Comparison of Some Blood indices among Chronic Renal Failure Patients Pre and Post Hemodialysis, Omer Alhag Saeed Center for Renal Disease and Surgery, Rufaa, Gezira State, Sudan (2017)

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Department of Hematology and Immunohaematology

Faculty of medical laboratory sciences

October 2017

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<tr>
<th>Name</th>
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</thead>
<tbody>
<tr>
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Date: 3 / 11 / 2017
Comparison of Some Blood indices among Chronic Renal Failure Patients Pre and Post Hemodialysis, *Omer Alhag Saeed*
Center for Renal Disease and Surgery, *Rufaa, Gezira State, Sudan (2017)*

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<table>
<thead>
<tr>
<th>Name</th>
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<th>Signature</th>
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</thead>
<tbody>
<tr>
<td>Dr. Albadawi Abdelbagi Talha</td>
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Declaration

I authorized that my dissertation **Comparison of Some Blood indices among Chronic Renal Failure Patients Pre and Post Hemodialysis, Omer Alhag Saeed Center for Renal Disease and Surgery, Rufaa, Gezira State, Sudan (2017)**, submitted by me, under the supervision of **Dr. Albadawi AbdElbagi Talha** and **Dr. Sanaa Elfatih Husieen** for the partial fulfillment for the award of Master degree in Medical Laboratory Sciences in Haematology and Immuno haematology. University of Gezira Faculty of Medical Laboratory Sciences Department of haematology and Immunohaematology; Wad-Medani, Sudan and this is original and it was not submitted in part or in full, in any printed or electronic means, and is not being considered elsewhere for publication.

Name and Signature of Candidate:

**Name:** Enas Awad Alameen Awadalah

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**Place:** Wad Medani

**Date:** 3/11/2017
Dedication

To my Darling husband and son,

To my mother and my father that never stop giving,

To my brother And all my friends my precious treasure
Acknowledgement

First, all praise is to Allah who has enabled me to complete this thesis in time. Next, I wish to express my sincere gratitude to both my supervisors, Dr. Albadawi Abdelbagi Talha (Principal supervisor), associate professor in haematology and Dr. Sanaa Elfatih Husieen (cosupervisor). I also wish to express special thanks to head of the laboratory and all staff of dialysis center for the facilitating the study of their patients. My darling thanks and gratitude for my husband Yasin Ahmed Mokhtar my gratitude is extended to my family my colleague and everyone who encouraged and supported me to perform work this.
Comparison of Some Blood indices among Chronic Renal Failure Patients Pre and Post Hemodialysis, Omer Alhag Saeed Center for Renal Disease and Surgery, Rufaa, Gezira State, Sudan (2017)

Enas Awad Alameen Awadalah

Abstract

Renal failure is a situation in which kidney fails to function adequately. There are two forms of renal failure: acute and chronic. End-Stage renal disease is the final stage of chronic renal failure where there is a progressive, irreversible deterioration in renal function which can be substituted by haemodialysis (HD), peritoneal dialysis, or transplantation. This cross sectional study was conducted in, Omer Alhag Saeed Center for Renal disease and Surgery Gezira State rufaa. To evaluate the effect of heamodialysis in complete blood count with focus on hemoglobin, haematocrit, red cell indices and platelets pre and post haemodialysis for patients of end stage renal disease. 40 patients were enrolled in this study, consisting of, 27 males represent (61%) and 23 females represent (39%). The mean age of male and female HD patients were 40±15 years and 35±12 years respectively, resulting in an overall mean age of 51 years for the population examined.

3 ml of blood were collected pre and post dialysis, in to tri-potassium Ethylene diamine tetra-acetic acid(K3 EDTA) anticoagulant containers about 10-15 minutes after the haemodialysis hemoglobin hematocrit, red cell indices was measured by using an automated blood cell counter (Sysmex KX 21N ).Data collected was analyzed by using Statistical packages for Social Science(SPSS). The result was statistically Significant difference in hemoglobin pre(10.2± 1.6) and post(10.9± 1.9),(P 0.000), hematocrit pre (32.4±5.1)and post(34.7±5.9)(P.value 0.00) , red cell indices MCV pre(90.7±6.0) and post(90.7±6.1), MCH pre (28.6 ± 2.6) post (28.6 ± 2.6) , MCHC pre (31.5 ± 1.2) post (31.4 ± 1.2) p.value (0.00) , platelet pre (205.9 ± 90.7) post (202.6 ± 84.8) p.value (0.00) .The results of the present study conclude that the most of the hematological parameters measured in HD patients, post-HD were significantly increased.
مقارنة بعض مؤشرات الدم بين مرضى الفشل الكلوي المزمن قبل وبعد غسيل الكلى، بمركز الحاج سعيد لأمراض وجراحة الكلى، رفاعة، ولاية الجزيرة، السودان (2017)

إيناس عوض الأمين عوض الله

ملخص الدراسة
الفشل الكلوي هو الوضع الذي فشل الكلى في العمل بشكل كاف. هناك نوعان من الفشل الكلوي: الحادة والمزمن. في نهاية المرحلة المرض الكلوي هو المرحلة النهائية من الفشل الكلوي المزمن حيث هناك تدهور تدريجي، لا رجعة فيه في وظيفة الكلى التي يمكن أن تكون الوظيفة البديلة لها هي غسيل الكلى، غسيل الكلي البرتيوني، أو زراعة الكلي. أجريت هذه الدراسة المقطعية في مركز عمر الحاج سعيد لمرض الكلى والجراحة ولاية الجزيرة رفاعة. لتقييم تأثير غسيل الكلى في عدد الدم الكامل مع التركيز على الهيموجلوبين، حجم الدم المضغوط، مؤشرات الخلية الحمراء والصفائح الدموية قبل وبعد غسيل الكلى للمرضى من المرحلة النهائية مرض الكلى. وقد تم تسجيل أربعين مريض في هذه الدراسة، ويتلفت من 27 ذكر تتمثل (61%) و 23 أنثى تتمثل (39%). وكان متوسط عمر المريض الذكور والإناث 40 ± 15 سنة و 35 ± 12 سنة على التوالي، مما أدى إلى متوسط العمر الإجمالي 51 سنة. جمعت 3 مل من الدم قبل وبعد الغسيل، في ثلاثي البوتاسيوم الإيثيلين ديمين رباعي حمض الخليك حاويات مضادة للخثر بعد حوالي 10-15 دقيقة من غسيل الكلى، ثم قبض مؤشرات الخلية الحمراء باستخدام عدد خلايا الدم الألي (سيسمكس N21 KX). حلت البيانات التي جمعت باستخدام الجرم الإحصائية للعلوم الاجتماعية. ووجدت النتيجة أن هناك فروق ذات دلالة إحصائية في الهيموجلوبين قبل (10.2 ± 1.9) و بعد (9.0 ± 1.7) متوسط حجم الخلية قبل (6.1 ± 0.7) و بعد (6.0 ± 0.8) متوسط كمية الهيموجلوبين في الخلية الواحدة قبل (28.6±2.3) و بعد (28.0±2.2) عدد الصفائح الدموية قبل (205.9±20.5) و بعد (202.6±20.8) ومتوسط كمية الهيموجلوبين في كل الخلايا قبل (31.5±1.2) و بعد (31.4±1.4). وتخلص هذه الدراسة إلى أن معظم المعلومات الدموية المقاسة في مرضى الغسيل الدموي الكلوي، قبل أو بعد زادت بشكل ملحوظ.
## List of content:

<table>
<thead>
<tr>
<th>Number</th>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supervision committee</td>
<td>I</td>
</tr>
<tr>
<td></td>
<td>Examining committee</td>
<td>II</td>
</tr>
<tr>
<td></td>
<td>Declaration</td>
<td>III</td>
</tr>
<tr>
<td></td>
<td>Dedication</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Acknowledgement</td>
<td>V</td>
</tr>
<tr>
<td></td>
<td>English Abstract</td>
<td>VI</td>
</tr>
<tr>
<td></td>
<td>Arabic Abstract</td>
<td>VI</td>
</tr>
<tr>
<td></td>
<td>List of content</td>
<td>VII</td>
</tr>
<tr>
<td></td>
<td>List of tables</td>
<td>VIII</td>
</tr>
<tr>
<td></td>
<td>List of figures</td>
<td>IX</td>
</tr>
<tr>
<td></td>
<td>List of abbreviations</td>
<td>X</td>
</tr>
</tbody>
</table>

## CHAPTER ONE

1.1 Introduction 1
1.2 Rational 2
1.3 Objectives 2
1.3.1 General objective 2
1.3.2 Specific objective: 2

## CHAPTER TWO (LITERATURE REVIEW)

2.1 Types of renal failure 3
2.1.1 Acute renal failure 3
2.1.2 Chronic kidney disease 3
2.2 A etiology of CKD 3
2.3 Diagnosis of CKD 4
2.4 Risk factor of CKD 4
2.5 Stage of CKD 4
2.5.1 End-stage renal disease 4
2.5.2 Management of ESRD 5
2.5.2.1 Peritoneal dialysis 5
2.5.2.2 Haemodialysis 5
2.5.2.3 Renal transplantation 6
2.6 Haematological changes in haemodialysis patients 7
2.6.1 Blood cells changes in haemodialysis patients 7
2.6.1.1 Erythrocytes 7
2.6.1.2 Thrombocytes 8
<table>
<thead>
<tr>
<th>Section</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1</td>
<td>Materials</td>
<td>9</td>
</tr>
<tr>
<td>3.1.1</td>
<td>Study design</td>
<td>9</td>
</tr>
<tr>
<td>3.1.2</td>
<td>Study area</td>
<td>9</td>
</tr>
<tr>
<td>3.1.3</td>
<td>Study population</td>
<td>9</td>
</tr>
<tr>
<td>3.1.4</td>
<td>sample size</td>
<td>9</td>
</tr>
<tr>
<td>3.1.5</td>
<td>Data collection</td>
<td>9</td>
</tr>
<tr>
<td>3.1.6</td>
<td>Sampling</td>
<td>9</td>
</tr>
<tr>
<td>3.1.7</td>
<td>Exclusion criteria</td>
<td>9</td>
</tr>
<tr>
<td>3.1.8</td>
<td>Inclusion criteria</td>
<td>9</td>
</tr>
<tr>
<td>3.1.9</td>
<td>Study period</td>
<td>9</td>
</tr>
<tr>
<td>3.1.10</td>
<td>Ethical consideration</td>
<td>10</td>
</tr>
<tr>
<td>3.1.11</td>
<td>Data presentation</td>
<td>10</td>
</tr>
<tr>
<td>3.2</td>
<td>Methodology</td>
<td>10</td>
</tr>
<tr>
<td>3.2.1</td>
<td>Requirement</td>
<td>10</td>
</tr>
<tr>
<td>3.2.2</td>
<td>Procedure</td>
<td>10</td>
</tr>
<tr>
<td>3.2.3</td>
<td>Test performed</td>
<td>11</td>
</tr>
<tr>
<td>3.2.4</td>
<td>Principles of instrument (Sysmex)</td>
<td>11</td>
</tr>
<tr>
<td>4.1</td>
<td>Result</td>
<td>12</td>
</tr>
<tr>
<td>4.2</td>
<td>Discussion</td>
<td>20</td>
</tr>
<tr>
<td>5.1</td>
<td>Conclusion</td>
<td>21</td>
</tr>
<tr>
<td>5.2</td>
<td>Recommendation</td>
<td>21</td>
</tr>
</tbody>
</table>

**References**

10
## List of Tables:

<table>
<thead>
<tr>
<th>Table</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table4.1</td>
<td>Differences between mean of Hb in renal failure patients pre and post HD.</td>
<td>13</td>
</tr>
<tr>
<td>Table4.2</td>
<td>Differences between mean of PCV in renal failure patients pre and post HD.</td>
<td>14</td>
</tr>
<tr>
<td>Table4.3</td>
<td>Differences between mean of MCV in renal failure patients pre and post HD.</td>
<td>15</td>
</tr>
<tr>
<td>Table4.4</td>
<td>Differences between mean of MCH in renal failure patients pre and post HD.</td>
<td>16</td>
</tr>
<tr>
<td>Table4.5</td>
<td>Differences between mean of MCHC in renal failure patients pre and post HD.</td>
<td>17</td>
</tr>
<tr>
<td>Table4.6</td>
<td>Differences between mean of PLT in renal failure patients pre and post HD.</td>
<td>18</td>
</tr>
<tr>
<td>Table4.7</td>
<td>Differences between mean of RBCS in renal failure patients pre and post HD.</td>
<td>19</td>
</tr>
</tbody>
</table>
List of Figures:

<table>
<thead>
<tr>
<th>Figure</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 4. 1</td>
<td>Mean of Hb in renal failure patients pre and post HD</td>
<td>13</td>
</tr>
<tr>
<td>Figure 4. 2</td>
<td>Mean of PCV in renal failure patients pre and post HD</td>
<td>14</td>
</tr>
<tr>
<td>Figure 4. 3</td>
<td>Mean of MCV in renal failure patients pre and post HD</td>
<td>15</td>
</tr>
<tr>
<td>Figure 4. 4</td>
<td>Mean of MCH in renal failure patients pre and post HD</td>
<td>16</td>
</tr>
<tr>
<td>Figure 4. 5</td>
<td>Mean of MCHC in renal failure patients pre and post HD</td>
<td>17</td>
</tr>
<tr>
<td>Figure 4. 6</td>
<td>Mean of PLTS in renal failure patients pre and post HD</td>
<td>18</td>
</tr>
<tr>
<td>Figure 4. 7</td>
<td>Mean of RBCS in renal failure patients pre and post HD</td>
<td>19</td>
</tr>
</tbody>
</table>
List of Abbreviations:

ARF : Acute renal failure
B.M : Bone marrow
CBC : Complete blood cell count
CKD: Chronic kidney disease
CRF : Chronic renal failure
EDTA: Ethylene diamine tetra acetic acid
EPO : Erythropoietin
ESRD : End-Stage Renal Disease
ESRF: End-Stage Renal failure
GFR: Glomerular filtration rate
Hb : Haemoglobin
Hct : Haematocrit
HD : Haemodialysis
HSCs: Hematopoietic stem cells
MCHC : Mean corpuscular hemoglobin concentration
MCV : Mean corpuscular volume
PD : Peritoneal dialysis
RBCs: Red blood cells
RRT: Renal replacement therapy
CHAPTER ONE
INTRODUCTION

1.1 Introduction

The kidneys are essential organs responsible for a multitude of bodily functions. The principle role of the kidney are the excretion of the waste products, including the nitrogenous wastes urea and uric acid; the regulation of the volume and composition of the body fluid; the production of the hormones-erythropoietin (EPO),rennin, and prostaglandins; the metabolism of vitamin D, and the small molecular weight proteins. Failure of the renal excretory function results in renal failure. When the kidneys are damage to such an extent that they can no longer maintain body fluid homeostasis, renal disease is described as having progressed to acute or chronic renal failure. Renal failure is defined as a deterioration of renal function that results in the retention of nitrogenous waste products in the blood and the body (azotemia) and decrease in the Glomerular filtration rate (GFR). Chronic anemia is the most profound hematologic alteration that accompanies renal failure. Anemia first appears when the GFR falls below 40 mL/minute and is present in most persons with ESRD. Several factors contribute to anemia in persons with chronic renal failure, including an erythropoietin deficiency, uremic toxins, and iron deficiency. The kidneys are the primary site for the production of the hormone erythropoietin, which controls red blood cell production. The accumulation of uremic toxins further suppresses red cell production in the bone marrow, and the cells that are produced have a shortened life span. Iron is essential for erythropoiesis. Many persons receiving maintenance hemodialysis also are iron deficient because of blood sampling and accidental loss of blood during dialysis. Other causes of iron deficiency include factors such as anorexia and dietary restrictions that limit intake. When untreated, anemia causes or contributes to weakness, fatigue, depression, insomnia, and decreased cognitive function. There is also increasing concern regarding the physiologic effects of anemia on cardiovascular function. The anemia of renal failure produces a decrease in blood viscosity and a compensatory increase in heart rate. The decreased blood viscosity also exacerbates peripheral vasodilatation and contributes to decreased vascular resistance. Most of hematological change appear in patient with renal failure under hemodialysis pre and post (Alghaythan, 2011).
1.2 Rational
Renal failure is one of the more common medical conditions affecting Sudanese people, associated with significant morbidity in chronic kidney disease (CKD). Also has major effects in blood components leading to different diseases. Consequently, patients should be carefully evaluated for the profile of anemia by measuring it.

1.3 Objectives:

1.3.1 General Objective:
To estimate full blood count pre and post dialysis in chronic renal failure.

1.3.2 Specific objective:
1- To estimate hemoglobin pre and post dialysis among chronic renal failure.
2- To estimate hematocrit, red cell indices, and platelet pre and post dialysis among chronic renal failure.
CHAPTER TWO
LITERATURE REVIEW

2.1 Types of renal failure

2.1.1 Acute Renal Failure

Acute Renal Failure (ARF) is a complex disorder that occurs in a variety of settings with clinical manifestations ranging from a minimal elevation in serum creatinine to an uric renal failure. ARF occurs suddenly and is usually initiated by underlying causes, for example dehydration, infection, serious injury to the kidney or the chronic use of over the counter pain medications like Tylenol (acetaminophen) or Advil (ibuprofen). ARF is often reversible with no lasting damage. The causes of ARF are classically divided into three categories: pre renal, post renal (or obstructive), and intrinsic (Jover et al., 2008).

2.1.2 Chronic kidney disease

Chronic kidney disease (CKD) is a critical and rapidly growing global health problem. CKD encompasses a spectrum of disease, ranging from mild renal damage, which can be asymptomatic and is only detected by blood and urine testing, through to end-stage disease, in which renal function is impaired to such an extent that the retention of metabolic waste products, salt and water becomes potentially fatal. CKD is characterized by progressive destruction of renal mass with irreversible sclerosis and loss of nephrons over a period of months to years, depending on the underlying etiology. It is defined as either renal damage or decreased renal function (decreased GFR) for three or more months (Gooneratne et al., 2008). CKD is characterized by an exceptionally high mortality rate, primarily due to Cardiovascular disease. CKD is usually silent until its late stages, and without aggressive screening, detection detection may not occur until immediately before symptomatic kidney failure develops (ST Peter, 2007).

2.2 A etiology of CKD

The most frequent causes of chronic renal failure (CRF) are diabetes mellitus (DM), present in 40-60% of all patients with CRF that progress to End-Stage Renal failure (ESRF); arterial hypertension, which affects 15-30%; and glomerulonephritis, which is seen in less than 10% of all cases. Only 2-3% of all CRF patients present renal polycystosis (Jover et al., 2008). In a recent prospective study of the patients admitted to dialysis in Najran, it was found that the mean age of the patients was 55 years and the main causes of CRF included Diabetic nephropathy, hypertension, unknown and obstructive uropathy as 28%, 24%, 23% and 8% respectively (Shaheen and Al-Khader, 2005).
2.3 Diagnosis of CKD
Chronic renal failure is the best parameter for assessing renal function. The standard for measuring GFR is inulin clearance, though in practice this is not the most widely used method. Creatinine clearance overestimates GFR, and precise collection of the 24-hour urine samples needed to calculate such clearance is not easy. As a result, in recent years effort has centered on the development of formulas (Modification of Diet in Renal Disease) capable of determining GFR as exactly as possible, based on a simple plasma sample used to determine serum creatinine concentration. These formulas take into account patient age (GFR undergoes a physiological decline with age), sex (120 ml/ min/1.73 m2 is considered normal in males, versus 100 ml/ min/1.73 m2 in females) and race. When evaluating the risk of progression of CRF and of the possible development of ESRF, it is necessary to detect and quantify proteinuria. In addition, in the early stages of CRF, normal or only slightly reduced GFR values may be observed despite an already manifest increase in protein excretion in urine. The latter is therefore more useful as an early marker of CRF (Jover et al., 2008).

2.4 Risk factor of CKD
Patients at higher risk for CKD include patients with diabetes, hypertension, or a family history of hypertension, diabetes, or CKD (Alghaythan, 2011).

2.5 Stage of CKD
Recent professional guidelines classify the severity of CKD in five stages, with stage 1 being the mildest and usually causing few symptoms and stage 5 being a severe illness with poor life expectancy if untreated. CRF requiring dialysis or transplantation is known as End-Stage Renal Disease (ESRD) (ST Peter, 2007).

2.5.1 End-Stage Renal Disease
End-Stage Renal Disease (ESRD) is the final stage of CRF where there is progressive, irreversible deterioration in renal function in which the body’s ability to maintain fluid and electrolyte balance fails, resulting in uremia. ESRD is characterized by a decrease in GFR and histologic evidence of less than 10% nephron function remaining (Michael and Gabriel, 2004). The ESRD patients require a regular course of dialysis or kidney transplantation to maintain life. Although dialysis is lifesaving and prolongs survival, it is only temporary and does not replace all of the renal functions. Allogeneic renal transplantation is the only current means to restore the whole renal functions, but its application is severely limited by donor shortage and immune-related problems. The National Kidney Foundation rated ESRD as the last and the fifth stage among CKD, which is based on the presence of kidney damage and level of kidney function whereas GFR <15 ml/min/1.75m² for ≥3
months. CRF occurs when the kidneys are operating at less than 50% of normal capacity. ESRD occurs when the kidneys are working at less than 10%–15% of normal capacity. Diabetic nephropathy is rapidly becoming a main cause of ESRD requiring dialysis (Shaheen and Al-Khader, 2005).

### 2.5.2 Management of ESRD

The treatment of ESRD is composed of conservative management and RRT. RRT is considered when conservative management fails to be effective against the progression of renal deterioration. RRT for ESRD patients include haemodialysis (HD), peritoneal dialysis (PD) and renal transplantation. The aim of RRT is to prolong the quantity of life without diminishing the quality of remaining years (Alghaythan, 2011).

#### 2.5.2.1 Peritoneal Dialysis

In Peritoneal dialysis, access to the body is gained through a catheter placed in the abdominal wall and inserted in the peritoneum. The dialysate (sterile electrolyte solution) is introduced through the catheter, and the peritoneal membrane filters the blood waste products via an osmotic mechanism. PD are efficacious in removing solutes and water depending on the clearance characteristic of the dialysis membrane (artificial or peritoneum, respectively) and the time allowed for exchange across the membrane. common side effect of PD is bacterial infection (peritonitis) can occur if the dialysis equipment is not clean (Alghaythan, 2011).

#### 2.5.2.2 Haemodialysis

Haemodialysis is a process of solute clearance based on diffusion across the membrane driven by a concentration gradient between the blood and dialysate. The total amount of solute transported per unit of time (clearance), depends on the molecular weight of the molecule, membrane characteristics (dialysance), dialysate flow, and blood flow. The goals of HD are to treat uremic symptoms (through removal of toxic metabolites), correct acid-base and electrolyte disturbances, maintain volume status, and, over the long term, improve quality of life, lower morbidity and mortality rates, and maintain nutritional stability. To reach these goals, patients with GFR of less than 10 to 15 mL/min undergo thrice-weekly HD (Jover et al., 2008). Credit for the first human dialysis treatment goes to Georg Haas, who in 1924 provided dialysis to a patient with ARF. This procedure was subsequently refined by Willem Kolff in the 1930s and 1940s and was used sporadically for patients with ARF. Problems with early dialysis therapy included clotting of blood in the extracorporeal circuit and difficulty in obtaining reliable access to the circulation. The availability of purified heparin in 1943 and the development of a Teflon arterio venous shunt by Belding Scribner and colleagues in 1960 were landmarks that led to the general applicability of HD.
to patients with CRF (Rosner, 2005). HD is performed three times a week for 3–4 h each session, but dialysis time for these sessions varies from patient to patient (Kusiak et al., 2005). In HD, blood filtration is carried out by a machine (dialyzer) equipped with a semi permeable membrane allowing passage of the excess fluids and waste products. HD requires establishing access to the vasculature through an arterio venous fistula, graft, or catheter, whereas PD requires establishing access to the peritoneal cavity using a peritoneal catheter. During HD, the patients receive anticoagulation, generally in the form of heparin, to facilitate blood cycling through the dialyzer, and for ensuring permeability of the vascular access (Jover et al., 2008). Dialysis involves the movement of solutes and solvent (water) across a semi permeable membrane (the dialyzer). Clinically, this occurs when blood is exposed to an extra corporeal semi permeable membrane, on the other side of which a solution of dialysate is flowing. Three major mechanisms govern the movement of molecules in this procedure: diffusion, ultra filtration, and convection. During a dialysis treatment, patients can be exposed to as much as 100 L of dialysate (Rosner, 2005). During HD, the blood passes through an extra-corporal circuit where metabolites are eliminated, the acid–base equilibrium is re-established and excess salt and water is removed. The process of diffusion exchanges solutes and metabolites across a semi-permeable membrane, separating the blood and dialysate. Water is removed from the body using a negative pressure gradient in a process called ultra called ultra-filtration. After transit through the dialyzer, the clean, filtered blood is returned to the body. A device called a hem dialyzer regulates the entire procedure (Kusiak et al., 2005). The side effect of HD is low blood pressure which caused by the drop in fluid level during dialysis. Low blood pressure can cause nausea and dizziness. Also, people receiving HD are increased risk of developing sepsis (blood poisoning) and high blood potassium level (Rosner, 2005).

2.5.2.3 Renal transplantation

The ideal solution for ESRD is transplantation but, as demand for donor organs, dialysis remains the only satisfactory alternative. Renal transplantation is the treatment of choice in patients with irreversible renal failure. Immediately before transplantation, and after the surgical operation, immunosuppressive therapy must be provided to avoid acute rejection. All transplant patients, with the exception of those receiving an organ from an identical twin, require life-long immunosuppressive therapy (Jover, et al. 2008).
2.6 Hematological changes in haemodialysis patients

The haematological changes in patients undergoing HD has been widely discussed in the literature. Some early reports indicate that any haematological changes in HD patients were insignificant, while most of the recent reports point to definite and significant haematological changes in these patients (Alghaythan, 2011)

2.6.1 Blood cells changes in haemodialysis patients

2.6.1.1 Erythrocytes

There are wide disagreements on the effect of HD on the RBCs, Hb, Hct, RDW and RBCs indices in patients with ESRD. Some reports found no change, others reported an increase while others reported decrease. An early research by Jackson et al., (1995), commented on the low-grade hemolytic anemia found in many patients undergoing HD. They explained that „non biocompatible” dialysis membranes may activate PMNs, leading to radicals production. After HD, the RBCs show increased membrane lipid peroxidation, reduced membrane fluidity, and increased osmotic fragility. This is likely to contribute to the low-grade hemolytic anemia reported in HD patients. Supplementation with vitamin E has been shown to prevent these changes and may therefore be a promising antioxidant in these patients. (Vickers et al., 1998), compared the RBCs count at 2, 15, 30 and 180 min after commencing dialysis and just before starting treatment. They did not find any change in the number of circulating RBCs when compared to pre-dialysis samples. This result was confirmed by (Malyszko et al., 1999), who studied the pre-and post-dialytic changes of the Hb and RBCs count in 53 ESRD patients under conventional HD. They also found that both these parameters did not differ significantly pre-and post-HD. Butt et al., (1999), did an extended study on the pre-and post-dialytic changes of the reticulocytes count, RBCs count, Hb and Hct levels in 60 ESRD patients under conventional HD. They reported that the mean of Hb, Hct levels and RBCs count were slightly increased ;while, they found no changes in mean reticulocytes count after HD (Malyszko, et al. 2001).
2.6.1.2 Thrombocytes

Studies of the effect of HD on thrombocytes also reveal contradictory results. Some researchers have reported that HD does not have any effect on thrombocytes while other researchers have described either increase or decrease in thrombocytes in HD patients after HD sessions (Tassies et al., 1995), studied the effect of HD on reticulated platelets during HD. They reported that HD decreased the percentage of RNA-rich platelets through elimination of the younger and more active platelets and worsened the thrombopathy present in uremic patients. Later on, another group of researcher (Sloand JA and Sloand EM, 1997) evaluated the platelets counts and mean platelets volume in twenty chronic HD patients. They found that there was no significant changes in platelets counts or mean platelets volume between pre-and post-dialysis samples. Vickers et al., (1998), assessed the platelets count at 2, 15, 30 and 180 min after commencing dialysis and after commencing dialysis and just before starting treatment. They also concluded that in comparison to the pre-dialysis samples, there was a small decrease in circulating platelets, at 15 and 30 min. However even this small decrease reverted to basal values by the end of the 3-hour period. Neuhaus et al., (2000), reported that two pediatric patients with ESRF develop heparin-induced cytopenia type II (HIT II) on HD. Both developed acute respiratory distress and chest pain within 30 min of initiating the 5th HD session. Marked clots were observed in the dialyzers. Substitution of heparin with the low molecular weight heparin dalteparin had no effect. Switching from anticoagulation to the heparinoid danaparoid resulted in immediate disappearance of all adverse effects, and further long-term HD was uneventful. They concluded that HIT II may occur in children on HD. HIT II is essentially a clinical diagnosis, as specific laboratory assays may be negative. Regular measurement of platelets during the 1st month after initiation of HD and prompt recognition of unusual clinical symptoms, i.e., respiratory distress and chest pain, will facilitate the diagnosis of HIT II. Once HIT II is suspected, heparin and low-molecular-weight heparins should be stopped immediately, and can successfully be substituted by the heparinoid danaparoid (Erdem, et al. 1996).
CHAPTER THREE
MATERIALS AND METHOD

3.1 Materials:

3.1.1 Study design:
Cross sectional laboratory based study was conducted to estimate Hemoglobin, hematocrit, red cells indices and platelet among hemodialysis patients.

3.1.2 Study area:
Study was conduct in Omer ALhage Saeed for renal disease and surgery Rufaa center of dialysis (Sudan).

3.1.3 Study population:
Hemodialysis Patients with chronic renal failure

3.1.4 Sample size:
Forty patients were included in this study

3.1.5 Data collection:
Questionnaire specifically designed to obtain information which helps in either including or excluding certain individuals in or from study.

3.1.6 sampling:
Blood sample obtained by using local anti septic for skin (70% ethanol) 3ml of venous blood collected from cases pre and post dialysis using a disposable sterile plastic syringe. The Blood draw in K3, EDTA container.
1. Patients providing informed consent.
2. Patients with documented chronic kidney disease treated under dialysis.

3.1.7 Exclusion criteria
1. Acute or chronic inflammatory disease.
2. Malignancy or known hematological disorder.
3. Recent severe hemorrhagic episode.

3.1.8 Study period:
From March to June 2017.
3.1.9 Ethical consideration:
informed consent was taken. Patients and treating doctor were informed by the results. Personnel identification data was kept secure.

3.1.10 Data presentation:
The results were presented as mean ± standard deviation. The Statistical analysis was performed using Statistical Package for Social Science (SPSS11.5). was performed using student t test to determine the effect of dialysis on Hb, PCV, RBCs indices and platelet count. Data presented in form of tables P.value was considered statistically significant.

3.2 Methodology:
3.2.1 Requirement:
- EDTA container.
- Syringe.
- Cotton.
- 70% Alcohol.
- A tourniquet

3.2.2 Procedure:
1- Participant was set up at right position for the collection.
2- The skin was cleaned by 70% alcohol and allowed to dry, to avoid stinging when the skin is penetrated.
3- A tourniquet was applied to the arm, tight sufficiently to distend the vein, but not so tightly to cause discomfort.
4- The needle was inserted, tourniquet removed and 3ml of the blood sample were collected in container with EDTA anticoagulant and mix gently.
5- The sample in EDTA bottle was places in the spiral mixer and allowed to mix very well. Whole blood mode was activated in the LCD screen, the sample number (code) was input via the key board and then the enter key.
6- Then, the cap was removed and inserted into the probe, on that condition, the start switch was pressed. The LCD screen displays analyzing, the sample was removed and recapped. The unit executes automatic analysis and displays the result on the screen.
3.2.3 Test performed:
Complete blood count CBC was done using Sysmex Automated Hematology Analyzer KX 21N series SN B 2010

3.2.4 Principles of instrument (Sysmex):

Detection Principle:
This instrument performs blood cell count by DC detection method.

Direct Current Detection Method:
Blood sample is aspirated, measured to a predetermined volume, diluted at the specified ratio, and then fed into each transducer. The transducer chamber has a minute hole called the aperture. On both side of the aperture, there are the electrodes between which flows direct current. Blood cells suspended in the diluted sample pass through the aperture, causing direct current resistance to change between the electrodes. As direct current resistance changes, the blood cell size is detected as electric pulses. Blood cell count is calculated by counting the pulses, and a histogram of blood cell sizes is plotted by determining the pulse sizes. Also, analyzing a histogram makes it possible to obtain various analysis data.
4.1 Results:
The results of this study are presented in four major sections. The first section includes the subject
characteristics (age, sex,), and the second section deals with the results of the measured red cells
parameters. One hundred adult patients, 27 males (61%) and 23 females (39%) in ESRF requiring
maintenance HD were recruited in our study. The mean age of male and female HD patients were
40±15 years and 35±12 years respectively, resulting in an overall mean age of 51 years for the
population examined. The differences between the mean of RBCs, Hb, Hct, and RBCs indices in
renal failure patients before and after HD sessions were calculated . All the measured indices
showed a significant increase statistically in values post- HD (P< 0.05).
The RBCs count increased post-HD (3.8 ± 0.6) when compared to the pre-HD count (3.5 ± 0.6)( P<
0.00); the Hb concentration increased post-HD (10.9 ± 1.9) when compared to that of the pre-HD
Hb concentration (10.2 ± 1.6)( P< 0.00); the Hct levels increased post HD (34.7 ± 5.9) when
compared to the pre-HD levels (32.4 ± 5.1)( P< 0.00); the MCV levels increased in post-HD (90.7±
6.1) when compared to the pre-HD levels (90.7 ± 6.0)( P< 0.00); the MCH levels no change post-
HD (28.6 ± 2.6) when compared to the pre-HD levels (28.6 ± 2.6)( P< 0.00); the MCHC levels
increased post HD (31.4 ± 1.2) when compared to the pre levels(31.4 ± 1.35)( P<0.00); The
platelets count showed statistical significant decrease post -HD (202.6 ± 84.8) when compared to
that of the pre-HD (205.9 ± 90.7)( P< 0.00).
Table 4.1 Differences between mean of Hb in renal failure patients pre and post hemodialysis

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb Pre - dialysis</td>
<td>10.2 ± 1.6</td>
<td>0.000</td>
</tr>
<tr>
<td>Hb Post – dialysis</td>
<td>10.9 ± 1.9</td>
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</tbody>
</table>

Figure 4.1 Mean of Hb in renal failure patients pre and post hemodialysis
Table 4.2 Differences between mean of PCV in renal failure patients pre and post hemodialysis.

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
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<tbody>
<tr>
<td>PCV Pre - dialysis</td>
<td>32.4 ± 5.1</td>
<td>0.000</td>
</tr>
<tr>
<td>PCV Post – dialysis</td>
<td>34.7 ± 5.9</td>
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</tbody>
</table>

Figure 4.2 Mean of PCV in renal failure patients pre and post hemodialysis
Table 4.3 Differences between mean of MCV in renal failure patients pre and post hemodialysis.

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCV Pre - dialysis</td>
<td>90.7 ± 6.1</td>
<td>0.000</td>
</tr>
<tr>
<td>MCV Post – dialysis</td>
<td>90.7 ± 6.1</td>
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</table>

Figure 4.3 Mean of MCV in renal failure patients pre and post hemodialysis
Table 4.4 Differences between mean of MCH in renal failure patients pre and post hemodialysis.

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
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<tbody>
<tr>
<td>MCH Pre - dialysis</td>
<td>28.6 ± 2.6</td>
<td>0.000</td>
</tr>
<tr>
<td>MCH Post – dialysis</td>
<td>28.6 ± 2.6</td>
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Figure 4.4 Mean of MCH in renal failure patients pre and post hemodialysis
### Table 4.5 Differences between mean of MCHC in renal failure patients pre and post hemodialysis

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCHC Pre – dialysis</td>
<td>31.5 ± 1.2</td>
<td></td>
</tr>
<tr>
<td>MCHC Post – dialysis</td>
<td>31.4 ± 1.2</td>
<td>0.000</td>
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![Figure 4.5](image)  

**Figure 4.5** Mean of MCHC in renal failure patients pre and post hemodialysis.
Table 4.6: Differences between mean of PLT in renal failure patient pre and post hemodialysis.

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
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<tbody>
<tr>
<td>PLT Pre – dialysis</td>
<td>205.9 ± 90.7</td>
<td></td>
</tr>
<tr>
<td>PLT Post – dialysis</td>
<td>202.6 ± 84.8</td>
<td>0.000</td>
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Figure 4.6: Mean of PLTs in renal failure patients pre and post hemodialysis.
Table 4.7 Differences between mean of RBCS in renal failure patient pre and post hemodialysis.

<table>
<thead>
<tr>
<th></th>
<th>Means ± SD</th>
<th>P. Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RBCs Pre – dialysis</td>
<td>3.5 ± 0.6</td>
<td>0.000</td>
</tr>
<tr>
<td>RBCs Post – dialysis</td>
<td>3.8 ± 0.9</td>
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</table>

Figure 4.7 Mean of RBCS in renal failure patients pre and post hemodialysis
4.2 Discussion:

Chronic renal failure is a major health problem and it greatly affects the economic and social status of affected patients. Dialysis treatment remains the principal method of treatment for correcting the renal dysfunction. HD increases longevity of patients with ESRD by removing the metabolic end products and excess of water (Costa et al., 2008). The results of this present study show that the patients with ESRD on regular HD display degrees of changes of various hematological parameters. The mean of each of RBCs count, Hb, Hct, and MCHc levels as measured in this study show a statistically significant increase in renal failure patients ’post- HD when compared to pre-HD levels. The increase of each RBCs count, Hb, Hct levels post-HD were explained by the fact that before HD, patients are usually hyper volemic and the values of each RBCs count, Hb, Hct levels are also lower. As ultra filtration takes place, RBCs count, Hb, and Hct values proportionally increase (Rangel et al., 2010). Our results agree with the findings of (Costa et al., 2008) found the similar results. (Pereira et al., 2010) reported similar observations though they did not find any significant change in the MCH values. They also reported significant decrease in the MCV value. The authors hypothesized that the increase in MCHC and decrease in MCV could be related to the erythrocyte membrane protein loss during the HD procedure. Recent studies (Jaroszyński et al., 2006; Rangel et al., 2010; Geller et al., 2010) have also reported that the mean of each Hb and Hct values show a statistically significant increase in renal failure patients post-HD when compared to pre-HD results. Thrombocytes are a nucleate fragments of megakaryocytes that are involved in the formation of blood clots (Sunitha and Munirathnam, 2008). The mean platelets counts in the current study showed a significance decrease in patients ’post-HD when compared to pre-HD counts. This finding is in agreement with (Yeniçoğlu et al; 2000), who reported a significant decrease in circulating platelets post-HD when compared to the pre-HD counts. The decrease in platelets counts post-HD may be due to either the HD procedure itself, through the interaction of blood with membranes that may activate complement (Galbusera et al.; 2009) or to the heparin used during dialysis was one of the factors accounting for the increased platelet aggregation after dialysis as previously reported by (Charvát et al., 1986). In a previous study (Docci et al, 1984) stated that the dialysis membrane composition is a major factor influencing hemodialysis-associated platelet loss. In contrast to our results, (Mohamed et al., 2008) found that there were no statistically significant in platelet count.
CHAPTER FIVE
CONCLUSION AND RECOMMENDATION

5.1 Conclusion
The results of this study indicate that most of the hematological parameters measured in HD patients, pre-or post-HD were noticed that there were changes in these parameters between the post-HD and pre-HD values.

- Hemoglobin level were increased post dialysis
- Hematocrit were increased post dialysis
- Platelets count were increased post dialysis

5.2 Recommendation

- Monitoring of the HD by hematological parameters.
- Help in preventing possible complications.
- Reducing the mortality/morbidity rate.
- Research studies are necessary to follow up patients through at least three HD sessions while continuously measuring their hematological parameters.
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