in a coma recovered consciousness following hemodialysis treatment, and she lived for another seven years, until she died from a different disease. The first dialysis patient to successfully be treated was she.

In 1945, University of Toronto researcher Gordon Murray devised a dialysis machine without help from any colleagues. Murray's device was more like contemporary machines, using flat plates that were unmovable, as opposed to Kolff's drum, which was rotary (McAlister 2005). Like Kolff, Murray's first triumph was with those who were very ill with renal failure (Murray *et al.* 1947). To improve on a comparable dialysis equipment (the Kolff) that was not as reliable, Nils Alwall of Lund University in Sweden enclosed

it in a stainless steel canister. The first completely feasible hemodialysis system could be created by providing a negative pressure to the outside canister, enabling the removal of fluids. The first patient to get treatment for acute renal illness was treated by Alwall on September 3, 1946 (Kurkus and Ostrowski 2019).

2.3.2 The mechanism of hemodialysis

An external filter, the dialyzer, with a semipermeable membrane is used to sift waste and surplus water during hemodialysis. The blood and dialysis fluid move in opposing directions, resulting in a counter-current flow gradient. A natural semipermeable membrane, the peritoneum, is used in peritoneal dialysis to extract both water and waste from the patient (the material or fluid that passes through the membrane of the dialysis) (Vadakedath and Kandi 2017, Al-Yassin 2018).

The basic idea behind dialysis is the transfer or diffusion of solute particles across a semipermeable membrane (diffusion). Urea and creatinine, in addition to other metabolic waste products, permeate through the dialysate (sodium bicarbonate ($NaHCO_3$), sodium chloride (NaCl), acid concentrate, and deionized water) because of a concentration gradient from the circulation. The rate of diffusion through the membrane depends on the size of the particles as they are diffused into the dialysate. Diffusion through the membrane is slower the greater the solute particle's size. The veins involved in this procedure are linked to the arteries, which transport oxygenated blood from the heart. This arteriovenous shunt makes the veins muscular enough to be pierced many times, while the pressure is also carefully controlled (Vadakedath and Kandi 2017). The diagrammatic representation of a dialyzer as shown in Figure 2.2.

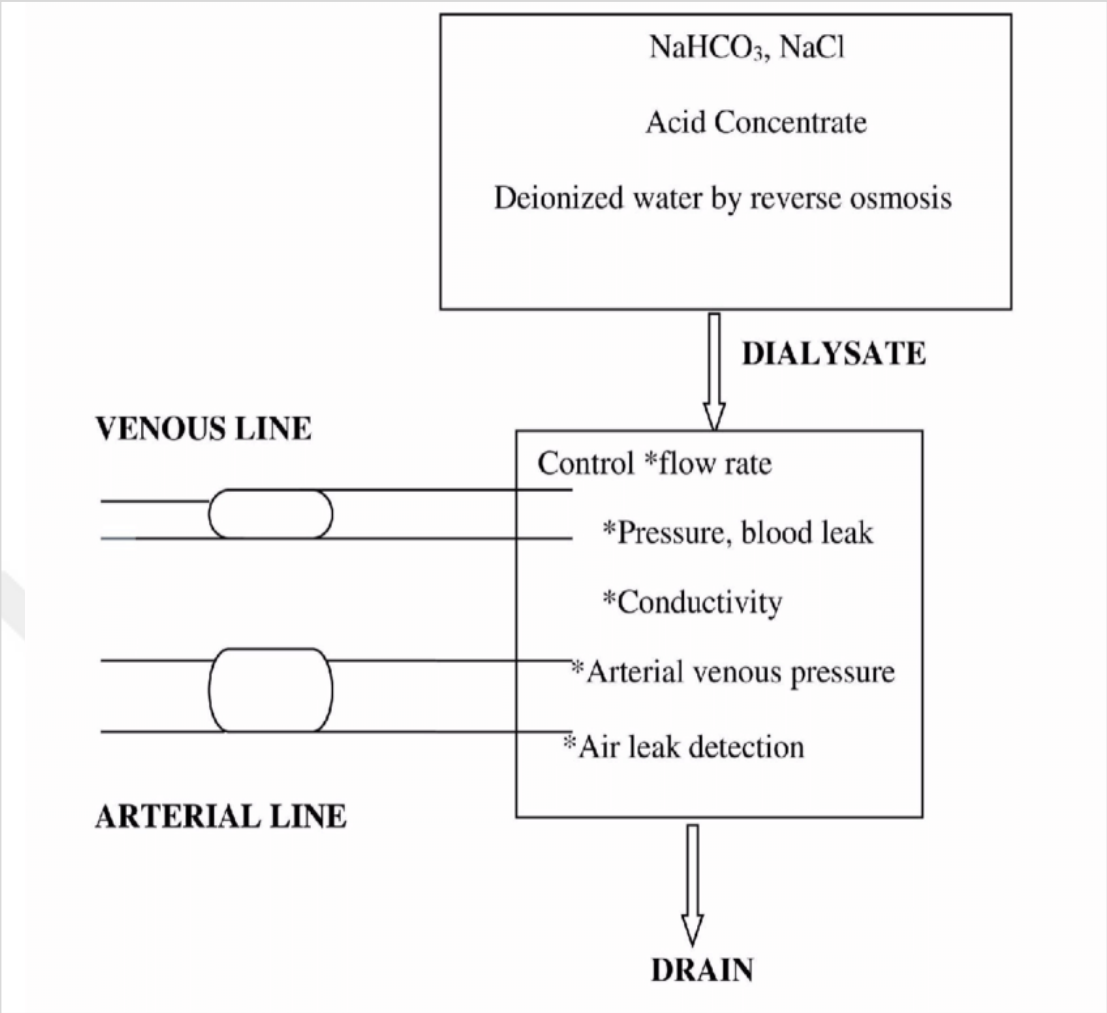

Figure 2.2 Diagrammatic representation of a dialyzer

2.3.3 Types of dialysis

Hemodialysis and peritoneal dialysis are the two major methods of dialysis, and they strip wastes and extra fluids from the blood in various forms (Liao *et al.* 2012). The dialyzer, an external filter with a semipermeable membrane, removes water and waste from the circulation by pumping blood outside the body. The dialysis is in the same direction as the blood. By increasing the concentration gradient of solutes between the blood and dialysate, the counter-current flow of blood and dialysate allows more urea and creatinine to be eliminated from the circulation. The blood solute concentrations (such potassium, phosphate, and urea) are much too high, but the dialysis solution has very little or none of them, and frequent dialysate replacement ensures they're kept to a minimum on this

side of the membrane. The dialysis fluid is not too dissimilar from healthy blood in terms of mineral contents, such as potassium and calcium. Bicarbonate penetration into the blood and neutralization of metabolic acidosis are facilitated by the slightly greater dialysis solution dosage given to patients with an altered pH balance and excess bicarbonate (Al-Yassin 2018).

Nephrologists often tailor dialysis fluid dosages to the requirements of the individual patient. In peritoneal dialysis, wastes and water are removed from the blood using the peritoneum as a semipermeable membrane. The peritoneal membrane and a dialysis-like dialysis solution in the abdominal cavity enable wastes and extra fluid to transfer from the circulation. A dialyzer's blood compartment has a partially permeable membrane. Patient blood is poured into it. A dialyzer consists of millions of microscopic hollow plastic fibers. The fiber wall is the semipermeable membrane (Figure 2.3).

Blood flow and dialysis solution movement both occur inside the fibers, whereas water and waste flow takes place between the dialysis solution and the fibers (Ahmad *et al.* 2008). The blood was then cleansed and recycled. To do ultrafiltration, the dialyzer membrane's hydrostatic pressure is elevated. The dialysate chamber of the dialyzer is usually put under negative pressure to accomplish this. One of the primary benefits of this 4-hour routine is that water and dissolved solutes may be transferred from blood to dialysate. This allows for the removal of several liters of additional fluid. Medicare payment restrictions mean that hemodialysis is only given three days a week in the United States, since in 2005, more than 2,500 individuals in the U.S. dialyzed at home on a regular basis for varying treatment durations. Dialysis five to seven times per week for six to eight hours has shown to be clinically beneficial (Ahmad *et al.* 2008).

Research has shown that this nocturnal daily hemodialysis technique, which is commonly called “NDHD,” results in a better small and large molecular clearance and a lower requirement for phosphate binders. While doing dialysis at home, long-term treatments are common, although at-home dialysis is a flexible modality that may be changed on a daily and weekly basis. Reports also claim that longer and more frequent dosages may be beneficial. One of the most common hospital treatments in the United States in 2011 was

hemodialysis, accounting for over 900,000 stays (a rate of 29 stays per 10,000 populations) (Ahmad *et al.* 2008, Al-Yassin 2018).

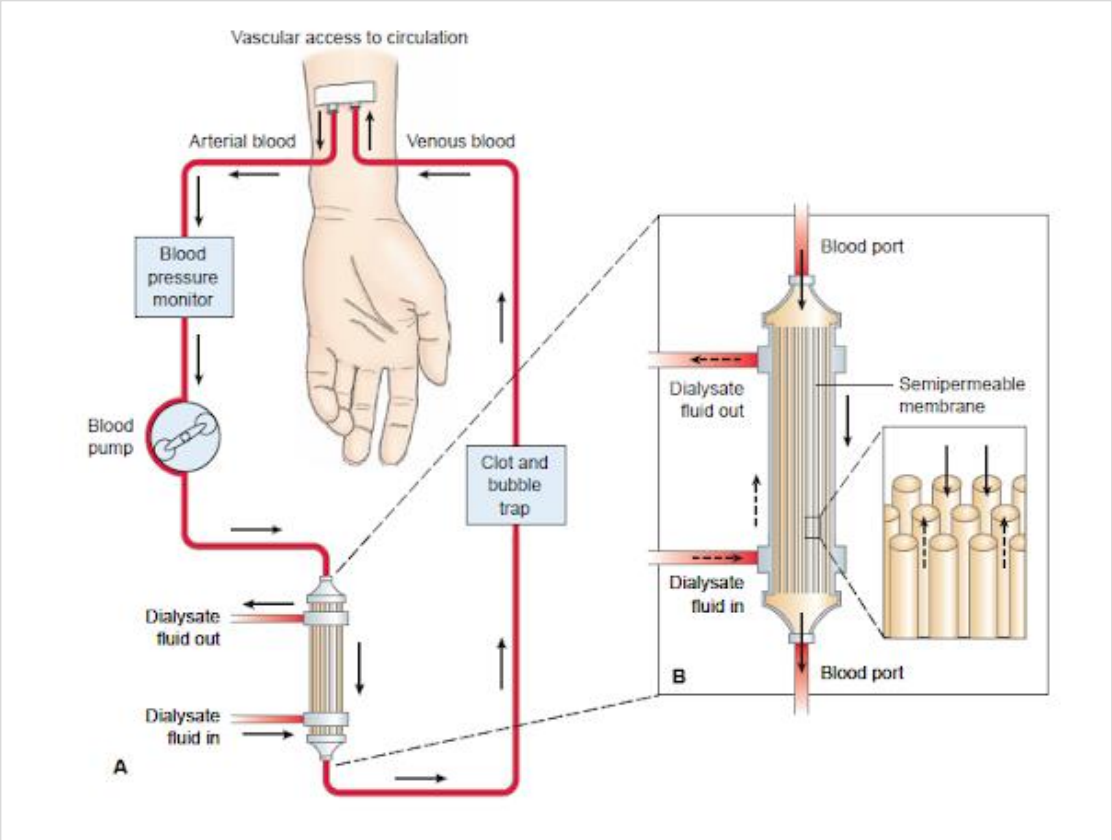

Figure 2.3 Hemodialysis system

2.3.4 Patient safety and technical advances

The risk of dying while undergoing hemodialysis has been greatly reduced, and such fatalities are almost unheard of. Thanks to enhanced dialysate delivery systems, better tracking devices, and automated protection mechanisms, complications have been minimized. The addition of more physiological bicarbonate-based dialysate, improved water standards, volumetric ultrafiltration controls, and computer-controlled sodium and potassium modeling are also technological advances. Some in-line devices are now able to track the flow of blood via the vascular access dynamically (Lopot *et al.* 2004), adjustments to haemoglobin concentration (to assess the replenishment of the vascular

system during ultrafiltration), as well as alterations to the electrical conductivity of the dialysate (to estimate the amount of solute being removed) (Maduell *et al.* 2008).

Because of this, dialysis machines with feedback-control systems now let you adjust the ultrafiltration rate in real time using a computer (Ward and Ronco 2009). By enabling consistent, automated temperature monitoring of dialysate, dialysis patients are more likely to have a steady body temperature, reducing the danger of an intradialytic hypotension (Maggiore *et al.* 2002). Studies in small patient populations indicate that in-line monitoring or feedback-control systems offer benefits, but major studies that meet rigid standards of research have not proven improved outcomes (Locatelli *et al.* 2005).

2.3.5 Complications of hemodialysis

Advanced Renal Education Program says that hemodialysis access problems include infections, aneurysms, venous hypertension, hematomas, heart failure, and persistent bleeding. The spread of infectious problems in vascular access in hemodialysis patients leads to considerable morbidity and death. Infection is responsible for 9.5-36% of hemodialysis patient mortality, according to previous estimates. In up to 48 to 73% of hemodialysis patients, vascular access site infections (mostly in patients with CVC) are confirmed to be the cause of all bacteremia. For tunnel cuffed catheters, the estimated risk of bacteremia is 2.3/1000 catheter days, indicating a 20 to 25% bacteremia risk over the average period of use. Infection occurs 10% of the time in AV grafts and 5% of the time in AV fistulas (Adhikari and Aryan 2019).

2.4 Health Problems With Water and Sodium Regulation

Emergency care doctors encounter electrolyte abnormalities of the most common type: water and sodium balance anomalies. 10% of patients in the emergency department suffer from hyponatremia, whereas 2% suffer from hypernatremia (Arampatzis *et al.* 2012).

It's not surprising that the vast majority of symptoms related to salt and water balance, and the nearness of the nervous system to the skull, are neurologic and life-threatening. Many of the fundamental concepts of understanding these disorders include fluid compartments, osmolality regulation, and the need of early diagnosis and appropriate treatment (Harring *et al.* 2014).

2.4.1 Physiology

The amount of water in the body, accounting for about 60% of total body weight in adults (Figure 2.4); varies depending on age and gender. The following equation may provide a more realistic depiction of TBW:

$$\text{TBW} = \text{weight (kg) X correction factor 1} \tag{2.1}$$

In total, the TBW is separated into two major components: intracellular fluid, around 40% of total body weight, and extracellular fluid (ECF), about 20% of total body weight. Two-thirds of the ECF is interstitial fluid, whereas one-third is intravascular fluid. 5% of total body weight is composed of the fluid within the vessels. The ECF's solute of choice is sodium, which has a concentration of 140 mEq/L in its normal state. Neurologic symptoms start to surface when salt concentrations in the skull change. These signs and symptoms can range from mild to life-threatening. Vasopressin and thirst control are the two main pathways that regulate sodium levels. An average stable adult requires around 1 to 3 litres of water a day to maintain adequate fluid retention (Kaplan and Kellum 2010, Kelen and Hsu 2011). This amount of water dissolves water, which is drained from the body through insensible losses and urine performance, from the respiratory tract 500 to 700 mL/day, from the skin 250 to 350 mL/d and from the faeces 100 mL/d (Kelen and Hsu 2011).

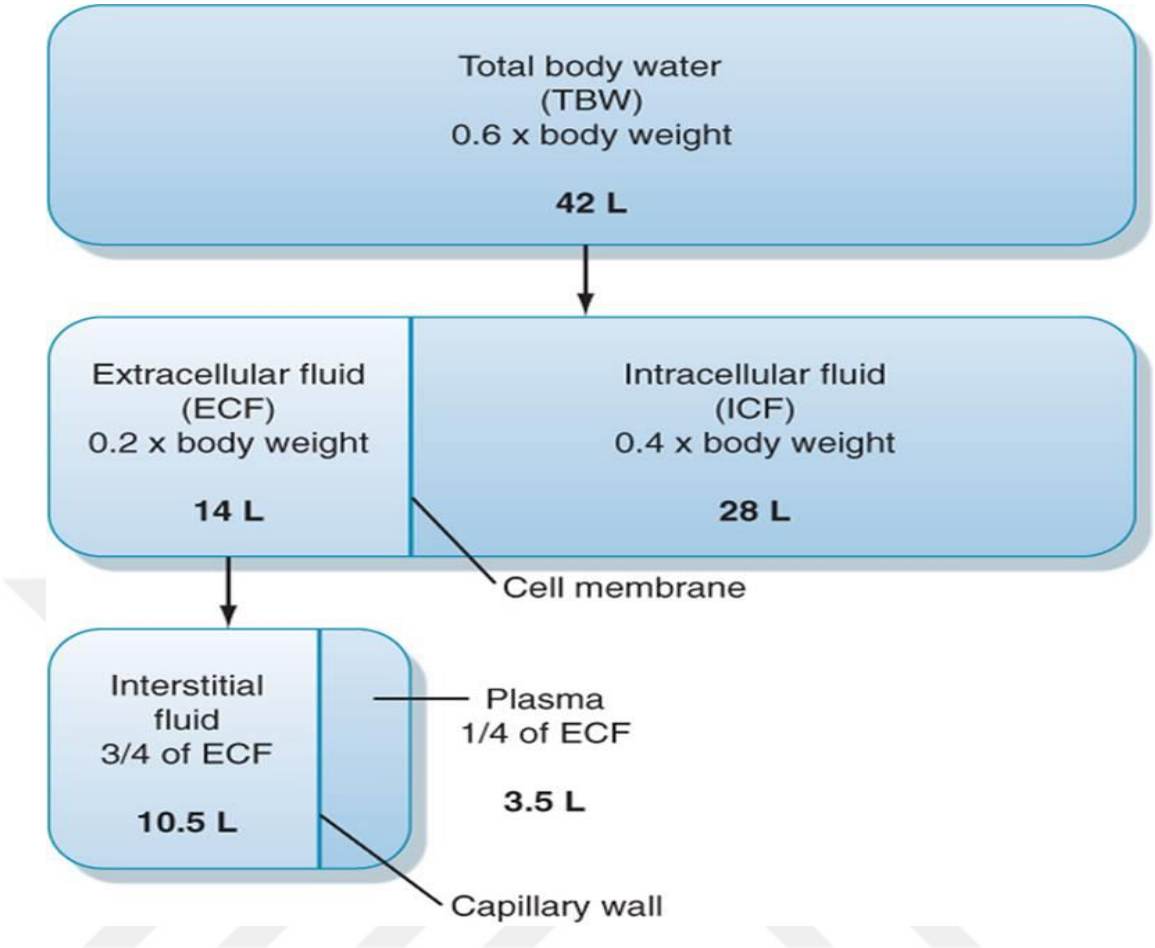

Figure 2.4 Relationship of fluid compartments to total body weight

2.4.2 Hyponatremia

Hospitalized patients and those with chronic medical problems often to have hyponatremia (sodium levels less than 135 mEq/L) or severe hyponatremia (levels below 125 mEq/L). Around 3 to 6 million people in the US have hyponatremia every year, and about a quarter of them need emergency care in the ER (Boscoe *et al.* 2006). According to Lee *et al.* (2000), It's estimated that 4% of all ER patients with adult medical conditions have hyponatremia, and 15% of patients admitted to the hospital are suffering from the condition. 10 Hyponatremia patients have an average mortality rate of 10% and an increased mortality risk of 33% compared to normal sodium levels. There is no correlation between the severity of hyponatremia and the likelihood of death in general (Hoorn and Zietse 2013).

2.4.2.1 Signs and symptoms

While some individuals may have no symptoms, others may have convulsions as a result of hyponatremia. An individual's odds of experiencing and overcoming cerebral edema, as well as the degree and pace of improvement in sodium levels, is often correlated with their symptoms. The following symptoms are often reported by patients: headaches, tiredness, vomiting, restlessness, anorexia, stomach pains, lethargy, and disorientation. The brain attempts to counteract cerebral edema by trying to release intracellular solutes as quickly as possible to avoid hyponatremia,12 and this becomes a therapeutic component. Patients with symptomatic hyponatremia will often come with some kind of neurologic complaint, although some may appear after a fall with severe injury (Arampatzis *et al.* 2012).

2.4.2.2 Pathogenesis

The total body weight of an average male, about 60% is made up of water, while the average female's water content is 50% of total body weight. Two-thirds of total body water (TBW) is intracellular fluid (ICF), whereas one-third is extracellular fluid (ECF). Interstitial fluid accounts for 26% of total body water (TBW). See Figure 2.4.

It is vital to understand that hyponatremia is often the result of increased extracellular water, not decreased extracellular sodium (Spasovski *et al.* 2014).

Hyponatremia is just another term that means water balance problem. Overall body water levels may vary (raise with congestive heart failure, CHF, and decrease with vomiting and diarrhea) (as in the syndrome of inappropriate ADH secretion SIADH). Because of an imbalance in the extracellular water and ions, hyponatremia results, regardless of overall body sodium (the major extracellular ions are sodium and chloride). Water flows freely through the intracellular and extracellular environments. As a consequence, the ECF and the ICF have the same osmolality. The equilibrium of water absorption and excretion determines serum sodium levels. Since ingested water is not adequately excreted in hyponatremia, serum sodium drops, while in hypernatremia, water consumption is insufficient (Verbalis *et al.* 2013).

2.4.3 Hypernatremia

Hypernatremia is more common than hyponatremia since its serum sodium content is more than 145 mEq/L. Hypernatremia is often observed in hospital patients, although approximately 0.2% of those visiting the emergency department also have it (Palevsky *et al.* 1996). Hypernatremia is often synonymous with intracellular dehydration exacerbated by a drop in TBW, as well as a reduction in free water consumption. Without enough water consumption to offset losses from intestinal, urine, and pulmonary lossesA patient will become thirstier and saltier until they drink free water, even if their kidney function is fine (Kovacs and Robertson 1992).

People with poor thirst-regulation and water-gathering capability, such as the elderly, youngsters, and those with disabilities (e.g., people on ventilators or in comas) are more prone to suffer from hypernatremia (Robertson *et al.* 1982).

2.4.3.1 Signs and symptoms

Hypernatremia symptoms are almost identical to those of hyponatremia since the conditions that cause both conditions are heavily focused on the central nervous system (Lindner and Funk 2013). When water seeps into the extracellular space, brain cells substantially shrink when hypernatremia is present. Cerebral hemorrhage is caused by the tearing of cerebral blood vessels (Adrogue and Madias 2000). Muscle cramps, hyperventilation, impaired glucose use, and decreased contractility of the left ventricle are all consequences of hypernatremia. 99 Patients and infants alike may exhibit symptoms of tiredness, exhaustion, or restlessness, or irritability. Neurologic testing may reveal signs of excessive muscle tension, stiff neck, spasmodic reflexes, asterixis, chorea, or seizures (Moritz and Ayus 2005).

Seizures, coma, or death may occur if the patient is not properly diagnosed and treated (Kelen and Hsu 2011). In one research, it was shown that individuals with hypernatremia had a decreased chance of receiving specialized dysnatremia therapy and a greater death rate in the hospital. While the absolute increase in serum sodium is important, it is also

necessary to note the speed of that increase, since this corresponds to the pace of dehydration of brain cells (Lindner *et al.* 2009).

2.4.3.2 Diagnosis of hypernatremia

A water deprivation test may be used to distinguish hypernatremia from diabetes insipidus, which is suspected in patients with diabetes insipidus (Fenske and Allolio 2012). Since it is both difficult and time-consuming, it is employed in circumstances when it is difficult to ascertain the diagnosis. It need a professional's help. The test is usually performed in a hospital environment on individuals who have central DI, have very low urine osmolality (less than 100 mOsm/kg/H^2O), and have severe polyuria. The patient is incapable of consuming food or beverages (NPO). The amount of urine produced in an hour and body weight are recorded. On entrance, all three of the aforementioned parameters are tested: urine osmolality, serum sodium, and serum osmolality. The first urine osmolality test is then performed hourly, while the latter two are performed every two hours (Fenske and Allolio 2012, Tinawi 2020).

3. MATERIALS AND METHODS

3.1 Materials

The study involved the devices and equipments which used in the lab as shown in Table 3.1.

Table 3.1 A list devices and equıpments used in the present study

Equıpments and Devices	Company	Country
Spectrophotometer	Biobase	India
Centrifuge	Memmert	Germany
Oven	Memmert	Germany
Water bath	Memmert	Germany
Shaking water bath	Memmert	Germany
Sysmex device	Sysmax	Japan
Pipette	BioSan	Germany
Different glasses	-----	China
Refrigerator	BEKO	Turkey

3.2 Study Design

People with CKD as diagnosed by a physician. Forty healthy individuals were chosen as the control group. Patients were assigned to five stages of CKD based on serum creatinine, which was used to assess the degree of their disease, with stage 1 being the least severe and stage 5 being end stage renal disease (ESRD).

3.3 Setting of The Study

From November 2020 to February 2021, 109 patients were confirmed to be sick with chronic renal disease at Al-Jumhuri Hospital. Private laboratories in Kirkuk, Iraq, were used to conduct the research.

3.4 Target Population

Chronic renal disease patients of both genders (65 males and 44 females) aged 20 to 71 years old who visit Al-jumhuri Hospital in Kirkuk, Iraq, are the target population. Each end-stage renal disease patient got four hours of hemodialysis treatment two or three times each week.

3.5 Collection of Blood Samples and Processing

Each patient's blood samples were taken by specially qualified nurses. In terms of the need for freshly collected blood samples, scientists disagree (Hall *et al.* 1995, Clarke and Higgins 2000). As a result, it is not necessary to use freshly collected blood. Every subject had five millilitres of venous blood drawn and separated into an EDTA tube (1.0 ml) (Figure 3.1) and gel vacutainer tube (4.0 ml) (Figure 3.2).

Vacutainer simple tubes were left for a brief period to cause blood to clot before being centrifuged at 4000 rpm for 10 minutes to extract transparent serum samples using the centrifuge as shown in the Figure 3.3. The isolated serum was inserted in five clear bottles, sealed, and held at -20 0C until the study was completed. Until use, the frozen serum samples were thawed at 4-8 C and gently mixed at room temperature.

Figure 3.1 EDTA (hematology tests) (CBC)

Figure 3.2 Gel vacutainer tube for chemistry tests

Figure 3.3 Centrifuge machine 800-8

3.6 Estimated Glomerular Filtration Rate (eGFR)

The serum creatinine test methodology used in this study follows the instructions from the manufacturer. The CKD-EPI equation was used to determine the concentration.

eGFR= 141 x (SCr/k) a x0.993Agex [1.018 if Female] x [1.159 if Black] (3.1)

- The values for serum creatinine for female and male were -0.329 and -0.411, respectively.
- If both male and female serum creatinine are above 0.9 and 0.7, respectively, a=- 1.209
- For female, k=0. 7, while for male, k=0. 9.
- Serum creatinine concentration =SCr.

3.7 Biochemical Tests

3.7.1 Kidney funtion parameters

3.7.1.1 Urea

Urease, an enzyme, transforms urea into ammonia and carbon dioxide. GlDH is able to convert ammonia to glutamate when NADH and oxoglutarate are present. The rate of the absorbance reduction due to the NADH oxidation to NAD+ in the presence of urea is modulated kinetically at 340 nm.

3.7.1.1.1 Test procedure

1. Prior to incubation, prepare working reagent, samples, and standards to 37ºC.
2. Measure the absorbance of the photometer with distilled water using the spectrophotometer as shown in Figure 3.4.
3. Transfer to a cuvette. According to the following Table 3.2.

Figure 3.4 Spectrophotometer

Table 3.2 Urea procedure steps

Pipette	Blank	Sample	Standard
Working reagent	1.0 ml	1.0 ml	1.0 ml
Standard	-	-	10 µl
Distilled Water	10 µl	-	-
Sample	-	10 µl	-

Mix and after 30 seconds of incubation at 37ºC, read against water the initial absorbance. Read again after exactly 60 seconds. Calculate the variation value of the absorbance of the sample and the standard at 340 nm.

3.7.1.1.2 Calculations

Urea concentration was calculated from the absorbance depending on the equation below:

Results= A2-A1 (sample) / A2-A1 (standard) x C Standard = mg/dl Total urea (3.2)

3.7.1.2 Creatinine

The technique uses a modified picrate reaction (Jaffe). Creatinine is known to produce a crimson matrix in alkaline circumstances by reacting with picrate ions. The complexity of formation increases as the concentration of creatinine in the sample rises.

3.7.1.2.1 Test procedure

1. Prior to incubation, prepare working reagent, samples, and standards to 37ºC.
2. Measure the absorbance of the photometer with distilled water using the spectrophotometer as shown in Figure 3.4.
3. Transfer to a cuvette. According to the following Table 3.3.

Table 3.3 Creatinine procedure steps

Pipette	Blank	Sample	Standard
Working reagent	1.0 ml	1.0 ml	1.0 ml
Standard	-	-	100 µl
Sample	-	100 µl	-

Place the sample (Ax1) and the standard in a 37°C incubator for 30 seconds and then read the absorbance of the sample (As1). Read the sample absorption (Ax2) and standard (As2) at 492 nm after precisely a minute after the first measurement.

3.7.1.2.2 Calculations

Creatinine concentration was calculated from the absorbance depending on the equation below:

Results= A2-A1 (sample) / A2-A1 (standard) x C Standard = mg/dl
Total creatinine (3.3)

3.7.2 Electrolytes

3.7.2.1 Sodium (Na^+)

It was formerly common to precipitate sodium as the triple salt, sodium uranyl zinc acetate, before using flame photometry and ion-selective electrodes to measure it in bodily fluids. Kolthoff's technique was launched in 1927, and it has since been used in a number of ways. Albanese and Lein (1948) used colorimetry to determine the solubilized residue directly, while Bradbury tested the yellow supernate for fading. This method is really an expansion of the technique already mentioned, which extracts a triple salt from a protein-free supernate. The specimen's sodium concentration corresponds to the reduction in the supernate-color reagent mixture's absorbance.

3.7.2.1.1 Test procedure

After each addition of the color reagent, pipet the following quantities (mL) into the indicated tubes and mix immediately. According to the following Table 3.4.

Table 3.4 Sodium procedure steps

Pipette	Blank	Sample	Standard
Reagent	1.0 ml	1.0 ml	1.0 ml
Standard	-	-	10 µl
Sample	-	10 µl	-

Incubate the tubes at room temperature for 10 minutes (15-30o C). Read and record Reagent Blank, Standard and Sample absorptions at 630 nm within 20 minutes.

3.7.2.1.2 Calculations

Sodium concentration was calculated from the absorbance depending on the equation below:

$$\text{Results} = \text{Abs (sample)} / \text{Abs (standard)} \times \text{C Standard} = \text{mEq/L Total Sodium} \quad (3.4)$$

3.7.2.2 Potassium (K^+)

For the purpose of monitoring electrolyte balance, potassium concentrations are used in the treatment and diagnosis of illnesses characterized by low or high potassium levels in the blood. Potassium is tested using a kinetic coupling assay device that utilizes pyruvate kinase and potassium. The pyruvate produced is transformed to lactate, and the NADH is turned to NAD in the process. As the serum potassium level goes up, the optical density at 380 nm decreases in proportion.

3.7.2.2.1 Test procedure

Pipette into clean dry testtubes labeled as blank (B), standard (S), and test (T), According the steps wich showing in Table 3.5.

Table 3.5 Potassium procedure steps

Pipette	Blank	Sample	Calibrator
R1 Reagent	1.0 mL	1.0 mL	1.0 mL
Calibrator	-	-	25 µL
Sample	-	25 µL	-
Mix, incubate for 5 minutes at 37ºC.			
R2 Reagent	250µL	250µL	250µL

Incubate for 1 minute at 37ºc and read at 405 nm (A1).
Incubate at 37ºc for 3 minutes and read (A2) at 405 nm.

3.7.2.2.2 Calculations

Potassium concentration was calculated from the absorbance depending on the equation below:

Results= A2-A1 (sample) / A2-A1 (standard) x C Standard = mEq/L Total Potassium (3.5)

3.8 Statistical Analysis

The data was statistically evaluated using the Minitab statistical programme, which was run on an SPSS and Microsoft Excel XP framework. The data was interpreted as mean SD (standard deviation), minimal, and maximum values in a straightforward manner.

To determine whether the variability between control and treatment groups was significant, a statistical analysis utilizing the Analysis of Variance (ANOVA) test was used. Tests utilizing Duncan's Multiple Range were used to compare the data's various methods.

Statistical significance was established at values higher than 0.05, whereas values less than 0.05 were deemed statistically significant.

$P > 0.05$	Non-significant
$P < 0.05$	Significant
$P < 0.01$	Highly significant (Beth *et al.* 2004).

4. RESULTS

4.1 Ages of Patients

The research showed that those with renal failure in the highest age group were 50-59 years, where it was 26.2%. Then, the age group was 60-69, as it was 22.1%, while the lowest age group of patients of renal failure was 30-39, where it was 5.4%, as shown in the Table 4.1.

Table 4.1 Age groups of in present study

Gender / Age (year)	Male	Female	Total
20-29	16(57.1%)	12(42.9%)	28(18.8%)
30-39	5(62.5%)	3(37.5%)	8(5.4%)
40-49	6(66.7%)	3(33.3%)	9(6%)
50-59	18(46.2%)	21(53.8%)	39(26.2%)
60-69	16(48.5%)	17(51.5%)	33(22.1%)
≥71	24(75%)	8(25%)	32(21.5%)
Total	85(57.04%)	64(42.96%)	149(100.0%)

4.2 Estimated Glomerular Filtration Rate (eGFR)

The research findings, which revealed percentage of healthy people in the current study was 40 (26.8%), while the number of patients with kidney failure was 83 (55.7%), while other stages of kidney disease were reported, the third stage included 9 (6%) patients while. The fourth stage included 17 (11.4%) patients, as shown in Table 4.2.

Table 4.2 The percentage of CKD stages in current study

Gender / Stages	Mean ± SD	Male	Female	Total
Stage 1	124.67±16.26 a	20(23.5%)	20(31.3%)	40 (26.8%)
Stage 2	———	———	———	———
Stage 3	32.75±3.18 b	6(7.1%)	3(4.7%)	9(6%)
Stage 4	18.57±2.72 c	9(10.6%)	8(12.5%)	17(11.4%)
Stage 5	12.01±2.44 d	50(58.8%)	33(51.5%)	83(55.7%)
Total	———	85(57.04%)	64(42.96%)	149(100.0%)

4.3 Kidney Functions

4.3.1 Creatinine levels

The results of the current study demonstrated significant differences between studied groups. Whereas, creatinine level in male (8.95±2.23) was showed significant (P≤0.05) increase compare with group control (0.86±0.21). Also, creatinine level in female (4.2±1.75) was showed significant (P≤0.05) increase compare with group control as shown in the Table 4.3 and Figure 4.1.

Table 4.3 Creatinine level in studied groups

Groups / Parameter	Control	Patients	
		Male	Female
S. creatinine	0.86±0.21 c	8.95±2.23 a	4.2±1.75 b
P-Value	0.000*		

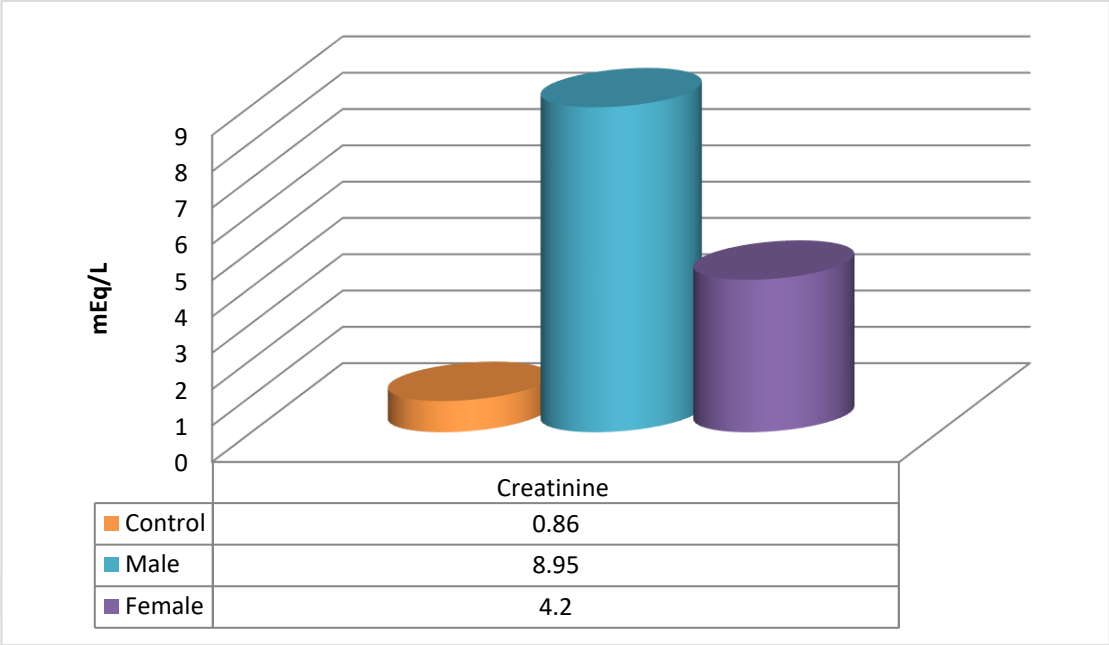

Figure 4.1 Creatinine level in studied groups

4.3.2 Urea levels

The results of the current study demonstrated significant differences between studied groups. Whereas, urea level in male (135.2±24.1) was showed significant (P≤0.05) increase compare with group control (20.5±6.19). Also, urea level in female (79.4±20.39) was showed significant (P≤0.05) increase compare with group control as shown in the Table 4.4 and Figure 4.2.

Table 4.4 Urea level in studied groups

Gender / Parameter	Control	Male	Female
S. urea	20.5±6.19 c	135.2±24.1 a	79.4±20.39 b
P-Value	0.000*		

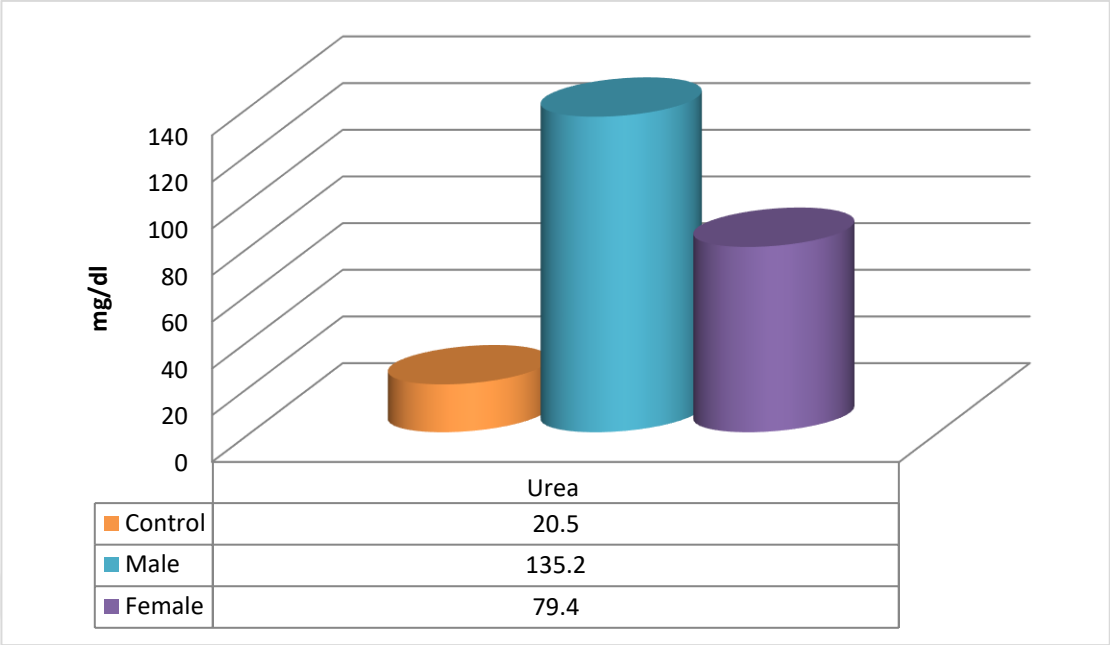

Figure 4.2 Urea level in studied groups

4.4 Electrolytes

4.4.1 Sodium level

The results of the current study demonstrated significant differences between studied groups. Whereas, sodium level in male (139.0±3.39) was showed significant (P≤0.05) decrease compare with group control (145.06±3.53). Also, sodium level in female (135.0±4.69) was showed significant (P≤0.05) decrease compare with group control as shown in the Table 4.5 and Figure 4.3.

Table 4.5 Sodium level in studied groups

Gender / Parameter	Control	Male	Female
S. sodium	145.06±3.53 a	139.0±3.39 b	135.0±4.69 b
P-Value	0.000*		

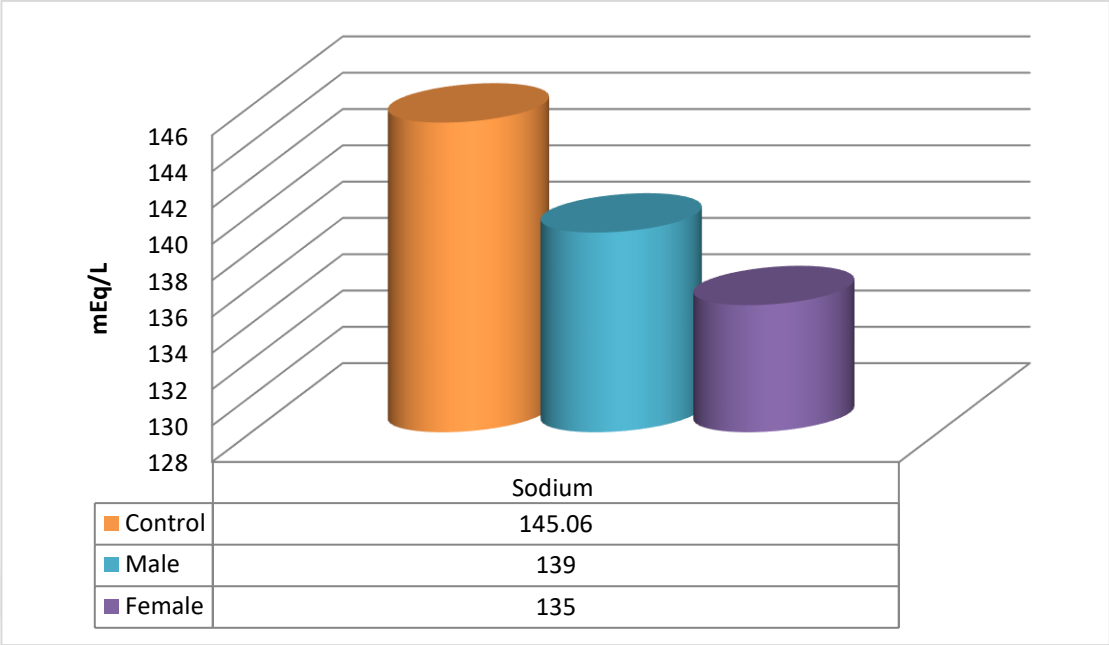

Figure 4.3 Sodium level in studied groups

4.4.2 Potassium level

The results of the current study demonstrated significant differences between studied groups. Whereas, potassium level in male (5.62±0.28) was showed significant (P≤0.05) increase compare with group control (3.56±0.23). Also, potassium level in female (4.92±0.29) was showed significant (P≤0.05) increase compare with group control as shown in the Table 4.6 and Figure 4.4.

Table 4.6 Potassium level in studied groups

Groups / Parameter	Control	Patients	
		Male	Female
S. potassium	3.56±0.23 a	5.62±0.28 b	4.92±0.29 b
P-Value	0.000*		

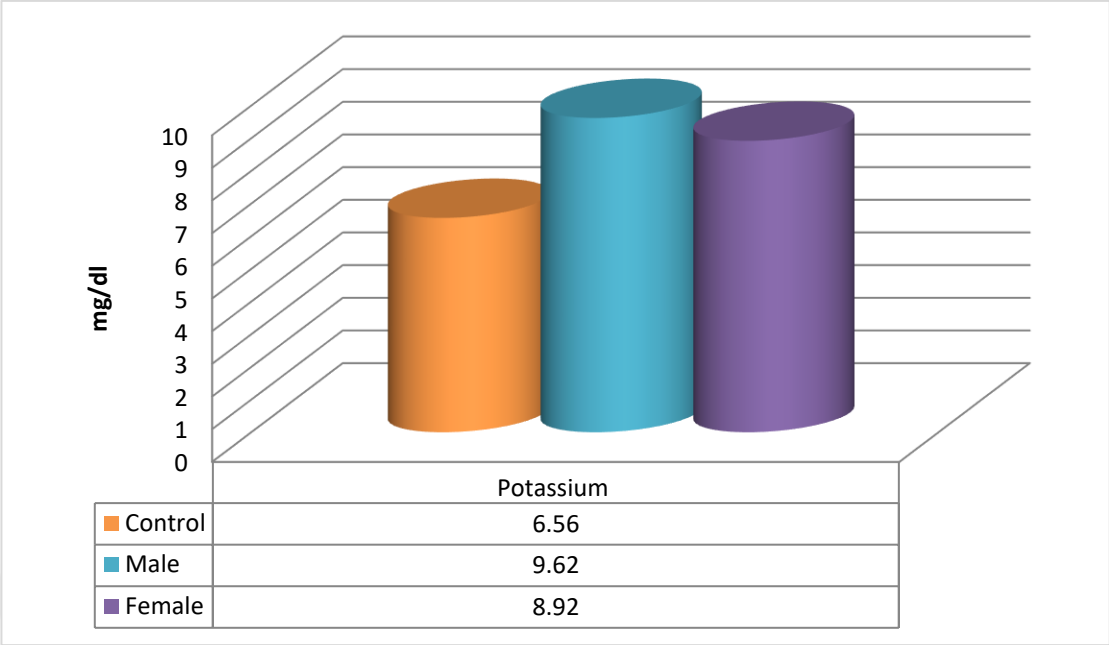

Figure 4.4 Potassium level in studied groups

4.5 Correlations

4.5.1 GFR and sodium

This study's findings showed a correlation that is positive between GFR and sodium, as it was observed that the lower the levels of GFR, the lower the levels of sodium and the levels of correlation were R^2= 0.9019, as shown in the Figure 4.5.

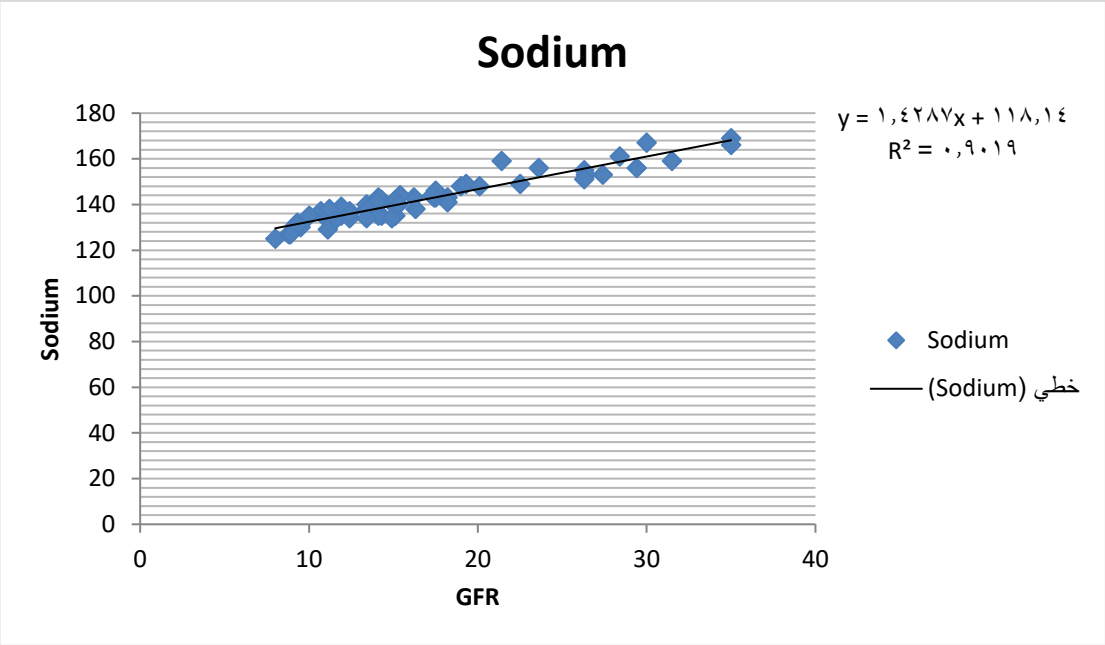

Figure 4.5 Correlation between GFR and sodium

4.5.2 GFR and potassium

The results of the current study showed an inverse correlation between GFR and potassium, as it was observed that the lower the levels of GFR, the higher the levels of potassium and the levels of correlation were R^2= 0.8498, as shown in the Figure 4.6.

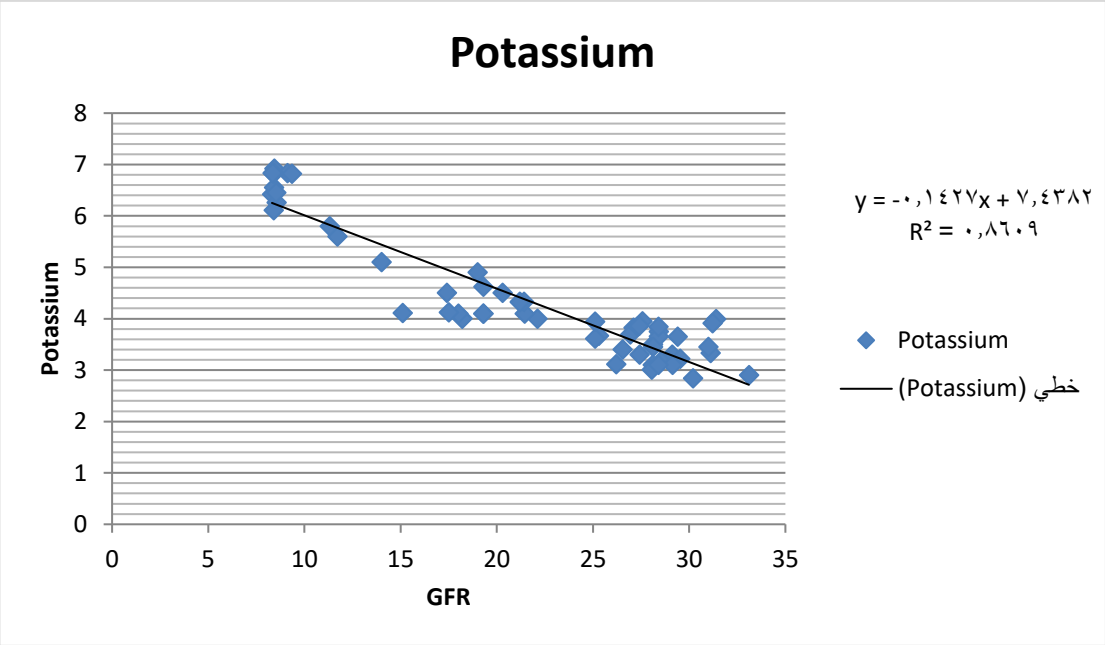

Figure 4.6 Correlation between GFR and potassium

4.6 Complete Blood Counts (CBC)

4.6.1 Red blood cells (RBC) count

The current study's findings revealed substantial variations between the groups surveyed. In contrast, the RBC count in males (3.51±0.41) was significantly lower ($P \leq 0.05$) than in the group control (5.47±0.32). In addition, the RBC ratio in females (3.83± 0.19) decreased significantly ($P \leq 0.05$) as compared to the group control, as seen in Table 4.7 and Figure 4.7.

Table 4.7 RBC count in studied groups

Gender / Parameter	Control	Patients	
		Male	Female
RBC ($X10^6$)	5.47±0.32 a	3.51±0.41 b	3.83± 0.19 b
P-Value	0.000*		

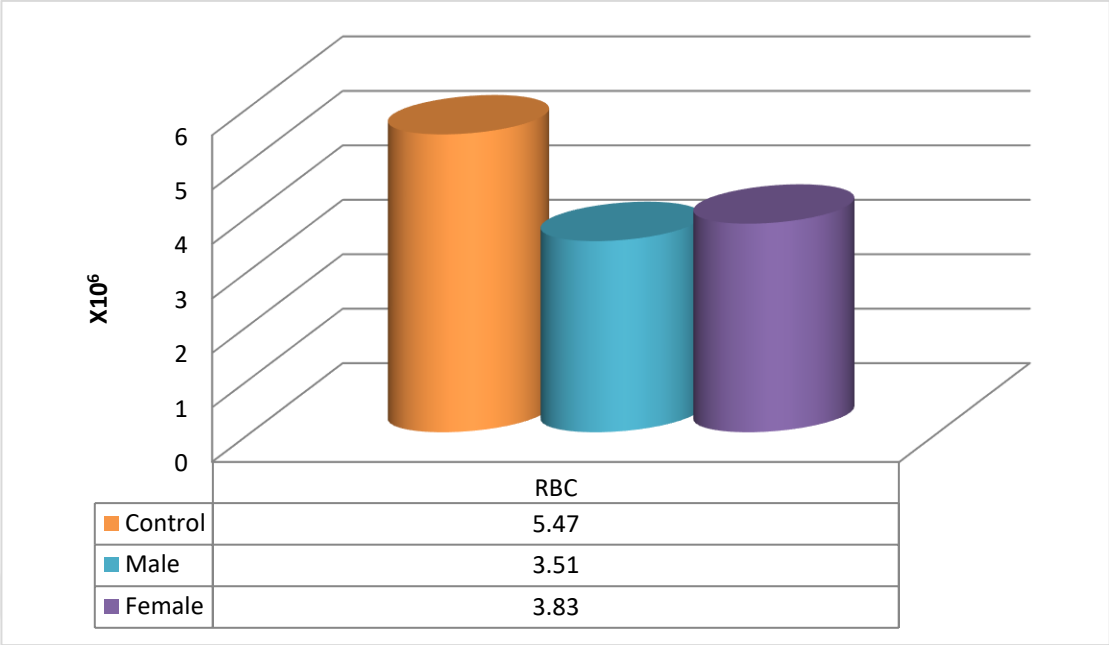

Figure 4.7 RBC count in studied groups

4.6.2 Hemoglobin (Hb) Level

The current study's findings revealed substantial variations between the groups surveyed. In contrast, Hb levels in males (7.54±1.25) were significantly lower (P≤0.05) than in the group control (13.47±0.62). In addition, the Hb level in females (9.81±1.37) was significantly lower (P≤0.05) than in the group control, as seen in Table 4.8 and Figure 4.8.

Table 4.8 Hb level in studied groups

Gender / Parameter	Control	Patients	
		Male	Female
Hb	13.47±0.62 a	7.54±1.25 b	9.81±1.37 b
P-Value	0.000*		

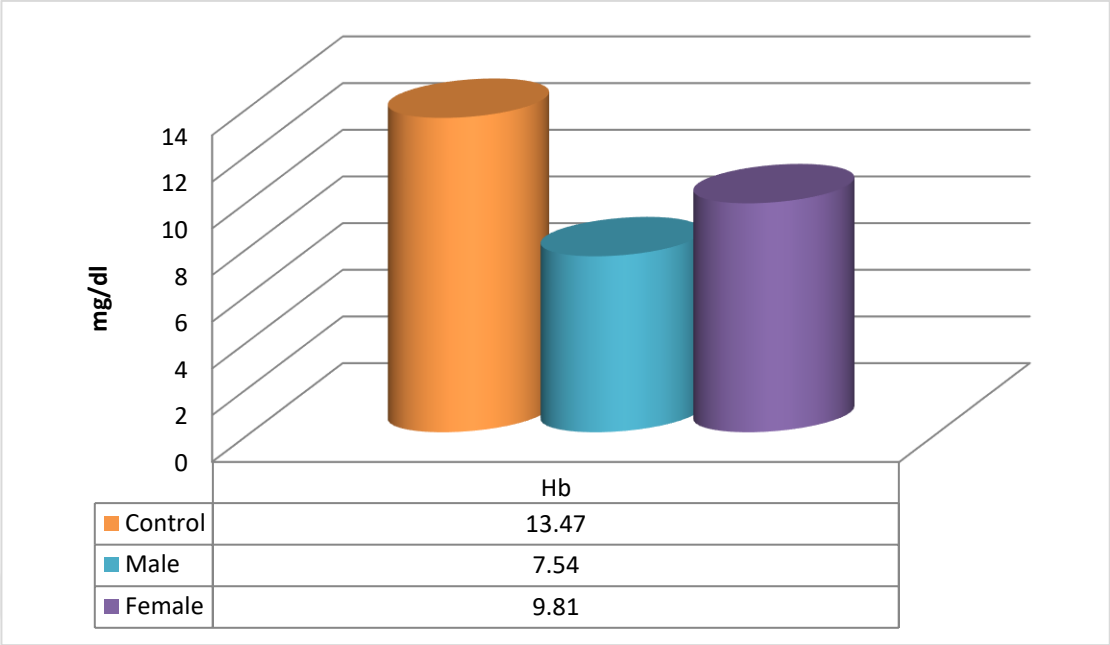

Figure 4.8 Hb level in studied groups

4.6.3 Packed cell volume (PCV)

The current study's findings revealed substantial variations between the groups surveyed. In contrast, PCV levels in males (27.32±2.1) were significantly lower (P≤0.05) than in the control group (41.86±3.05). In addition PCV levels in females (31.74±3.1) were significantly lower (P≤0.05) than in the control group as shown in the Table 4.9 and Figure 4.9.

Table 4.9 PCV level in studied groups

Gender / Parameter	Control	Patients	
		Male	Female
PCV	41.86±3.05 a	27.32±2.1 b	31.74±3.1 b
P-Value	0.000*		

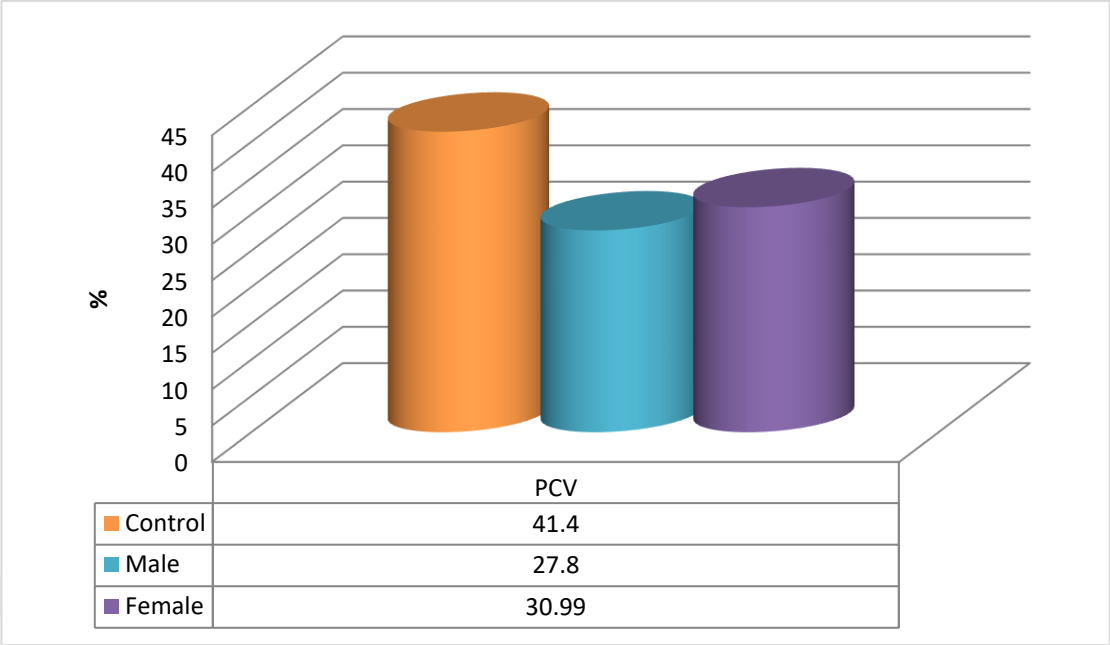

Figure 4.9 PCV level in studied groups

4.6.4 Mean corpuscular volume (MCV)

The current study's findings revealed substantial variations between the groups surveyed. In contrast, the MCV amount in males (84.92±4.17) was found to be non-significant (P≤0.05) change compared with control group (87.13±5.1). In addition, the MCV% in females (86.13±5.5) was non-significant (P≤0.05) change compared with control group as shown in the Table 4.10 and Figure 4.10.

Table 4.10 MCV level in studied groups

Gender / Parameter	Control	Patients	
		Male	Female
MCV	87.13±5.1 a	84.92±4.17 a	86.13±5.5 a
P-Value	0.908		

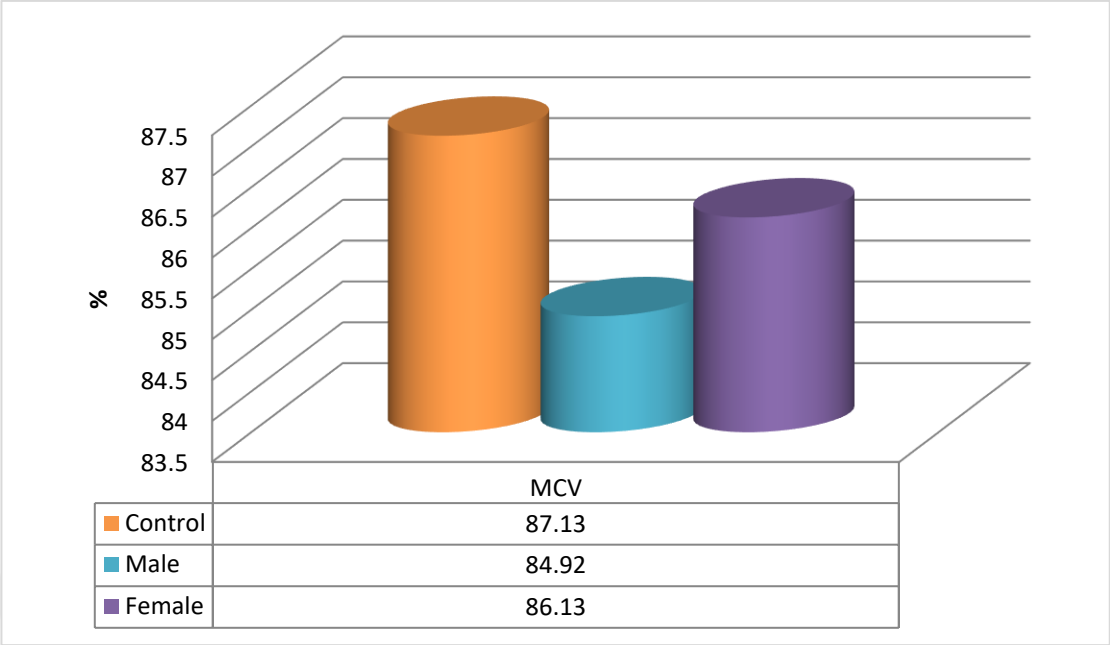

Figure 4.10 MCV level in studied groups

5. DISCUSSION

5.1 Kidney Function (Urea & Creatinine)

People with chronic renal disease (CKD) suffer from kidney failure that worsens with time that necessitates long-term treatment with renal replacement therapy. Haemodialysis is a form of renal replacement therapy in which waste materials such as creatinine, urea, and excess water are eliminated from the body. CKD was also found to be more common in males than females during the search, which agrees based on the results and et al (2014) in Pakistan (Hida *et al.* 1985) found that people between the ages of 40 and 60 are more affected. The cause may be due to arthritis, diabetes, or other age-related improvements. The removal of waste during dialysis is often dependent on proper dialysis scheduling, patient awareness, and patient dialyzer and dietary patterns (Hayrullah *et al.* 2011).

Leafy green vegetables and meat are known to put a strain on the kidneys, resulting in a rise in serum urea and creatinine levels (Kaysen *et al.* 2012). It was also discovered that middle-aged men are more susceptible to CKD. To find a plausible solution, the molecular origin of this interaction must be investigated (Schieppati and Remuzzi 2005). Long-term kidney failure results in decreased renal clearance, or glomerular filtration rate (GFR), causing substances like urea and creatinine to build up in the blood.

According to the findings of this report, the amount of creatinine in the patients' population was significantly higher than in the group control, and men were more affected than women. This finding is in line with the results of Al- Rawi *et al.* (2014), who found a substantial rise in creatinine concentration in the serum of patients with permanent renal disease (7.643.57 mg/dL) as compared to the control group, where the average creatinine concentration was (0.880.07 mg/dL). The fact that creatinine is one of the metabolic wastes that is normally excreted by urine may account for the high concentration of creatinine in the serum of peoples with chronic renal disease. The concentration of creatinine in the blood serum is inversely proportional to the rate of glomerular filtration; a small reduction in the glomerular filtration rate causes a rise in the creatinine concentration in the blood (Zilva *et al.* 1988).

While the rate of decline in kidneys function (–1.64 mL/min/1.73m^2) seems to be sluggish, the average baseline eGFR was (22.5 mL/min/1.73m^2). The same increases in serum creatinine (mg/dL) resulted in smaller changes in eGFR in early stages of CKD (Yuste *et al.* 2013).

In addition, in contrast to the healthy group, the amount of urea and serum creatinine in CKD patients was slightly higher ($p \leq 0.01$) than in the healthy group. The other experiments agreed with these findings (Souza *et al.* 2011). Skoreck and Porth explained that the kidneys remove vital solutes such as serum creatinine and blood urea, and that urea was found in the blood of CKD patients as the first organic solvent. When the kidneys lose their capacity to expel nitrogenous wastes from the bloodstream, these contaminants build up, causing urea and creatinine levels to increase (Skorecki *et al.* 2006, Porth 2007).

Increased protein consumption, shocking, and gastrointestinal bleeding all contribute to a spike in blood urea and serum creatinine levels (Anderson 1996). When renal function drops to about 25-50%, blood urea levels rise, making it a responsive predictor of renal disease (Sharma *et al.* 2011) However, Moses and Johnkennedy (2013) found that creatinine levels increased marginally as a result of kidney injury, resulting in a decrease in GFR due to kidney inflammation. Often, creatinine was used to assess kidney failure and the development of CKD (Stevens *et al.* 2009).

The kidneys' decreased filtration rate causes them to be unable to filter blood and remove waste, which results in a buildup of waste and toxic substances in the blood, which causes renal failure (Israa and Mohammed 2013, Delanaye *et al.* 2017).

5.2 Sodium Concentration

Sodium is used to assess electrolyte balance and kidney function, as well as to control chronic or acute hypernatremia (high blood sodium) or hyponatremia (low blood sodium).

Salt consumption may need to be restricted in patients with elevated blood pressure and chronic kidney disease (Armbruster 2013).

The new findings revealed that the amount of sodium concentration in the blood serum was lower in the treatment population as comparison to the group control. This findings is in accordance with the study of Al-Rubaei *et al.* (2010) who stated that the sodium concentration (134.724.2 mmol/l) in the patients group was significantly lower than the sodium concentration (142.532.5 mmol/l) in the control group, and that the cause for this is due to the absence of sodium absorption a second time after filtering as a result of renal tubule inflammation (Reynolds *et al.* 2006). As well as a decrease in the activity of $Na/^{+}K^{+}$-ATPase enzyme, this leads to a decrease in the sodium exchange with hydrogen ions in the renal tubules (Tietz *et al.* 1986).

The elimination of natural or near-natural diuresis contributes to the elimination of sodium, and thus its level in the body decreases, and the occurrence of Odema leads to a dilution of sodium within the body, and thus a reduction of its level as well as an increase in acidity in the body causes sodium loss by causing disruption to the tubules, which causes sodium loss (Al-Rawi *et al.* 2014).

We found that sodium decreased in the treatment population as compared to the stable group, with statistically important variations ($p \leq 0.05$). These findings support the findings of another study (Berl 2008), This discovery has shown that individuals with CKD are at a greater risk of developing hyponatremia because of their limited ability to concentrate or dilute urine. Furthermore, nutrient restriction and multi-drug drugs are normal, all of which can contribute to sodium imbalances. Because of hypotonic fluid absorption or insufficient water, hyponatremia is more common in dialysis patients.

In either case, metabolic acidosis is linked to the development of kidney failure (Dober *et al.* 2015). Chronic kidney disease can be linked to sodium intake, according to a report (Levin *et al.* 2009). According to another study, People with chronic renal disease are more prone to suffer from hypertension because of their diseased kidneys' inability to

expel salt (Koomans *et al.* 1985). Those two studies agree with current results related to sodium level.

5.3 Potassium Concentration

Potassium is a cation that is needed for proper heart and muscle function. The kidneys are important for keeping potassium levels in check. A potassium levels that is too high (hyperkalemia) or too low (hypokalemia) can be dangerous and must be managed to return to the usual reference interval. Potassium levels in patients with progressive CKD may be elevated (Armbruster 2013). Potassium levels in the blood rise as a result of the renal failure, potassium accumulates in the blood leading to a reduction in kidney activity. The kidneys were unable to eliminate the excess potassium that the body needed, resulting in an excess of potassium levels (Yusuf *et al.* 2016).

The current findings revealed that the amount of potassium in the blood serum of the treatment group was slightly higher at the level of chance ($P \leq 0.05$) than that of the group control. This finding is in line with the findings of Al-Rubaei *et al.* (2010), who found that the potassium concentration (5.91.19 mmol/l) in the patients group was significantly higher than the potassium concentration (4.013.24 mmol/l) in the control group. This is attributable to the kidneys' important role in excreting approximately 90% of the potassium daily into the urine, so the chronic renal dialysis disease has a weakness in regulating this mechanism and thus disorder this process (Rastergar and Soleimani 2001).

Also, in patients with Uremia, an elevated amount of the hormone aldosterone, whose secretion increases due to a rise in the Renin enzyme, contributes to a decrease in potassium and an increase in sodium, but also to an increase in acidity that competes with the mechanism of potassium excretion out of the body, and a decrease in the level of sodium. One of the catalysts for an improvement in enzyme resonance is an excess of potassium (Ahmed and Weisberg 2001). When the glomerular filtration rate (GFR) exceeds 60 ml/min per 1.73 m^2, hyperkalemia is uncommon; however, it becomes more frequent as the GFR decreases. Pseudohyperkalemia, temporary changes in potassium caused by cell transfer, and drug-induced disruption of potassium excretion are the most

common causes of hyperkalemia in people with retained GFR (Foley *et al.* 2008, Drawz *et al.* 2012).

According to a new report, chronic kidney failure causes 95% of filtered sodium ions to be reabsorbed (Sterns *et al.* 2010). The kidneys may not be able to extract excess potassium from the blood in some forms of chronic renal disease (NKDEP 2011). In patients with chronic renal disease, the mechanism of renal potassium ion excretion is impaired. In either case, it can rise in tandem with degrading renal function. As a result, increased potassium ion levels may have induced potassium ion excretion (Hsieh *et al.* 2011). A more recent study uncovered a link between chronic renal illness and hyperkalemia, or high blood potassium levels. Studies have revealed that vascular smooth muscle cells in chronic renal disease patients exhibit less calcium receptors (Barle 1974).

As opposed to the health reference group, the current study results indicate a drop in sodium levels before dialysis. Previous research backs up these conclusions (Ravel *et al.* 2016, Luo *et al.* 2016). By contrast, the kidneys may generate angiotensin when the sodium level in the blood is low, which leads to aldosterone production from the adrenal cortex, which in turn encourages sodium reabsorption in the renal tubules from the glomerular filtrate. As renal failure progresses, the quantity of water in the blood rises, resulting in a decrease in blood flow. Increased salt levels make blood pressure more vulnerable, decreasing the amount of sodium that affects high blood pressure (Dekker *et al.* 2016).

5.4 Complete Blood Count (CBC)

The current study's findings revealed that the proportion of packed cell volume and haemoglobin levels in the treatment group is significantly lower than in the group control Anemia is demonstrated by low PCV and haemoglobin content in patients with chronic renal disease, and several studies have shown that anaemia is a complication of chronic kidney failure (Hsu *et al.* 2001, Costa *et al.* 2008). It is believed that kidney failure causes anaemia by decreasing the release of the hormone erythropoietin, which induces the formation of red blood cells in the bone marrow (Brunelli and Berns 2009).

The accumulation of nitrogenous waste products in the blood, which prevents the formation of erythrocyte cells in the bone marrow, is one of the causes of anaemia in people having chronic renal failure (Besarab *et al.* 1989). Another significant explanation is anaemia, which is caused by a lack of iron, folic acid, vitamins B6 and B12, several additional well-known causes of anemia Patients with chronic renal illness are prone to hunger, and in turn are more likely to suffer from anemia due to lack of appetite. As in those with persistent kidney failure, the potential for internal bleeding that isn't immediately apparent puts them at risk of becoming anemic (Bickford 2002).

In terms of MCV, the findings revealed that the MCV% of the patient group was non-significant change compared lower with group control. The cause is due to lack of appetite and anaemia, as well as the attending physician's dietary, which plays a major role in lowering MCV in the patients' population, and protein loss by urine, which also helps to lower MCV in patients.

6. CONCLUSIONS AND RECOMMENDATION

6.1 Conclusions

- Creatinine level in chronic kidney disease patients showed significant increase compare with healthy persons.
- Urea level in chronic kidney disease patients showed significant increase compare with healthy persons.
- Sodium level in chronic kidney disease patients showed significant decrease compare with healthy persons.
- Potassium level in chronic kidney disease patients showed significant increase compare with healthy persons.
- The present research findings revealed positive correlation between GFR and sodium.
- The present research findings revealed inverse correlation between GFR and potassium.

6.2 Recommendation

- Further studying electrolyte disorder in CKD patient.
- Monitoring electrolyte status in CKD and especially Hemodialysis.

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