Estimation of Urea and Creatinine Pre and Post Dialysis in Patients with Chronic Renal Failure, Gezira Hospital for Renal Disease and Surgery, Gezira State, Sudan

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(B.Sc. in Clinical Chemistry, Omdurman Islamic University 2009)

A Dissertation

Submitted to University of Gezira in Partial Fulfillment of the Requirements for the Award of the Degree of Master of Science in Clinical Chemistry

Department of Clinical Chemistry

Faculty of Medical Laboratory Sciences

University of Gezira

May 2015
Estimation of Urea and Creatinine Pre and Post Dialysis in Patients with Chronic Renal Failure, Gezira Hospital for Renal Disease and Surgery, Gezira State, Sudan

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<table>
<thead>
<tr>
<th>Name</th>
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<th>Signature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usz: Abubakr Hassan Atta Almawla</td>
<td>Main supervisor</td>
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</tbody>
</table>

Date: 24/5/ 2015
Estimation of Urea and Creatinine Pre and Post Dialysis in Patients with Chronic Renal Failure, Gezira Hospital for Renal Disease and Surgery, Gezira State, Sudan

AzzamMirghaniElsaiedAlmahy

Examination Committee:

<table>
<thead>
<tr>
<th>Name</th>
<th>Position</th>
<th>Signature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dr. Abubakr Hassan Atta Almawla</td>
<td>Chairman</td>
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<tr>
<td>Dr. Omer Balla Ibrahim Abd Elgadir</td>
<td>External examiner</td>
<td>...........</td>
</tr>
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<td>Dr. Ameer Mohammed Dafalla</td>
<td>Internal examiner</td>
<td>...........</td>
</tr>
</tbody>
</table>

Date: 24/5/ 2015
**Dedication**

*My parents:*

Thank you for your unconditional support with my studies. I’m honored to have you as my parents.

Thank you for allowing me a chance to prove and improve myself all my walks of life.

I love you

*My family:*

Thank you for believing in me, for allowing me to further my studies.

To my friends and all people support me.

And of course, all my colleagues in master program Batch (1).

If I forgot anyone, please don’t ever doubt my dedication and love for ALL
The great thanks for god first.

I extend my sincere thanks and gratitude to all of helped me in completing this dissertation and provided me with information and special thanks and appreciation to my supervisors Dr: Abubakr Hassan and Dr. Albadawi Abdellbagi Talha, University of Gezira, Faculty of Medical Laboratory Sciences, Department of Clinical Chemistry who follow and support me in all stages of this dissertation.

Also, I’d like to thank my college Elham Awad and all staff in Omer Elhag Musa Center for Kidney Diseases and Surgery Rufaa.

Our thanks are also extended to the all staff in Gezira Hospital for Renal Disease and Surgery.

Finally I never forget all staff in clinical chemistry department, Collage and Post graduate studies department.
Estimation of Urea and Creatinine Pre and Post Dialysis in Patients with Chronic Renal Failure, Gezira Hospital for Renal Disease and Surgery, Gezira State, Sudan

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Abstract

Chronic kidney disease is an irreversible loss of renal function for at least three months. It is accompanied by profound changes in urea and creatinine levels in the blood. These changes contribute to serious complications such as glomerulonephritis, hypertension, diabetes mellitus and other inherited diseases such as polycystic kidney disease. This study aims to estimate blood urea and serum creatinine levels in patients with chronic renal failure pre and post dialysis to evaluate the efficacy of dialysis among these patients. This study was carried out during the period of February 2015 to April 2015 in Gezira State in units of dialysis (Gezira Hospital for Renal Disease and Surgery) and (Omer Elhag Musa Center for Kidney Diseases and Surgery Rufaa). 100 patients were selected of chronic renal failure their age range between 21-71 years. Two samples were collected from each patient; first sample is selected before dialysis and the other after dialysis in lithium heparin container to measure level of urea and creatinine to determine the efficacy of dialysis on urea and creatinine. Serum urea and serum creatinine level were measured using Mindray BS200 Chemistry Auto Analyzer. This study showed significant decrease in urea and creatinine level after dialysis and showed that level of urea is more decreased after dialysis compared to creatinine, and from this study hemodialysis is a preferred technique to correct urea and creatinine in chronic renal failure. During the study it was also observed that Chronic Kidney Disease is more common in male than in female. People between 40 to 60 years are more affected with
CKD. More studies were recommended to confirm these results and screening program was also recommended to reduce the burden and to prevent the complication of chronic kidney disease. Estimation of urea and creatinine pre and post dialysis in renal failure patients was highly recommended as routine test.
قياس مستوى البولينا والكرياتينين قبل وبعد الغسيل الكلوي لمرضى الفشل الكلوي المزمن

مستشفى جراحة الكلى، ولاية الجزيرة، السودان

عزام ميرغني السيد الماحي

ملخص الدراسة

الفشل الكلوي المزمن هو عبارة عن فقدان مستمر وغير رجعي لوظائف الكلى لفترة لا تقل عن ثلاثة أشهر مما يؤثر هذا الفقدان على مستوى البولينا والكرياتينين في الدم، هذه التغيرات في مستوي البولينا والكرياتينين قد تسبب في حدوث مضاعفات خطيرة جدا مثل أمراض الالتهاب الكلوي وإمراض ضغط الدم ومرض السكري وداء الكلى المتعدد الكيسات. هدفت هذه الدراسة إلى قياس مستوى معدلي اليوريا والكرياتينين عند المرضى ذوي الفشل الكلوي المزمن قبل وبعد عملية الغسيل الدموي يوم كفاءة الغسيل الدموي عند هؤلاء المرضى. أجريت هذه الدراسة في الفترة من فبراير 2015 حتى أبريل 2015 في ولاية الجزيرة في مراكز غسيل الكلى (مستشفى الجزيرة لأمراض وجراحه الكلي بود مدنى) ومركز عمر الحاج موسى لأمراض وجراحه الكلى برفاعه على الاشخاص الذين يعانون من مرض الفشل الكلوي المزمن الذين تتراوح أعمارهم بين (21-70) سنة. تم اختيار 100 مريض مصاب بالفشل الكلوي المزمن وتم قياس مستوى البولينا والكرياتينين وذلك بعد أن تم جمع عينتين من كل مريض واحد قبل الفحص الكلوي والثانية بعدة في حاوية الليثيوم هبارين وذلك لتحديد كفاءة الغسيل الكلوي ومدى تأثيره على معدلي البولينا والكرياتينين. تم قياس مستوى البولينا والكرياتينين معمليا باستخدام جهاز التحليل الكيميائي ميندراي بي اس 200. هذه الدراسة أوضحت أن هناك نقصان مؤثر في معدلي البولينا والكرياتينين بعد الغسيل الكلوي وأوضحت أيضا أن مستوى البولينا أكثر نقصان من مستوى الكرياتينين بعد الغسيل الكلوي. ومن هذه الدراسة أيضا نرى أن الغسيل الدموي هو أفضل علاج لتصحيح معدلي البولينا والكرياتينين عند المرضى ذوي الفشل الكلوي المزمن كما أضحى أيضا من خلال هذه الدراسة أن معظم مرضى الفشل الكلوي المزمن غالبهم من الرجال مباشرة مع النساء ووجدنا أن غالبية المرضى المصابون ينتمون إلى هذا المرض في الاعمار المتوسطة (40-60 سنة). اوصت هذه الدراسة بإجراء عدد من الدراسات حول هذا الموضوع لتاكيد النتائج هذه الدراسة، كما أوصت إجراء فحوصات ومتابعة للمرضى لمنع حدوث مضاعفات، كما أوصت بإجراء فحصي البولينا والكرياتينين قبل وبعد الغسيل الدموي كفحص روتيني لكل المرضى في مركز الغسيل.
# Table of contents

<table>
<thead>
<tr>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supervision Committee</td>
<td>I</td>
</tr>
<tr>
<td>Examining Committee</td>
<td>II</td>
</tr>
<tr>
<td>Dedication</td>
<td>III</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>IV</td>
</tr>
<tr>
<td>Abstract in English</td>
<td>V</td>
</tr>
<tr>
<td>Abstract in Arabic</td>
<td>VI</td>
</tr>
<tr>
<td>Table of content</td>
<td>VII</td>
</tr>
<tr>
<td>List of tables</td>
<td>X</td>
</tr>
<tr>
<td>List of figures</td>
<td>XI</td>
</tr>
<tr>
<td>List of abbreviation</td>
<td>XII</td>
</tr>
<tr>
<td>Chapter one: Introduction</td>
<td></td>
</tr>
<tr>
<td>Renal failure</td>
<td>1</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>1</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>2</td>
</tr>
<tr>
<td>Dialysis</td>
<td>2</td>
</tr>
<tr>
<td>Types of dialysis</td>
<td>3</td>
</tr>
<tr>
<td>Urea</td>
<td>3</td>
</tr>
<tr>
<td>Creatinine</td>
<td>3</td>
</tr>
<tr>
<td>Rational</td>
<td>4</td>
</tr>
<tr>
<td>Objectives</td>
<td>4</td>
</tr>
<tr>
<td>Chapter two: Literature review</td>
<td></td>
</tr>
<tr>
<td>The Kidney</td>
<td>5</td>
</tr>
<tr>
<td>Renal anatomy</td>
<td>5</td>
</tr>
<tr>
<td>Renal physiology</td>
<td>8</td>
</tr>
<tr>
<td>Glomerular filtrations</td>
<td>8</td>
</tr>
<tr>
<td>Tubular function</td>
<td>8</td>
</tr>
<tr>
<td>Proximal convoluted tubule</td>
<td>8</td>
</tr>
<tr>
<td>Loop of Henle</td>
<td>9</td>
</tr>
<tr>
<td>Countercurrent Multiplier System</td>
<td>9</td>
</tr>
<tr>
<td>Distal Convoluted Tubule</td>
<td>10</td>
</tr>
<tr>
<td>Anti-Diuretic Hormone</td>
<td>11</td>
</tr>
<tr>
<td>Aldosterone</td>
<td>11</td>
</tr>
<tr>
<td>Collecting Duct</td>
<td>11</td>
</tr>
<tr>
<td>Renal failure</td>
<td>13</td>
</tr>
<tr>
<td>Classification of renal failure</td>
<td>13</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>13</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>13</td>
</tr>
<tr>
<td>Signs and symptoms</td>
<td>14</td>
</tr>
<tr>
<td>Treatment</td>
<td>15</td>
</tr>
<tr>
<td>Dialysis</td>
<td>15</td>
</tr>
<tr>
<td>History</td>
<td>15</td>
</tr>
<tr>
<td>Principle</td>
<td>16</td>
</tr>
<tr>
<td>Types</td>
<td>16</td>
</tr>
<tr>
<td>Hemodialysis</td>
<td>16</td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
<td>17</td>
</tr>
<tr>
<td>Laboratory diagnosis</td>
<td>18</td>
</tr>
<tr>
<td>Urea</td>
<td>18</td>
</tr>
<tr>
<td>Biochemistry</td>
<td>18</td>
</tr>
<tr>
<td>Clinical application</td>
<td>18</td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>19</td>
</tr>
<tr>
<td>Creatinine</td>
<td>19</td>
</tr>
<tr>
<td>Biochemistry</td>
<td>19</td>
</tr>
<tr>
<td>Clinical application</td>
<td>20</td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>20</td>
</tr>
<tr>
<td>Chapter three: Material and methods</td>
<td></td>
</tr>
<tr>
<td>Study area and study population</td>
<td>21</td>
</tr>
<tr>
<td>Study design</td>
<td>21</td>
</tr>
<tr>
<td>Sample size</td>
<td>21</td>
</tr>
<tr>
<td>Data collection</td>
<td>21</td>
</tr>
<tr>
<td>Ethical approval</td>
<td>22</td>
</tr>
<tr>
<td>Materials and equipment</td>
<td>22</td>
</tr>
<tr>
<td>Blood sampling and Collection</td>
<td>22</td>
</tr>
<tr>
<td>Methods</td>
<td>23</td>
</tr>
<tr>
<td>Urea</td>
<td>23</td>
</tr>
<tr>
<td>Creatinine</td>
<td>23</td>
</tr>
<tr>
<td>Statistical analysis</td>
<td>24</td>
</tr>
<tr>
<td>Chapter four: Result and Discussion</td>
<td>25</td>
</tr>
<tr>
<td>Results</td>
<td>25</td>
</tr>
<tr>
<td>Discussion</td>
<td>32</td>
</tr>
<tr>
<td>Conclusion</td>
<td>33</td>
</tr>
<tr>
<td>Recommendation</td>
<td>33</td>
</tr>
<tr>
<td>References</td>
<td>34</td>
</tr>
<tr>
<td>Appendixes</td>
<td>37</td>
</tr>
</tbody>
</table>
## List of tables

<table>
<thead>
<tr>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.1 Procedure of urea</td>
<td>23</td>
</tr>
<tr>
<td>3.2 Procedure of creatinine</td>
<td>24</td>
</tr>
<tr>
<td>Title</td>
<td>Page</td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>2.1 Cross Section of the Kidney</td>
<td>7</td>
</tr>
<tr>
<td>2.2 Components of the Nephron</td>
<td>12</td>
</tr>
<tr>
<td>4.1 Distribution of the study population according to gender</td>
<td>26</td>
</tr>
<tr>
<td>4.2 Distribution of the study population according to age groups</td>
<td>26</td>
</tr>
<tr>
<td>4.3 Distribution of the study population according to co morbidity</td>
<td>27</td>
</tr>
<tr>
<td>4.4 Pre-dialysis serum urea level in CKD patients</td>
<td>28</td>
</tr>
<tr>
<td>4.5 post-dialysis serum urea level in CKD patients</td>
<td>29</td>
</tr>
<tr>
<td>4.6 pre-dialysis serum creatinine level in CKD patients</td>
<td>30</td>
</tr>
<tr>
<td>4.7 post- dialysis serum creatinine level in CKD patients</td>
<td>31</td>
</tr>
</tbody>
</table>
### List of abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADH</td>
<td>Anti-Diuretic Hormone</td>
</tr>
<tr>
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</tr>
<tr>
<td>BUN</td>
<td>Blood Urea Nitrogen</td>
</tr>
<tr>
<td>CKD</td>
<td>Chronic Kidney Disease</td>
</tr>
<tr>
<td>DM</td>
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</tr>
<tr>
<td>GDH</td>
<td>Glutamate De Hydrogenates</td>
</tr>
<tr>
<td>GFR</td>
<td>Glomerular Filtration Rate</td>
</tr>
<tr>
<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>KDIGO</td>
<td>Kidney Disease Improving Global Outcomes</td>
</tr>
<tr>
<td>NAD</td>
<td>Nicotine amide Adenine Dinucleotide</td>
</tr>
<tr>
<td>NADH</td>
<td>Nicotine amide Dinucleotide de Hydrogenates</td>
</tr>
<tr>
<td>NPN</td>
<td>Non Protein Nitrogen</td>
</tr>
<tr>
<td>SPSS</td>
<td>Statistical Package for the Social Sciences</td>
</tr>
</tbody>
</table>
CHAPTER ONE

INTRODUCTION

1-1 Renal failure:

Renal failure is a condition in which the kidneys fail to remove metabolic end-products from the blood and regulate the fluid, electrolyte, and pH balance of the extracellular fluids. The underlying cause may be renal disease, systemic disease, or urologic defects of non-renal origin. Renal failure can occur as an acute or a chronic disorder. Acute renal failure is abrupt in onset and often is reversible if recognized early and treated appropriately. In contrast, chronic renal failure is the end result of irreparable damage to the kidneys. It develops slowly, usually over the course of a number of years (Levy and Viscose, 1996).

1-1-1 Acute Renal Failure:

Acute renal failure (ARF) represents a rapid decline in renal function sufficient to increase blood levels of nitrogenous wastes and impair fluid and electrolyte balance. It is a common threat to seriously ill persons in intensive care units, with a mortality rate ranging from 42% to 88% (Levy and Viscose, 1996). Although treatment methods such as dialysis and renal replacement methods are effective in correcting life-threatening fluid and electrolyte disorders, the mortality rate associated with acute renal failure has not changed substantially since the 1960s (Thadhani et al, 1996; Brady et al, 2000). This probably is because acute renal failure is seen more often in older persons than before, and because it frequently is superimposed on other life-threatening conditions, such as trauma, shock, and sepsis. The most common indicator of acute renal failure is azotemia, an accumulation of nitrogenous wastes (urea nitrogen, uric acid, and creatinine) in the blood. In acute renal failure the glomerular filtration rate (GFR) is decreased. As a result, excretion of nitrogenous wastes is reduced and fluid and electrolyte balance cannot be maintained. Persons with acute renal failure often are asymptomatic, and the condition is diagnosed by observation of elevations in blood urea nitrogen (BUN) and creatinine (Brady et al, 2000).
1-1-2 Chronic Renal Failure:

Unlike acute renal failure, chronic renal failure represents progressive and irreversible destruction of kidney structures. As recently as 1965, many patients with chronic renal failure progressed to the final stages of the disease and then died. The high mortality rate was associated with limitations in the treatment of renal disease and with the tremendous cost of ongoing treatment (Rettig, 1996). In 1972, federal support began for dialysis and transplantation through a Medicare entitlement program. Technologic advances in renal replacement therapy (i.e., dialysis therapy and transplantation) have improved the outcomes for persons with renal failure. In the United States, there are approximately 400,000 persons with end-stage renal disease who are living today, a product of continued research and advances in treatment methods (National Kidney and Urological Information Center, 2001).

Chronic renal failure can result from a number of conditions that cause permanent loss of nephrons, including diabetes, hypertension, glomerulonephritis, and polycystic kidney disease. Typically, the signs and symptoms of renal failure occur gradually and do not become evident until the disease is far advanced. This is because of the amazing compensatory ability of the kidneys. As kidney structures are destroyed, the remaining nephrons undergo structural and functional hypertrophy, each increasing its function as a means of compensating for those that have been lost.

It is only when the few remaining nephrons are destroyed that the manifestations of renal failure become evident (Cotran et.al, 1999).

1.2 Dialysis:

In medicine, dialysis (from Greek dialusis ,meaning dissolution ,dia meaning through, and lysis ,meaning loosening or splitting) is a process for removing waste and excess water from the blood ,and is used primarily as an artificial replacement for lost kidney function in people with kidney failure(Pendse et.al,2008 ).
1.2.1 Types of Dialysis:

1.2.1.1 Hemodialysis (HD)
Blood is filtered outside the body. Hemodialysis removes wastes and excess fluid outside your body. During a hemodialysis treatment, blood is removed from your body and pumped by a machine through a Dialyzer. The dialyzer is the semipermeable membrane that cleans your blood.

1.2.1.2 Peritoneal dialysis (PD)
Blood is cleaned inside the body. Peritoneal dialysis cleans your blood and removes extra fluids using one of your body’s own membranes, the peritoneal membrane, as the filter. The peritoneal membrane is the lining that surrounds the peritoneum or abdominal cavity, which contains your stomach, spleen, liver and intestines.

1.3 Urea:
The Non Protein Nitrogenous (NPN) compound present in highest concentration in the blood is urea. Urea is the major excretory product of protein metabolism. It is formed in the liver from amino group (-NH₂) and free ammonia generated during protein catabolism (Bishop et.al, 2008)

1.4 Creatinine:
Creatine is synthesized primarily in the liver from arginine, glycine, and methionine. It is then transported to other tissues such as muscle, where it is converted to creatine phosphate, which serves as a high-energy source. Creatine phosphate loses phosphoric acid and creatine loses water to form creatinine, which diffuses into the plasma and is excreted in the urine (Bishop et.al, 2008).
1.5 Rational:
chronic renal diseases now account for the majority of global morbidity and mortality, rather than infectious disease, dialysis has a number of important commercial and industrial application and play a crucial role maintaining the health of human, dialysis is very important treatment in patients with kidney disease that can help purify the blood and remove waste.

1.6 Objectives:

1.6.1 General objective
To evaluate the efficacy of hemodialysis in patients with chronic renal failure in correcting blood urea and serum creatinine.

1.6.2 Specific objectives
- To estimate blood urea pre and post dialysis in patients with chronic renal failure.
- To estimate serum creatinine level pre and post dialysis in patients with chronic renal failure.
CHAPTER TWO

LITERATURE REVIEW

2.1 The kidneys:
The kidneys are vital organs that perform a variety of important functions. The most prominent functions are removal of unwanted substances from plasma (both waste and surplus), homeostasis (maintenance of equilibrium) of the body’s water, electrolyte and acid-base status, and participation in hormonal regulation. In the clinical laboratory, kidney function tests are used in assessment of renal disease, water balance, and acid-base disorders and in situations of trauma, head injury, surgery, and infectious disease (Bishop et al., 2008).

2.2 Renal anatomy:
The kidneys are paired, bean-shaped organs located retro peritoneal on either side of the spinal column. Macroscopically, a fibrous capsule of connective tissue encloses each kidney. When dissected longitudinally, two regions can be clearly discerned—an outer region called the cortex and an inner region called the medulla. The pelvis can also be seen (figure 2.1). It is a basin like cavity at the upper end of the ureter into which newly formed urine passes. The bilateral ureters are thick-walled canals, connecting the kidneys to the urinary bladder. Urine is temporarily stored in the bladder until voided from the body by way of the urethra. Nephrons is the functional units of the kidney that can only be seen microscopically. Each kidney contains approximately 1 million nephrons. Each nephron is a complex apparatus comprised of five basic parts:

- The glomerulus—a capillary tuft surrounded by the expanded end of a renal tubule known as Bowman’s capsule. Each glomerulus is supplied by an afferent arteriole

Carrying the blood in and an efferent arteriole carrying the blood out. The efferent arteriole branches into per tubular capillaries that supply the tubule.
■ The proximal convoluted tubule—located in the cortex.

■ The long loop of Henle—composed of the thin descending limb, which spans the medulla, and the ascending limb, which is located in both the medulla and the cortex, composed of a region that is thin and then thick.

■ The distal convoluted tubule—located in the cortex.

■ The collecting duct—formed by two or more distal convoluted tubules as they pass back down through the cortex and the medulla to collect the urine that drains from each nephron. Collecting ducts eventually merge and empty their contents into the renal pelvis (Bishop et.al, 2008).
Figure (2.1): Cross Section of the Kidney (Bishop et.al, 2008).
2.3 Renal physiology:

There are three basic renal system functions: glomerular filtration, tubular secretion, and tubular reabsorption (Bishop et al, 2008).

2.3.1 Glomerular filtrations:

The glomerulus is the first part of the nephron and functions to filter incoming blood. Several factors facilitate filtration. One factor is the unusually high pressure in the glomerular capillaries, which is a result of their position between two arterioles. This sets up a steep pressure difference across the walls. Another factor is the semipermeable glomerular basement membrane, which has a molecular size cutoff value of approximately 66,000 Da, about the molecular size of albumin. This means that water, electrolytes, and small dissolved solutes, such as glucose, amino acids, low-molecular-weight proteins, urea, and creatinine, pass freely through the basement membrane and enter the proximal convoluted tubule. Other blood constituents, such as albumin; many plasma proteins; cellular elements; and protein-bound substances, such as lipids and bilirubin, are too large to be filtered. In addition, because the basement membrane is negatively charged, negatively charged molecules, such as proteins, are repelled. Of the 1200–1500 mL of blood that the kidneys receive each minute (approximately one quarter of the total cardiac output), the glomerulus filters out 125–130 mL of an essentially protein-free, cell-free fluid, called glomerular filtrate. The volume of blood filtered per minute is the glomerular filtration rate (GFR), and its determination is essential in evaluating renal function (Bishop et al, 2008).

2.3.2 Tubular function:

2.3.2.1 Proximal convoluted tubule:

The proximal tubule is the next part of the nephron to receive the now cell-free and essentially protein-free blood. This filtrate contains waste products, which are toxic to the body above a certain concentration, and substances that are valuable to the body. One function of the proximal tubule is to return the bulk of each valuable substance back to
the blood circulation. Thus, 75% of the water, sodium, and chloride; 100% of the glucose (up to the renal threshold); almost all of the amino acids, vitamins, and proteins; and varying amounts of urea, uric acid, and ions, such as magnesium, calcium, potassium, and bicarbonate, are reabsorbed. Almost all (98%–100%) of uric acid, a waste product, is actively reabsorbed, only to be secreted at the distal end of the proximal tubule. When the substances move from the tubular lumen to the peritubular capillary plasma, the process is called tubular reabsorption. With the exception of water and chloride ions, the process is active; that is, the tubular epithelial cells use energy to bind and transport the substances across the plasma membrane to the blood. The transport processes that are involved normally have sufficient reserve for efficient reabsorption, but they are saturable. When the concentration of the filtered substance exceeds the capacity of the transport system, the substance is then excreted in the urine. The plasma concentration above which the substance appears in urine is known as the renal threshold, and its determination is useful in assessing both tubular function and nonrenal disease states. A renal threshold does not exist for water because it is always transported passively through diffusion down a concentration gradient. A second function of the proximal tubule is to secrete products of kidney tubular cell metabolism, such as hydrogen ions, and drugs, such as penicillin. The term tubular secretion is used in two ways: (1) tubular secretion describes the movement of substances from peritubular capillary plasma to the tubular lumen, and (2) tubular secretion also describes when tubule cells secrete products of their own cellular metabolism into the filtrate in the tubular lumen. Transport across the membrane of the cell is again either active or passive (Bishop et al, 2008).

2.3.2.2 Loop of Henle

2.3.2.2.1 Countercurrent Multiplier System

The osmolality in the medulla in this portion of the nephron increases steadily from the corticomedullary junction inward and facilitates the reabsorption of water, sodium, and chloride. The hyperosmolality that develops in the medulla is continuously maintained by the loop of Henle, a hairpin-like loop between the proximal tubule and the distal convoluted tubule. The opposing flows in the loop, the downward flow in the descending
limb, and the upward flow in the ascending limb, is termed a countercurrent flow. To understand how the hyperosmolality is maintained in the medulla, it is best to look first at what happens in the ascending limb. Sodium and chloride are actively and passively reabsorbed into the medulla interstitial fluid along the entire length of the ascending limb. Because the ascending limb is relatively impermeable to water, little water follows and the medulla interstitial fluid becomes hyperosmotic compared with the fluid in the ascending limb. The fluid in the ascending limb becomes hypotonic or dilute as sodium and chloride ions are reabsorbed without the loss of water, so the ascending limb is often called the diluting segment. The descending limb, in contrast to the ascending limb, is highly permeable to water and does not reabsorb sodium and chloride. The high osmolality of the surrounding interstitial medulla fluid is the physical force that accelerates the reabsorption of water from the filtrate in the descending limb. Interstitial hyperosmolality is maintained because the ascending limb continues to pump sodium and chloride ions into it. This interaction of water leaving the descending loop and sodium and chloride leaving the ascending loop to maintain a high osmolality within the kidney medulla produces hypoosmolal urine as it leaves the loop. This process is called the countercurrent multiplier system (Bishop et al, 2008).

2.3.2.3 Distal Convoluted Tubule:

The distal convoluted tubule is much shorter than the proximal tubule, with two or three coils that connect to a collecting duct. The filtrate entering this section of the nephron is close to its final composition. About 95% of the sodium and chloride ions and 90% of water have already been reabsorbed from the original glomerular filtrate. The function of the distal tubule is to effect small adjustments to achieve electrolyte and acid-base homeostasis. These adjustments occur under the hormonal control of both antidiuretic hormone (ADH) and aldosterone.
2.3.2.4 Anti Diuretic Hormone (ADH):

ADH is a peptide hormone secreted by the posterior pituitary, mainly in response to increased blood osmolality; ADH is also released when blood volume decreases by more than 5%–10%. Large decreases of blood volume will stimulate ADH secretion even when plasma osmolality is decreased. ADH stimulates water reabsorption.

The walls of the distal collecting tubules are normally impermeable to water (like the ascending loop of Henle), but they become permeable to water in ADH.

Water diffuses passively from the lumen of the tubules, resulting in more concentrated urine and decreased plasma osmolality (Bishop et.al, 2008).

2.3.2.5 Aldosterone:

This hormone is produced by the adrenal cortex under the influence of the renin–angiotensin mechanism. Its secretion is triggered by decreased blood flow or blood pressure in the afferent renal arteriole and by decreased plasma sodium. Aldosterone stimulates sodium reabsorption in the distal tubules and potassium and hydrogen ion secretion. Hydrogen ion secretion is linked to bicarbonate regeneration and ammonia secretion, which also occur here. In addition to these ions, small amounts of chloride ions are reabsorbed (Bishop et.al, 2008).

2.3.2.6 Collecting Duct:

The collecting ducts are the final site for either concentrating or diluting urine. The hormones ADH and aldosterone act on this segment of the nephron to control reabsorption of water and sodium. Chloride and urea are also reabsorbed here. Urea plays an important role in maintaining the hyper osmolality of the renal medulla.
Because the collecting ducts in the medulla are highly permeable to urea, urea diffuses down its concentration gradient out of the tubule and into the medulla interstitium, increasing its osmolality (Bishop et.al, 2008).

Figure (2.2): Components of the Nephron (Bishop et.al, 2008).
2.4 Renal failure:

Also (kidney failure or renal insufficiency) is a medical condition in which the kidneys fail to adequately filter waste products from the blood (Medline Plus, 2012).

Renal failure is mainly determined by a decrease in glomerular filtration rate, the rate at which blood is filtered in the glomeruli of the kidney. This is detected by a decrease in or absence of urine production or determination of waste product (urea and creatinine) in the blood (Liao, et.al, 2012).

2.4.1 Classification of renal failure:

2.4.1.1 Acute renal failure:

Is a rapidly progressive loss of renal function (Medical Encyclopedia, 2012), generally characterized by oliguria (decrease urine production, quantified as less than 400 ml per day in adults) and fluid and electrolyte imbalance (Klahr, et.al, 1998).

The causes of acute kidney injury include accidents, injuries, or complications from surgeries in which the kidneys are deprived of normal blood flow for extended periods of time. Heart bypass surgery is an example of one such procedure. Drug overdoses, accidental or from chemical overloads of drugs such as antibiotics or chemotherapy, may also cause the onset of acute kidney injury. Unlike chronic kidney disease, however, the kidneys can often recover from acute kidney injury, allowing the patient to resume a normal life. People suffering from acute kidney injury require supportive treatment until their kidneys recover function, and they often remain at increased risk of developing future kidney failure (Nkudic, 2012).

2.4.1.2 Chronic renal failure:

Unlike acute renal failure, chronic renal failure is a progressive loss in renal function over a period of months or years. The symptoms of worsening kidney function are not specific, and might include feeling generally unwell and experiencing a reduced appetite (national kidney foundation, 2002).
In chronic renal failure there is a steady and continued decrease in renal clearance or glomerular filtration rate (GFR), which leads to the gathering of urea, creatinine and other chemicals in the blood. According to the Kidney Disease Improving Global Outcomes (KDIGO) declaration GFR of less than 60 mL/minute/1.73 m2 is the indication of CKD. KDIGO additional classified the CKD in different stages which are: GFR 30 to 60 mL/minute as stage three; GFR 15 to 30 mL/minute as stage four; and GFR less than 15 mL/minute as stage five of CKD (Levey et.al, 2005). In stage five level of serum creatinine is greater than 5.0 mg/dl in men, and greater than 4.0 mg/dl in women (Couchoud et.al, 1999).

The most common causes of CKD are diabetes mellitus and long-term, uncontrolled hypertension (Kes et.al.2011) Polycystic kidney disease is another well known cause of CKD. Overuse of common drugs such as aspirin, ibuprofen, and acetaminophen (paracetamol) can also cause chronic kidney damage (Perneger, et.al.1994). Some infectious diseases, such as hantavirus, can attack the kidneys, causing kidney failure(Appel, et.al.2012).

2.4.2 Signs and symptoms:

Symptoms can vary from person to person. Someone in early stage kidney disease may not feel sick. When kidneys fail to filter properly, waste accumulates in the blood and the body, a condition called azotemia. Very low levels of azotaemia may produce few, if any, symptoms. If the disease progresses, symptoms become noticeable (if the failure is of sufficient degree to cause symptoms). Renal failure accompanied by noticeable symptoms is termed uraemia (Grinsted, 2005).

Symptoms of kidney failure include the following:

- High levels of urea in the blood, which can result in: Vomiting and/or diarrhea, which may lead to dehydration, Nausea, Weight loss, Nocturnal urination and Blood in the urine (Amgen Inc.2009).

A buildup of phosphates in the blood that diseased kidneys cannot filter out may cause Itching, Bone damage and Muscle cramps (caused by low levels of calcium which can be associated with hyper phosphatemia (Dr Andy Stein 2007).
Failure of kidneys to remove excess fluid may cause swelling of the legs, ankles, feet, face and/or hands, Shortness of breath due to extra fluid on the lungs (may also be caused by anemia) and Polycystic kidney disease.

Healthy kidneys produce the hormone erythropoietin that stimulates the bone marrow to make oxygen-carrying red blood cells. As the kidneys fail, they produce less erythropoietin, resulting in decreased production of red blood cells to replace the natural breakdown of old red blood cells. As a result, the blood carries less hemoglobin, a condition known as anemia. This can result in: Feeling tired and/or weak, Memory problems, Difficulty concentrating, Dizziness and Low blood pressure (The PD Companion et.al, 2008).

**Other symptoms include:**
Appetite loss, a bad taste in the mouth, Difficulty sleeping, Darkening of the skin, Excess protein in the blood (Katzung and Bertram, 2007).

2.4.3 Treatment:

The two types of treatment for kidney failure are dialysis or transplantation (Bishop et.al, 2008).

2.5 Dialysis:

In medicine, dialysis (from greek dialusis, meaning dissolution, dia, meaning through, and lysis, meaning loosening or splitting) is a process for removing waste and excess water from the blood, and is used primarily as an artificial replacement for lost kidney function in people with kidney failure (Pendse et.al, 2008).

2.5.1 History:

Dialysis as we know it has its roots in the 20 century. The 1940s: Inspiration, war and progress Dr. Willem Kolff is considered the father of dialysis. This young Dutch physician constructed the first dialyzer (artificial kidney) in 1943. The road to Kolff’s creation of an artificial kidney began in the late 1930s when he was working in a small ward at the University of Groningen Hospital in the Netherlands. There, Kolff watched helplessly as a young man died slowly of kidney failure. Kolff decided to find a way to make a machine that would do the work of the kidneys. A Dutch physician, Willem...
Johan Kolff, constructed the first working dialyzer in 1943 during the Nazi occupation of the Netherlands. Due to the scarcity of available resources, Kolff had to improvise and build the initial machine using sausage casings, beverage cans, a washing machine, and various other items that were available at the time. Over the following two years, [1943-1945] Kolff used his machine to treat 16 patients suffering from acute kidney failure, but the results were unsuccessful. Then, in 1945, a 67-year-old comatose woman regained consciousness following 11 hours of hemodialysis with the dialyzer, and lived for another seven years before dying from an unrelated condition. She was the first-ever patient successfully treated with dialysis (Kolff, 2009).

Thanks to the efforts of Kolff and Scribner and other medical pioneers like them, people with chronic kidney disease are now able to live full and productive lives.

**2.5.2 Principle:**

Dialysis works on the principles of the diffusion of solutes and ultrafiltration of fluid across a semi-permeable membrane. Diffusion is a property of substances in water; substances in water tend to move from an area of high concentration to an area of low concentration. Blood flows by one side of a semi-permeable membrane, and a dialysate, or special dialysis fluid, flows by the opposite side. A semipermeable membrane is a thin layer of material that contains holes of various sizes, or pores. Smaller solutes and fluid pass through the membrane, but the membrane blocks the passage of larger substances (for example, red blood cells, large proteins). This replicates the filtering process that takes place in the kidneys, when the blood enters the kidneys and the larger substances are separated from the smaller ones in the glomerulus (Louis et.al, 2006)

**2.5.3 Types:**

The two main types of dialysis, hemodialysis and peritoneal dialysis, remove wastes and excess water from the blood in different ways (Pendse, et.al, 2008)

**2.5.3.1 Hemodialysis:**

Hemodialysis removes wastes and water by circulating blood outside the body through an external filter, called an dialyzer, that contains a semipermeable membrane. The blood flows in one direction and the dialysate flows in the opposite. The counter-current flow of
the blood and dialysate maximizes the concentration gradient of solutes between the blood and dialysate, which helps to remove more urea and creatinine from the blood.

In hemodialysis, the patient's blood is pumped through the blood compartment of a dialyzer, exposing it to a partially permeable membrane. The dialyzer is composed of thousands of tiny hollow synthetic fibers. The fiber wall acts as the semipermeable membrane. Blood flows through the fibers, dialysis solution flows around the outside of the fibers, and water and wastes move between these two solutions (Ahmad et.al, 2008).

The cleansed blood is then returned via the circuit back to the body. Ultrafiltration occurs by increasing the hydrostatic pressure across the dialyzer membrane. This usually is done by applying a negative pressure to the dialysate compartment of the dialyzer. This pressure gradient causes water and dissolved solutes to move from blood to dialysate, and allows the removal of several liters of excess fluid during a typical 4-hour treatment. In the United States, hemodialysis treatments are typically given in a dialysis center three times per week (due in the United States to Medicare reimbursement rules) (Rocco, 2007).

2.5.3.2 Peritoneal dialysis:

In peritoneal dialysis, wastes and water are removed from the blood inside the body using the peritoneum as a natural semipermeable membrane. Wastes and excess water move from the blood, across the peritoneal membrane, and into a special dialysis solution, called dialysate, in the abdominal cavity (Pendse et.al, 2008).

In peritoneal dialysis, a sterile solution containing glucose (called dialysate) is run through a tube into the peritoneal cavity, the abdominal body cavity around the intestine, where the peritoneal membrane acts as a partially permeable membrane. The peritoneal membrane or peritoneum is a layer of tissue containing blood vessels that lines and surrounds the peritoneal, or abdominal, cavity and the internal abdominal organs (stomach, spleen, liver, and intestines) (Blake P et.al,2008 ). Diffusion and osmosis drive waste products and excess fluid through the peritoneum into the dialysate until the dialysate approaches equilibrium with the body's fluids. Then the dialysate is drained, discarded, and replaced with fresh dialysate (Louis et.al, 2005).
2.6 Laboratory diagnosis:

2.6.1 Urea:

The Non Protein Nitrogenous (NPN) compound present in highest concentration in the blood is urea. Urea is the major excretory product of protein metabolism. It is formed in the liver from amino group (-NH\textsubscript{2}) and free ammonia generated during protein catabolism (Bishop et.al., 2008).

2.6.1.1 Biochemistry:

Protein metabolism produces amino acids that can be oxidized to produce energy or stored as fat and glycogen. These processes release nitrogen which is converted to urea and excreted as a waste product. Following synthesis in the liver, urea is carried in the blood to the kidney, where it is readily filtered from the plasma by the glomerulus. Most of the urea in the glomerular filtrate is excreted in the urine, although some urea is reabsorbed by passive diffusion during passage of the filtrate through the renal tubules. The amount reabsorbed depends on the urine flow rate and extent of hydration. Small quantities of urea (<10% of the total) are excreted through the gastrointestinal tract and skin. The concentration of urea in the plasma is determined by the protein content of the diet, the rate of protein catabolism, and renal function and perfusion (Bishop et.al., 2008).

2.6.1.2 Clinical application:

Measurement of urea is used to evaluate renal function, to assess hydration status, to determine nitrogen balance, to aid in the diagnosis of renal disease, and to verify adequacy of dialysis (Bishop et.al., 2008).

2.6.1.3 Pathophysiology:

An elevated concentration of urea in the blood is called azotemia. Very high plasma urea concentration accompanied by renal failure is called uremia or the uremic syndrome. This condition is eventually fatal if not treated by dialysis or transplantation.

Conditions causing increase plasma urea are classified according to the cause into three main categories: prerenal, renal and post renal (Bishop et.al, 2008).
Prerenal azotemia is a result of reduced renal blood flow. Less blood is delivered to the kidney; consequently, less urea is filtered. Causative factors include congestive heart failure, shock, hemorrhage and dehydration (Bishop et.al, 2008).

Decreased renal function causes an increase in plasma urea concentration as a result of compromised urea excretion. Renal causes of elevated urea include acute and chronic renal failure, glomerular nephritis and tubular necrosis. Post renal azotemia can be due to obstruction of urine flow anywhere in the urinary tract by renal calculi, tumors of the bladder or prostate or severe infection. The major causes of decreased plasma urea concentration include low protein intake and severe liver disease (Bishop et.al, 2008).

2.6.2 Creatinine:

Creatinine is formed from creatine and creatine phosphate in muscle and is excreted into the plasma at a constant rate related to muscle mass. It is commonly used to assess renal filtration function (Bishop et.al, 2008).

2.6.2.1 Biochemistry:

Creatine is synthesized primarily in the liver from arginine, glycine, and methionine. It is then transported to other tissues such as muscle, where it is converted to creatine phosphate, which serves as a high-energy source. Creatine phosphate loses phosphoric acid and creatine loses water to form creatinine, which diffuses into the plasma and is excreted in the urine (Bishop et.al, 2008).

2.6.2.2: Clinical application:

Measurement of creatinine concentration is used to determine the sufficiency of kidney function, to determine the severity of kidney damage and to monitor the progression of kidney disease (Bishop et.al, 2008).
2.6.2.3 Pathophysiology:

An elevated creatinine concentration is associated with abnormal renal function, especially as it relates to glomerular function. Plasma concentration of creatinine is inversely proportional to the clearance of creatinine. Therefore, when plasma creatinine concentration is elevated, GFR is decreased, indicating renal damage (Bishop et.al, 2008).
CHAPTER THREE
MATERIAL and METHODS

3.1 Study area and study population:
Gezira State is situated between Blue Nile and the White Nile in the East-Central Region of the country and is bordered to the North of Khartoum State; South Sennar State; East of the Gadarif State and West White Nile State. The area approximately is 25,549.2 square kilometers with population 3,796,000 inhabitants (sudan.gov.sd, 2012). The state contains seven localities and Wad Medani city is a capital of the State. This study was conducted in Gezira State in Gezira Hospital for Renal Disease and Surgery and Omer Elhag Musa Center for Kidney Diseases and Surgery Rufaa. The hospitals admitted different patients coming from all over Gezira state and other neighboring state namely Sennar and Gadarif.
The other location is Omer Elhag Mosa Center which admitted different patient from Rufaa city and neighboring villages.

3.2 Study design:
A hospital based cross-sectional study conducted in 100 patients with chronic renal failure.

3.3 Sample size:
The study includes 100 samples collected from chronic renal failure patients and selected randomly. 30 blood samples pre and post dialysis were taken from chronic renal failure patients in Gezira Hospital for Renal Disease and Surgery while the remaining samples about 70 samples were taken from Omer Elhag Musa Center for Kidney Diseases and Surgery Rufaa.

3.4 Data collection:
The data were collected by using a questionnaire. A questionnaire was designed to include all information needed.
3.5 Ethical approval:

The study proposal got an approval from the University of Gezira Faculty of Medical Lab. Sciences.

3.6 Materials and equipment:

The following materials and equipment’s were utilized in this study

- Mindary BS-200 ,chemistry analyzer cormay ,Poland serial number.8393/2008
- Cotton.
- Syringe with needle.
- Spirit swab.
- Gloves.
- Cuvette.
- Centrifuge (Model: TDL -4, Serial No. 08).
- Pipettes- 10 μl, 100 μl, 1000μl.
- Test - tubes 100 mm 12 mm.
- Anticoagulant (lithium heparin)

3.7 Blood Sampling and Collection:

For pre and post dialysis analysis in patient with chronic renal failure, blood of 100 patients was collected from Gezira Hospital for Renal Disease and Surgery and Omer Elhag Musa center for Kidney Diseases and Surgery Rufaa.

3 ml of the blood was obtained from each patient before and after dialysis.

After collection, placed the blood in lithium heparin tube, then centrifuge to separate serum and was used for the estimation of creatinine and urea.
3.8 Methods:

3.8.1 Urea:

3.8.1.1: principle:
Urea was measured by diacetyl monoxime colorimetric method and Berthelot reaction. In this method the urea is converted to ammonia by an enzyme called urease. The ammonia produced is combined with 2-oxoglutarate and NADH in the presence of Glutamate Dehydrogenase (GDH), which yields L-Glutamate and NAD. The decrease in NADH absorbance is proportional to the urea concentration.

Table (3.1) Procedure of urea:

<table>
<thead>
<tr>
<th>Volume</th>
<th>Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reagent1</td>
</tr>
<tr>
<td></td>
<td>Reagent2</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Times</td>
<td>10s</td>
</tr>
<tr>
<td>Filter</td>
<td>Main</td>
</tr>
<tr>
<td>Analytical type</td>
<td>kinetic</td>
</tr>
</tbody>
</table>

3.8.2: Creatinine:

3.8.2.1. Principle:
Creatinine was estimated by the Jaffe reaction, a calorimetric procedure in which creatinine forms a yellow orange complex in alkaline solution with picric acid. This colored complex is determined photometrically. The intensity of produced colored is directly proportional to the amount of creatinine in the sample.
Table (3.2) Procedure of creatinine:

<table>
<thead>
<tr>
<th>volume</th>
<th>Sample</th>
<th>25 μL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Reagent 1</td>
<td>200 μL</td>
</tr>
<tr>
<td></td>
<td>Reagent 2</td>
<td>50 μL</td>
</tr>
<tr>
<td>Time</td>
<td>3s</td>
<td></td>
</tr>
<tr>
<td>Filter</td>
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<td>510nm</td>
</tr>
<tr>
<td>Analytical type</td>
<td>Fixed time</td>
<td></td>
</tr>
</tbody>
</table>

3.9 Statistical analysis:

All data collected from chronic renal failure and the result analyzed by Statistical Package for the Social Sciences (SPSS) program (version 20).
CHAPTER FOUR
RESULTS AND DISCUSSION

4.1 Results:

4.1.1 Gender:

In 100 patient with chronic renal failure in this study, 72 were males and 28 were female. figure (4.1).

Figure (4.1): Distribution of the study population according to gender.
### 4.1.2 Age group:

The age group of the study population 21-30 (11%), 31-40 (19%), 41-50 (26%), 51-60 (31%), and more than 60 (13%).

![Figure (4.2): Distribution of the study population according to age groups.](image)
4.1.3 Co morbidity:

The co morbidity of the study population was hypertension and DM is account (40%), hypertension (20%), DM (20%) and others account (20%).

Figure (4.3): distribution of the study population according to co morbidity
4.1.4 Pre dialysis serum urea level:

In CKD patients, pre-dialysis serum urea level was significantly higher than normal range (20-40 mg/dl).

From 70-100 mg/dl (9%), 101-150 mg/dl (27%), 151-250 (54%) and more than 250 represent (10%).

**Figure (4.4):** Pre-dialysis serum urea level in CKD patients
4.1.5 Post dialysis serum urea level:

There was a clear reduction in urea levels after dialysis, in most of the patients it was reduced to 51-100mg/dl represent (63%), 1to 50 mg/dl represent (20%) ,151-200mg/dl represent (7%) and more than 200mg/dl represent (10%) (Figure 4.5).

![Figure 4.5](image.png)

**Figure (4.5):** post- dialysis serum urea level in CKD patients
4.1.6 Pre-dialysis Serum Creatinine:

Serum creatinine level was higher than normal range (up to 1.4 mg/dl) in CKD patients undergoing dialysis. Most of the patients have serum creatinine level between 7.6-12 mg/dl (51%), 12.1-15mg/dl (25%), 6.1-7.5mg/dl(17%) and more than 15mg/dl represent (7%) before dialysis.

Figure (4.6): pre-dialysis serum creatinine level in CKD patients
4.1.7 Post-dialysis Serum Creatinine:

Dialysis has positive impact on serum creatinine level and reduced its level towards normal value. In figure (10) show the results, most of the patient (52%) had serum creatinine level between 4.6-7.5mg/dl after dialysis, 1.6-4.5mg/dl represent (20%) and 7.6-10.5 (18%).

Factors like age, sex and physical status of person also effect serum creatinine level (Lascano M E and Poggio, 2010).

**Figure (4.7):** post-dialysis serum creatinine level in CKD patients
4.2 Discussion:

This study was carried out to estimate the levels of urea and creatinine pre and post dialysis in patients with chronic renal failure to study the efficacy of dialysis.

200 samples were collected from 100 patients, two blood samples collected from each patient one before dialysis and the other after dialysis, about 72 samples of them were collected from males and 28 samples were collected from females.

In this study, the mean of plasma urea pre dialysis was (178, 17 mg/dl) while after dialysis was (102, 78 mg/dl), and the mean of plasma creatinine pre dialysis was (10.06 mg/dl) while after dialysis was (6.26 mg/dl).

The chronic renal failure is more common in males (72%) than females (28%) may be because males are more exposed to risk factors and works in some hard jobs, and these findings agreed with Hida M et.al, 1985 and Noor et.al, 2014.

Chronic renal failure is more distributed in middle ages patients between 31-50 is most affected (57%) due to this age is more exposed to infection with diabetes mellitus and hypertention, this study agreed with Noor et.al, 2014.

In chronic renal failure pre dialysis serum urea level was significantly higher than normal range (20-40 mg/dl). Most of the patient (54%) had serum urea level between 151-250 mg/dl because the kidney is not properly work and then accumulate these waste product, this finding is agree with study Noor et.al, 2014.

After dialysis there was a clear reduction in blood urea level, in most of the patients it was reduced 51-150 mg/dl (63%) and 1 to 50 mg/dl (20%) due to eliminate the waste product by dialysis.

In chronic renal failure pre dialysis serum creatinine level was higher than normal range (up to 1.4 mg/dl). Most of the patients have serum creatinine level between 7, 6-12 mg/dl (51%) and 12, 1-15 mg/dl (25%) before the dialysis.

Dialysis has positive impact on serum creatinine level and reduced its level, most of the patients (72%) had serum creatinine level below 7, 5 mg/dl after the dialysis.
CHAPTER FIVE

Conclusion and Recommendation

5.1 Conclusion

In conclusion, the hemodialysis reduces the levels of blood urea and serum creatinine significantly.

Hemodialysis is a prefer technique to correct blood urea and serum creatinine in chronic renal failure patients.

People between 40 to 60 years were more affected with CKD.

Hypertension and diabetes mellitus are most common cause to chronic renal failure.

5.2 Recommendation:

Estimation the levels of blood urea and serum creatinine were recommended to be done pre and post dialysis in all patients on dialysis as routine test.

The affection of dialysis depends on many other factors which considered as important substance. Need more researches.

More studies are recommended to confirm these results.
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Noor ul Amin, Raja Tahir Mahmood* , M. Javaid Asad, Mudassar Zafar, and Asad Mehmood Raja, April 2014.
APPENDIX

University of Gezira
Faculty of medical laboratory sciences
Estimation of UREA AND CREATININE PRE AND POST DIALYSIS
IN PATIENTS with chronic renal failure

Questionnaire:
No of patient:………………
Name of patient:………..
Sex:
Male (…. )                      female(….)
Age:...........
Location:..........  
Duration of dialysis:……
Number time of dialysis /week:……...
Heart disease    Yes (  )    No (  )
Liver disease    Yes (  )    No (  )
Renal disease    Yes (  )    No (  )
Diabetes mellitus Yes (  )    No (  )
Hypertensive     Yes (  )    No (  )
Others:………………
Laboratory investigation:
Urea:
Pre dialysis (.....Mg/dl)
Post dialysis (.....Mg/dl)
Creatinine:
Pre dialysis (.....Mg/dl)
Post dialysis (.....Mg/dl)               Date:………………
Mindray BS-200 Chemistry Auto Analyzer